The University Of Sheffield.

The approach-avoidance systems across adolescence: Relationships with risk-taking and anxiety

Philippa Howsley

A thesis submitted in partial fulfilment of the requirements for the degree of Doctor of Philosophy

The University of Sheffield
Faculty of Science
Department of Psychology

October 2016

Abstract

Adolescents are more likely to engage in potentially harmful risk-taking behaviours and experience elevated anxiety levels than both children and adults. The simultaneous increase in risk-taking and anxiety during adolescence seems paradoxical given that high anxiety levels are associated with reduced risk-taking in adults. Despite this, many gaps remain in our current understanding of why risk-taking behaviours and anxiety levels simultaneously increase during adolescence. This doctoral work was therefore designed to provide novel insights into the simultaneous increase of risk-taking and anxiety during adolescence. Specifically, this doctoral work aimed to examine how the relationship between risk-taking and anxiety changes across the course of adolescence (Chapter 3), and whether age-related changes in the electroencephalography (EEG) correlates of the approach-avoidance systems can account for increases in risk-taking and anxiety during adolescence (Chapters 4 and 5). To these ends, a large cohort of male and female preadolescents (9-12 years), mid-adolescents (13-17 years), and late adolescents (18-23 years) participated in a series of behavioural and EEG studies. The first study (Chapter 3) found that the relationship between risk-taking and anxiety followed gender-specific developmental trajectories. Chapter 3 also revealed significant age- and gender-related changes in risk-taking and anxiety in this sample of adolescents. The second study (Chapter 4) used EEG to investigate whether age-related changes in reward- and threat-related anticipatory activity could partially account for the developmental differences in risk-taking and anxiety found in this sample of adolescents (Chapter 3). The findings suggested that adolescents have both reward- and threat-related anticipatory biases. Thus, these findings challenge current theories asserting that adolescents are hyporesponsive to threats. The final study (Chapter 5) used EEG to examine the development of spontaneous alpha and its relationship to risk-taking behaviours throughout adolescence. While alpha was not reliably associated with risk-taking, this study provides novel insights into the development of spontaneous alpha during adolescence.

Declaration

This thesis comprises original work that has not previously been presented for an award at this, or any other, university. The work presented in this thesis was completed by the candidate under the supervision of Dr Liat Levita and Professor Glenn Waller.

The following paper is based on the work contained in this thesis:

> Chapter 4: Howsley, P., & Levita, L. (under review). Anticipatory representations of reward and threat in perceptual areas from early to late adolescence.

Acknowledgements

This doctoral work would not have been possible without the wisdom and guidance of my primary supervisor, Liat Levita. I am very thankful for your mentorship and support over the last few years. I would like to extend my thanks to my secondary supervisor, Glenn Waller, for your support and insightful feedback. Thank you also to Liz Milne and Peter Totterdell for overseeing my progress and approving my confirmation review!

I am very grateful to the families and students who volunteered to take part in my research studies. Without their participation this research would not exist.

I would like to acknowledge the work of Anna Kolesnik, who assisted with data collection as part of the Sheffield Undergraduate Research Experience scheme. Thank you for your help and for being good company in the lab!

Above all else, I am incredibly thankful to my family for their overwhelming love and support. A special thank you to my husband, Gordon, for your endless love, patience, and encouragement. And for making my figures look so lovely!

Contents

List of figures

List of tables

Chapter 1

Literature review

1.1. Introduction

Adolescents are more likely than children and adults to engage in potentially harmful risky behaviours (Burnett, Bault, Coricelli & Blakemore, 2010; Steinberg *et al*., 2008) and experience elevated anxiety levels (Abe & Suzuki, 1986). High anxiety levels in adults are associated with reduced levels of risk-taking behaviours (Giorgetta *et al*., 2012; Maner *et al*., 2007), and therefore the simultaneous increase in risk-taking behaviours and anxiety levels during adolescence seems paradoxical. Several neurobiological models have been proposed to explain the increases in risk-taking behaviours during adolescence (Casey *et al*., 2010; Ernst, Pine & Hardin, 2006; Nelson, Leibenluft, McClure & Pine, 2005; Steinberg, 2008). Casey's neurobiological model also attempts to explain the increases in emotionality, i.e., greater levels of anxiety and negative affect, during adolescence (Casey *et al*., 2010). Despite this, many gaps remain in our current understanding of why risk-taking behaviours and anxiety levels simultaneously increase during adolescence. This doctoral work was therefore designed to provide novel insights into the simultaneous increase of risk-taking and anxiety during adolescence. Specifically, this doctoral work aimed to examine how the relationship between risk-taking and anxiety changes across the course of adolescence (Chapter 3), and whether age-related changes in the EEG correlates of the approach-avoidance systems can account for the development of risk-taking and anxiety during adolescence (Chapters 4 and 5). Notably, the same cohort of adolescents took part in all three studies included in this doctoral work in order to explore the simultaneous increase in risk-taking and anxiety from multiple behavioural and EEG perspectives.

This review first defines adolescence as a transitional period (Chapter 1.2) and outlines the evidence for the simultaneous increase in risk-taking and anxiety during adolescence (Chapter 1.3). Next, this review explores how the brain structurally changes during adolescence (Chapter 1.4), and critically evaluates the current neurobiological models of adolescence suggesting that the development of the approach-avoidance systems contributes to the increases in risk-taking and anxiety during adolescence (Chapter 1.5). The next section then discusses what is currently known about the approach-avoidance systems during adolescence (Chapter 1.6). In the penultimate section, this review discusses what is currently known about the EEG correlates of the approach-avoidance systems during adolescence, and how the EEG studies in this doctoral work extend the existing literature (Chapter 1.7). Finally, this review briefly summarises each of the following chapters (Chapter 1.8).

1.2. Adolescence: A transitional period

Adolescence typically begins around 10 years of age (World Health Organization, 2003), and extends beyond the teenage years well into the third decade of life (Dahl, 2004; Steinberg, 2008). Importantly, adolescence is not an isolated period in development, but a transitional phase that bridges the gap between childhood and adulthood. Therefore, to understand the discrete changes that occur during adolescence, it is critical that the transitions into and out of adolescence are examined (Casey, Getz & Galvan, 2008a; Spear, 2000).

Successful transitions between childhood and adulthood are crucial for individuals to become healthy and productive members of society (Arnett, 2001; Spear, 2000). While the majority of adolescents negotiate the transition between childhood and adulthood successfully, studies have consistently documented that adolescents engage in significantly more risk-taking behaviours and experience elevated levels of anxiety compared to both children and adults (Abe & Suzuki, 1986; Burnett *et al*., 2010; Steinberg *et al*., 2008). Notably, the observation that adolescents take more risks and experience greater levels of anxiety is not new; Hall's (1904) seminal work proposed that adolescence is a period of storm and stress.

1.3. The storm and stress of adolescence

1.3.1. The storm of adolescence: Increase in risk-taking behaviours

Hall (1904) coined the term 'storm of adolescence' to refer to the increase in risk-taking behaviours that is widely observed during adolescence. Risk-taking behaviours are behaviours that have high subjective desirability as well as high potential harm or loss (Irwin, 1990). Such behaviours include unprotected sex, illicit drug use, and delinquency (Arnett, 1992; Eaton *et al*., 20010; Irwin, 1990). A plethora of studies have found that risk-taking behaviours increase during the transition from childhood to adolescence, peak in adolescence, and subsequently decrease during the transition from adolescence to adulthood (Burnett *et al*., 2010; Donovan & Jessor, 1985; Eaton *et al*., 2010; Eshel, Nelson, Blair, Pine & Ernst, 2007; Figner, Mackinlay, Wilkening & Weber, 2009; Gullone, Moore, Moss & Boyd, 2000; Kann *et al*., 1999; Windle *et al*., 2008). Studies have also reported that males are more likely than females to engage in risky behaviours during all developmental stages (Byrnes, Miller & Schafer, 1999; Gullone *et al*., 2000; Turner & McClure, 2003; Van Leijenhorst, Westenberg & Crone, 2008).

The developmental changes in risk-taking behaviours are thought to largely result from age-related changes in sensation seeking and impulsivity (Steinberg *et al*., 2008). Sensation seeking refers to a high desire to seek out novel, varied, complex, and intense experiences and sensations, and a willingness to take risks for the sake of such experiences and sensations (Zuckerman, 1994). Like risk-taking behaviours, sensation seeking follows an inverted u-shaped developmental trajectory, whereby sensation-seeking increases from childhood to adolescence, peaks in adolescence, and decreases from adolescence to adulthood (Harden & Tucker-Drob, 2011; Shulman, Harden, Chein & Steinberg, 2014; Shulman, Harden, Chein & Steinberg, 2015; Steinberg *et al*., 2008; Zuckerman, Eysenck & Eysenck, 1978). Compared to sensation seeking and risk-taking behaviours, impulsivity refers to a lack of self-control, rapid decision-making, and a tendency to act without adequate thought (Moeller, Barratt, Dougherty, Schmitz & Swann, 2001; Steinberg *et al*., 2008). Several studies have reported that impulsivity decreases, as impulse control increases, throughout childhood and adolescence (Casey *et al*., 1997; Harden & Tucker-Drob, 2011; Shulman *et al*., 2014, 2015; Smith, Xiao & Bechara, 2012; Steinberg *et al*., 2008). The combination of elevated sensation seeking tendencies and immature self-regulatory capacities is thought to make adolescence a particularly vulnerable time for engaging in risky behaviours (Casey *et al*., 2010; Steinberg *et al*., 2008).

Risk-taking is a normal, even adaptive, part of adolescent development (Steinberg & Morris, 2001). Nevertheless, adolescent risk-taking can lead to serious short- and long-term negative consequences, including injuries, substance abuse, and sexually transmitted diseases (Casey, Jones & Hare, 2008b; Dahl, 2004; Eaton *et al*., 2010). Dahl (2004) proposed that there is a health paradox during adolescence, whereby adolescents are at their physical prime and yet have the highest rates of preventable injuries and mortality of any age group; injuries are the main cause of adolescent death (Viner, 2011), and mortality rates increase by two hundred per cent during adolescence (Dahl, 2004). Adolescent boys are at the greatest risk, with three times as many adolescent boys dying from preventable injuries as adolescent girls (Miniño, 2010). The findings that males take more risks and are more likely to die from preventable injuries underscore the importance of examining gender differences in risk-taking behaviours. In addition to having potentially costly outcomes for individuals, adolescent risk-taking also has potentially harmful and costly consequences for society. For instance, approximately 1,245 adolescents visit accident

and emergency departments in the UK each week for alcohol-related treatments (Newbury-Birch *et al*., 2009), and the UK spends approximately £4 billion on youth crime and antisocial behaviour every year (Natale, 2010).

1.3.2. The stress of adolescence: Increase in anxiety levels

Hall (1904) also defined adolescence as a period of heightened stress. More recent work has also suggested that adolescents experience high levels of stress due to the sheer number of changes that they undergo (Casey *et al*., 2010; Spear, 2000). Such changes include sexual maturation, changing social demands, reduced parental influence, more salient peer and romantic relationships, enhanced cognitive abilities, and greater economic independence (Buchanan, Eccles & Becker, 1992; Spear, 2000).

Consistent with this idea, a number of studies have reported that self-report anxiety symptoms reach a lifetime peak in typically developing adolescents (Abe & Suzuki, 1986). Adolescents are also more likely than any other age group to develop an anxiety disorder (Kessler *et al*., 2005), and many adulthood anxiety disorders manifest and develop during adolescence (Kessler *et al*., 2005; Pine, Cohen, Gurley, Brook & Ma, 1998). Epidemiological studies report that anxiety disorders are the most prevalent psychiatric disorders during adolescence (Fergusson, Horwood & Lynskey, 1993; Merikangas *et al*., 2010), and that 31.9 per cent of adolescents meet the criteria for an anxiety disorder at any one time (Merikangas *et al*., 2010). Importantly, anxious adolescents are at an increased risk for long-term negative consequences, including educational underachievement, substance abuse, anxiety and mood disorders, and suicidal behaviour as young adults (Pine *et al*., 1998; Woodward & Fergusson, 2001). Gender differences have also been reported; females frequently experience higher anxiety levels and are twice as likely to be diagnosed with an anxiety disorder compared to males during all developmental stages (Abe & Suzuki, 1986; Lewinsohn, Gotlib, Lewinsohn, Seeley & Allen, 1998; Van Oort, Greaves-Lord, Verhulst, Ormel & Huizink, 2009).

These findings highlight the importance of understanding why adolescence is a vulnerable period for experiencing elevated anxiety levels and developing an anxiety disorder, particularly for females. It is important to note that while adolescents are at increased risk for developing an anxiety disorder, the majority of adolescents do not. Accordingly, this doctoral work focused on anxiety levels in typically developing adolescents.

1.3.3. The paradox: The simultaneous increase in risk-taking and anxiety

The simultaneous increase in risk-taking behaviours and anxiety levels during adolescence seems paradoxical since anxiety acts as a brake on risk-taking in adults (Broman-Fulks, Urbaniak, Bondy & Toomey, 2014; Giorgetta *et al*., 2012; Lorian & Grisham, 2010; Maner *et al*., 2007; Maner & Schmidt, 2006). Despite appearing paradoxical, it is possible that the simultaneous increase in risk-taking and anxiety during adolescence has an evolutionary purpose (Casey *et al*., 2008a, 2008b; Spear, 2000; Spear, 2009; Steinberg, 2008). From an evolutionary perspective, sensation seeking tendencies and risk-taking behaviours peak during adolescence to encourage adolescents to leave the family home to seek out unrelated sexual partners. Since exploring novel environments has the potential to be dangerous, adolescents are thought to have elevated anxiety levels in order to be vigilant to potential threats. Importantly, elevated anxiety levels are not pathological when they facilitate the avoidance of danger (Steimer, 2002). Thus, the evolutionary interpretation of adolescent risk-taking and anxiety accounts for why risk-taking behaviours and anxiety levels simultaneously increase in typically developing adolescents.

While the simultaneous increase in risk-taking and anxiety during adolescence may have evolutionary antecedents, the relationship between risk-taking and anxiety in adolescents remains unclear. Initial work indicates that risk-taking behaviours are positively associated with anxiety levels during adolescence, whereby anxious adolescents take more risks than non-anxious adolescents (Comeau, Stewart & Loba, 2001; Patton *et al*., 1996; Reynolds *et al*., 2013; Richards *et al*., 2015). However, no study to date has included younger and older comparison groups, and therefore how the relationship between risk-taking and anxiety changes during the course of adolescence is currently unknown. Moreover, gender differences in the relationship between risk-taking and anxiety have been largely overlooked. Since differences between male and female adolescents in risk-taking and anxiety have been frequently observed (Byrnes *et al*., 1999; Lewinsohn *et al*., 1998), it is possible that exploring differences between males and females will reveal gender-specific relationships between risk-taking and anxiety. To these ends, the first study in this thesis aimed to examine how the relationship between risk-taking and anxiety changes across the course of adolescence in male and female adolescents aged 9-23 years (Chapter 3).

Hall (1904) attributed the storm and stress of adolescence entirely to the biological changes of puberty. Puberty is a developmental period encompassing the

physical changes that are necessary for sexual maturation (Spear, 2000). While adolescence is thought to begin at the age of 10 years for all individuals (World Health Organization, 2003), the onset of puberty varies markedly between individuals; puberty can begin any time between the ages of 8 and 13 years for healthy females and between 9 and 14 years for healthy males (Sørensen *et al.,* 2013). The age of pubertal onset is also thought to be highly heritable (~60%; Sørensen *et al.,* 2013). Thus, these findings show that chronological age and pubertal stage are highly dissociable during adolescence (Marshall & Tanner, 1969, 1970).

While Hall's (1904) claim is clearly an oversimplification, there is converging evidence that puberty has significant affects on risk-taking and anxiety during adolescence that are independent of chronological age. Specifically, more advanced pubertal stage and earlier pubertal onset are linked to greater engagement in risk-taking behaviours and higher levels of anxiety (Collado, MacPherson, Kurdziel, Rosenberg & Lejuez, 2014; Costello, Sung, Worthman, & Angold, 2007; Patton *et al*., 2004; Reardon, Leen-Feldner & Hayward, 2009). The finding that more advanced pubertal stage is associated with greater levels of risk-taking and anxiety is in line with the evolutionary perspective on adolescent risk-taking and anxiety; it is possible that as the body is reaching sexual maturation, adolescents are increasingly motivated to leave the family home to seek out potential sexual partners. Overall, these findings demonstrate the need to disentangle the affects of chronological age and puberty on the development of risk-taking behaviours and anxiety levels during adolescence. Accordingly, the first study in this thesis aimed to explore the development of risk-taking and anxiety as a function of both age and puberty, in addition to examining the relationships between risk-taking and anxiety during adolescence (Chapter 3).

1.4. The adolescent brain

Despite the human brain reaching ninety five per cent of its total volume by six years of age, the brain undergoes substantial structural changes during late childhood and adolescence (Giedd *et al*., 1999; Gogtay *et al*., 2004; Sowell, Trauner, Gamst & Jernigan, 2002). Consequently, a number of neurobiological models have been proposed to explain the increases in risk-taking behaviours during adolescence (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008). Casey's model (Casey *et al*., 2010) also accounts for the increases in emotionality, i.e., greater levels of anxiety and negative affect, reported during adolescence. Before the neurobiological

models are outlined and evaluated (Chapter 1.5), this section outlines what is currently known about the structural changes that occur in the brain during childhood and adolescence.

Structural magnetic resonance imaging (sMRI) studies conducted over the last two decades have consistently reported that the human brain matures in a back-to-front order, with posterior cortical regions maturing earlier in development than anterior cortical regions. More recent work has also reported that subcortical structures, such as the nucleus accumbens and amygdala, mature earlier in development than cortical prefrontal regions (Mills, Goddings, Clasen, Giedd & Blakemore, 2014). Thus, compared to other cortical and subcortical regions, the prefrontal cortex (PFC) undergoes a protracted development during childhood and adolescence, which extends throughout the third decade of life (Giedd *et al*., 1999; Giedd, 2004; Gotgay *et al*., 2004; Huttenlocher, 1979; Sowell *et al*., 2002).

The structural changes that occur during childhood and adolescence involve both white and grey matter. White matter is largely composed of myelinated axons. Myelin is a fatty sheath that forms around the axon of neurons, creating an electrically insulating layer. The myelin sheath enables neurons to transmit electrical impulses at a faster rate (Morell & Quarles, 1999). Several studies have reported that white matter increases across all areas of the cortex throughout childhood, adolescence, and young adulthood (Barnea-Goraly *et al*., 2005; Giedd *et al*., 1999; Giedd, 2004). These increases in white matter during childhood and adolescence are thought to result from increases in myelination and/or axon size (Segalowitz, Santesso & Jetha, 2010). White matter tracts between frontal cortical and other cortical regions, as well as between frontal cortical and subcortical structures, also continue to develop across childhood, adolescence, and young adulthood (Hagmann *et al*., 2010; Liston *et al*., 2006).

Compared to white matter, grey matter contains neural cell bodies, unmyelinated axons, dendrites, glia, and blood vessels. Early work investigating the development of grey matter reported that grey matter in most cortical areas follows a non-linear trajectory during childhood and adolescence, whereby cortical grey matter in the frontal, parietal, and temporal lobes steadily increases throughout childhood, peaks in early adolescence, and decreases throughout middle and late adolescence (Giedd *et al*., 1999; Giedd, 2004; Gotgay *et al*., 2004; Sowell *et al*., 2002). However, more recent work has failed to report an inverted u-shaped developmental trajectory of cortical grey matter (Mills *et al*., 2016). Instead, recent studies show that cortical

grey matter volume is greatest in childhood and decreases steadily throughout the second and third decade of life (Mills *et al*., 2016). Despite these discrepancies, cortical grey matter has been shown to develop first in posterior sensory regions and last in anterior higher-order regions (Paus, 2005). There is also evidence that grey matter in subcortical structures, namely the amygdala and nucleus accumbens, matures earlier in development than grey matter in cortical prefrontal regions (Mills *et al*., 2014).

Taken together, these findings reveal that the adolescent brain undergoes considerable changes throughout adolescence, and demonstrate that the PFC has a protracted development compared to other cortical and subcortical regions. The loss of grey matter during adolescence is thought to reflect the removal of superfluous synapses and the reorganisation of remaining synapses (Crews, He & Hodge, 2007). Hence, the reduction of grey matter combined with the augmentation of white matter during adolescence is thought to underlie faster and more efficient neural processing (Casey *et al*., 2008b; Casey, Tottenham, Liston & Durston, 2005; Paus, 2005). Moreover, improvements in cortico-cortical and cortico-subcortical connectivity during adolescence are thought to enhance interregional processing and global efficiency (Hagmann *et al*., 2010; Liston *et al*., 2006).

Gender differences in structural brain development have also been reported. Total cerebral volume is approximately 10 per cent larger in males compared to females, and peaks earlier in females (10.5 years) than in males (14.5 years) (Giedd *et al*., 1999; Lenroot *et al*. 2007). Gender-specific developmental trajectories have also been found for cortical grey matter; grey matter in frontal and parietal regions reaches maximum volume 1-2 years earlier in females compared to males (Giedd *et al*., 1999; Lenroot *et al*. 2007). Moreover, several studies have reported significant gender differences in grey matter density in subcortical structures, including the amygdala, hippocampus, striatum, hypothalamus, and cerebellum (Giedd *et al*., 1996; Tiemeier *et al*., 2010). White matter has also been reported to follow gender-specific developmental trajectories; Lenroot *et al*. (2007) found that white matter volume increased at a faster rate in males compared to females during childhood and adolescence. Such sexual dimorphisms in grey and white matter during adolescence suggest that the increases in gonadal hormones (testosterone, oestrogen, progesterone) during puberty may play an important role in structural brain development (Blakemore, Burnett & Dahl, 2010; Lenroot & Giedd, 2010). Despite this, very few studies have examined the affects of puberty on structural brain

development.

Initial research suggests that puberty has significant affects on the development of cortical and subcortical grey matter (Bramen *et al*., 2011; Goddings *et al*., 2014; Neufang *et al*., 2009; Peper *et al*. 2009). However, a consistent relationship between puberty and grey matter development is yet to emerge. Peper *et al*. (2009) found that gonadal hormones influenced global grey matter density in male and female adolescents aged 10-15 years; testosterone levels were positively associated with global grey matter density in males whereas oestrogen levels were negatively associated with global grey matter density in females. In addition to having affects on global grey matter density, puberty has also been shown to affect specific cortical and subcortical regions. For example, Goddings *et al*. (2014) reported that pubertal stage significantly influenced the structural development of several subcortical structures in male and female adolescents aged 7-20 years; more advanced pubertal stage was positively associated with grey matter volume in the amygdala and hippocampus but negatively associated with grey matter volume in the nucleus accumbens, caudate, putamen, and globus pallidus. Consistently, Neufang *et al*. (2009) found that testosterone levels were positively associated with grey matter volume in the amygdala for males and females aged 8-15 years. Interestingly, Bramen *et al*. (2011) found a positive association between pubertal stage and grey matter volume in the amygdala and hippocampus for boys aged 11-14 years but not for females. In contrast to Goddings *et al*. (2014), Neufang *et al*. (2009) also found that testosterone levels were negatively associated with grey matter volume in the hippocampus for males and females. Furthermore, Neufang *et al*. (2009) reported a negative association between testosterone levels and grey matter volume in parietal regions for males, and a positive association between oestrogen levels and grey matter volume in limbic regions for females. While these findings are not entirely consistent, they provide clear evidence that puberty has significant affects on cortical and subcortical grey matter development during adolescence.

A recent review has also suggested that puberty has significant affects on the development of white matter (Ladouceur, Peper, Crone & Dahl, 2012). For instance, Perrin *et al*. (2008) reported that the influence of testosterone on white matter volume was greater in boys with shorter versions of the androgen receptor gene. Collectively, these findings reveal that puberty has considerable affects on white and grey matter development during adolescence. Notably, puberty-related changes in the brain can

either be permanent (organisational changes) or temporary (activational changes) where gonadal hormones induce acute changes that are reversed once the hormones are removed (Blakemore *et al*., 2010; Sisk & Zehr, 2005). While these findings provide initial insights into the role of puberty in structural brain development, considerably more work is needed to identify the region- and gender-specific affects that puberty has on grey and white matter development during adolescence, as well as to establish whether these affects are permanent or temporary.

1.5. Neurobiological models of adolescence

Given that the brain undergoes substantial structural changes during adolescence, several neurobiological models have been proposed in an attempt to explain why risk-taking behaviours peak during adolescence (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008). Casey's model also aims to explain why adolescents experience heightened emotionality, i.e., greater levels of anxiety and negative affect, compared to children and adults (Casey *et al*., 2010). All the neurobiological models implicate the developmental mismatch of immature prefrontal cortical regions and more mature subcortical limbic regions in the heightened levels of risk-taking and emotionality observed during adolescence. Thus, the neurobiological models extend previous suggestions that elevated levels of risk-taking and emotionality during adolescence result solely from the protracted development of the PFC (Mills *et al*., 2014). The neurobiological models are outlined in the first four sections below. The final section critically evaluates the models.

1.5.1. Casey's dual systems model

Casey's dual systems model (Casey *et al*., 2008a, 2008b, 2010; Somerville, Jones & Casey, 2010) aims to explain the peak in risk-taking behaviours and emotionality during adolescence. Casey's model focuses on the relative maturity of cortical prefrontal regions and subcortical limbic regions, namely the ventral striatum and amygdala, during development. While prefrontal regions are paramount for cognitive control, impulse control, rational decision-making, and emotional regulation, limbic regions such as the ventral striatum and amygdala are critical for motivational and emotional processes (Cardinal, Parkinson, Hall & Everitt, 2002; Miller & Cohen, 2001).

Casey's model proposes that the relative maturity of prefrontal and limbic regions during childhood and adulthood is balanced; both prefrontal and limbic

regions are structurally immature in children and structurally mature in adults. However, the protracted development of the PFC compared to limbic regions leads to an imbalance in maturity between prefrontal and limbic regions during adolescence, whereby limbic regions are more structurally mature than prefrontal regions (Figure 1.1). As well as maturing earlier in development, Casey's model proposes that limbic regions are more responsive to rewards and threats during adolescence compared to childhood and adulthood.

Casey's model predicts that prefrontal regions can regulate limbic activity in unemotional 'cold' contexts during adolescence. Critically however, adolescents' undeveloped prefrontal regions are thought to be unable to regulate limbic activity in emotional 'hot' contexts when the limbic system has been highly activated by salient stimuli. Thus, the combination of an underdeveloped PFC and greater subcortical activity to salient stimuli is thought to underlie higher levels of risk-taking, sensation seeking, and emotionality during adolescence, particularly in 'hot' contexts.

There is substantial evidence from sMRI studies that the PFC has a prolonged development compared to other cortical and subcortical structures (Giedd *et al.*, 1999; Giedd, 2004; Gotgay *et al.*, 2004; Huttenlocher, 1979; Mills *et al.*, 2014; Sowell *et al.*,

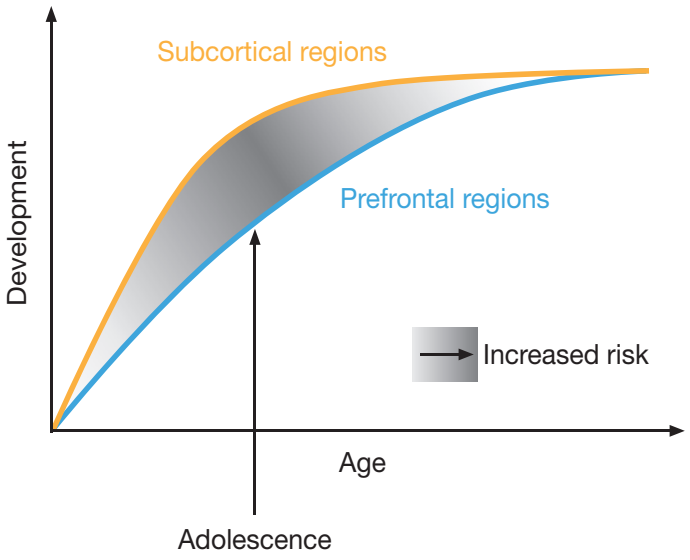

Figure 1.1 Casey's dual systems model (Casey *et al.*, 2008a; Casey *et al.*, 2008b; Casey *et al.*, 2010; Somerville *et al.*, 2010). This schematic model illustrates the prolonged development of cortical prefrontal regions relative to subcortical limbic regions (amygdala and ventral striatum). The imbalance in maturity between prefrontal and limbic regions is hypothesised to make adolescence a vulnerable period for engaging in risky behaviours and experiencing high levels of emotionality. Figure adapted from Somerville *et al.* (2010).

2002; see Chapter 1.4). Functional magnetic resonance imaging (fMRI) studies have also documented age-related changes in PFC function during childhood, adolescence, and adulthood; when completing cognitive tasks, children and adolescents recruit larger and more diffuse prefrontal regions than adults (Bunge, Dudukovic, Thomason, Vaidya & Gabrieli, 2002; Casey *et al*., 1997; Casey, Giedd & Thomas, 2000; Durston & Casey, 2006; Luna *et al*., 2001; Rubia *et al*., 2000; Tamm, Menon & Reiss, 2002). These studies have also reported that task-relevant PFC activity, i.e., activity correlated with task performance, becomes more focal with age and task-irrelevant PFC activity diminishes (e.g., Casey *et al*., 1997). These age-related changes in prefrontal activity are thought to result from the increases in white matter and decreases in grey matter that occur during adolescence, which together improve the speed and efficiency of neural processing (Blakemore & Choudhury, 2006; Hagmann *et al*., 2010). Thus, greater prefrontal activity during cognitive tasks in children and adolescents is thought to reflect less efficient neural processing in comparison to adults. Greater prefrontal activity during cognitive tasks in children and adolescents is also thought to reflect more effortful attention (Casey *et al*., 2000).

In addition to adolescents having an underdeveloped PFC, Casey's model suggests that adolescents' limbic regions are more responsive to rewards and threats compared to children and adults. Consistent with this idea, fMRI studies have reported that subcortical limbic regions are hypersensitive to rewards during adolescence; adolescents have greater activity in the ventral striatum and amygdala when anticipating and receiving rewarding or appetitive stimuli compared to both children and adults (Barkley-Levenson & Galván, 2014; Braams, van Duijvenvoorde, Peper & Crone, 2015; Galván *et al*., 2006; Galván, Hare, Voss, Glover & Casey, 2007; Hare *et al*., 2008; Padmanabhan, Geier, Ordaz, Teslovich & Luna, 2011; Van Leijenhorst *et al*., 2010). Critically, these studies have also shown that the amount of subcortical activity in response to rewards is positively associated with engagement in risky behaviours (Barkley-Levenson & Galván, 2014; Braams *et al*., 2015; Galván *et al*., 2007). Thus, these studies suggest that greater subcortical activity during adolescence is one mechanism underlying the increase in risk-taking. While fewer studies have examined adolescents' neural responses to threatening or aversive stimuli, initial work suggests that adolescents are also hypersensitive to threats. Specifically, fMRI studies have reported that adolescents have greater amygdala and ventral striatum activity when anticipating and encountering threatening or aversive stimuli compared to children

and adults (Britton *et al*., 2013; Galván & McGlennen, 2013; Hare *et al*., 2008). Notably, high levels of anxiety are associated with increased sensitivity and vigilance towards potential threats (Bouton *et al*., 2001; Mineka & Oehlberg, 2008). Hence, taken together, these studies provide compelling evidence in support of Casey's model, and suggest that adolescents are hypersensitive to rewards and threats, and that this hypersensitivity may partially underlie greater levels of risk-taking and anxiety during adolescence.

Importantly however, the central premise of Casey's dual systems model is that adolescents are able to regulate their emotions and behaviour in unemotional 'cold' contexts, but not in emotional 'hot' contexts. Accordingly, several studies have compared adolescents' prefrontal and limbic responses in neutral 'cold' contexts and emotional 'hot' contexts (Chein, Albert, O'Brien, Uckert & Steinberg, 2011; Hare *et al*., 2008; Lau *et al*., 2011; Monk *et al*., 2003). In line with Casey's model, Chein *et al*. (2011) found that adolescents (14-18 years) had greater ventral striatum and orbitofrontal cortex activity, but reduced lateral PFC activity, than young adults (24-29 years) while engaging in a risk-taking task. Critically, these age-dependent differences were only observed when adolescents' peers were present, but not when adolescents were alone. Similar findings have also been observed for threatening stimuli (Hare *et al*., 2008; Lau *et al*., 2011; Monk *et al*., 2003). For instance, Hare *et al*. (2008) found that adolescents (13-18 years) had greater amygdala activity and reduced ventral PFC activity in response to threatening, but not calm, facial expressions compared to young adults (19-32 years). Thus, the evidence to date appears to support Casey's dual systems model, and suggests that adolescents are hypersensitive to both rewards and threats.

1.5.2. Steinberg's dual systems model

Steinberg (Steinberg, 2008, 2010) developed the dual systems model (DSM) to explain the neural mechanisms underlying the increase in risk-taking behaviours and sensation seeking tendencies during the transition from childhood to adolescence, and the subsequent decrease in risk-taking and sensation seeking during the transition from adolescence to adulthood. In contrast to Casey's model, the DSM focuses exclusively on adolescent risk-taking, and is therefore limited in its ability to comprehensively explain adolescent behaviour and emotion. Despite this focus, the DSM provides important insights into why risk-taking behaviours and sensation seeking tendencies peak during adolescence.

The DSM is very similar to Casey's dual systems model in that the DSM focuses on the relative maturity of two brain systems: a 'cognitive-control' system and a 'socioemotional' system. The cognitive-control system comprises the lateral PFC, lateral parietal cortex, and anterior cingulate cortex. By contrast, the socioemotional system encompasses limbic and paralimbic regions, namely the amygdala, ventral striatum, orbitofrontal cortex, medial PFC, and superior temporal sulcus. Consistent with Casey's model, the DSM asserts that the socioemotional system matures much earlier in development than the cognitive-control system, leading to an imbalance in maturity between the two systems during adolescence. The DSM also proposes that the socioemotional system is more active in response to affective stimuli in adolescents compared to children and adults. Thus, according to both Steinberg's and Casey's dual systems models, the increase in risk-taking and reward-seeking behaviours during the transition from childhood to adolescence results from a more mature and active socioemotional system combined with an underdeveloped cognitive-control system that is unable to regulate the socioemotional system in emotionally salient situations. As the cognitive-control system matures and activity in the socioemotional system decreases, the cognitive-control system is increasingly able to regulate the socioemotional system in emotionally salient situations. Accordingly, risk-taking behaviours decline during the transition from adolescence to young adulthood.

The DSM draws from the same evidence base as Casey's model, suggesting that the socioemotional system matures earlier in development than the cognitive-control system, and is more active in response to appetitive stimuli in adolescents compared to children and adults (Barkley-Levenson & Galván, 2014; Braams *et al*., 2015; Galván *et al*., 2007). However, the DSM also asserts that the influx of gonadal hormones during early adolescence is largely responsible for the elevated activity in the socioemotional system in response to rewards during adolescence. In support of this idea, fMRI studies have reported that pubertal stage and gonadal hormone levels are associated with subcortical activity when receiving rewards (Braams *et al*., 2015; De Macks *et al*., 2011). For instance, Braams *et al*. (2015) found that self-reported pubertal stage and testosterone levels were positively associated with reward-related nucleus accumbens activity in 8-17 year olds after controlling for chronological age. Similarly, De Macks *et al*. (2011) reported that testosterone levels were positively associated with reward-related dorsal and ventral striatal activity after controlling for chronological

age in 10-16 year olds. Together, these findings show that increases in pubertal stage and testosterone levels are associated with increases in reward-related subcortical activity. It has therefore been suggested that the changes in gonadal hormones during puberty influence the way adolescents respond to rewards, which subsequently drives adolescents to engage in greater levels of sensation seeking and risk-taking behaviours (De Macks *et al*., 2011).

While the DSM does not address the increase in anxiety levels during adolescence, it is important to note that puberty has also been associated with threat-related brain activity in adolescents (Forbes, Phillips, Silk, Ryan & Dahl, 2011; Moore *et al*., 2012). For instance, Moore *et al*. 2012 found that self-reported pubertal stage in adolescents aged 13 years was positively correlated with activity in the amygdala, extrastriate cortex, and thalamus in response to fearful and angry faces. Collectively, these preliminary studies suggest that more advanced pubertal stage is associated with increases in reward- and threat-related neural activity. Thus, the influences of puberty and age on reward- and threat-related brain activity need to be disentangled in adolescent research. Accordingly, the EEG study reported in Chapter 4 explored adolescents' anticipatory responses to rewarding and threatening outcomes as a function of both age and puberty.

Consistent with Casey's model, the DSM also emphasises the importance of 'hot' and 'cold' contexts in adolescent risk-taking. In particular, the DSM proposes that the presence of peers considerably increases adolescent risk-taking, and shifts the context from 'cold' to 'hot'. In support of this idea, there is substantial behavioural evidence showing that adolescents take more risks when they are with peers compared to when they are alone (Chein *et al*., 2011; Gardner & Steinberg, 2005; Simons-Morton, Lerner & Singer, 2005). For instance, Gardner and Steinberg (2005) found that mid-adolescents (13-16 years) and late adolescents (18-22 years) took significantly more risks on a computerised driving game when they were in the presence of peers compared to when they were on their own. In direct contrast, adults (24+ years) made the same number of risky decisions when they were on their own as when they were in the presence of peers.

fMRI studies also support the idea that peers encourage risk-taking during adolescence. For instance, Chein *et al*. (2011) found that mid-adolescents (14-18 years) had more ventral striatum and orbitofrontal cortex activity while they were completing a risk-taking task in the presence of peers compared to when they were

alone. Crucially, greater activity in the ventral striatum and orbitofrontal cortex was associated with subsequent risky behaviour. Notably, these effects were not observed for young adults (24-29 years). Consistently, Smith, Steinberg, Strang and Chein (2015) found that adolescents had increased striatal activity when receiving rewards during peer observation compared to young adults. These findings suggest that peers increase adolescent risk-taking by increasing the salience of rewards (Smith *et al.*, 2015). In sum, these studies provide cogent evidence that the context, and the presence of peers in particular, can have significant affects on adolescent risk-taking.

1.5.3. The Triadic Model

Ernst *et al.* (2006) developed the Triadic Model to explain the increases in risk-taking and reward-orientated behaviours during adolescence. While Casey and Steinberg propose dual system models, the Triadic Model asserts that behaviour is motivated by three distinct systems: an approach, reward-based system driven by the nucleus accumbens (ventral striatum); an avoidance threat-based system driven by the amygdala; and a regulatory system driven by the PFC.

The Triadic Model is primarily concerned with explaining how representations of stimuli are translated into approach and avoidance behaviours. According to the

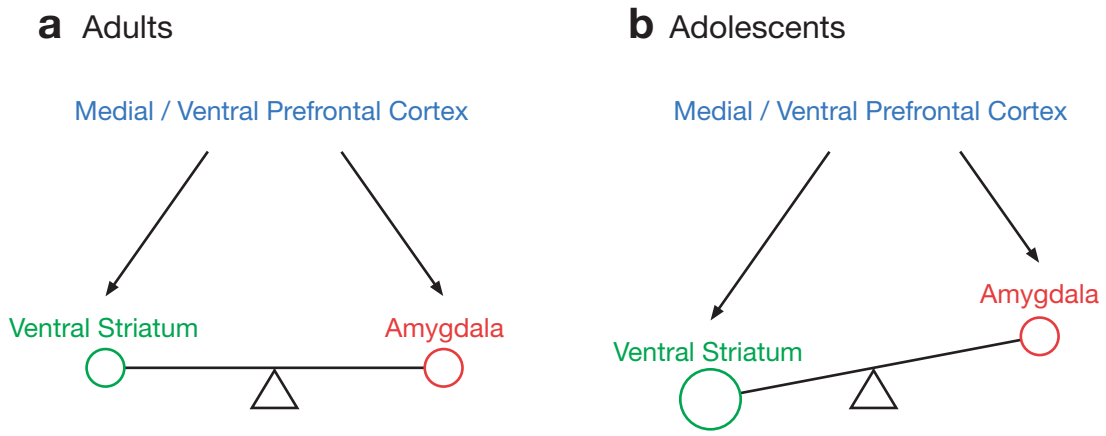

Figure 1.2 The Triadic Model (Ernst *et al.*, 2006). The Triadic Model asserts that behaviour is motivated by three distinct systems: an approach, reward-based system driven by the nucleus accumbens (ventral striatum); an avoidance threat-based system driven by the amygdala; and a regulatory system driven by the PFC. In adults (**a**), the approach and avoidance systems are balanced, and therefore the PFC is able to exert equal control over the approach-avoidance systems. However, in adolescents (**b**), the approach system is stronger than the avoidance system, and therefore the PFC is unable to exert equal control over the approach-avoidance systems. The arrows represent the relative control of the PFC over the ventral striatum and amygdala. Figure adapted from Ernst and Fudge (2009).

Triadic Model, the coding of approach and avoidance signals, by the nucleus accumbens and amygdala, respectively, is balanced in adults. Hence, during adulthood, the prefrontal regulatory system is able to exert equal control over the approach and avoidance systems. In contrast to adults, adolescents are thought to have a stronger approach system and a weaker avoidance system, and therefore the coding of approach and avoidance signals is imbalanced during adolescence. Consequently, the approach system prevails over the avoidance system in adolescence, and the immature prefrontal regulatory system is unable to exert equal control over the approach and avoidance systems. Thus, according to the Triadic Model, adolescents have a hyperresponsive approach system and a hyporesponsive avoidance system (Figure 1.2). This imbalance during adolescence is thought to result in a strong desire to approach rewarding stimuli, and consequently engage in higher levels of risk-taking behaviours.

In support of the Triadic Model, there is considerable evidence suggesting that adolescents are hyperresponsive to rewards and have a stronger approach system than both children and adults (see Chapter 1.5.1). However, research examining adolescents' sensitivity to threats is limited. Consistent with the Triadic Model's predictions, there is some evidence from behavioural studies indicating that adolescents are highly sensitive to rewards but less sensitive to threats. For example, in a large sample of 10-30 year olds, Cauffman *et al*. (2010) found that avoidance behaviours on the Iowa Gambling Task, i.e., avoiding playing from disadvantageous card decks, increased with age. In contrast, approach behaviours, i.e., playing from advantageous card decks, were greatest in middle and late adolescents (14-21 years). The authors interpreted the findings by suggesting that adolescents have a strong approach system paired with a weak, immature avoidance system.

In contrast to these behavioural findings, initial fMRI evidence suggests that adolescents are hyperresponsive to threatening stimuli (Britton *et al*., 2013; Galván & McGlennen, 2013; Hare *et al*., 2008; see Chapter 1.5.1), and not hyporesponsive to threats as the Triadic Model predicts. Moreover, recent EEG findings suggest that adolescents (12-15 years) are more sensitive to potential threats than young adults (18-32 years) (Levita, Howsley, Jordan & Johnston, 2014). Finally, adolescents are more likely than any other age group to experience elevated anxiety levels and be diagnosed with an anxiety disorder (Abe & Suzuki, 1986; Kessler *et al*., 2005; Merikangas *et al*., 2010; Pine *et al*., 1998). Anxiety is characterised by increased

attention to, and excessive avoidance of, stimuli perceived to be harmful or threatening (Bouton, Mineka & Barlow, 2001; Mineka & Oehlberg, 2008). Hence, these findings collectively point towards adolescents being hypersensitive to threats. Despite this, adolescents' sensitivity to threat has been scarcely studied, and thus considerably more behavioural, fMRI, and EEG research is needed to shed light on how sensitivity to threat develops across adolescence, and how sensitivity to threat corresponds to sensitivity to reward during different developmental stages.

While Casey's and Steinberg's dual systems models focus on the maturity and activity of two neural systems, the Triadic Model proposes that three distinct nodes are critical in driving adolescent behaviour, namely the PFC, nucleus accumbens, and amygdala. All the neurobiological models assert that the PFC is pivotal for regulating behaviours and emotions, and that the nucleus accumbens and amygdala are critical for motivational and emotional processes. However, the Triadic Model suggests that the nucleus accumbens and amygdala have specific roles in processing rewarding and threatening stimuli, respectively. While early studies implicated the ventral striatum (nucleus accumbens) in reward processing (e.g., Hollerman, Tremblay & Schultz, 1998; Schultz, Apicella, Scarnati & Ljungberg, 1992) and the amygdala in threat processing (e.g., Adolphs, Tranel, Damasio & Damasio, 1995), more recent studies have shown that the striatum has a role in processing threatening and aversive stimuli (Jensen *et al*., 2003; Levita, Hoskin & Champi, 2012; Pohlack, Nees, Ruttorf, Schad & Flor, 2012; Seymour, Daw, Dayan, Singer & Dolan, 2007), and the amygdala has a role in processing rewarding and appetitive stimuli (Baxter & Murray, 2002; Gottfried, O'Doherty & Dolan, 2003). Thus, contemporary work suggests that the striatum and amygdala have complementary but distinct roles in value-based encoding, updating value representations, outcome expectancy, and reinforcement learning that extend beyond the reward-threat distinction (Cardinal, Parkinson, Hall & Everitt, 2002; Costa, Dal Monte, Lucas, Murray & Averbeck, 2016; Pohlack *et al*., 2012; Somerville, van den Bulk & Skwara, 2014). Hence, the Triadic Model is currently limited in its ability to accurately describe adolescents' neurobiological responses to reward and threat. In order to establish a comprehensive and precise understanding of the neural circuitry underlying adolescent risk-taking and anxiety, it is vital that the neurobiological models of adolescence continue to evolve and incorporate contemporary empirical findings.

1.5.4. Social Information Processing Network

As noted by the DSM (Chapter 1.5.2), peers become increasingly important during adolescence, and are thought to contribute to the high levels of risk-taking behaviours frequently observed in this developmental period (Chein *et al.*, 2011; Gardner & Steinberg, 2005; Simons-Morton *et al.*, 2005). Accordingly, Nelson *et al.* (2005) developed the Social Information Processing Network (SIPN) model in an attempt to explain why social cues and peer relationships become more salient during adolescence (Figure 1.3). The SIPN proposes that three distinct neural nodes are responsible for guiding social behaviour: the detection node; the affective node; and the cognitive-regulation node. The detection node is comprised of visual processing areas, including the inferior occipital cortex, inferior regions of the temporal cortex, intraparietal sulcus, fusiform face area, superior temporal sulcus, and anterior temporal cortex, and is responsible for identifying the social properties of a stimulus. By comparison, the affective node encompasses the amygdala, nucleus accumbens, hypothalamus, septum, the bed nucleus of the stria terminalis, and orbitofrontal cortex. Thus, the affective node is comparable to the subcortical limbic regions

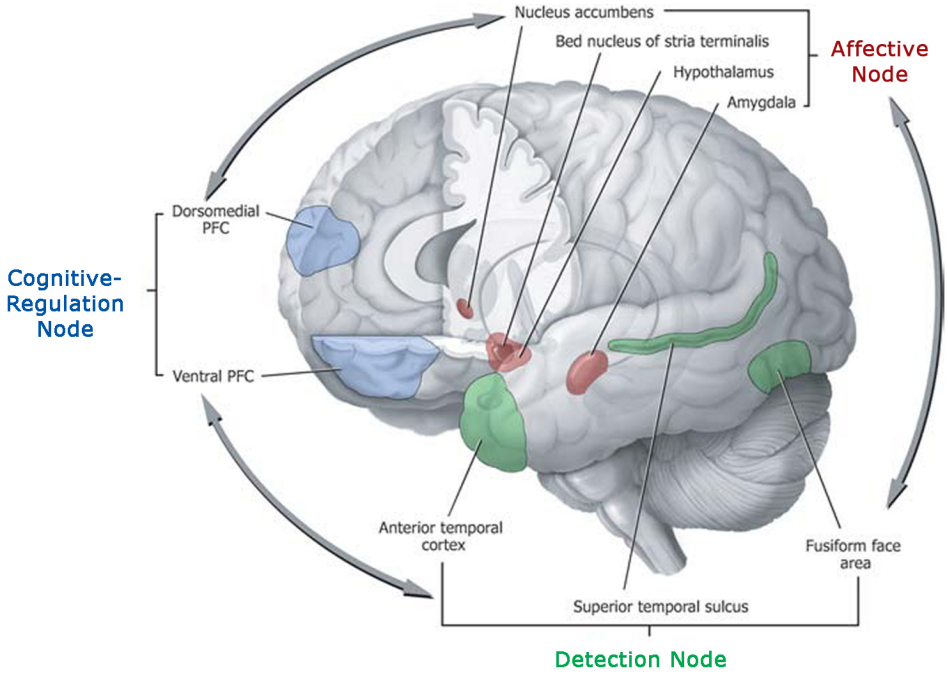

Figure 1.3 The Social Information Processing Network (Nelson *et al.*, 2005). A schematic depiction of the regions contained within the detection node (green), affective node (red), and cognitive-regulation node (blue). The grey arrows represent that the nodes are highly interactive. Figure reprinted with permission from Nelson *et al.* (2005).

discussed in the other neurobiological models (Casey *et al*., 2010; Ernst *et al*., 2006; Steinberg, 2008). The affective node is important for determining the value of a social stimulus, and whether that stimulus should be approached or avoided. Finally, the cognitive-regulation node includes the dorsomedial PFC and ventral PFC, and has three core functions: to perceive others' mental states; to inhibit prepotent responses; and to generate goal-directed behaviours towards socially rewarding stimuli and away from socially threatening stimuli. The three nodes are highly interconnected and function as an interactive neural network.

The visual processing areas that comprise the detection node have largely reached structural maturity by adolescence (Gogtay *et al*., 2004). Therefore, the SIPN suggests that heightened sensitivity to social stimuli during adolescence is driven by changes in the affective and cognitive-regulation nodes. In particular, the SIPN suggests that the affective node becomes hyperresponsive to social stimuli in adolescents relative to children and adults. In line with the DSM, the SIPN has a strong emphasis on the role of puberty in the hyperresponsivity of the affective node to salient stimuli. Consistent with the other models (Casey *et al*., 2010; Ernst *et al*., 2006; Steinberg, 2008), the SIPN proposes that the immaturity of the cognitive-regulation node combined with a more active affective mode results in greater sensitivity to social stimuli during adolescence. The SIPN asserts that adolescents are increasingly able to regulate their responses to social information as the PFC matures.

Notably, the SIPN is the only neurobiological model to incorporate posterior visual regions. This is an improvement over the other models since a number of studies using EEG (Levita *et al*., 2014; Li, Li & Luo, 2005; Pizzagalli, Greischar & Davidson, 2003) and fMRI (Harry, Williams, Davis & Kim, 2013; Lang *et al*., 1998) have reported that activity in visual processing regions is greater in response to affective stimuli compared to neutral stimuli. Moreover, the amount of threat-related activity in visual processing regions is thought to be modulated by participants' anxiety levels (Li *et al*., 2005). These findings suggest that salient stimuli not only modulate activity in prefrontal and limbic regions, but also in posterior visual regions (Pizzagalli *et al*., 2003). Hence, if adolescents are hyperresponsive to rewards and threats as the current fMRI literature suggests (Barkley-Levenson & Galván, 2014; Braams *et al*., 2015; Britton *et al*., 2013; Galván *et al*., 2006; Galván & McGlennen, 2013; Hare *et al*., 2008), it is possible that adolescents would also have greater levels of activity in posterior visual regions when anticipating and receiving rewards and threats.

Despite including a posterior detection node, the SIPN asserts that adolescents' increased sensitivity to social stimuli results from developmental differences in the affective and cognitive-regulation nodes given that posterior visual areas structurally mature early in development (Gogtay *et al*., 2004). Thus, all the current neurobiological models overlook posterior regions when explaining the increases in risk-taking and emotionality during adolescence. Critically however, structural maturity does not imply functional maturity; a brain region with a similar structure in children and adults may not function in the same way (Ghetti & Bauer, 2012). Moreover, many behaviours result from dynamic neural networks that include both structurally mature and structurally immature regions in adolescents. Thus, it is plausible that a structurally immature node, such as the PFC, will have a cascading effect on the rest of a neural network. Adolescent work should therefore examine all brain regions that have been implicated in affective processing, even if they are structurally mature.

1.5.5. Critical evaluation of the models

The neurobiological models have provided testable models of adolescent development, and have therefore significantly enhanced our current understanding of why engagement in risk-taking behaviours and levels of emotionality peak during adolescence. Despite this, the neurobiological models have a number of limitations that need to be addressed.

First, while Casey's model and the Triadic Model focus on adolescents' sensitivity to reward and threat, Steinberg's DSM and the SIPN only focus on adolescents' sensitivity to reward. Crucially, in order to understand why risk-taking behaviours and anxiety levels simultaneously increase during adolescence, adolescents' sensitivity to both reward and threat need to be examined. Notably, Casey's model asserts that adolescents are hypersensitive to both rewards and threats, whereas the Triadic Model suggests that adolescents are hypersensitive to rewards but hyposensitive to threats. Given that threat-related behaviours and neural responses have not been widely studied in adolescents, and the initial EEG and fMRI evidence suggests that adolescents are hypersensitive to threats (Britton *et al*., 2013; Galván & McGlennen, 2013; Hare *et al*., 2008; Levita *et al*., 2014), the claim that adolescents are hyposensitive to threats (Ernst *et al*., 2006) is currently unsubstantiated. Hence, substantially more work is needed to explore adolescents' sensitivity to threat, and to

establish how the development of threat sensitivity corresponds to the development of reward sensitivity throughout adolescence.

Second, despite adolescence being a transitional period between childhood and adulthood (Casey *et al*., 2008a; Spear, 2000), the Triadic Model and SIPN only account for changes between adolescence and adulthood. Thus, the Triadic Model and SIPN are unable to explain how adolescence is a distinct developmental period from childhood; establishing how adolescents are distinct from children is essential for understanding why risk-taking behaviours and anxiety levels increase during the transition from childhood to adolescence (Casey *et al*., 2008a; Spear, 2000).

Third, most of the evidence testing the neurobiological models has resulted from fMRI and nonhuman animal studies. However, surprisingly little EEG work has examined adolescents' sensitivity to reward or threat. EEG has a number of benefits over fMRI (see Chapter 1.7). Therefore, investigating the approach-avoidance systems using EEG should provide novel insights into the neural processes underlying the approach-avoidance systems during adolescence (see Chapter 1.7).

Fourth, despite the SIPN including a posterior detection node, Nelson *et al*. (2005) assert that the increased salience of social stimuli during adolescence results solely from age-related changes in prefrontal and limbic regions. Hence, all the neurobiological models only implicate cortical prefrontal and subcortical limbic structures when explaining the increases in risk-taking behaviours (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008), and increases in emotionality (Casey *et al*., 2010), during adolescence. However, as discussed in Chapter 1.5.4, several EEG and fMRI studies in adults have reported that rewarding and threatening stimuli modulate activity in posterior brain regions (Harry *et al*., 2013; Lang *et al*., 1998; Levita *et al*., 2014; Li *et al*., 2005; Pizzagalli *et al*., 2003). Moreover, structural maturity does not necessarily imply functional maturity (Ghetti & Bauer, 2012). Thus, if adolescents are hyperresponsive to rewards and threats, as the current literature suggests, it is possible that posterior regions will also have a role in biasing adolescents towards rewards and threats. Therefore, more work is needed to identify the role of posterior brain regions in adolescent development.

Finally, all the neurobiological models overlook potential gender differences in adolescent development. This is surprising since there are considerable gender differences in risk-taking behaviours, anxiety levels, and brain development during adolescence (Byrnes *et al*., 1999; Lenroot & Giedd, 2010; Lewinsohn *et al*., 1998).

Critically, if the neurobiological models are going to inform future interventions aimed at reducing adolescent risk-taking and anxiety, it is important that the neurobiological models aim to establish and incorporate any gender differences in brain structure and activity associated with gender-specific behavioural outcomes. Therefore, the gender differences in risk-taking, anxiety, and related brain activity warrant further investigation.

This doctoral research was designed with these limitations in mind. The overall aim of this doctoral research was to examine the simultaneous increase in risk-taking behaviours and anxiety levels during adolescence. To this end, a large cohort of male and female preadolescents (9-12 years), mid-adolescents (13-17 years), and late adolescents (18-23 years) participated in a series of behavioural and EEG studies. Specifically, the first study aimed to examine how the relationship between risk-taking and anxiety changes across the course of adolescence (Chapter 3). The second two studies (Chapters 4 and 5) aimed to investigate whether age-related changes in the EEG correlates of the approach-avoidance systems could account for the development of risk-taking and anxiety found in this sample of adolescents.

These three studies addressed the limitations of the neurobiological models in the following ways. First, this doctoral work examined the development of risk-taking behaviours and anxiety levels, as well as age-related changes in the EEG correlates of the approach and avoidance systems, during adolescence. Second, participants aged 9-23 years took part in this research in order to examine the transitions from preadolescence to mid-adolescence and from mid-adolescence to late adolescence. Risk-taking behaviours and anxiety levels are thought to peak in mid-adolescence (Abe & Suzuki, 1986; Burnett *et al*., 2010; Steinberg *et al*., 2008), and thus examining the transitions from preadolescence to mid-adolescence and from mid-adolescence to late adolescence provided a way to examine the effects that are unique to mid-adolescence. Third, the neural correlates of the approach and avoidance systems were examined using EEG in order to provide novel insights into the neural processes associated with the approach-avoidance systems during adolescence. Fourth, the role of posterior regions in anticipating rewarding and threatening outcomes was assessed using EEG in Chapter 4. Finally, each of the three studies overtly tested potential gender differences. Consistent with Steinberg's DSM and the SIPN, this doctoral work also had a strong focus on the influence of puberty on risk-taking, anxiety, and brain activity given that puberty has been shown to affect adolescent risk-taking, anxiety,

structural brain development, and reward- and threat-related neural activity (Braams *et al*., 2015; Collado *et al*., 2014; Goddings *et al*., 2014; Ladouceur *et al*., 2012; Moore *et al*., 2012; Reardon *et al*., 2009).

1.6. The approach-avoidance systems during adolescence

The neurobiological models of adolescence are based on the premise that behaviour is driven by an approach reward-based system and an avoidance threat-based system. All the neurobiological models (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008) assert that the increase in risk-taking and reward-orientated behaviours during adolescence is driven by a hyperresponsive approach system and that adolescents are hypersensitive to rewards compared to children and adults. Notably, the Triadic Model and Casey's dual systems model also suggest that the avoidance system is pivotal in adolescent behaviour. In particular, the Triadic Model suggests that adolescent risk-taking is driven by a hyperresponsive approach system combined with a hyporesponsive avoidance system. In direct contrast, Casey's dual systems suggests that adolescents have a hyperresponsive avoidance system and a hyperresponsive approach system, which underlie greater levels of emotionality and risk-taking during adolescence, respectively.

The idea that behaviour is motivated by two antagonist systems has been agreed upon for decades (Davidson, 1984, 1992; Elliot, 2006; Fowles, 1987; Gray, 1975; Lewin, 1935; Mackintosh, 1974; Mowrer, 2006; Schneirla, 1959; Skinner, 1948), and as such several approach-avoidance models have been proposed (Corr & McNaughton, 2012; Elliot, 2006; Fowles, 1987; Gray, 1975; Mackintosh, 1974; Mowrer, 1960). Despite the different approach-avoidance models using different terminology for the approach and avoidance systems (e.g., behavioural activation system versus behavioural inhibition system; reward versus punishment; approach versus withdrawal), there is consensus among the models that behaviour is driven by two distinct approach-avoidance systems that are primarily concerned with survival.

The approach system is activated by appetitive or rewarding stimuli and drives behaviour towards desirable outcomes (Berridge & Kringelbach, 2008; Elliot, 2006). The approach system is therefore thought to underlie sensation seeking and risk-taking behaviours (Cloninger, 1987; Zuckerman & Kuhlman, 2000). In contrast to the approach system, the avoidance system is activated by aversive or threatening stimuli and drives behaviour away from undesirable outcomes (Corr, 2013; Davidson, 1992;

Elliot, 2006). Anxiety disorders are characterised by increased attention to, and excessive avoidance of, stimuli perceived to be harmful or threatening (Bouton *et al*., 2001; Mineka & Oehlberg, 2008); thus, the avoidance system is thought to have a critical role in the aetiology and maintenance of anxiety disorders (Salkovskis, 1991).

The peak in risk-taking behaviours and anxiety levels during adolescence suggests that the approach and avoidance systems are hyperresponsive to rewards and threats, respectively, in adolescents (Casey *et al*., 2010). Indeed, there is substantial evidence to suggest that adolescents are highly motivated by rewards, and that adolescents have a stronger approach system compared to children and adults (Barkley-Levenson & Galván, 2014; Braams *et al*., 2015; Galván *et al*., 2006; Galván *et al*., 2007; Hare *et al*., 2008; Padmanabhan *et al*., 2011; Van Leijenhorst *et al*., 2010). However, the role of the avoidance system in motivating adolescent behaviour has received considerably less attention. This may be because examining avoidance and threat-related behaviours in children and adolescents is surrounded by considerable ethical constraints. Moreover, it may be because the neurobiological models of adolescence have largely focused on reward-orientated behaviours. As previously discussed (Chapter 1.5.1), the initial fMRI evidence suggests that subcortical structures, such as the amygdala and nucleus accumbens, are more responsive to threatening stimuli in adolescents compared to both children and adults (Britton *et al*., 2013; Galván & McGlennen, 2013; Hare *et al*., 2008). Recent EEG findings have also shown that adolescents are more responsive to visual cues that predict a threatening outcome compared to young adults (Levita *et al*., 2014). Thus, in light of the current literature, the claim that adolescents have a weak, hyporesponsive avoidance system (Ernst *et al*., 2006; Ernst, Daniele & Frantz, 2011) is unfounded. More developmental work is needed to determine how sensitivity to threat and avoidance behaviours change from late childhood to young adulthood in order to establish whether adolescents have a stronger, weaker, or equivalent avoidance system compared to children and adults. The studies reported in Chapters 4 and 5 provide an initial step towards establishing how the EEG correlates of approach and avoidance systems change during the course of adolescence.

1.7. EEG correlates of the approach-avoidance systems

fMRI studies have been extremely useful in identifying the neural structures that have a role in processing approach- and avoidance-related stimuli. These studies

have consistently reported that subcortical limbic regions, particularly the ventral striatum (nucleus accumbens) and amygdala, and cortical prefrontal regions are pivotal in approach-avoidance processes (Adolphs *et al*., 1995; Knutson, Fong, Adams, Varner & Hommer, 2001; Levita *et al*., 2012; O'Doherty *et al*., 2004; Robinson, Charney, Overstreet, Vytal & Grillon, 2011). Notably, fMRI studies are limited in their ability to establish the precise timing of neural processes; fMRI measures neural activity indirectly by detecting changes in blood oxygenation, and consequently has a temporal resolution in the order of seconds. Critically however, neural activity occurs within milliseconds, and thus a millisecond-by-millisecond account of the neural processes associated with the approach-avoidance systems is currently missing from the literature. Compared to fMRI, the EEG signal directly reflects neural activity and therefore has excellent temporal resolution in the order of milliseconds rather than seconds (Davidson, Jackson, & Larson, 2000). Thus, using EEG should provide novel and much needed insights into the neural correlates of the approach-avoidance systems during adolescence. Furthermore, EEG is particularly well suited to developmental studies. In particular, the environment in which EEG is recorded is less hostile than fMRI; fMRI is noisy, and involves lying down in a confined space for a long period of time. Moreover, fMRI studies usually have high attrition rates in developmental studies (Ulmer & Jansen, 2010), and are considerably more expensive than EEG studies. Hence, EEG is a useful tool for examining the development of the approach-avoidance systems across childhood, adolescence, and adulthood.

There are several ways of decomposing the EEG signal to examine the EEG correlates of the approach-avoidance systems. Two methods were used in this doctoral work. First, event-related potentials (ERPs) were used to explore adolescents' anticipatory neural responses to visual cues that predicted rewarding and threatening outcomes (Chapter 4). ERPs are neural responses that are time locked to specific stimuli. ERPs measure the magnitude of neural activity (amplitude, μV) as well as the timing of neural activity relative to the stimulus onset (latency, ms). ERPs are composed of several peaks and troughs, which index particular perceptual and cognitive processes. Crucially, many ERPs are sensitive to appetitive and aversive stimuli; therefore, ERPs provide a way to examine how the brain processes affective information. Second, resting frontal EEG activity within the alpha frequency band (8-13 Hz) was examined in relation to adolescents' risk-taking behaviours (Chapter 5). There is a long-standing biphasic motivational theory suggesting that frontal alpha

asymmetry reflects the lateralisation of the approach-avoidance systems (Davidson, 1984, 1992). Based on the finding that alpha is inversely related to cortical activity (Haegens, Nácher, Luna, Romo & Jensen, 2011; Shagass, 1972), Davidson (1984, 1992) proposed that relatively greater left cortical activity compared to right cortical activity is associated with reward-related behaviours and the approach system, whereas relatively greater right cortical activity compared to left cortical activity is associated with threat-related behaviours and the avoidance system. There is considerable support for this theory in infants and adults (e.g., Davidson & Fox, 1989; Thibodeau, Jorgensen & Kim, 2006; Wheeler, Davidson & Tomarken, 1993). However, whether the same is true for adolescents is yet to be determined. The following sections briefly outline and discuss what is currently known about the ERPs (Chapter 1.7.1) and resting frontal alpha activity (Chapter 1.7.2) associated with the approach-avoidance systems in adolescents and adults.

1.7.1. Event-related potentials

The vast majority of studies examining the neural correlates of the approach-avoidance systems have used fMRI. Thus, the ERP components associated with the approach-avoidance systems remain largely unexplored. In an attempt to address this, the second study in this doctoral research used an instrumental conditioning task to examine adolescents' anticipatory ERP responses to rewards and threats (Chapter 4).

Suboptimal action selection during adolescence has been proposed as a potential mechanism underlying elevated levels of risk-taking and anxiety in this age group (Casey *et al.*, 2008b). Crucially, actions are guided by the anticipation of outcomes (Elsner & Hommel, 2001); thus, biases in outcome anticipation are thought to have knock-on effects on action selection, preparation, and implementation (Freese & Amaral, 2005; Hegdé & Felleman, 2007; Lamme & Roelfsema, 2000; Lang & Bradley, 2010; Sugase, Yamane, Ueno & Kawano, 1999; Vuilleumier, 2005). Therefore, biases in anticipatory neural activity to rewarding and threatening outcomes may underlie suboptimal action selection, and hence greater levels of risk-taking and anxiety, during adolescence. In line with this idea, fMRI studies have reported that adolescents have greater activity in subcortical limbic regions when anticipating rewarding and threatening outcomes, and that this activity is correlated with risk-taking behaviours and anxiety levels (Braams *et al.*, 2015; Galván *et al.*, 2006; Hare *et al.*, 2008). By contrast, studies examining the ERP correlates of reward and threat

anticipation in adolescents are scarce.

Instrumental conditioning tasks provide a way to examine anticipatory neural activity associated with rewarding and threatening outcomes that are conditional on actions. Accordingly, an instrumental conditioning task was used to measure adolescents' anticipatory neural responses to rewards and threats in the second study in this thesis (Chapter 4). Instrumental conditioning (Skinner, 1948) builds on Thorndike's law of effect, which asserts that actions associated with rewarding or pleasant outcomes are likely to be repeated while actions associated with punishing or unpleasant outcomes are not (Thorndike, 1905). Thus, while both instrumental and classical conditioning are concerned with learning to associate a neutral stimulus with an appetitive or aversive outcome, only instrumental conditioning is concerned with modifying behaviour through the use of rewarding and punishing outcomes.

Skinner (1948) proposed that each behaviour is composed of three parts: the discriminative stimulus (S^D); the learned response; and the reinforcer or punisher. The S^D is a cue that is associated with specific outcomes that are conditional on particular responses. For example, one response to an S^D may result in a punishment while a different response to the same S^D may result in the avoidance of a punishment. Examples of S^D include pictures, lights, sounds, and odours. The learned response refers to a behavioural response to a S^D that is modified by the associated outcomes. Once a response has been made, the behaviour is either reinforced or punished. Reinforcers increase the likelihood of a behaviour being repeated, whereas punishers decrease the likelihood of a behaviour being repeated. Thus, the outcome associated with a response determines whether the behaviour is likely to be repeated. S^D can be associated with four potential outcomes: positive reinforcement (receiving a rewarding outcome); negative reinforcement (avoiding a punishing outcome); positive punishment (receiving a punishing outcome); and negative punishment (removing a rewarding outcome). Notably, reinforcers and punishers can be primary or secondary. Primary reinforcers and punishers elicit automatic and involuntary biological reflexes, and include food, sex, pain, and loud noises. By contrast, secondary reinforcers and punishers have been previously paired with a primary reinforcer or another conditioned reinforcer, and include winning and losing money or points.

To the author's knowledge, no study to date has explored adolescents' anticipatory ERP responses to rewarding stimuli, and only one study has examined adolescents' anticipatory ERP responses to threatening stimuli (Levita *et al*., 2014). In

Levita *et al*.'s (2014) study, adolescents (12-15 years) and young adults (18-32 years) completed an instrumental avoidance task in which participants responded to S^D that predicted the onset of a loud, aversive tone. Both adolescents and young adults had greater N170 ERP amplitudes to S^D that predicted a threatening outcome compared to control cues. Critically, potentiation of the N170 to avoidance cues was greater in adolescents than young adults. This finding is in direct contrast with the Triadic Model, which asserts that adolescents have a weaker avoidance system compared to adults (Ernst *et al*., 2006). The N170 is an early visual ERP component, which is thought to originate from the fusiform face area and superior temporal gyrus (Sadeh, Podlipsky, Zhdanov & Yovel, 2010). Thus, the findings from Levita *et al*. (2014) extend the current neurobiological models of adolescence to suggest that heightened emotionality during adolescence may result from a widely distributed neural network that includes occipitotemporal regions in addition to prefrontal and limbic regions.

Our previous findings are an important first step towards understanding the ERP correlates of avoidance behaviours in adolescents (Levita *et al*., 2014). However, these findings need extending to assess not only how adolescents respond to S^D that predict threatening outcomes, but also to S^D that predict rewarding outcomes; examining adolescents' neural responses to rewarding and threatening stimuli in the same task will provide a way to directly test the suggestion that adolescents' have a hyperresponsive approach system combined with a hyporesponsive avoidance system (Ernst *et al*., 2006). Our previous findings also need extending to examine the ERP correlates of the approach-avoidance systems across the course of adolescence, rather than in two discrete age groups; adolescence is a developmental period representing the transition from childhood to adulthood, and therefore the transitions into and out of adolescence need to be examined in order to identify the changes that are unique to adolescence (Casey *et al*., 2008a; Spear, 2000). Finally, exploring gender differences in adolescents' anticipatory neural activity to S^D that predict rewards and threats may shed some light on why adolescent males are particularly prone to engaging in more risky behaviours and adolescent females are more likely to experience elevated anxiety levels (Byrnes *et al*., 1999; Lewinsohn *et al*., 1998). Accordingly, the second study in this doctoral research extended our previous ERP findings (Levita *et al*., 2014) by assessing anticipatory neural responses to S^D that predicted rewarding and threatening outcomes in a large sample of male and female adolescents aged 9-23 years (Chapter 4).

Since one of the main benefits of EEG is excellent temporal resolution, Chapter 4 also aimed to examine both early and late ERP components in order to investigate the timing of anticipatory neural responses to rewards and threats. To that end, the late positive potential (LPP) was assessed in addition to the N170. The LPP is a slow, positive ERP component that emerges 300-400 ms post stimulus onset over centroparietal regions. Like the N170, the LPP has greater amplitudes for positively and negatively valenced stimuli compared to neutral stimuli (Cuthbert, Schupp, Bradley, Birbaumer & Lang, 2000; Schupp *et al*., 2000; Schupp, Junghofer, Weike & Hamm, 2004), and is therefore a good candidate for exploring the later stages of reward and threat anticipation.

1.7.2. EEG frontal alpha asymmetry

The PFC is thought to have a critical role in the approach-avoidance systems; the PFC is highly connected to other cortical and subcortical regions (Fuster, 2001; Miller & Cohen, 2001), and is therefore able to integrate information, regulate emotions, and direct behaviour in accordance with current goals and task demands (Matsumoto, Suzuki & Tanaka, 2003; Powell & Redish, 2016; Ridderinkhof, Van Den Wildenberg, Segalowitz & Carter, 2004). There is considerable evidence from fMRI studies to suggest that the PFC is engaged when adults anticipate and receive rewards (Knutson *et al*., 2001; Knutson, Fong, Bennett, Adams & Hommer, 2003) and threats (Bishop, Duncan, Brett & Lawrence, 2004; Robinson *et al*., 2011). As discussed earlier (Chapter 1.4), the PFC undergoes a protracted development and is therefore relatively immature during adolescence. Thus, it has been suggested that the PFC has a weaker role in the approach-avoidance systems in adolescents compared to adults (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008). In line with this idea, Lau *et al*. (2011) found that activity in the dorsolateral PFC was important for discriminating between threat and safety cues in adults but not in adolescents.

The role of the PFC in the approach-avoidance systems has also been extensively studied using EEG. There is a long-standing theory that frontal cortical asymmetry reflects the lateralisation of approach-avoidance processes, as indexed by resting EEG alpha activity (Coan & Allen, 2004; Davidson, 1992; Harmon-Jones, Gable & Peterson, 2010). Frontal cortical asymmetry is measured using alpha asymmetry scores, whereby alpha activity recorded over the right frontal cortex is subtracted from alpha activity recorded over the left frontal cortex. Alpha activity is thought to have an

inverse relationship with cortical activity; greater alpha activity reflects reduced cortical activity and reduced alpha activity reflects greater cortical activity (Haegens *et al.,* 2011; Shagass, 1972). Based on these findings, Davidson proposed that a greater tendency to approach rewarding or appetitive stimuli is associated with relatively greater left frontal cortical activity compared to right frontal cortical activity, whereas a greater tendency to avoid threatening or aversive stimuli is associated with relatively greater right frontal cortical activity compared to left frontal cortical activity (Davidson, 1984, 1992; Tomarken, Davidson & Henriques, 1990; Tomarken, Davidson, Wheeler & Doss, 1992; Wheeler *et al*., 1993).

There is considerable evidence for the approach-avoidance lateralisation of the PFC in infants, children, and adults. In particular, relatively greater left cortical activity has been associated with risk-taking behaviours and sensation seeking tendencies in adults (Coan & Allen, 2003; Santesso *et al.*, 2008; Sutton & Davidson, 1997; Wheeler *et al*., 1993), and relatively greater right cortical activity has been associated with anxiety in infants, children, and adults (Davidson & Fox, 1989; Smit, Posthuma, Boomsma & De Geus, 2007; Thibodeau *et al*., 2006). However, very few studies have examined frontal asymmetry in adolescents, and it therefore remains unclear whether frontal asymmetry is a marker of adolescent risk-taking and anxiety. Since risk-taking behaviours and anxiety levels are widely reported to peak during adolescence (Abe & Suzuki, 1986; Burnett *et al*., 2010; Steinberg *et al*., 2008), examining frontal alpha asymmetry in adolescents may provide new insights into the role of the PFC in the approach-avoidance systems during adolescence. Consequently, the final study in this thesis (Chapter 5) aimed to explore the relationships between frontal alpha asymmetry and risk-taking behaviours across the different stages of adolescence adolescence.

1.8. Thesis outline

This doctoral research sought to explore the simultaneous increase in risk-taking and anxiety during adolescence (Abe & Suzuki, 1986; Burnett *et al*., 2010; Steinberg *et al*., 2008). To that end, a large cohort of male and female preadolescents (9-12 years), mid-adolescents (13-17 years), and late adolescents (18-23 years) participated in a series of behavioural and EEG studies examining how the relationship between risk-taking and anxiety changes during the course of adolescence (Chapter 3), and the age-related changes in the EEG correlates of the approach-avoidance systems

(Chapters 4 and 5).

Chapter 2 details the participant cohort, as well as the behavioural and EEG methods used in this doctoral research.

Chapter 3 presents a behavioural study that aimed to examine the gender differences in the relationship between risk-taking behaviours and anxiety levels during adolescence. Chapter 3 also aimed to investigate the age- and gender-related differences in risk-taking, sensation seeking, impulsivity, and anxiety in this sample of adolescents. Finally, Chapter 3 aimed to disentangle the effects of age and puberty on the development of risk-taking, sensation seeking, impulsivity, and anxiety.

Next, Chapter 4 aimed to examine how anticipatory neural responses to reward and threat change during the course of adolescence. This study built on our previous EEG work showing that adolescents (12-15 years) have greater potentiation of the N170 ERP component to visual cues predicting threatening outcomes in comparison to young adults (18-32 years) (Levita *et al*., 2014). Chapter 4 extended our previous findings by examining two ERPs that are modulated by motivationally salient stimuli, the N170 and LPP, while participants completed an instrumental task in which they emitted or omitted a motor response to obtain rewards and avoid losses. The relationships between ERP amplitudes and measures of pubertal stage, risk-taking behaviours, and anxiety levels were also explored.

Finally, given that relatively greater left and right frontal cortical activity is thought to be associated with approach- and avoidance-orientated behaviours, respectively (Davidson, 1984, 1992), Chapter 5 aimed to explore the development of frontal alpha asymmetry during adolescence and its relationship to measures of risk-taking. Chapter 5 also aimed to investigate how the cortical sources of resting EEG alpha change as a function of age, gender, and puberty during adolescence.

Chapter 6 summarises and discusses the findings from this doctoral work, and considers directions for future research.

Chapter 2

General methodology

Abstract

This chapter details the behavioural and EEG measures used in this doctoral research. The same cohort of participants took part in each of the three studies. Participants were categorised into three age groups: preadolescents aged 9-12 years; mid-adolescents aged 13-17 years; and late adolescents aged 18-23 years. Risk taking behaviours were measured using the Balloon Analogue Risk Task (BART; Lejuez *et al*., 2002) and Youth Risk Behaviour Surveillance Survey (YRBSS; Aklin, Lejuez, Zvolensky, Kahler & Gwadz, 2005). Sensation seeking and impulsivity were measured using the Brief Sensation Seeking Scale (BSSS; Hoyle, Stephenson, Palmgreen, Lorch & Donohew, 2002) and a Go/NoGo task, respectively. Anxiety was measured using the State Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983), and depression was measured using the Hospital Anxiety and Depression Scale (HADS-D; Zigmond & Snaith, 1983). Participants aged 9-17 years also completed the Pubertal Development Scale (PDS; Petersen, Crockett, Richards & Boxer, 1988). Two EEG tasks were used to measure the EEG correlates of the approach-avoidance systems: an instrumental conditioning task measured anticipatory ERP responses to visual cues that predicted rewarding and threatening outcomes (Chapter 4), and a resting state EEG session measured spontaneous alpha (Chapter 5).

2.1. Participants

The same cohort of participants took part in each of the three studies included in this doctoral research (Chapters 3, 4, and 5). Examining the development of risk-taking behaviours, anxiety levels, and the approach-avoidance systems in the same cohort provided a way to explore the simultaneous increase in risk-taking and anxiety from multiple behavioural and EEG perspectives.

In total, 105 participants aged 9-23 years took part in this research. Participants were categorised into three age groups: preadolescents aged 9-12 years; mid-adolescents aged 13-17 years; and late adolescents aged 18-23 years (Table 2.1). Similar age groups have been widely used in behavioural and fMRI studies (e.g., Chein *et al*., 2011; Hare *et al*., 2008; Romer *et al*., 2009; Van Leijenhorst *et al*., 2010), and were therefore deemed appropriate for the current research. Adolescence begins around 10 years of age (World Health Organization, 2003), and thus preadolescence reflects the transitional period between childhood and adolescence. Compared to preadolescence, mid-adolescence reflects the teenage years, and is the developmental period where individuals are most likely to engage in risk-taking behaviours and experience elevated anxiety levels (Abe & Suzuki, 1986; Burnett *et al*., 2010; Steinberg *et al*., 2008). Finally, late adolescence reflects the later stages of adolescence, where individuals are transitioning between adolescence and young adulthood. Given that the human brain continues to mature across the third decade of life (Huttenlocher, 1979; Petanjek *et al*., 2011; Sowell, Thompson, Holmes, Jernigan & Toga 1999; Sowell, Thompson, Tessner & Toga, 2001), it has been suggested that adolescence extends until at least 25 years of

Table 2.1
Participant demographics

			Age (years)		Handedness		IQ	
Age Group	Gender	*n*	*M*	*SD*	*M*	95% CI	*M*	95% CI
Preadolescents	Females	18	10.78	1.22	0.66	0.56, 0.75	114.39	108.85, 119.32
9-12 years	Males	19	10.26	1.28	0.79	0.71, 0.87	114.68	108.37, 120.32
	All	37	10.51	1.26	0.73	0.66, 0.78	114.54	110.67, 118.41
Mid-adolescents	Females	16	14.69	1.54	0.68	0.58, 0.78	106.25	101.38, 110.31
13-17 years	Males	16	14.94	1.48	0.72	0.46, 0.90	107.25	103.81, 111.00
	All	32	14.81	1.49	0.70	0.57, 0.81	106.75	103.81, 109.44
Late adolescents	Females	20	20.45	1.47	0.77	0.70, 0.84	111.55	106.85, 116.20
18-23 years	Males	16	21.00	1.55	0.80	0.69, 0.89	114.00	110.67, 117.19
	All	36	20.69	1.51	0.78	0.73, 0.83	112.62	109.14, 115.94

Note. Handedness = Edinburgh Handedness Inventory; IQ = FSIQ-2 WASI-II scores; 95% CI = 95% bootstrapped confidence intervals.

age (Steinberg, 2008).

It should be noted that grouping participants into categorical age groups rather than using age as a continuous variable can be problematic in developmental research; there are considerable individual differences in the developmental trajectories of children and adolescents (Steinberg & Morris, 2001), and thus categorising participants into age groups may result in potentially interesting and important developmental differences being missed. Despite this, categorising participants into age groups provides a way to directly examine the discrete changes that occur within and between different developmental stages. Therefore, categorising participants into age groups was the preferred approach in this research.

In order to meaningfully compare the behaviour and EEG activity of preadolescents, mid-adolescents, and late adolescents, all participants needed to complete the same measures. Consequently, the behavioural and EEG measures detailed in this chapter were designed for use with participants aged 9-23 years old.

2.1.1. Inclusion criteria

Participants were recruited through the University of Sheffield, UK and local advertising. Prior to taking part, all participants over the age of 18 years and parents of participants aged 9-17 years completed an extensive screening form to ensure that only typically developing adolescents were included in this research. The screening form ensured that participants had normal or corrected-to-normal vision, normal hearing, no current or previous neurological, psychiatric, or medical conditions, were not currently taking medication, and were native English speakers. Due to potential differences in brain lateralisation between right- and left-handers (Bourne, 2008), only right-handed individuals were invited to take part. Handedness was measured using the Edinburgh Handedness Inventory (Oldfield, 1971; Table 2.1). An *Age Group* (preadolescents, mid-adolescents, late adolescents) by *Gender* (females, males) ANOVA found no main effects of *Age Group* ($F(2, 99) = 1.08$, $p = 0.343$, $\eta_p^2 = 0.02$) or *Gender* ($F(1, 99) = 1.87$, $p = 0.174$, $\eta_p^2 = 0.02$), and the *Age Group* by *Gender* interaction was non-significant ($F(2, 99) = 0.52$, $p = 0.594$, $\eta_p^2 = 0.01$), suggesting that handedness was equivalent across groups.

2.1.2. Sample size

The sample size was based on similar developmental EEG studies (e.g., Batty &

Taylor, 2006; Levita *et al*., 2014; Taylor, McCarthy, Saliba & Degiovanni, 1999). Overall, there were at least 14 participants per group available for statistical analysis in each EEG study (Chapters 4 and 5).

2.2. Procedure

Participants completed two visits within a two-week period. Prior to taking part in the first visit, informed consent was received from all participants, as well as from a parent or guardian of all participants under the age of 18 years. Participants carried out all tasks in a private room in the presence of an experimenter. Participants received £5 following each visit, and were fully debriefed upon completion of the second visit. Notably, a number of participants only completed the first visit, and were therefore fully debriefed after the first session. The research took place in the Department of Psychology, University of Sheffield, and was approved by the Department of Psychology, University of Sheffield ethics committee.

During the first visit, participants completed a battery of self-report questionnaires, two behavioural tasks measuring risk-taking propensity and impulsivity, and an IQ test. The self-report questionnaires measured participants' anxiety and depression levels, recent engagement in risk-taking behaviours, and sensation seeking tendencies. Participants aged 9-17 years also completed a self-report questionnaire assessing their current pubertal stage. The self-report questionnaires were completed using the online survey platform Qualtrics, and were presented in a random order to cancel out any order effects. Similarly, the order in which participants completed the questionnaires, behavioural tasks, and IQ test was counterbalanced across the different groups. The first visit lasted approximately one hour. During the second visit, participants had their brain activity measured using EEG while they completed an instrumental conditioning task (Chapter 4) and a short resting state session (Chapter 5). The second visit lasted approximately two hours. The following sections outline the behavioural (Chapter 2.3) and EEG (Chapter 2.4) measures in more detail.

2.3. Behavioural measures

2.3.1. Risk-taking behaviours

Risk-taking behaviours are frequently measured using retrospective self-report questionnaires that ask participants to self-report their engagement in a range of risky

behaviours, such as alcohol use, illicit drug use, and risky sexual behaviour. Thus, measuring risk-taking behaviours in children and adolescents raises a number of ethical and practical concerns. The primary concern when measuring risk-taking behaviours in developmental samples is whether it is ethical to ask participants to report their engagement in a range of risk-taking behaviours when it may expose individuals to behaviours they were previously unaware of. Adolescents may also be reluctant to answer questions about their engagement in risk-taking behaviours honestly due to the fear of potential negative consequences if a parent or guardian found out. Alternatively, some adolescents may lack the insight to provide an accurate report of their own behaviour. Finally, age-dependent differences in risk-taking behaviours may emerge due to older individuals having more opportunities to engage in certain risky behaviours, such as alcohol use and illicit drug use, rather than risk-taking propensity differing between age groups (Ladouceur *et al*., 2000; Lejuez *et al*., 2002; Lejuez, Aklin, Zvolensky & Pedulla, 2003).

In light of these concerns, behavioural tasks have been developed to measure risk-taking propensity rather than actual risk-taking behaviours. Therefore, behavioural tasks eliminate the ethical and practical issues associated with using self-report questionnaires to measure risk-taking behaviours in developmental samples (Lejuez *et al*., 2002, 2003). In particular, behavioural tasks do not ask participants to self-report their engagement in risky behaviours. Thus, behavioural tasks do not expose adolescents to unknown risky behaviours, require adolescents to accurately report their engagement in risky behaviours, or cause adolescents to worry about the potential negative consequences associated with answering honestly. Moreover, behavioural tasks eliminate the potential confound of finding age-related changes in risk-taking behaviours associated with accessibility to risks. To these ends, the Balloon Analogue Risk Task (Lejuez *et al*., 2002) was used in this research to assess participants' risk-taking propensity. An age-appropriate self-report questionnaire, the Youth Risk Behaviour Surveillance Survey (Aklin *et al*., 2005), was also administered to measure actual risk-taking behaviours. These measures are discussed further below.

2.3.1.1. Balloon Analogue Risk Task (BART)

The BART (Lejuez *et al*., 2002) is a widely used computerised behavioural task that measures risk-taking propensity in children, adolescents, and adults. The BART was developed to be an ecologically valid measure of risk-taking, whereby risk-taking

is advantageous up to a certain point, but risk-taking past this point is disadvantageous (Lejuez *et al.*, 2002). Thus, the BART accounts for risk-taking behaviours having the potential to be both advantageous and disadvantageous.

Both adult (BART; Lejuez *et al.*, 2002) and youth (BART-Y; Lejuez *et al.*, 2007) versions of the BART have been developed. In the BART and BART-Y, participants pump up a virtual balloon to win money or points, respectively. Since money may have a different meaning for younger compared to older adolescents (Barkley-Levenson & Galván, 2014), the BART-Y was used to measure risk-taking propensity in all participants in this doctoral research (Figure 2.1). Notably, in the original version of the BART-Y, the total number of points won is rewarded with a small, medium, or large monetary prize. Therefore, the original version of the BART-Y was modified slightly so that participants' performance on the task was not associated with a monetary reward. Similar modified versions of the BART-Y have been used previously

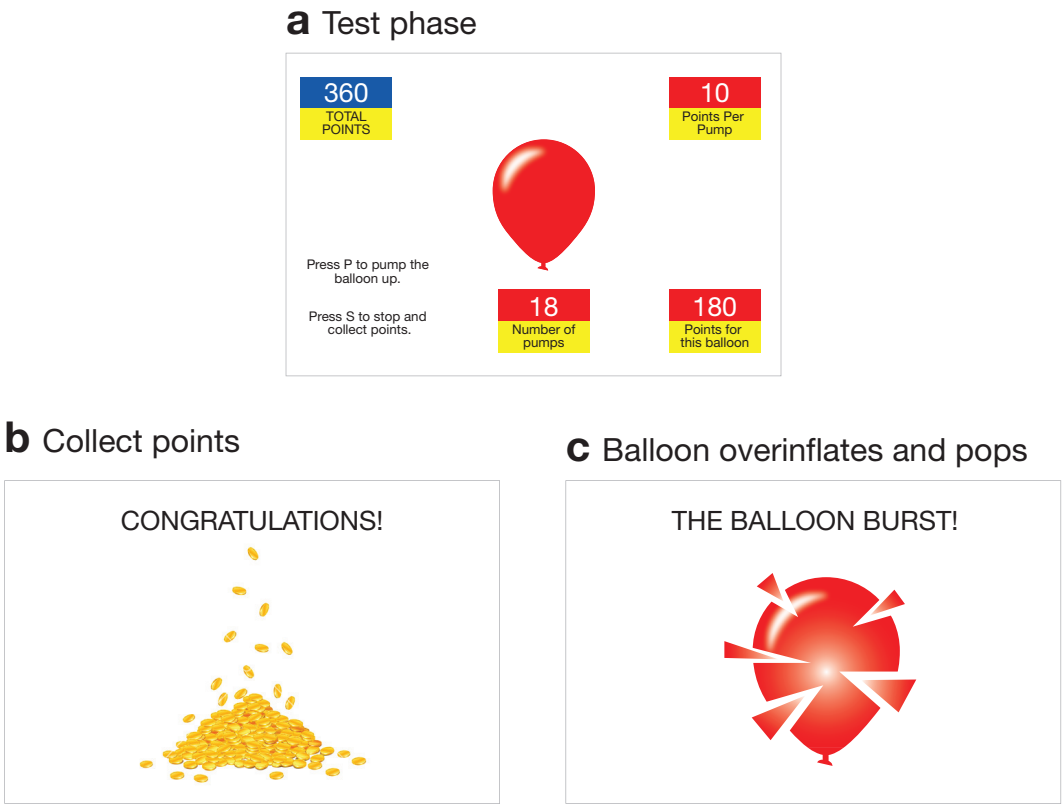

Figure 2.1 The Balloon Analogue Risk Task (BART). Participants pump up a virtual balloon to win points (**a**). Each balloon has a unique breakpoint where the balloon overinflates and pops. Thus, there are two outcomes: participants stop pumping up the balloon to collect their points before the balloon breakpoint (**b**); or, participants pump up the balloon past the balloon breakpoint and lose their points for that trial (**c**). The two outcome screens (**b**, **c**) were displayed for 2 seconds.

in adolescent samples (e.g., Vaca *et al*., 2013). In the modified version of the BART-Y used in this doctoral research, participants pumped up a virtual balloon in order to win points (Figure 2.1a). Participants won 10 points for each pump. Each balloon had a different breakpoint where the balloon would overinflate and pop. The probability that the first pump would pop the balloon was 1/128, while the probability the second pump would pop the balloon was 1/127, and so on. Thus, each pump involved greater risk, but also greater potential reward. The average balloon breakpoint was 64 pumps. Participants could stop the trial at any point and collect their points (Figure 2.1b). However, if participants pumped up the balloon to its breakpoint, the balloon popped and participants did not win any points for that trial (Figure 2.1c). One of two feedback screens informed participants of the outcome. As well as receiving visual feedback, participants heard the sound of coins when they won points and the sound of a balloon bursting when the balloon popped. In total, there were 30 balloons.

The BART yields three dependent measures: the average number of adjusted pumps for unpopped balloons; the total number of points won on the task; and the total number of popped balloons. The adjusted, rather than unadjusted, pumps are assessed since the number of pumps per balloon is constrained by the breakpoint of each balloon. The adjusted number of pumps is calculated for each unpopped balloon by dividing the number of pumps by the balloon's unique breakpoint, and multiplying by 100 ((*n* pumps / balloon breakpoint) * 100). A greater risk-taking propensity is indicated by a greater number of average adjusted pumps for unpopped balloons, total number of points won, or popped balloons. The modified version of the BART-Y was programmed and delivered using E-Prime 2.0.

To examine whether the modified BART-Y was an appropriate measure of risk-taking propensity in late adolescents, the BART-Y was piloted with 10 individuals aged 18-25 years (M_{age} = 19.20 years, SD_{age} = 2.10; 7 females). On average, the number of adjusted pumps per balloon was 54.38. Lejuez *et al.* (2002) reported an average of 25.00 and 30.50 adjusted pumps per balloon on the BART across 30 balloons for females and males aged 18-25 years, respectively. It is possible that participants in this pilot study took more risks on the BART-Y because points, rather than money, were used as the incentive. Indeed, Bornovalova et al. (2009) reported that the incentive used on the BART modulates the level of risk-taking in late adolescents (18-21 years). Consistent with previous work (Lejuez *et al.*, 2002), the average number of popped balloons in this pilot study was 9.80. Finally, the average number of points won on the BART-Y was

7513. Previous studies using the BART-Y have not reported the average number of points won and it is therefore unclear whether the number of points won on this task is comparable to other studies. In addition, different studies using the BART-Y attribute a different number of points to each pump. For example, Lejuez *et al.* (2007) awarded 1 point per pump whereas Vigil-Colet (2007) awarded 5 points per pump. Nonetheless, given that the number of points won is dependent on the number of adjusted pumps and the number of popped balloons, and the number of adjusted pumps and popped balloons were consistent with previous studies, it follows that the number of points won would also be in line with previous research.

To assess whether risk-taking changed during the course of the BART, risk-taking on the first, second, and final 10 balloons were compared (Table 2.2). Repeated measures ANOVAs were used to compare risk-taking during the first, second, and final 10 balloons. Consistent with previous work (Lejuez *et al.*, 2002, 2007), the average number of pumps ($F(2, 18) = 1.06$, $p = 0.368$, $\eta_p^2 = 0.11$), the number of points won ($F(2, 18) = 0.40$, $p = 0.676$, $\eta_p^2 = 0.04$), and the number of popped balloons ($F(2, 18) = 0.11$, $p = 0.895$, $\eta_p^2 = 0.04$) did not differ across the first, second, and final 10 balloons, suggesting that risk-taking remained constant across the duration of the task. In light of this, only the total scores were analysed in this research to minimise the number of statistical tests.

Table 2.2
BART pilot data

	First 10 Balloons		Second 10 Balloons		Final 10 Balloons		Total	
	M	95% CI	*M*	95% CI	*M*	95% CI	*M*	95% CI
BART Pumps	54.33	45.47, 63.19	51.89	40.24, 63.55	56.91	46.44, 67.38	54.38	45.01, 63.74
BART Points	2443	1731.59, 3154.41	2398	1838.85, 2957.15	2672	2258.70, 3085.30	7513	6366.45, 8659.55
BART Balloons	3.10	1.96, 4.24	3.30	1.61, 4.99	3.40	2.09, 4.71	9.80	6.45, 13.15

Note. BART Pumps = BART number of adjusted pumps; BART Points = BART total points won; BART Balloons = BART number of popped balloons; 95% CI = 95% bootstrapped confidence intervals.

2.3.1.2. Youth Risk Behaviour Surveillance Survey (YRBSS)

The YRBSS (Center for Disease Control, 2001) is a self-report questionnaire that measures health-risk behaviours. The YRBSS was developed for use with children and adolescents, and therefore minimises the ethical concerns surrounding using self-report questionnaires to measure risk-taking behaviours in developmental samples (see Chapter 2.3.1). The YRBSS was chosen over other risk-taking self-report questionnaire since it has been used widely in developmental research. In particular, the YRBSS has been extensively used in the United States to assess the epidemiology of health-related behaviours in adolescents (e.g., Eaton *et al*., 2010), and has been used to assess the validity of the BART in both adolescent and adult samples (Aklin *et al*., 2005; Lejuez *et al*., 2002, 2003, 2007). A 10-item version of the YRBSS has been developed for use in adolescent research (Aklin *et al*., 2005), and therefore the 10-item version was administered to all participants in this research. In the 10-item version of the YRBSS, participants are asked to indicate whether or not they have engaged in the following behaviours during the previous twelve months: drunk alcohol; smoked a cigarette; used any illegal drug; gambled for real money; had sexual intercourse without a condom; stolen anything from a store; carried a weapon outside of their home; been in a physical fight; ridden in a car without a seatbelt; ridden a bicycle or motorcycle without wearing a helmet. Responses were coded 1 for yes and 0 for no, and summed together to compute a total score (maximum score of 10). Greater scores on the YRBSS reflect greater engagement in real world risk-taking behaviours during the previous twelve months.

2.3.2. Sensation seeking

Age-related changes in sensation seeking tendencies are thought to contribute to the developmental differences in risk-taking behaviours (Steinberg *et al*., 2008; see Chapter 1.3.1 and Chapter 3.1.1.1). Participants' sensation seeking tendencies were therefore measured using the Brief Sensation Seeking Scale (BSSS; Hoyle *et al*., 2002). The BSSS is a shortened version of Form V of the Sensation Seeking Scale (Zuckerman *et al*., 1978), and was designed to be an age-appropriate measure of sensation seeking in adolescents. The BSSS has been validated in a large sample of adolescents aged 13-17 years (Hoyle *et al*., 2002), and is widely used with adolescents and adults aged 9-75 years (e.g., Eachus, 2004; MacPherson, Magidson, Reynolds, Kahler & Lejuez, 2010; Palmgreen, Donohew, Lorch, Hoyle & Stephenson, 2001).

The BSSS asks individuals to respond to the following statements: I would like to explore strange places; I get restless when I spend too much time at home; I like to do frightening things; I like wild parties; I would like to take off on a trip with no pre-planned routes or timetables; I prefer friends who are excitingly unpredictable; I would like to try bungee jumping; I would love to have new and exciting experiences, even if they are illegal. Responses are collected on a five-point Likert scale and summed together to create a total score (maximum score of 40). Greater scores indicate greater sensation seeking tendencies.

2.3.3. Impulsivity

Impulsivity has also been implicated in adolescent risk-taking (Steinberg *et al*., 2008; see Chapter 1.3.1 and Chapter 3.1.1.1). Accordingly, impulsivity was measured along with sensation seeking tendencies and risk-taking behaviours. The Go/NoGo task is widely used in developmental work to measure impulsivity (e.g., Bezdjian, Baker, Lozano & Raine, 2009; Casey *et al.*, 1997; Hare *et al.,* 2008; Johnstone, Pleffer, Barry, Clarke & Smith, 2005), and was therefore selected to measure impulsivity in this sample of adolescents. The Go/NoGo task used in this research (Figure 2.2) was

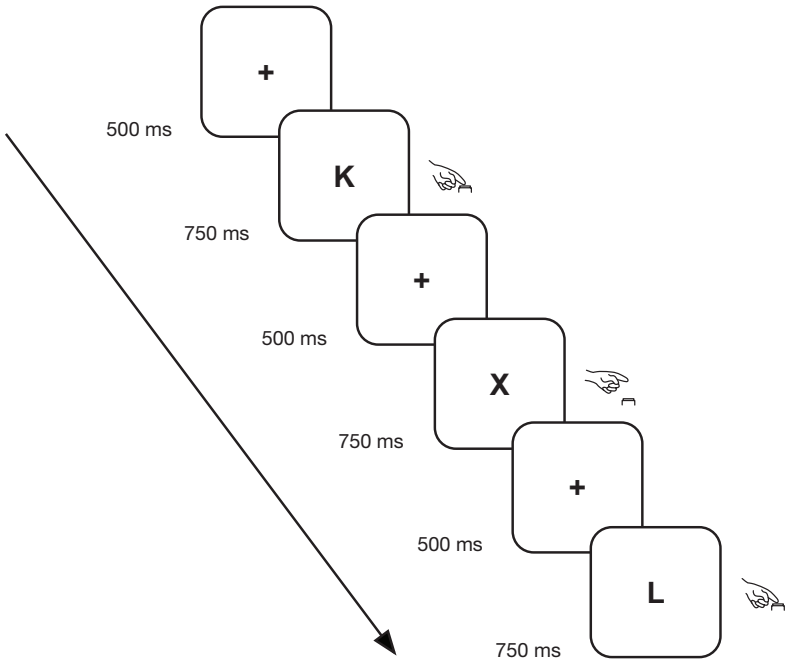

Figure 2.2 The Go/NoGo task. Participants were instructed to make a motor response to every letter except 'X'. Each stimulus was presented for 750 ms and there was a 500 ms intertrial interval between each stimulus.

developed on the basis of previous developmental work (Casey *et al.*, 1997; Hare *et al.,* 2008). In this version of the Go/NoGo task, black letters presented on a white background were used as the stimuli, and participants were instructed to make a motor response using a mouse button as quickly as possible to all letters except 'X'. Letters were displayed for 750 ms and there was a 500 ms intertrial interval. Participants responded to 120 letters in total. Go trials occurred at a higher frequency (n = 90) than NoGo trials (n = 30) to bias participants towards making a motor response. Performance on the Go/NoGo task was assessed using Go Accuracy (the percentage of correct responses to Go trials), NoGo Accuracy (the percentage of correct responses to NoGo trials), and Reaction Time (ms) to correct Go trials. The Go/NoGo was programmed and delivered using E-Prime 2.0.

The Go/NoGo task was piloted with 10 late adolescents aged 18-25 years (M_{age} = 19.20 years, SD_{age} = 2.10; 7 females). Accuracy was very high for both Go (M = 99.33%, 95% CI [98.78, 99.78]) and NoGo trials (M = 87.33%, 95% CI [79.67, 94.24]). As expected, a paired t-test revealed that late adolescents made more errors for NoGo trials compared to Go trials ($t(9) = 3.86$, $p = 0.004$). The average reaction time to correct Go trials was 386 ms (95% CI [351, 421]). These findings are consistent with previous studies measuring impulsivity using the Go/NoGo task in 18-25 year olds (e.g., Hirose *et al.*, 2012).

2.3.4. Anxiety

Anxiety was measured using the State-Trait Anxiety Inventory (STAI; Spielberger *et al.*, 1983). The STAI is a 40-item questionnaire that measures state (STAI-S) and trait (STAI-T) anxiety levels. There are child and adult versions of the STAI; the child version is used to assess anxiety levels in individuals aged 9-12 years, while the adult version is used to assess anxiety levels in individuals aged 13+ years. Given that this research aimed to examine the anxiety levels of individuals aged 9-23 years, it was investigated whether the child version of the STAI was an appropriate measure of anxiety in individuals aged 18-23 years to enable all participants to complete the same measure of anxiety. To that end, 300 individuals aged 18-41 years (M_{age} = 22.07 years, SD_{age} = 3.77; 216 females) completed the child and adult versions of the STAI online using Qualtrics. Participants were recruited through the University of Sheffield e-mail research volunteer list. Since the maximum raw scores on the child and adult versions of the STAI are 60 and 80, respectively, raw scores were converted

to percentages to enable direct comparisons between the two scales. Paired t-tests revealed that STAI-S scores measured using the child version (M = 59.17%, 95% CI [57.95, 60.40]) were significantly higher than STAI-S scores measured using the adult version (M = 53.03%, 95% CI [51.27, 54.79]) ($t(299) = 10.86$, $p < 0.001$). Similarly, STAI-T scores measured using the child version (M = 64.15%, 95% CI [62.59, 65.71]) were significantly higher than STAI-T scores measured using the adult version (M = 57.23%, 95% CI [55.65, 58.81]) ($t(299) = 12.09$, $p < 0.001$). These results indicate that the child version of the STAI is not an appropriate tool for measuring anxiety in late adolescents aged 18-23 years. Therefore, in this research, participants aged 9-12 years completed the child version of the STAI, and participants aged 13-23 years completed the adult version, as recommended by the STAI manual (Spielberger *et al*., 1983). Higher STAI scores indicate greater anxiety levels.

2.3.5. Depression

Anxiety and depression are highly comorbid in adolescents (Brady & Kendall, 1992) and young adults (Hirschfeld, 2001). Thus, participants' depression levels were measured using the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) to control for the influence of depression on participants' anxiety levels. The HADS is a 14-item scale that measures anxiety (HADS-A) and depression (HADS-D). The HADS has been validated in large samples of adolescents aged 10-19 years (Chan, Leung, Fong, Leung & Lee, 2010; White, Leach, Sims, Atkinson & Cottrell, 1999) and adults (Bjelland, Dahl, Haug & Neckelmann, 2002). Since the STAI was used to measure anxiety, only the HADS-D was analysed. Higher HADS-D scores indicate greater levels of depression.

2.3.6. Pubertal development

Puberty has been shown to influence risk-taking behaviours, anxiety levels, structural brain development, and reward- and threat-related neural activity (Braams *et al*., 2015; Collado *et al*., 2014; Goddings *et al*., 2014; Ladouceur *et al*., 2012; Moore *et al*., 2012; Reardon *et al*., 2009). Thus, this doctoral work had a strong focus on pubertal development. Notably, there is considerable variability in the onset of puberty; puberty can begin any time between the ages of 8 and 13 years for healthy females and between 9 and 14 years for healthy males (Sørensen *et al.,* 2013). Hence, chronological age and pubertal stage are highly dissociable during adolescence (Marshall & Tanner,

1969; Marshall & Tanner, 1970). Puberty lasts for approximately 4.5 years (Pinyerd & Zipf, 2005), and therefore by the age of 17 years puberty is largely complete for the majority of males and females (Braams *et al.*, 2015; Dorn & Biro, 2011). The three studies in this doctoral research therefore examined the relationship between pubertal stage and the specific dependent variables (e.g., risk-taking behaviours, anxiety levels, reward- and threat-related brain activity, spontaneous alpha) in a continuous sample of 9-17 year olds while controlling for chronological age.

In line with previous research (Braams *et al.*, 2015; Steinberg *et al.*, 2008), pubertal development was only assessed in preadolescents (9-12 years) and mid-adolescents (13-17 years). The Pubertal Development Scale (PDS; Petersen *et al.*, 1988) was used to measure current pubertal stage, and is a short self-report questionnaire that is designed for use with children and adolescents aged 9-18 years. The PDS has been extensively used to measure pubertal stage in both behavioural and fMRI studies (e.g., Braams *et al.*, 2015; Steinberg *et al.*, 2008). The PDS is also highly positively correlated with other pubertal measures, including physical examinations and hormone levels, and is therefore thought to be a reliable measure of current pubertal stage (Shirtcliff, Dahl & Pollak, 2009). For the PDS, males and females rated their growth in height, skin changes, and body hair growth. Males also rated changes in their voice and facial hair, while females indicated their menarche and breast development. Each item was rated on a four-point scale: 1 = not yet started; 2 = barely started; 3 = changes are underway; 4 = seems complete. Points for each item were averaged to give a PDS score. A higher PDS score indicates a more advanced pubertal stage.

2.3.7. Cognitive ability

There is some evidence that cognitive ability is positively associated with risk-taking behaviours (Dohmen, Falk, Huffman & Sunde, 2010). Consequently, the Wechsler Abbreviated Scale of Intelligence two-subtest version (WASI-II; Wechsler, 2011) was administered to all participants. Scores from the Vocabulary and Matrix Reasoning subtests were summed to yield the Full Scale IQ (FSIQ-2) score. Higher FSIQ-2 scores reflect greater cognitive abilities. The WASI-II can be administered to participants aged 6-90 years, making it an ideal measure of cognitive ability in developmental studies. FSIQ-2 group means are reported in Table 2.1.

2.4. EEG measures

Neural activity was measured using EEG. Measuring neural activity using EEG has many benefits, and developmental research is particularly well suited to EEG (see Chapter 1.7). This section first details the EEG recording (Chapter 2.4.1), and then describes the two EEG tasks used in this doctoral research (Chapter 2.4.2). Finally, this section outlines the preprocessing stream for the two EEG studies (Chapter 2.4.3).

2.4.1. EEG recording

The EEG signals were recorded using Biosemi Active Two 64 channel + common mode sense (CMS) / driven right leg (DRL) electrode caps and Biosemi 'Pin-Type' Ag-AgCl active electrodes. The electrode caps were fitted according to the 10/20 electrode system. Electrooculography (EOG) signals were also recorded using four Biosemi flat active electrodes placed on participants' temples and above and below their left eye. EEG and EOG signals were amplified using the Biosemi ActiveTwo AD-Box. Electrode offsets were stable and kept below ±25 μV. EEG signals were recorded continuously with a sampling rate of 2048 Hz. To reduce the affects of environmental electrical noise, participants sat in a quiet, electrically shielded room. The room was kept cool using air conditioning to minimise slow drifts in the recording resulting from sweat.

2.4.2. EEG tasks

Participants completed an instrumental conditioning task (Chapter 4) and a resting state session (Chapter 5) while their brain activity was recorded using EEG.

2.4.2.1. Instrumental conditioning task

Chapter 4 presents an ERP study whereby participants completed an instrumental task in which they responded to visual cues that predicted either a rewarding or threatening outcome. The instrumental conditioning task used in this study was an extension of a validated avoidance paradigm used previously in a developmental EEG study (Levita *et al*., 2014) and adult fMRI study (Levita *et al*., 2012).

The instrumental task used in Chapter 4 was composed of a reward block and an avoidance block (Figure 2.3). Both the reward and avoidance blocks included two S^D and two control stimuli. The S^D predicted a positive outcome (winning 10 points) in the reward block and a negative outcome (losing 10 points) in the avoidance block. In the reward block, one of S^D required participants to emit an action to win 10 points

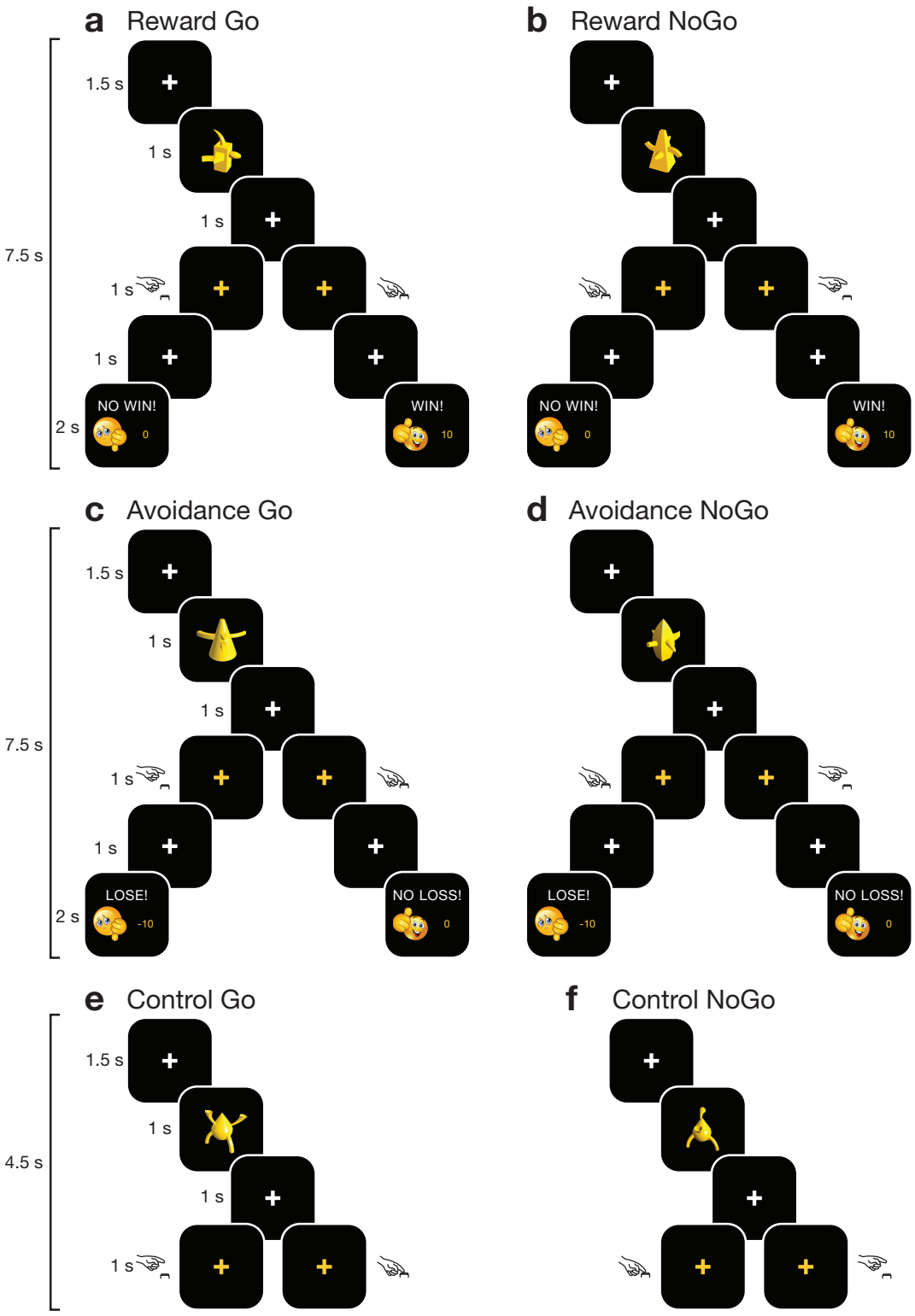

Figure 2.3 The instrumental conditioning task. The instrumental task was composed of a reward block (**a**, **b**, **e**, **f**) and avoidance block (**c**, **d**, **e**, **f**). Both the reward and avoidance blocks included two discriminative stimuli (S^D) (**a**, **b**, **c**, **d**) and two control stimuli (**e**, **f**). The S^D predicted a positive outcome (winning 10 points) in the reward block and a negative outcome (losing 10 points) in the avoidance block. For all conditions, participants were presented with a white fixation cross, followed by the visual cue. Participants were required to wait until the yellow fixation cross appeared on the screen before emitting or withholding their motor response. For S^D, participants saw one of two feedback screens indicating whether or not they had made the correct response. For control cues, participants received no feedback since control cues were not associated with a positive or negative outcome.

(Reward^{Go}; Figure 2.3a), while the other S^D required participants to omit an action to win 10 points (Reward^{NoGo}; Figure 2.3b). Participants only won points if they made the correct motor response to the S^D. In order to increase the potential threat associated with losing points, the reward block always occurred before the avoidance block. In the avoidance block, one of the S^D required participants to emit an action to avoid losing 10 points (Avoidance^{Go}; Figure 2.3c), while the other S^D required participants to omit an action to avoid losing 10 points (Avoidance^{NoGo}; Figure 2.3d). Participants lost 10 points for each incorrect response. Importantly, this instrumental task used a continuous schedule of reinforcement, whereby participants' behaviour was always reinforced.

The two control stimuli were consistent across the reward and avoidance blocks. Participants were required to emit an action for one of the control stimuli (Control^{Go}; Figure 2.3e) and omit an action for the other control stimulus (Control^{NoGo}; Figure 2.3f). Participants were told that the control cues were not associated with a rewarding or threatening outcome, but were included to ensure that they were paying attention throughout the task. The control stimuli provided comparison conditions to examine reinforcement-dependent potentiation to the S^D. For both the S^D and control stimuli, participants were told to emit or omit their motor response while the yellow cross was displayed on the screen. This provided a way to separate out neural activity associated with motor processes from anticipatory processes. For the S^D only, participants received feedback about their response in order to reinforce the correct response-outcome contingency. The feedback included whether they had made the correct motor response and their running total points score. Each stimulus was presented 72 times in both the reward and avoidance blocks. The stimuli were presented in a pseudorandom order, with the same stimulus not being presented more than twice consecutively. Each block was split into four 8-minute sections to allow participants to take regular breaks.

2.4.2.1.1. Visual stimuli and apparatus

The visual stimuli resembled greebles, and were created by Scott Yu and provided by Michael J. Tarr for the Neural Basis of Cognition and Department of Psychology, Carnegie Mellon University (http://www.tarrlab.org). Since early visual ERPs, such as the N170, are sensitive to the low-level visual properties of stimuli (Eimer, 2011; Rossion & Caharel, 2011), the visual stimuli used in this task were

matched for orientation, luminosity, size, and contrast, and counterbalanced across participants. Matlab 2012a was used to program and deliver the instrumental task. The task was delivered using a Viglen Intel Pentium 4 3 GHz computer and presented on a Viglen Omnino III monitor with a 1024 by 768 pixel resolution and 60 Hz refresh rate. The visual stimuli were presented on a black background, and motor responses were recorded using the space bar. For the duration of the task participants were seated approximately 70 cm away from the computer monitor in a dimly lit room shielded by a Faraday cage.

2.4.2.1.2. Reinforcer

Points were used as the reinforcer of behaviour in this instrumental conditioning task. Notably, the current study extended previous versions of the instrumental task so that the rewards and threats were salient, equivalent, and age-appropriate for individuals aged 9-23 years. Hence, the reinforcer used in this version of the instrumental task was changed from previous versions; Levita *et al*. (2014) used a loud aversive tone and Levita *et al*. (2012) used aversive pictures. In addition to aversive tones and aversive pictures, previous instrumental tasks in humans have used electric shocks (Delgado, Jou, LeDoux & Phelps, 2009), money (Bjork, Smith, Chen & Hommer, 2010; Forbes *et al*., 2010), and points (Schneider *et al*., 2012) as the reinforcers of behaviour. A primary aim of this study was to compare adolescents' anticipatory neural responses to both rewards and threats, and therefore comparable rewarding and threatening reinforcers were essential. Pictures pose an ethical problem in developmental work since the pictures 18-23 year olds find rewarding and threatening are not age-appropriate for younger adolescents. Furthermore, money may have a different meaning for younger adolescents compared to older adolescents (Barkley-Levenson & Galván, 2014). Moreover, there are no appetitive stimuli that equate to electric shocks and aversive tones. In comparison, points have been used successfully with adolescents in behavioural studies using the BART-Y (Lejuez *et al*., 2007) and in fMRI studies using instrumental tasks (Schneider *et al*., 2012). Therefore, points were judged to be the most suitable reinforcer of behaviour for this research.

2.4.2.1.3. Practice trials

Minimising behavioural differences between age groups can be advantageous when examining age-related differences in neural activity because age-dependent

differences in neural activity are more likely to reflect the motivational or affective content of the task, rather than developmental differences in task demands (Casey, 2002). The ERP study reported in Chapter 4 aimed to investigate age-related differences in anticipatory neural activity to rewards and threats, rather than age-related differences in task demands. To that end, potential age-related differences in task performance were minimised by requiring participants to learn the task contingencies; before the start of each block participants had to get 75% of the practice trials correct and verbally recall the correct response to each stimulus.

2.4.2.1.4. Pilot study

The instrumental conditioning task was piloted with 10 late adolescents. One participant failed to complete the task and therefore 9 participants aged 18-21 years (M_{age} = 19.33 years, SD_{age} = 1.22; 6 females) were included in the analyses. All participants completed the task to a high degree of accuracy (> 88% correct). Table 2.3 displays the means and 95% confidence intervals for accuracy and reaction time for each of the conditions.

Table 2.3
Instrumental conditioning task pilot data

		Accuracy (%)		Reaction Time (ms)	
Block	Condition	*M*	95% CI	*M*	95% CI
Reward	$Reward^{Go}$	96.93	94.78, 99.07	373	322, 424
	$Reward^{NoGo}$	97.52	94.15, 100.89		
	$Control^{Go}$	96.05	93.79, 98.31	362	305, 419
	$Control^{Go}$	97.81	94.86, 100.75		
Avoidance	$Avoidance^{Go}$	97.95	96.12, 99.78	336	276, 396
	$Avoidance^{NoGo}$	99.27	98.73, 99.80		
	$Control^{Go}$	97.22	96.04, 98.40	335	277, 393
	$Control^{NoGo}$	99.27	98.24, 100.29		

Note. 95% CI = 95% bootstrapped confidence intervals.

To assess whether accuracy differed between the conditions, repeated measures ANOVAs were conducted for the reward and avoidance blocks, with *Condition* (reward block: $Reward^{Go}$, $Reward^{NoGo}$, $Control^{Go}$, $Control^{NoGo}$; avoidance block: $Avoidance^{Go}$, $Avoidance^{NoGo}$, $Control^{Go}$, $Control^{NoGo}$) as the within-group factor. For the

reward block, no main effect of *Condition* was found ($F(3, 24) = 1.01$, $p = 0.407$, $\eta_p^2 = 0.11$). By contrast, the main effect *Condition* reached significance for the avoidance block ($F(3, 24) = 5.43$, $p = 0.005$, $\eta_p^2 = 0.40$). Pairwise comparisons revealed very small but significant differences; participants made slightly more errors in the $Control^{Go}$ condition compared to the $Avoidance^{NoGo}$ ($p = 0.001$) and $Control^{NoGo}$ ($p = 0.015$) conditions, indicating that more errors were made to conditions that required a motor action compared to conditions that required inhibition of a motor action. Repeated measures ANOVAs were also conducted to assess reaction time to Go conditions, with *Condition* (reward block: $Reward^{Go}$, $Control^{Go}$; avoidance block: $Avoidance^{Go}$, $Control^{Go}$) as the within-group factor. Only correct trials were included in the reaction time analyses. No main effects of *Condition* were found for the reward block ($F(1, 8) = 1.49$, $p = 0.257$, $\eta_p^2 = 0.16$) or avoidance block ($F(1, 8) = 0.09$, $p = 0.769$, $\eta_p^2 = 0.01$), suggesting that reaction times were equivalent across the different conditions. Since accuracy and reaction times were largely comparable across conditions, and participants completed the task with a high degree of accuracy, this task was used to assess adolescents' anticipatory neural responses to rewards and threats (Chapter 4).

2.4.2.2. Resting state EEG

Chapter 5 presents an EEG study examining the cortical sources of resting alpha and their relationship to pubertal stage and risk-taking behaviours during adolescence. On the basis of previous adolescent and adult studies (Black *et al*., 2014; Schutter, de Haan & van Honk, 2004; Wheeler *et al*., 1993), six one-minute intervals of resting state EEG data were recorded. Participants were instructed to keep their eyes open for three one-minute sessions and closed for three one-minute intervals in an alternating order (open-closed-open-closed-open-closed). There was a 10 second buffer between intervals to allow the experimenter to verbally instruct participants to open or close their eyes. The resting state EEG data were recorded in the same session as the instrumental conditioning task (Chapter 4).

2.4.3. EEG preprocessing

All EEG preprocessing was conducted offline. EEG data were downsampled from 2048 to 512 Hz using Biosemi's decimator tool. Importantly, Biosemi's decimator tool applies a fifth order sinc filter to prevent aliasing. ERPLAB 5.0 (Lopez-Calderon & Luck, 2014) and EEGLAB 13.5.4b (Delorme & Makeig, 2004) were used to preprocess

the EEG signals. Figure 2.4 depicts the preprocessing streams for the ERP (Chapter 4) and resting state (Chapter 5) data. The subsequent paragraphs briefly outline the preprocessing steams for the ERP and resting state data.

The downsampled ERP data were imported into EEGLAB using the vertex (Cz) as the reference. Trials with incorrect behavioural responses were removed from the data to ensure that only trials with correct behavioural responses were included in the ERP analyses. ERPLAB was used to remove the direct current offset and band-pass filter the continuous EEG data between 0.1 and 30 Hz. EEGLAB was used for the subsequent analyses. The continuous data were epoched between -200 and 800 ms, and electrode channels that resulted in the rejection of more than 25% of trials were deleted. Next, occular artefacts were corrected (see Chapter 2.4.3.1), and deleted electrode channels were interpolated to ensure that all participants had 64 channels available for analysis. Epochs were then baseline corrected between -200 and 0 ms, and epochs with amplitude differences larger than ±150 μV were rejected to remove any additional artefacts from the data (see Chapter 2.4.3.1). All participants had a minimum of 25 epochs per condition following artefact rejection. The epochs for each condition were subsequently averaged together to create grand average ERP waveforms for each group. Finally, the N170 and LPP were quantified and statistically analysed (see Chapter 4.2.6 for details).

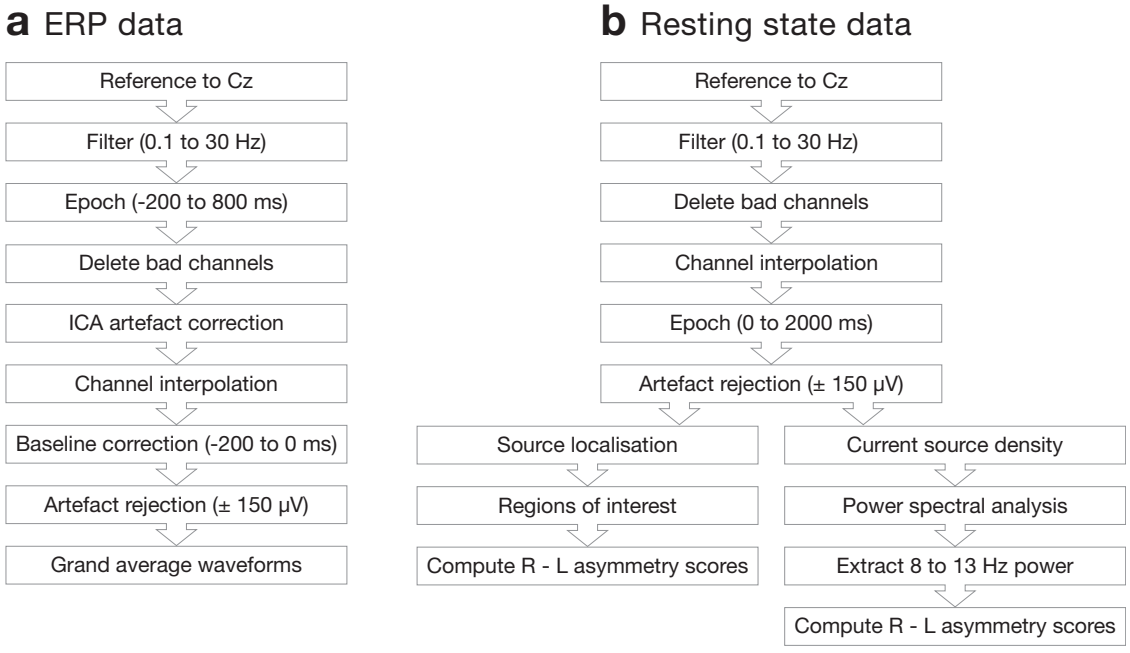

Figure 2.4 Preprocessing stream for the ERP (**a**) and resting state (**b**) data.

The resting state data comprised both eyes-open and eyes-closed conditions. The eyes-open and eyes-closed conditions were analysed separately but followed an identical processing stream. In the same way as the ERP data, the downsampled resting state data were imported into EEGLAB using the vertex (Cz) as the reference. ERPLAB was used to remove the direct current offset and band-pass filter the continuous EEG data between 0.1 and 30 Hz. EEGLAB was used for the subsequent analyses. Electrode channels that resulted in the rejection of more than 25% of data were deleted and subsequently interpolated to ensure that all participants had 64 electrode channels available for analysis. In line with similar studies (e.g., Black *et al*., 2014; Harmon-Jones & Allen, 1998), the continuous resting state data were epoched into 367 2-second epochs that overlapped by 0.5 seconds to prevent data loss. Next, epochs with amplitude differences larger than ±150 μV were rejected to remove artefacts from the data. The cleaned, epoched data were then subjected to further processing to localise the cortical sources of spontaneous alpha (see Chapter 2.4.3.3 and Chapter 5.2.5.1 for details) and to compute frontal alpha asymmetry scores (see Chapter 2.4.3.2 and Chapter 5.2.5.2 for details).

The following sections discuss artefact rejection and correction (Chapter 2.4.3.1), referencing and current source density (Chapter 2.4.3.2), and source localisation (Chapter 2.4.3.3) in more detail.

2.4.3.1. Artefact rejection and correction

Although EEG intends to exclusively record electrical neural activity, the electrodes also record electrical activity resulting from non-neural sources. Typical EEG artefacts include eye blinks, lateral eye movements, sweating, muscle movement such as jaw clenching, poor electrode impedances, and electrical noise. Artefacts considerably reduce the signal-to-noise ratio (SNR) of the EEG signal, and therefore need to be removed. Many artefacts can be minimised during recording by instructing participants to remain as still as possible, ensuring the electrodes are well connected, and keeping the room cool to minimise slow drifts resulting from sweat. However, it is impossible to prevent all artefacts, and thus many artefacts require removal during preprocessing. High-pass filters (< 1 Hz) can remove very low frequency artefacts such as sweating, and low-pass filters (> 30 Hz) can remove high frequency artefacts such as electrical noise. Moreover, noisy channels resulting from poor electrode connections can be removed. However, ocular and muscular artefacts need to be removed using

artefact rejection and/or artefact correction. Artefact rejection identifies and rejects epochs or periods of continuous data where the EEG signals reach a particular amplitude threshold. While artefact rejection aims to improve the SNR by removing noise from the EEG signals, artefact rejection can actually result in poor SNR because a considerable amount of neural activity is rejected along with the artefact. This is a particular issue in developmental work since children tend to move more than adults, and thus there tend to be a greater number of artefacts in the EEG signal.

In an attempt to address this, artefacts can be corrected rather than rejected. Compared to artefact rejection, artefact correction involves isolating and correcting artefacts in specific channels instead of rejecting all EEG channels. Artefact correction therefore prevents the unnecessary loss of data, and thus improves the SNR. While artefact correction does not work well for irregular and unpredictable artefacts, artefact correction is a very reliable technique for removing large and predictable artefacts such as eye blinks and lateral eye movements (Jung *et al.*, 1998; Jung *et al.*, 2000). Independence components analysis (ICA) is a widely used technique for correcting ocular artefacts. ICA works by decomposing the EEG channel data into maximally independent components that reflect distinct signals.

Both artefact rejection and artefact correction were used to remove artefacts from the ERP data (Chapter 4). ICA was used to identify and correct occular eye movements and artefact rejection was used to remove other artefacts, such as muscle

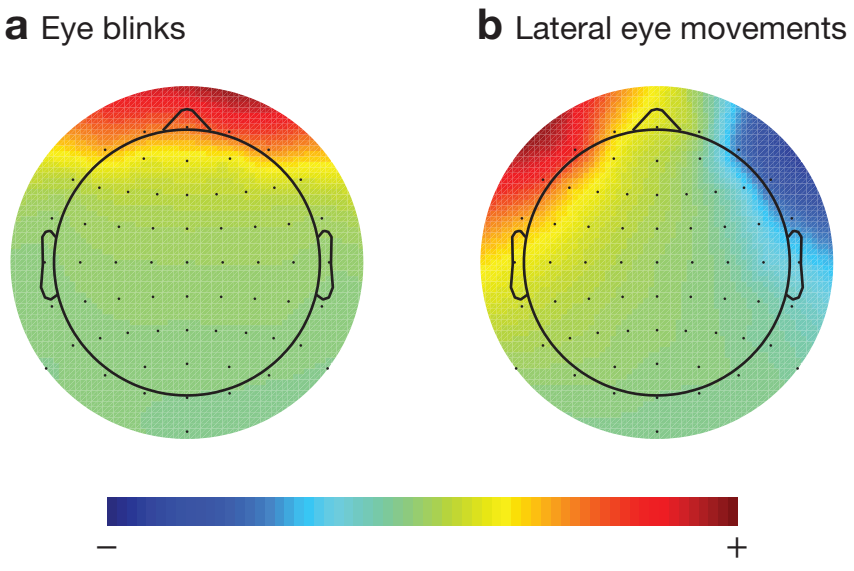

Figure 2.5 Example independent components for eye blinks (**a**) and lateral eye movements (**b**).

movement. Based on recent recommendations (Luck, Lopez-Calderon, Huang & Fo, 2013), trials with eye blinks occurring within 200 ms of the stimulus onset were rejected prior to running the ICA to ensure that only epochs where participants had seen the visual stimulus were included in the analyses. After the data had been filtered and epoched, and noisy electrode channels had been delected, the channels were decomposed using ICA into independent components (the number of channels minus one determines the number of independent components in EEGLAB). The two components for participants' eye blinks and lateral eye movements were identified and corrected (Figure 2.5). ICA data were then transformed back into channel data for the remaining analyses.

In comparison to the ERP data, only artefact rejection was used for the resting state data (Chapter 5). The resting state data directly compared spontaneous alpha when participants' eyes were open and closed. Since eye movements could only be corrected in the eyes-open condition, applying artefact correction to the resting state data would result in considerable differences in the preprocessing stream between the eyes-open and eyes-closed conditions. Accordingly, only artefact rejection (±150 μV threshold) was used for the resting state data.

2.4.3.2. Referencing and current source density

For most EEG systems, the EEG signal at each electrode results from three electrodes: active (A), ground (G), and reference (R). Specifically, the EEG signal at each electrode reflects the potential difference between the active electrode and ground electrode (A − G), as well as the potential difference between the reference electrode and ground electrode (R − G). Thus, the EEG signal reflects (A − G) − (R − G) = A − R. The ground electrode is required for amplifying the very small EEG signals, and does so through a ground circuit in an amplifier. However, the ground circuit introduces electrical noise into the EEG signal, which needs to be removed. The reference electrode is therefore used to measure the potential difference between the reference electrode and ground electrode in order to cancel out the ground signal entirely (Luck, 2014).

Hence, most EEG studies are required to use a reference electrode during recording. In this research, the EEG signals were recorded using the BioSemi ActiveTwo system. Rather than a ground electrode, the BioSemi Active Two system uses an active CMS electrode and a passive DRL electrode. The CMS electrode is

similar to the ground electrode in that the potential differences between each active electrode and the CMS electrode are recorded. However, the DRL electrode introduces a very small voltage into the scalp to drive the average scalp potential down so that it is comparable to the potential of the amplifier ground circuit. This removes the need for a reference electrode during recording since the potential differences between the scalp and amplifier are minimised during recording. Nevertheless, EEG signals recorded using BioSemi need referencing offline during preprocessing in order to maximise the SNR. Common EEG references include the average reference, the vertex, linked mastoids, and linked ears. When using reference electrodes, the reference electrode should ideally be placed at an electrically neutral site (Rossion & Jacques, 2012). However, there is no electrically neutral site on the scalp, and therefore the choice of reference significantly influences the ERP waveform (Luck, 2014). The vertex was selected to be the reference for the ERP data since it is not biased towards one hemisphere and does not introduce a lot of noise, such as muscle movement, into the data (Luck, 2014).

In addition to examining ERPs, this doctoral work examined frontal alpha asymmetry (Chapter 5). Recent work has shown that frontal alpha asymmetry scores that have been transformed to current source density (CSD) estimates, rather than referenced to standard references such as the average reference or linked mastoids, are significantly less influenced by extraneous factors such as the time of year and day (Velo, Stewart, Hasler, Towers & Allen, 2012). The benefits of using CSD over reference electrodes for EEG data have also been extensively documented by several reviews (Hagemann, 2004; Kamarajan, Pandey, Chorlian & Porjesz, 2015; Kayser & Tenke, 2015a; Kayser & Tenke, 2015b). Crucially, CSD is reference-free, and represents a higher resolution and spatially enhanced topography of the neural generators that underlie the scalp-recorded signals (Burle *et al*., 2015; Kayser *et al*., 2006; Kayser & Tenke, 2015b; Tenke & Kayser, 2005). In light of these benefits, the frontal alpha asymmetry data in this doctoral work were transformed to CSD estimates. In order to maximise the SNR, the data were imported into EEGLAB using the vertex (Cz) as the reference and subsequently transformed to CSD estimates.

EEG signals are transformed to CSD estimates by computing the second spatial derivative of the scalp voltage distribution. The second spatial derivate is computed by first measuring the voltage difference between each electrode and the average voltage of the surrounding electrodes (first derivative), and then computing the difference of

each newly created difference value and the average difference values from surrounding electrodes (second derivative) (Luck, 2014). Thus, CSD represents the outward flow of current at each point on the scalp instead of the potential differences between each active electrode and the reference electrode. Therefore, CSD transformed scalp topographies amplify contributions from local sources and attenuate volume-conducted contributions from more distal sources (Burle *et al.*, 2015; Kayser *et al.*, 2006; Kayser & Tenke, 2015b; Tenke & Kayser, 2005). Volume conduction refers to the transmission of current through different types of biological tissue, such as grey matter, cerebral spinal fluid, skin, and the skull. The different types of biological matter have different conductive properties, which causes the current to spread out through the brain as the current travels from the neural source to the scalp electrode. Consequently, the EEG signal at each electrode does not necessarily represent the activity of local brain sources, but the activity from a number of spatially dispersed sources. CSD therefore improves the spatial resolution of the scalp-recorded EEG signals by reducing the negative impact of volume conduction. CSD is measured in $\mu V/cm^2$ and is therefore a measure of both magnitude and space.

2.4.3.3. Source localisation

Source localisation was used to examine age- and gender-related differences in the cortical sources of spontaneous alpha (Chapter 5). A number of source localisation solutions have been developed to estimate the cortical sources underlying scalp-recorded EEG activity. One extensively used and widely validated source localisation

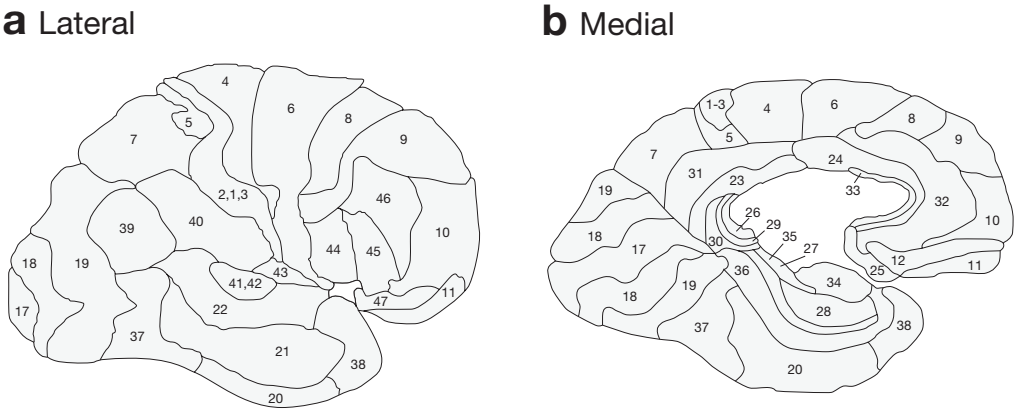

Figure 2.6 Human Brodmann areas for the lateral (**a**) and medial (**b**) cortical surface.

solution is sLORETA (standardized low-resolution brain electromagnetic tomography; Pascual-Marqui, 2002). sLORETA provides a three-dimensional distributed, linear, minimum norm inverse solution that computes CSD estimates from scalp-recorded EEG activity. sLORETA partitions the cortical grey matter into 6239 voxels (5 mm resolution) using the electrode positions established by the Montreal Neurological Institute 152 template, and defines regions of interest (ROIs) using Brodmann areas (BA) (Figure 2.6). Critically, sLORETA has been validated by numerous combined EEG-fMRI studies (e.g., Olbrich *et al*., 2009; Mobascher *et al*., 2009), and independent reviews have found sLORETA to be highly reliable (Greenblatt, Ossadtchi & Pflieger, 2005; Grech *et al*., 2008; Sekihara, Sahani & Nagarajan, 2005). Consequently, sLORETA was used in this doctoral research to estimate how the cortical sources of spontaneous alpha change during the course of adolescence.

2.5. Statistical approach

IBM SPSS Statistics 22.0 was used for all statistical analyses. All statistical tests were two-tailed, and the significance level was set at $p < 0.05$. Statistical tests were bootstrapped where possible, and the false discovery rate was used to correct for multiple comparisons.

2.5.1. Bootstrapping

Behavioural and EEG outcome measures frequently have skewed distributions. While some statistical tests, such as analysis of variance, are robust to violations in normality, many are not. Transforming data and using non-parametric statistics are widely used solutions for skewed data. However, transforming data does not always normalise data distributions and non-parametric statistics are not always appropriate (Field, 2009). Bootstrapping is an alternative solution for dealing with skewed data (Efron & Tibshirani, 1993). Bootstrapping is based on the premise of statistical sampling, whereby a sample is used to estimate the characteristics of an entire population. In bootstrapping, the sample is treated as a population from which smaller 'bootstrap' samples are selected. The statistical test of interest is calculated in each bootstrap sample. The sampling distribution of the statistic can then be estimated. The standard error (the standard deviation of the sampling distribution) can be used to determine the 95% bootstrapped confidence intervals and *p* values. Bootstrapping can therefore derive robust estimates of standard errors and confidence intervals for

means, t-tests, correlation coefficients, and regression coefficients. IBM SPSS Statistics can bootstrap the standard errors and confidence intervals for means, t-tests, correlation coefficients, regression coefficients, and ANOVA pairwise comparisons with the exception of ANOVA tests that include within-group factors. Thus, all means, t-tests, correlation coefficients, regression coefficients, and ANOVA pairwise comparisons, with the exception of repeated-measures and mixed-design ANOVAs, in this doctoral research were bootstrapped using bias-corrected and accelerated 95% confidence intervals based on 1000 samples, in line with current recommendations (Field, 2009). Bootstrapped confidence intervals and *p* values are reported where analyses have been bootstrapped.

2.5.2. False discovery rate

Computing multiple comparisons without correcting the significance level considerably inflates the Type I error rate. The traditional approach to the multiple comparisons problem is to control the familywise error rate (FWER). The Bonferroni correction is the most common approach, and controls the FWER by dividing the significance level (usually 0.05) by the number of statistical tests. However, when conducting a large number of comparisons, the Bonferroni correction is very conservative, which inflates the Type II error rate.

An alternative method to the Bonferroni correction is the Benjamini-Hochberg (B-H) correction (Benjamini & Hochberg, 1995). The B-H correction has more statistical power than the Bonferroni correction because the B-H correction controls the false discovery rate (FDR) rather than the FWER. The FDR reflects the proportion of significant results that are false positives. For instance, an FDR of 0.1 implies that 10% of significant findings are false positives. The B-H correction ranks the *p* values for each 'family' of statistical tests from smallest to largest. The *p* values are then systematically compared against the B-H thresholds. The B-H thresholds are computed by dividing the rank of a particular *p* value by the total number of statistical tests to be computed, and then multiplying by the FDR. The smallest *p* value needs to reach the first and most stringent threshold before any other *p* value can be deemed significant. If the smallest *p* value reaches the significance threshold, the second smallest *p* value is evaluated. If the second smallest *p* value reaches its threshold, the third smallest *p* value is evaluated, and so on.

When the B-H procedure is used with an FDR greater than 0.05, it is possible

for tests to be significant even though their *p* value is greater than 0.05. The FDR is largely dependent on the type of research being conducted; an FDR of 0.05 or smaller is appropriate for specific hypothesis-driven experiments, whereas an FDR of 0.1 or 0.2 is more suitable for exploratory research since it helps to generate hypotheses for further investigation (McDonald, 2014). The studies presented in this doctoral research were designed to generate hypotheses for future research, and therefore an FDR of 0.1 was used. All correlations, regressions, and ANOVA pairwise comparisons in this research were corrected for multiple comparisons using the B-H procedure. B-H corrections were conducted in Microsoft Excel using the bootstrapped *p* values.

2.6. Summary

The same cohort of participants took part in each of the three studies. In total, 105 participants aged 9-23 years took part in this research. Participants were categorised into three age groups: preadolescents aged 9-12 years; mid-adolescents aged 13-17 years; and late adolescents aged 18-23 years. Risk taking behaviours were measured using the BART (Lejuez *et al*., 2002) and YRBSS (Aklin *et al*., 2005). Sensation seeking and impulsivity were measured using the BSSS (Hoyle *et al*., 2002) and a Go/NoGo task, respectively. Anxiety was measured using the STAI (Spielberger *et al*., 1983), and depression was measured using the HADS-D (Zigmond & Snaith, 1983). Participants aged 9-17 years also completed the PDS (Petersen *et al*., 1988) to measure their current pubertal stage. Two EEG tasks were used to measure the EEG correlates of the approach-avoidance systems during adolescence: an instrumental conditioning task measured anticipatory neural responses to visual cues that predicted rewarding and threatening outcomes (Chapter 4), and a resting state EEG session measured spontaneous alpha (Chapter 5).

Chapter 3

Gender differences in the relationships between risk-taking and anxiety during adolescence

Abstract

This study first aimed to investigate the age- and gender-related differences in risk-taking, sensation seeking, impulsivity, and anxiety adolescents aged 9-23 years. Next, this study aimed to examine gender differences in the relationship between risk-taking and anxiety during distinct stages of adolescence in order to shed light on the simultaneous increase in risk-taking and anxiety during adolescence. Finally, this study aimed to examine the influence of puberty on the development of risk-taking, sensation seeking, impulsivity, and anxiety in male and female adolescents aged 9-17 years while controlling for chronological age. To these ends, 37 preadolescents (9-12 years; 18 females), 32 mid-adolescents (13-17 years; 16 females), and 36 late adolescents (18-23 years; 20 females) completed a battery of self-report questionnaires measuring real world risk-taking (YRBSS), sensation seeking (BSSS), and anxiety levels (STAI), and two behavioural tasks measuring risk-taking propensity (BART) and impulsivity (Go/NoGo). Participants aged 9-17 years also completed a self-report questionnaire measuring their current pubertal stage (PDS). This study found that BART risk-taking increased from preadolescence to late adolescence. By contrast, YRBSS risk-taking increased from preadolescence to mid-adolescence for males and females, but from mid-adolescence to late adolescence for females only. Sensation seeking remained stable across adolescence, but impulsivity decreased from preadolescence to mid-adolescence. Anxiety levels were greater in preadolescents compared to mid-adolescents and late adolescents, and in females compared to males. Critically, the relationship between risk-taking and anxiety changed significantly during the course of adolescence, and followed different developmental trajectories for males and females. Finally, more advanced pubertal stage was associated with greater levels of YRBSS risk-taking for males and females aged 9-17 years. Overall, the findings from this study emphasise the need to tease out the effects of age, gender, and puberty on the development of risk-taking, sensation seeking, impulsivity, and anxiety during adolescence.

3.1. Introduction

This study had three aims. Firstly, this study aimed to investigate the age- and gender-related differences in risk-taking behaviours, sensation seeking, impulsivity, and anxiety levels in a sample of typically developing preadolescents (9-12 years), mid-adolescents (13-17 years), and late adolescents (18-23 years). Secondly, this study aimed to examine the gender differences in the relationship between risk-taking behaviours and anxiety levels during preadolescence, mid-adolescence, and late adolescence. Finally, this study aimed to examine the influence of puberty on the development of risk-taking behaviours, sensation seeking, impulsivity, and anxiety levels in male and female adolescents aged 9-17 years while controlling for chronological age.

The introduction to this study begins by outlining how risk-taking behaviours, sensation seeking tendencies, impulsivity, and anxiety levels change across adolescence (Chapter 3.1.1). This introduction then discusses what is currently known about the relationship between risk-taking and anxiety in adults and adolescents (Chapter 3.1.2), and the role of puberty in the development of risk-taking, sensation seeking, impulsivity, and anxiety during adolescence (Chapter 3.1.3). Finally, this introduction outlines the aims and hypotheses of the current study (Chapter 3.1.4).

3.1.1. Development of risk-taking behaviours, sensation seeking, impulsivity, and anxiety levels during adolescence

3.1.1.1. Risk-taking behaviours, sensation seeking, and impulsivity

A number of methodologies have been used to examine how risk-taking behaviours change throughout development. The most widely used method is retrospective self-report questionnaires, where individuals are asked to report their recent engagement in a range of risky behaviours. There is converging evidence from studies using self-report questionnaires showing that engagement in risk-taking behaviours increases from childhood to adolescence, peaks in adolescence, and decreases from adolescence to adulthood. This inverted u-shaped trajectory has been found for a range of risky behaviours, such as substance use, unprotected sex, carrying a weapon, and physical fighting (Donovan & Jessor, 1985; Eaton *et al*., 2010; Gullone *et al*., 2000; Kann *et al*., 1999; Windle *et al*., 2008).

While self-report questionnaires have provided important insights into the development of risk-taking behaviours, there are a number of ethical and practical

issues with using self-report questionnaires to measure risk-taking behaviours in developmental samples (see Chapter 2.3.1). In light of these concerns, there has been a shift towards developing behavioural tasks that measure risk-taking behaviours in laboratory settings (e.g., Burnett *et al*., 2010; Collado *et al*., 2014). Behavioural tasks are particularly useful for measuring risk-taking behaviours in children and adolescents because they are able to minimise or eliminate many of the potential confounds associated with using self-report questionnaires in developmental samples (Ladouceur *et al*., 2000; Lejuez *et al*., 2002, 2003). However, it is important to note that behavioural tasks measure risk-taking propensity rather than real world risk-taking behaviours, and may therefore tap into a different construct of risk-taking. Despite this concern, performance on behavioural risk-taking tasks has been shown to correlate with self-report questionnaires measuring recent engagement in risky behaviours in both adolescents and adults (Lejuez *et al*., 2002, 2007). Thus, using behavioural tasks in addition to self-report questionnaires should provide a more comprehensive, and less confounded, understanding of how risk-taking behaviours develop throughout adolescence.

The first aim of this study was to investigate the age- and gender-related differences in risk-taking behaviours, sensation seeking, impulsivity, and anxiety levels in a sample of typically developing preadolescents (9-12 years), mid-adolescents (13-17 years), and late adolescents (18-23 years). In light of the previous literature, risk-taking behaviours in the current study were measured using an extensively used and well-validated behavioural task, the BART (Lejuez *et al.,* 2002), and an age-appropriate retrospective self-report questionnaire, the YRBSS (Aklin *et al*., 2005).

The BART requires participants to pump up a virtual balloon in order to win money or points (see Chapter 2.3.1.1 for more details regarding the BART). The BART has been used to measure risk-taking behaviours in children, adolescents, and young adults (Aklin *et al*., 2005; Lejuez *et al*., 2002, 2003, 2007; South, Dana, White & Crowley, 2011), and is therefore an appropriate behavioural task for measuring risk-taking behaviours in developmental samples. Consistent with studies using self-report questionnaires, studies using behavioural tasks to measure the development of risk-taking behaviours have also reported that risk-taking follows an inverted u-shaped developmental trajectory, whereby risk-taking behaviours increase from childhood to adolescence, peak in adolescence, and decrease from adolescence to adulthood (Burnett *et al*., 2010; Eshel *et al*., 2007; Figner *et al*., 2009). However, no study to date

has examined BART risk-taking in a sample with an age range wide enough to assess how risk-taking develops across the entire course of adolescence. Therefore, using the BART to measure risk-taking behaviours in a normative sample of adolescents aged 9-23 years will provide novel insights into how risk-taking behaviours, as measured by the BART, develop throughout adolescence.

It has been suggested that the developmental trajectory of risk-taking behaviours, as measured by both self-report questionnaires and behavioural tasks, is largely dependent on age-related changes in sensation seeking and impulsivity during childhood and adolescence (Steinberg *et al*., 2008). While sensation seeking and impulsivity are frequently combined into a single construct (Zuckerman, 1996), there is considerable evidence to suggest that sensation seeking and impulsivity are conceptually and empirically distinct constructs that follow different developmental trajectories during adolescence (Harden & Tucker-Drob, 2011; Shulman *et al*., 2014, 2015; Steinberg *et al*., 2008). Sensation seeking refers to a high desire to seek out novel, varied, complex, and intense experiences and sensations, and a willingness to take risks for the sake of such experiences and sensations (Zuckerman, 1994). In comparison, impulsivity refers to a lack of self-control, rapid decision-making, and a tendency to act without adequate thought (Moeller *et al*., 2001; Steinberg *et al*., 2008). Sensation seeking has been shown to follow the same trajectory as risk-taking behaviours, whereby sensation seeking increases from childhood to adolescence, peaks in adolescence, and decreases from adolescence to adulthood (Harden & Tucker-Drob, 2011; Shulman *et al*., 2014, 2015; Steinberg *et al*., 2008; Zuckerman *et al*., 1978). By contrast, studies have consistently reported that impulsivity steadily decreases, as impulse control increases, during childhood and adolescence (Casey *et al*., 1997; Harden & Tucker-Drob, 2011; Shulman *et al*., 2014; Shulman *et al*., 2015; Smith *et al*., 2012; Steinberg *et al*., 2008).

Based on these findings, it has been proposed that the peak in risk-taking behaviours during adolescence results from an increased motivation to seek out novel and exciting experiences combined with immature self-regulatory abilities that are unable to modulate such tendencies (Casey *et al*., 2008b; Steinberg *et al*., 2008). In the same way, the decrease in risk-taking from adolescence to adulthood is thought to result from a reduction in sensation seeking tendencies combined with improvements in impulse control and self-regulation (Steinberg *et al*., 2008). Accordingly, the current study measured participants' sensation seeking tendencies and impulsiveness along

with their risk-taking behaviours in order to assess the contributions of sensation seeking and impulsivity to the development of risk-taking behaviours in this sample of adolescents.

Notably, gender differences in risk-taking behaviours are frequently observed using both self-report and behavioural measures, with males being more likely to engage in risk-taking behaviours than females throughout the lifespan (Byrnes *et al*., 1999; Gullone *et al*., 2000; Turner & McClure, 2003; Van Leijenhorst *et al*., 2008). Consistently, studies have also reported that males exhibit higher levels of sensation seeking (Cross, Cyrenne & Brown, 2013) and impulsivity (Chapple & Johnson, 2007) compared to females, which may partially explain why males engage in more risk-taking behaviours than females.

The developmental trajectories of risk-taking, sensation seeking, and impulsivity are considered to be well established (Steinberg *et al*., 2008). However, only a handful of studies have measured the development of risk-taking, sensation seeking, and impulsivity in a cohort of adolescents with an age range wide enough to examine how these constructs change across the course of adolescence (Steinberg *et al*., 2008). Moreover, these studies have largely overlooked potential gender differences in the development of risk-taking, sensation seeking, and impulsivity. Consequently, examining the development of risk-taking, sensation seeking, and impulsivity in a sample of male and female adolescents aged 9-23 years will not only reveal how these constructs develop in this sample of adolescents, but will also add to the current evidence base of how these constructs develop across the course of adolescence for males and females.

3.1.1.2. Anxiety levels

Adolescence is not only a vulnerable period for engaging in potentially harmful risk-taking behaviours, but also for developing an anxiety disorder and experiencing elevated anxiety levels. Anxiety disorders are more likely to emerge in adolescence than any other time of life (Kessler *et al*., 2005), and are the most prevalent psychiatric disorders during adolescence (Merikangas *et al*., 2010). A recent epidemiological study conducted in the United States reported that as many as 32 per cent of adolescents meet the criteria for an anxiety disorder at any one time (Merikangas *et al*., 2010).

As well as being at an increased risk for developing an anxiety disorder, adolescents are also at more likely to experience heightened anxiety levels compared

to children and adults; a number of studies have found that self-report anxiety symptoms reach a lifetime peak during adolescence (Abe & Suzuki, 1986). In contrast to these findings, several studies have reported that anxiety symptoms in typically developing samples remain relatively stable across childhood and adolescence (Bosquet & Egeland, 2006; Gullone, King & Ollendick, 2001). Moreover, one study has reported that anxiety levels initially decrease from preadolescence to early adolescence (10-13 years), but subsequently increase across mid-adolescence (14-17 years) (Van Oort *et al*., 2009). Critically, these studies did not include participants older than 17 years, and are therefore unable to determine whether the children and adolescents in these samples had greater or equivalent anxiety levels than late adolescents and young adults. Nevertheless, these studies suggest that there are considerable individual differences in anxiety levels in typically developing children and adolescents.

The current study focused exclusively on typically developing adolescents given that the majority of adolescents have not been diagnosed with, or meet the criteria for, an anxiety disorder. Notably, it has been widely reported that females are more likely to be diagnosed with an anxiety disorder and experience greater levels of anxiety compared to males throughout the lifespan (Abe & Suzuki, 1986; Lewinsohn *et al*., 1998; Van Oort *et al*., 2009). Therefore, the current study aimed to examine both the age- and gender-related differences in state and trait anxiety levels in adolescents aged 9-23 years. Given that anxiety is highly comorbid with depression during childhood, adolescence, and adulthood (Brady & Kendall, 1992; Hirschfeld, 2001), participants' depression levels were also measured in the current study in order to control for the influence of depression on participants' anxiety levels.

3.1.2. The relationship between risk-taking and anxiety during adolescence

The literature discussed so far provides cogent evidence that adolescence is associated with significant increases in both risk-taking behaviours and anxiety levels. In adults, high levels of anxiety are associated with reduced risk-taking behaviours, as measured by both self-report questionnaires and behavioural tasks (Broman-Fulks *et al*., 2014; Giorgetta *et al*., 2012; Lorian & Grisham, 2010; Maner *et al*., 2007; Maner & Schmidt, 2006). Thus, the simultaneous increase in risk-taking behaviours and anxiety levels during adolescence seems paradoxical. However, it has been suggested that the simultaneous increase in risk-taking and anxiety during adolescence may have an

evolutionary purpose (Casey *et al*., 2008a, 2008b; Spear, 2000, 2009; Steinberg, 2008; see Chapter 1.3.3). The idea that the simultaneous increase in risk-taking and anxiety during adolescence has evolutionary antecedents is supported by nonhuman animal work; many mammalian species, including mice, rats, and nonhuman primates, show adolescent-specific changes in risk-taking, sensation seeking, and responses to stress (Laviola, Macri, Morley-Fletcher & Adriani, 2003; Spear, 2000). For instance, there is evidence that adolescent mice are more hyperactive and exhibit greater levels of novelty and sensation seeking behaviours compared to younger and older mice (Adriani, Chiarotti & Laviola, 1998). Moreover, there is evidence that adolescent rats are more sensitive to stress than adult rats; Doremus-Fitzwater, Varlinskaya and Spear (2009) found that adolescent rats had stress-induced reductions in body weight and less habituation of stress-induced corticosterone after being repeatedly restrained compared to adult rats.

Despite the simultaneous increase in risk-taking and anxiety seeming paradoxical, research assessing the relationship between risk-taking behaviours and anxiety levels during adolescence is limited. Initial work suggests that the relationship between risk-taking and anxiety is fundamentally different in adolescents compared to adults. Specifically, higher levels of anxiety in adolescents are associated with greater levels of real world risk-taking, as measured by self-report questionnaires (Comeau *et al*., 2001; Patton *et al*., 1996). Studies using behavioural tasks have also reported that higher levels of anxiety in adolescents are associated with greater levels of risk-taking, but only when adolescents experience acute social stress (Reynolds *et al*., 2013; Richards *et al*., 2015). Thus, instead of higher anxiety levels being associated with reduced risk-taking behaviours as reported in adults (e.g., Giorgetta *et al*., 2012), higher anxiety levels in adolescents appear to be associated with greater levels of risk-taking.

Previous studies examining the relationship between risk-taking and anxiety in adolescents provide preliminary evidence that the relationship between risk-taking and anxiety is different in adolescents compared to adults. However, the studies to date have been conducted with discrete age groups within adolescence. For example, Richards *et al*. (2015) examined the relationship between risk-taking and anxiety in a sample of adolescents aged 15-18 years. Critically, no study has yet examined how the relationship between risk-taking and anxiety changes throughout adolescence. Middle adolescence (13-17 years) is frequently reported to be the age group when risk-taking

behaviours and anxiety levels peak (e.g., Abe & Suzuki, 1986; Burnett *et al*., 2010). Therefore, examining the changes in the relationship between risk-taking and anxiety from preadolescence to mid-adolescence, as well as from mid-adolescence to late adolescence, should provide insights into why mid-adolescence is a particularly vulnerable period for experiencing elevated levels of risk-taking and anxiety. Furthermore, most studies investigating the relationship between risk-taking and anxiety in adolescents have not examined potential differences between males and females. This is surprising since there are considerable gender differences in risk-taking behaviours and anxiety levels during adolescence; males typically take more risks and females frequently have higher levels of anxiety (Byrnes *et al*., 1999; Lewinsohn *et al*., 1998). The current literature is therefore unable to shed light on how the relationship between risk-taking and anxiety changes during adolescence, and whether there are gender differences in such relationships. Importantly, high levels of risk-taking and anxiety during adolescence have been associated with a range of short- and long-term adverse consequences (Miniño, 2010; Newbury-Birch *et al*., 2009; Pine *et al*., 1998; Viner, 2012; Woodward & Fergusson, 2001). In order for interventions aimed at reducing the high levels of risk-taking and anxiety during adolescence to be most effective, we need a comprehensive understanding of the specific age- and gender-related differences in the relationship between risk-taking and anxiety. To these ends, the second aim of this study was to examine the potential gender differences in the relationship between risk-taking and anxiety in preadolescents (9-12 years), mid-adolescents (13-17 years), and late adolescents (18-23 years).

3.1.3. The influence of puberty on risk-taking behaviours, sensation seeking, impulsivity, and anxiety during adolescence

Most studies examining the development of risk-taking, sensation seeking, impulsivity, and anxiety have focused on how such constructs change as a function of age across childhood, adolescence, and young adulthood. However, there is a body of work suggesting that puberty has significant affects on the development of risk-taking behaviours, sensation seeking, and anxiety levels during adolescence (Collado *et al*., 2014; Martin *et al*., 2002; Reardon *et al*., 2009). Developmental sMRI and fMRI studies also suggest that puberty has significant affects on adolescent brain development (Braams *et al*., 2015; Bramen *et al*., 2011; De Macks *et al*., 2011; Forbes *et al*., 2011; Goddings *et al*., 2014; Moore *et al*., 2012; Neufang *et al*., 2009; Peper *et al*., 2009; see

Chapter 1.4). Crucially, the onset of puberty varies markedly between individuals; puberty can begin any time between the ages of 8 and 13 years for healthy females and between 9 and 14 years for healthy males (Sørensen *et al.,* 2013). Thus, chronological age and pubertal development are highly dissociable during adolescence (Marshall & Tanner, 1969, 1970). For instance, two adolescents of the same age may be at very different pubertal stages.

Therefore, while examining the development of risk-taking, sensation seeking, impulsivity, and anxiety across preadolescence, mid-adolescence, and late adolescence will extend our current understanding of how these constructs develop throughout adolescence, the findings will not be able to determine whether the developmental trajectories are due to chronological age, puberty, or a combination of the two. Moreover, since there are large individual differences in the age of pubertal onset, it is possible that grouping adolescents into discrete age groups (i.e., preadolescence, mid-adolescence, and late adolescence) will confound the developmental trajectories of risk-taking, sensation seeking, impulsivity, and anxiety. Accordingly, the final aim of this study was to examine whether pubertal stage was associated with risk-taking behaviours, sensation seeking, impulsivity, and anxiety levels in male and female adolescents aged 9-17 years while controlling for chronological age. The following sections briefly outline and evaluate what is currently known about the influence of puberty on risk-taking behaviours, sensation seeking, impulsivity, and anxiety levels during adolescence.

The empirical work to date suggests that earlier pubertal onset and more advanced pubertal stage are associated with greater levels of risk-taking behaviours during adolescence, after controlling for chronological age (Collado *et al*., 2014; Costello *et al*., 2007; Faden, Ruffin, Newes-Adeyi, & Chen, 2009; Patton *et al*., 2004). The majority of studies assessing the relationship between puberty and risk-taking have used self-reported questionnaires to measure engagement in risky behaviours. However, as discussed earlier, using self-report questionnaires to measures risk-taking in developmental samples can be problematic (Ladouceur *et al*., 2000; Lejuez *et al*., 2002; Lejuez *et al*., 2003; see Chapter 2.3.1). To date, only one study has examined the relationship between puberty and risk-taking using both a behavioural task (BART) and self-report questionnaire (YRBSS) (Collado *et al*., 2014). Collado *et al*. (2014) found that more advanced pubertal stage predicted greater risk-taking behaviours, as measured by both the BART and YRBSS, in adolescents aged 11-15 years. Notably, this

study did not explore potential gender differences in the relationship between puberty and risk-taking. Given that females usually begin puberty 1-2 years before males (Marshall & Tanner, 1969, 1970), and males tend to take more risks than females (Byrnes *et al*., 1999), it is possible that there will be gender-specific trajectories between puberty and risk-taking. Collado *et al*.'s (2014) study therefore needs extending to explore potential gender differences in the relationship between puberty and risk-taking.

Consistent with risk-taking, puberty has been shown to have significant affects on the development of sensation seeking during adolescence, whereby more advanced pubertal stage is associated with higher levels of sensation seeking, after controlling for chronological age (Forbes & Dahl, 2010; Martin *et al*., 2002). Notably, puberty is thought to contribute more to the increases in sensation seeking during adolescence than chronological age (Forbes & Dahl, 2010; Martin *et al*., 2002; Spear, 2000; Steinberg, 2008). Steinberg's DSM (Steinberg, 2008) and the SIPN (Nelson *et al*., 2005) propose that the gonadal hormones released during puberty have significant affects on neural activity within the socioemotional system (i.e., brain regions critical for affective processing). This increase in activity is thought to motivate adolescents to seek out rewarding and novel experiences, which in turn increases engagement in sensation seeking and risk-taking behaviours. Thus, it is possible that the relationships between puberty and measures of risk-taking and sensation seeking result from the influence of gonadal hormones on brain activity (Nelson *et al*., 2005; Steinberg, 2008).

In contrast to risk-taking and sensation seeking, the relationship between puberty and impulsivity is largely unknown. Initial evidence suggests that puberty has no affect on impulsivity in male and female adolescents aged 10-16 years (Steinberg *et al*., 2008). However, in order to determine the reliability of this finding, the relationship between puberty and impulsivity requires further investigation.

Finally, in line with risk-taking and sensation seeking, earlier pubertal onset and more advanced pubertal stage has been associated with higher levels of anxiety, after controlling for chronological age (Deardorff *et al*., 2007; Ge, Conger & Elder, 1996; Hayward *et al*., 1992; Patton *et al*., 1996; Reardon *et al*., 2009). Consistently, more advanced pubertal stage is also associated with greater sensitivity to threat (Moore *et al*., 2012; Quevedo, Benning, Gunnar & Dahl, 2009). Crucially, the relationship between puberty and anxiety appears to be largely gender-specific; most

studies have found a relationship between puberty and anxiety for females, but not for males (Reardon *et al*., 2009).

Collectively, these findings support the idea that there is a paradox during adolescence, whereby more advanced pubertal stage is associated with increases in risk-taking and sensation seeking behaviours in addition to increases in anxiety levels. Notably, previous studies examining the relationships between puberty and levels of risk-taking, sensation seeking, impulsivity, and anxiety have focused on linear relationships (e.g., Collado *et al*., 2014; Ge *et al*., 1996). However, risk-taking behaviours, sensation seeking, and anxiety levels have been shown to follow non-linear developmental trajectories across adolescence, whereby risk-taking behaviours, sensation seeking, and anxiety levels peak in middle adolescence (e.g., Abe & Suzuki, 1986; Burnett *et al*., 2010; Steinberg *et al*., 2008). Hence, our current understanding of the influence of puberty on risk-taking, sensation seeking, and anxiety may be limited by the exclusive use of linear relationships. Accordingly, the current study examined both the linear and nonlinear relationships between pubertal stage and measures of risk-taking, sensation seeking, impulsivity, and anxiety.

3.1.4. The current study

In the current study, 37 preadolescents aged 9-12 years (18 females, 19 males), 32 mid-adolescents aged 13-17 years (16 females, 16 males), and 36 late adolescents aged 18-23 years (20 females, 16 males) completed a battery of self-report questionnaires measuring recent real world risk-taking behaviours, sensation seeking tendencies, anxiety levels, and depression levels, two behavioural tasks measuring risk-taking propensity and impulsivity, and an IQ test. Preadolescents and mid-adolescents also completed a self-report questionnaire measuring their current pubertal stage.

This study had three aims. Firstly, this study aimed to investigate the age- and gender-related differences in risk-taking behaviours, sensation seeking, impulsivity, and anxiety levels across preadolescence, mid-adolescence, and late adolescence. Based on the research discussed above, it was predicted that risk-taking behaviours and sensation seeking tendencies would increase from preadolescence to mid-adolescence, peak in mid-adolescence, and subsequently decrease from mid-adolescence to late adolescence (e.g., Burnett *et al*., 2010; Shulman *et al*., 2014; Steinberg *et al*., 2008). It was also predicted that males would take more risks and have

greater sensation seeking tendencies than females during preadolescence, mid-adolescence, and late adolescence (Byrnes *et al*., 1999; Cross *et al*., 2013). Secondly, it was predicted that impulsivity would decrease from preadolescence to late adolescence for both females and males (Harden & Tucker-Drob, 2011; Steinberg *et al*., 2008). Finally, it was predicted that anxiety levels would be greater during mid-adolescence compared to preadolescence and late adolescence (Abe & Suzuki, 1986). It was also predicted that females would have greater anxiety levels than males, irrespective of age (Lewinsohn *et al*., 1998).

Secondly, this study aimed to examine the gender differences in the relationship between risk-taking behaviours and anxiety levels during preadolescence, mid-adolescence, and late adolescence. It was predicted that anxiety would have differential affects on risk-taking behaviours during preadolescence, mid-adolescence, and late adolescence. Based on the literature discussed above, the following predictions were made: (1) higher levels of anxiety would be associated with reduced levels of BART and real-world risk-taking in late adolescents (e.g., Giorgetta *et al*., 2012; Maner *et al*., 2007); (2) higher levels of anxiety would be associated with greater levels of BART and real world risk-taking in mid-adolescents (e.g., Patton *et al*., 1996; Reynolds *et al*., 2013); (3) no directional hypothesis was made for preadolescents since the relationship between risk-taking and anxiety in 9-12 year olds is currently unknown; (4) no directional hypotheses were made for males and females since gender differences in the relationship between risk-taking and anxiety have been scarcely studied.

Finally, this study aimed to examine the influence of puberty on the development of risk-taking behaviours, sensation seeking, impulsivity, and anxiety levels in male and female adolescents aged 9-17 years while controlling for chronological age. It was predicted that more advanced pubertal stage would be associated with greater levels of risk-taking, sensation seeking, and anxiety (Collado *et al*., 2014; Martin *et al*., 2002; Reardon *et al*., 2009). In contrast, it was predicted that pubertal stage would not be associated with impulsivity (Steinberg *et al*., 2008). Lastly, it was predicted that puberty would be associated with risk-taking and sensation seeking in males and females, but anxiety only in females (Reardon *et al*., 2009).

3.2. Method

3.2.1. Participants

One hundred and five participants aged 9-23 years took part in this study. Participants were split into three age groups: preadolescents aged 9-12 years; mid-adolescents aged 13-17 years; and late adolescents aged 18-23 years (Table 2.1). Detailed participant information is reported in Chapter 2.1.

3.2.2. Measures

3.2.2.1. Risk-taking behaviours

Risk-taking behaviours were measured using the BART (Lejuez *et al*., 2002) and YRBSS (Aklin *et al*., 2005) (see Chapter 2.3.1).

3.2.2.2. Sensation seeking

Sensation seeking was measured using the BSSS (Hoyle *et al*., 2002; see Chapter 2.3.2).

3.2.2.3. Impulsivity

Impulsivity was measured using a Go/NoGo task (see Chapter 2.3.3).

3.2.2.4. Anxiety and depression

Anxiety and depression were measured using the STAI (Spielberger *et al*., 1983) and HADS-D (Zigmond & Snaith, 1983), respectively (see Chapter 2.3.4 and Chapter 2.3.5).

3.2.2.5. Pubertal development

Current pubertal stage was assessed in females and males aged 9-17 years using the PDS (Petersen *et al*., 1988; see Chapter 2.3.6). The PDS was found to be highly reliable for both females (α = 0.87) and males (α = 0.89). An independent t-test revealed that PDS scores were significantly higher for females (M = 2.72, 95% CI = [2.38, 3.02]) compared to males (M = 2.29, 95% CI = [2.04, 2.56]) ($t(67)$ = 2.20, p = 0.031). This finding is consistent with previous work (Marshall & Tanner, 1969, 1970) and suggests that females in this sample were at a slightly later stage in their pubertal development compared to males.

3.2.2.6. Cognitive ability

Participants' cognitive ability was measured using the WASI-II (Wechsler, 2011; see Chapter 2.3.7) in order to control for potential affects of cognitive ability on risk-taking behaviours. The means and 95% confidence intervals for participants' IQ scores are presented in Table 2.1. A two-way ANOVA (*Age Group*, *Gender*) was used to compare IQ scores between groups. A main effect of *Age Group* was found ($F(2, 99) = 5.84$, $p = 0.004$, $\eta_p^2 = 0.11$), indicating that mid-adolescents had significantly lower IQ scores than preadolescents ($p = 0.002$) and late adolescents ($p = 0.004$). By contrast, IQ scores did not differ between preadolescents and late adolescents ($p = 0.486$). No main effect of *Gender* was found ($F(1, 99) = 0.42$, $p = 0.517$, $\eta_p^2 = 0.00$), and the *Age Group* by *Gender* interaction was non-significant ($F(2, 99) = 0.11$, $p = 0.893$, $\eta_p^2 = 0.00$).

3.2.3. Procedure

The procedure is reported in Chapter 2.2.

3.3. Results

3.3.1. Age and gender differences in risk-taking behaviours, sensation seeking, impulsivity, and anxiety

The first aim of this study was to examine the age and gender differences in risk-taking behaviours, sensation seeking tendencies, impulsivity, and anxiety levels in this sample of adolescents. Age and gender differences were examined using two-way ANOVAs (*Age Group*, *Gender*). Since IQ scores differed significantly between age groups, IQ scores were included as a covariate in all ANOVAs. ANOVA pairwise comparisons were bootstrapped and corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5). Figure 3.1 depicts the means and bootstrapped 95% confidence intervals for the BART, YRBSS, BSSS, Go/NoGo task, STAI, HADS-D, and STAI controlling for HADS-D. Supplementary Table 3.1 (Appendix 1) reports the means and bootstrapped 95% confidence intervals for the BART and YRBSS, Supplementary Table 3.2 (Appendix 1) reports the means and bootstrapped 95% confidence intervals for the BSSS and Go/NoGo, and Supplementary Table 3.3 (Appendix 1) reports the means and bootstrapped 95% confidence intervals for the STAI, HADS-D, and STAI controlling for HADS-D.

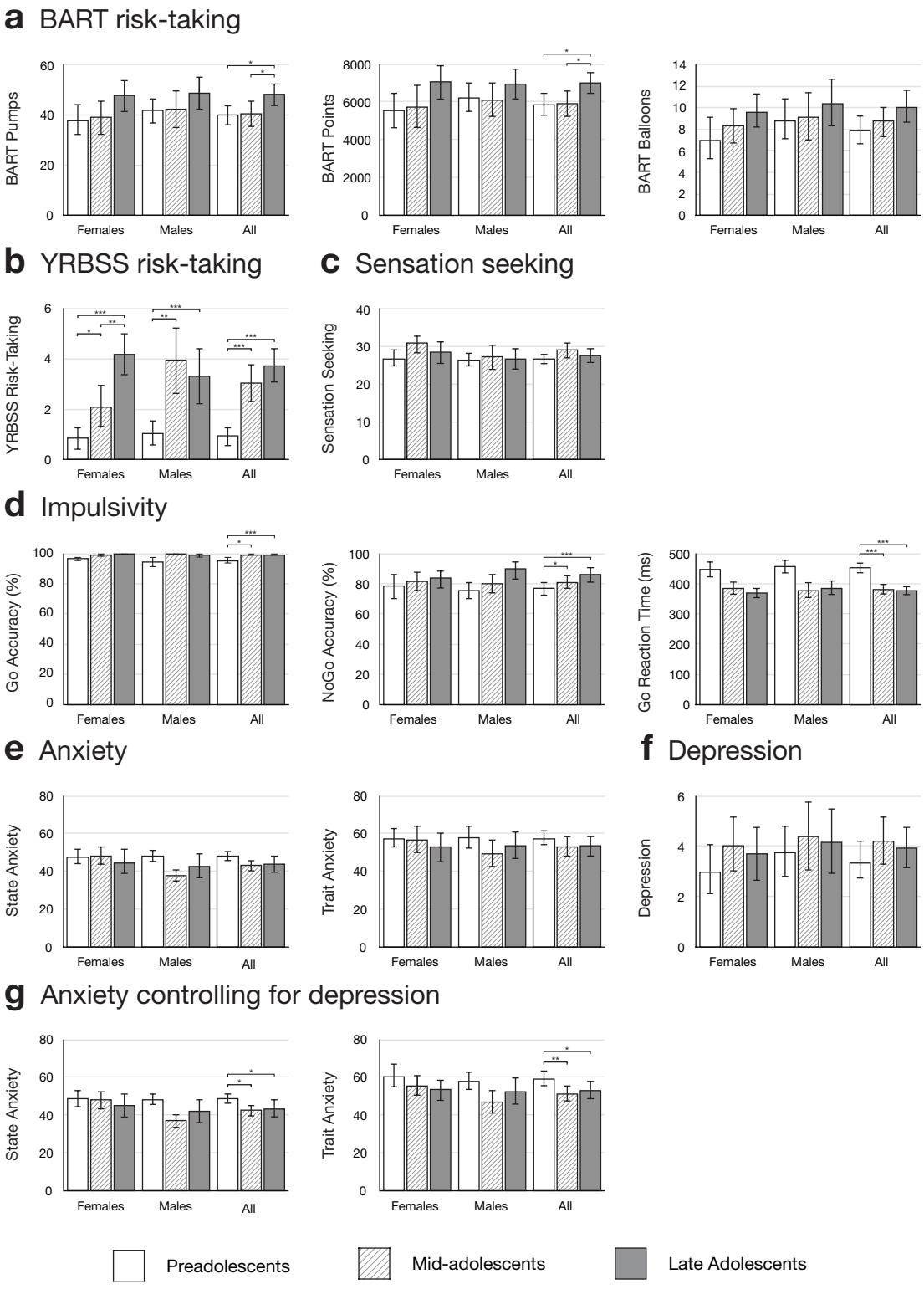

Figure 3.1 Group means for risk-taking propensity (BART; **a**), real world risk-taking (YRBSS; **b**), sensation seeking (BSSS; **c**), impulsivity (Go accuracy, NoGo accuracy, Go reaction time; **d**), anxiety (STAI state anxiety, STAI trait anxiety; **e**), depression (HADS-D; **f**), and anxiety controlling for depression (**g**). Error bars represent the bootstrapped 95% confidence intervals. All means are adjusted for IQ. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

3.3.1.1. Age and gender differences in risk-taking behaviours

3.3.1.1.1. BART risk-taking

Overall, BART risk-taking was equivalent for preadolescents and mid-adolescents, but increased from mid-adolescence to late adolescence. Unexpectedly, no gender effects were found for BART risk-taking.

For BART adjusted pumps, a main effect of *Age Group* was found ($F(2, 98) = 4.18$, $p = 0.018$, $\eta_p^2 = 0.08$), revealing that late adolescents pumped up each balloon more than preadolescents ($p = 0.011$) and mid-adolescents ($p = 0.025$). No difference was found between preadolescents and mid-adolescents in the number of adjusted pumps per balloon ($p = 0.789$). The main effect of *Gender* ($F(1, 98) = 1.14$, $p = 0.289$, $\eta_p^2 = 0.01$), and the *Age Group* by *Gender* interaction ($F(2, 98) = 0.11$, $p = 0.899$, $\eta_p^2 = 0.00$) were non-significant for BART adjusted pumps. Similarly, for BART total points, a main effect of *Age Group* was found ($F(2, 98) = 4.17$, $p = 0.018$, $\eta_p^2 = 0.08$), showing that late adolescents won more points on the BART compared to preadolescents ($p = 0.012$) and mid-adolescents ($p = 0.020$). No difference was found between preadolescents and mid-adolescents in the total number of points won on the BART ($p = 0.920$). The main effect of *Gender* ($F(1, 98) = 0.78$, $p = 0.378$, $\eta_p^2 = 0.01$), and the *Age Group* by *Gender* interaction ($F(2, 98) = 0.43$, $p = 0.652$, $\eta_p^2 = 0.01$) were non-significant for BART total points. By contrast, for BART popped balloons, the main effects of *Age Group* ($F(2, 98) = 2.50$, $p = 0.087$, $\eta_p^2 = 0.05$) and *Gender* ($F(1, 98) = 1.89$, $p = 0.172$, $\eta_p^2 = 0.02$), as well as the *Age Group* by *Gender* interaction ($F(2, 98) = 0.18$, $p = 0.834$, $\eta_p^2 = 0.00$) were non-significant.

3.3.1.1.2. YRBSS risk-taking

The YRBSS revealed significant age- and gender-related changes in this sample of adolescents. A significant main effect of *Age Group* was found for YRBSS risk-taking ($F(2, 98) = 23.38$, $p < 0.001$, $\eta_p^2 = 0.32$), revealing that preadolescents took significantly fewer real world risks compared to both mid-adolescents ($p = 0.001$) and late adolescents ($p = 0.001$). No significant difference was found between mid-adolescents and late adolescents in the number of real world risks taken ($p = 0.198$), suggesting that risk-taking increased during the transition from preadolescence to mid-adolescence, but not from mid-adolescence to late adolescence. While no main effect of *Gender* was found ($F(1, 98) = 1.19$, $p = 0.279$, $\eta_p^2 = 0.01$), there was a significant *Age Group* by *Gender* interaction ($F(2, 98) = 4.78$, $p = 0.010$, $\eta_p^2 = 0.09$). To further

investigate this interaction, two one-way ANOVAs were conducted with *Age Group* as the between-group variable. Separate ANOVAs were conducted for females and males. For females, a main effect of *Age Group* was found ($F(2, 50) = 20.16$, $p < 0.001$, $\eta_p^2 = 0.45$), showing that preadolescent females took significantly fewer risks in the previous 12 months compared to mid-adolescent females ($p = 0.028$) and late adolescent females ($p = 0.001$). Mid-adolescent females also took significantly fewer risks in the previous 12 months compared to late adolescent females ($p = 0.002$). Together, these findings indicate that, for females, engagement in real world risk-taking increased from preadolescence to mid-adolescence, as well from mid-adolescence to late adolescence. For males, the main effect of *Age Group* was significant ($F(2, 47) = 11.29$, $p < 0.001$, $\eta_p^2 = 0.33$), revealing that preadolescent males took significantly fewer risks in the previous 12 months compared to mid-adolescent males ($p = 0.002$) and late adolescent males ($p = 0.001$). However, no significant difference was found between mid-adolescent and late adolescent males ($p = 0.383$), suggesting that, for males, engagement in real world risk-taking increased from preadolescence to mid-adolescence, but not from mid-adolescence to late adolescence.

The YRBSS yields a composite score reflecting engagement in a wide range of risk-taking behaviours, including substance use, unsafe sexual activity, aggressive and illegal behaviours, and road safety behaviours. Thus, chi-squared tests were used to assess whether specific risky behaviours were driving the overall age effects in YRBSS risk-taking. Chi-squared tests were computed for each item on the YRBSS. Overall age differences were assessed separately for females and males, and significant results were followed up with additional chi-squared tests to test differences between preadolescents and mid-adolescents, preadolescents and late adolescents, and mid-adolescents and late adolescents. Figure 3.2 depicts the percentage of females and males in each age group engaging in the different risks measured by the YRBSS, and Table 3.1 reports the chi-squared coefficients.

The chi-squared analyses revealed that female and male late adolescents engaged in significantly more alcohol use, tobacco use, illicit drug use, unsafe sex, and not wearing a car seatbelt compared to preadolescents. Notably, the age at which engagement in these behaviours increased differed between females and males. For females, tobacco use, illicit drug use, and unsafe sex only increased between mid-adolescence and late adolescence, but not from preadolescence to mid-adolescence.

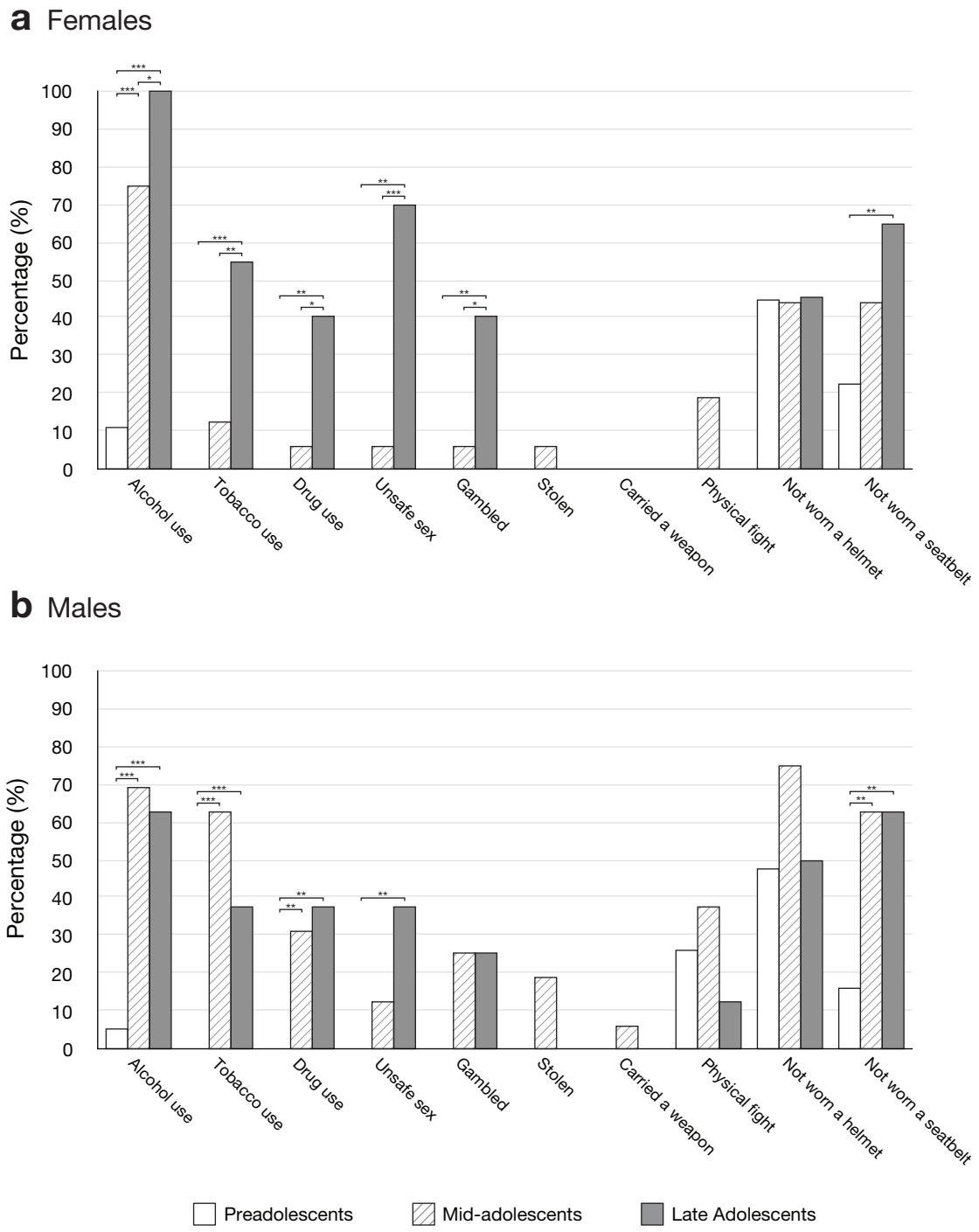

Figure 3.2 The percentage of female (**a**) and male (**b**) preadolescents, mid-adolescents, and late adolescents engaging in each item on the YRBSS during the previous 12 months. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

By contrast, alcohol use and not wearing a car seatbelt increased from preadolescence to mid-adolescence as well as from mid- adolescence to late adolescence for females. For males, alcohol use, tobacco use, illicit drug use, and not wearing a car seatbelt increased from preadolescence to mid-adolescence, but not from mid-adolescence to late adolescence. By comparison, unsafe sex increased from preadolescence to mid-adolescence as well as between mid-adolescence and late adolescence for males. Overall, these findings suggest that engagement in real world risk-taking predominantly increased at different ages for males and females; engagement in real world risk-taking largely increased during the transition from mid-adolescence to late adolescence for females, but during the transition from preadolescence to mid-adolescence for males. These findings are consistent with the ANOVA results showing that real world risk-taking increased between preadolescence and late adolescence for females but only between preadolescence and mid-adolescence for males.

Table 3.1
Chi-squared coefficients for the YRBSS

			Age Group Differences		
Item	Gender	Overall	Preadolescents vs. Mid-adolescents	Mid-adolescents vs. Late Adolescents	Preadolescents vs. Late Adolescents
Alcohol use	Females	33.51***	14.28***	5.63*	30.71***
	Males	17.84***	15.54***	0.14	13.20***
Tobacco use	Females	17.35***	2.39	6.96**	13.93***
	Males	16.17***	16.63***	2.00	8.60***
Drug use	Females	12.69**	1.16	5.40*	9.12**
	Males	8.51*	6.93**	0.14	8.60**
Unsafe sex	Females	28.39***	1.16	14.86***	19.95**
	Males	9.41**	2.52	2.67	5.36**
Gambled	Females	12.69**	1.16	5.40*	9.12**
	Males	5.63	-	-	-
Stolen	Females	2.42	-	-	-
	Males	6.97*	3.90	3.31	0.00
Carried a weapon	Females	- [a]	-	-	-
	Males	2.23	-	-	-
Physical fight	Females	7.54*	3.70	4.09	0.00
	Males	2.64	-	-	-
Not worn a helmet	Females	0.01	-	-	-
	Males	3.15	-	-	-
Not worn a seatbelt	Females	7.03*	1.79	1.63	7.01**
	Males	10.51**	8.12**	0.00	8.12**

Note. Follow up analyses were only computed if there was a significant main effect. [a] No females reported carrying a weapon and therefore the Chi-squared coefficient could not be computed. Chi-squared analyses were bootstrapped using 1000 samples. The B-H procedure was used to correct for multiple comparisons and had an FDR of 0.1. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Gambling and fighting also showed significant main effects for females. Consistent with tobacco use, illicit drug use, and unsafe sex, gambling increased significantly from mid-adolescence to late adolescence, but not from preadolescence to mid-adolescence. An overall main effect of fighting was also revealed for females, but no differences were found between age groups. Similarly, an overall main effect of stealing was found for males, but no differences were found between age groups. Finally, no main effects of carrying a weapon or wearing a helmet were found for either females or males.

3.3.1.2. Age and gender differences in sensation seeking

No age or gender effects were found for sensation seeking; the main effects of *Age Group* ($F(2, 98) = 1.77$, $p = 0.176$, $\eta_p^2 = 0.04$) and *Gender* ($F(1, 98) = 3.69$, $p = 0.058$, $\eta_p^2 = 0.04$), as well as the *Age Group* by *Gender* interaction ($F(2, 98) = 0.94$, $p = 0.394$, $\eta_p^2 = 0.02$), were non-significant.

3.3.1.3. Age and gender differences in impulsivity

Overall, preadolescents were significantly slower at responding to Go trials, and made significantly more errors on the Go/NoGo task compared to mid-adolescents and late adolescents.

For Go reaction time, a main effect of *Age Group* was found ($F(2, 98) = 31.10$, $p < 0.001$, $\eta_p^2 = 0.39$), showing that preadolescents were significantly slower at responding to Go trials compared to mid-adolescents ($p = 0.001$) and late adolescents ($p = 0.001$). No significant difference in reaction time was found between mid-adolescents and late adolescents ($p = 0.720$). The main effect of *Gender* ($F(1, 98) = 0.45$, $p = 0.504$, $\eta_p^2 = 0.01$) and the *Age Group* by *Gender* interaction ($F(2, 98) = 0.60$, $p = 0.550$, $\eta_p^2 = 0.01$) for reaction time were non-significant.

Go/NoGo accuracy was analysed using a mixed ANOVA in order to examine potential differences between the Go and NoGo conditions. *Condition* (Go, NoGo) was the within-group factor, and *Age Group* (preadolescents, mid-adolescents, late adolescents) and *Gender* (females, males) were the between-group factors. As with the other ANOVAs, IQ was included as a covariate. A main effect of *Age Group* was found ($F(2, 98) = 7.77$, $p = 0.001$, $\eta_p^2 = 0.14$), revealing that preadolescents made significantly more errors on the Go/NoGo task compared to mid-adolescents ($p = 0.037$) and late adolescents ($p < 0.001$). No significant difference in accuracy was found between mid-

adolescents and late adolescents (p = 0.126). No main effects of *Gender* (F(1, 98) = 0.02, p = 0.094, η_p^2 = 0.00) or *Condition* (F(1, 98) = 1.34, p = 0.268, η_p^2 = 0.01) were found. Finally, the *Age Group* by *Gender* (F(2, 98) = 1.27, p = 0.287, η_p^2 = 0.03), *Condition* by *Age Group* (F(2, 98) = 2.29, p = 0.107, η_p^2 = 0.05), *Condition* by *Gender* (F(1, 98) = 0.22, p = 0.637, η_p^2 = 0.00), and *Condition* by *Age Group* by *Gender* (F(2, 98) = 1.22, p = 0.299, η_p^2 = 0.02) interactions were non-significant. Taken together with reaction time, these findings show that performance on the Go/NoGo task did not differ between males and females, but significantly improved between preadolescence and mid-adolescence, and subsequently plateaued across mid-adolescence and late adolescence.

3.3.1.4. Age and gender differences in anxiety

3.3.1.4.1. State anxiety

For state anxiety, the main effects of *Age Group* (F(2, 98) = 2.10, p = 0.128, η_p^2 = 0.04) and *Gender* (F(1, 98) = 3.58, p = 0.061, η_p^2 = 0.04), as well as the *Age Group* by *Gender* interaction (F(2, 98) = 2.42, p = 0.094, η_p^2 = 0.05), were non-significant, suggesting that state anxiety levels were stable across adolescence. However, when controlling for participants' depression levels, the main effect of *Age Group* became significant (F(2, 97) = 3.87, p = 0.024, η_p^2 = 0.07). Pairwise comparisons revealed that preadolescents had greater state anxiety levels than mid-adolescents (p = 0.015) and late adolescents (p = 0.024). By comparison, mid-adolescents and late adolescents had comparable state anxiety levels (p = 0.708). A main effect of *Gender* was also found when controlling for depression (F(1, 97) = 6.31, p = 0.014, η_p^2 = 0.06), showing that females (M = 47.29, 95% CI [44.23, 50.72]) had greater state anxiety levels than males (M = 42.39, 95% CI [39.99, 44.82]). The *Age Group* by *Gender* interaction remained non-significant when controlling for depression (F(2, 97) = 2.52, p = 0.086, η_p^2 = 0.05).

3.3.1.4.2. Trait anxiety

Trait anxiety followed the same pattern as state anxiety. Before controlling for depression, the main effects of *Age Group* (F(2, 98) = 1.09, p = 0.340, η_p^2 = 0.02) and *Gender* (F(1, 98) = 0.54, p = 0.464, η_p^2 = 0.01), as well as the *Age Group* by *Gender* interaction (F(2, 98) = 0.84, p = 0.436, η_p^2 = 0.02), were non-significant. However, when controlling for depression, the main effect of *Age Group* became significant (F(2, 97) = 4.63, p = 0.012, η_p^2 = 0.09), showing that that preadolescents had greater trait anxiety levels than mid-adolescents (p = 0.007) and late adolescents (p = 0.016). In

comparison, mid-adolescents and late adolescents had equivalent trait anxiety levels (p = 0.603). A trend was found for the main effect of *Gender* (F(1, 97) = 3.53, p = 0.063, η_p^2 = 0.04), suggesting that females (M = 56.52, 95% CI [53.14, 59.99]) had greater trait anxiety levels than males (M = 52.43, 95% CI [48.94, 56.42]). The *Age Group* by *Gender* interaction remained non-significant when controlling for depression (F(2, 97) = 1.11, p = 0.333, η_p^2 = 0.02).

3.3.2. Relationships between risk-taking and anxiety during adolescence

The second aim of this study was to examine the relationship between risk-taking behaviours and anxiety levels during preadolescence, mid-adolescence, and late adolescence. To examine the relationships between risk-taking and anxiety, Pearson correlation coefficients were computed for female and male preadolescents, mid-adolescents, and late adolescents. Given that anxiety and depression are highly comorbid during adolescence (Brady & Kendall, 1992; Hirschfeld, 2001), correlations were subsequently recomputed with depression scores included as a covariate. Since IQ scores differed significantly between preadolescents, mid-adolescents, and late adolescents, IQ scores were included as a covariate in all correlations. Correlations were bootstrapped and corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5). The correlation coefficients are reported in Supplementary Table 3.4 (Appendix 1).

3.3.2.1. Relationships between BART risk-taking and anxiety

This section reports the relationships between BART risk-taking and anxiety. Overall, higher state anxiety levels were associated with lower levels of BART risk-taking in late adolescent females. In direct contrast, higher state anxiety levels were associated with greater levels of BART risk-taking in preadolescent females. Finally, higher trait anxiety levels were associated with lower levels of BART risk-taking in preadolescent males. All other relationships were non-significant.

3.3.2.1.1. BART risk-taking and state anxiety

As predicted, greater levels of state anxiety were associated with reduced risk-taking on the BART for late adolescent females. Specifically, state anxiety was moderately negatively correlated with the number of adjusted pumps (r = -0.50, p = 0.031), the number of points won (r = -0.54, p = 0.018), and the number of popped

balloons (r = -0.50, p = 0.031). Notably, these relationships became non-significant after controlling for depression (adjusted pumps: r = -0.48, p = 0.042; total points: r = -0.43, p = 0.072; popped balloons: r = -0.39, p = 0.107).

Contrary to predictions, no correlations were found between state anxiety and BART risk-taking for mid-adolescent females.

In direct contrast to late adolescent females, state anxiety was moderately positively correlated with the number of adjusted pumps (r = 0.61, p = 0.009), the number of points won (r = 0.53, p = 0.031), and the number of popped balloons (r = 0.50, p = 0.039) for preadolescent females. These findings suggest that greater levels of state anxiety were associated with increased risk-taking on the BART for preadolescent females. The relationships between state anxiety and the number of adjusted pumps (r = 0.62, p = 0.011) and number of popped balloons (r = 0.55, p = 0.029) held after controlling for depression. However, the relationship between state anxiety and the number of points won failed to reach significance after correction (r = 0.53, p = 0.036).

Compared to females, no relationships between state anxiety and BART risk-taking were found for preadolescent, mid-adolescent, or late adolescent males.

3.3.2.1.2. BART risk-taking and trait anxiety

Trait anxiety was moderately negatively correlated with the number of adjusted pumps for preadolescent males (r = -0.56, p = 0.016), suggesting that greater levels of trait anxiety were associated with reduced risk-taking on the BART for preadolescent males. However, this relationship failed to reach significance after controlling for depression (r = -0.44, p = 0.076).

No relationships between trait anxiety and BART risk-taking were found for male mid-adolescents or late adolescents. Similarly, no relationships between trait anxiety and BART risk-taking were found for preadolescent, mid-adolescent, or late adolescent females.

3.3.2.2. Relationships between YRBSS risk-taking and anxiety

This section reports the relationships between YRBSS risk-taking and anxiety. Overall, higher trait anxiety levels were associated with greater levels of YRBSS risk-taking in preadolescent females. All other relationships between YRBSS risk-taking and trait or state anxiety levels were non-significant.

3.3.2.2.1. YRBSS risk-taking and state anxiety

No relationships between state anxiety and YRBSS risk-taking were found for any group.

3.3.2.2.2. YRBSS risk-taking and trait anxiety

Trait anxiety was positively associated with real-world risk-taking for preadolescent females (r = 0.68, p = 0.003), suggesting that greater levels of trait anxiety were associated with increased real world risk-taking for preadolescent females. This relationship held after controlling for depression (r = 0.65, p = 0.006).

No relationships between trait anxiety and YRBSS risk-taking were found for mid-adolescent or late adolescent females. Similarly, no relationships between trait anxiety and YRBSS risk-taking were found for preadolescent, mid-adolescent, or late adolescent males.

3.3.3. The influence of puberty on risk-taking behaviours, sensation seeking, impulsivity, and anxiety

The previous sections provide insights into how risk-taking, sensation seeking, impulsivity, and anxiety, and the relationships between risk-taking and anxiety, change as a function of age and gender during the course of adolescence. However, studies have also reported that puberty has considerable affects on the development of risk-taking, sensation seeking, and anxiety (Collado *et al*., 2014; Martin *et al*., 2002; Reardon *et al*., 2009). Accordingly, the final aim of this study was to examine the linear and quadratic relationships between pubertal stage and measures of risk-taking, sensation seeking, impulsivity, and anxiety while controlling for chronological age. Since there are large individual differences in the age of pubertal onset (Sørensen *et al.,* 2013), categorising participants into age groups may confound any potential relationships between puberty and risk-taking, sensation seeking, impulsivity, and anxiety. Thus, the relationships between puberty and risk-taking, sensation seeking, impulsivity, and anxiety were assessed in females (n = 34, M_{age} = 12.62, SD_{age} = 2.40) and males (n = 35, M_{age} = 12.40, SD_{age} = 2.72) aged 9-17 years using age as a continuous variable.

Since many developmental trajectories are non-linear, hierarchical polynomial regression analyses were conducted to assess the linear and quadratic relationships between pubertal stage, risk-taking, sensation seeking, impulsivity, and anxiety. The

dependent variables were BART adjusted pumps, BART total points, BART popped balloons, YRBSS risk-taking, BSSS sensation seeking, Go accuracy, NoGo accuracy, Go reaction time, STAI state anxiety, and STAI trait anxiety. Age was entered into the first block of the regression analyses as a control variable, and PDS scores were entered into the second block. Separate regression analyses were conducted to assess the linear and quadratic relationships; age and PDS scores were used to examine the linear relationships, and squared age and PDS scores were used to assess the quadratic relationships. To control for the affect of depression on anxiety levels, STAI regression analyses were recomputed with HADS-D scores included as a covariate; HADS-D scores were entered into the first block, age was entered into the second block, and PDS scores were entered into the third block. Supplementary Table 3.5 (Appendix 1) reports the intercorrelations, and Supplementary Table 3.6 (Appendix 1) reports the linear and quadratic regression coefficients for risk-taking (BART, YRBSS), sensation seeking (BSSS), impulsivity (Go/NoGo), anxiety (STAI), and anxiety controlling for depression (STAI HADS-D).

An independent t-test revealed no significant differences in IQ scores between females (M = 110.56, 95% CI [106.81, 114.16]) and males (M = 111.29, 95% CI [107.72, 114.74]) aged 9-17 years ($t(67) = 0.28$, $p = 0.778$). Thus, IQ scores were not included as a control variable in the regression analyses. The regression coefficients were bootstrapped and corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5).

3.3.3.1. Risk-taking behaviours and puberty

3.3.3.1.1. BART risk-taking and puberty

No linear or quadratic relationships were found between puberty and BART risk-taking for females or males.

3.3.3.1.2. YRBSS risk-taking and puberty

In contrast to BART risk-taking, real world risk-taking was significantly predicted by pubertal stage. Real world risk-taking was predicted by the linear PDS term for both females (ΔR^2 = 10.2%, $F(1, 31) = 4.63$, $p = 0.039$) and males (ΔR^2 = 15.5%, $F(1, 32) = 14.13$, $p = 0.001$). These findings show that real world risk-taking increased with more advanced pubertal stage in both females and males.

Real world risk-taking was also predicted by the quadratic PDS term for both

females (ΔR^2 = 13.5%, $F(1, 31) = 6.48$, $p = 0.016$) and males (ΔR^2 = 15.0%, $F(1, 32) = 14.41$, $p = 0.001$). The partial regression plots (Figure 3.3) revealed gender-specific trajectories for males and females. For males, real world risk-taking increased from early to mid puberty and began to plateau towards the later stages of puberty. For females, real world risk-taking steadily increased with more advanced pubertal stage.

3.3.3.2. Sensation seeking and puberty

No linear or quadratic relationships were found between puberty and sensation seeking for females or males.

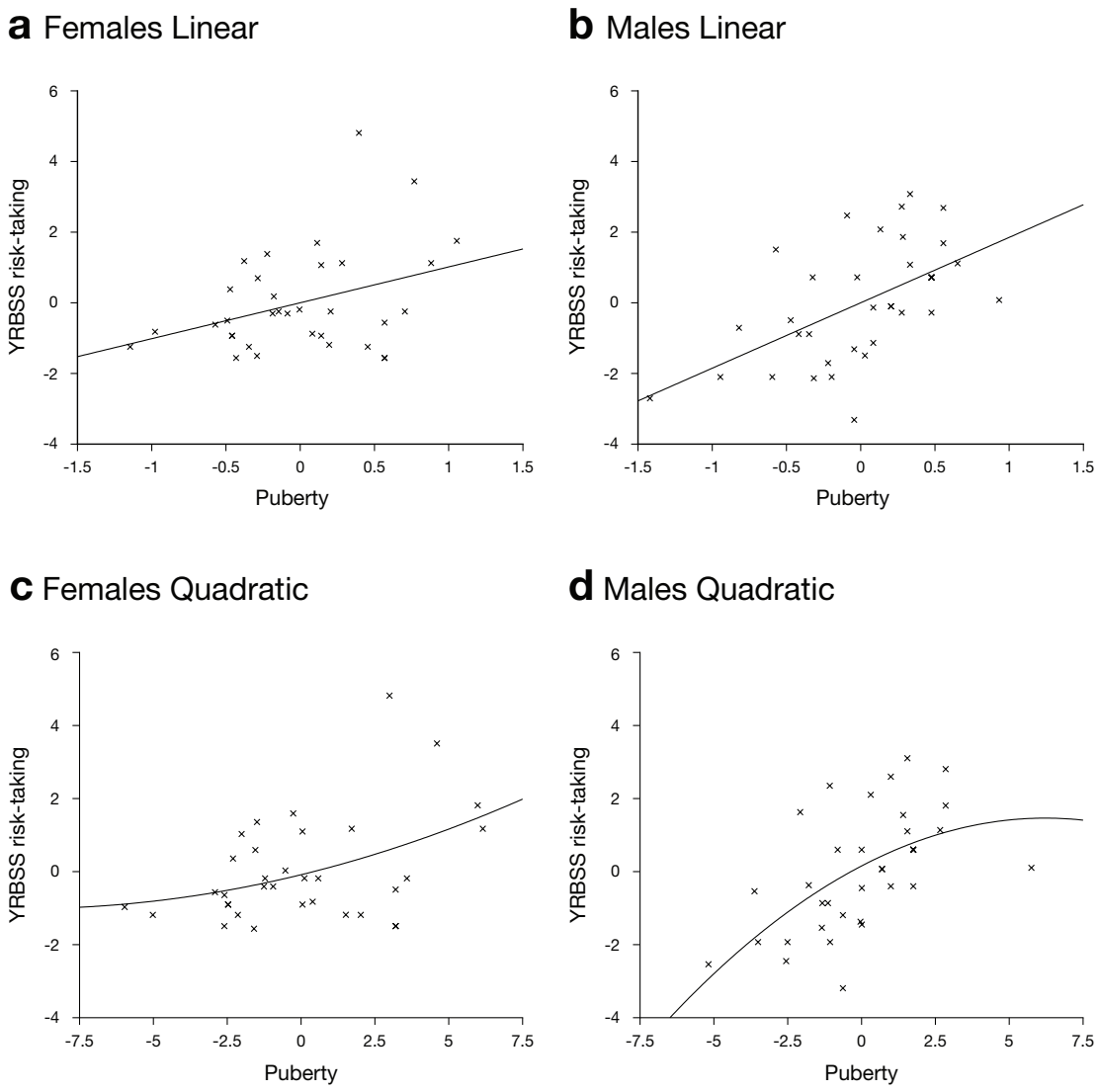

Figure 3.3 Partial regression plots of the residuals for the linear (**a**, **b**) and quadratic (**c**, **d**) relationships between puberty and YRBSS risk-taking controlling for chronological age for female (**a**, **c**) and male (**b**, **d**) adolescents aged 9-17 years.

3.3.3.3. Impulsivity and puberty

No linear or quadratic relationships were found between puberty and impulsivity for females or males.

3.3.3.4. Anxiety and puberty

3.3.3.4.1. State anxiety and puberty

No linear or quadratic relationships were found between puberty and state anxiety for females or males.

3.3.3.4.2. Trait anxiety and puberty

No linear or quadratic relationships were found between puberty and trait anxiety for females or males.

3.3.4. Relationships between risk-taking, sensation seeking, and impulsivity

Finally, the relationships between risk-taking behaviours (BART adjusted pumps, BART total points, BART popped balloons, YRBSS), sensation seeking (BSSS), and impulsivity (Go RT, Go accuracy, NoGo accuracy) were assessed to examine whether risk-taking, sensation seeking, and impulsivity were related or distinct constructs in this sample of adolescents (Supplementary Table 3.7, Appendix 1). Pearson correlation coefficients were conducted for male and female preadolescents, mid-adolescents, and late adolescents. Correlations were bootstrapped and corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5).

As predicted, YRBSS risk-taking was highly positively correlated with sensation seeking for mid-adolescent males (r = 0.73, p = 0.002) and late adolescent females (r = 0.78, p < 0.001), suggesting that greater sensation seeking tendencies were associated with increased real world risk-taking in mid-adolescent males and late adolescent females. The number of popped balloons on the BART was also negatively correlated with Go accuracy for preadolescent males (r = -0.52, p = 0.021), suggesting that greater accuracy to Go trials during the Go/NoGo task was associated with reduced BART risk-taking in preadolescence males. These findings are consistent with previous findings (e.g., Donohew *et al.*, 2000; Lejuez *et al.*, 2002; Steinberg, 2008) and the study predictions. Critically, most groups did not show relationships between the BART, YRBSS, and BSSS, suggesting that the BART, YRBSS, and BSSS may have largely

measured distinct constructs in this sample of adolescents.

3.4. Discussion

This study had three aims. The first aim of the study was to investigate the age- and gender-related differences in risk-taking behaviours, sensation seeking, impulsivity, and anxiety levels in a sample of typically developing preadolescents (9-12 years), mid-adolescents (13-17 years), and late adolescents (18-23 years). The results showed that BART risk-taking remained stable across preadolescence and mid-adolescence, but increased from mid-adolescence to late adolescence for both males and females. By comparison, gender-specific trajectories were observed for the YRBSS, with real world risk-taking increasing from preadolescence to mid-adolescence for males and females, but from mid-adolescence to late adolescence for females only. There was also evidence that males engaged in real world risky behaviours earlier in development than females. Unexpectedly, sensation seeking tendencies were equivalent across all groups. However, as predicted, impulsivity decreased from preadolescence to mid-adolescence, but remained stable across mid-adolescence and late adolescence. Finally, females reported greater anxiety levels than males as expected, but unexpectedly, preadolescents had greater anxiety levels than mid-adolescents and late adolescents.

The second aim of this study was to examine gender differences in the relationship between risk-taking behaviours and anxiety levels during preadolescence, mid-adolescence, and late adolescence. The findings showed that higher levels of anxiety were associated with reduced levels of risk-taking in late adolescent females, but not in late adolescent males. By comparison, higher levels of anxiety were associated with increased levels of risk-taking in preadolescent females, and reduced levels of risk-taking in preadolescent males. Critically, anxiety was not associated with risk-taking in either mid-adolescent females or males.

Finally, this study aimed to examine the influence of puberty on the development of risk-taking, sensation seeking, impulsivity, and anxiety in male and female adolescents aged 9-17 years while controlling for chronological age. The results revealed linear and quadratic relationships between pubertal stage and real world risk-taking behaviours for both males and females, suggesting that more advanced pubertal stage was associated with greater levels of real world risk-taking. By contrast, pubertal stage was not associated with BART risk-taking, sensation seeking, impulsivity, or

anxiety in either males or females. This discussion is organised in accordance with the study aims.

3.4.1. *Age and gender differences in risk-taking, sensation seeking, impulsivity, and anxiety*

The first aim of this study was to examine the age- and gender-related differences in risk-taking behaviours, sensation seeking, impulsivity, and anxiety in a normative sample of adolescents aged 9-23 years. Notably, only a few studies have measured the development of risk-taking, sensation seeking, impulsivity, and anxiety in a cohort of adolescents with an age range wide enough to examine how these constructs change across the course of adolescence (Steinberg *et al*., 2008). Moreover, these studies have largely overlooked potential gender differences. Hence, the findings from this study provide novel insights into how these constructs develop throughout adolescence for males and females.

3.4.1.1. Risk-taking behaviours

To account for the potential confounds associated with self-report questionnaires (Ladouceur *et al*., 2000; Lejuez *et al*., 2002; Lejuez *et al*., 2003), participants completed a behavioural task that measured risk-taking propensity (BART) in addition to a self-report questionnaire that measured recent engagement in a range of risky behaviours (YRBSS). Based on recent findings (e.g., Burnett *et al*., 2010; Steinberg *et al*., 2008), it was predicted that risk-taking behaviours, as measured by the BART and YRBSS, would increase from preadolescence to mid-adolescence, peak in mid-adolescence, and begin to decrease from mid-adolescence to late adolescence.

Contrary to previous studies (Burnett *et al*., 2010; Eshel *et al*., 2007; Figner *et al*., 2009) and the study predictions, BART risk-taking remained stable across preadolescence and mid-adolescence, but increased from mid-adolescence to late adolescence. The BART was originally developed to simulate real world risk-taking, whereby risk-taking is advantageous up to a certain point (i.e., the balloon breakpoint) but risk-taking past this point is disadvantageous (Lejuez *et al*., 2002). The BART measures risk-taking propensity using three dependent variables: the average number of adjusted pumps for unpopped balloons; the total points won; and the total number of popped balloons. In the current study, greater BART risk-taking in late adolescents

was indexed by a greater number of adjusted pumps for unpopped balloons and total points won. Crucially, no age differences were found for the number of popped balloons. Therefore, the finding that late adolescents pumped up each balloon more and won more points but did not pop more balloons than preadolescents and mid-adolescents suggests that late adolescents were using a more optimal strategy to maximise gain, rather than simply taking more indiscriminate risks.

This is the first study to examine the development of risk-taking propensity from preadolescence to late adolescence using the BART. Notably, studies that have reported a peak in risk-taking propensity during adolescence using behavioural tasks have largely used probabilistic gambling tasks (e.g., Burnett *et al*., 2010; Eshel *et al*., 2007; Figner *et al*., 2009). Thus, it is possible that only certain behavioural tasks are able to elicit the peak in risk-taking propensity during mid-adolescence. Future work is therefore needed to determine how the development of risk-taking propensity relates to specific task demands.

Gender differences in BART risk-taking were also expected; it was predicted that males would take more risks on the BART compared to females, irrespective of age. In contrast to this prediction, no gender differences were observed in BART risk-taking in the current study. While gender differences in BART risk-taking have been found in young adults (Lighthall, Mather & Gorlick, 2009), most adolescent and adult studies have either not examined gender differences or have reported small to no effects (Aklin *et al*., 2005; Cazzell, Li, Lin, Patel & Liu, 2012; Lejuez *et al*., 2002; Lejuez *et al*., 2003; Lejuez *et al*., 2007). Notably, Lighthall *et al*. (2009) only found gender differences on the BART when participants experienced acute stress; stress increased risk-taking in males but reduced risk-taking in females. Thus, the lack of gender differences in the current study could reflect that participants completed the BART in isolation under conditions of low arousal.

In contrast to BART risk-taking, but in accordance with the study predictions and previous work (Donovan & Jessor, 1985; Eaton *et al*., 2010; Gullone *et al*., 2000; Kann *et al*., 1999; Windle *et al*., 2008), real world risk-taking, as measured by the YRBSS, increased from preadolescence to mid-adolescence for both females and males. Real world risk-taking also increased from mid-adolescence to late adolescence for females, suggesting that engagement in real world risk-taking followed gender-specific trajectories.

Interestingly, specific risks were found to be driving the overall changes in

YRBSS risk-taking over the adolescent period. Alcohol use, tobacco use, illicit drug use, unsafe sex, and not wearing a car seatbelt contributed to the age-related changes in real world risk-taking in both females and males. Gambling also contributed to the age-related changes in females. By contrast, stealing, carrying a weapon, physical fighting, and not wearing a helmet while riding a bicycle or motorcycle did not significantly change during the course of adolescence. These findings suggest that the specific risks underlying the increase in real world risk-taking during adolescence were largely comparable for male and female adolescents. Crucially however, the age at which engagement in these risks primarily increased differed for females and males; engagement in real world risk-taking increased from mid-adolescence to late adolescence for females but from preadolescence to mid-adolescence for males. Thus, these findings suggest that, in this sample of adolescents, males engaged in risk-taking behaviours earlier in development than females.

Based on previous work (Burnett *et al*., 2010; Steinberg *et al*., 2008), it was predicted that real world risk-taking behaviours would decrease from mid-adolescence to late adolescence for both females and males. However, in contrast to the study predictions, real world risk-taking continued to increase from mid-adolescence to late adolescence for females. In comparison to females, there was some evidence that real world risk-taking was beginning to decrease in males; no age-related changes were found between mid-adolescence and late adolescence for males. It is possible that the late adolescents in this sample were not old enough for a reduction in risk-taking behaviours to be detected; most studies that have found a reduction in risk-taking behaviours from adolescence to late adolescence/young adulthood have included participants aged 25 years and above (e.g., Burnett *et al*., 2010; Eshel *et al*., 2007; Figner *et al*., 2009). Hence, this sample of adolescents may have been too young to detect a decrease in risk-taking behaviours. Future work examining the developmental trajectory of risk-taking should therefore endeavour to include an additional age group aged 25 years and above.

Together, these findings reveal that BART risk-taking and YRBSS risk-taking followed different developmental trajectories in this sample of adolescents. Current theories of adolescent risk-taking assert that adolescent decision-making cannot be fully understood without acknowledging the context in which a decision involving risk is being made (Casey *et al*., 2010; Miller & Brynes, 1997; Steinberg, 2008). Specifically, adolescents are thought to be capable of making similar judgments to adults in low

arousal contexts (when they experience 'cold cognition'), but make suboptimal, risky decisions in high arousal contexts (when they experience 'hot cognition') (Figner *et al*., 2009). Studies have consistently reported that the social context significantly influences adolescent risk-taking; adolescents take significantly more risks when they are in the presence of peers compared to when they are in isolation (Gardner & Steinberg, 2005), and most adolescent risk-taking and criminal activity occurs in groups (Zimring, 1981). Thus, the presence of peers is thought to shift the context from low arousal to high arousal (Steinberg, 2008). In light of these findings, it is possible to speculate that distinct developmental trajectories were observed for the BART and YRBSS in the current study because the BART and YRBSS measured risk-taking behaviours originating from different social contexts (Aklin *et al*., 2005); the BART was completed in isolation under conditions of low arousal whereas risk-taking behaviours measured by the YRBSS are likely to have occurred in the presence of peers and therefore under conditions of high arousal. Finally, no relationships were observed between the BART and YRBSS for any group, supporting the idea that the BART and YRBSS were measuring distinct constructs of risk-taking in the current study.

3.4.1.2. Sensation seeking and impulsivity

The developmental trajectory of risk-taking behaviours, as measured by both behavioural tasks and self-report questionnaires, is thought to be largely dependent on age-related changes in sensation seeking and impulsivity during childhood and adolescence (Steinberg *et al*., 2008). Participants in the current study were therefore asked to complete measures of sensation seeking (BSSS) and impulsivity (Go/NoGo task).

On the basis of previous studies (Harden & Tucker-Drob, 2011; Shulman *et al*., 2014; Steinberg *et al*., 2008), it was predicted that sensation seeking would increase from preadolescence to mid-adolescence, peak in mid-adolescence, and decrease from mid-adolescence to late adolescence. Unexpectedly however, sensation seeking remained stable across preadolescence, mid-adolescence, and late adolescence. Notably, the BSSS had highly variable Cronbach's alphas (0.38–0.81) in this sample (Supplementary Table 3.2, Appendix 1). The Cronbach's alphas were very low in preadolescents, which is particularly problematic because studies have consistently reported that sensation seeking tendencies begin to increase during preadolescence for both males and females (Shulman *et al*., 2015). The BSSS was also not correlated

with the BART or YRBSS for most groups. Together, these findings point towards the BSSS being a poor measure of sensation seeking in this sample of adolescents. This is surprising since the BSSS has been used to measure sensation seeking in individuals aged 9-75 years (e.g., Eachus, 2004; MacPherson *et al*., 2010; Palmgreen *et al*., 2001). Despite this, the findings from the current study suggest that the BSSS is not an optimal tool for measuring sensation seeking tendencies in children and early adolescents.

Impulsivity has also been implicated in adolescent risk-taking; immature impulse control and self-regulatory abilities are thought to be unable to modulate the increases in sensation seeking during adolescence (Steinberg *et al*., 2008). In contrast to risk-taking behaviours and sensation seeking, impulsivity has been shown to decrease across adolescence, as impulse control improves (Harden & Tucker-Drob, 2011; Shulman *et al*., 2014; Shulman *et al*., 2015; Steinberg *et al*., 2008). Consistent with previous work and the study predictions, impulsivity decreased from preadolescence to mid-adolescence, but remained stable across mid-adolescence to late adolescence. Specifically, preadolescents took longer to respond to Go trials and made significantly more errors on the Go/NoGo task than both mid-adolescents and late adolescents. Notably, some studies have shown that impulsivity continues to reduce from mid-adolescence into late adolescence (Shulman *et al*., 2015). However, a reduction in impulsivity from mid-adolescence to late adolescence is thought to depend on the complexity and difficulty of the task (Davidson, Amso, Anderson & Diamond, 2006; Steinberg *et al*., 2008). Taken together with risk-taking, the findings from the current study support previous work showing that impulsivity and risk-taking behaviours have distinct developmental trajectories across adolescence, whereby impulsivity decreases and risk-taking increases during the course of adolescence (Steinberg *et al*., 2008).

3.4.1.3. Anxiety levels

As well as being a vulnerable period for engaging in risk-taking behaviours, there is also evidence that typically developing adolescents are at an increased risk for experiencing heightened levels of anxiety compared to children and adults (Abe & Suzuki, 1986). Studies have also consistently reported that females are at greater risk than males for experiencing high anxiety levels at all developmental stages (Abe & Suzuki, 1986; Lewinsohn *et al*., 1998; Van Oort *et al*., 2009). Consistent with previous

work and the study predictions, females in the current study had greater state and trait anxiety levels compared to males, irrespective of age. However, contrary to previous work and the study predictions, mid-adolescents did not have greater state or trait anxiety levels than either preadolescents or late adolescents; rather, preadolescents had greater state and trait anxiety levels than both mid-adolescents and late adolescents.

The finding that preadolescents had greater state and trait anxiety levels than mid-adolescents and late adolescents was unexpected. Since there is not a questionnaire that can accurately measure anxiety symptoms in individuals aged 9-23 years, the current study administered the child version of the STAI to preadolescents and the adult version of the STAI to mid-adolescents and late adolescents, in line with the manual recommendations (Spielberger *et al*., 1983). The STAI child and adult versions have maximum scores of 60 and 80, respectively, and thus the raw scores were converted to percentages in order to equate the two measures. The findings that preadolescents had greater anxiety levels than both mid-adolescents and late adolescents may therefore reflect that the child version of the STAI measured a different construct of anxiety to the adult version. However, there is some empirical evidence that preadolescents have greater anxiety levels than mid-adolescents. For instance, Van Oort *et al*. (2009) found that anxiety levels decreased from preadolescence to early adolescence (10-13 years). Thus, it is possible that the preadolescents in this sample did have greater anxiety levels than mid-adolescents and late adolescents. Regardless of whether the child and adult versions of the STAI measured different constructs of anxiety or preadolescents had greater anxiety levels, mid-adolescents did not have greater anxiety levels than late adolescents. Therefore, the findings from this study show that anxiety levels, as measured by the STAI, did not peak in mid-adolescence. Taken together, the findings from the current study demonstrate that experiencing heightened anxiety levels during middle adolescence is not inevitable and universal (Arnett, 1999; Dahl, 2004), as has sometimes been suggested (Hall, 1904).

Notably, the age and gender differences in state and trait anxiety were only observed when depression levels were controlled for. This is consistent with studies showing that the STAI measures negative affect in addition to generalised anxiety (Andrade, Gorenstein, Vieira Filho, Tung & Artes, 2001; Bados, Gómez-Benito & Balaguer, 2010), and that anxiety and depression are highly comorbid during

adolescence (Brady & Kendall, 1992; Hirschfeld, 2001). These findings therefore emphasise the importance of controlling for depression when examining the development of anxiety during adolescence.

3.4.2. Relationships between risk-taking and anxiety

The findings discussed in the previous section outline the developmental trajectories of risk-taking behaviours and anxiety levels in this sample of adolescents. However, based on the existing literature, it remains unclear how anxiety levels relate to risk-taking behaviours during the different stages of adolescence for males and females. Accordingly, the second aim of this study was to examine gender differences in the relationship between risk-taking behaviours and anxiety levels during preadolescence, mid-adolescence, and late adolescence.

Contrary to the study predictions, no relationships between risk-taking and anxiety were found for either female or male mid-adolescents. These findings initially appear inconsistent with previous studies reporting that greater anxiety levels are associated with greater levels of risk-taking on the BART (Reynolds *et al*., 2013; Richards *et al*., 2015). However, the studies assessing relationships between anxiety and BART risk-taking only found a relationship between risk-taking and anxiety when adolescents were exposed to acute social stress (Reynolds *et al*., 2013; Richards *et al*., 2015). It is therefore possible that BART risk-taking was not associated with anxiety in mid-adolescents because participants completed the BART in isolation and were not exposed to acute stress.

The lack of a relationship between risk-taking and anxiety in mid-adolescents is also inconsistent with previous studies reporting that high levels of anxiety are associated with greater engagement in real world risk-taking behaviours during mid-adolescence (Comeau *et al*., 2001; Patton *et al*., 1996). These previous studies have proposed the self-medication hypothesis as the mechanism underlying the positive relationship between risk-taking and anxiety, whereby individuals engage in high levels of risky behaviours to alleviate symptoms of negative affect (Comeau *et al*., 2001; Patton *et al*., 1996). Thus, it is possible that no relationships were found between real world risk-taking and anxiety in mid-adolescents in the current study because mid-adolescents' anxiety levels were not particularly high in this sample; mid-adolescents did not have higher anxiety levels than preadolescents or late adolescents. Hence, it is possible that elevated anxiety levels in mid-adolescents are needed to

elicit a positive relationship between risk-taking and anxiety. This idea is consistent with Reynolds *et al*. (2013) and Richards *et al*.'s (2015) findings, where a positive relationship between risk-taking and anxiety was only found when mid-adolescents were exposed to acute social stress. Despite this, it is unclear from the literature whether high levels of anxiety in mid-adolescence are needed to elicit a relationship between risk-taking and anxiety; the literature to date has not compared how the relationship between risk-taking and anxiety changes throughout adolescence, and how this relationship relates to participants' levels of anxiety.

There is substantially more research examining the relationship between risk-taking and anxiety in undergraduate samples. These studies have reliably reported that higher levels of anxiety in late adolescents/young adults are associated with reduced levels of risk-taking (Broman-Fulks *et al*., 2014; Giorgetta *et al*., 2012; Lorian & Grisham, 2010; Maner *et al*., 2007; Maner & Schmidt, 2006). Consistent with these findings and the study predictions, higher state anxiety levels were associated with reduced BART risk-taking in late adolescent females. Given that anxiety enhances sensitivity to potential threats (MacLeod & Mathews, 1988; Martin, Williams & Clark, 1991), it is possible that higher levels of anxiety in late adolescent females were associated with greater sensitivity to potential losses on the BART, and consequently a reduced desire to engage in risky behaviours. Interestingly, the late adolescent females did not have greater anxiety levels than the other groups in this sample. Moreover, late adolescents' state and trait anxiety levels were comparable to the STAI standard scores (Spielberger *et al*., 1983). Therefore, the negative relationship between risk-taking and anxiety in late adolescent females suggests that the relationship between risk-taking and anxiety changes during the transition from mid-adolescence to late adolescence, rather than particularly high anxiety levels being associated with a reduction in risk-taking.

Notably, no relationship was found between risk-taking and anxiety for late adolescent males. Since males develop more slowly than females (Lenroot & Giedd, 2010), it is possible that the late adolescent males included in this study were too young to detect a negative relationship between risk-taking and anxiety. Alternatively, it is possible that the relationship between risk-taking and anxiety in undergraduate samples is predominantly driven by females. Previous work has largely overlooked gender differences in the relationship between risk-taking and anxiety in late adolescents/young adults, and therefore whether this is the case remains unclear.

Consequently, future work is needed to compare the relationship between risk-taking and anxiety between late adolescents (18-24 years) and young adults (25-30 years), and to examine the potential gender differences in such relationships, in order to determine why no relationship between risk-taking and anxiety was found in this sample of late adolescent males.

In comparison to the mid- and late adolescents, no directional hypotheses were made for preadolescents since the relationship between risk-taking and anxiety had not been previously studied in 9-12 year olds. While higher levels of state anxiety were associated with reduced BART risk-taking in late adolescent females, higher levels of state anxiety were associated with increased BART risk-taking in preadolescent females. Trait anxiety was also positively associated with real world risk-taking in preadolescent females. Thus, these findings reveal that the relationship between risk-taking and anxiety followed a distinct developmental trajectory for females, whereby risk-taking and anxiety were positively associated in preadolescence, not associated in mid-adolescence, and negatively associated in late adolescence.

In contrast to preadolescent females, greater trait anxiety levels in preadolescent males were associated with reduced BART risk-taking. Together, these findings suggest that the relationship between risk-taking and anxiety is gender-specific in 9-12 year olds. However, given that this is the first study to explore the relationship between risk-taking and anxiety in preadolescents, future work is needed to replicate these findings. Further work is also needed to understand the potential mechanisms that may underlie the gender-specific relationships between risk-taking and anxiety in preadolescents. Understanding the relationship between risk-taking and anxiety in preadolescents is particularly important because interventions designed to prevent high levels of risk-taking and anxiety in middle adolescence need to be implemented during preadolescence.

Collectively, these findings show that the relationship between risk-taking and anxiety changes considerably during the course of adolescence, and follows gender-specific developmental trajectories. Importantly, depression significantly influenced many of the relationships between risk-taking and anxiety in both preadolescents and late adolescents. As discussed earlier, the STAI is thought to measure negative affect in addition to generalised anxiety (Andrade *et al*., 2001; Bados *et al*., 2010), which may explain why many of the relationships between risk-taking and anxiety changed when controlling for depression.

3.4.3. The influence of puberty on adolescent risk-taking, sensation seeking, impulsivity, and anxiety

The previous sections provide insights into how risk-taking, sensation seeking, impulsivity, and anxiety, and the relationships between risk-taking and anxiety, change as a function of age and gender during the course of adolescence. Notably, studies have also reported that puberty has considerable affects on the development of risk-taking, sensation seeking, and anxiety (Collado *et al*., 2014; Martin *et al*., 2002; Reardon *et al*., 2009). Given that the age of pubertal onset varies considerably between individuals (Sørensen *et al.,* 2013), it is possible that categorising participants into age groups may distort the potential relationships between puberty and risk-taking, sensation seeking, impulsivity, and anxiety. Thus, the final aim of this study was to examine the linear and quadratic relationships between pubertal stage and measures of risk-taking, sensation seeking, impulsivity, and anxiety in male and female adolescents aged 9-17 years while controlling for chronological age.

The linear and quadratic relationships between pubertal stage and real world risk-taking were significant for males and females. The linear relationships revealed that greater levels of real world risk-taking were associated with more advanced pubertal stage for both males and females. These findings are highly consistent with previous studies (Collado *et al*., 2014; Faden *et al*., 2009). The quadratic relationships were also significant, and revealed gender-specific trajectories for males and females. For males, risk-taking increased from early to mid puberty, and subsequently plateaued across mid to late puberty. In contrast, risk-taking steadily increased with more advanced pubertal stage in females. These findings are consistent with the age-related changes in YRBSS risk-taking, where risk-taking behaviours tailed off during mid-adolescence for males but continued to increase between mid- and late adolescence for females. Notably, pubertal development explained between 10.2 and 15.5 per cent of the variance in real world risk-taking after controlling for chronological age, suggesting that puberty had a considerable influence on real world risk-taking during adolescence.

In contrast to YRBSS risk-taking, BART risk-taking was not significantly predicted by pubertal stage. The current study does not therefore support recent findings showing a relationship between puberty and BART risk-taking in 11-15 year olds (Collado *et al*., 2014). This discrepancy in findings may reflect slight differences in task demands; participants in Collado *et al*.'s (2014) study received a monetary prize

that corresponded to their total number of points whereas participants in the current study did not receive a monetary prize. As discussed earlier (Chapter 3.4.1.1), the BART and YRBSS were not correlated for any group in this cohort, and therefore the inconsistency in findings between the BART and YRBSS may reflect that the BART and YRBSS were measuring different constructs of risk-taking.

In contrast to previous studies that have reported a strong positive relationship between puberty and sensation seeking (Forbes & Dahl, 2010; Martin *et al*., 2002), puberty was not associated with sensation seeking in the current study. However, as discussed above (Chapter 3.4.1.2), it is possible that the lack of relationships between sensation seeking and puberty, as well as between sensation seeking and chronological age, result from the BSSS being a poor measure of sensation seeking in this sample of adolescents. Puberty was also not associated with impulsivity in the current study. This finding supports previous work suggesting that puberty and impulsivity are not associated (Steinberg *et al*., 2008)

Consistent with the study predictions and recent findings (Reardon *et al*., 2009), anxiety was not associated with puberty for males. However, contrary to previous work (Reardon *et al*., 2009) and the study predictions, anxiety was not associated with pubertal stage for females. Thus, the findings from the current study suggest that anxiety levels did not increase as a function of age or puberty during the course of adolescence. It is possible that puberty was not related to anxiety in this sample of adolescents because anxiety levels were not particularly high; all groups had anxiety levels that were comparable to the STAI standard scores (Spielberger *et al*., 1983). However, future work is needed to determine whether high anxiety levels are needed to elicit a relationship between puberty and anxiety. Notably, there is also considerable evidence that girls who begin puberty earlier than their peers are at an increased risk of experiencing heightened anxiety levels (Ge *et al*., 1996; Reardon *et al*., 2009). Therefore, it is possible that earlier pubertal onset is more robustly associated with anxiety than current pubertal stage.

It is beyond the scope of this study to determine the mechanisms underlying the relationships between puberty and real world risk-taking in adolescent females and males. However, it would be too simplistic to assume that the hormonal changes associated with puberty are directly responsible for the relationships between puberty and risk-taking observed in this study. The neurobiological models of adolescence propose that greater levels of activity in the socioemotional system underlie the

increases in reward-orientated and risk-taking behaviours during adolescence (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008). Critically, Steinberg's DSM (Steinberg, 2008) and the SPIN (Nelson *et al*., 2005) assert that these increases in activity in the socioemotional system result from gonadal hormones released during puberty. Thus, the interactions between pubertal hormones, brain activity, and risk-taking behaviours may be one mechanism underlying the relationship between pubertal stage and real world risk-taking observed in the current study. However, it has also been suggested that there are social and psychological factors that modulate the relationships between puberty and behaviour (Brooks-Gunn & Graber, 1994). Further work is needed to identify the puberty-dependent and puberty-independent affects on adolescent development, as well as the mechanisms that underlie such affects.

3.4.4. Study limitations

The findings from the current study need to be considered in light of the study limitations. First, the PDS was selected to measure pubertal status since it is widely used, highly reliable, and non-invasive (Shirtcliff *et al*., 2009). However, the PDS requires participants to self-report their own pubertal development, which has the potential to lead to inaccurate assessments, particularly in younger adolescents.

Second, the tools that are currently available to measure risk-taking, sensation seeking, and anxiety in developmental samples are not optimal. Measures in developmental studies need to be able to accurately measure constructs across wide age ranges that span multiple developmental periods. In the current study, the YRBSS was used to assess real world risk-taking behaviours. However, the YRBSS was a binary measure that assessed whether or not adolescents had engaged in a limited range of risky behaviours during the previous 12 months. Hence, the YRBSS is unable to provide a comprehensive assessment of adolescents' engagement in risk-taking behaviours. For instance, the YRBSS considers an adolescent who has had one alcoholic drink during the previous 12 months as equal to an adolescent who drinks alcohol on a regular basis. Moreover, the YRBSS asks participants to report their engagement in 10 specific behaviours. Consequently, the YRBSS is unable to assess adolescents' engagement in risky behaviours that are not included in the YRBSS. Due to the limitations of self-report questionnaires, the BART was used to measure risk-taking propensity. However, as discussed earlier (Chapter 3.4.1.1), the BART may not

be good measure of risk-taking behaviours in developmental studies; it is possible that age-related differences emerge due to participants' ability to develop an optimal strategy. Moreover, the BSSS had very poor Cronbach's alphas in preadolescents and may therefore not be an appropriate measure of sensation seeking tendencies in children and young adolescents. Finally, the child and adult versions of the STAI were used in this study because there is no available measure that can accurately assess anxiety symptoms in a sample of individuals aged 9-23 years. However, using the child and adult versions may have led to confounds, whereby participants who used the child version (preadolescents) self-reported higher levels of anxiety than participants who used the adult version (mid-adolescents, late adolescents). Together, these shortcomings highlight the need for tools that can accurately measure levels of risk-taking, sensation seeking, and anxiety throughout late childhood, early adolescence, middle adolescence, late adolescence, and young adulthood.

3.4.5. Conclusion

Despite the caveats, the current study provides novel insights into how risk-taking behaviours, sensation seeking, impulsivity, and anxiety levels change as a function of age, gender, and puberty during adolescence. Moreover, the current study represents an important step towards understanding the gender-specific developmental trajectories for the relationship between risk-taking and anxiety during adolescence. Notably, the neurobiological models of adolescence posit that age-related changes in reward and threat sensitivity underlie the changes in risk-taking, sensation seeking, and anxiety during adolescence (Casey *et al.*, 2010; Ernst *et al.*, 2006; Nelson *et al.*, 2005; Steinberg, 2008). Accordingly, the next study presented in this thesis (Chapter 4) examined adolescents' anticipatory ERP responses to rewarding and threating outcomes in the same cohort of adolescents in order to investigate whether anticipatory neural responses to reward and threat correspond to the age- and gender-related changes observed in the current study.

Chapter 4

Anticipatory neural responses to reward and threat across adolescence

Abstract

This study examined whether age-related changes in reward- and threat-related anticipatory processes can partially account for the developmental differences in risk-taking behaviours and anxiety levels found in this sample of adolescents (Chapter 3). This was investigated by examining reinforcement-dependent potentiation to discriminative stimuli (S^D) that predicted rewarding or threatening outcomes. To that end, early (N170) and late (LPP) ERPs that are modulated by motivationally salient stimuli were examined in 30 preadolescents (9-12 years; 15 females), 30 mid-adolescents (13-17 years; 15 females), and 34 late adolescents (18-23 years; 18 females) while they completed an instrumental task in which they emitted or omitted a motor response to obtain rewards and avoid losses. The LPP, but not the N170, showed age, but not gender, differences in reinforcement-dependent potentiation; preadolescents, mid-adolescents, and late adolescents showed potentiation to S^D that predicted a threat, but only preadolescents and mid-adolescents showed potentiation to S^D that predicted a reward. Notably, LPP potentiation was not modulated by participants' pubertal stage, risk-taking behaviours, or anxiety levels. Overall, these findings provide evidence that adolescents show anticipatory biases not just to reward-related cues but also to avoidance-related cues. These findings therefore challenge the neurobiological models asserting that adolescent behaviour is solely modulated by a reward bias.

4.1. Introduction

This study had four aims. Firstly, this study aimed to examine age- and gender-related changes in the reinforcement-dependent potentiation of the N170 and LPP ERPs to discriminative stimuli (S^D) that predicted rewarding or threatening outcomes in adolescents aged 9-23 years. Secondly, this study aimed to examine the functional significance of N170 and LPP reinforcement-dependent potentiation by assessing the relationships between ERP potentiation to S^D that predicted rewarding and threatening outcomes and participants' reaction times for corresponding conditions. Thirdly, this study aimed to assess whether N170 and LPP potentiation to rewarding and threatening outcomes was associated with participants' risk-taking behaviours and anxiety levels, respectively. Finally, this study aimed to examine the relationships between pubertal stage and ERP potentiation to S^D that predicted rewarding and threatening outcomes in male and female adolescents aged 9-17 years while controlling for chronological age.

The introduction to this study begins by outlining the premise of the current study (Chapter 4.1.1). This introduction then discusses what is currently known about the development and functional significance of the N170 (Chapter 4.1.2) and LPP (Chapter 4.1.3), and the benefits of using instrumental tasks to explore anticipatory ERP responses to rewarding and threatening outcomes (Chapter 4.1.4). Next, this introduction discusses the potential role of puberty in the development of the N170 and LPP (Chapter 4.1.5), and finally, this introduction outlines the aims and hypotheses of the current study (Chapter 4.1.6).

4.1.1. Current models of the approach-avoidance systems during adolescence

The Triadic Model of adolescence asserts that adolescent risk-taking behaviour results from a hyperresponsive approach system combined with a hyporesponsive avoidance system (Ernst *et al*., 2006; Ernst *et al*., 2011). In contrast to this idea, we recently found that adolescents (12-15 years) had greater reinforcement-dependent potentiation of the N170 to S^D that predicted a threatening outcome than young adults (18-32 years) (Levita *et al*., 2014). This finding raised three interesting questions that warrant further investigation. First, how does enhanced potentiation to S^D that predict threatening outcomes fit with the neurobiological models suggesting that adolescents have a hyporesponsive avoidance system (Ernst *et al*., 2006; Ernst *et al*., 2011)? Second, do adolescents also show enhanced potentiation to S^D that predict rewarding

outcomes, and if so, how does the relationship between reward- and threat-related potentiation change during the course of adolescence? Third, are there gender differences in potentiation to S^D that predict rewarding and threatening outcomes that can help explain why males are more likely to engage in risk-taking behaviours and females are more likely to experience greater anxiety levels (Byrnes *et al.*, 1999; Lewinsohn *et al.*, 1998)?

The current study was designed to address these questions, and examined reinforcement-dependent potentiation to visual cues that predicted rewarding and threatening outcomes in males and females during three distinct stages of adolescence: preadolescence (9-12 years), mid-adolescence (13-17 years), and late adolescence (18-23 years). In order to examine the time course of these processes, reinforcement-dependent potentiation was indexed by early (N170) and late (LPP) ERPs that have been shown to be modulated by motivationally salient stimuli (Keil *et al.*, 2002; Levita *et al.*, 2014; Rellecke, Sommer & Schacht, 2013; Sabatinelli, Lang, Keil & Bradley, 2007; Schupp *et al.*, 2000).

4.1.2. N170

The N170 is a negative visual component that peaks approximately 170 ms post stimulus onset. N170 amplitudes are maximal in occipitotemporal regions, with source localisation studies reporting that the N170 component originates from the superior temporal sulcus and fusiform gyrus (Sadeh *et al.*, 2010). The N170 has been shown to reflect early visual processing and has greater amplitudes in response to human faces compared to non-face stimuli (Bentin, Allison, Puce, Perez & McCarthy, 1996; Jeffreys, 1989). Notably, greater N170 amplitudes are also elicited by conditioned and unconditioned emotional stimuli compared to control stimuli, including learned danger cues (Dolan, Heinze, Hurlemann & Hinrichs, 2006; Levita *et al.*, 2014; Pizzagalli *et al.*, 2003) and emotional facial expressions (Batty & Taylor, 2003; Blau, Maurer, Tottenham & McCandliss, 2007; Hinojosa, Mercado & Carretié, 2015; Rellecke *et al.*, 2013; Schyns, Petro & Smith, 2007). Potentiation of early visual components, such as the N170, to motivationally salient stimuli is thought to result from dynamic re-entrant processing loops between the amygdala and visual cortex (Dolan, 2002; Vuilleumier, 2005). These re-entrant processing loops modulate cortical firing in the visual cortex to facilitate the processing of salient stimuli (Keil *et al.*, 2009; Sabatinelli, Lang, Bradley, Costa & Keil, 2009).

Developmentally, the N170 has been identified in children as young as 4 years old (Batty & Taylor, 2006; Taylor *et al*., 2001). It has been widely reported that N170 amplitudes reduce in size and become more negative during the transition from late childhood to adulthood (Hileman, Henderson, Mundy, Newell & Jaime, 2011; Levita *et al*., 2014; Taylor *et al*., 1999). These age-related changes in N170 amplitudes are thought to reflect developments in white and grey matter during childhood and adolescence (see Chapter 1.4). The N170 seems to have a similar function throughout development, with face and emotional stimuli eliciting larger N170 amplitudes than non-face and unemotional stimuli (Levita *et al*., 2014; Taylor *et al*., 2001).

4.1.3. LPP

In contrast to the N170, the LPP is a slow, positive component that emerges 300-400 ms post stimulus onset and usually lasts for the duration of the stimulus presentation (Cuthbert *et al*., 2000). However, like the N170, studies have reported that the LPP is potentiated in response to salient stimuli; LPP amplitudes are greater for positive and negative visual stimuli compared to neutral visual stimuli (Cuthbert *et al*., 2000; Schupp *et al*., 2000; Schupp, Junghofer, Weike & Hamm, 2004). It has therefore been suggested that the LPP reflects selective attention to motivationally salient stimuli (Cuthbert *et al*., 2000; Schupp, Flaisch, Stockburger & Junghöfer, 2006). The LPP has maximal amplitude around the midline over the parietal cortex. Despite this, a wide neural network generates the LPP, which involves concurrent activity in brain regions associated with visual and attentional processing, including the lateral occipital, parietal, and inferotemporal cortices, as well as in brain regions associated with emotional processing, including the orbitofrontal cortex, insula, anterior cingulate cortex, ventral striatum, and amygdala (Liu, Huang, McGinnis-Deweese, Keil & Ding, 2012; Moratti, Saugar & Strange, 2011; Sabatinelli, Keil, Frank, & Lang, 2013; Sabatinelli *et al*., 2007). It has been suggested that greater coupling between these regions underlies enhanced LPP amplitudes to emotional stimuli compared to neutral stimuli (Moratti *et al*., 2011).

Like the N170, the LPP has been identified in children as young as 4 years old (Hua *et al*., 2014). Since the N170 and LPP have both been observed in young children, they are good candidates for examining how neural responses to salient stimuli change across development (Nelson & McCleery, 2008). The LPP is thought to have a similar function in children, adolescents, and adults, whereby positively and negatively

valenced visual stimuli elicit larger LPP amplitudes than neutral visual stimuli (Dennis & Hajcak, 2009; Hajcak & Dennis, 2009; Hua *et al*., 2014; Kujawa, Klein & Hajcak, 2012; Kujawa, Klein & Proudfit, 2013; Solomon, DeCicco & Dennis, 2012). Developmental differences have also been observed, with LPP amplitudes decreasing throughout childhood and adolescence (Kujawa *et al*., 2012; MacNamara *et al*., 2016). In addition, the topography of the LPP changes with age; maximal LPP amplitudes shift from occipitoparietal regions in children and adolescents (Kujawa *et al*., 2012; Kujawa *et al*., 2013) to more centroparietal regions in adults (Hajcak, Weinberg, MacNamara & Foti, 2012). The developmental changes in the LPP are consistent with the changes in brain structure, function, and re-organisation during adolescence (Thompson *et al*., 2000). The human brain matures in a back-to-front fashion, with occipital areas maturing first and prefrontal areas maturing last (Gogtay *et al*., 2004); the PFC undergoes a protracted development across childhood and adolescence that extends well into the third decade of life (Gotgay *et al*., 2004; Huttenlocher, 1979; see Chapter 1.4). A broad frontal-parietal-occipital-limbic network has been shown to generate and modulate the LPP (Liu *et al*., 2012; Moratti *et al*., 2011). Thus, it is possible that changes in LPP topography are the result of the LPP becoming more reliant on prefrontal regions as the PFC matures.

4.1.4. N170 and LPP: classical and instrumental conditioning

This study aimed to examine the age- and gender-related changes in anticipatory representations to rewarding and threatening outcomes during the course of adolescence, as indexed by the N170 and LPP. Associative learning tasks, i.e., classical and instrumental conditioning, provide an ideal way of dissociating the neural activity associated with anticipatory processes from neural activity associated with consummatory processes (see Chapter 1.7.1). Aversive classical conditioning tasks in adults have revealed that both the N170 (Dolan *et al*., 2006; Pizzagalli *et al*., 2003) and LPP (Franken, Huijding, Nijs & van Strien, 2011; Pastor *et al*., 2015; Pizzagalli *et al*., 2003) are potentiated to conditioned stimuli that predict the onset of a threatening unconditioned stimulus compared to control stimuli. These findings therefore suggest that both early sensory and late cognitive processes are modulated when adults anticipate threatening outcomes. Potentiation of such processes is thought to reflect motivated attention, where cues that signal potential threat engage attentional resources in order to facilitate appropriate survival behaviours (Lang,

Bradley, & Cuthbert, 1997). In contrast to aversive classical conditioning, the ERP components modulated by reward anticipation in appetitive classical conditioning tasks remain largely unexplored. Initial evidence in adults suggests that the P3b, a late positive component related to the LPP, has greater amplitudes to visual cues signalling a rewarding outcome compared to a neutral outcome (Franken, van Strien, Bocanegra & Huijding, 2011).

Crucially, actions are guided by the anticipation of outcomes (Elsner & Hommel, 2001). Thus, biases in outcome anticipation are thought to have a cascading effect on action selection, preparation, and implementation (Hegdé & Felleman, 2007; Freese & Amaral, 2005; Lamme & Roelfsema, 2000; Lang & Bradley, 2010; Sugase *et al*., 1999; Vuilleumier, 2005). Indeed, biases in outcome anticipation and suboptimal action selection are thought to underlie high levels of risk-taking and anxiety. Specifically, engagement in risky behaviours and related pathologies such as gambling disorder and substance abuse are thought to be driven by a strong preference for immediate over delayed rewards, which often results in impulsive behaviour (Petry, 2001). Moreover, anxiety disorders are characterised by avoidance behaviours, whereby individuals actively or passively avoid situations or stimuli they anticipate to be threatening or harmful (Salkovskis, 1991).

Instrumental conditioning tasks provide a way to examine anticipatory neural activity to cues that signal the onset of rewarding or threatening outcomes that are conditional on actions (Skinner, 1948; see Chapter 1.7.1). Instrumental tasks therefore provide a way to examine the functional significance of reward- and threat-related anticipatory neural activity, and its possible role in guiding action-outcome behaviours. Accordingly, this study used an instrumental task to investigate reinforcement-dependent potentiation to S^D that predicted either rewarding or threatening outcomes to determine whether reward- and threat-related anticipatory activity could account for the developmental differences in risk-taking and anxiety found in this sample of adolescents (Chapter 3).

There is currently a scarcity of ERP studies examining anticipatory neural activity using instrumental tasks in both adolescents and adults. To the author's knowledge, only one study has examined modulation of the N170 in an instrumental task (Levita *et al*., 2014), and no study to date has examined modulation of the LPP in an instrumental task. However, there is some evidence in adults that the P3b has greater amplitudes to visual cues that signal a rewarding outcome in instrumental

tasks (Broyd *et al*., 2012; Goldstein *et al*., 2006; Santesso *et al*., 2012). Thus, the current study provides novel insights into the role of the N170 and LPP in reward- and threat-related anticipatory processes associated with specific response-outcome contingencies.

Notably, fMRI studies have reported that mid-adolescents have greater activity in subcortical limbic regions when anticipating rewarding and threatening outcomes in instrumental tasks compared to younger and older participants (Galván & McGlennen, 2013; Padmanabhan *et al*., 2011). Studies have also reported that reward- and threat-related subcortical activity is associated with risk-taking behaviours and anxiety levels during adolescence (Barkley-Levenson & Galván, 2014; Braams *et al*., 2015; Galván *et al*., 2007; Hare *et al*., 2008). Thus, examining reward- and threat-related potentiation of the N170 and LPP in preadolescents, mid-adolescents, and late adolescents provided a way to assess whether N170 and LPP potentiation peaks during mid-adolescence compared to early and late adolescence, and whether this peak was associated with participants' risk-taking behaviours and anxiety levels.

4.1.5. The influence of puberty on N170 and LPP potentiation

The onset of puberty varies markedly between individuals; puberty can begin any time between the ages of 8 and 13 years for healthy females and between 9 and 14 years for healthy males (Sørensen *et al.,* 2013). Thus, chronological age and pubertal development are highly dissociable during adolescence (Marshall & Tanner, 1969; Marshall & Tanner, 1970). Notably, there is converging evidence to suggest that more advanced pubertal status is linked to greater levels of reward- and threat-related neural activity, risk-taking behaviours, and anxiety levels (Braams *et al*., 2015; Collado *et al*., 2014; Moore *et al*., 2012; Reardon *et al*., 2009). Consistently, pubertal stage was positively associated with real world risk-taking behaviours in this sample of adolescents (Chapter 3). Despite this, the influence of puberty on ERPs that are modulated by affective stimuli has received surprisingly little attention. To date, no study has investigated the relationship between puberty and N170 amplitude, and only one study has assessed the relationship between puberty and LPP amplitude (Nelson, Perlman, Hajcak, Klein & Kotov, 2015). Interestingly, Nelson *et al*. (2015) reported no affect of pubertal stage on LPP amplitudes in response to affective pictures. However, this study was limited to females aged 13.5–15.5 years. Therefore, the influence of puberty on the development of the LPP and N170 during adolescence requires further

investigation. Consequently, the current study aimed to examine the relationships between pubertal stage and ERP potentiation to S^D that predict rewarding and threatening outcomes in male and female adolescents aged 9-17 years while controlling for chronological age.

4.1.6. The current study

In the current study, 31 preadolescents aged 9-12 years (16 females, 15 males), 30 mid-adolescents aged 13-17 years (15 females, 15 males), and 34 late adolescents aged 18-23 years (18 females, 16 males) completed an instrumental conditioning task while having their brain activity measured using EEG. In this task, participants learned to emit or omit a motor response to S^D to either obtain a reward (winning points) or avoid a threat (losing points). Participants also had to emit or omit a motor response in response to two control cues that did not predict the onset of a rewarding or threatening outcome. The control cues provided a way to determine whether age-related changes in the N170 and LPP resulted from differences in the potentiation of anticipatory responses to S^D or from developmental differences in motor, motor-preparation, or visual processes associated with an instrumental procedure. Participants also completed a battery of self-report questionnaires measuring risk-taking, anxiety, and depression, and a behavioural task measuring risk-taking tendencies (Chapter 3).

This study had four aims. The primary aim of this study was to examine age- and gender-related changes in the potentiation of N170 and LPP amplitudes to S^D compared to control stimuli. The age- and gender-related differences in N170 and LPP amplitudes were also explored. Based on the research discussed above, it was predicted that: (1) N170 and LPP amplitudes would be potentiated to S^D predicting rewarding and threatening outcomes compared to control cues; (2) reinforcement-dependent potentiation of the N170 and LPP would be greater in mid- adolescents compared to preadolescents and late adolescents; (3) irrespective of condition, N170 and LPP amplitudes would decrease from preadolescence to late adolescence (Hileman *et al*., 2011; Kujawa *et al*., 2012; Levita *et al*., 2014; MacNamara *et al*., 2016); (4) males would show greater N170 and LPP reinforcement-dependent potentiation to S^D predicting a rewarding outcome, whereas females would show greater N170 and LPP reinforcement-dependent potentiation to S^D predicting a threatening outcome (Byrnes *et al*., 1999; Lewinsohn *et al.*, 1998).

The second aim of this study was to examine the functional significance of N170 and LPP reinforcement-dependent potentiation by assessing the relationships between ERP potentiation to S^D and participants' reaction times for corresponding conditions. It was predicted that reaction time would be negatively associated with N170 and LPP potentiation for all groups. Moreover, if mid-adolescents are hypersensitive to rewards (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg *et al*., 2008) and hypersensitive to threats (Casey *et al*., 2010; Levita *et al*., 2014), it is possible that mid-adolescents will have stronger associations between reward- and threat-related ERP potentiation and reaction times (i.e., greater ERP potentiation associated with faster reactions times) than preadolescents and late adolescents.

The third aim of this study was to assess whether N170 and LPP potentiation to rewarding and threatening outcomes was associated with risk-taking behaviours and anxiety levels, respectively. It was predicted that N170 and LPP potentiation to rewarding outcomes would be positively associated with risk-taking behaviours, and N170 and LPP potentiation to threatening outcomes would be positively associated with anxiety levels. It was also predicted that mid-adolescents would show stronger associations between ERP potentiation and measures of risk-taking and anxiety than preadolescents and late adolescents, and that females would be more likely to show relationships between threat-related activity and anxiety whereas males would be more likely to show relationships between reward-related activity and risk-taking.

The fourth and final aim of this study was to examine the relationships between pubertal stage and ERP potentiation to S^D that predict rewarding and threatening outcomes in male and female adolescents aged 9-17 years while controlling for chronological age. Since more advanced pubertal stage has been associated with greater levels of reward- and threat-related neural activity (Braams *et al*., 2015), it was predicted that more advanced pubertal status would be associated with greater levels of N170 and LPP potentiation to S^D that predict rewarding and threatening outcomes.

4.2. Method

4.2.1. Participants

The same cohort of participants took part in this study as in Chapter 3. In total, ninety-five volunteers aged 9-23 years old participated in the current study. One

participant (female aged 10 years) was excluded due to excessive EEG artefacts. Participant demographics for the final sample are displayed in Table 4.1. Participants were split into three age groups: preadolescents aged 9-12 years; mid-adolescents aged 13-17 years; and late adolescents aged 18-23 years. Detailed participant information is reported in Chapter 2.1.

4.2.2. Procedure

The procedure is reported in Chapter 2.2.

4.2.3. Instrumental conditioning task

The instrumental conditioning task used in this study was an extension of a validated avoidance paradigm used previously in a developmental EEG study (Levita *et al*., 2014) and fMRI study in adults (Levita *et al*., 2012). The instrumental task was composed of a reward block and an avoidance block. Both the reward and avoidance blocks included two S^D and two control stimuli. The S^D predicted a positive outcome (winning 10 points) in the reward block ($Reward^{Go}$, RG; $Reward^{NoGo}$, RN) and a negative outcome (losing 10 points) in the avoidance block ($Avoidance^{Go}$, AG; $Avoidance^{NoGo}$, AN). Participants were required to make the correct motor action (either emitting or

Table 4.1
Participant demographics

				Risk-taking behaviours *M* [95% CI]				Anxiety *M* [95% CI]	
Age Group	Gender	*n*	Age *M* (*SD*)	BART Pumps	BART Points	BART Balloons	YRBSS	STAI-T	STAI-T HADS-D
Preadolescents	Females	15	10.80 (1.26)	38.24 [31.56, 45.18]	5542.67 [4513.01, 6528.28]	7.07 [5.13, 9.40]	0.53 [0.20, 0.87]	54.89 [50.44, 59.34]	58.19 [51.87, 65.49]
	Males	15	10.53 (1.30)	40.90 [34.49, 46.68]	6024.00 [5165.89, 6810.72]	9.00 [7.13, 11.12]	0.93 [0.47, 1.40]	58.11 [52.58, 64.22]	58.88 [54.59, 63.47]
Mid-adolescents	Females	15	14.67 (1.59)	38.29 [32.04, 45.01]	5700.67 [4694.40, 6797.81]	8.13 [6.73, 9.60]	1.87 [1.27, 2.53]	54.67 [48.19, 61.48]	55.44 [50.81, 60.23]
	Males	15	14.80 (1.42)	41.11 [34.31, 47.51]	5949.33 [4957.31, 6836.17]	8.67 [6.73, 10.56]	4.00 [2.80, 5.13]	49.33 [41.91, 57.34]	46.31 [39.61, 52.66]
Late Adolescents	Females	18	20.39 (1.38)	46.63 [40.23, 53.12]	7034.44 [6054.39, 7936.30]	9.28 [7.89, 10.67]	4.22 [3.50, 4.94]	52.29 [45.14, 59.73]	52.52 [47.25, 57.51]
	Males	16	21.00 (1.55)	48.96 [42.30, 55.39]	6997.50 [6308.23, 7722.86]	10.50 [8.50, 12.36]	3.25 [2.25, 4.25]	53.52 [46.80, 59.84]	51.55 [45.06, 59.02]

Note. BART Pumps = BART number of adjusted pumps; BART Points = BART total points won; BART Balloons = BART number of burst balloons; YRBSS = Youth Risk Behaviour Surveillance Survey; STAI-T = State Trait Anxiety Inventory-Trait Anxiety; STAI-T HADS = State Trait Anxiety Inventory-Trait Anxiety controlling for Hospital Anxiety and Depression Scale-Depression Scale. 95% = bootstrapped 95% confidence intervals.

omitting an action) in order to win points (reward block) or avoid losing points (avoidance block). The two control stimuli were not associated with rewarding or threatening outcomes (Control^{Go}, CG; Control^{NoGo}, CN). See Chapter 2.4.2.1 for full details regarding the instrumental conditioning task.

4.2.4. Apparatus

The apparatus is reported in Chapter 2.4.2.1.1.

4.2.5. EEG recording

The EEG recording is reported in Chapter 2.4.1.

4.2.6. EEG preprocessing

The EEG preprocessing stream is reported in Chapter 2.4.3. Once the EEG data had been cleaned, epoched, and averaged, electrodes were clustered on the basis of the topographical maps and previous studies (e.g., Blau *et al.*, 2007; Cuthbert *et al.*, 2000; Hua *et al.*, 2014) in order to reduce the number of statistical comparisons. The N170 was clustered using electrodes O1, PO3, and PO7 in the left hemisphere, and O2, PO4, and PO8 in the right hemisphere. Selecting both right and left hemisphere electrode clusters for the N170 provided a way to assess laterality effects. The LPP was identified

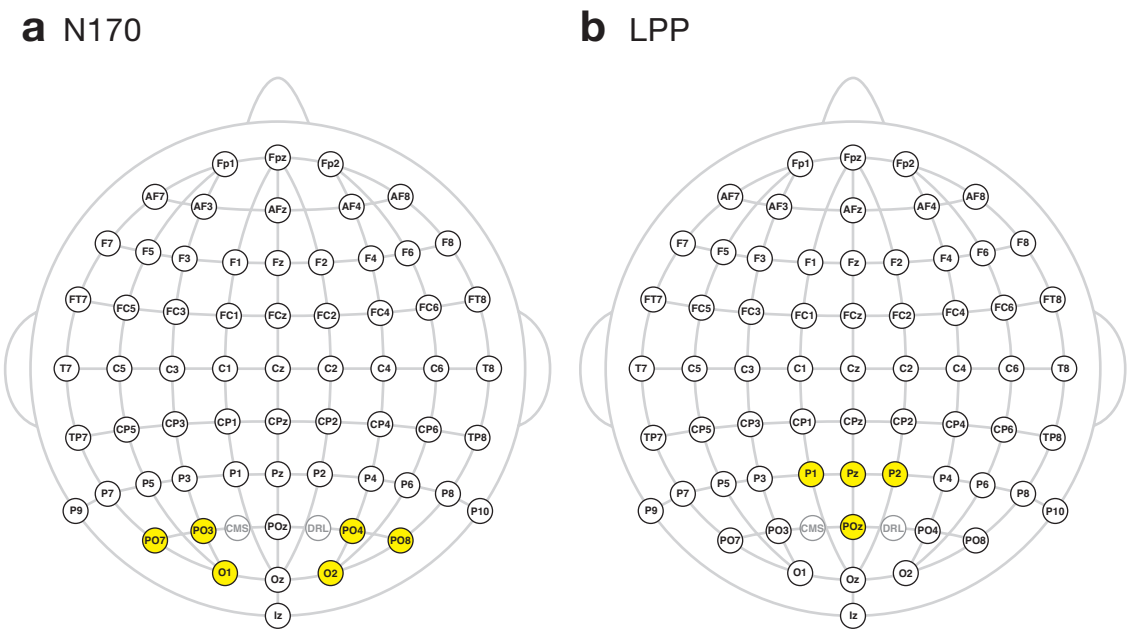

Figure 4.1 Electrode clusters for the N170 (**a**) and LPP (**b**).

at central parietal electrodes and therefore Pz, P1, P2, and POz were clustered for the LPP analyses. Figure 4.1 displays the electrode clusters for the N170 and LPP. Based on previous ERP work (Blau *et al*., 2007; Cuthbert *et al*., 2000; Kujawa *et al*., 2012) and the ERP waveforms, time windows of 150-220 ms and 400-700 ms were selected for the N170 and LPP, respectively. Since peak ERP amplitudes are highly influenced by noise (Luck, 2014; Woodman, 2010), rectified area under the curve within each time window was used to quantify ERP amplitudes.

4.2.7. Behavioural measures

4.2.7.1. Risk-taking behaviours

Risk-taking behaviours were measured using the BART (Lejuez *et al*., 2002) and YRBSS (Aklin *et al*., 2005) (see Chapter 2.3.1).

4.2.7.2. Anxiety and depression

Anxiety was measured using the STAI (Spielberger *et al*., 1983; see Chapter 2.3.4). Only the STAI-T was used in the current study since participants completed the behavioural measures on a different day to the EEG session. Depression was measured using the HADS-D (Zigmond & Snaith, 1983; Chapter 2.3.5).

4.2.7.3. Pubertal development

Pubertal development was assessed in 9-17 year olds using the PDS (Petersen *et al*., 1988; see Chapter 2.3.6). Consistent with previous studies (Marshall & Tanner, 1969; Marshall & Tanner, 1970), an independent t-test revealed that PDS scores were slightly higher for females aged 9-17 years (M = 2.73, 95% CI [2.41, 3.02]) compared to males aged 9-17 years (M = 2.30, 95% CI [2.03, 2.58]) ($t(58) = 2.05$, $p = 0.045$), indicating that females were at a slightly later stage in their pubertal development compared to males.

4.3. Results

4.3.1. Instrumental task performance

Task performance was indexed by accuracy and reaction time. Accuracy scores reflect the percentage of correct responses for each condition. Reaction times were measured for conditions that required a motor response ($Reward^{Go}$, $Avoidance^{Go}$, $Control^{Go}$), and reflect the time it took for participants to make a motor response while

the yellow fixation cross was displayed on the screen. Only trials with correct behavioural responses were included in the reaction time analyses.

4.3.1.1. Accuracy

Figure 4.2 and Supplementary Table 4.1 (Appendix 2) display the means and 95% bootstrapped confidence intervals for task accuracy. To assess the age- and gender-related differences in task accuracy, two mixed-design ANOVAs were conducted with *Condition* (reward block: Reward^{Go}, Reward^{NoGo}, Control^{Go}, Control^{NoGo}; avoidance block: Avoidance^{Go}, Avoidance^{NoGo}, Control^{Go}, Control^{NoGo}) as the within-group factor, and *Gender* (females, males) and *Age Group* (preadolescents, mid-adolescents, late adolescents) as the between-group factors. Separate ANOVAs were conducted for the reward and avoidance blocks. Greenhouse-Geisser corrections are reported where sphericity has been violated. ANOVA pairwise comparisons were corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5).

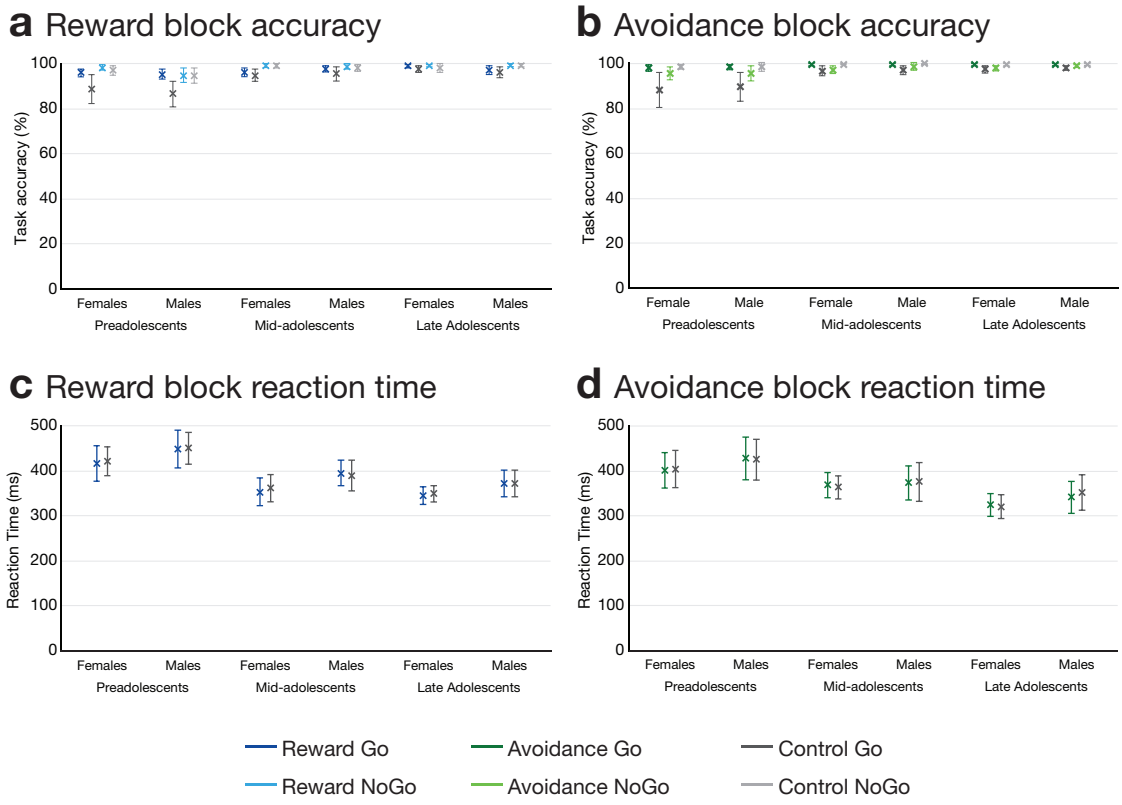

Figure 4.2 Group means for task accuracy for the reward block (**a**) and avoidance block (**b**), and for reaction time for the reward block (**c**) and avoidance block (**d**). Error bars represent 95% bootstrapped confidence intervals.

Task accuracy was very high, with all groups responding correctly to at least 85% of trials for all conditions. Despite this, the ANOVAs revealed small, but significant, differences between conditions and age groups for the reward and avoidance blocks. The findings show that preadolescents made significantly more errors in both the reward and avoidance blocks compared to mid-adolescents and late adolescents. Furthermore, all groups made significantly more errors in response to conditions that required a motor response compared to conditions that required inhibition of a motor response. No gender differences were observed in task accuracy.

4.3.1.1.1. Reward block

The ANOVA for the reward block revealed a main effect of *Age Group* ($F(2, 88) = 14.50$, $p < 0.001$, $\eta_p^2 = 0.25$), revealing that preadolescents ($M = 93.82$, 95% CI [92.59, 95.04]) made slightly more errors than both mid-adolescents ($M = 97.35$, 95% CI [96.12, 98.57]) ($p < 0.001$) and late adolescents ($M = 98.15$, 95% CI [97.00, 99.30]) ($p < 0.001$). By contrast, no difference was found between mid-adolescents and late adolescents ($p = 0.345$). A main effect of *Condition* was also found ($F(1.64, 143.91) = 36.01$, $p < 0.001$, $\eta_p^2 = 0.29$). The pairwise comparisons revealed that all conditions significantly differed from each other ($Reward^{Go}$: $M = 96.80$, 95% CI [96.13, 97.46]; $Reward^{NoGo}$: $M = 98.14$, 95% CI [97.57, 98.70]; $Control^{Go}$: $M = 93.14$, 95% CI [91.64, 94.64]; $Control^{NoGo}$: $M = 97.68$, 95% CI [96.90, 98.45]), except $Reward^{NoGo}$ and $Control^{NoGo}$ ($p = 0.146$). These findings reveal that, across age groups, participants made more errors in response to $Control^{Go}$ cues compared to the other conditions ($Reward^{Go}$, $Reward^{NoGo}$, $Control^{NoGo}$). Participants also made more errors in response to $Reward^{Go}$ cues than for $Reward^{NoGo}$ and $Control^{NoGo}$ cues. Finally, no differences were found between the $Reward^{NoGo}$ and $Control^{NoGo}$ cues.

The *Condition* and *Age Group* main effects for the reward block were qualified by a *Condition* by *Age Group* interaction ($F(3.27, 143.91) = 7.46$, $p < 0.001$, $\eta_p^2 = 0.15$). This interaction was followed-up with separate repeated measures ANOVAs for each of the age groups, with *Condition* as the within-group factor. The follow-up ANOVAs revealed a main effect of *Condition* for preadolescents ($F(1.35, 39.02) = 20.03$, $p < 0.001$, $\eta_p^2 = 0.41$), mid-adolescents ($F(1.98, 57.47) = 12.16$, $p < 0.001$, $\eta_p^2 = 0.30$) and late adolescents ($F(1.62, 53.36) = 4.89$, $p = 0.003$, $\eta_p^2 = 0.13$). These findings show that all groups made significantly more errors in response to $Control^{Go}$ cues compared to the other conditions ($Reward^{Go}$, $Reward^{NoGo}$, $Control^{NoGo}$). Mid-adolescents and late

adolescents also made more errors in response to Reward^{Go} cues than cues requiring inhibition of a motor response (Reward^{NoGo}, Control^{NoGo}).

4.3.1.1.2. Avoidance block

The ANOVA for the avoidance block also revealed a main effect of *Age Group* ($F(2, 88) = 12.24$, $p < 0.001$, $\eta_p^2 = 0.22$), revealing that preadolescents ($M = 95.30$, 95% CI [94.18, 96.41]) made slightly more errors than both mid-adolescents ($M = 98.51$, 95% CI [97.40, 99.62]) ($p < 0.001$) and late adolescents ($M = 98.77$, 95% CI [97.72, 99.81]) ($p < 0.001$). No difference was found between mid-adolescents and late adolescents ($p = 0.739$). A main effect of *Condition* was also found ($F(1.37, 120.59) = 31.63$, $p < 0.001$, $\eta_p^2 = 0.26$). The pairwise comparisons revealed that all conditions significantly differed from each other (Avoidance^{Go}: $M = 97.32$, 95% CI [96.55, 98.09]; Avoidance^{NoGo}: $M = 99.03$, 95% CI [98.71, 99.36]; Control^{Go}: $M = 94.50$, 95% CI [92.89, 96.10]; Control^{NoGo}: $M = 99.25$, 95% CI [98.90, 99.59]), except Avoidance^{NoGo} and Control^{NoGo} ($p = 0.146$). Consistent with the reward block, these findings reveal that, across age groups, participants made more errors in response to Control^{Go} cues compared to the other conditions (Avoidance^{Go}, Avoidance^{NoGo}, Control^{NoGo}). Participants also made more errors in response to Avoidance^{Go} cues than for Avoidance^{NoGo} and Control^{NoGo} cues. Finally, no differences were found between the Avoidance^{NoGo} and Control^{NoGo} cues. Taken together with the reward block, these findings suggest that participants made more errors in response to cues requiring a motor response than to cues requiring inhibition of a motor response.

A *Condition* by *Age Group* interaction qualified the main effects of *Condition* and *Age Group* ($F(2.74, 120.59) = 8.37$, $p < 0.001$, $\eta_p^2 = 0.16$). The *Condition* by *Age Group* interaction was followed-up with separate repeated-measures ANOVAs for each of the age groups, with *Condition* as the within-group factor. The follow-up ANOVAs revealed a main effect of *Condition* for preadolescents ($F(1.28, 37.05) = 15.78$, $p < 0.001$, $\eta_p^2 = 0.35$), mid-adolescents ($F(1.73, 50.24) = 14.64$, $p < 0.001$, $\eta_p^2 = 0.34$) and late adolescents ($F(2.06, 67.98) = 11.79$, $p < 0.001$, $\eta_p^2 = 0.26$). These findings show that all groups made significantly more errors in response to Control^{Go} cues compared to the other conditions (Avoidance^{Go}, Avoidance^{NoGo}, Control^{NoGo}). All groups also made more errors in response to Avoidance^{Go} cues than to cues requiring inhibition of a motor response (Avoidance^{NoGo}, Control^{NoGo}). Late adolescents also made more errors in response to Avoidance^{NoGo} cues compared to Control^{NoGo} cues.

4.3.1.2. Reaction time

Figure 4.2 and Supplementary Table 4.1 (Appendix 2) display the means and 95% bootstrapped confidence intervals for task reaction time. Age- and gender-related differences in reaction time were assessed using two mixed-design ANOVAs with *Condition* (reward block: Reward^{Go}, Control^{Go}; avoidance block: Avoidance^{Go}, Control^{Go}) as the within-group factor, and *Gender* (females, males) and *Age Group* (preadolescents, mid-adolescents, late adolescents) as the between-group factors. Separate ANOVAs were conducted for the reward and avoidance blocks. No main or interaction effects were found for reaction time for either the reward or avoidance block, indicating that reaction times were equivalent across groups.

4.3.2. N170

4.3.2.1. N170 scalp topography

The N170 topographical maps are displayed in Figure 4.3. Consistent with the N170 literature (Levita *et al*., 2014; Rossion *et al*., 2000), the N170 topographical maps revealed maximal activity in occipitotemporal regions. Developmental patterns were also observed, with neural activity in occipitotemporal regions reducing in magnitude across adolescence. There were similar patterns of neural activity for S^D and control stimuli, with the difference maps revealing minimal differences between conditions.

4.3.2.2. N170 amplitude

Grand average waveforms and group means for the N170 are displayed in Figures 4.4 and 4.5 for the reward block and Figures 4.6 and 4.7 for the avoidance block. Age- and gender-related differences in N170 amplitudes were assessed using mixed-design ANOVAs, with *Condition* (reward block: Reward^{Go}, Reward^{NoGo}, Control^{Go}, Control^{NoGo}; avoidance block: Avoidance^{Go}, Avoidance^{NoGo}, Control^{Go}, Control^{NoGo}) and *Laterality* (left hemisphere, right hemisphere) as the within-group factors, and *Gender* (females, males) and *Age Group* (preadolescents, mid-adolescents, late adolescents) as the between-group factors. Since the reward block always preceded the avoidance block, separate ANOVAs were conducted for the reward and avoidance blocks to account for potential time effects. Greenhouse-Geisser corrections are reported where sphericity has been violated. ANOVA pairwise comparisons were corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5).

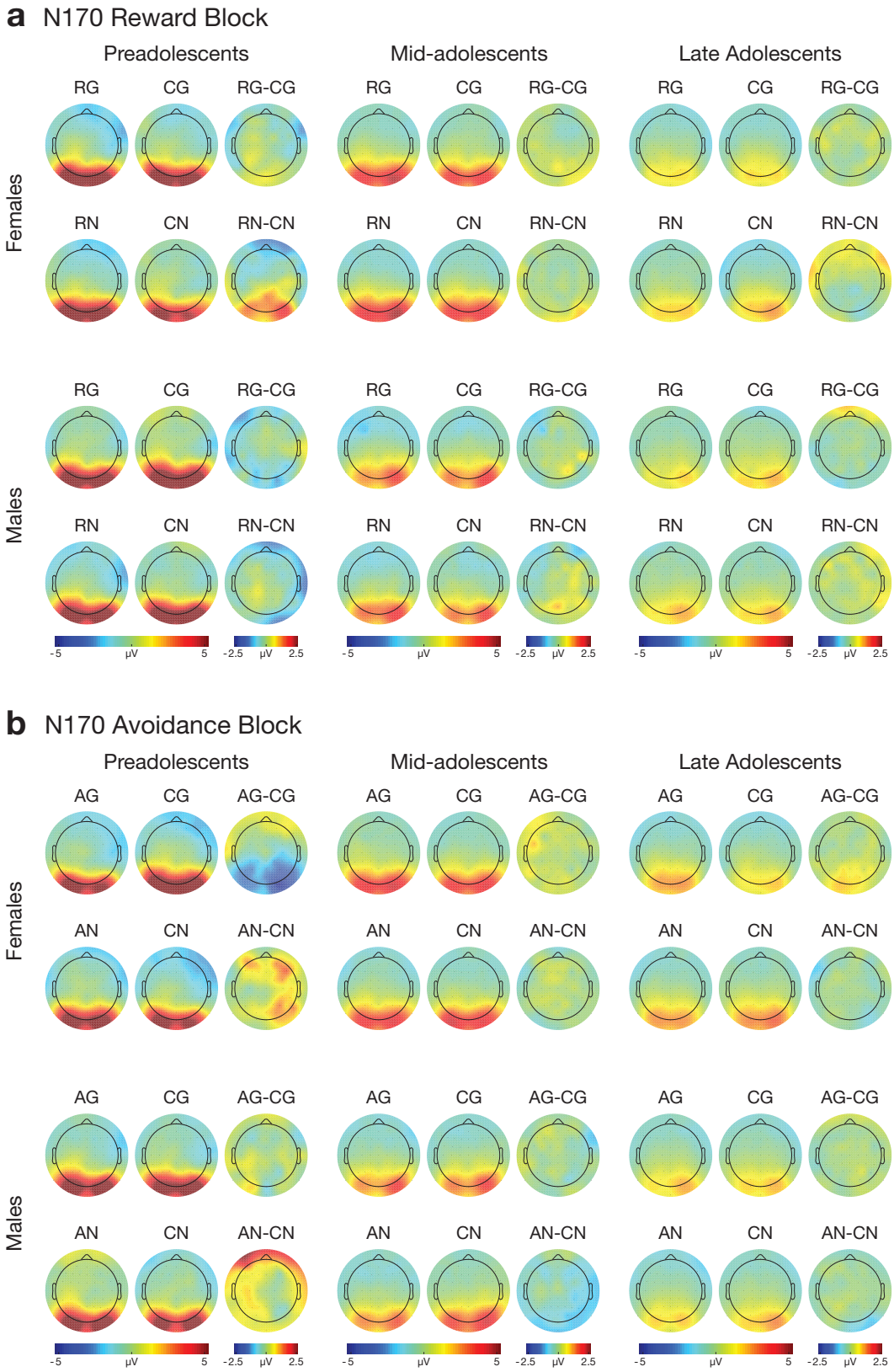

Figure 4.3 Scalp topography of the N170 (150-220 ms) for the reward block (**a**) and avoidance block (**b**). Topographical maps are shown for the S^D and control stimuli, as well as for the differences in activity between S^D and their respective control condition. RG = Reward^{Go}; RN = Reward^{NoGo}; AG = Avoidance^{Go}; AN = Avoidance^{NoGo}; CG = Control^{Go}; CN = Control^{NoGo}.

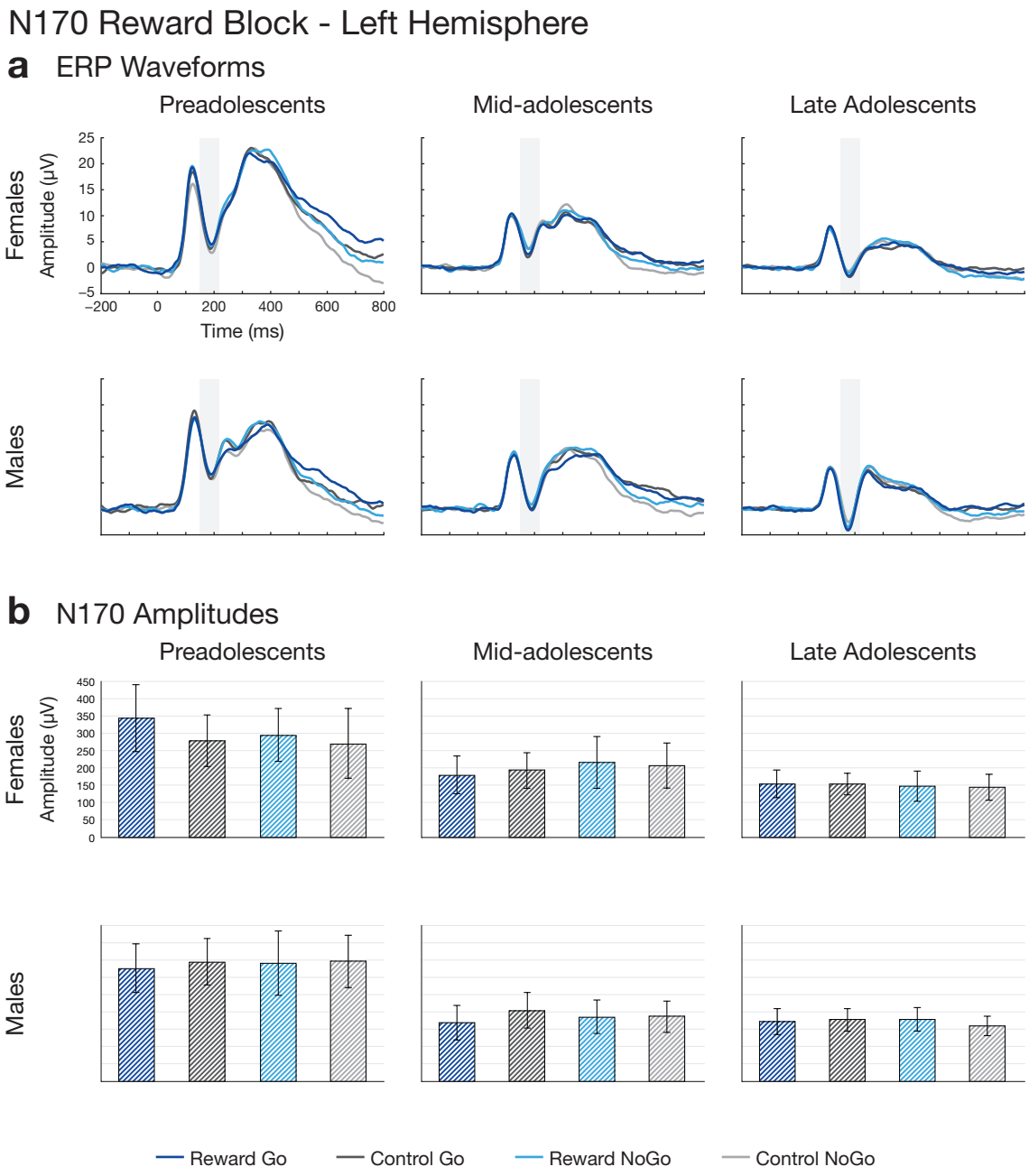

Figure 4.4 Grand averaged ERP waveforms (**a**) and N170 amplitude group means (**b**) during the reward block at the left occipitotemporal electrode cluster. The N170 was identified between 150-220 ms post stimulus onset, and is illustrated by the grey area on the ERP waveforms. Rectified area under the curve was used to quantify N170 amplitudes. Error bars represent 95% bootstrapped confidence intervals.

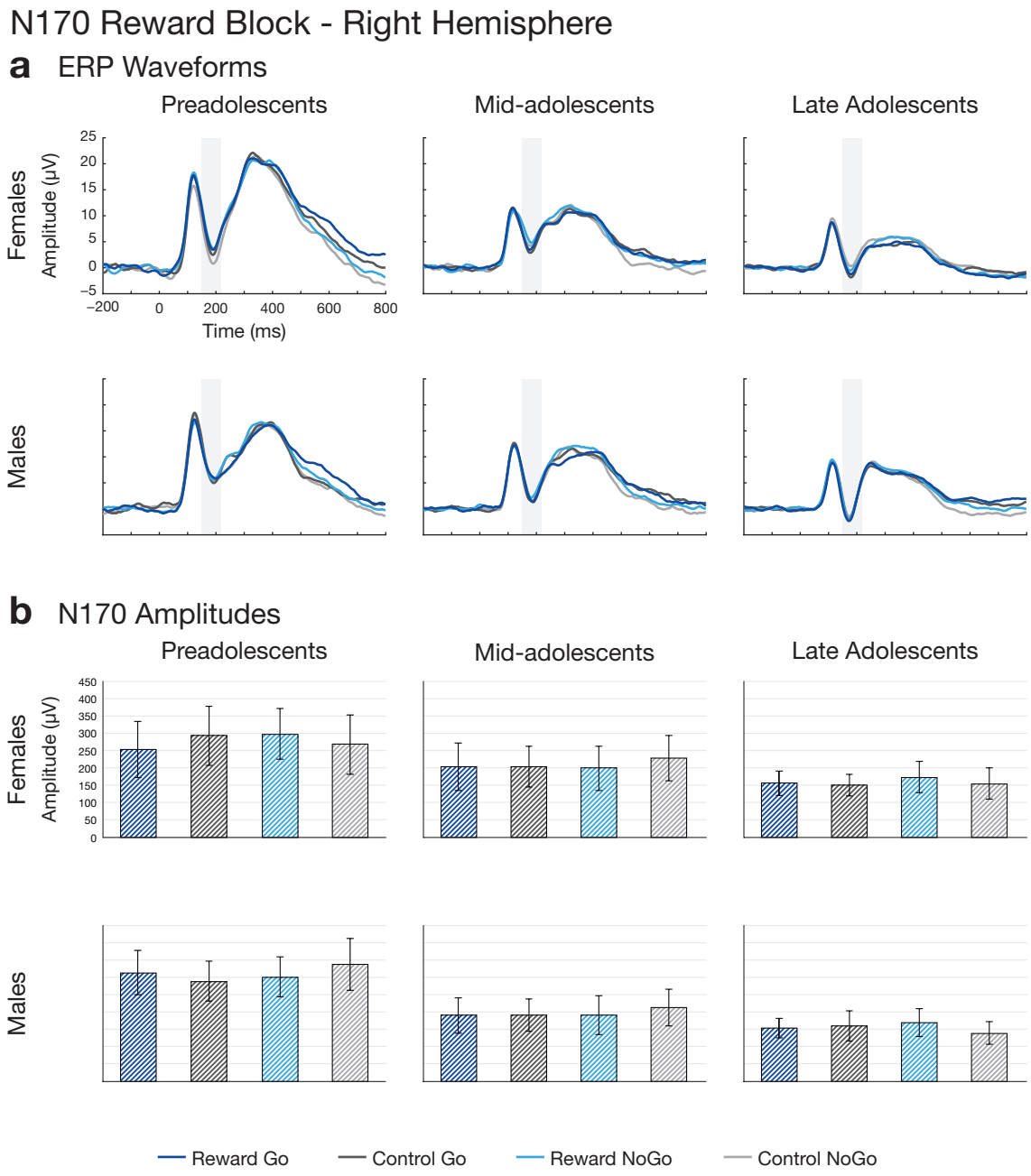

Figure 4.5 Grand averaged ERP waveforms (**a**) and N170 amplitude group means (**b**) during the reward block at the right occipitotemporal electrode cluster. The N170 was identified between 150-220 ms post stimulus onset, and is illustrated by the grey area on the ERP waveforms. Rectified area under the curve was used to quantify N170 amplitudes. Error bars represent 95% bootstrapped confidence intervals.

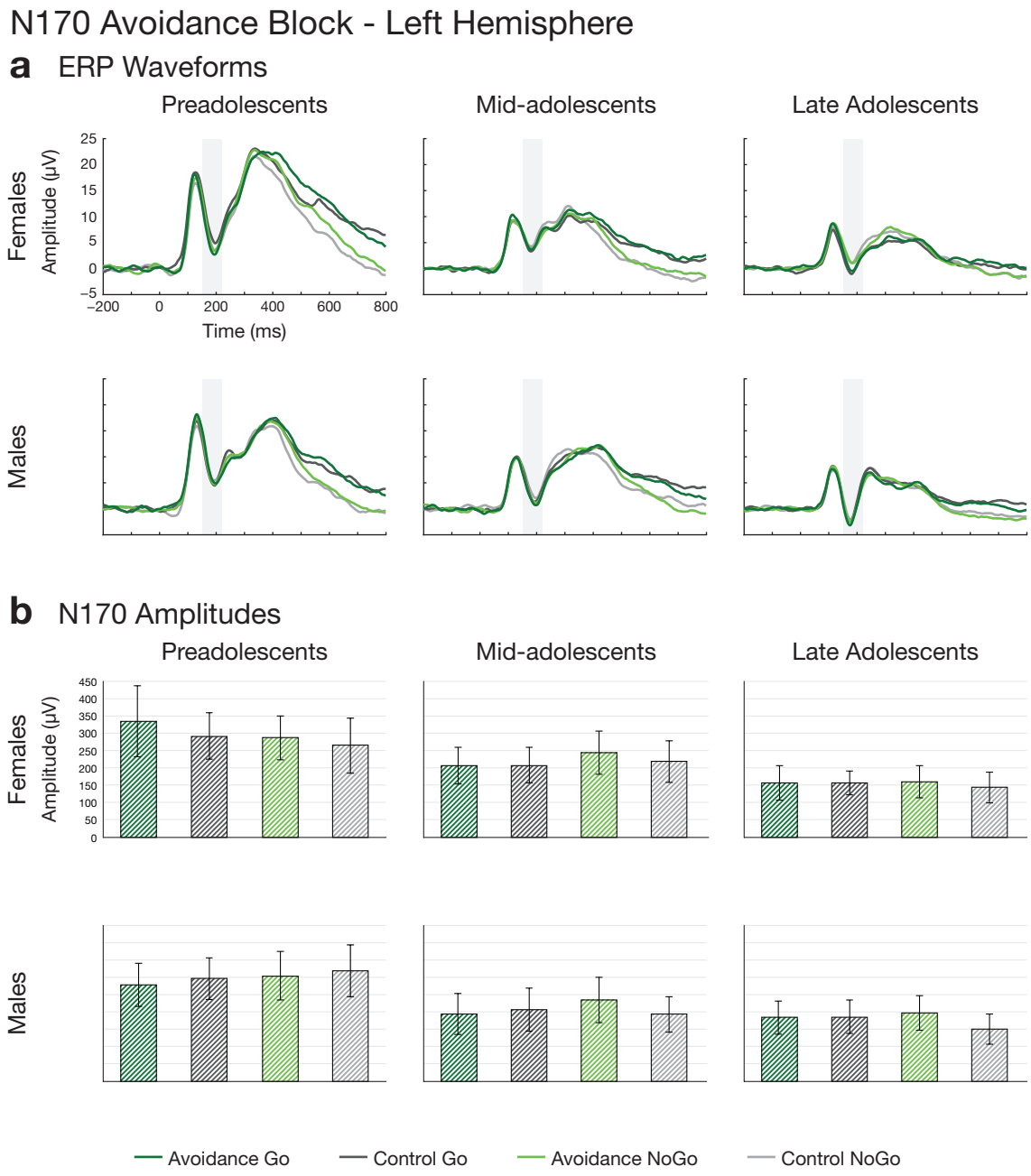

Figure 4.6 Grand averaged ERP waveforms (**a**) and N170 amplitude group means (**b**) during the avoidance block at the left occipitotemporal electrode cluster. The N170 was identified between 150-220 ms post stimulus onset, and is illustrated by the grey area on the ERP waveforms. Rectified area under the curve was used to quantify N170 amplitudes. Error bars represent 95% bootstrapped confidence intervals.

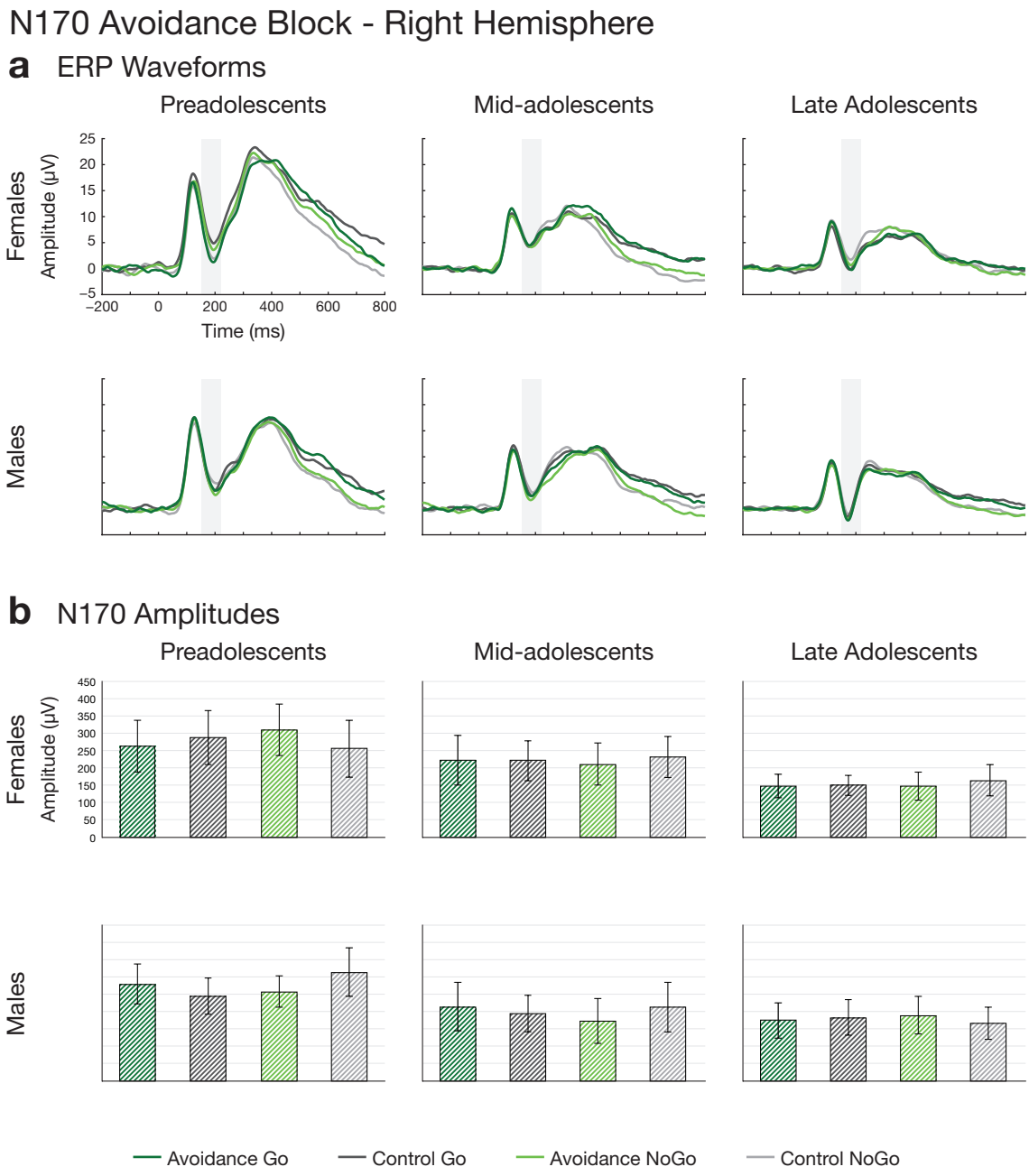

Figure 4.7 Grand averaged ERP waveforms (**a**) and N170 amplitude group means (**b**) during the avoidance block at the right occipitotemporal electrode cluster. The N170 was identified between 150-220 ms post stimulus onset, and is illustrated by the grey area on the ERP waveforms. Rectified area under the curve was used to quantify N170 amplitudes. Error bars represent 95% bootstrapped confidence intervals.

The main effect of *Age Group* was significant for the reward block ($F(2, 88) = 12.80$, $p < 0.001$, $\eta_p^2 = 0.23$) and the avoidance block ($F(2, 88) = 8.27$, $p < 0.001$, $\eta_p^2 = 0.16$). For the reward block, preadolescents had greater N170 amplitudes ($M = 307.80$, 95% CI [265.72, 349.88]) compared to both mid-adolescents ($M = 203.37$, 95% CI [161.29, 245.45]) ($p = 0.001$) and late adolescents ($M = 164.23$, 95% CI [124.63, 203.83]) ($p < 0.001$). No difference was found between mid-adolescents and late adolescents for N170 amplitudes in the reward block ($p = 0.182$). Similarly, for the avoidance block, preadolescents had greater N170 amplitudes ($M = 285.46$, 95% CI [240.99, 329.93]) compared to both mid-adolescents ($M = 206.45$, 95% CI [161.98, 250.92]) ($p = 0.014$) and late adolescents ($M = 161.44$, 95% CI [119.60, 203.28]) ($p < 0.001$). No difference was found between mid-adolescents and late adolescents for N170 amplitudes in the avoidance block ($p = 0.146$). These findings show that N170 amplitudes decreased from preadolescence to mid-adolescence in both the reward and avoidance blocks. No main effects of *Condition* or *Gender* were found for either the reward and avoidance blocks. Moreover, no interactions were found for the reward or avoidance blocks. Overall, these findings indicate that the N170 was not potentiated by visual cues predicting either a reward or threat.

4.3.3. LPP

4.3.3.1. LPP scalp topography

LPP topographical maps are displayed in Figure 4.8. In accordance with previous work (Dennis & Hajcak, 2009; Hajcak & Dennis, 2009; Kujawa *et al.*, 2012; Kujawa *et al.*, 2013; Solomon *et al.*, 2012), the LPP scalp topographical maps revealed maximal activity over central centroparietal regions for all groups. Developmental differences were also observed, with neural activity reducing in magnitude and becoming more focal in centroparietal regions throughout adolescence.

4.3.3.2. LPP amplitude

Grand average waveforms and group means for the LPP are displayed in Figures 4.9 and 4.10 for the reward and avoidance blocks, respectively. Age- and gender-related effects in LPP amplitudes were investigated using mixed-design ANOVAs, with *Condition* (reward block: $\text{Reward}^{\text{Go}}$, $\text{Reward}^{\text{NoGo}}$, $\text{Control}^{\text{Go}}$, $\text{Control}^{\text{NoGo}}$; avoidance block: $\text{Avoidance}^{\text{Go}}$, $\text{Avoidance}^{\text{NoGo}}$, $\text{Control}^{\text{Go}}$, $\text{Control}^{\text{NoGo}}$) as the within-group factor, and *Gender* (females, males) and *Age Group* (preadolescents, mid-adolescents, late

adolescents) as the between-group factors. As with the N170, separate ANOVAs were conducted for the reward and avoidance blocks to account for potential time effects. Greenhouse-Geisser corrections are reported where sphericity has been violated. To reduce the number of statistical tests, comparisons were made between S^D and their corresponding control condition to assess reinforcement-dependent potentiation ($Reward^{Go}$-$Control^{Go}$, $Reward^{NoGo}$-$Control^{NoGo}$, $Avoidance^{Go}$-$Control^{Go}$, $Avoidance^{NoGo}$-$Control^{NoGo}$), as well as within S^D and control stimuli to assess the effects of action selection on LPP amplitudes ($Reward^{Go}$-$Reward^{NoGo}$, $Avoidance^{Go}$-$Avoidance^{NoGo}$, $Control^{Go}$-$Control^{NoGo}$). The B-H procedure (Benjamini & Hochberg, 1995) was used to correct ANOVA pairwise comparisons (see Chapter 2.5).

For the reward block, a main effect of *Condition* was found ($F(3, 264) = 13.78$, $p < 0.001$, $\eta_p^2 = 0.14$). Pairwise comparisons revealed that LPP amplitudes were significantly greater for $Reward^{Go}$ cues ($M = 1004.54$, 95% CI [892.36, 1116.71]) compared to $Control^{Go}$ cues ($M = 902.15$, 95% CI [799.73, 1004.58]) ($p = 0.006$), and for $Reward^{NoGo}$ cues ($M = 884.83$, 95% CI [777.86, 991.80]) compared to $Control^{NoGo}$ cues ($M = 744.84$, 95% CI [655.13, 834.56]) ($p = 0.001$). This shows that, across all groups, LPP amplitudes were potentiated in response to cues that predicted a rewarding outcome compared to control cues. Furthermore, $Reward^{Go}$ cues had significantly larger LPP amplitudes than $Reward^{NoGo}$ cues ($p = 0.005$), and $Control^{Go}$ cues had significantly larger LPP amplitudes than $Control^{NoGo}$ cues ($p < 0.001$), showing that cues requiring a motor response had significantly larger LPP amplitudes than cues requiring the inhibition of a motor response. A main effect of *Age Group* was also found for the reward block ($F(2, 88) = 18.78$, $p < 0.001$, $\eta_p^2 = 0.30$). Pairwise comparisons revealed that, irrespective of condition, preadolescents had greater LPP amplitudes ($M = 1248.45$, 95% CI [1088.62, 1408.27]) compared to both mid-adolescents ($M = 828.26$, 95% CI [668.44, 988.09]) ($p < 0.001$) and late adolescents ($M = 575.56$, 95% CI [425.18, 725.95]) ($p < 0.001$). Mid-adolescents also had greater LPP amplitudes compared to late adolescents ($p = 0.025$). These findings reveal that LPP amplitudes decreased from preadolescence to late adolescence in the reward block.

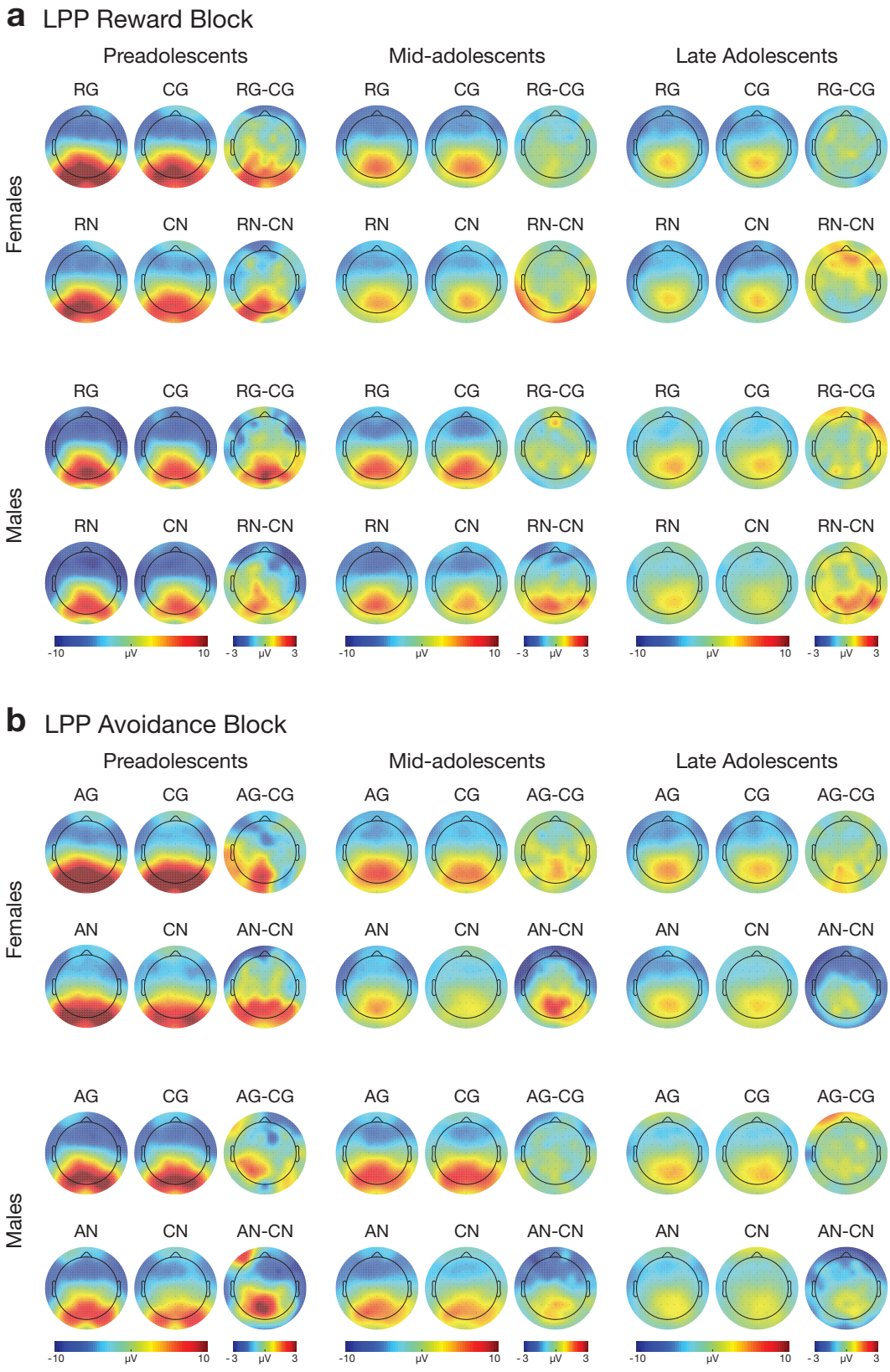

Figure 4.8 Scalp topography of the LPP (400-700 ms) for the reward block (**a**) and avoidance block (**b**). Topographical maps are shown for the S^D and control stimuli, as well as for the differences in activity between S^D and their respective control condition. RG = Reward^{Go}; RN = Reward^{NoGo}; AG = Avoidance^{Go}; AN = Avoidance^{NoGo}; CG = Control^{Go}; CN = Control^{NoGo}.

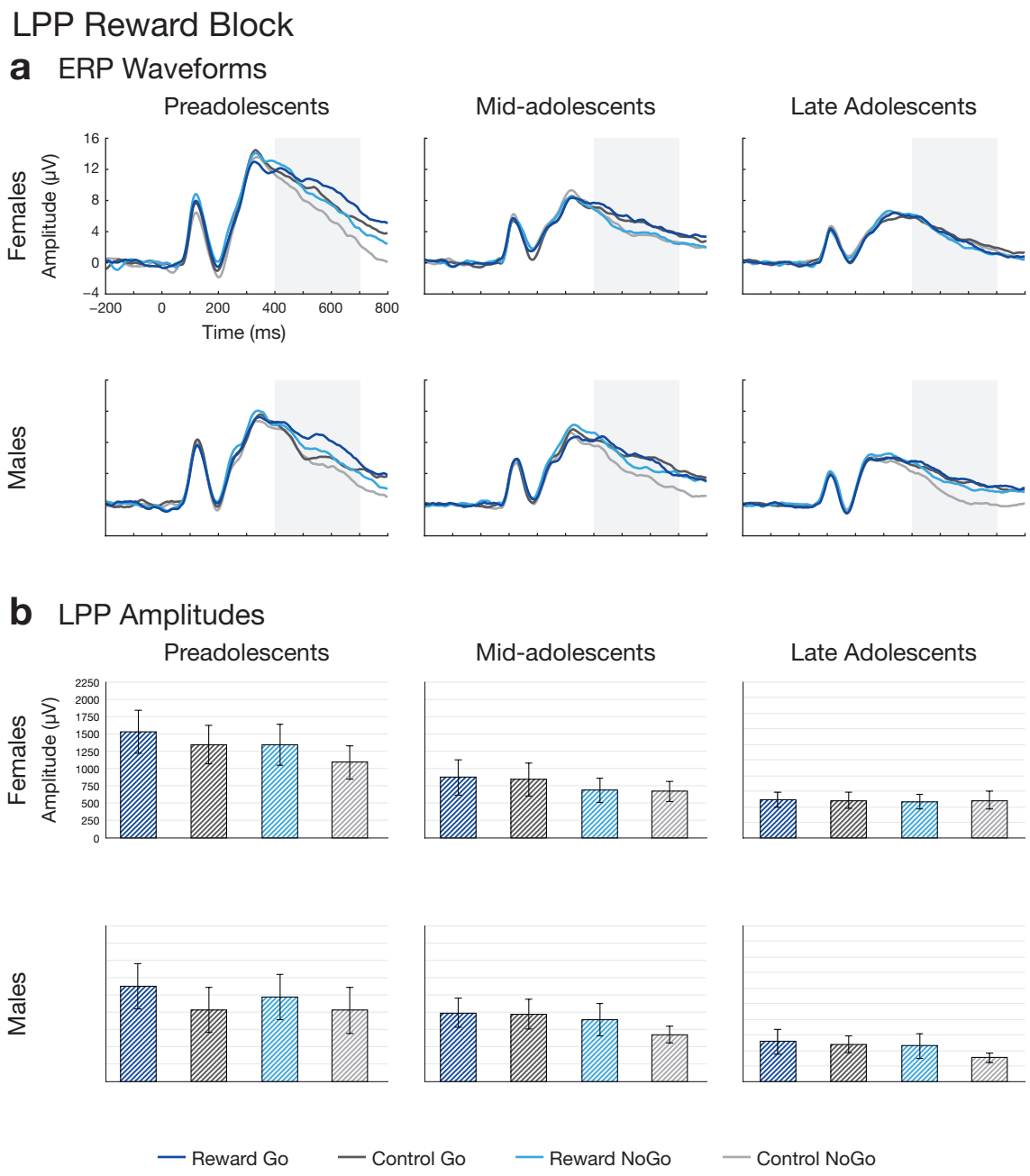

Figure 4.9 Grand averaged ERP waveforms (**a**) and LPP amplitude group means (**b**) during the reward block at the centroparietal electrode cluster. The LPP was identified between 400-700 ms post stimulus onset, and is illustrated by the grey area on the ERP waveforms. Rectified area under the curve was used to quantify LPP amplitudes. Error bars represent 95% bootstrapped confidence intervals.

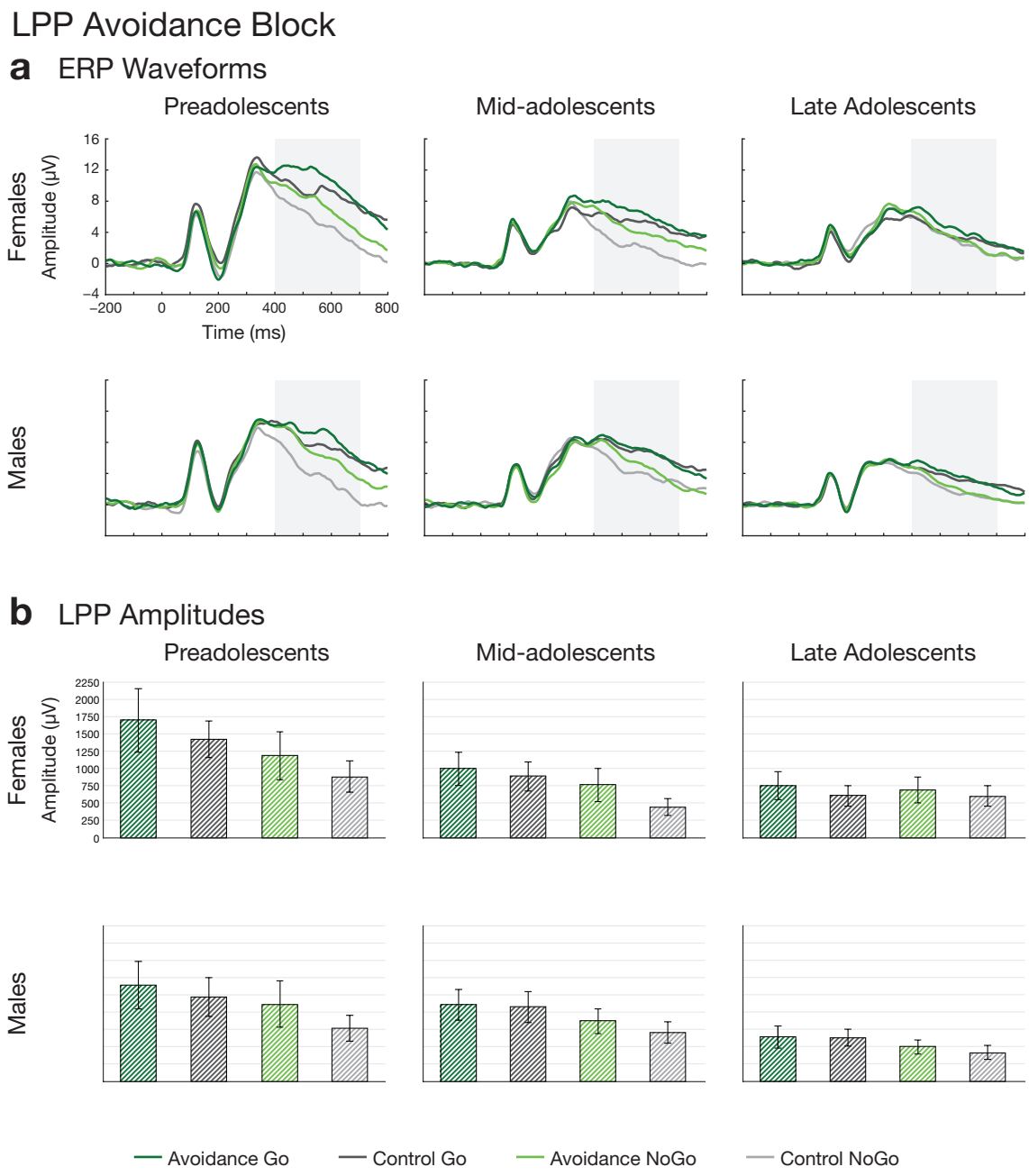

Figure 4.10 Grand averaged ERP waveforms (**a**) and LPP amplitude group means (**b**) during the avoidance block at the centroparietal electrode cluster. The LPP was identified between 400-700 ms post stimulus onset, and is illustrated by the grey area on the ERP waveforms. Rectified area under the curve was used to quantify LPP amplitudes. Error bars represent 95% bootstrapped confidence intervals.

The *Condition* by *Group* interaction for the reward block almost reached significance ($F(2, 88) = 2.02$, $p = 0.064$, $\eta_p^2 = 0.04$). To further examine this interaction, a repeated measures ANOVA was conducted for each age group with *Condition* as the within-group factor. A main effect of *Condition* was found for preadolescents ($F(3, 87) = 6.57$, $p < 0.001$, $\eta_p^2 = 0.19$) and mid-adolescents ($F(3, 87) = 6.39$, $p = 0.001$, $\eta_p^2 = 0.18$), but not for late adolescents ($F(2.04, 67.31) = 2.09$, $p = 0.131$, $\eta_p^2 = 0.01$). Pairwise comparisons revealed that preadolescents had greater LPP amplitudes for Reward^{Go} cues ($M = 1453.36$, 95% CI [1199.51, 1707.21]) compared to Control^{Go} cues ($M = 1193.10$, 95% CI [949.03, 1437.17]) ($p = 0.007$), and for Reward^{NoGo} cues ($M = 1285.50$, 95% CI [1048.94, 1522.06]) compared to Control^{NoGo} cues ($M = 1061.82$, 95% CI [823.73, 1299.92]) ($p = 0.010$). In comparison, mid-adolescents only had greater LPP amplitudes for Reward^{NoGo} cues ($M = 791.59$, 95% CI [610.78, 972.39]) compared to Control^{NoGo} cues ($M = 677.79$, 95% CI [559.50, 796.07]) ($p = 0.031$); no difference was found between Reward^{Go} ($M = 932.08$, 95% CI [729.32, 1134.85]) and Control^{Go} cues ($M = 911.59$, 95% CI [724.40, 1098.78]) for mid-adolescents ($p = 0.725$). All other main effects and interactions for the reward block were non-significant.

For the avoidance block, a main effect of *Condition* was found ($F(3, 264) = 48.28$, $p < 0.001$, $\eta_p^2 = 0.35$). Pairwise comparisons showed that LPP amplitudes were greater for Avoidance^{Go} cues ($M = 1100.36$, 95% CI [967.53, 1233.19]) compared to Control^{Go} cues ($M = 974.42$, 95% CI [876.31, 1072.52]) ($p = 0.004$), and for Avoidance^{NoGo} cues ($M = 855.93$, 95% CI [736.01, 975.85]) compared to Control^{NoGo} cues (M = 637.63, 95% CI [557.60, 717.66]) ($p < 0.001$). Furthermore, Avoidance^{Go} cues had significantly larger LPP amplitudes than Avoidance^{NoGo} ($p < 0.001$), and Control^{Go} cues had significantly larger LPP amplitudes than Control^{NoGo} cues ($p < 0.001$), showing that cues requiring a motor response had significantly larger LPP amplitudes than cues requiring the inhibition of a motor response. A main effect of *Age Group* for the avoidance block was also found ($F(2, 88) = 12.86$, $p < 0.001$, $\eta_p^2 = 0.23$). Pairwise comparisons revealed that preadolescents had greater LPP amplitudes ($M = 1211.78$, 95% CI [1038.40, 1385.16]) compared to mid-adolescents ($M = 859.17$, 95% CI [685.80, 1032.55]) ($p = 0.005$) and late adolescents ($M = 605.30$, 95% CI [442.16, 768.44]) ($p < 0.001$). Mid-adolescents also had greater LPP amplitudes compared to late adolescents ($p = 0.037$). These findings are consistent with the reward block, and show that LPP amplitudes decreased from preadolescence to late adolescence.

The main effects of *Condition* and *Age Group* for the avoidance block were

qualified by a *Condition* by *Age Group* interaction ($F(6, 264) = 5.75$, $p < 0.001$, $\eta_p^2 = 0.12$). To further examine this interaction, a repeated measures ANOVA was conducted for each age group with *Condition* as the within-group factor. A main effect of *Condition* was found for preadolescents ($F(3, 87) = 22.28$, $p < 0.001$, $\eta_p^2 = 0.44$), mid-adolescents ($F(3, 87) = 22.28$, $p < 0.001$, $\eta_p^2 = 0.44$) and late adolescents ($F(3, 99) = 4.95$, $p = 0.003$, $\eta_p^2 = 0.13$). Pairwise comparisons revealed that preadolescents had greater LPP amplitudes for Avoidance^{Go} cues ($M = 1547.61$, 95% CI [1207.03, 1888.20]) compared to Control^{Go} cues ($M = 1320.16$, 95% CI [1089.37, 1550.94]) ($p = 0.034$), and for Avoidance^{NoGo} cues ($M = 1153.44$, 95% CI [847.23, 1459.65]) compared to Control^{NoGo} cues ($M = 825.91$, 95% CI [628.77, 1023.04]) ($p = 0.001$). By comparison, mid-adolescents and late adolescents only had greater LPP amplitudes for Avoidance^{NoGo} cues (mid-adolescents: $M = 820.41$, 95% CI [651.57, 989.25]; late adolescents: $M = 599.54$, 95% CI [463.94, 735.13]) compared to Control^{NoGo} cues (mid-adolescents: $M = 577.90$, 95% CI [454.26, 701.53]; late adolescents: $M = 514.57$, 95% CI [409.45, 619.69]) (mid-adolescents: $p < 0.001$; late adolescents: $p = 0.046$). In contrast to preadolescents, no differences in LPP amplitude were found between Avoidance^{Go} (mid-adolescents: $M = 1054.63$, 95% CI [870.07, 1239.18]; late adolescents: $M = 702.29$, 95% CI [547.64, 856.93]) and Control^{Go} cues (mid-adolescents: $M = 983.76$, 95% CI [808.84, 1158.68]; late adolescents: $M = 618.69$, 95% CI [509.86, 727.52]) for either mid-adolescents ($p = 0.208$) or late adolescents ($p = 0.137$). All other main effects and interactions for the avoidance block were non-significant.

In summary, no gender differences were observed for LPP amplitudes. However, significant age effects were found in both the reward and avoidance blocks, with LPP amplitudes decreasing from preadolescence to late adolescence. Main effects of *Condition* were also observed in both the reward and avoidance blocks, revealing that S^D had significantly greater LPP amplitudes compared to their corresponding control stimulus (Reward^{Go} > Control^{Go}, Reward^{NoGo} > Control^{NoGo}, Avoidance^{Go} > Control^{Go}, Avoidance^{NoGo} > Control^{NoGo}). Furthermore, in both the reward and avoidance blocks, conditions that required a motor response had larger LPP amplitudes than conditions that required inhibition of a motor response (Reward^{Go} > Reward^{NoGo}, Avoidance^{Go} > Avoidance^{NoGo}, Control^{Go} > Control^{NoGo}). For the avoidance block, a *Condition* by *Age Group* interaction revealed that LPP amplitudes for preadolescents were potentiated to S^D that required emitting an action to avoid losing points, as well as to S^D that required omitting an action to avoid losing points. By comparison, LPP

amplitudes for mid-adolescents and late adolescents were only potentiated to the S^D that required omitting an action to avoid losing points. For the reward block, there was a trend towards a *Condition* by *Age Group* interaction. This interaction indicated that LPP potentiation for the reward block followed the same pattern as the avoidance block for preadolescents and mid-adolescents. However, LPP amplitudes were not potentiated for late adolescents in the reward block (see Figure 4.11 for a summary).

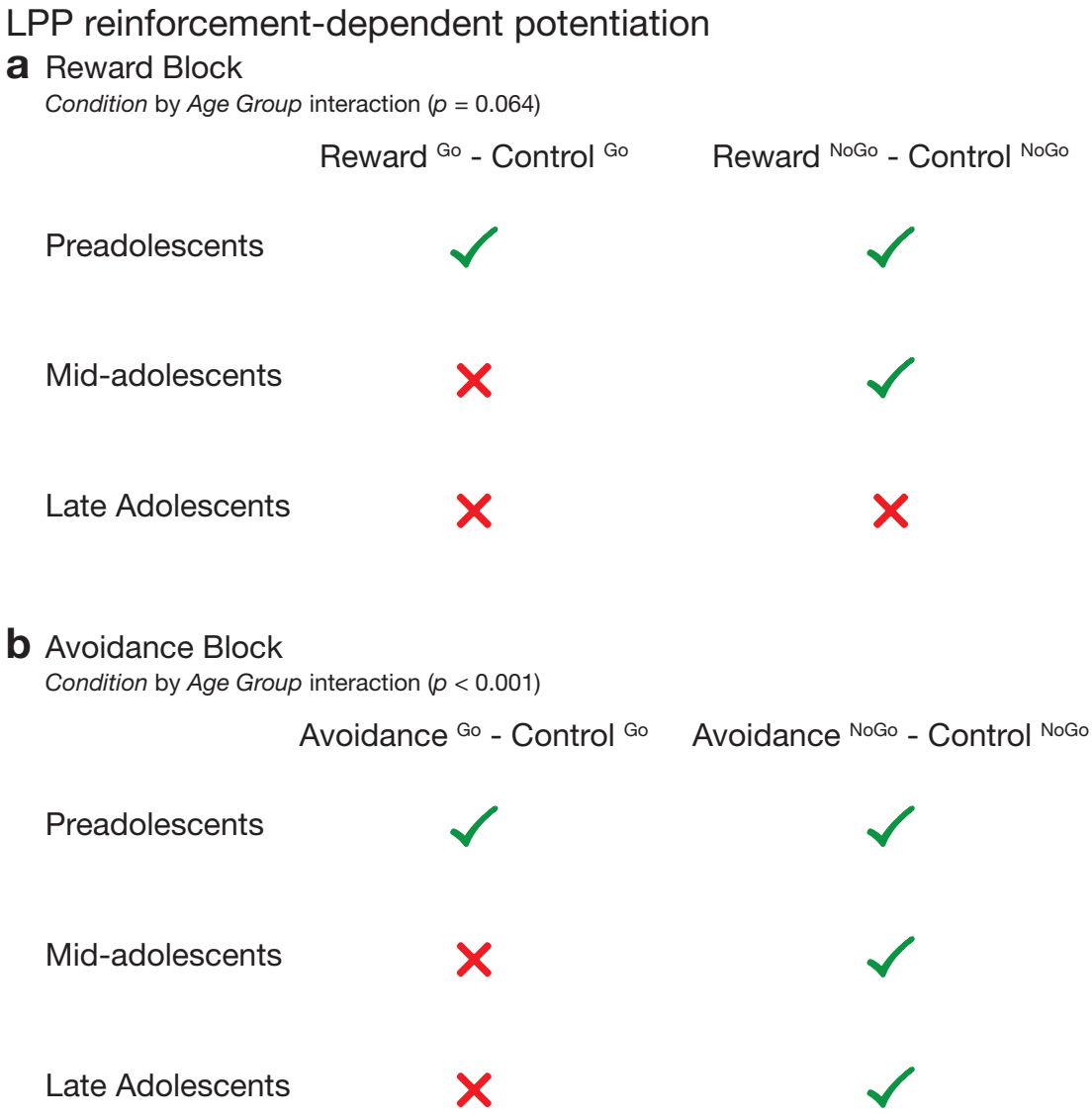

Figure 4.11 Summary of the *Condition* by *Age Group* interactions for the reward block (**a**) and avoidance block (**b**).

4.3.4. Relationships between LPP potentiation and behavioural measures

The second and third aims of this study were to explore the relationships between ERP potentiation and behavioural measures. Since the N170 was not potentiated to S^D that predicted rewarding or threatening outcomes, the following analyses were limited to the LPP. In order to isolate LPP activity that was unique to the S^D, difference waves were computed by subtracting control conditions from their corresponding S^D ($Reward^{Go}$-$Control^{Go}$, $Reward^{NoGo}$-$Control^{NoGo}$, $Avoidance^{Go}$-$Control^{Go}$, $Avoidance^{NoGo}$-$Control^{NoGo}$). Pearson correlation coefficients were used to explore the relationships between LPP difference waves and behavioural measures. To explore potential age- and gender-related effects, correlations were conducted separately for age groups and genders. Correlations were bootstrapped and corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5).

4.3.4.1. Relationships between LPP potentiation and task reaction time

The second aim of this study was to examine the functional significance of ERP potentiation to S^D. The S^D were visual cues that signalled the onset of rewarding or threatening outcomes that were conditional on participants making the correct motor responses. Given that actions are guided by the anticipation of outcomes (Elsner & Hommel, 2001), it is possible that the magnitude of participants' anticipatory responses to S^D would be associated with how quickly they responded to target stimuli. To examine this idea, the functional significance of the LPP was examined by assessing the corresponding relationships between participants' reaction times and LPP potentiation for conditions that required a motor response. Specifically, LPP $Reward^{Go}$ difference waves were correlated with reaction times to $Reward^{Go}$ and $Control^{Go}$ target stimuli. Similarly, LPP $Avoidance^{Go}$ difference waves were correlated with reaction times to $Avoidance^{Go}$ and $Control^{Go}$ target stimuli. Contrary to predictions, no relationships between LPP potentiation and reaction time were found for the reward or avoidance blocks. The Pearson correlation coefficients are reported in Supplementary Table 4.2 (Appendix 2).

4.3.4.2. Relationships between LPP potentiation and measures of risk-taking and anxiety

The third aim of this study was to assess whether ERP potentiation to S^D that predicted rewarding and threatening outcomes was associated with risk-taking

behaviours and anxiety levels, respectively. Thus, the relationships between LPP potentiation to S^D predicting a rewarding outcome and risk-taking behaviours (BART Pumps, BART Points, BART Balloons, YRBSS), as well as the relationships between LPP potentiation to S^D predicting a threatening outcome and anxiety levels (STAI-T and STAI-T controlling for depression), were explored. Contrary to the predictions, no relationships were found for any group between LPP difference waves associated with rewarding outcomes and risk-taking behaviours. Similarly, no relationships were found for any group between LPP difference waves associated with threatening outcomes and anxiety levels. The Pearson correlation coefficients are reported in Supplementary Table 4.3 (Appendix 2).

4.3.5. The influence of puberty on N170 and LPP potentiation

The analyses so far have provided insights into the development of the N170 and LPP in response to S^D and control stimuli throughout adolescence, and the relationships between LPP potentiation and measures of reaction time, risk-taking, and anxiety. Notably, puberty is thought to contribute to the age-related changes in reward- and threat-related activity during adolescence. Accordingly, the final aim of this study was to examine whether the degree of ERP potentiation was associated with pubertal stage. Since there are large individual differences in the age of pubertal onset (Marshall & Tanner, 1969; Marshall & Tanner, 1970), it is possible that categorising participants into age groups may have confounded the developmental trajectories of the N170 and LPP. Moreover, it is possible that examining the development of the N170 as a function of puberty rather than age will reveal developmental differences in N170 potentiation. To these ends, the relationships between pubertal stage and potentiation of the LPP and N170 were examined in a continuous sample of females (n = 30, M_{age} = 12.73, SD_{age} = 2.42) and males (n = 30, M_{age} = 12.67, SD_{age} = 2.55) aged 9-17 years.

The same approach was used in this study as in Chapter 3. Since several studies have reported that the developmental trajectories of reward- and threat-related neural activity are non-linear (Braams *et al.*, 2015; Hare *et al.*, 2008), hierarchical polynomial regression analyses were conducted to assess the linear and quadratic relationships between puberty and potentiation of the N170 and LPP. To tease out the confounding affects of chronological age on pubertal development, age was entered into the first block of the regression analyses as a control variable, and PDS scores were entered into

the second block. Separate regression analyses were conducted to assess the linear and quadratic relationships; raw age and PDS scores were used to examine the linear relationships, and squared age and PDS scores were used to assess the quadratic relationships. The regression coefficients were bootstrapped and corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5).

Contrary to predictions, the regression analyses revealed that pubertal stage did not predict N170 or LPP potentiation in either male or female adolescents aged 9-17 years. These findings suggest that the N170 was not modulated in this task, and that the developmental differences in reward- and threat-related LPP potentiation resulted from changes in age rather than pubertal stage. The intercorrelations for the N170 and LPP are reported in Supplementary Table 4.4 (Appendix 2). The regression coefficients for the N170 and LPP are reported in Supplementary Tables 4.5 and 4.6, respectively (Appendix 2).

4.4. Discussion

This study aimed to examine age- and gender-related differences in anticipatory representations to rewards and threats as measured by early occipitotemporal (N170) and late centroparietal (LPP) ERPs to S^D that predicted rewarding or threatening outcomes. This study stemmed from our previous work (Levita *et al*., 2014), and was designed to address the following questions: (1) how does enhanced potentiation to S^D that predict threatening outcomes fit with the neurobiological models suggesting that adolescents have a hyporesponsive avoidance system (Ernst *et al*., 2006; Ernst *et al*., 2011)?; (2) do adolescents also show enhanced potentiation to S^D that predict rewarding outcomes, and if so, how does the relationship between reward- and threat-related potentiation change during the course of adolescence?; and (3) are there gender differences in potentiation to S^D that predict rewarding and threatening outcomes that can help explain why males are more likely to engage in risk-taking behaviours and females are more likely to experience greater anxiety levels (Byrnes *et al*., 1999; Lewinsohn *et al*., 1998)? To these ends, preadolescents (9-12 years), mid-adolescents (13-17 years), and late adolescents (18-23 years) completed an instrumental conditioning task while having their brain activity measured using EEG.

Contrary to the study predictions and our previous findings (Levita *et al*., 2014),

the N170 was not potentiated to S^D that predicted rewarding outcomes or to S^D that predicted threating outcomes for any age group. In contrast, LPP amplitudes were potentiated to S^D that predicted rewarding outcomes and to S^D that predicted threatening outcomes relative to the control stimuli. Critically, the magnitude of LPP potentiation to the S^D was found to change across the three stages of adolescence, and was different in response to stimuli that required a motor response compared to stimuli that required inhibition of a motor response. In contrast to the study predictions, LPP potentiation to S^D predicting rewarding and threatening outcomes was not modulated by pubertal stage, task reaction time, risk-taking behaviours, or anxiety levels for any group.

4.4.1. LPP

An instrumental task was used to examine anticipatory neural activity to visual cues predicting rewarding and threatening outcomes. As predicted, and in line with previous aversive classical conditioning studies (Pastor *et al*., 2015; Pizzagalli *et al*., 2003), LPP amplitudes were potentiated in centroparietal areas for S^D that predicted a threatening outcome compared to control stimuli. Notably, LPP amplitudes were also potentiated in centroparietal areas for S^D that predicted a rewarding outcome compared to control stimuli. Together, these findings support previous developmental and adult studies showing that LPP amplitudes are greater in response to primary reinforcers, such as pleasant and unpleasant pictures, compared to neutral stimuli, in passive picture viewing tasks (e.g., Cuthbert *et al*., 2000; Hajcak & Dennis, 2009).

Critically, the reinforcement-dependent potentiation of the LPP changed during the course of adolescence. Specifically, preadolescents showed LPP potentiation to the S^D in both the reward and avoidance blocks, regardless of whether they had to emit or omit a motor action. By comparison, mid-adolescents showed LPP potentiation to the S^D in both the reward and avoidance blocks, but only to S^D that required inhibition of a motor action. In contrast, late adolescents only showed LPP potentiation to the S^D in the avoidance block, and only for S^D that required inhibition of a motor action. Thus, in contrast to both preadolescents and mid-adolescents, late adolescents only showed LPP potentiation to the S^D when they were faced with the threat of losing the points they had earned in the preceding reward block.

Previous studies have reported that the salience of primary and secondary reinforcers, including appetitive and aversive tastes (Galván & McGlennen, 2013) and

money (Barkley-Levenson & Galván, 2014), decreases from mid-adolescence to late adolescence. It is therefore feasible that the salience of the reinforcer contributed to the age-related differences in LPP potentiation found in the current study, whereby the salience of the reinforcer decreased from preadolescence to late adolescence. Thus, the reinforcer used in the current study may have had different motivational affects on preadolescents, mid-adolescents and late adolescents, which may have, in turn, influenced the strength of instrumental conditioning, and consequently the magnitude of LPP amplitudes in response to the S^D.

Developmental differences in task performance may have also contributed to the age-related changes in LPP potentiation; preadolescents made significantly more errors during the reward and avoidance blocks and had significantly more LPP potentiation to S^D associated with rewarding and threatening outcomes compared to mid-adolescents and late adolescents. Notably, preadolescents made the most errors in response to $Control^{Go}$ stimuli in the reward and avoidance blocks, but their LPP amplitudes for the $Reward^{Go}$ and $Avoidance^{Go}$ S^D were greater than their LPP amplitudes for $Control^{Go}$ stimuli. These findings suggest that, in preadolescents, the magnitude of the LPP did not directly reflect task performance. Furthermore, task performance was equivalent for mid-adolescents and late adolescents. However, mid-adolescents, but not late adolescents, showed reward-related LPP potentiation. While age-related differences in task performance may have contributed to LPP amplitudes, the findings point towards LPP potentiation being predominantly modulated by the rewarding and threatening outcomes associated with the S^D.

It is important to note that, in contrast to the study predictions, LPP potentiation did not peak in middle adolescence. Previous fMRI studies that have reported a peak in reward- and threat-related anticipatory activity in mid-adolescents have either used primary reinforcers such as appetitive and aversive tastes (Galván & McGlennen, 2013) or secondary reinforcers such as money (Braams *et al*., 2015). In this task, points, rather than money, were used as the secondary reinforcer since money may have a different meaning for younger individuals compared to older individuals (Barkley-Levenson & Galván, 2014). Therefore, the discrepancy between this and previous studies could result from the current study using points as the reinforcer. It is possible that more ecologically valid secondary reinforcers, such as money, or primary reinforcers that have high evolutionary significance are needed to elicit the peak in neural activity during mid-adolescence.

While no differences in the pattern of activation were found between the reward and avoidance blocks for preadolescents and mid-adolescents, LPP potentiation in late adolescents was observed only in response to the S^D that predicted a potential threat, but not a potential reward. Interpreted in light of the theory of loss aversion (Tversky & Kahneman, 1992), this finding suggests that the threat of a loss may have been more salient than acquiring a reward during late adolescence. Crucially, these findings do not support the idea that adolescent behaviour is driven by hyperresponsive approach system and hyporesponsive avoidance system (Ernst *et al*., 2006; Ernst *et al*., 2011). Instead, these findings support Casey's dual systems model (Casey *et al*., 2010), and suggest that preadolescents and mid-adolescents are motivated by both rewards and threats. Moreover, these findings suggest that late adolescents are more motivated by threats than rewards.

Biases in anticipatory processes to rewards and threats are thought to contribute to high levels of risk-taking behaviours and anxiety, respectively (Petry, 2001; Salkovskis, 1991). Thus, the relationships between LPP potentiation to S^D that predicted rewarding outcomes and participants' risk-taking behaviours, and the relationships between LPP potentiation to S^D that predicted threating outcomes and participants' anxiety levels, were explored in this sample of adolescents. Unexpectedly, no relationships were found between adolescents' risk-taking behaviours and LPP potentiation to S^D that predicted a rewarding outcome. Similarly, no relationships were found between adolescents' anxiety levels and LPP potentiation to S^D that predicted a threatening outcome. These findings are in contrast to fMRI studies showing that subcortical neural activity associated with reward and threat anticipation is positively correlated with risk-taking behaviours and anxiety levels, respectively (Barkley-Levenson & Galván, 2014; Braams *et al*., 2015; Galván *et al*., 2007), as well as theories implicating reward anticipation in risk-taking and threat anticipation in anxiety (Petry, 2001; Salkovskis, 1991). As discussed earlier, it is possible that winning and losing points was not salient enough or of high enough evolutionary significance to tap into the mechanisms underlying risk-taking behaviours and anxiety levels during adolescence.

In the current study, stimuli requiring a motor response evoked larger LPP amplitudes compared to stimuli requiring inhibition of a motor response in all groups. Since the LPP is thought to reflect selective attention to motivationally salient stimuli (Cuthbert *et al*., 2000; Schupp *et al*., 2000; Schupp *et al*., 2004), it is possible that

conditions requiring a motor response elicited greater attentional resources in preparation for motor action. To examine whether LPP potentiation was functionally implicated in preparing for action selection, the relationships between LPP amplitudes and reaction times for Go conditions were explored. Contrary to expectations, no relationships between LPP amplitudes and reaction times were found. It is possible that the design of the task precluded us from seeing an effect, as there was a delay between when the visual cues were presented and when participants were required to make a motor response. It has been suggested that the LPP reflects global inhibition of neural activity in the visual cortex, which in turn, facilitates more selective processing of the salient stimulus (Brown, van Steenbergen, Band, de Roover & Nieuwenhuis, 2012). Hence, in this study, the stimuli requiring a motor response could be evoking a greater attentional bias compared to stimuli requiring inhibition of a motor response. How and whether this attentional bias can affect subsequent decision making mechanisms and action selection is yet to be determined. Further work is also needed to determine why LPP potentiation was found in response to S^D requiring a motor response and inhibition of a motor response in preadolescents, but only to S^D requiring inhibition of a motor response in mid- and late adolescents.

Consistent with previous developmental studies (e.g., Kujawa *et al*., 2012; MacNamara *et al*., 2016), LPP amplitudes were found to decrease from preadolescence to late adolescence for S^D and control stimuli. The scalp maps also revealed that LPP topography changed with age, whereby LPP activation shifted from occipitoparietal to centroparietal regions from preadolescence to late adolescence. In addition, the scalp maps showed that LPP activity became less bilateral and more focal with age. These age-related changes in LPP amplitude and topography are in accordance with previous findings (Dennis & Hajcak, 2009; Hajcak & Dennis, 2009; Kujawa *et al*., 2012; Kujawa *et al*., 2013; Solomon *et al*., 2012), and are thought to reflect brain maturation during adolescence. The human brain matures in a back-to-front fashion, with higher order prefrontal regions developing last (Gotgay *et al*., 2004; Mills *et al*., 2014). As such, compared to other cortical and subcortical regions, the PFC undergoes significant changes throughout childhood and adolescence (see Chapter 1.4). The maturation of the PFC during adolescence is thought to underlie considerable improvements in cognitive control and information processing (Luna, Garver, Urban, Lazar & Sweeney, 2004; Yurgelun-Todd, 2007). Thus, the reduction in LPP amplitude across adolescence may reflect more efficient processing as the brain matures. This is consistent with the

behavioural findings in this study showing that preadolescents made significantly more errors on the task compared to mid-adolescents and late adolescents. Given that both top-down prefrontal areas and bottom-up occipitoparietal areas modulate the LPP (Moratti *et al*., 2011), it is possible that the changes in LPP topography observed in this and other studies (Hajcak & Dennis, 2009; Kujawa *et al*., 2012; Kujawa *et al*., 2013) are the result of the LPP becoming more reliant on prefrontal regions as the PFC matures.

4.4.2. N170

While the LPP was used to examine late anticipatory representations to S^D that predicted rewarding and threating outcomes, the N170 was used to examine early anticipatory representations. The visual stimuli used in this instrumental task were greeble-like and elicited clear N170 ERPs. This is consistent with previous studies showing that greebles evoke the N170 component (Levita *et al*., 2014; Rossion, Gauthier, Goffaux, Tarr & Crommelinck, 2002). However, in contrast to the study predictions, no differences in N170 amplitude were found between S^D and control stimuli for any age group. This finding initially appears inconsistent with previous studies showing early modulation within ventral visual pathways to motivationally salient stimuli (Dolan *et al*., 2006; Levita *et al*., 2014; Pizzagalli *et al*., 2003). One plausible explanation for the discrepancy in findings is that a secondary rather than a primary reinforcer was used in the current study. Previous classical and instrumental conditioning tasks that have shown modulation of the N170 to conditioned stimuli have used primary reinforcers where the threat was imminent (Dolan *et al*., 2006; Levita *et al*., 2014; Pizzagalli *et al*., 2003). In the current study, the S^D predicted the delayed onset of a secondary reinforcer (points) that was conditional on specific motor responses. It is therefore possible that stimuli that have high evolutionary significance, i.e., primary reinforcers, are needed to engage very early attentional mechanisms. In order to disentangle neural activity associated with anticipatory processes from neural activity associated with motor responses, participants were required to wait for a target stimulus (yellow cross) before making their response. Hence, the type of reinforcer used (primary *vs* secondary), as well as the time between the stimulus and response (immediate *vs* delayed), may account for the discrepancies observed between this and previous studies (Dolan *et al*., 2006; Levita *et al*., 2014; Pizzagalli *et al*., 2003). Further work is needed to identify the task contingencies and

reinforcers that elicit reinforcement-dependent potentiation in early visual processing areas during adolescence, as well as to identify the functional significance of such potentiation.

Despite not finding reinforcement-dependent potentiation of the N170, this study found age-related changes in N170 amplitude that were independent of condition. In line with previous studies (Hileman *et al*., 2011; Taylor *et al*., 1999) and the study predictions, the N170 decreased in amplitude from preadolescence to mid-adolescence. Reductions in N170 amplitude during childhood and adolescence are thought to result from increases in white matter and decreases in grey matter (see Chapter 1.4). While the scalp topographical plots indicated an additional reduction in N170 amplitude from mid- to late adolescence, this effect was not statistically significant.

4.4.3. The influence of puberty on N170 and LPP potentiation

The final aim of the current study was to explore the linear and non-linear relationships between puberty and ERP potentiation in male and female adolescents aged 9-17 years while controlling for chronological age. Contrary to predictions, current pubertal stage was not associated with N170 or LPP potentiation. This was surprising since puberty has been shown to have significant affects on reward- and threat-related neural activity (Braams *et al*., 2015; Moore *et al*., 2012). Despite this, the finding that LPP amplitudes were not related to current pubertal stage is consistent with previous findings (Nelson *et al*., 2015); Nelson *et al*. (2015) found no evidence for LPP amplitudes in response to affective pictures being modulated by pubertal stage in 13.5-15.5 year old females. The current study therefore extends Nelson *et al*.'s (2015) findings by examining LPP amplitudes to conditioned stimuli in male and female adolescents aged 9-17 years. Collectively, the evidence to date suggests that pubertal stage is not associated with LPP or N170 amplitudes during adolescence. Nevertheless, future studies should continue to explore potential relationships between ERPs and puberty in order to establish whether there are any circumstances under which puberty influences reward- and threat-related ERP activity.

4.4.4. Study limitations

The current study has a number of limitations that need highlighting. First, the *Condition* by *Age Group* interaction effect for the reward block just failed to reach

significance. Moreover, the LPP waveforms and bar graphs (Figure 4.9) indicated that males were largely driving the overall reward-related LPP potentiation effects for the mid-adolescents. Despite this, no gender differences emerged from the statistical analyses. Together, these findings suggest that there was high variability in this sample of adolescents and not enough power to detect potentially interesting effects. Future work should therefore aim to explore the development of reward- and threat-related potentiation in larger samples of adolescents. Second, due to time constraints, participants did not receive feedback for control stimuli. Consequently, this study was unable to explore reward- and threat-related consummatory processes, and compare anticipatory and consummatory responses during the different stages of adolescence.

4.4.5. Conclusion

Research has repeatedly shown that adolescents are highly motivated by rewards (Casey *et al.*, 2010; Ernst *et al.*, 2006; Nelson *et al.*, 2005; Steinberg, 2008). However, research examining the extent to which adolescents are motivated by avoiding potential threats is limited. The findings reported here suggest that preadolescents and mid-adolescents are motivated by both obtaining rewards and avoiding loss. The current study therefore provides initial evidence that anticipatory biases are at least as strong for avoidance-related cues as they are for reward-related cues during early and middle adolescence. Hence, these findings do not support the suggestion that adolescents are hyposensitive to threat (Ernst *et al.*, 2006). While further work is required to examine the functional significance of these findings, the results suggest that future studies should not only focus on sensitivity to reward during adolescence, but also on sensitivity to threat.

It has been suggested that frontal cortical asymmetry, as indexed by resting EEG alpha activity, reflects the lateralisation of approach-avoidance processes (Davidson, 1984, 1992). In support of this idea, many studies have reported that frontal cortical asymmetry is a marker of risk-taking behaviours and anxiety levels in adults (Coan & Allen, 2003; Santesso *et al.*, 2008; Sutton & Davidson, 1997; Thibodeau *et al.*, 2006; Wheeler *et al.*, 1993). However, whether the same is true for adolescents is yet to be determined. Given that the first study in this doctoral work found age-related increases in risk-taking, but not anxiety, the final study reported in this doctoral work (Chapter 5) examined the development of frontal asymmetry, and its relationship to measures of risk-taking behaviours. Chapter 5 also explored how the cortical sources

of resting alpha change as a function of age, gender, and puberty during the course of adolescence.

Chapter 5

Cortical sources of spontaneous alpha during adolescence: Relationships with puberty and risk-taking

Abstract

This study aimed to investigate how the cortical sources of spontaneous alpha during eyes-open and eyes-closed conditions change during the course of adolescence as a function of age and pubertal stage. Notably, there is a long-standing theory asserting that relatively greater left frontal cortical activity is associated with reward-orientated behaviours and relatively greater right frontal neural activity is associated with avoidance-orientated behaviours, as indexed by spontaneous EEG alpha activity (Davidson, 1984, 1994). While there is considerable evidence for this theory in adults, research examining frontal asymmetry and its relationship to risk-taking and anxiety in adolescents is limited. The first study in this doctoral work found that risk-taking behaviours, but not anxiety levels, increased throughout adolescence in this sample (Chapter 3). Hence, the final aim of this study was to examine whether frontal asymmetry could account for the developmental differences in risk-taking behaviours found in this sample of adolescents. To these ends, 29 preadolescents (9-12 years; 14 females), 29 mid-adolescents (13-17 years; 14 females), and 33 late adolescents (18-23 years; 17 females) had their resting brain activity measured using EEG during eyes-open and eyes-closed conditions. Overall, the findings revealed that the cortical sources of alpha changed considerably during the course of adolescence, and that more advanced pubertal development predicted reduced alpha activity in male, but not female, adolescents. Unexpectedly, frontal asymmetry was found to not be a reliable marker of risk-taking behaviours in this sample of adolescents.

5.1. Introduction

This study had three aims. Firstly, this study aimed to investigate the age- and gender-related changes in the cortical sources of spontaneous alpha in adolescents aged 9-23 years. Secondly, this study aimed to examine the relationship between puberty and the development of spontaneous alpha in male and female adolescents aged 9-17 years while controlling for chronological age. Finally, this study aimed to examine the functional significance of spontaneous alpha in this sample of adolescents. It has been suggested that frontal cortical asymmetry, i.e., relatively greater left or right frontal cortical activity, reflects the lateralisation of approach-avoidance processes, as indexed by spontaneous alpha (Coan & Allen, 2004; Davidson, 1984, 1992; Harmon-Jones *et al*., 2010; Tomarken *et al*., 1990; Tomarken *et al*., 1992; Wheeler *et al*., 1993). In support of this idea, many studies have reported that frontal cortical asymmetry is associated with risk-taking behaviours and anxiety levels in adults (Coan & Allen, 2003; Santesso *et al.*, 2008; Sutton & Davidson, 1997; Tomarken *et al*., 1990; Tomarken *et al*., 1992; Wheeler *et al*., 1993). However, whether the same is true for adolescents is yet to be determined. Notably, the same cohort of participants took part in this study as in Chapters 3 and 4. Since the first study in this doctoral work (Chapter 3) found that risk-taking behaviours, but not anxiety levels, increased throughout adolescence, the current study aimed to explore whether frontal asymmetry could account for the developmental differences in risk-taking behaviours found in this sample of adolescents.

The introduction to this study will first define the alpha rhythm (Chapter 5.1.1), and outline the development of spontaneous alpha from infancy to adulthood (Chapter 5.1.2). This introduction will then discuss what is currently known about functional significance of spontaneous alpha (Chapter 5.1.3) and the role of spontaneous alpha in the approach-avoidance systems (Chapter 5.1.4). Next, this introduction will outline and evaluate the human and nonhuman animal work investigating the cortical sources of alpha (Chapter 5.1.5), and discuss why examining puberty in relation to the development of alpha warrants investigation (Chapter 5.1.6). Finally, this introduction will outline the aims and hypotheses of the current study (Chapter 5.1.7).

5.1.1. The alpha rhythm

Hans Berger first described the EEG alpha rhythm in 1929. Berger observed that

the alpha rhythm oscillated between 8-13 Hz and had maximal amplitude over occipital regions when individuals were awake and resting with their eyes closed. Amplitude indexes the magnitude of an oscillation, and refers to the vertical displacement between the peak of an oscillation and its rest position. Amplitude is measured in μV and can be squared to yield a power value (μV^2). It has been reported that alpha amplitude (or power) is inversely related to cortical activity, whereby increases in alpha amplitude reflect decreases in cortical activity, and decreases in alpha amplitude reflect increases in cortical activity (Haegens *et al.,* 2011; Shagass, 1972).

While amplitude refers to the magnitude of an oscillation, frequency refers to the number of oscillations per second. The alpha rhythm has a frequency range of 8-13 Hz and a peak frequency of 10 Hz in healthy adults (Klimesch, 1999). Peak alpha frequency reflects the maximal power value within the alpha frequency band. Individual alpha peak frequency can also be measured, and reflects the centre of gravity, rather than the peak, within the alpha frequency band (Klimesch, 1999). The amplitude and frequency of the alpha rhythm can be measured either at rest (spontaneous alpha) or in response to specific events (event-related alpha). Spontaneous alpha reflects the endogenous fluctuations of cortical activity within the alpha frequency band, and is recorded while participants are awake and resting with their eyes open or closed. Compared to spontaneous alpha, event-related alpha responses are evoked by a sensory or cognitive event. Event-related alpha is frequently measured during short (~1 second) resting periods that occur before the onset of a trial or between the presentation of a warning stimulus and a test period (Başar, 2012; Klimesch, 1999). Both amplitude and frequency measures have been extensively used to establish how spontaneous alpha develops throughout the lifespan, as well as to examine the functional significance of spontaneous and event-related alpha (Klimesch, 1999).

Notably, spontaneous alpha recorded from frontal scalp electrodes is thought to reflect the approach-avoidance systems, and has been associated with a number of approach- and avoidance-related behaviours and emotions, such as risk-taking and anxiety (Coan & Allen, 2004; Davidson, 1984, 1992; Harmon-Jones *et al*., 2010; Tomarken *et al*., 1990; Tomarken *et al*., 1992; Wheeler *et al*., 1993). Consequently, this study focused on spontaneous alpha rather than event-related alpha. Specifically, the current study aimed to investigate whether frontal alpha asymmetry could account for

the age- and gender-related changes in risk-taking behaviours found in this sample of adolescents (Chapter 3).

5.1.2. Development of spontaneous alpha

A number of studies have shown that the amplitude and frequency of spontaneous alpha develop considerably throughout childhood and adolescence (Dustman, Shearer & Emmerson, 1999; Katada, Ozaki, Suzuki & Suhara, 1981). A posterior-dominant rhythm (PDR) that resembles alpha can be observed in infants aged only a few months old (Stroganova, Orekhova & Posikera, 1999). The peak frequency of the PDR steadily increases from approximately 3 Hz in infancy to the adult frequency of 10 Hz in early adolescence (Lindsley, 1939; Marshall, Bar-Haim & Fox, 2002; Niedermeyer, 1997; Somsen, van't Klooster, van der Molen, van Leeuwen & Licht, 1997). The increase in alpha frequency during childhood and adolescence is thought to result from the augmentation of white matter (Segalowitz *et al*., 2010; Whitford *et al*., 2007; see Chapter 1.4). In addition to the increase in alpha frequency, the amplitude of the alpha rhythm decreases across all areas of the scalp during childhood and adolescence (Chiang, Rennie, Robinson, van Albada & Kerr, 2011; Dustman *et al*., 1999; Yordanova & Kolev, 1997). The EEG signal largely reflects the simultaneous firing of pyramidal neurons in cortical grey matter (Davidson *et al*., 2000). Thus, the reduction in alpha amplitude is thought to result from the decrease in cortical grey matter during adolescence (Segalowitz *et al*., 2010; Whitford *et al*., 2007; see Chapter 1.4). The reduction in alpha amplitude may also partially result from the increase in skull thickness during childhood and adolescence (Hagemann, Hewig, Walter & Naumann, 2008).

5.1.3. Function of spontaneous alpha

The functional significance of spontaneous alpha has been investigated ever since it was first discovered. It was initially thought that alpha rhythm had little functional relevance and simply reflected the idling of the brain (Adrian & Matthews, 1934; Pfurtscheller, Stancak & Neuper, 1996). However, while the functional significance of alpha is still being explored and debated, the empirical work to date unanimously suggests that alpha activity does not reflect cortical idling (Cooper, Croft, Dominey, Burgess & Gruzelier, 2003). Instead, the current evidence suggests that spontaneous alpha has an important and active role in cognitive and memory

processes (Klimesch, 1999; Palva & Palva, 2007).

Preliminary support for the role of spontaneous alpha in cognitive and memory processes comes from studies assessing how the frequency of alpha changes across the lifespan (Klimesch, 1999). The frequency of spontaneous alpha increases throughout infancy, childhood, and adolescence (Lindsley, 1939; Niedermeyer, 1997), and is thought to result from the increase in white matter during childhood and adolescence. This, in turn, is thought to underlie more efficient and faster neural processing (Klimesch, 1999), and thus improvements in cognitive functioning. After peaking in adolescence, the frequency of alpha subsequently decreases across adulthood, from approximately 10 Hz in 20 year olds to 8 Hz in 70 year olds (Dustman *et al*., 1999; Obrist, 1979). This decrease in alpha frequency has been suggested to reflect age-related reductions in memory performance and cognitive processing (Klimesch, 1999). Indeed, Li, Sun and Jiao (1996) found that the reduction in alpha frequency in individuals aged 46 to 80 years was correlated with performance on a range of executive tasks. In sum, the changes in alpha frequency throughout the lifespan are thought to correspond to age-related changes in cognitive and memory performance (Klimesch, 1999).

Empirical work in atypical populations also supports the idea that spontaneous alpha has a functional role in cognitive and memory processes. Compared to healthy controls, slower alpha frequencies and reduced or absent alpha power are observed across a number of developmental, psychiatric, and neurodegenerative conditions, such as autism spectrum disorder, schizophrenia, and Alzheimer's disease (Alfimova & Uvarova, 2008; Babiloni *et al*., 2009; Leuchter, Spar, Walter & Weiner, 1987). Overall, the evidence to date collectively suggests that spontaneous alpha is associated with cognitive and memory processes in both typical and atypical populations (Klimesch, 1999).

Alpha frequency and power have also been shown to reliably predict performance on cognitive and memory tasks in typically developing populations (e.g., Klimesch, Doppelmayr, Schimke & Ripper, 1997; Klimesch, Schimke & Pfurtscheller, 1993). Peak alpha frequency is highly variable between individuals, and this variability is thought to underlie the individual differences in performance on a variety of cognitive and memory tasks (Klimesch, 1999). In particular, individuals with higher peak alpha frequencies have faster reaction times to target stimuli and perform better on memory tasks compared to individuals with lower peak alpha frequencies (Clark *et*

al., 2004; Haegens, Cousijn, Wallis, Harrison & Nobre, 2014; Klimesch, Schimke, Ladurner & Pfurtscheller, 1990; Klimesch *et al*., 1993; Surwillo, 1963; Surwillo, 1971). Higher peak alpha frequencies have also been associated with higher reading performance in children (Suldo, Olson & Evans, 2002).

Event-related alpha studies can assess the extent to which alpha desynchronises (decreases) during a test period relative to a resting period. These studies have reported that alpha desynchronisation is greater in individuals with better task performance (Boiten, Sergeant & Geuze, 1992; Klimesch *et al.*, 1997; Van Winsun, Sergeant & Geuze, 1984). Event-related studies have also reported that alpha desynchronises in cortical areas important for task performance, but synchronises (increases) in cortical areas that are not relevant for a particular task (Klimesch *et al*., 1997). Consequently, current theories regarding the functional significance of alpha suggest that alpha has a functional role in active inhibition and the direction of attention (Foxe & Snyder, 2011; Jensen, Bonnefond & VanRullen, 2012; Klimesch, Sauseng & Hanslmayr, 2007), both of which are critical for optimal task performance (Jensen & Mazaheri, 2010).

5.1.4. Frontal alpha asymmetry

There is a biphasic motivational theory proposing that frontal cortical activity in the left and right hemispheres reflects lateralisation of the approach and avoidance systems, respectively (Davidson, 1984, 1992). Alpha amplitude (or power) is inversely related to cortical activity, whereby increases in alpha amplitude reflect decreases in cortical activity, and decreases in alpha amplitude reflect increases in cortical activity (Haegens *et al.,* 2011; Shagass, 1972). Thus, spontaneous alpha activity recorded over the PFC is used as an index of frontal cortical activity (Davidson, 1984, 1992). Alpha asymmetry scores are calculated by subtracting left alpha power from right alpha power between symmetrical pairs of electrodes. Based on the assumption that alpha power is inversely related to cortical activity (Haegens *et al.,* 2011; Shagass, 1972), positive alpha asymmetry scores reflect greater left cortical activity relative to right cortical activity. This is because positive alpha asymmetry scores result from less alpha activity measured at the left electrode compared to at the right electrode. In comparison, negative alpha asymmetry scores reflect greater right cortical activity relative to left cortical activity, and result from less alpha activity measured at the right electrode compared to at the left electrode. Finally, alpha asymmetry scores of 0

reflect equivalent alpha and cortical activity measured at the right and left electrode sites.

In support of this theory, there is a large body of evidence suggesting that a greater tendency to approach rewarding or appetitive stimuli is associated with relatively greater left frontal cortical activity compared to right frontal cortical activity, whereas a greater tendency to avoid threatening or aversive stimuli is associated with relatively greater right frontal cortical activity compared to left frontal cortical activity (Coan & Allen, 2004; Davidson, 1984, 1992; Harmon-Jones *et al*., 2010; Tomarken *et al*., 1990; Tomarken *et al*., 1992; Wheeler *et al*., 1993). Notably, the lateralisation of frontal cortical activity is based on the approach-avoidance distinction rather than the positive-negative valence distinction. For example, Harmon-Jones and Allen (1998) found that anger, a negatively valenced emotion, was associated with greater left frontal cortical activity, indicating that left frontal activity was associated with the approach system rather than positively valenced emotions.

Most of the work to date examining frontal alpha asymmetry has been conducted in undergraduate samples. Thus, whether frontal alpha asymmetry can account for the age-related changes in risk-taking and anxiety during adolescence remains unclear. In Chapter 3, this doctoral work examined the age- and gender-related changes in risk-taking behaviours and anxiety levels across preadolescence, mid-adolescence, and late adolescence. It was predicted that risk-taking behaviours and anxiety levels would peak in mid-adolescence (Abe & Suzuki, 1986; Burnett *et al*., 2010; Steinberg *et al*., 2008). As expected, real world risk-taking behaviours increased significantly from preadolescence to mid-adolescence for males and females. Real world risk-taking also increased from mid-adolescence to late adolescence for females. BART risk-taking also increased from mid-adolescence to late adolescence. By contrast, anxiety levels did not increase from preadolescence to mid-adolescence; instead, preadolescents had greater anxiety levels than mid-adolescents and late adolescents. Hence, the current study focused on whether frontal asymmetry could account for the developmental differences in risk-taking behaviours observed in this sample of adolescents.

The neurobiological models assert that adolescent risk-taking behaviour is driven by a hyperresponsive approach reward-based system compared to children and adults (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008). Hence, if frontal alpha asymmetry reflects the approach-avoidance systems (Davidson,

1984, 1992), it should be possible to test this assertion by exploring age-related changes in frontal alpha asymmetry. Consequently, this study examined the age- and gender-related differences in frontal alpha asymmetry, and the relationship between frontal alpha asymmetry and risk-taking behaviours in male and female adolescents aged 9-23 years. The following sections outline what is currently known about the relationship between frontal asymmetry and the approach system in adult and adolescent samples (Chapter 5.1.4.1), as well as methodological considerations in frontal alpha asymmetry research (Chapter 5.1.4.2).

5.1.4.1. Left frontal cortical activity and the approach system

There is a large body of work conducted with undergraduate and young adult samples suggesting that greater left frontal cortical activity is associated with approach sensitivity and approach-related behaviours such as risk-taking and sensation seeking (Coan & Allen, 2003; Santesso *et al.*, 2008; Sutton & Davidson, 1997; Wheeler *et al.*, 1993). For instance, Santesso *et al.* (2008) found that high levels of sensation seeking were associated with relatively greater left cortical activity in 18-26 year olds. Notably however, not all studies have reported that relatively greater left frontal cortical activity is associated with approach behaviours. For example, Schutter *et al.* (2004) found that more risky decision-making on a behavioural task was associated with relatively greater right frontal cortical activity in 18-26 year olds. This indicates that the relationships between the approach system and left frontal cortical activity may not be as robust as previously suggested (Davidson, 1984, 1992).

In contrast to adult work, research assessing the relationships between frontal asymmetry and the approach system in adolescents is scarce. Initial evidence in mid and late adolescents (14-21 years) suggests that high approach sensitivity is associated with relatively greater left frontal cortical activity (Black *et al.*, 2014). This finding is consistent with adult studies (e.g., Coan & Allen, 2003; Santesso *et al.*, 2008; Sutton & Davidson, 1997; Wheeler *et al.*, 1993). However, Black *et al.*'s (2014) study needs extending in two ways. First, Black *et al.* (2014) examined the relationships between approach sensitivity and frontal alpha asymmetry in a single group of adolescents aged 14-21 years that spanned two developmental periods (i.e., middle and late adolescence). Critically, adolescence is a transitional period, and therefore the relationships between frontal alpha asymmetry and approach-related behaviours need to be examined during the distinct phases of adolescence rather than in a single age

group. Second, Black *et al*. (2014) failed to explore potential gender differences in the relationships between approach sensitivity and frontal asymmetry. Notably, there are considerable gender differences in risk-taking behaviours and brain development during adolescence (Byrnes *et al*., 1999; Lenroot & Giedd, 2010). Indeed, risk-taking behaviours in the current sample of adolescents followed gender-specific trajectories; real world risk-taking increased from preadolescence to mid-adolescence for both males and females, but also from mid-adolescence to late adolescence for females (see Chapter 3). It is therefore possible that gender-specific relationships will emerge between frontal asymmetry and approach-related behaviours in this sample of adolescents.

To these ends, the current study aimed to examine the relationships between frontal asymmetry and risk-taking behaviours, as measured by the BART and YRBSS, in typically developing male and female adolescents aged 9-23 years. Although sensation seeking has also been associated with frontal asymmetry in adult samples (Santesso *et al.*, 2008), sensation seeking tendencies in the current sample of adolescents, as measured using the BSSS, did not change as a function of age, gender, or puberty. Thus, the relationships between frontal asymmetry and sensation seeking were not explored in the current study.

5.1.4.2. Methodological considerations in frontal alpha asymmetry research

While there is a large body of evidence for the approach-avoidance lateralisation of frontal cortical activity, there are a number of methodologies used in frontal alpha asymmetry research that need considering. First, frontal asymmetry research has predominantly focused on two pairs of frontal scalp electrodes (F4-F3 and F8-F7; e.g., Black *et al*., 2014; Harmon-Jones *et al*., 2008; Santesso *et al.*, 2008; Schutter *et al*., 2004). However, the PFC accounts for a large proportion of cortical grey matter that contains anatomically and functionally distinct structures, including the medial PFC (mPFC), dorsolateral PFC (DLPFC), and orbitofrontal cortex (OFC). Accordingly, it has been suggested that a more fine-grained analysis of the PFC is needed in frontal asymmetry research (Miller, Crocker, Spielberg, Infantolino & Heller, 2015). Recent advancements in EEG source localisation have provided reliable methods for estimating cortical activity in specific structures. Consequently, to explore frontal asymmetry and its relationship to risk-taking in specific frontal structures, the current study computed frontal asymmetry scores using source

localised frontal cortical activity in three ROIs: mPFC, DLPFC, and OFC.

In order to compare the source localised frontal asymmetry scores to the existing literature, frontal asymmetry scores were also computed using the two most widely reported scalp electrode pairs in frontal asymmetry research, F4-F3 and F8-F7 (Black *et al.*, 2014; Harmon-Jones *et al.*, 2008; Santesso *et al.*, 2008; Schutter *et al.*, 2004). While the vast majority of previous studies have referenced frontal alpha activity using traditional references, such as linked mastoids, a number of recent papers have documented the benefits of CSD transformed data over traditional references for frontal alpha asymmetry research (see Chapter 2.4.3.2 for details). Hence, this study applied a CSD transformation to the EEG data before computing frontal asymmetry scores with the scalp electrodes.

Finally, most studies examining frontal asymmetry in children, adolescents, and adults have correlated the average of the eyes-open and eyes-closed conditions with approach- and avoidance-related behaviours and emotions (e.g., Santesso *et al.*, 2008; Schutter *et al.*, 2004; Wiedemann *et al.*, 1999). Recent work in children and young adults shows that opening the eyes leads to an increase in skin conductance levels and a global reduction of alpha power, indicating that the shift from eyes-closed to eyes-open results in an increase in arousal (Barry, Clarke, Johnstone & Brown, 2009; Barry, Clarke, Johnstone, Magee & Rushby, 2007). Moreover, as will be discussed in Chapter 5.1.5, there are considerable differences in the cortical and subcortical sources of spontaneous alpha during eyes-open and eyes-closed conditions (Feige *et al.*, 2005; Lüchinger, Michels, Martin & Brandeis, 2011; Wu, Eichele & Calhoun, 2010). It is therefore surprising that eyes-open and eyes-closed conditions are often regarded as equivalent baseline conditions and combined in frontal asymmetry studies. Thus, the current study examined frontal asymmetry and its relationship to risk-taking behaviours separately for eyes-open and eyes-closed conditions.

5.1.5. Cortical and subcortical sources of spontaneous alpha

While the studies discussed in the previous sections provide insights into the functional significance of alpha, they are unable to shed light on the cortical and subcortical sources underlying spontaneous alpha. The underlying generators of spontaneous alpha have been estimated in adults using EEG source localisation (Cuspineda *et al.*, 2009; Knyazev, Slobodskoj-Plusnin, Bocharov & Pylkova, 2011; Laufs *et al.*, 2003), magnetoencephalography (MEG) (Hari, Salmelin, Mäkelä, Salenius

& Helle, 1997), combined positron emission tomography (PET)-EEG (Sadato *et al*., 1998; Schreckenberger *et al*., 2004), and combined fMRI-EEG (De Munck *et al*., 2007; Feige *et al*., 2005; Goldman, Stern, Engel Jr & Cohen, 2002; Lüchinger *et al*., 2011; Moosmann *et al*., 2003; Wu *et al*., 2010). The thalamus and occipital-parietal cortices are most frequently reported as the generators of spontaneous alpha in adults when the eyes are closed. Specifically, spontaneous alpha during eyes-closed conditions is positively correlated with the BOLD signal in the thalamus and negatively correlated with the BOLD signal in occipital-parietal regions (Cuspineda *et al*., 2009; De Munck *et al*., 2007; Feige *et al*., 2005; Goldman *et al*., 2002; Knyazev *et al*., 2011; Lüchinger *et al*., 2011; Moosmann *et al*., 2003). Negative correlations between frontal and temporal cortices and spontaneous alpha during eyes-closed conditions have also been reported in adults (Cuspineda *et al*., 2009; De Munck *et al*., 2007; Goldman *et al*., 2002; Wu *et al*., 2010).

Animal work has reported that the alpha rhythm can be detected slightly earlier in the thalamus than in the cortex (Da Silva & Van Leeuwen, 1977), suggesting that the thalamus generates the alpha rhythm and subsequently induces synchronised alpha activity in the cortex (Steriade, Gloor, Llinas, Da Silva & Mesulam, 1990). Moreover, the findings that the BOLD response in frontal, temporal, parietal, and occipital cortical regions is correlated with spontaneous alpha are consistent with the thalamus having strong, reciprocal connections to most cortical regions (Höhl-Abrahão & Creutzfeldt, 1991). Together, these findings provide clear evidence that thalamo-cortical networks are critical in generating spontaneous alpha (Da Silva, Van Lierop, Schrijer & Van Leeuwen, 1973). However, there is also evidence that cortico-cortical networks that are independent of thalamic input are also involved in generating spontaneous alpha; Da Silva, Vos, Mooibroek and Van Rotterdam (1980) found cortio-cortical alpha coherences, i.e., spectral associations between separate cortical regions, after eliminating cortio-cortical coherences attributable to the thalamus. Therefore, the converging evidence from human and nonhuman animal studies suggests that spontaneous alpha is generated by a combination of thalamo-cortical and cortico-cortical networks.

Most studies examining the sources of spontaneous alpha have exclusively examined the sources of alpha during eyes-closed conditions. However, a handful of studies have examined the differences between eyes-open and eyes-closed conditions in the cortical and subcortical sources of spontaneous alpha (Feige *et al*., 2005;

Lüchinger *et al.*, 2011; Wu *et al.*, 2010). For instance, Lüchinger *et al.* (2011) found that alpha power was positively correlated with the BOLD signal in the thalamus and negatively correlated with the BOLD signal in occipital and parietal regions during the eyes-closed condition. Alpha power was also negatively correlated with the BOLD signal in occipital and parietal regions during the eyes-open condition, but also in frontal regions. These findings suggest that a wider neural network is engaged when the eyes are open compared to when the eyes are closed. Other studies have reported that the shift from eyes-closed to eyes-open significantly reduces the haemodynamic response in the thalamus, as well as in frontal, temporal, parietal, and occipital cortical regions (Feige *et al*., 2005; Wu *et al.*, 2010). Together, these findings indicate that there are considerable differences in the sources of alpha between eyes-open and eyes-closed conditions.

In contrast to adult work, the sources of spontaneous alpha in adolescents have been scarcely studied. To the author's knowledge, only one study to date has compared the sources of spontaneous alpha in adolescents and adults (Lüchinger *et al*., 2011). Lüchinger *et al*. (2011) recorded EEG and fMRI simultaneously in adolescents aged 15 years and young adults aged 25 years. Unexpectedly, no age differences between adolescents and adults were found in the sources of spontaneous alpha during the eyes-closed condition or eyes-open condition. While Lüchinger *et al*.'s (2011) study represents a first step in understanding the sources of spontaneous alpha during adolescence, only adolescents aged 15 years and adults aged 25 years participated in the study. Consequently, the findings from this study cannot be generalised to adolescents of other ages, and are unable to explain whether the sources of spontaneous alpha change during the course of adolescence, i.e., throughout preadolescence, mid-adolescence, and late adolescence. Spontaneous alpha is disrupted in several psychiatric disorders that often emerge during adolescence, including anxiety, major depression, and schizophrenia (Babiloni *et al*., 2009; Grin-Yatsenko, Baas, Ponomarev & Kropotov, 2009; Kessler *et al*., 2005; Knyazev, Savostyanov & Levin, 2004). Thus, understanding how the sources of spontaneous alpha mature in typical developmental populations is critical for understanding how spontaneous alpha deviates from the norm in atypical populations. Furthermore, Lüchinger *et al*. (2011) did not explore potential gender differences in the sources of spontaneous alpha; the adolescent and young adult age groups were composed of both males (n = 8) and females (n = 10). Given that the brain matures more quickly in

females than males during adolescence (Lenroot & Giedd, 2010; see Chapter 1.4), it is possible that the cortical sources of spontaneous alpha will differ between adolescent males and females. To these ends, this study aimed to examine the cortical sources of spontaneous alpha in frontal (mPFC, DLPFC, OFC) and occipital ROIs using EEG source localisation in a sample of male and female adolescents aged 9-23 years.

5.1.6. The influence of puberty on the development of spontaneous alpha

Examining the cortical sources of spontaneous alpha during preadolescence, mid-adolescence, and late adolescence will provide insights into how the cortical sources of spontaneous alpha change as a function of age and gender throughout adolescence. However, there is increasing evidence showing that puberty has specific affects on the structural development of cortical and subcortical grey matter in human adolescents that are independent of chronological age (Bramen *et al*., 2011; Goddings *et al*., 2014; Neufang *et al*., 2009; see Chapter 1.4). Since the alpha rhythm is generated by cortical and subcortical grey matter structures (Da Silva *et al.*, 1980; Lüchinger *et al.*, 2011; see Chapter 5.1.5), it is possible that the influence puberty has on grey matter development may in turn affect the development of spontaneous alpha. Despite this, no study to date has explored the relationships between puberty and spontaneous alpha. Accordingly, this study also aimed to examine the relationships between puberty and spontaneous alpha in the frontal (mPFC, DLPFC, OFC) and occipital ROIs in male and female adolescents aged 9-17 years. Since the development of grey matter in several cortical and subcortical brain structures is nonlinear (Gogtay *et al*., 2004; Mills *et al*., 2014; Mills *et al*., 2016), both the linear and nonlinear relationships between puberty and spontaneous alpha were explored.

5.1.7. The current study

The same cohort of participants took part in this study as in Chapters 3 and 4. In the current study, preadolescents aged 9-12 years, mid-adolescents aged 13-17 years, and late adolescents aged 18-23 years had their resting brain activity measured using EEG during eyes-open and eyes-closed conditions. Participants' resting brain activity was measured during the same EEG session as the instrumental conditioning task (Chapter 4). Participants' risk-taking behaviours, as indexed by the BART and YRBSS (Chapter 3), were examined in relation to participants' frontal alpha asymmetry scores.

The first aim of this study was to examine the cortical sources of spontaneous alpha in male and female preadolescents, mid-adolescents, and late adolescents. Firstly, it was predicted that the cortical sources of spontaneous alpha would be located in occipital-parietal regions during the eyes-closed condition (De Munck *et al*., 2007; Lüchinger *et al.*, 2011). By comparison, it was predicted that the cortical sources of alpha would be more widely distributed and located in frontal regions as well as in occipital-parietal regions during the eyes-open condition (Lüchinger *et al.*, 2011). Secondly, it was predicted that alpha power in the occipital ROI would be greater in the eyes-closed condition compared to the eyes open-condition (Feige *et al*., 2005; Wu *et al.*, 2010). Thirdly, it was predicted that alpha power would decrease in all ROIs from preadolescence to late adolescence in both the eyes-open and eyes-closed conditions (Chiang *et al*., 2011; Dustman *et al*., 1999; Yordanova & Kolev, 1997).

The second aim of this study was to examine the linear and nonlinear relationships between puberty and spontaneous alpha in the frontal and occipital ROIs in male and female adolescents aged 9-17 years while controlling for chronological age. Since cortical and subcortical grey matter reduces across adolescence (Gogtay *et al*., 2004; Mills *et al*., 2014; Mills *et al*., 2016), it was predicted that more advanced pubertal stage would be associated with reduced levels of alpha in males and females in the eyes-open and eyes-closed conditions.

The third and final aim of this study was to examine whether frontal alpha asymmetry could account for the developmental differences in risk-taking behaviours observed in this sample of adolescents aged 9-23 years (Chapter 3). Based on previous work (Black *et al*., 2014; Coan & Allen, 2003; Santesso *et al.*, 2008; Sutton & Davidson, 1997; Wheeler *et al*., 1993), it was predicted that relatively greater left frontal cortical activity would be associated with risk-taking behaviours, and that the relationships between frontal asymmetry and risk-taking behaviours would follow the developmental trajectory of risk-taking behaviours reported in Chapter 3.

5.2. Method

5.2.1. Participants

The same cohort of participants took part in this study as in Chapters 3 and 4. In total, ninety-five volunteers aged 9-23 years participated in this study. Three participants were excluded from analyses due to insufficient data following artefact rejection (3 females aged 9, 17, and 21 years). Participants were split into three age

groups: preadolescents aged 9-12 years; mid-adolescents aged 13-17 years; and late adolescents aged 18-23 years. Descriptive statistics for the final sample are reported in Table 5.1. Detailed participant information is reported in Chapter 2.1.

Table 5.1
Participant demographics

				Risk-taking behaviours *M* [95% CI]				Anxiety levels [a] *M* [95% CI]	
Age Group	Gender	*n*	Age *M* (*SD*)	BART Pumps	BART Points	BART Balloons	YRBSS	STAI-T	STAI-T HADS-D
Preadolescents	Females	14	10.93 (1.21)	37.27 [30.18, 45.38]	5349.29 [4256.41, 6502.03]	7.14 [5.02, 9.62]	0.43 [0.21, 0.64]	54.41 [50.00, 58.81]	58.11 [50.50, 65.98]
	Males	15	10.53 (1.30)	40.90 [35.53, 46.51]	6024.00 [5254.36, 6759.79]	9.00 [7.13, 11.08]	0.93 [0.53, 1.40]	58.11 [50.57, 65.22]	58.97 [54.47, 63.71]
Mid-adolescents	Females	14	14.50 (1.51)	38.35 [32.15, 43.77]	5713.57 [4636.31, 6635.84]	8.21 [6.34, 10.00]	1.79 [1.21, 2.43]	54.82 [48.53, 61.65]	55.82 [50.53, 61.15]
	Males	15	14.80 (1.42)	41.11 [34.69, 47.73]	5949.33 [5076.75, 6787.21]	8.67 [6.73, 10.80]	4.00 [2.80, 5.20]	49.33 [42.71, 56.21]	46.40 [39.65, 53.23]
Late Adolescents	Females	17	20.35 (1.41)	46.29 [39.28, 53.50]	6954.12 [5774.75, 7971.07]	9.18 [7.88, 10.59]	4.29 [3.41, 5.29]	52.94 [45.00, 60.72]	52.67 [46.75, 57.87]
	Males	16	21.00 (1.55)	48.96 [41.72, 55.83]	6997.50 [6111.94, 7875.11]	10.05 [8.56, 12.72]	3.25 [2.19, 4.34]	53.52 [46.33, 61.02]	51.64 [45.79, 58.09]

Note. BART Pumps = BART number of adjusted pumps; BART Points = BART total points won; BART Balloons = BART number of popped balloons; YRBSS = Youth Risk Behaviour Surveillance Survey; STAI-T = State Trait Anxiety Inventory-Trait Anxiety; STAI-T HADS = State Trait Anxiety Inventory-Trait Anxiety controlling for Hospital Anxiety and Depression Scale-Depression Scale; 95% CI = bootstrapped 95% confidence intervals; [a] = percentage scores.

5.2.2. Procedure

The procedure is reported in Chapter 2.2.

5.2.3. Behavioural measures

5.2.3.1. Risk-taking behaviours

Risk-taking behaviours were measured using the BART (Lejuez *et al*., 2002) and YRBSS (Aklin *et al*., 2005) (see Chapter 2.3.1).

5.2.3.2. Pubertal development

Pubertal development was assessed in 9-17 year olds using the PDS (Petersen *et al*., 1988; see Chapter 2.3.6). Consistent with previous work (Marshall & Tanner,

1969; Marshall & Tanner, 1970), an independent t-test revealed that PDS scores were higher for females aged 9-17 years (M = 2.73, 95% CI [2.45, 3.00]) compared to males aged 9-17 years (M = 2.30, 95% CI [2.02, 2.55]) ($t(56) = 2.05$, $p = 0.050$), indicating that females were at a slightly later stage in their pubertal development compared to males.

5.2.4. EEG recording

The EEG recording is reported in Chapter 2.4.1.

5.2.5. EEG preprocessing

The EEG preprocessing stream is reported in Chapter 2.4.3. The cleaned, epoched resting state data for the eyes-open and eyes-closed conditions were subjected to further processing in order to localise the cortical sources of spontaneous alpha (see Chapter 5.2.5.1) and compute frontal alpha asymmetry scores (see Chapter 5.2.5.2).

5.2.5.1. sLORETA source localisation

sLORETA (Pascual-Marqui, 2002; see Chapter 2.4.3.3 for details) was used to estimate the cortical sources of spontaneous alpha in the eyes-open and eyes-closed conditions. Artefact-free epochs were given as an input to sLORETA and used to compute the EEG cross-spectra for each participant. The cross-spectra were then used to estimate the corresponding three-dimensional CSD for the alpha frequency band (8-

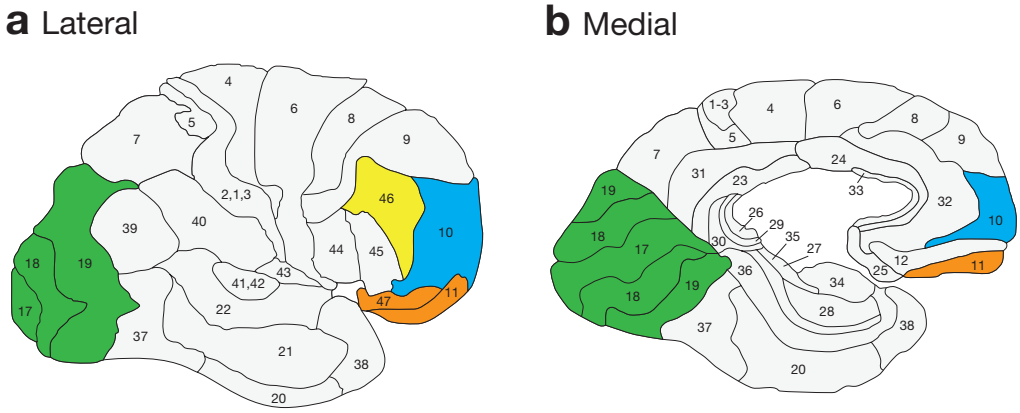

Figure 5.1 Human Brodmann areas (BA) for the lateral (**a**) and medial (**b**) surface. The highlighted BA reflect the regions of interest used in this study. BA10 = medial prefrontal cortex (blue); BA46 = dorsolateral prefrontal cortex (yellow); BA11 and BA47 = orbitofrontal cortex (orange); BA17, BA18, and BA19 = occipital cortex (green).

13 Hz). ROIs were used to examine alpha CSD in frontal and occipital areas. Brodmann areas (BA) were used to define the ROIs, and included the medial PFC (mPFC; BA10), dorsolateral PFC (DLPFC; BA46), orbitofrontal cortex (OFC; BA11 and BA47), and occipital cortex (BA17, BA18, and BA19) (Figure 5.1).

5.2.5.2. Frontal alpha asymmetry

Two methods were used to examine the relationships between frontal alpha asymmetry and participants' risk-taking behaviours. First, frontal asymmetry scores were calculated with frontal ROIs. Asymmetry scores were calculated for each frontal ROI (mPFC, DLPFC, and OFC) by subtracting average alpha CSD in the left hemisphere from average alpha CSD in the right hemisphere. Second, frontal asymmetry scores were computed using scalp electrodes in order to compare the current study with the existing frontal asymmetry literature. Scalp asymmetry scores were computed on the basis of previous work (e.g., Black *et al*., 2014; Harmon-Jones & Allen, 1998; Schutter *et al*., 2004). First, the artefact-free epochs were transformed to reference-free CSD estimates ($\mu V/cm^2$) using a spherical spline surface Laplacian via the CSD toolbox (Kayser & Tenke, 2006; Perrin, Pernier, Bertrand & Echallier, 1989; see Chapter 2.4.3.2 for more details regarding CSD). Next, a fast Fourier Transform, using a Hamming window with tapered edges, was applied to each epoch to compute estimates of

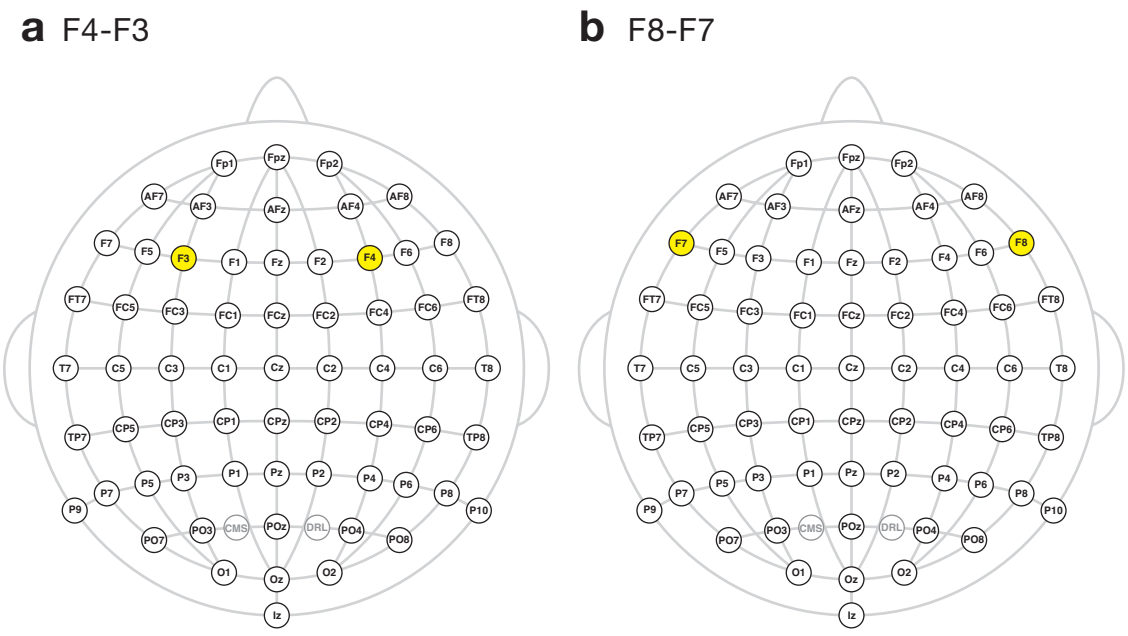

Figure 5.2 Electrode pairs F4-F3 (**a**) and F8-F7 (**b**) used for the scalp asymmetry scores.

spectral power (μV^2). Spectral power values were then converted to power density (μV^2/Hz) in the alpha frequency band (8-13 Hz). Power density values for the alpha frequency band were then averaged across all epochs and natural log transformed. Scalp asymmetry scores were calculated by subtracting left log average alpha power density scores from right log average alpha power density scores for symmetrical electrode pairs. The electrode pairs F4-F3 and F8-F7 (Figure 5.2) were selected to examine frontal alpha asymmetry since they are the most widely used electrode pairs in both adolescent (Black *et al*., 2014) and young adult (Harmon-Jones *et al*., 2008; Santesso *et al.*, 2008; Schutter *et al*., 2004) frontal asymmetry research.

Alpha power is inversely related to cortical activity (Haegens *et al.,* 2011; Shagass, 1972). Thus, for both the ROIs and scalp electrodes, positive asymmetry scores reflect greater left cortical activity relative to right cortical activity, and negative asymmetry scores reflect greater right cortical activity relative to left cortical activity. Asymmetry scores of 0 reflect equivalent cortical activity in the left and right hemispheres.

5.3. Results

5.3.1. Cortical sources of spontaneous alpha during adolescence

5.3.1.1. Scalp and sLORETA CSD maps

The scalp (2D) and sLORETA (3D) CSD maps provided initial insights into how the spatial distribution of alpha changed during the course of adolescence. The scalp topographical maps (Figure 5.3) indicated that posterior alpha was greater in the eyes-closed compared to eyes-open condition for all groups. The scalp maps also suggested that posterior alpha reduced in magnitude from preadolescence to late adolescence during the eyes-open and eyes-closed conditions.

The sLORETA maps for the eyes-open (Figure 5.4) and eyes-closed (Figure 5.5) conditions revealed insights into the cortical sources underlying the scalp-recorded alpha activity during the different stages of adolescence. The sLORETA maps indicated that occipital-parietal regions were primarily responsible for generating alpha during the eyes-closed condition. By comparison, the sLORETA maps suggested that a much wider neural network, including occipital, parietal, temporal, and frontal regions, was engaged during the eyes-open condition. Consistent with the scalp maps, the sLORETA maps also indicated that alpha reduced in magnitude from preadolescence to late adolescence during the eyes-open and eyes-closed conditions.

5.3.1.2. Age and gender differences in the cortical sources of spontaneous alpha

Mixed-design ANOVAs were used to examine age- and gender-related differences in the cortical sources of spontaneous alpha during the eyes-open and eyes-closed conditions. Separate ANOVAs were conducted for each ROI (mPFC, DLPFC, OFC, occipital cortex), with *Condition* (eyes-open, eyes-closed) as the within-group factor, and *Age Group* (preadolescents, mid-adolescents, late adolescents) and *Gender* (females, males) as the between-group factors. Significant interactions were followed up using bootstrapped t-tests. ANOVA pairwise comparisons and t-tests were corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5). Figure 5.6 displays the group means and bootstrapped 95% confidence intervals for alpha CSD in the mPFC, DLPFC, OFC, and occipital cortex during the eyes-open and eyes-closed conditions.

5.3.1.2.1. Medial PFC (mPFC)

A main effect of *Condition* was found ($F(1, 85) = 12.14$, $p = 0.001$, $\eta_p^2 = 0.13$), revealing that mPFC alpha was greater in eyes-open ($M = 0.09$, 95% CI [0.07, 0.10]) than eyes-closed ($M = 0.06$, 95% CI [0.05, 0.07]). A main effect of *Age Group* was also found ($F(2, 85) = 7.18$, $p = 0.001$, $\eta_p^2 = 0.14$), showing that preadolescents ($M = 0.10$, 95% CI [0.08, 0.12]) had greater mPFC alpha than both mid-adolescents ($M = 0.05$, 95% CI [0.03, 0.07]) ($p = 0.001$) and late adolescents ($M = 0.06$, 95% CI [0.04, 0.08]) ($p = 0.003$). No difference in mPFC alpha was found between mid-adolescents and late adolescents ($p = 0.632$).

The main effects of *Condition* and *Age Group* were qualified by a *Condition* by *Age Group* interaction ($F(2, 85) = 3.52$, $p = 0.034$, $\eta_p^2 = 0.08$). Paired t-tests revealed that preadolescents had equivalent mPFC alpha in eyes-open ($M = 0.11$, 95% CI [0.08, 0.14]) and eyes-closed ($M = 0.09$, 95% CI [0.07, 0.12]) ($t(28) = 1.58$, $p = 0.126$). Similarly, mid-adolescents had equivalent mPFC alpha in eyes-open ($M = 0.06$, 95% CI [0.05, 0.07]) and eyes-closed ($M = 0.05$, 95% CI [0.04, 0.06]) ($t(28) = 1.44$, $p = 0.161$). By contrast, late adolescents had significantly more mPFC alpha in eyes-open ($M = 0.09$, 95% CI [0.06, 0.13]) compared to eyes-closed ($M = 0.03$, 95% CI [0.02, 0.03]) ($t(32) = 2.93$, $p = 0.006$). No main effect of *Gender* was found ($F(1, 85) = 1.08$, $p = 0.301$, $\eta_p^2 = 0.13$), and all other interactions were non-significant.

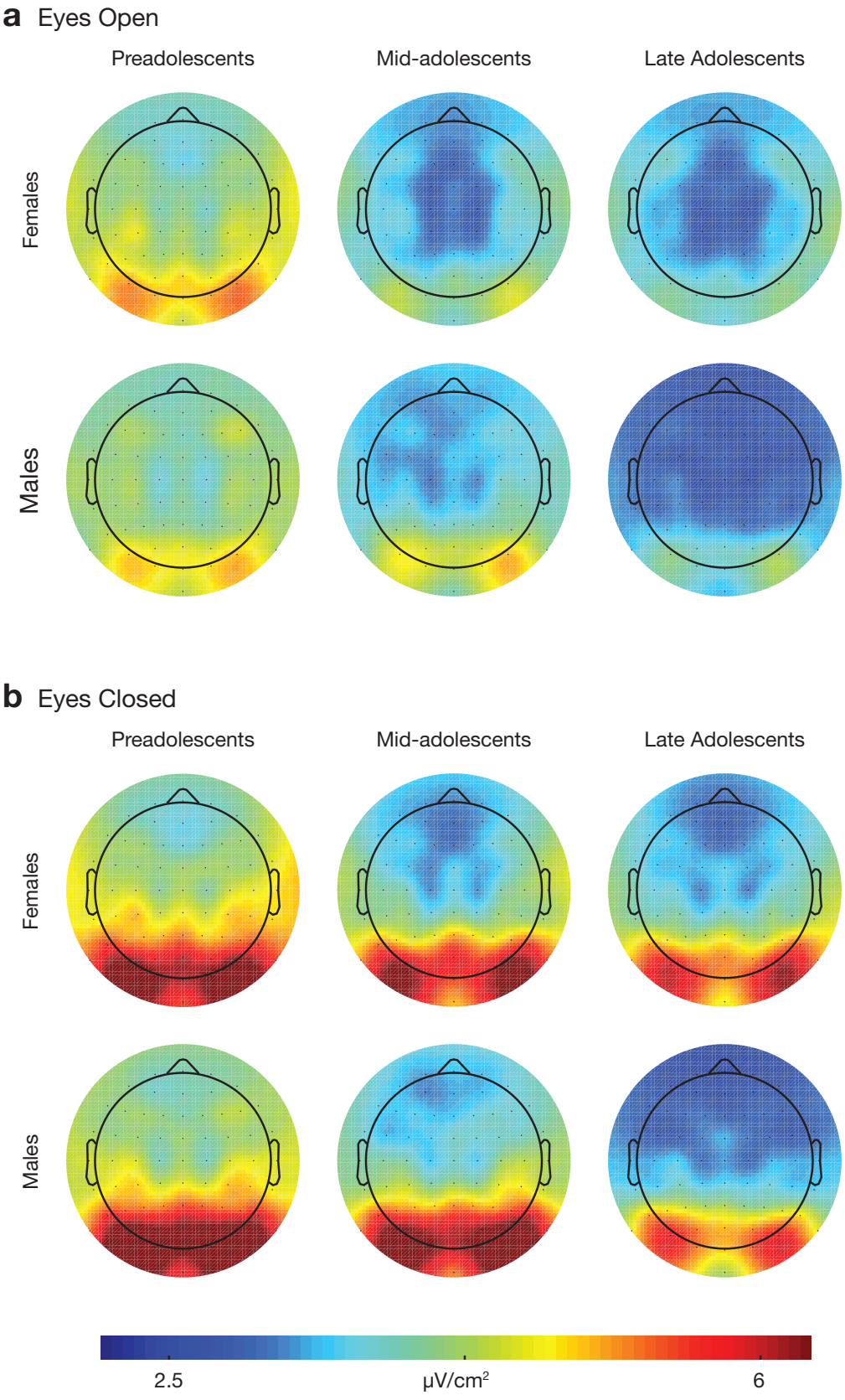

Figure 5.3 Scalp (2D) CSD maps for the alpha frequency band (8-13 Hz) during the eyes-open (**a**) and eyes-closed (**b**) conditions.

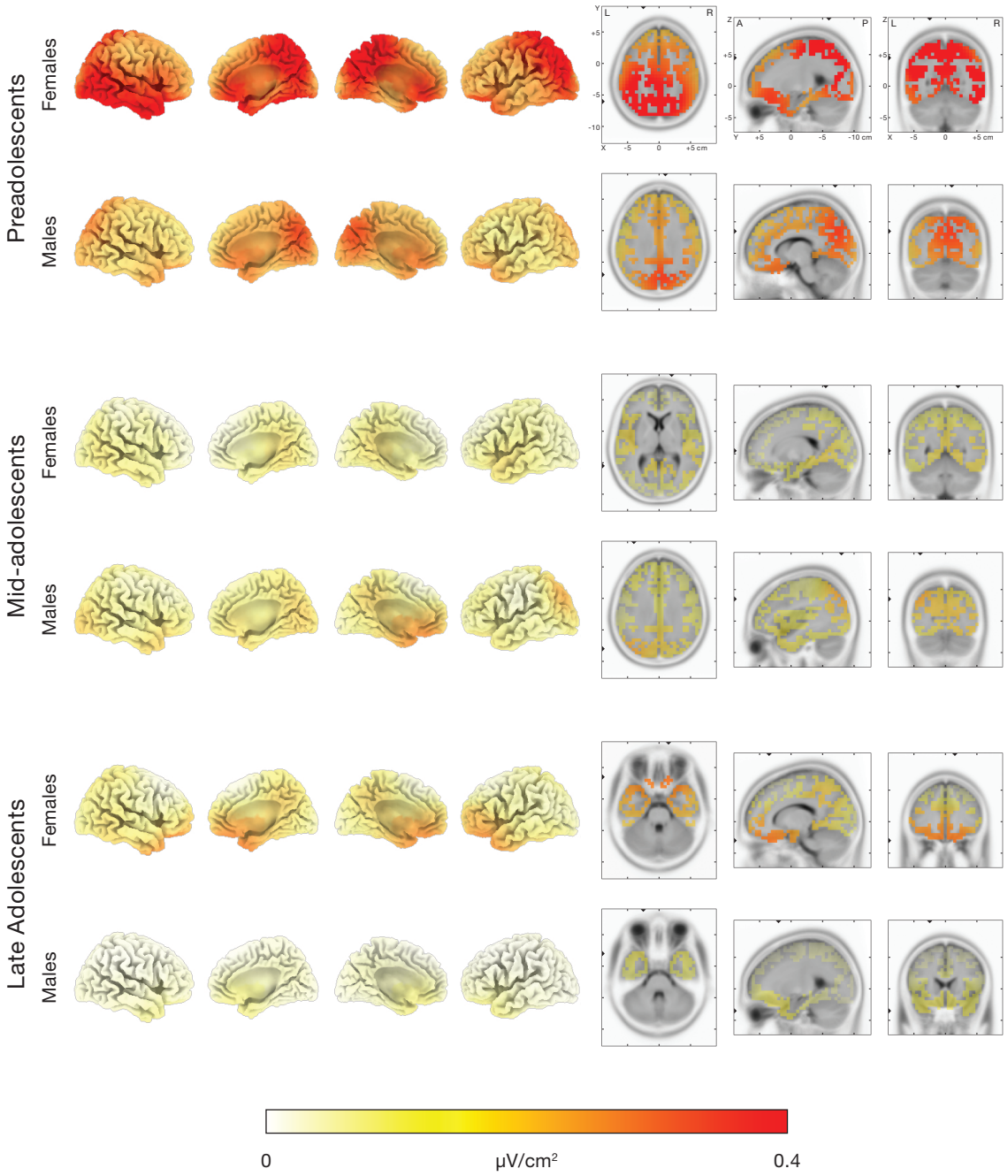

Figure 5.4 sLORETA (3D) CSD maps for the alpha frequency band (8-13 Hz) during the eyes-open condition.

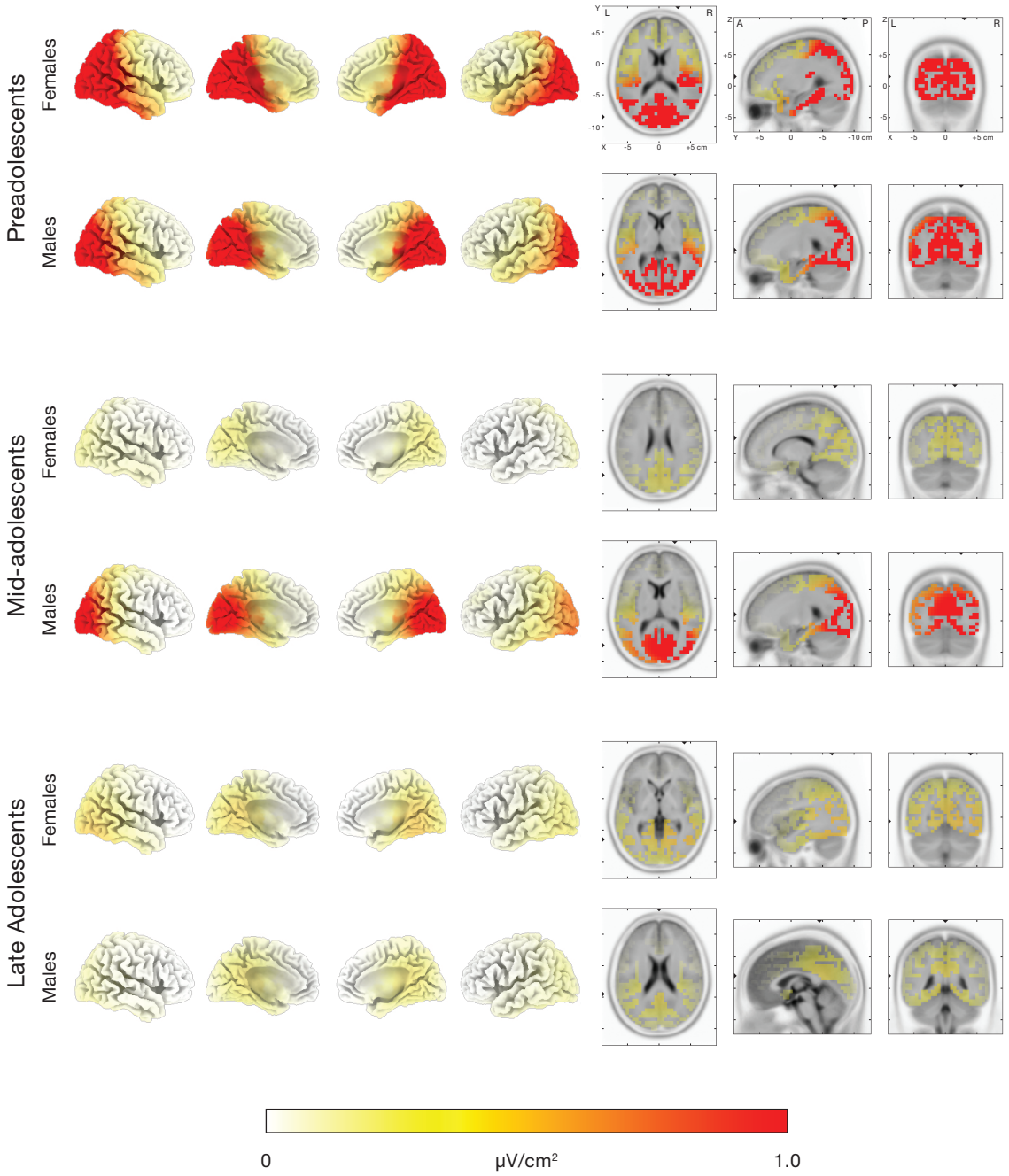

Figure 5.5 sLORETA (3D) CSD maps for the alpha frequency band (8-13 Hz) during the eyes-closed condition.

5.3.1.2.2. Dorsolateral PFC (DLPFC)

A main effect of *Condition* was found ($F(1, 85) = 10.01$, $p = 0.002$, $\eta_p^2 = 0.11$), showing that DLPFC alpha was greater in eyes-open ($M = 0.09$, 95% CI [0.07, 0.11]) compared to eyes-closed ($M = 0.06$, 95% CI [0.05, 0.08]). A main effect of *Age Group* was also found ($F(2, 85) = 9.97$, $p < 0.001$, $\eta_p^2 = 0.19$), revealing that preadolescents ($M = 0.11$, 95% CI [0.09, 0.13]) had more DLPFC alpha compared to both mid-adolescents ($M = 0.06$, 95% CI [0.04, 0.08]) ($p < 0.001$) and late adolescents ($M = 0.06$, 95% CI [0.04, 0.08]) ($p < 0.001$). No difference in DLPFC alpha was found between mid-adolescents and late adolescents ($p = 0.927$). No main effect of *Gender* was found ($F(1, 85) = 0.70$, $p = 0.405$, $\eta_p^2 = 0.01$), and all interactions were non-significant.

5.3.1.2.3. Orbitofrontal cortex (OFC)

A main effect of *Age Group* was found ($F(2, 85) = 6.15$, $p = 0.003$, $\eta_p^2 = 0.13$), revealing that preadolescents ($M = 0.15$, 95% CI [0.12, 0.18]) had more OFC alpha

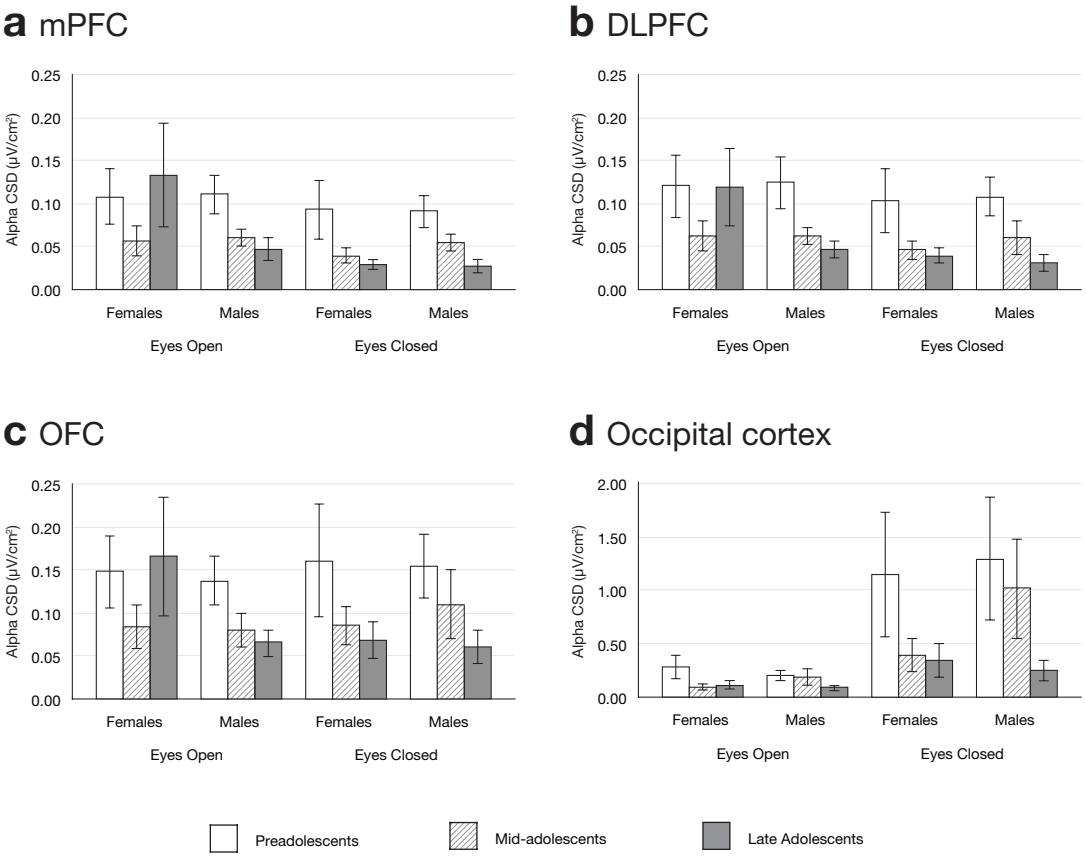

Figure 5.6 Group means for alpha CSD in the mPFC (**a**), DLPFC (**b**), OFC (**c**), and occipital cortex (**d**) during the eyes-open and eyes-closed conditions. Error bars represent 95% bootstrapped confidence intervals.

compared to both mid-adolescents (M = 0.09, 95% CI [0.06, 0.12]) (p = 0.003) and late adolescents (M = 0.09, 95% CI [0.06, 0.12]) (p = 0.003). No difference in OFC alpha was found between mid-adolescents and late adolescents (p = 0.989).

While no main effect of *Condition* was found (F(1, 85) = 0.25, p = 0.615, η_p^2 = 0.00), the *Condition* by *Age Group* interaction was significant (F(2, 85) = 3.49, p = 0.035, η_p^2 = 0.08). The paired t-tests revealed that OFC alpha was equivalent in eyes-open (M = 0.14, 95% CI [0.12, 0.17]) and eyes-closed (M = 0.16, 95% CI [0.12, 0.20]) for preadolescents (t(28) = 7.80, p = 0.442). Similarly, OFC alpha was equivalent in eyes-open (M = 0.08, 95% CI [0.06, 0.10]) and eyes-closed (M = 0.10, 95% CI [0.08, 0.12]) for mid-adolescents (t(28) = 1.47, p = 0.154). By contrast, a trend indicated that late adolescents had significantly more OFC alpha in eyes-open (M = 0.12, 95% CI [0.08, 0.17]) compared to eyes-closed (M = 0.06, 95% CI [0.05, 0.08]) (t(32) = 1.89, p = 0.068). No main effect of *Gender* was found (F(1, 85) = 1.16, p = 0.285, η_p^2 = 0.13), and all other interactions were non-significant.

5.3.1.2.4. Occipital cortex

A main effect of *Condition* was found (F(1, 85) = 38.24, p < 0.001, η_p^2 = 0.31), revealing that occipital alpha was greater in eyes-closed (M = 0.74, 95% CI [0.54, 0.95]) compared to eyes-open (M = 0.16, 95% CI [0.13, 0.20]). These findings suggest that alpha was greater in the occipital cortex during the eyes-closed conditions compared to the eyes-open condition. This is in direct contrast with the findings from the mPFC, DLPFC, and OFC where alpha was greater in eyes-open compared to eyes-closed. A main effect of *Age Group* was also found (F(2, 85) = 7.64, p < 0.001, η_p^2 = 0.15), showing that preadolescents (M = 0.73, 95% CI [0.53, 0.93]) had significantly more occipital alpha than mid-adolescents (M = 0.42, 95% CI [0.23, 0.62]) (p = 0.032) and late adolescents (M = 0.20, 95% CI [0.01, 0.38]) (p < 0.001). No difference in occipital alpha was found between mid-adolescents and late adolescents (p = 0.101).

The main effects of *Condition* and *Age Group* were qualified by a *Condition* by *Age Group* interaction (F(2, 85) = 6.21, p = 0.003, η_p^2 = 0.13). However, the paired t-tests revealed the same pattern of findings for each age group, whereby occipital alpha was greater during the eyes-closed condition compared to the eyes-open condition. In particular, occipital alpha was greater in eyes-closed (M = 1.22, 95% CI [0.70, 1.85]) than eyes-open (M = 0.24, 95% CI [0.18, 0.32]) for preadolescents (t(28) = 3.87, p = 0.001). Similarly, occipital alpha was greater in eyes-closed (M = 0.71, 95% CI [0.44,

1.06]) than eyes-open (M = 0.15, 95% CI [0.10, 0.22]) for mid-adolescents ($t(28) = 4.15$, $p < 0.001$). Occipital alpha was also greater in eyes-closed (M = 0.30, 95% CI [0.21, 0.40]) than eyes-open (M = 0.10, 95% CI [0.07, 0.12]) for late adolescents ($t(32) = 4.29$, $p < 0.001$). No main effect of *Gender* was found ($F(1, 85) = 0.98$, $p = 0.326$, $\eta_p^2 = 0.011$), and all other interactions were non-significant.

5.3.2. The influence of puberty on the development of spontaneous alpha

The findings so far suggest that frontal and occipital alpha change significantly during the course of adolescence. However, it is also possible that the development of spontaneous alpha is affected by puberty since several studies have reported that puberty influences the development of cortical and subcortical grey matter (Bramen *et al*., 2011; Goddings *et al*., 2014; Neufang *et al*., 2009). Accordingly, the second aim of this study was to explore the influence of puberty on the development of spontaneous alpha in male and female adolescents aged 9-17 years while controlling for chronological age. Given that the age of pubertal onset is highly variable between individuals (Sørensen *et al.,* 2013), it is possible that categorising adolescents into age groups may confound any potential relationships between puberty and spontaneous alpha. Thus, the relationships between puberty and spontaneous alpha were examined in a continuous sample of females (n = 28; M_{age} = 12.71, SD_{age} = 2.26) and males (n = 30; M_{age} = 12.67, SD_{age} = 2.55) aged 9-17 years.

The same approach was used in this study as in Chapters 3 and 4. Since many developmental trajectories during childhood and adolescence are non-linear, hierarchical polynomial regression analyses were conducted to assess the linear and quadratic relationships between puberty and alpha CSD. Regression analyses were conducted with the four ROIs as the dependent variables (mPFC, DLPFC, OFC, occipital cortex). Separate regression analyses were conducted for the eyes-open and eyes-closed conditions. To tease out the confounding affects of chronological age on pubertal development, age was entered into the first block of the regression analyses as a control variable, and PDS scores were entered into the second block. Separate regression analyses were conducted to assess the linear and quadratic relationships; raw age and PDS scores were used to examine the linear relationships, and squared age and PDS scores were used to assess the quadratic relationships. Regression coefficients were bootstrapped and corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5). The intercorrelations between the

variables are reported in Supplementary Table 5.1 (Appendix 3).

5.3.2.1. Females

Supplementary Table 5.2 (Appendix 3) reports the regression coefficients for eyes-open and eyes-closed for females. No linear or quadratic relationships were found between puberty and spontaneous alpha for either the eyes-open or eyes-closed condition.

5.3.2.2. Males

Supplementary Table 5.3 (Appendix 3) reports the regression coefficients for the eyes-open and eyes-closed conditions for males. Contrary to females, puberty significantly predicted alpha CSD in males. Frontal alpha CSD was significantly predicted by puberty in the eyes-closed condition, but not in the eyes-open condition. In particular, the linear pubertal term significantly predicted mPFC alpha (ΔR^2 = 14.7%, $F(1, 27) = 5.43$, $p = 0.028$), DLPFC alpha (ΔR^2 = 17.8%, $F(1, 27) = 7.09$, $p = 0.013$), and OFC alpha (ΔR^2 = 18.8%, $F(1, 27) = 6.76$, $p = 0.015$). The quadratic pubertal term also significantly predicted mPFC alpha (ΔR^2 = 15.1%, $F(1, 27) = 5.66$, $p = 0.025$), DLPFC alpha (ΔR^2 = 17.6%, $F(1, 27) = 6.85$, $p = 0.014$), and OFC alpha (ΔR^2 = 19.8%, $F(1, 27) = 7.22$, $p = 0.012$).

Occipital alpha was also significantly predicted by the linear (ΔR^2 = 17.7%, $F(1, 27) = 5.99$, $p = 0.021$) and quadratic (ΔR^2 = 16.9%, $F(1, 27) = 5.67$, $p = 0.025$) pubertal terms in the eyes-closed condition. In contrast to the frontal ROIs, occipital alpha was significantly predicted by the linear (ΔR^2 = 26.8%, $F(1, 27) = 10.18$, $p = 0.004$) and quadratic (ΔR^2 = 20.6%, $F(1, 27) = 7.21$, $p = 0.012$) pubertal terms in the eyes-open condition.

Together, these findings revealed that alpha in the frontal and occipital ROIs decreased with advancing pubertal stage in males. These findings indicate that puberty has significant affects on the development of spontaneous alpha during adolescence that are independent of chronological age but dependent on gender. The partial regression plots are presented in Figure 5.7 for the frontal ROIs and in Figure 5.8 for the occipital ROI.

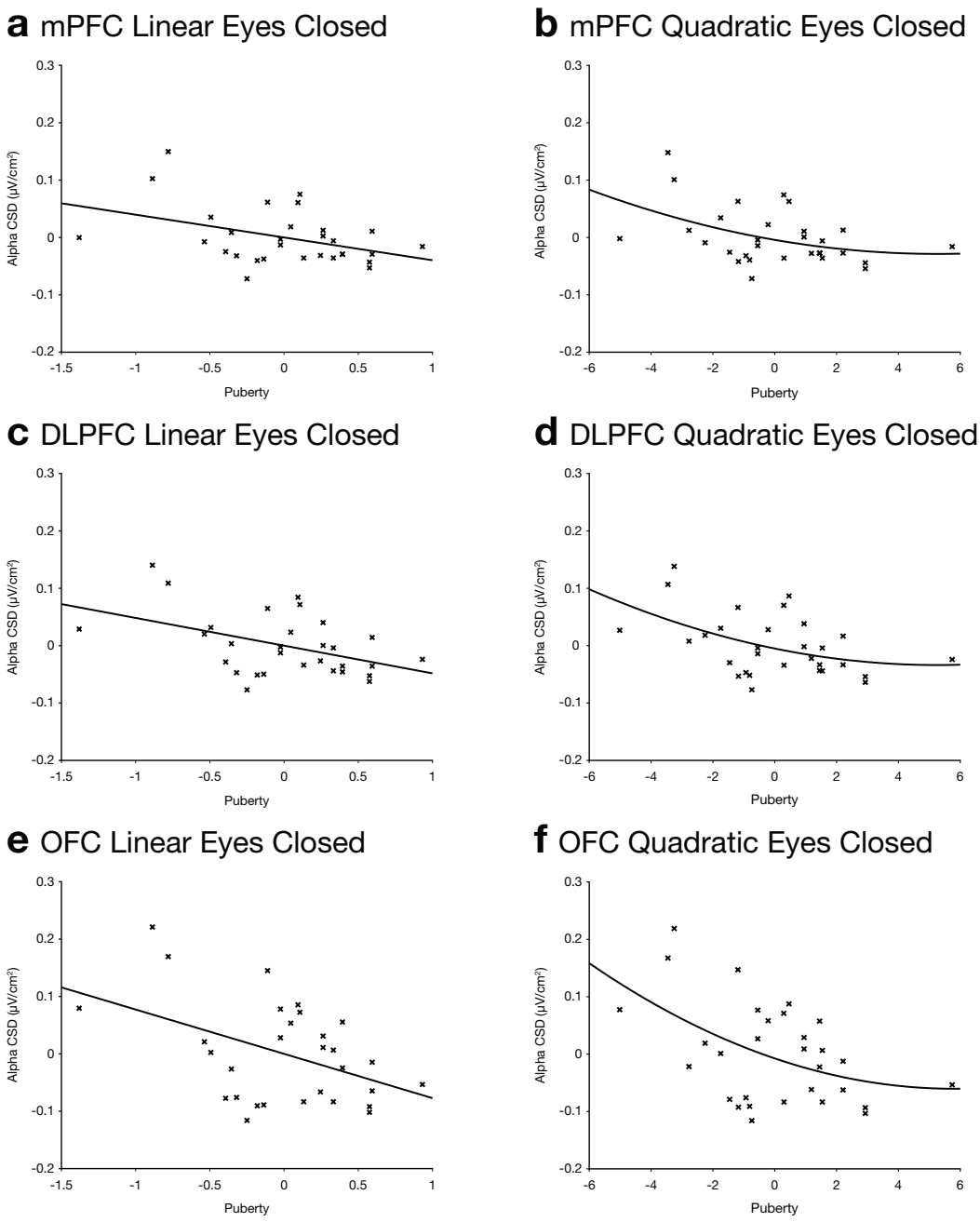

Figure 5.7 Partial regression plots of the residuals for the linear and quadratic relationships between puberty and alpha CSD in the mPFC (**a**, **b**), DLPFC (**c**, **d**), and OFC (**e**, **f**) during the eyes-closed condition controlling for chronological age for male adolescents aged 9-17 years.

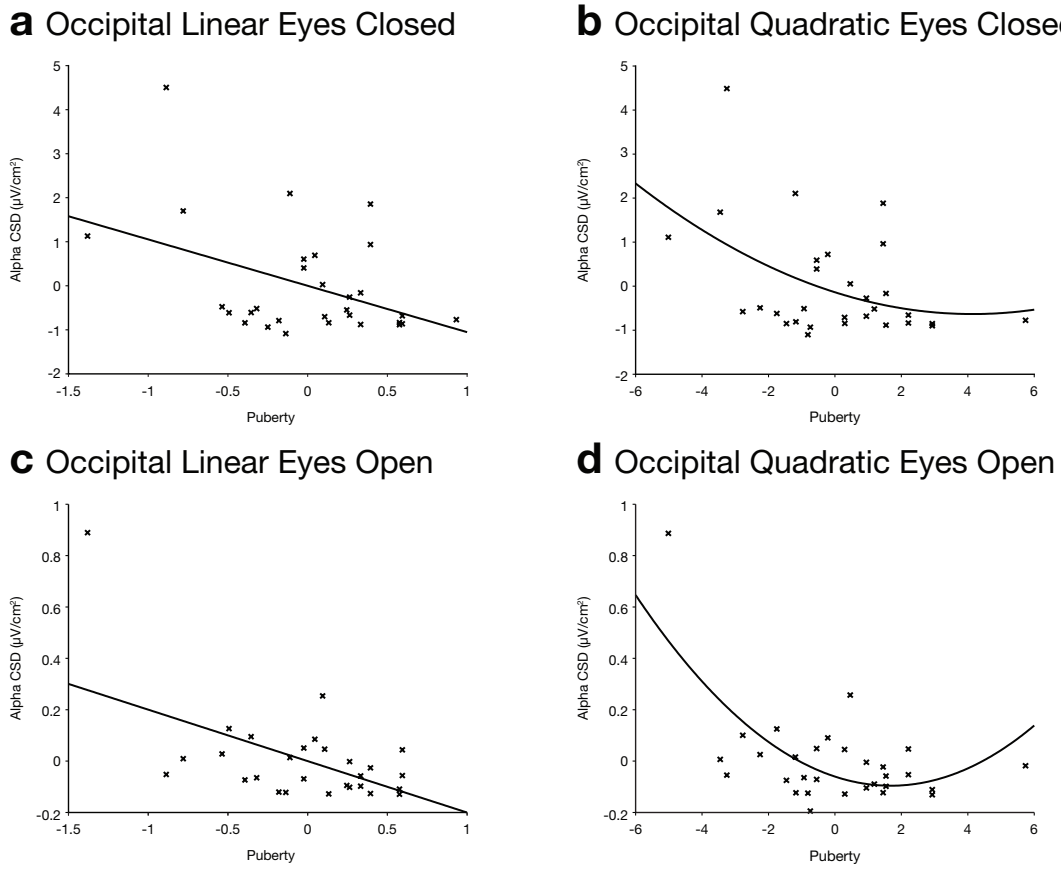

Figure 5.8 Partial regression plots of the residuals for the linear and quadratic relationships between puberty and alpha CSD in the occipital cortex during the eyes-closed condition (**a**, **b**) and eyes-open condition (**c**, **d**) controlling for chronological age for male adolescents aged 9-17 years.

5.3.3. Frontal alpha asymmetry

Frontal cortical asymmetry, as indexed by spontaneous alpha, is thought to reflect lateralisation of the approach-avoidance systems, whereby greater left frontal cortical activity reflects approach-related processes and greater right frontal cortical activity reflects avoidance-related processes. Risk-taking behaviours, as measured by the YRBSS and BART, increased throughout adolescence in this sample (Chapter 3). Therefore, the final aim of this study was to explore whether frontal asymmetry could account for the developmental differences in risk-taking behaviours in this sample of adolescents. Frontal alpha asymmetry was examined using the frontal ROIs (mPFC, DLPFC, OFC) and frontal scalp electrodes (F4-F3, F8-F7). This section examines the age- and gender-related differences in frontal asymmetry (Chapter 5.3.3.1), and

relationships between frontal asymmetry and risk-taking behaviours (Chapter 5.3.3.2).

5.3.3.1. Age- and gender-related differences in frontal alpha asymmetry

Mixed-design ANOVAs were used to examine age- and gender-related differences in frontal asymmetry during the eyes-open and eyes-closed conditions. Separate ANOVAs were conducted for each ROI (mPFC, DLPFC, OFC) and electrode pair (F4-F3, F8-F7) with *Condition* (eyes-open, eyes-closed) as the within-group factor, and *Age Group* (preadolescents, mid-adolescents, late adolescents) and *Gender* (females, males) as the between-group factors. ANOVA pairwise comparisons were corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5).

5.3.3.1.1. Frontal alpha asymmetry: ROIs

Figure 5.9 displays the group means and bootstrapped 95% confidence intervals for alpha asymmetry in the mPFC, DLPFC, OFC during the eyes-open and eyes-closed conditions. No main or interaction effects were found for the frontal alpha asymmetry scores computed using the ROIs (mPFC, DLPFC, OFC). These findings therefore suggest that levels of asymmetry in the frontal ROIs were comparable across groups.

5.3.3.1.2. Frontal alpha asymmetry: scalp electrodes

Figure 5.10 displays the group means and bootstrapped 95% confidence intervals for alpha asymmetry scores for scalp electrodes F4-F3 and F8-F7 during the eyes-open and eyes-closed conditions. No main or interaction effects were found for the frontal alpha asymmetry scores computed using the scalp electrodes (F4-F3, F8-F7). These findings are consistent with the ROI analysis (Chapter 5.3.3.1.1), and suggest that levels of asymmetry in the scalp electrodes were comparable across groups.

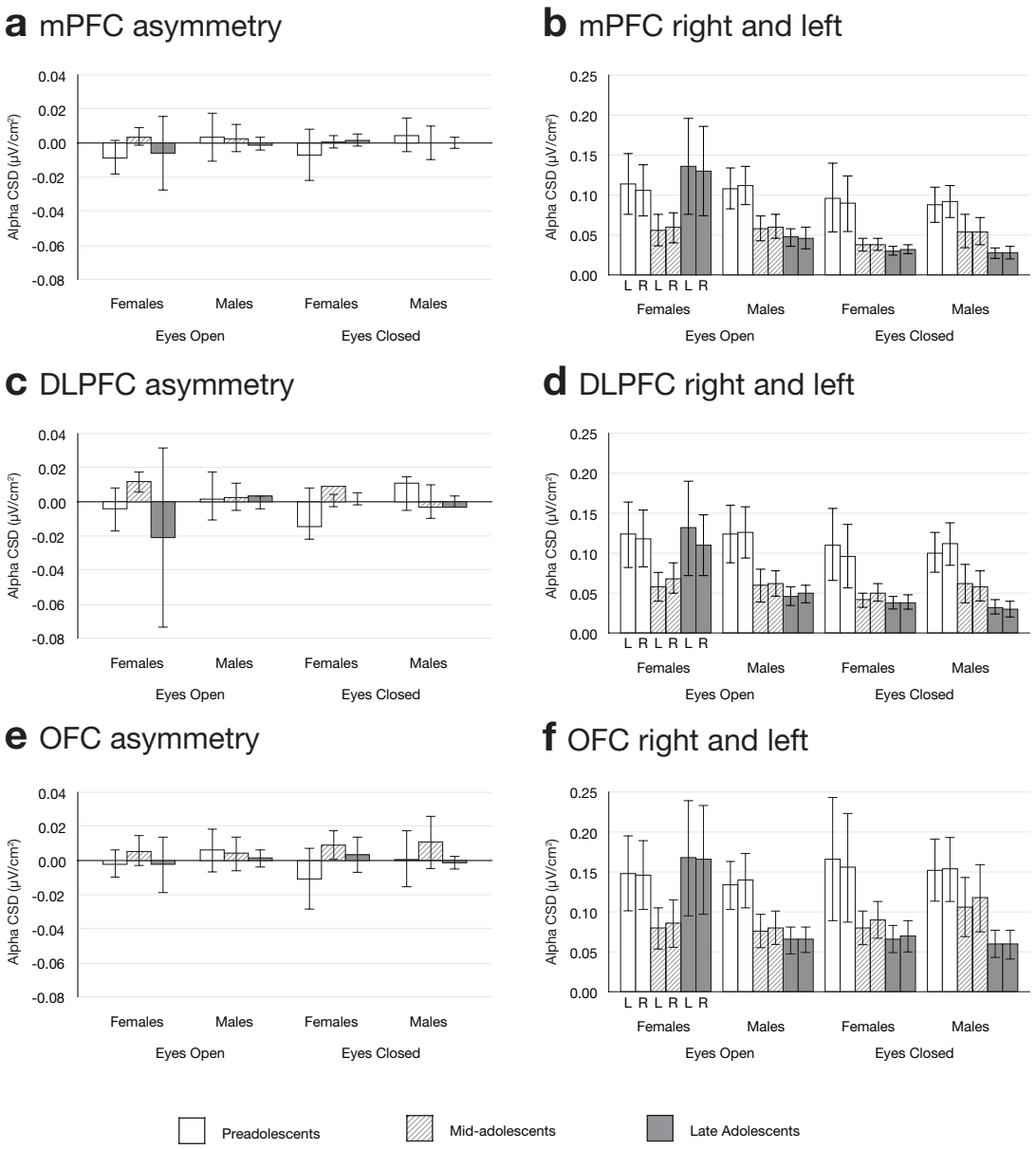

Figure 5.9 Group means for alpha CSD asymmetry and alpha CSD in the right and left hemispheres in the mPFC (**a**, **b**), DLPFC (**c**, **d**), and OFC (**e**, **f**) for the eyes-open and eyes-closed conditions. Error bars represent 95% bootstrapped confidence intervals.

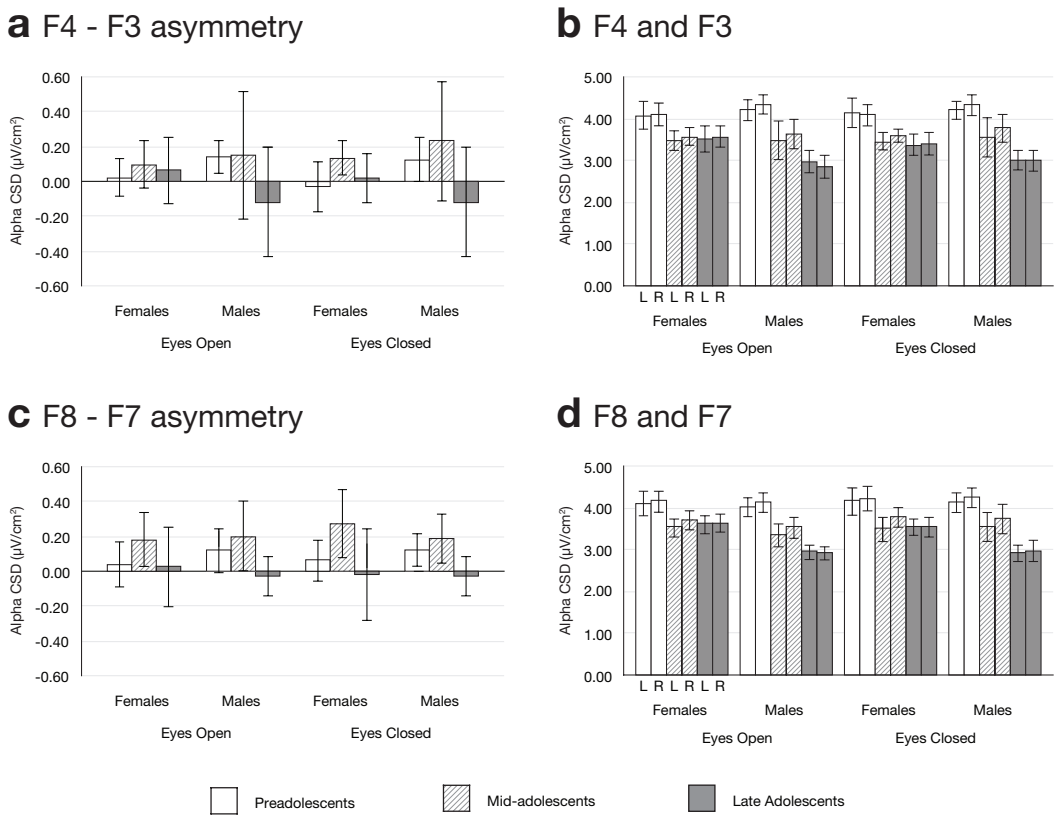

Figure 5.10 Group means for alpha CSD asymmetry and alpha CSD in the right and left hemisphere for scalp electrode pairs F4-F3 (**a**, **b**) and F8-F7 (**c**, **d**) during the eyes-open and eyes-closed conditions. Error bars represent 95% bootstrapped confidence intervals.

5.3.3.2. Relationships between frontal alpha asymmetry and risk-taking behaviours

Next, relationships between frontal alpha asymmetry and risk-taking behaviours were explored. Risk-taking behaviours were measured using the BART and YRBSS (Chapter 3). The BART yields three dependent measures: the average number of adjusted pumps for unpopped balloons; the total number of points won on the task; and the total number of popped balloons. By contrast, the YRBSS yields one dependent measure that reflects participants' recent engagement in a range of risky behaviours. Higher BART and YRBSS scores reflect greater levels of risk-taking (see Chapter 2.3.1). Correlations were conducted separately for age groups and genders, and were bootstrapped and corrected for multiple comparisons using the B-H procedure (Benjamini & Hochberg, 1995; see Chapter 2.5).

5.3.3.2.1. Frontal alpha asymmetry and risk-taking: ROIs

The Pearson correlation coefficients between ROI frontal alpha asymmetry scores (mPFC, DLPFC, OFC) and participants' risk-taking behaviours (BART, YRBSS) are presented in Supplementary Table 5.4 (Appendix 3). The group means and bootstrapped 95% confidence intervals for the ROI frontal asymmetry scores are presented in Figure 5.9. In summary, significant correlations were found between ROI frontal alpha asymmetry scores and risk-taking behaviours for female and male mid-adolescents and male late adolescents. By comparison, no correlations were found between ROI frontal alpha asymmetry scores and risk-taking behaviours for female or male preadolescents or female late adolescents.

For mid-adolescent females, DLPFC alpha asymmetry was positively correlated with YRBSS risk-taking during the eyes-open condition (r = 0.63, p = 0.016) and with the total number of points won on the BART during the eyes-closed condition (r = 0.57, p = 0.032). Together, these findings support the study predictions and previous work (Black *et al*., 2014), and suggest that greater YRBSS and BART risk-taking was associated with relatively more left frontal cortical activity in the DLPFC in mid-adolescent females.

Consistent with mid-adolescent females, risk-taking behaviours were also positively associated with relatively more left frontal cortical activity for mid-adolescent males. Specifically, OFC alpha asymmetry was positively correlated with the total number of points won on the BART during the eyes-open condition (r = 0.57, p = 0.027). This finding is consistent with the study predictions and previous work (Black *et al*., 2014), and suggests that greater BART risk-taking was associated with relatively more left frontal cortical activity in the OFC in mid-adolescent males.

In contrast to mid-adolescent females and males, for late adolescent males, DLPFC alpha asymmetry was negatively correlated with YRBSS risk-taking during the eyes-open condition (r = -0.57, p = 0.023). This finding is contrary to predictions and previous work (Black *et al*., 2014), and suggests that greater YRBSS risk-taking was associated with relatively more right frontal cortical activity in the DLPFC in late adolescent males.

5.3.3.2.2. Frontal alpha asymmetry and risk-taking: scalp electrodes

In order to compare this study to the existing literature, frontal asymmetry was also assessed using the most widely used scalp electrode pairs, F4-F3 and F8-F7 (Black

et al., 2014; Harmon-Jones *et al*., 2008; Santesso *et al*., 2008; Schutter *et al*., 2004). The Pearson correlation coefficients between scalp frontal asymmetry (F4-F3, F8-F7) and participants' risk-taking behaviours (BART, YRBSS) are presented in Supplementary Table 5.5 (Appendix 3). The group means and bootstrapped 95% confidence intervals for the scalp frontal asymmetry scores are presented in Figure 5.10. Contrary to previous studies (e.g., Black *et al*., 2014; Santesso *et al*., 2008) and the study predictions, no correlations were found between the frontal asymmetry scores using the scalp electrodes (F4-F3, F8-F7) and risk-taking behaviours for any group.

5.4. Discussion

This study had three aims. The first aim of this study was to identify how the cortical sources of spontaneous alpha during eyes-open and eyes-closed conditions change throughout adolescence for males and females. In order to account for the potential affects of puberty on the development of spontaneous alpha, the second aim of this study was to assess the relationship between pubertal stage and spontaneous alpha in male and female adolescents aged 9-17 years. Finally, frontal alpha asymmetry is thought to reflect lateralisation of the approach-avoidance systems in adults (Davidson, 1984, 1992). However, whether the same is true for adolescents remains unclear. Since Chapter 3 revealed significant age- and gender-related differences in risk-taking behaviours in this sample of adolescents, the third and final aim of this study was to explore whether frontal alpha asymmetry could account for the developmental differences in risk-taking observed in this sample. To these ends, preadolescents aged 9-12 years, mid-adolescents aged 13-17 years, and late adolescents aged 18-23 years had their resting brain activity measured using EEG during eyes-open and eyes-closed conditions. Overall, the findings revealed that the cortical sources of alpha changed considerably during the course of adolescence, and that more advanced pubertal development was associated with reduced levels of alpha CSD in males aged 9-17 years. Unexpectedly however, frontal asymmetry was found to not be a reliable marker of risk-taking in this sample of adolescents.

Alpha CSD was examined in four ROIs: mPFC, DLPFC, OFC and occipital cortex. The mPFC, DLPFC, and OFC were selected for two reasons. First, several studies have reported that frontal regions are involved in the generation of spontaneous alpha in adults (Cuspineda *et al*., 2009; De Munck *et al*., 2007; Goldman *et al*., 2002; Feige *et al*., 2005; Lüchinger *et al*., 2011; Wu *et al*., 2010). The human brain

matures in a back-to-front order, with prefrontal regions developing last. Thus, compared to other cortical and subcortical regions, the PFC has a protracted development across adolescence (Gotgay *et al*., 2004; Mills *et al*., 2014). The PFC is therefore a good candidate for examining potential age-related differences in the cortical sources of spontaneous alpha during adolescence. Second, frontal alpha asymmetry has been widely implicated in the approach-avoidance systems, whereby relatively greater left frontal cortical activity is associated with the approach system and relatively greater right frontal cortical activity is associated with the avoidance system (Coan & Allen, 2004; Davidson, 1984, 1992; Harmon-Jones *et al*., 2010; Tomarken *et al*., 1990; Tomarken *et al*., 1992; Wheeler *et al*., 1993). Compared to the frontal ROIs, the occipital cortex was selected as a ROI because it is thought to be the primary cortical generator of the alpha rhythm in adults (Cuspineda *et al*., 2009; De Munck *et al*., 2007; Feige *et al*., 2005; Goldman *et al.*, 2002; Lüchinger *et al*., 2011; Moosmann *et al*., 2003). Hence, examining the development of occipital alpha provided important insights into how the primary cortical generator of spontaneous alpha develops across adolescence. This discussion is organised in accordance with the study aims.

5.4.1. Cortical sources of spontaneous alpha throughout adolescence

The first aim of this study was to examine the cortical sources of spontaneous alpha in frontal and occipital ROIs during eyes-open and eyes-closed conditions in male and female preadolescents, mid-adolescents, and late adolescents. As predicted, developmental reductions in alpha CSD were observed in all ROIs; preadolescents had more alpha CSD in the mPFC, DLPFC, OFC, and occipital cortex compared to both mid-adolescents and late adolescents, irrespective of condition. No differences in alpha CSD were found between mid-adolescents and late adolescents, suggesting that frontal and occipital alpha CSD decreased from preadolescence to mid-adolescence, but remained largely stable across middle and late adolescence. These findings support previous studies showing that alpha power decreases across all areas of the scalp during childhood and adolescence (Chiang *et al*., 2011; Dustman *et al*., 1999; Yordanova & Kolev, 1997). Such decreases in cortical alpha power are thought to reflect the reduction of cortical grey matter and augmentation of white matter during adolescence (Segalowitz *et al*., 2010; Whitford *et al*., 2007).

Critically, considerable differences in the cortical sources of spontaneous alpha

were found between the eyes-open and eyes-closed conditions for all groups. The sLORETA maps revealed that the sources of alpha were primarily localised to occipital, parietal, and occipito-temporal regions in the eyes-closed condition (Figure 5.5). In comparison to the eyes-closed condition, the sLORETA maps revealed that the sources of alpha were more widespread in the eyes-open condition for all groups, and included occipital, parietal, temporal, and frontal regions (Figure 5.4). The ANOVAs for the ROIs also revealed significant differences between the eyes-closed and eyes-open conditions; there was significantly more alpha CSD in the occipital cortex during the eyes-closed condition, and significantly more alpha CSD in the mPFC and DLPFC during the eyes-open condition for all groups.

The finding that the occipital and parietal cortices were the primary cortical sources of spontaneous alpha during the eyes-closed condition for all groups is highly consistent with previous studies (Goldman *et al*., 2002; Lüchinger *et al*., 2011; Moosmann *et al*., 2003). Substantially less work has examined the differences in the cortical sources of alpha between eyes-open and eyes-closed conditions. Despite this, the findings from this study are largely consistent with previous work; the current study found that alpha CSD was significantly greater in occipital cortical regions during the eyes-closed condition compared to the eyes-open condition (Wu *et al*., 2010), and that the cortical generators of alpha were more widespread during the eyes-open condition compared to the eyes-closed condition (Lüchinger *et al*., 2011).

Interestingly, developmental differences were found in frontal alpha CSD during the eyes-open condition. Specifically, late adolescents had more alpha CSD in the mPFC and OFC during the eyes-open condition compared to the eyes-closed condition. By contrast, alpha CSD was equivalent across the eyes-open and eyes-closed conditions in the mPFC and OFC for preadolescents and mid-adolescents.

Alpha activity is inversely related to cortical activity (Haegens *et al.,* 2011; Shagass, 1972), and a number of studies have reported that alpha synchronises (increases) in task irrelevant areas and desynchronises (decreases) in task relevant areas (Klimesch *et al*., 1997). Hence, current theories suggest that alpha has an active role in inhibiting irrelevant brain activity to facilitate more efficient neural processing (Foxe & Snyder, 2011; Jensen *et al*., 2012; Klimesch, 1999; Klimesch *et al*., 2007; Palva & Palva, 2007; see Chapter 5.1.3). Thus, the finding that late adolescents had greater levels of alpha CSD in the PFC than preadolescents and mid-adolescents when their eyes were open may reflect that late adolescents had greater levels of inhibition in the

PFC. Notably, the PFC has a protracted development across childhood and adolescence that extends into the third decade of life (Gotgay *et al*., 2004; Mills *et al*., 2014). The structural development of the PFC is thought to underlie age-related changes in PFC function; fMRI studies have consistently reported that PFC activity during executive tasks reduces and becomes more focal as the PFC structurally develops (Casey *et al*., 1997; see Chapter 1.5.1). It is therefore possible to speculate that a greater level of alpha in the PFC during late adolescence and adulthood is one mechanism underlying more efficient neural processing, and may therefore contribute to the improvements in cognitive and memory processes observed throughout childhood and adolescence. In support of this idea, late adolescents performed better than preadolescents on the Go/NoGo task, a measure of executive function and response inhibition, (Chapter 3) and previous studies have reported that executive functions that are reliant on the PFC develop throughout adolescence and into late adolescence/young adulthood (Casey *et al*., 2010; Blakemore & Choudhury, 2006; Steinberg *et al*., 2008). However, future work is needed to establish the role of frontal alpha activity in the development of cognitive and memory processes throughout childhood and adolescence.

Compared to the mPFC and OFC, alpha CSD in the DLPFC was not significantly greater in the eyes-open condition compared to the eyes-closed condition for late adolescents. Despite this, there was some evidence that the DLPFC showed the same developmental trend as the mPFC and OFC (see Figure 5.6). The DLPFC is one of the final brain structures to mature and may not reach full maturity until the end of the third decade of life (Huttenlocher, 1979; Petanjek *et al*., 2011; Sowell *et al*., 1999, 2001). Thus, it is feasible that alpha CSD in the DLPFC was not greater in late adolescents during the eyes-open condition compared to the eyes-closed condition because the DLPFC may have been relatively less mature than the mPFC and OFC due to its protracted development.

For the first time, this study assessed the cortical sources of alpha during eyes-open and eyes-closed conditions in a sample of preadolescents, mid-adolescents, and late adolescents. The findings from this study therefore give preliminary insights into how the cortical sources of alpha change throughout adolescence. The only other study to compare the sources of spontaneous alpha between adolescents and adults reported no age-related differences (Lüchinger *et al*., 2011). In contrast, the current study found significant developmental differences in the frontal sources of alpha. Importantly, there are two key differences between the current study and Lüchinger *et*

al.'s (2011) study that may account for the discrepancy in findings; Lüchinger *et al*. (2011) did not explore potential gender differences and only included adolescents aged 15 years and adults aged 25 years. By comparison, the current study examined age-related and gender-related differences in adolescents aged 9-23 years.

Notably, while the current study reported significant age differences in the cortical sources of alpha, no significant gender differences were found. Despite this, the sLORETA maps indicated that there were differences between males and females in the cortical sources of alpha. In particular, for occipital alpha CSD in the eyes-closed condition, mid-adolescent males more strongly resembled preadolescents whereas mid-adolescent females more strongly resembled late adolescents (see Figure 5.6). Moreover, the age-related changes in mPFC and OFC alpha CSD in late adolescents appeared to be driven by females (see Figure 5.6). These findings are consistent with what is currently known about brain development during adolescence, whereby females mature slightly faster than males (Lenroot & Giedd, 2010; see Chapter 1.4). Finally, Figure 5.6 also suggested that there was a u-shaped developmental trajectory for frontal alpha CSD during the eyes open condition for females but not males; frontal alpha CSD appeared to reduce from preadolescence to mid-adolescence and subsequently increase from mid-adolescence to late adolescence. It is possible that the lack of statistical significance for such gender-related differences reflects high levels of variability in this sample. High levels of variability in resting state studies is thought to result from participants engaging in a range of internal processes, such as spontaneous thoughts, memory retrieval, future planning, or daydreaming (Fox, Spreng, Ellamil, Andrews-Hanna & Christoff, 2015).

5.4.2. The influence of puberty on the development of spontaneous alpha

Recent work has shown that puberty has significant affects on the development of cortical and subcortical grey matter during adolescence that are independent of chronological age (Bramen *et al*., 2011; Goddings *et al*., 2014; Neufang *et al*., 2009). Since the alpha rhythm is generated by cortical and subcortical grey matter structures (Da Silva *et al*., 1980; De Munck *et al*., 2007; Feige *et al*., 2005; Goldman *et al*., 2002; Lüchinger *et al*., 2011; Moosmann *et al*., 2003; Wu *et al*., 2010), it is possible that the influence puberty has on grey matter development also affects the development of spontaneous alpha. Despite this, no study to date has examined the influence of puberty on the development of spontaneous alpha. Accordingly, the second aim of this

study was to examine the relationships between pubertal stage and spontaneous alpha in frontal and occipital ROIs in male and female adolescents aged 9-17 years old while controlling for chronological age. Since the development of many behaviours and brain structures are nonlinear, this study examined the linear and nonlinear relationships between alpha and pubertal development.

Interestingly, relationships between spontaneous alpha and pubertal stage were only observed for males. For males, both the linear and quadratic terms revealed that more advanced pubertal stage was associated with less alpha CSD in the frontal ROIs during the eyes-closed condition (Figure 5.7) and in the occipital ROI during the eyes-open and eyes-closed conditions (Figure 5.8). Puberty accounted for between 14.7 and 26.8 per cent of the variance in alpha CSD, suggesting that pubertal stage had a considerable influence on the development of frontal and occipital alpha in this sample of adolescent males.

Given that the relationships were found only in males, it is possible to speculate that the relationships between puberty and spontaneous alpha found in this study resulted from puberty-specific changes in testosterone levels. Several studies have reported that testosterone levels are associated with the development of cortical grey matter in adolescent males (see Chapter 1.4). Notably, Neufang *et al*. (2009) found that testosterone levels were negatively associated with grey matter volume in the parietal cortex in males aged 8-15 years. The parietal cortex is thought to be one of the primary cortical generators of spontaneous alpha during eyes-open and eyes-closed conditions (Cuspineda *et al*., 2009; De Munck *et al*., 2007; Feige *et al*., 2005; *et al*., 2011; Moosmann *et al*., 2003). Consistently, the sLORETA maps in the current study showed that, for all groups, the parietal-occipital cortices were the primary cortical generator of alpha in the eyes-closed condition (Figure 5.5), and were one of the cortical generators in the eyes-open condition (Figure 5.4). While the current study measured pubertal stage instead of testosterone levels, studies have shown that testosterone levels increase with advancing pubertal stage (Biro, Lucky, Huster & Morrison, 1995; Shirtcliff *et al*., 2009). Moreover, spontaneous alpha is thought to be generated by cortico-cortical neural networks, in addition to cortico-thalamic networks (Da Silva *et al*., 1980). Thus, it is possible that the affect of testosterone on parietal grey matter (Neufang *et al*., 2009) may partially account for the findings reported in the current study, whereby more advanced pubertal stage was associated with less alpha in frontal and occipital cortical regions.

A number of studies have also reported that puberty has an affect on the development of subcortical grey matter in males; more advanced pubertal stage in males has been positively associated with grey matter volume in the amygdala and hippocampus (Bramen *et al*., 2011, Goddings *et al*., 2014) and negatively associated with grey matter volume in the nucleus accumbens, caudate, putamen, and globus pallidus (Goddings *et al*., 2014). However, it is currently unknown whether testosterone and pubertal stage have significant affects on the structural development of the thalamus. The thalamus is a subcortical structure critical for the generation of the alpha rhythm (Da Silva & Van Leeuwen, 1977; Steriade et al., 1990), and is therefore a likely candidate underlying the relationship between puberty and alpha activity found in the current study. Future work is therefore needed to examine the relationship between puberty and the structural development of the thalamus, as well as between puberty and the generation of spontaneous alpha originating from the thalamus.

5.4.3. Frontal alpha asymmetry: relationships with risk-taking

There is a highly influential biphasic motivational theory proposing that frontal cortical activity, as indexed by spontaneous alpha, in the left and right hemispheres reflects lateralisation of approach and avoidance processes, respectively (Davidson, 1984, 1992). While there is considerable evidence for frontal lateralisation in adults, very little work has been conducted in adolescents. The study reported in Chapter 3 revealed that risk-taking behaviours, but not anxiety levels, increased during the course of adolescence. Accordingly, the final aim of the current study was to explore the development of frontal alpha asymmetry and its relationship to risk-taking behaviours during preadolescence, mid-adolescence, and late adolescence to investigate whether frontal asymmetry could account for the developmental differences in risk-taking found in this sample of adolescents.

In contrast to the study predictions and previous work (Black *et al*., 2014; Coan & Allen, 2003; Santesso *et al*., 2008; Sutton & Davidson, 1997; Wheeler *et al*., 1993), no relationships between frontal alpha asymmetry and risk-taking behaviours were found for the scalp electrodes for any group. This is surprising since there has been considerable empirical support in 18-25 years that greater left frontal cortical activity is associated with approach sensitivity and approach-related behaviours, such as sensation seeking and risk-taking behaviours (Coan & Allen, 2003; Santesso *et al*.,

2008; Sutton & Davidson, 1997; Wheeler *et al*., 1993).

Compared to the scalp asymmetry scores, several relationships emerged between frontal alpha asymmetry and risk-taking behaviours for the frontal ROIs. However, while the relationships between frontal asymmetry and risk-taking were in the expected direction for mid-adolescent females and males, the pattern of findings were sparse and unsystematic. In particular, for mid-adolescent females, DLPFC alpha CSD was positively associated with YRBSS risk-taking in the eyes-open condition and BART risk-taking in the eyes-closed condition. In comparison, for mid-adolescent males, OFC alpha CSD was positively associated with BART risk-taking in the eyes-open condition. In contrast to mid-adolescent females and males, the relationships for the late adolescent males were in the unexpected direction, whereby YRBSS risk-taking was associated with relatively greater right DLPFC activity during the eyes-open condition. Moreover, no relationships between frontal asymmetry and risk-taking were observed for late adolescent females, or preadolescents. Overall, these findings do not reveal a clear pattern of relationships between frontal asymmetry and risk-taking behaviours. Moreover, the relationships that did emerge do not correspond to the development risk-taking behaviours found in this sample of adolescents; YRBSS risk-taking increased from preadolescence to mid-adolescence for males and females, and from mid-adolescence to late adolescence for females. BART risk-taking also increased from mid-adolescence to late adolescence for both males and females (Chapter 3).

Notably, the only study to assess relationships between frontal asymmetry and the approach system in adolescents recruited individuals who had higher or lower than average sensitivity to reward (Black *et al*., 2014). By contrast, participants in the current study were not recruited on the basis of reward sensitivity. Thus, it is feasible that the discrepancy between the current study and Black *et al.*'s (2014) study results from differences in adolescents' sensitivity to reward. It is possible that exploring frontal asymmetry in adolescents who are highly sensitive to rewards or in adolescents who engage in higher than average levels of risk-taking will provide more useful insights into frontal asymmetry as a potential mechanism underlying risk-taking behaviours during adolescence.

The neurobiological models of adolescence assert that adolescent risk-taking behaviour is driven by a hyperresponsive approach reward-based system (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008). Moreover, the Triadic Model proposes that adolescents also have a hyporesponsive avoidance threat-based

system (Ernst *et al*., 2006). Hence, if frontal alpha asymmetry reflects the approach-avoidance systems (Davidson, 1984, 1992), it should be possible to test these assertions by exploring age-related changes in frontal alpha asymmetry. However, this study found no significant age-related changes in frontal alpha asymmetry. There was some evidence of greater left hemisphere activity in mid-adolescents, particularly at electrodes F8-F7 (Figure 5.10), but this effect did not reach statistical significance. Thus, the frontal asymmetry findings from the current study cannot shed light on the relative dominance of the approach or avoidance systems, and their relationship to risk-taking behaviours, during adolescence.

The development of frontal alpha asymmetry, and its relationship to risk-taking behaviours was assessed in the current study using CSD transformed scalp data and frontal ROIs (mPFC, DLPFC, OFC). CSD transformed data attenuate the impact of distal and widely distributed brain electrical sources, and therefore provide a more spatially accurate representation of the sources underlying the EEG signal. Furthermore, CSD transformed resting state data are less influenced by uncontrolled circadian and seasonal factors compared to traditional electrodes. Thus, CSD transformed alpha asymmetry data compared to referenced alpha asymmetry data are more likely to reflect neural activity than extraneous factors (Velo *et al*., 2012). Although CSD transformed scalp data provide a more accurate spatial representation of the sources underlying the EEG signal, the sources of EEG activity remain unknown. Therefore, the current study also examined frontal alpha asymmetry using source localised ROIs in order to provide more comprehensive investigation into the potential structures underlying scalp-recorded frontal alpha asymmetry. Finally, the current study examined frontal alpha asymmetry during eyes-open and eyes-closed conditions given that there are considerable differences in the sources of alpha between eyes-open and eyes-closed conditions (Feige *et al*., 2005; Lüchinger *et al*., 2011; Wu *et al*., 2010), and opening the eyes reduces global alpha power and increases skin conductance levels (Barry *et al*., 2007; Barry *et al*., 2009). Thus, the current study employed novel methods to explore the development of frontal alpha asymmetry and its relationship to risk-taking behaviours. For the reasons outlined above, future work should aim to investigate frontal alpha asymmetry using CSD transformed scalp data and source localised ROIs during eyes-open and eyes-closed conditions.

5.4.4. Study limitations

The findings reported in this study provide novel insights into the cortical sources of spontaneous alpha, and their relationship to pubertal development and risk-taking behaviours, during adolescence. However, there are a number of limitations of the current study that need to be considered. Firstly, the sLORETA solution space is limited to cortical grey matter and is therefore unable to examine subcortical structures, such as the thalamus. Moreover, this study selected frontal and occipital cortical ROIs to examine the development of alpha during adolescence. However, the sLORETA maps indicated that parietal and temporal cortical regions might also be involved in the generation of spontaneous alpha. Thus, this study is limited in its capacity to examine all the potential sources of alpha throughout adolescence. Future work should therefore aim to examine the cortical and subcortical generators of alpha in adolescents using combined EEG-fMRI to provide a greater understanding of how both the cortical and subcortical sources of alpha develop during the course of adolescence.

Secondly, participants in resting state studies are instructed to relax with their eyes open or closed. It is therefore plausible that the participants in this study were engaging in any one of a number of internal processes, including spontaneous thoughts, future planning, memory retrieval, or daydreaming (Fox *et al*., 2015). Hence, the cortical sources associated with resting state activity may reflect a number of different processes. Indeed, the high levels of variability in this study may reflect that preadolescents, mid-adolescents, and late adolescents were engaging in a variety of different internal processes. Future work should therefore aim to tease out the cortical and subcortical sources of alpha associated with different internal processes. One approach would be to measure alpha while participants engage in a series of internal processes, such as memory retrieval and future planning. Critically however, younger participants may not have the capabilities to engage in such internally directed thoughts, and there is no overt, reliable measure to determine which internal process participants engaged in. Event-related studies may therefore provide a more accurate way of assessing the cortical and subcortical sources of alpha involved in distinct internal processes.

5.4.5. Conclusion

The current findings go beyond that of previous studies by exploring the age-

and gender-related differences in the cortical sources of spontaneous alpha in a sample of typically developing adolescents, and their relation to puberty and risk-taking behaviours. Moreover, in contrast to the vast majority of previous studies, the current study examined differences in alpha CSD between eyes-open and eyes-closed conditions. Overall, the current study demonstrates that there are considerable differences in the cortical sources of alpha during eyes-open and eyes-closed conditions, and that the cortical sources of alpha change significantly during the course of adolescence. The current study also revealed that there are gender-specific affects of puberty on the development of spontaneous alpha. Finally, the current study did not find frontal asymmetry to be a reliable marker of risk-taking in a sample of typically developing adolescents.

The majority of previous studies examining the cortical and subcortical sources of spontaneous alpha, and the relationship between frontal alpha asymmetry and approach- and avoidance-related behaviours have largely focused on the adult brain. Notably, Başar (2012) has suggested that a comprehensive understanding of alpha can be only achieved by investigating alpha in maturing, evolving, emotional, and pathological brains. The current study therefore provides an important step towards understanding the development and function of spontaneous alpha in the typically developing brain. Nonetheless, a great deal more research needs to be conducted before we have a complete understanding of the development and function of alpha in the maturing brain, and the mechanisms through which alpha influences cognitive and memory processes.

Chapter 6

Summary and general discussion

6.1. Introduction

This chapter first outlines the main findings from this doctoral research and discusses how this doctoral work extends the existing literature (Chapter 6.2). This chapter subsequently proposes directions for future research (Chapter 6.3) and draws final conclusions (Chapter 6.4).

6.2. Summary and general discussion

There is a large body of work suggesting that adolescents take more risks and experience greater levels of anxiety than children and adults (Abe & Suzuki, 1986; Burnett *et al*., 2010; Spear, 2000; Steinberg *et al*., 2008). Critically, high levels of risk-taking and anxiety during adolescence are associated with numerous short- and long-term adverse outcomes (Dahl, 2004; Pine *et al*., 1998; Woodward & Fergusson, 2001), and thus the mechanisms underlying adolescent risk-taking and anxiety warrant investigation. The primary aim of this thesis was therefore to investigate the simultaneous increase in risk-taking behaviours and anxiety levels during adolescence. Specifically, this doctoral work aimed to examine how the relationship between risk-taking and anxiety changes across the course of adolescence (Chapter 3), and whether age-related changes in the EEG correlates of the approach-avoidance systems can account for the development of risk-taking and anxiety during adolescence (Chapters 4 and 5). Notably, the same cohort of adolescents (N = 105) took part in all the studies in this doctoral work so that the simultaneous increase in risk-taking and anxiety could be examined from multiple behavioural and EEG perspectives. Participants were categorised into three age groups: preadolescence (9-12 years), mid-adolescence (13-17 years), and late adolescence (18-23 years), and took part in the following studies:

- Gender differences in the relationships between risk-taking and anxiety during preadolescence, mid-adolescence, and late adolescence (Chapter 3).
- Anticipatory ERP responses to rewarding and threatening outcomes during preadolescence, mid-adolescence, and late adolescence (Chapter 4).
- The development of spontaneous alpha and its relationship to risk-taking behaviours during preadolescence, mid-adolescence, and late adolescence (Chapter 5).

A number of neurobiological models have been proposed to explain the

increases in risk-taking behaviours during adolescence (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008). Casey's dual systems model (Casey *et al*., 2010) also accounts for the increases in emotionality, i.e., greater levels of anxiety and negative affect, during adolescence. While these models have considerably improved our understanding of why risk-taking behaviours and anxiety levels increase during adolescence, the neurobiological models are limited in a number of ways. First, while Casey's dual systems model (Casey *et al*., 2010) and the Triadic Model (Ernst *et al*., 2006) focus on adolescents' sensitivity to reward and threat, Steinberg's dual systems model (DSM; Steinberg, 2008) and the Social Information Processing Network (SIPN; Nelson *et al*., 2005) only focus on sensitivity to reward. Second, despite adolescence being a transitional period between childhood and adulthood (Casey *et al*., 2008a; Spear, 2000), the Triadic Model and SIPN only account for changes between adolescence and adulthood. Third, most of the evidence testing the neurobiological models has resulted from fMRI and nonhuman animal studies. Fourth, the neurobiological models only implicate cortical prefrontal and subcortical limbic structures in adolescent development. Finally, all the neurobiological models overlook potential gender differences in adolescent development. The studies in this doctoral work were therefore designed to address these limitations. The following sections discuss how this doctoral work addressed these limitations, as well as the potential directions for future work.

6.2.1. The approach-avoidance systems during adolescence

The neurobiological models of adolescence are based on the premise that behaviour is driven by an approach reward-based system and an avoidance threat-based system. The approach system is sensitive to rewarding or appetitive stimuli, and drives behaviour towards rewarding or desirable outcomes. The approach system is therefore thought to underlie sensation seeking and risk-taking behaviours. In contrast, the avoidance system is sensitive to threatening or aversive stimuli, and drives behaviour away from threatening or undesirable outcomes. The avoidance system is therefore associated with anxiety-related behaviours (Bouton *et al*., 2001; Elliot, 2006; Zuckerman & Kuhlman, 2000). All the neurobiological models (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008) assert that the increase in risk-taking behaviours during adolescence is driven by a hyperresponsive approach system and that adolescents are hypersensitive to rewards. Notably, the

Triadic Model and Casey's dual systems model also assert that the avoidance system has a pivotal role in adolescent behaviour. In particular, the Triadic Model suggests that adolescent risk-taking is driven by a hyperresponsive approach system combined with a hyporesponsive avoidance system. While the Triadic Model accounts for the increases in risk-taking, Casey's dual systems model accounts for the increases in risk-taking and emotionality during adolescence. Consequently, Casey's model proposes that adolescents have a hyperresponsive approach system and a hyperresponsive avoidance system.

While there is substantial evidence that adolescents are highly motivated by rewards and have a hyperresponsive approach system (e.g., Barkley-Levenson & Galván, 2014; Braams *et al*., 2015; Galván *et al*., 2006; Galván *et al*., 2007; Hare *et al*., 2008; Padmanabhan *et al*., 2011; Van Leijenhorst *et al*., 2010), empirical work examining the development of the avoidance system and adolescents' sensitivity to threat is sparse. Moreover, in order to investigate how the development of the approach system corresponds to the development of the avoidance system during adolescence, the approach and avoidance systems need to be examined using a within-subject design. Despite this, there are only a handful of studies assessing the development of approach and avoidance systems using a within subject design (e.g., Galván & McGlennen, 2013; Hare *et al*., 2008), and there is currently no work examining the development of the approach and avoidance systems in the same paradigm using EEG. Hence, this doctoral work used EEG to investigate the development of the approach and avoidance systems throughout adolescence (Chapters 4 and 5). Thus, this doctoral work makes a significant contribution to the existing literature by providing the first insights into how the EEG correlates of the approach-avoidance systems change throughout the adolescent period.

The second study in this doctoral work (Chapter 4) was designed to directly examine the EEG correlates of the approach-avoidance systems during three distinct stages of adolescence. This study built on our previous EEG findings showing that adolescents (12-15 years) have greater potentiation of the N170 ERP component to visual cues that predicted a threatening outcome compared to young adults (18-32 years) (Levita *et al*., 2014). Notably, this finding contradicts the suggestion that adolescents have a hyporesponsive avoidance system (Ernst *et al*., 2006). While our previous study provided important insights into the EEG correlates of the avoidance system during adolescence and young adulthood, it was unable to assess how the

avoidance system changes during the course of adolescence, and how the avoidance system compares to the approach system throughout this period.

Accordingly, this doctoral work used EEG to examine anticipatory responses to rewarding (approach system) and threatening (avoidance system) outcomes in preadolescents, mid-adolescents, and late adolescents (Chapter 4). Two ERPs that are modulated by motivationally salient stimuli, the N170 (Levita *et al*., 2014; Rellecke *et al*., 2013) and LPP (Keil *et al*., 2002; Sabatinelli *et al*., 2007; Schupp *et al*., 2000) were used to explore participants' anticipatory responses to rewards and threats. In contrast to the study hypotheses, N170 amplitudes were not modulated by stimuli that predicted rewards or threats in any group. However, in contrast to the N170, LPP amplitudes were potentiated in response to visual cues predicting rewarding and threatening outcomes compared to neutral visual cues. Significantly, the LPP revealed that anticipatory representations of the approach and avoidance systems followed different developmental trajectories during adolescence. Specifically, preadolescents showed greater LPP potentiation to visual cues predicting rewarding and threatening outcomes than both mid-adolescents and late adolescents. Moreover, mid-adolescents showed LPP potentiation to visual cues predicting rewarding outcomes, whereas late adolescents did not. In contrast, mid-adolescents and late adolescents showed comparable LPP potentiation to visual cues predicting threatening outcomes.

Together, these findings suggest that the adolescents in this sample were not hyporesponsive to threats, as predicted by Ernst's Triadic Model. Instead, these findings suggest that preadolescents and mid-adolescents were more sensitive to potential rewards than late adolescents, but all groups were sensitive to potential threats. Given that adolescents were sensitive to both rewards and threats, these findings are most consistent with Casey's neurobiological model. Moreover, since preadolescents and mid-adolescents were more sensitive to reward-related cues than late adolescents, this research is also largely consistent with Steinberg's DSM, which posits that adolescents are more sensitive to rewards than both children and adults. However, in contrast to the study predictions and neurobiological models of adolescence (Casey *et al*., 2008; Ernst *et al*., 2011; Steinberg, 2008), reward- and threat-related LPP activity was not greater in mid-adolescents compared to preadolescents and late adolescents. Hence, this doctoral research does not support the idea that reward- and threat-related brain activity peaks in middle adolescence. These findings therefore suggest that the development of reward and threat sensitivity

may be more complex than simply increasing from childhood to adolescence and decreasing from adolescence to adulthood.

It is possible that the developmental trajectory and responsivity of the approach and avoidance systems is largely dependent on the nature of the specific rewards and threats. It is therefore possible that this doctoral work did not find a peak in reward- or threat-related LPP potentiation during mid-adolescence because the reinforcer of behaviour in the instrumental task (winning and losing points) was not salient or motivating enough for mid-adolescents. fMRI studies that have reported a peak in reward- and threat-related activity in mid-adolescence have predominantly used money (e.g., Braams *et al*., 2015) or emotional faces (e.g., Hare *et al*., 2008). Future work investigating neural responses to a range of primary and secondary reinforcers in a large cohort of typically and atypically developing children, adolescents, and young adults would be extremely valuable for fMRI and EEG studies examining the developmental trajectory of reward- and threat-related neural activity (see Chapter 6.3.4).

Notably, the findings from this doctoral research are not of direct relevance to SIPN since the studies were not conducted in a social context. However, there is now strong evidence to suggest that peers have a significant influence on adolescent risk-taking behaviours and reward-related brain acitivty. Hence, future work should aim to examine how ERP correlates of approach and avoidance behaviours vary as a function of social context during the different stages of adolescence (see Chapter 6.3.3).

The approach and avoidance systems are thought to underlie risk-taking and anxiety, respectively (Cloninger, 1987; Salkovskis, 1991; Zuckerman & Kuhlman, 2000). However, the reward- and threat-related LPP potentiation in this doctoral research was not associated with measures of risk-taking or anxiety for any group. It is possible that winning and losing points was not salient enough to tap into the neural mechanisms underlying risk-taking and anxiety. Alternatively, it is possible that the measures of risk-taking and anxiety used in this doctoral research were not optimal (see Chapter 6.3.4). Thus, this doctoral research highlights the importance of considering the salience of specific rewards and threats when interpreting the developmental trajectories of the approach-avoidance systems, as well as the validity of measures currently used to assess risk taking and anxiety during different developmental stages.

Importantly, the study reported in Chapter 4 did not directly compare scalp

distributions between the reward and avoidance blocks since the reward block always preceeded the avoidance block. Hence, any comparisons made between the reward and avoidance blocks may have been confounded by potential time and fatigue effects. Thus, while this study provides some important insights into how the approach and avoidance systems develop during the course of adolescence, future work is needed to identify how the development of the reward system directly compares to the development of the avoidance system. To this end, future studies will need to design tasks that are able to compute reward x threat interactions.

The third study in this doctoral work (Chapter 5) also aimed to examine the EEG correlates of the approach-avoidance systems during adolescence by investigating frontal asymmetry. It has been suggested that PFC lateralisation reflects the approach-avoidance distinction, whereby greater cortical activity in the left PFC relative to the right PFC reflects the approach system and greater cortical activity in the right PFC relative to the left PFC reflects the avoidance system, as indexed by spontaneous alpha (Davidson, 1984, 1992). Based on this premise, it should be possible to test the relative strength of the approach and avoidance systems throughout adolescence. Hence, the third study in this doctoral work examined the development of source localised and scalp-recorded frontal spontaneous alpha in preadolescents, mid-adolescents, and late adolescents. This study also examined the relationships between PFC alpha asymmetry and adolescents' risk-taking behaviours. However, this study did not reveal clear developmental trajectories of the approach-avoidance systems during adolescence, or systematic relationships between frontal alpha asymmetry and risk-taking behaviours. Thus, the final study in this doctoral work did not shed light on the development of the approach-avoidance systems, and the role of the approach-avoidance systems in risk-taking behaviours, during adolescence. Future work examining the developmental of frontal asymmetry, and the relationships between frontal asymmetry and risk-taking behaviours, in atypical populations may provide more useful insights (see Chapter 6.3.1).

Despite not providing insights into the approach-avoidance systems during adolescence, the study presented in Chapter 5 examined, for the first time, how the cortical sources of spontaneous alpha during eyes-open and eyes-closed conditions develop throughout adolescence. Notably, the findings revealed developmental differences in the frontal sources of alpha; late adolescents had significantly more alpha CSD in the mPFC and OFC during the eyes-open condition compared to the

eyes-closed condition. By contrast, alpha CSD was equivalent in the eyes-open and eyes-closed conditions in the mPFC and OFC for preadolescents and mid-adolescents. Moreover, preadolescents had greater levels of alpha CSD in the mPFC, DLPFC, OFC, and occipital cortex compared to both mid-adolescents and late adolescents, irrespective of condition. Critically, these findings highlight the need to treat adolescence as a transitional period, rather than a single snapshot in time (Casey *et al*., 2008a; Spear, 2000).

6.2.2. Adolescence as a transitional period

As discussed above, adolescence is a developmental period that reflects the transition from childhood to adulthood. Thus, the transitions into and out of adolescence need to be examined in order to understand adolescent-specific changes that may underlie high levels of risk-taking and anxiety (Casey *et al*., 2008a; Spear, 2000). Despite this, most studies assessing adolescent development only include a single adolescent group (e.g., 15-18 years; Richards *et al*., 2015), or a discrete adolescent group and a discrete adult group (e.g., 11-16 years and 24-40 years; Sebastian *et al*., 2011). Hence, these studies only tap into specific developmental periods, and are unable to assess how behaviours and neural responses change across the course of adolescence and how adolescents differ from children. Understanding how adolescents differ from children is critical for understanding why risk-taking behaviours and anxiety levels are widely reported to increase during the transition from childhood to adolescence (Casey *et al*., 2008a; Spear, 2000). In order to test the development of risk-taking behaviours, anxiety levels, and the approach-avoidance systems throughout adolescence, a large cohort of adolescents aged 9-23 years took part in this doctoral work. Critically, the transitions into and out of adolescence were examined by categorising participants into three age groups: preadolescence (9-12 years), mid-adolescence (13-17 years), and late adolescence (18-23 years). As expected, including a younger age group (preadolescents) and an older age group (late adolescents) provided important insights into the development of risk-taking behaviours, anxiety levels, and the approach-avoidance systems across adolescence.

The first study (Chapter 3) in this doctoral work found that preadolescents had greater levels of anxiety compared to both mid-adolescents and late adolescents. Preadolescents also took significantly fewer real world risks than both mid-adolescents and late adolescents. By contrast, both preadolescents and mid-

adolescents took significantly fewer risks on the BART compared to late adolescents. Age-related changes in impulsivity were also found; impulsivity decreased from preadolescence to mid-adolescence, but not from mid-adolescence to late adolescence. Thus, including a younger and older comparison group provided important insights into how anxiety, risk-taking, and impulsivity changed from preadolescence to mid-adolescence, and from mid-adolescence to late adolescence. Chapter 3 also examined, for the first time, how the relationship between risk-taking and anxiety changes during the course of adolescence. Crucially, this study revealed significant differences in the relationship between risk-taking and anxiety during the different stages of adolescence. Although the findings from this study are not entirely consistent with previous work (e.g., Abe & Suzuki, 1986; Burnett *et al*., 2010; Richards *et al*., 2015; Steinberg et al., 2008), the findings extend the existing literature by exploring the development of anxiety, risk-taking, sensation seeking, and impulsivity, and the relationships between risk-taking and anxiety, in a normative sample with an age range wide enough to determine how these constructs change during the entire course of adolescence.

Including a younger comparison group in the second study in this doctoral work (Chapter 4) also provided vital information about the development of anticipatory ERP responses to visual cues predicting rewarding outcomes. LPP amplitudes were potentiated to visual cues predicting rewarding outcomes in mid-adolescents but not in late adolescents. Critically however, the LPP was also potentiated to visual cues predicting rewarding outcomes in preadolescents. Moreover, there was evidence that preadolescents were more responsive to visual cues predicting rewarding outcomes than mid-adolescents. Thus, without a younger comparison group, it would not have been possible to determine whether LPP potentiation to reward-related cues resulted from heightened sensitivity to stimuli predicting potential rewards during middle adolescence, as predicted by the neurobiological models (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008), or a developmental trait extending from childhood. Hence, the findings from this study underscore the importance of including younger comparison groups when attempting to establish the behaviours and neural responses that are unique to adolescence.

Notably, the results from all three studies in this doctoral work show that preadolescence is a particularly distinct developmental period compared to both mid-adolescence and late adolescence. As discussed earlier, Chapter 3 revealed that

preadolescents were more anxious, risk-averse, impulsive, and showed distinctive relationships between risk-taking and anxiety compared to mid-adolescents and late adolescents. Additionally, the two EEG studies showed that preadolescents were more responsive to rewards and threats, as indexed by the LPP (Chapter 4), and had greater levels of alpha CSD in frontal and occipital regions (Chapter 5) compared to mid-adolescents and late adolescents. Together, these findings emphasise the need to tease out the early stages of adolescence from middle and late adolescence, and show that there are considerable developments in anxiety, risk-taking, and EEG activity during the transitional period between childhood and adolescence.

Therefore, one of the major strengths of this doctoral work is that the transitions from late childhood/preadolescence to mid-adolescence in addition to the transitions from mid-adolescence to late adolescence/young adulthood were examined. Critically, the Triadic Model and SIPN account for changes in the approach-avoidance systems between adolescence and adulthood, but not between childhood and adolescence. Thus, the Triadic Model and SIPN need to be updated in line with Casey and Steinberg's dual systems models to account for the developmental changes in the approach-avoidance systems from childhood to adolescence, as well as from adolescence to adulthood.

Another major strength of examining the developmental trajectories of risk-taking, anxiety, and brain activity in a sample of 9-23 year olds is that a large amount of normative data has been generated. In particular, the developmental trajectories of risk-taking behaviours as measured by a self-report questionnaire and behavioural task, sensation seeking, impulsivity, anxiety, and anxiety controlling for depression were investigated in male and female adolescents aged 9-23 years old. No study to the author's knowledge has explored the development of all these constructs in male and female adolescents with such a wide age range. Hence, these findings go beyond the existing literature by exploring how the developmental trajectories of these constructs develop within a single cohort of individuals during the entire course of adolescence. Moreover, Chapter 3 provides the first insights into how the relationship between risk-taking and anxiety changes throughout the course of adolescence, and the gender differences in such relationships. Chapter 4 also provides normative data of how N170 and LPP amplitudes in response to visual cues in an instrumental task develop across the course of adolescence. Finally, for the first time, Chapter 5 explored how the cortical sources of spontaneous alpha during eyes-open and eyes-closed conditions

develop throughout adolescence for males and females.

6.2.3. Using EEG to measure adolescent development

The vast majority of work assessing adolescents' neural responses to rewards and threats has used fMRI. These fMRI studies have provided vital insights into the neural structures involved in the approach-avoidance systems during adolescence, and how reward- and threat-related brain activity changes during the course of adolescence. However, unlike fMRI, EEG directly reflects the activity of neuronal populations (Davidson *et al.*, 2000), and can therefore provide a millisecond-by-millisecond account of reward- and threat-related anticipatory and consummatory neural activity during the different stages of adolescence. Thus, EEG studies are able to provide a complementary perspective on the neural mechanisms underlying the approach and avoidance systems during adolescence. This doctoral work therefore used EEG to examine the neural correlates of the approach-avoidance systems during adolescence.

In order to examine the timing of reward- and threat-related activity during adolescence, Chapter 4 investigated anticipatory responses to visual cues predicting rewarding and threatening outcomes using early (N170) and late (LPP) ERPs that are modulated by motivationally salient stimuli (Keil *et al.*, 2002; Levita *et al.*, 2014; Rellecke *et al.*, 2013; Sabatinelli *et al.*, 2007; Schupp *et al.*, 2000). Critically, only LPP amplitudes were potentiated, suggesting that the instrumental task used in this study did not tap into early neural systems that are primarily concerned with survival. These findings therefore underscore the importance of using EEG to shed light on the timing of reward- and threat-related processes during adolescence.

While fMRI has superior spatial resolution to EEG, CSD (see Chapter 2.4.3.2) and recent advances in source localisation (see Chapter 2.4.3.3) improve the spatial resolution of EEG and provide ways to estimate the cortical generators underlying the scalp-recorded EEG signals (Kayser & Tenke, 2015b; Pascual-Marqui, 2002). Compared to traditional referencing, CSD estimates provide sharper and more accurate topographies that are more likely to reflect the neural sources underlying the scalp-recorded activity (Burle *et al.*, 2015; Kayser *et al.*, 2006; Kayser & Tenke, 2015b; Tenke & Kayser, 2005). Accordingly, the resting alpha (Chapter 5) data was transformed to CSD. Chapter 5 also used source localisation to identify the cortical sources of spontaneous alpha throughout adolescence.

While CSD was only applied to the resting state data in this doctoral work, an increasing number of studies are applying CSD transformations to ERP data (see Kamarajan *et al*., 2014). The advantages of CSD for ERP research are comparable to the advantages for frequency data. Critically, CSD estimates are reference-free and therefore provide a way to compare studies without the confound of the choice of reference electrode, i.e., one study using average reference and another study using linked mastoids. Given that the choice of reference electrode significantly influences the shape of the ERP waveform (Luck, 2014), using CSD estimates in ERP studies should eliminate potential quantitative or statistical ambiguities caused by the choice of reference electrodes, and allow ERP studies to more accurately compare their results. To these ends, future work examining the ERP correlates of approach and avoidance behaviours should aim to apply CSD transformations.

Notably, EEG is particularly well suited to developmental studies. In particular, the environment in which EEG is recorded is less hostile than that of fMRI; fMRI is noisy, and involves lying down in a narrow space for a long period of time. Moreover, fMRI studies usually have high attrition rates in developmental studies (Ulmer & Jansen, 2010), and are considerably more expensive than EEG studies. Hence, combined with superior temporal resolution and methods that can improve the spatial resolution, the findings reported in this doctoral work demonstrate that EEG is a valid and useful tool for exploring the neural correlates of the approach-avoidance systems throughout adolescence.

6.2.4. The role of posterior regions in adolescent development

The neurobiological models only implicate cortical prefrontal and subcortical limbic structures when explaining the increases in risk-taking behaviours (Casey *et al*., 2010; Ernst *et al*., 2006; Nelson *et al*., 2005; Steinberg, 2008), and increases in emotionality (Casey *et al*., 2010), during adolescence. However, as discussed in Chapter 1.5.4, several EEG and fMRI studies in adults have reported that rewarding and threatening stimuli modulate activity in posterior brain regions (Harry *et al*., 2013; Lang *et al*., 1998; Levita *et al*., 2014; Li *et al*., 2005; Pizzagalli *et al*., 2003). Thus, if adolescents are hyperresponsive to rewards and threats, as the current literature suggests, it is possible that posterior regions will also have a role in biasing adolescents towards rewards and threats. Our previous findings revealed that adolescents had greater N170 potentiation to visual cues predicting threat compared

to young adults (Levita *et al*., 2014). Since the N170 is thought to be generated by structures located in occipitotemporal areas, namely the fusiform face area and superior temporal sulcus (Sadeh *et al*., 2010), our previous findings support the suggestion that posterior regions are important in biasing adolescents' attention towards potential threats. The second study in this doctoral work (Chapter 4) was designed to replicate and extend these findings. Unexpectedly, the N170 was not modulated by visual cues predicting threat, or by visual cues predicting reward, in mid-adolescents in this doctoral work. Thus, this doctoral work appears to not support the suggestion that posterior regions are important in biasing adolescents' attention towards potential reward and threat. However, the N170 was not potentiated for any age group, suggesting that early activity in posterior visual regions was not modulated by the instrumental task in this study. It is possible that stimuli of high evolutionary significance, i.e., primary reinforcers, are required to tap into these early ventral visual pathways. However, future work is needed to examine whether this is the case (see Chapter 6.3.4).

In contrast to the N170, the LPP was modulated by visual cues predicting rewarding and threatening outcomes. Several studies have reported that a wide neural network generates the LPP, including posterior regions that are critical for visual processing (Liu *et al*., 2012; Moratti *et al*., 2011; Sabatinelli *et al*., 2007; Sabatinelli *et al*., 2013). Hence, it is possible that the greater levels of LPP potentiation in preadolescents and mid-adolescents compared to late adolescents partially resulted from greater levels of activity in posterior regions. Thus, these findings provide some evidence for the idea that posterior regions are important for reward- and threat-related activity during adolescence.

6.2.5. Gender differences in adolescent development

The role of gender in risk-taking behaviours, anxiety levels, and reward- and threat-related neural activity has been largely overlooked in adolescent research. Moreover, the neurobiological models do not provide any insights into the role of gender in adolescent development. This is surprising since a number of studies have reported significant gender differences in risk-taking behaviours, anxiety levels, and brain development during adolescence (Byrnes *et al*., 1999; Giedd *et al*., 1999; Lenroot *et al*. 2007; Lewinsohn *et al*., 1998). In an attempt to address this, each of the three studies included in this doctoral work overtly investigated gender differences.

As expected, significant gender differences were found for risk-taking behaviours and anxiety levels (Chapter 3). Specifically, real world risk-taking increased from preadolescence to mid-adolescence for both males and females, but from mid-adolescence to late adolescence for females only. Females also self-reported higher levels of state and trait anxiety than males, irrespective of age. Additionally, gender-specific trajectories were found for the relationship between risk-taking and anxiety. While higher levels of anxiety were associated with reduced levels of risk-taking for late adolescent females, no relationship between risk-taking and anxiety was found for late adolescent males. Hence, these findings suggest that anxiety acted as a brake on risk-taking for late adolescent females, but not for late adolescent males. Moreover, higher levels of anxiety were associated with greater levels of risk taking in preadolescent females, but reduced levels of risk-taking in preadolescent males. Overall, these findings suggest that the relationship between risk-taking and anxiety during preadolescence and late adolescence is gender-specific. Together, these findings emphasise the importance of exploring gender differences when examining risk-taking behaviours and anxiety levels during adolescence.

In contrast, the EEG studies in this doctoral work did not reveal any statistically significant gender differences. Specifically, no significant gender differences emerged for the N170 or LPP (Chapter 4). Despite this, there was some evidence of a trend for gender differences in the LPP waveforms; the waveforms indicated that males largely drove the overall effect of reward-related LPP potentiation for the mid-adolescents. In the same way as Chapter 4, no gender differences emerged from the statistical analyses for spontaneous alpha (Chapter 5). However, the sLORETA tomography maps, particularly for the occipital cortex during the eyes-closed condition, strongly suggested that the development of spontaneous alpha was faster in females compared to males; mid-adolescent males more closely resembled the preadolescents, whereas mid-adolescent females more closely resembled the late adolescents. This observation is consistent with what is known about structural brain development, whereby females mature approximately 1-2 years faster than males (Giedd *et al*., 1999; Lenroot *et al*. 2007). It is feasible that high levels of variability between participants prevented gender differences from emerging in the statistical analyses. Critically, if the neurobiological models are going to inform future interventions aimed at reducing adolescent risk-taking and anxiety, it is important that the neurobiological models aim to establish and incorporate any potential gender

differences in brain structure and activity that are associated with gender-specific behavioural outcomes. Hence, future studies should continue to explore the potential gender differences in risk-taking, anxiety, and related brain activity during adolescence.

6.2.6. The influence of puberty on adolescent development

Consistent with Steinberg's DSM and the SIPN, this doctoral work had a strong focus on examining the influence of puberty on risk-taking, anxiety, and brain activity given that puberty has been shown to affect adolescent risk-taking, anxiety, structural brain development, and reward- and threat-related neural activity (Braams *et al*., 2015; Collado *et al*., 2014; Goddings *et al*., 2014; Ladouceur *et al*., 2012; Moore *et al*., 2012; Reardon *et al*., 2009). Pubertal stage had unique affects on recent real world risk-taking behaviours for both males and females aged 9-17 years, whereby more advanced pubertal stage was associated with greater engagement in recent real world risk-taking behaviours (Chapter 3). More advanced pubertal stage was also associated with less alpha CSD in frontal and occipital regions, but only for males (Chapter 5). For both risk-taking and spontaneous alpha, puberty accounted for between 10.2 and 26.8 per cent of the variance, suggesting that pubertal stage had a considerable influence on the development of real world risk-taking behaviours and spontaneous alpha during adolescence. Notably, puberty did not significantly influence sensation seeking, impulsivity, anxiety (Chapter 3), or reward- and threat-related anticipatory ERP activity (Chapter 4). These findings indicate that puberty may not have universal effects on adolescent development. Future work is therefore needed to establish the puberty-dependent and puberty-independent effects on behaviour, brain activity, and brain structure during adolescence. Early pubertal onset has been associated with greater engagement in risky behaviours and higher levels of anxiety (Downing & Bellis, 2009; Reardon *et al*., 2009), and thus it is possible that examining the timing of pubertal onset will also reveal insights into the influence puberty on behaviour, brain activity, and brain structure.

6.3. Directions for future research

This field of research is relatively new, and therefore there is a lot of scope for future research. This section reviews some of the directions for future research that have emerged from this doctoral work.

6.3.1. Application to atypical populations

Given that the majority of individuals negotiate the transition from childhood to adulthood successfully, this doctoral work examined the developmental trajectories of risk-taking behaviours, anxiety levels, the relationship between risk-taking and anxiety, reward- and threat-related brain activity, and spontaneous alpha in a normative sample of adolescents aged 9-23 years. However, there is a significant minority of adolescents who engage in very high levels of potentially harmful risky behaviours or who develop an anxiety disorder. It is possible that the developmental trajectories explored in this doctoral work, such as the relationship between risk-taking and anxiety, reward- and threat-related anticipatory neural activity, and spontaneous alpha, would be considerably different in these atypical populations compared to typically developing populations. For instance, several studies have reported that spontaneous alpha is disrupted in a number of developmental and psychiatric conditions, including anxiety disorders (Knyazev *et al*., 2004). Importantly, interventions aimed at reducing high levels of risk-taking and anxiety in adolescents need to be informed by studies assessing adolescents who are at an increased risk for engaging in potentially harmful risk-taking behaviours and developing an anxiety disorder. Hence, exploring the typical and atypical developmental trajectories of risk-taking, anxiety, and related brain activity is important for understanding why some adolescents are more likely to engage in potentially harmful behaviours and develop an anxiety disorder than others. Future work should therefore aim to establish how the developmental trajectories of risk-taking, anxiety, and related brain activity differ between typical and atypical populations.

6.3.2. Young adult participants

This doctoral work included participants aged 9-23 years in order to assess the transitions from preadolescence to mid-adolescence as well as from mid-adolescence to late adolescence. Thus, this doctoral work was able to examine the development of risk-taking, anxiety, and related brain activity across the entire course of adolescence. Despite this, this doctoral work would have benefited from a young adult age group, aged between 24-30 years. For instance, it is possible that including a young adult age group would have revealed a negative relationship between risk-taking and anxiety for males comparable to the relationship observed in late adolescent females. Moreover, it is possible that risk-taking behaviours would have shown an inverted u-shaped

trajectory, whereby risk-taking increased from preadolescence to mid-adolescence/late adolescence and decreased from late adolescence to young adulthood (Chapter 3). Future work should therefore endeavour to assess risk-taking behaviours, anxiety levels, and related brain activity in participants aged 9-30 years in order to assess the age-related changes between late adolescence and young adulthood, in addition to the age-related changes between late childhood and adolescence.

6.3.3. The influence of peers on adolescent risk-taking

There is now considerable evidence from both human and nonhuman animal studies to suggest that adolescents not only engage in more social interactions, but also find them more rewarding (Douglas, Varlinskaya & Spear, 2004; Larson & Richards, 1991). Consistently, a number of studies have reported that the presence of peers significantly increases adolescents' engagement in risk-taking behaviours (Gardner & Steinberg, 2005) and reward-related brain activity (Chein *et al*., 2011; Smith *et al*., 2015). Thus, the social context, and the presence of peers in particular, appear to have a marked impact on adolescent risk-taking (Nelson *et al*., 2005; Steinberg, 2008).

Notably, the studies reported in this doctoral work were conducted only in the presence of an experimenter. However, it is possible that replicating these studies in a social context, i.e., in the presence of peers, would lead to different conclusions. For instance, previous studies have reported that adolescents take more risks on behavioural risk-taking tasks when in the presence of their peers (Gardner & Steinberg, 2005). Thus, it is possible that the relationship between BART risk-taking and anxiety would differ depending on the social context. Future work should therefore aim to establish how the relationship between risk-taking and anxiety, and reward- and threat-related EEG activity, changes across the course of adolescence as a function of social context.

6.3.4. Development of appropriate measures

Studies assessing behavioural or brain development require measures that can accurately and appropriately measure constructs in samples with a wide age range that span a number of developmental periods (i.e., preadolescence, mid-adolescence, and late adolescence). However, tools that are able to accurately measure constructs across several developmental periods are scarce. Thus, while the measures used in this

doctoral work were selected to be appropriate for individuals aged 9-23 years, further work is needed to improve the measures that are currently available.

First, the ERP study reported in Chapter 4 examined age-related changes in anticipatory responses to visual cues predicting rewarding and threatening outcomes. Previous work in humans assessing neural responses to rewards and threats in instrumental tasks have either used pictures (e.g., Levita *et al*., 2012), loud tones (e.g., Levita *et al*., 2014), electric shocks (e.g., Delgado *et al*., 2009), or money (e.g., Bjork *et al*, 2010; Forbes *et al*., 2010). A primary aim of this study was to compare adolescents' anticipatory neural responses to rewards and threats, and therefore comparable rewarding and threatening reinforcers were essential. While pictures and money can be used to assess neural responses to both rewards (appetitive pictures, winning money) and threats (aversive pictures, losing money), there are a number of ethical and practical problems with using pictures and money in development work (see Chapter 2.4.2.1.2). Moreover, it is difficult to match tones on valence, and there is no appetitive reinforcer for electric shocks. In light of other developmental studies using points successfully (Lejuez *et al*., 2007), this study deemed that winning and losing points as the positive and negative reinforcers, respectively, would be age-appropriate and comparable.

However, in contrast to our previous findings (Levita *et al*., 2014), the N170 was not potentiated to visual cues predicting a threatening outcome. Our previous study used a primary reinforcer, namely a loud aversive tone, as the reinforcer of behaviour. Hence, it is possible that only primary reinforcers can modulate neural activity in early visual processing streams. Moreover, it is possible that LPP reinforcement-dependent potentiation did not peak in mid-adolescence because using points as the reinforcer was not salient enough for that age group. In order to test these ideas, further work is needed to explore the developmental trajectories of anticipatory and consummatory neural activity in response to a range of primary and secondary reinforcers in typically and atypically developing children, adolescents, and young adults.

Second, the self-report questionnaires and behavioural tasks used in this doctoral work were selected on the basis of age-appropriateness, use in the field, and validity. To that end, the YRBSS and BART were used to measure risk-taking behaviours, the BSSS was used to measure sensation seeking, and the STAI was used to measure anxiety. However, these measures were limited in a number of ways. While the YRBSS is widely used in adolescent research, the YRBSS only assesses a finite

number of risk-taking behaviours on a binary scale. Thus, the YRBSS is unable to provide a comprehensive overview of the risky behaviours that adolescents are engaging in. The development of a risk-taking scale whereby individuals do not report their engagement in specific risky behaviours, but instead report to what extent they engage in risky behaviours in comparison to their peers may provide an alternative way of examining the development of real world risk-taking during adolescence.

To account for the potential confounds associated with self-report questionnaires, a behavioural task was used to measure risk-taking propensity. The BART was selected to measure risk-taking propensity because it is extensively used in behavioural and fMRI studies. Despite this, the expected inverted u-shaped trajectory of risk-taking behaviours did not emerge. Previous studies that have reported such a trajectory have largely used probabilistic gambling tasks (Burnett *et al*., 2010; Eshel *et al*., 2007; Figner *et al*., 2009), which could account for the discrepancy in findings. However, no study to date has examined the development of risk-taking propensity using a variety of behavioural tasks. Conducting such a study in a large cohort of individuals aged 9-30 years would provide useful insights into how task demands affect the development of risk-taking propensity, and would shed light on why some studies report an inverted u-shaped trajectory in risk-taking propensity and others do not.

There was also evidence from this doctoral work that the BSSS was a poor measure of sensation seeking in this sample. It is therefore possible that some of the more unexpected findings in this doctoral work, i.e., no age- or gender-related differences in sensation seeking (Chapter 3), resulted from the BSSS being a poor measure of sensation seeking. Similarly, this doctoral work used the child and adult versions of the STAI given that there is no single measure of anxiety that can accurately assess anxiety in a sample of 9-23 year olds. The STAI child and adult versions have maximum scores of 60 and 80, respectively, and thus the raw scores were converted to percentages in order to equate the two measures. The finding that preadolescents had greater anxiety levels than both mid-adolescents and late adolescents may therefore reflect that the child version of the STAI measured a different construct of anxiety to the adult version.

Finally, it is possible that developments in basic motor and perceptual functions contribute to the age-related changes in risk-taking behaviours during adolescence. While this doctoral work aimed to control for such differences by

including control stimuli in the instrumental conditioning paradigm (Chapter 4), this research did not examine how developmental differences in basic motor and perceptual functions influence enagagement in risk-taking behaviours. Thus, future work is needed to investigate the relationship between basic motor and perceptual functions and risk-taking behaviours during the different stages of adolescence.

Critically, if studies are going to provide useful insights into the development of risk-taking and anxiety across adolescence, we need behavioural measures that can accurately assess risk-taking and anxiety in individuals with a wide age range. It is possible that many of the conflicting results between studies result from differences in task demands, such as the specific rewards and threats used, or the tools used to measure risk-taking and anxiety. Thus, there is a great need for future work to establish how responses to rewards and threats develop across adolescence, and to develop accurate and age-appropriate measures of risk-taking and anxiety so that studies can meaningfully compare their findings.

6.3.5. Replication in larger cross-sectional studies

While this doctoral research recruited a relatively large sample (N = 105), the sample size of each experimental group (e.g., preadolescent females) was small. In addition, developmental research tends to have high levels of variability since developmental trajectories across childhood and adolescence are highly individualised. Thus, this doctoral research may not have had the statistical power to detect small and potentially interesting effects. For instance, there was evidence for gender effects in the EEG studies (Chapters 4 and 5) that did not reach statistical significance (see Chapter 6.2.5).

Notably, outliers were not removed in this doctoral work due to the small sample size of each experimental group. Hence, it is possible that outliers may have influenced the correlation and regression analyses. In particular, there is some evidence from the eyes-open partial regression plot in Chapter 5 (Figure 5.8) that there were outliers in the data, which may have influenced the analyses. Since behavioural and EEG data are often non-normally distributed and developmental studies usually have high levels of variability, all analyses in this doctoral research (with the exception of ANOVAs with within-subject factors) were bootstrapped. As discussed in Chapter 2.5.1, bootstrapping is a non-parametric approach that provides robust estimates of standard errors, confidence intervals, correlation coefficients, and regression

coefficients without assuming a normal distribution (Field, 2009). Therefore, the findings reported in this doctoral research should be relatively robust to outliers. Nevertheless, outliers were not removed from the data and the findings should be interpreted with this in mind.

Finally, this doctoral work pursued several novel lines of research and consequently reported a number of original findings. For example, the behavioural study reported in Chapter 3 was the first study to examine the relationship between risk-taking and anxiety in male and female preadolescents, mid-adolescents, and late adolescents. Interestingly, this study found gender-specific developmental trajectories for the relationship between risk-taking and anxiety. In light of such findings, the small sample sizes of each experimental group, and the potential influence of outliers, the findings reported this doctoral research need replicating in larger cross-sectional samples to assess their validity and reliability.

6.3.6. Longitudinal studies

In addition to conducting cross-sectional studies to replicate the findings, longitudinal studies would be extremely valuable in establishing the developmental trajectories of risk-taking, anxiety, and related brain activity. There are considerable individual differences in child and adolescent development, with individuals reaching developmental milestones at different ages and having unique social and family environments (Steinberg & Morris, 2001). Hence, cross-sectional designs may introduce additional variance from extraneous variables, which could confound and conceal potentially important developmental differences (Blakemore & Choudhury, 2006). In order to minimise such variance and increase statistical power, cross-sectional studies can recruit larger cohorts, tightly control the experimental design and data collection, and use rigorous processing and statistical analysis techniques. However, longitudinal designs also provide a way to reduce the variance in developmental samples; longitudinal studies test the same cohort of participants repeatedly over a set period time, and are therefore able to minimise the variance attributable to extraneous variables. Longitudinal designs also provide a way to test whether risk-taking behaviours, anxiety levels, and related brain activity increase from childhood to adolescence and decrease from adolescence to young adulthood in the same individual, which would provide key insights into how behaviour and brain activity changes throughout adolescence. Despite the benefits of longitudinal studies

over cross-sectional studies, longitudinal studies are time-consuming, expensive, and have high attrition rates. Thus, future research should aim to utilise a combination of longitudinal studies and large cross-sectional studies in order to provide a more accurate and comprehensive understanding of adolescent development.

6.4. Final conclusions

This doctoral work aimed to provide a significant contribution to the literature by examining, for the first time, how the relationship between risk-taking and anxiety, and the EEG correlates of the approach-avoidance systems, change across the course of adolescence. Critically, this doctoral work does not support the suggestion that adolescents are hyporesponsive to threats (Ernst *et al*., 2006). Moreover, this doctoral work highlights the need to explore adolescents' sensitivity to both rewards and threats, as well as the transitions into and out of adolescence, in order to gain a comprehensive understanding of how the approach-avoidance systems develop across adolescence.

Importantly, there is now a large body of work to suggest that adolescence is a particularly vulnerable time for engaging in potentially harmful risky behaviours, experiencing high levels of anxiety, and developing a mental health condition (Abe & Suzuki, 1986; Burnett *et al*., 2010; Kessler *et al*., 2005; Steinberg *et al*., 2008). Critically, high levels of risk-taking and anxiety during adolescence have been associated with a number of short- and long-term adverse consequences (Dahl, 2004; Pine *et al*., 1998; Viner, 2011; Woodward & Fergusson, 2001). Research is beginning to uncover the factors and neurobiological mechanisms that underlie the increases in risk-taking behaviours and anxiety levels during adolescence. However, considerably more work is needed to examine the complex interactions between the brain, behaviour, puberty, gender, and social context in order to provide a complete and accurate understanding of adolescent development (Dahl, 2004).

References

Abe, K., & Suzuki, T. (1986). Prevalence of some symptoms in adolescence and maturity: Social phobias, anxiety symptoms, episodic illusions and idea of reference. *Psychopathology*, *19*(4), 200-205.

Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. R. (1995). Fear and the human amygdala. *Journal of Neuroscience*, *15*(9), 5879-5891.

Adrian, E. D., & Matthews, B. H. C. (1934). The Berger rhythm: Potential changes from the occipital lobes in man, *Brain*, *57*, 354–385.

Adriani, W., Chiarotti, F., & Laviola, G. (1998). Elevated novelty seeking and peculiar d-amphetamine sensitization in periadolescent mice compared with adult mice. *Behavioral Neuroscience*, *112*(5), 1152-1166.

Aklin, W. M., Lejuez, C. W., Zvolensky, M. J., Kahler, C. W., & Gwadz, M. (2005). Evaluation of behavioral measures of risk taking propensity with inner city adolescents. *Behaviour Research and Therapy*, *43*(2), 215-228.

Alfimova, M. V., & Uvarova, L. G. (2008). Changes in EEG spectral power on perception of neutral and emotional words in patients with schizophrenia, their relatives, and healthy subjects from the general population. *Neuroscience and Behavioral Physiology*, *38*(5), 533-540.

Andrade, L., Gorenstein, C., Vieira Filho, A. H., Tung, T. C., & Artes, R. (2001). Psychometric properties of the Portuguese version of the State-Trait Anxiety Inventory applied to college students: Factor analysis and relation to the Beck Depression Inventory. *Brazilian Journal of Medical and Biological Research*, *34*(3), 367-374.

Arnett, J. (1992). Reckless behavior in adolescence: A developmental perspective. *Developmental Review*, *12*(4), 339-373.

Arnett, J. (1999). Adolescent storm and stress, reconsidered. *American Psychologist*, *54*(5), 317-326.

Arnett, J. (2001). Conceptions of the transition to adulthood: Perspectives from adolescence through midlife. *Journal of Adult Development*, *8*(2), 133-143.

Babiloni, C., Frisoni, G. B., Pievani, M., Vecchio, F., Lizio, R., Buttiglione, M., ... & Rossini, P. M. (2009). Hippocampal volume and cortical sources of EEG alpha rhythms in mild cognitive impairment and Alzheimer disease. *Neuroimage*, *44*(1), 123-135.

Bados, A., Gómez-Benito, J., & Balaguer, G. (2010). The state-trait anxiety inventory,

trait version: Does it really measure anxiety?. *Journal of Personality Assessment*, *92*(6), 560-567.

Barkley-Levenson, E., & Galván, A. (2014). Neural representation of expected value in the adolescent brain. *Proceedings of the National Academy of Sciences*, *111*(4), 1646-1651.

Barnea-Goraly, N., Menon, V., Eckert, M., Tamm, L., Bammer, R., Karchemskiy, A., ... & Reiss, A. L. (2005). White matter development during childhood and adolescence: A cross-sectional diffusion tensor imaging study. *Cerebral Cortex*, *15*(12), 1848-1854.

Barry, R. J., Clarke, A. R., Johnstone, S. J., & Brown, C. R. (2009). EEG differences in children between eyes-closed and eyes-open resting conditions. *Clinical Neurophysiology*, *120*(10), 1806-1811.

Barry, R. J., Clarke, A. R., Johnstone, S. J., Magee, C. A., & Rushby, J. A. (2007). EEG differences between eyes-closed and eyes-open resting conditions. *Clinical Neurophysiology*, *118*(12), 2765-2773.

Başar, E. (2012). A review of alpha activity in integrative brain function: Fundamental physiology, sensory coding, cognition and pathology. *International Journal of Psychophysiology*, *86*(1), 1-24.

Batty, M., & Taylor, M. J. (2003). Early processing of the six basic facial emotional expressions. *Cognitive Brain Research*, *17*(3), 613-620.

Batty, M., & Taylor, M. J. (2006). The development of emotional face processing during childhood. *Developmental Science*, *9*(2), 207-220.

Baxter, M. G., & Murray, E. A. (2002). The amygdala and reward. *Nature Reviews Neuroscience*, *3*(7), 563-573.

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society. Series B (Methodological)*, *57*(1), 289-300.

Bentin, S., Allison, T., Puce, A., Perez, E., & McCarthy, G. (1996). Electrophysiological studies of face perception in humans. *Journal of Cognitive Neuroscience*, *8*(6), 551-565.

Berger, H. (1929). Über das elektrenkephalogramm des menschen. *European Archives of Psychiatry and Clinical Neuroscience*, *87*(1), 527-570.

Berridge, K. C., & Kringelbach, M. L. (2008). Affective neuroscience of pleasure: Reward in humans and animals. *Psychopharmacology*, *199*(3), 457-480.

Bezdjian, S., Baker, L. A., Lozano, D. I., & Raine, A. (2009). Assessing inattention and impulsivity in children during the Go/NoGo task. *British Journal of Developmental Psychology*, *27*(2), 365-383.

Biro, F. M., Lucky, A. W., Huster, G. A., & Morrison, J. A. (1995). Pubertal staging in boys. *Journal of Pediatrics*, *127*(1), 100-102.

Bishop, S., Duncan, J., Brett, M., & Lawrence, A. D. (2004). Prefrontal cortical function

and anxiety: controlling attention to threat-related stimuli. *Nature Neuroscience*, *7*(2), 184-188.

Bjelland, I., Dahl, A. A., Haug, T. T., & Neckelmann, D. (2002). The validity of the Hospital Anxiety and Depression Scale: An updated literature review. *Journal of Psychosomatic Research*, *52*(2), 69-77.

Bjork, J. M., Smith, A. R., Chen, G., & Hommer, D. W. (2010). Adolescents, adults and rewards: comparing motivational neurocircuitry recruitment using fMRI. *PloS One*, *5*(7), e11440.

Black, C. L., Goldstein, K. E., LaBelle, D. R., Brown, C. W., Harmon-Jones, E., Abramson, L. Y., & Alloy, L. B. (2014). Behavioral approach system sensitivity and risk taking interact to predict left-frontal EEG asymmetry. *Behavior Therapy*, *45*(5), 640-650.

Blakemore, S. J., Burnett, S., & Dahl, R. E. (2010). The role of puberty in the developing adolescent brain. *Human Brain Mapping*, *31*(6), 926-933.

Blakemore, S. J., & Choudhury, S. (2006). Development of the adolescent brain: Implications for executive function and social cognition. *Journal of Child Psychology and Psychiatry*, *47*(3-4), 296-312.

Blau, V. C., Maurer, U., Tottenham, N., & McCandliss, B. D. (2007). The face-specific N170 component is modulated by emotional facial expression. *Behavioral and Brain Functions*, *3*, 7.

Boiten, F., Sergeant, J., & Geuze, R. (1992). Event-related desynchronization: The effects of energetic and computational demands. *Electroencephalography and Clinical Neurophysiology*, *82*(4), 302-309.

Bornovalova, M. A., Cashman-Rolls, A., O'Donnell, J. M., Ettinger, K., Richards, J. B., & Lejuez, C. W. (2009). Risk taking differences on a behavioral task as a function of potential reward/loss magnitude and individual differences in impulsivity and sensation seeking. *Pharmacology Biochemistry and Behavior*, *93*(3), 258-262.

Bosquet, M., & Egeland, B. (2006). The development and maintenance of anxiety symptoms from infancy through adolescence in a longitudinal sample. *Development and Psychopathology*, *18*(02), 517-550.

Bourne, V. J. (2008). Examining the relationship between degree of handedness and degree of cerebral lateralization for processing facial emotion. *Neuropsychology*, *22*(3), 350-356.

Bouton, M. E., Mineka, S., & Barlow, D. H. (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychological Review*, *108*(1), 4-32.

Braams, B. R., Van Duijvenvoorde, A. C., Peper, J. S., & Crone, E. A. (2015). Longitudinal changes in adolescent risk-taking: A comprehensive study of neural responses to rewards, pubertal development, and risk-taking behavior. *Journal of Neuroscience*, *35*(18), 7226-7238.

Brady, E. U., & Kendall, P. C. (1992). Comorbidity of anxiety and depression in children and adolescents. *Psychological Bulletin*, *111*(2), 244.

Bramen, J. E., Hranilovich, J. A., Dahl, R. E., Forbes, E. E., Chen, J., Toga, A. W., ... & Sowell, E. R. (2011). Puberty influences medial temporal lobe and cortical gray matter maturation differently in boys than girls matched for sexual maturity. *Cerebral Cortex*, *21*(3), 636-646.

Britton, J. C., Grillon, C., Lissek, S., Norcross, M. A., Szuhany, K. L., Chen, G., ... & Pine, D. S. (2013). Response to Learned Threat: An fMRI study in adolescent and adult anxiety. *American Journal of Psychiatry*, *170*(10), 1195-1204.

Broman-Fulks, J. J., Urbaniak, A., Bondy, C. L., & Toomey, K. J. (2014). Anxiety sensitivity and risk-taking behavior. *Anxiety, Stress, and Coping*, *27*(6), 619-632.

Brooks-Gunn, J., & Graber, J. A. (1994). Puberty as a biological and social event: Implications for research on pharmacology. *Journal of Adolescent Health*, *15*(8), 663-671.

Brown, S. B., Van Steenbergen, H., Band, G. P., De Rover, M., & Nieuwenhuis, S. (2012). Functional significance of the emotion-related late positive potential. *Frontiers in Human Neuroscience*, 6, 33.

Broyd, S. J., Richards, H. J., Helps, S. K., Chronaki, G., Bamford, S., & Sonuga-Barke, E. J. (2012). An electrophysiological monetary incentive delay (e-MID) task: a way to decompose the different components of neural response to positive and negative monetary reinforcement. *Journal of Neuroscience Methods*, *209*(1), 40-49.

Buchanan, C. M., Eccles, J. S., & Becker, J. B. (1992). Are adolescents the victims of raging hormones? Evidence for activational effects of hormones on moods and behavior at adolescence. *Psychological Bulletin*, *111*(1), 62-107.

Bunge, S. A., Dudukovic, N. M., Thomason, M. E., Vaidya, C. J., & Gabrieli, J. D. (2002). Immature frontal lobe contributions to cognitive control in children: Evidence from fMRI. *Neuron*, *33*(2), 301-311.

Burle, B., Spieser, L., Roger, C., Casini, L., Hasbroucq, T., & Vidal, F. (2015). Spatial and temporal resolutions of EEG: Is it really black and white? A scalp current density view. *International Journal of Psychophysiology*, *97*(3), 210-220.

Burnett, S., Bault, N., Coricelli, G., & Blakemore, S. J. (2010). Adolescents' heightened risk-seeking in a probabilistic gambling task. *Cognitive Development*, *25*(2), 183-196.

Byrnes, J. P., Miller, D. C., & Schafer, W. D. (1999). Gender differences in risk taking: A meta-analysis. *Psychological Bulletin*, *125*(3), 367-383.

Cardinal, R. N., Parkinson, J. A., Hall, J., & Everitt, B. J. (2002). Emotion and motivation: The role of the amygdala, ventral striatum, and prefrontal cortex. *Neuroscience and Biobehavioral Reviews*, *26*(3), 321-352.

Casey, B. J. (2002). Windows into the human brain. *Science*, *296*(5572), 1408-1409.

Casey, B. J., Getz, S., & Galvan, A. (2008a). The adolescent brain. *Developmental Review*, *28*(1), 62-77.

Casey, B. J., Giedd, J. N., & Thomas, K. M. (2000). Structural and functional brain

development and its relation to cognitive development. *Biological Psychology*, *54*(1), 241-257.

Casey, B. J., Jones, R. M., & Hare, T. A. (2008b). The adolescent brain. *Annals of the New York Academy of Sciences*, *1124*(1), 111-126.

Casey, B. J., Jones, R. M., Levita, L., Libby, V., Pattwell, S. S., Ruberry, E. J., ... & Somerville, L. H. (2010). The storm and stress of adolescence: Insights from human imaging and mouse genetics. *Developmental Psychobiology*, *52*(3), 225-235.

Casey, B. J., Tottenham, N., Liston, C., & Durston, S. (2005). Imaging the developing brain: What have we learned about cognitive development?. *Trends in Cognitive Sciences*, *9*(3), 104-110.

Casey, B. J., Trainor, R. J., Orendi, J. L., Schubert, A. B., Nystrom, L. E., Giedd, J. N., ... & Forman, S. D. (1997). A developmental functional MRI study of prefrontal activation during performance of a go-no-go task. *Journal of Cognitive Neuroscience*, *9*(6), 835-847.

Cauffman, E., Shulman, E. P., Steinberg, L., Claus, E., Banich, M. T., Graham, S., & Woolard, J. (2010). Age differences in affective decision making as indexed by performance on the Iowa Gambling Task. *Developmental Psychology*, *46*(1), 193-207.

Cazzell, M., Li, L., Lin, Z. J., Patel, S. J., & Liu, H. (2012). Comparison of neural correlates of risk decision making between genders: An exploratory fNIRS study of the Balloon Analogue Risk Task (BART). *Neuroimage*, *62*(3), 1896-1911.

Centers for Disease Control and Preventions. (2001). *Youth Risk Behavior Surveillance.* Atlanta, GA: Centers for Disease Control and Preventions.

Chan, Y. F., Leung, D. Y., Fong, D. Y., Leung, C. M., & Lee, A. M. (2010). Psychometric evaluation of the Hospital Anxiety and Depression Scale in a large community sample of adolescents in Hong Kong. *Quality of Life Research*, *19*(6), 865-873.

Chapple, C. L., & Johnson, K. A. (2007). Gender differences in impulsivity. *Youth Violence and Juvenile Justice*, *5*(3), 221-234.

Chein, J., Albert, D., O'Brien, L., Uckert, K., & Steinberg, L. (2011). Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry. *Developmental Science*, *14*(2), F1-F10.

Chiang, A. K. I., Rennie, C. J., Robinson, P. A., Van Albada, S. J., & Kerr, C. C. (2011). Age trends and sex differences of alpha rhythms including split alpha peaks. *Clinical Neurophysiology*, *122*(8), 1505-1517.

Clark, C. R., Veltmeyer, M. D., Hamilton, R. J., Simms, E., Paul, R., Hermens, D., & Gordon, E. (2004). Spontaneous alpha peak frequency predicts working memory performance across the age span. *International Journal of Psychophysiology*, *53*(1), 1-9.

Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants. A proposal. *Archives of General Psychiatry*, *44*(6), 573-

588.

Coan, J. A., & Allen, J. J. (2003). Frontal EEG asymmetry and the behavioral activation and inhibition systems. *Psychophysiology*, *40*(1), 106-114.

Coan, J. A., & Allen, J. J. (2004). Frontal EEG asymmetry as a moderator and mediator of emotion. *Biological Psychology*, *67*(1), 7-50.

Collado, A., MacPherson, L., Kurdziel, G., Rosenberg, L. A., & Lejuez, C. W. (2014). The relationship between puberty and risk taking in the real world and in the laboratory. *Personality and Individual Differences*, *68*, 143-148.

Comeau, N., Stewart, S. H., & Loba, P. (2001). The relations of trait anxiety, anxiety sensitivity, and sensation seeking to adolescents' motivations for alcohol, cigarette, and marijuana use. *Addictive Behaviors*, *26*(6), 803-825.

Cooper, N. R., Croft, R. J., Dominey, S. J., Burgess, A. P., & Gruzelier, J. H. (2003). Paradox lost? Exploring the role of alpha oscillations during externally vs. internally directed attention and the implications for idling and inhibition hypotheses. *International Journal of Psychophysiology*, *47*(1), 65-74.

Corr, P. J. (2013). Approach and avoidance behaviour: Multiple systems and their interactions. *Emotion Review*, *5*(3), 285-290.

Corr, P. J., & McNaughton, N. (2012). Neuroscience and approach/avoidance personality traits: A two stage (valuation–motivation) approach. *Neuroscience and Biobehavioral Reviews*, *36*(10), 2339-2354.

Costa, V. D., Dal Monte, O., Lucas, D. R., Murray, E. A., & Averbeck, B. B. (2016). Amygdala and Ventral Striatum Make Distinct Contributions to Reinforcement Learning. *Neuron*, *92*(2), 505–517.

Costello, E. J., Sung, M., Worthman, C., & Angold, A. (2007). Pubertal maturation and the development of alcohol use and abuse. *Drug and Alcohol Dependence*, *88*(Suppl. 1), S50-S59.

Crews, F., He, J., & Hodge, C. (2007). Adolescent cortical development: A critical period of vulnerability for addiction. *Pharmacology Biochemistry and Behavior*, *86*(2), 189-199.

Cross, C. P., Cyrenne, D. L. M., & Brown, G. R. (2013). Sex differences in sensation-seeking: A meta-analysis. *Scientific Reports*, *3*, 2486.

Cuspineda, E. R., Machado, C., Virues, T., Martínez-Montes, E., Ojeda, A., Valdés, P. A., ... & Valdes, L. (2009). Source analysis of alpha rhythm reactivity using LORETA imaging with 64-channel EEG and individual MRI. *Clinical EEG and Neuroscience*, *40*(3), 150-156.

Cuthbert, B. N., Schupp, H. T., Bradley, M. M., Birbaumer, N., & Lang, P. J. (2000). Brain potentials in affective picture processing: Covariation with autonomic arousal and affective report. *Biological Psychology*, *52*(2), 95-111.

Dahl, R. E. (2004). Adolescent brain development: A period of vulnerabilities and opportunities. Keynote address. *Annals of the New York Academy of Sciences*, *1021*, 1-22.

Da Silva, F. L., & Van Leeuwen, W. S. (1977). The cortical source of the alpha rhythm. *Neuroscience Letters*, *6*(2-3), 237-241.

Da Silva, F. L., Van Lierop, T. H. M. T., Schrijer, C. F., & Van Leeuwen, W. S. (1973). Organization of thalamic and cortical alpha rhythms: Spectra and coherences. *Electroencephalography and Clinical Neurophysiology*, *35*(6), 627-639.

Da Silva, F. L., Vos, J. E., Mooibroek, J., & Van Rotterdam, A. (1980). Relative contributions of intracortical and thalamo-cortical processes in the generation of alpha rhythms, revealed by partial coherence analysis. *Electroencephalography and Clinical Neurophysiology*, *50*(5), 449-456.

Davidson, R. J. (1984). Hemispheric asymmetry and emotion. In K. R. Scherer & P. Ekman (Eds.), *Approaches to emotion* (Chapter 2). Hillsdale, NJ: Lawrence Erlbaum Associates.

Davidson, R. J. (1992). Anterior cerebral asymmetry and the nature of emotion. *Brain and Cognition*, *20*(1), 125-151.

Davidson, M. C., Amso, D., Anderson, L. C., & Diamond, A. (2006). Development of cognitive control and executive functions from 4 to 13 years: Evidence from manipulations of memory, inhibition, and task switching. *Neuropsychologia*, *44*(11), 2037-2078.

Davidson, R. J., & Fox, N. A. (1989). Frontal brain asymmetry predicts infants' response to maternal separation. *Journal of Abnormal Psychology*, *98*(2), 127-131.

Davidson, R. J., Jackson, D., & Larson, C. L. (2000). Human electroencephalography. In J. T. Cacioppo, L. G. Tassinary, & G. Berntson, (Eds.), *Handbook of psychophysiology* (Chapter 2). Cambridge, United Kingdom: Cambridge University Press.

Deardorff, J., Hayward, C., Wilson, K. A., Bryson, S., Hammer, L. D., & Agras, S. (2007). Puberty and gender interact to predict social anxiety symptoms in early adolescence. *Journal of Adolescent Health*, *41*(1), 102-104.

Delgado, M. R., Jou, R. L., LeDoux, J. E., & Phelps, E. A. (2009). Avoiding negative outcomes: Tracking the mechanisms of avoidance learning in humans during fear conditioning. *Frontiers in Behavioral Neuroscience*, *3*, 3.

Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, *134*(1), 9-21.

De Macks, Z. A. O., Moor, B. G., Overgaauw, S., Güroğlu, B., Dahl, R. E., & Crone, E. A. (2011). Testosterone levels correspond with increased ventral striatum activation in response to monetary rewards in adolescents. *Developmental Cognitive Neuroscience*, *1*(4), 506-516.

De Munck, J. C., Goncalves, S. I., Huijboom, L., Kuijer, J. P. A., Pouwels, P. J. W., Heethaar, R. M., & Da Silva, F. L. (2007). The hemodynamic response of the

alpha rhythm: An EEG/fMRI study. *Neuroimage*, *35*(3), 1142-1151.

Dennis, T. A., & Hajcak, G. (2009). The late positive potential: A neurophysiological marker for emotion regulation in children. *Journal of Child Psychology and Psychiatry*, *50*(11), 1373-1383.

Dohmen, T., Falk, A., Huffman, D., & Sunde, U. (2010). Are risk aversion and impatience related to cognitive ability?. *American Economic Review*, *100*(3), 1238-1260.

Dolan, R. J. (2002). Emotion, cognition, and behavior. *Science*, *298*(5596), 1191-1194.

Dolan, R. J., Heinze, H. J., Hurlemann, R., & Hinrichs, H. (2006). Magnetoencephalography (MEG) determined temporal modulation of visual and auditory sensory processing in the context of classical conditioning to faces. *Neuroimage*, *32*(2), 778-789.

Donohew, L., Zimmerman, R., Cupp, P. S., Novak, S., Colon, S., & Abell, R. (2000). Sensation seeking, impulsive decision-making, and risky sex: Implications for risk-taking and design of interventions. *Personality and Individual Differences*, *28*(6), 1079-1091.

Donovan, J. E., & Jessor, R. (1985). Structure of problem behavior in adolescence and young adulthood. *Journal of Consulting and Clinical Psychology*, *53*(6), 890.

Doremus-Fitzwater, T. L., Varlinskaya, E. I., & Spear, L. P. (2009). Social and non-social anxiety in adolescent and adult rats after repeated restraint. *Physiology and Behavior*, *97*(3), 484-494.

Dorn, L. D., & Biro, F. M. (2011). Puberty and its measurement: A decade in review. *Journal of Research on Adolescence*, *21*(1), 180-195.

Douglas, L. A., Varlinskaya, E. I., & Spear, L. P. (2004). Rewarding properties of social interactions in adolescent and adult male and female rats: Impact of social versus isolate housing of subjects and partners. *Developmental Psychobiology*, *45*(3), 153-162.

Downing, J., & Bellis, M. A. (2009). Early pubertal onset and its relationship with sexual risk taking, substance use and anti-social behaviour: A preliminary cross-sectional study. *BMC Public Health*, *9*, 446.

Durston, S., & Casey, B. J. (2006). What have we learned about cognitive development from neuroimaging?. *Neuropsychologia*, *44*(11), 2149-2157.

Dustman, R. E., Shearer, D. E., & Emmerson, R. Y. (1999). Life-span changes in EEG spectral amplitude, amplitude variability and mean frequency. *Clinical Neurophysiology*, *110*(8), 1399-1409.

Eachus, P. (2004). Using the Brief Sensation Seeking Scale (BSSS) to predict holiday preferences. *Personality and Individual Differences*, *36*(1), 141-153.

Eaton, D. K., Kann, L., Kinchen, S., Shanklin, S., Ross, J. Hawkins, J., ... Wechsler, H. (2010). Youth risk behavior surveillance – United States, 2009. *Morbidity and Mortality Weekly Report Surveillance Summaries*, *59*(5), 1-142.

Efron, B., & Tibshirani, R. J. (1993). *An introduction to the bootstrap*. New York, NY: Chapman & Hall.

Eimer, M. (2011). The face-sensitivity of the n170 component. *Frontiers in Human Neuroscience*, *5*, 119.

Elliot, A. J. (2006). The hierarchical model of approach-avoidance motivation. *Motivation and Emotion*, *30*(2), 111-116.

Elsner, B., & Hommel, B. (2001). Effect anticipation and action control. *Journal of Experimental Psychology: Human Perception and Performance*, *27*(1), 229-240.

Ernst, M., Daniele, T., & Frantz, K. (2011). New perspectives on adolescent motivated behavior: Attention and conditioning. *Developmental Cognitive Neuroscience*, *1*(4), 377-389.

Ernst, M., Pine, D. S., & Hardin, M. (2006). Triadic model of the neurobiology of motivated behavior in adolescence. *Psychological Medicine*, *36*(03), 299-312.

Eshel, N., Nelson, E. E., Blair, R. J., Pine, D. S., & Ernst, M. (2007). Neural substrates of choice selection in adults and adolescents: Development of the ventrolateral prefrontal and anterior cingulate cortices. *Neuropsychologia*, *45*(6), 1270-1279.

Faden, V. B., Ruffin, B., Newes-Adeyi, G., & Chen, C. (2009). The relationship among pubertal stage, age, and drinking in adolescent boys and girls. *Journal of Child and Adolescent Substance Abuse*, *19*(1), 1-15.

Feige, B., Scheffler, K., Esposito, F., Di Salle, F., Hennig, J., & Seifritz, E. (2005). Cortical and subcortical correlates of electroencephalographic alpha rhythm modulation. *Journal of Neurophysiology*, *93*(5), 2864-2872.

Fergusson, D. M., Horwood, L. J., & Lynskey, M. T. (1993). Prevalence and comorbidity of DSM-III-R diagnoses in a birth cohort of 15 year olds. *Journal of the American Academy of Child and Adolescent Psychiatry*, *32*(6), 1127-1134.

Field, A. (2013). *Discovering statistics using SPSS* (3rd ed.). London, United Kingdom: Sage Publications.

Figner, B., Mackinlay, R. J., Wilkening, F., & Weber, E. U. (2009). Affective and deliberative processes in risky choice: Age differences in risk taking in the Columbia Card Task. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *35*(3), 709-730.

Forbes, E. E., & Dahl, R. E. (2010). Pubertal development and behavior: hormonal activation of social and motivational tendencies. *Brain and Cognition*, *72*(1), 66-72.

Forbes, E. E., Phillips, M. L., Silk, J. S., Ryan, N. D., & Dahl, R. E. (2011). Neural systems of threat processing in adolescents: Role of pubertal maturation and relation to measures of negative affect. *Developmental Neuropsychology*, *36*(4), 429-452.

Forbes, E. E., Ryan, N. D., Phillips, M. L., Manuck, S. B., Worthman, C. M., Moyles, D. L., ... & Dahl, R. E. (2010). Healthy adolescents' neural response to reward: Associations with puberty, positive affect, and depressive symptoms. *Journal of*

the American Academy of Child and Adolescent Psychiatry, *49*(2), 162-172.

Fowles, D. C. (1987). Application of a behavioral theory of motivation to the concepts of anxiety and impulsivity. *Journal of Research in Personality*, *21*(4), 417-435.

Fox, K. C., Spreng, R. N., Ellamil, M., Andrews-Hanna, J. R., & Christoff, K. (2015). The wandering brain: Meta-analysis of functional neuroimaging studies of mind-wandering and related spontaneous thought processes. *Neuroimage*, *111*, 611-621.

Foxe, J. J., & Snyder, A. C. (2011). The role of alpha-band brain oscillations as a sensory suppression mechanism during selective attention. *Frontiers in Psychology*, *2*, 154.

Franken, I. H., Huijding, J., Nijs, I. M., & Van Strien, J. W. (2011). Electrophysiology of appetitive taste and appetitive taste conditioning in humans. *Biological Psychology*, *86*(3), 273-278.

Franken, I. H., Van Strien, J. W., Bocanegra, B. R., & Huijding, J. (2011). The P3 event-related potential as an index of motivational relevance. *Journal of Psychophysiology*, *25*, 32-39.

Freese, J. L., & Amaral, D. G. (2005). The organization of projections from the amygdala to visual cortical areas TE and V1 in the macaque monkey. *Journal of Comparative Neurology*, *486*(4), 295-317.

Fuster, J. M. (2001). The prefrontal cortex – an update: Time is of the essence. *Neuron*, *30*(2), 319-333.

Galván, A., Hare, T. A., Parra, C. E., Penn, J., Voss, H., Glover, G., & Casey, B. J. (2006). Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *Journal of Neuroscience*, *26*(25), 6885-6892.

Galván, A., Hare, T., Voss, H., Glover, G., & Casey, B. J. (2007). Risk-taking and the adolescent brain: Who is at risk?. *Developmental Science*, *10*(2), F8-F14.

Galván, A., & McGlennen, K. M. (2013). Enhanced striatal sensitivity to aversive reinforcement in adolescents versus adults. *Journal of Cognitive Neuroscience*, *25*(2), 284-296.

Gardner, M., & Steinberg, L. (2005). Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: An experimental study. *Developmental Psychology*, *41*(4), 625-635.

Ge, X., Conger, R. D., & Elder, G. H. (1996). Coming of age too early: Pubertal influences on girls' vulnerability to psychological distress. *Child Development*, *67*(6), 3386-3400.

Ghetti, S., & Bauer, P. J. (2012). *Origins and development of recollection: Perspectives from psychology and neuroscience*. New York, NY: Oxford University Press.

Giedd, J. N. (2004). Structural magnetic resonance imaging of the adolescent brain. *Annals of the New York Academy of Sciences*, *1021*(1), 77-85.

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., ... & Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience*, *2*(10), 861-863.

Giedd, J. N., Vaituzis, A. C., Hamburger, S. D., Lange, N., Rajapakse, J. C., Kaysen, D., ... & Rapoport, J. L. (1996). Quantitative MRI of the temporal lobe, amygdala, and hippocampus in normal human development: Ages 4–18 years. *Journal of Comparative Neurology*, *366*(2), 223-230.

Giorgetta, C., Grecucci, A., Zuanon, S., Perini, L., Balestrieri, M., Bonini, N., ... & Brambilla, P. (2012). Reduced risk-taking behavior as a trait feature of anxiety. *Emotion*, *12*(6), 1373.

Goddings, A. L., Mills, K. L., Clasen, L. S., Giedd, J. N., Viner, R. M., & Blakemore, S. J. (2014). The influence of puberty on subcortical brain development. *Neuroimage*, *88*, 242-251.

Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., ... & Thompson, P. M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Sciences of the United States of America*, *101*(21), 8174–8179.

Goldman, R. I., Stern, J. M., Engel Jr, J., & Cohen, M. S. (2002). Simultaneous EEG and fMRI of the alpha rhythm. *Neuroreport*, *13*(18), 2487-2492.

Goldstein, R. Z., Cottone, L. A., Jia, Z., Maloney, T., Volkow, N. D., & Squires, N. K. (2006). The effect of graded monetary reward on cognitive event-related potentials and behavior in young healthy adults. *International Journal of Psychophysiology*, *62*(2), 272-279.

Gottfried, J. A., O'Doherty, J., & Dolan, R. J. (2003). Encoding predictive reward value in human amygdala and orbitofrontal cortex. *Science*, *301*(5636), 1104-1107.

Gray, J. A. (1975). *Elements of a two-process theory of learning*. New York, NY: Academic Press.

Greenblatt, R. E., Ossadtchi, A., & Pflieger, M. E. (2005). Local linear estimators for the bioelectromagnetic inverse problem. *IEEE transactions on signal processing, 53*(9), 3403-3412.

Grin-Yatsenko, V. A., Baas, I., Ponomarev, V. A., & Kropotov, J. D. (2009). EEG power spectra at early stages of depressive disorders. *Journal of Clinical Neurophysiology*, *26*(6), 401-406.

Guitart-Masip, M., Huys, Q. J., Fuentemilla, L., Dayan, P., Duzel, E., & Dolan, R. J. (2012). Go and no-go learning in reward and punishment: Interactions between affect and effect. *Neuroimage*, *62*(1), 154-166.

Gullone, E., King, N. J., & Ollendick, T. H. (2001). Self-reported anxiety in children and adolescents: A three-year follow-up study. *Journal of Genetic Psychology*, *162*(1), 5-19.

Gullone, E., Moore, S., Moss, S., & Boyd, C. (2000). The adolescent risk-taking questionnaire development and psychometric evaluation. *Journal of Adolescent*

Research, *15*(2), 231-250.

Haegens, S., Cousijn, H., Wallis, G., Harrison, P. J., & Nobre, A. C. (2014). Inter-and intra-individual variability in alpha peak frequency. *Neuroimage*, *92*, 46-55.

Haegens, S., Nácher, V., Luna, R., Romo, R., & Jensen, O. (2011). α-Oscillations in the monkey sensorimotor network influence discrimination performance by rhythmical inhibition of neuronal spiking. *Proceedings of the National Academy of Sciences*, *108*(48), 19377-19382.

Hagemann, D. (2004). Individual differences in anterior EEG asymmetry: Methodological problems and solutions. *Biological Psychology*, *67*(1), 157-182.

Hagemann, D., Hewig, J., Walter, C., & Naumann, E. (2008). Skull thickness and magnitude of EEG alpha activity. *Clinical Neurophysiology*, *119*(6), 1271-1280.

Hagmann, P., Sporns, O., Madan, N., Cammoun, L., Pienaar, R., Wedeen, V. J., ... & Grant, P. E. (2010). White matter maturation reshapes structural connectivity in the late developing human brain. *Proceedings of the National Academy of Sciences*, *107*(44), 19067-19072.

Hajcak, G., & Dennis, T. A. (2009). Brain potentials during affective picture processing in children. *Biological Psychology*, *80*(3), 333-338.

Hajcak, G., Weinberg, A., MacNamara, A., & Foti, D. (2012). ERPs and the study of emotion. In S. J. Luck & E. S. Kappenman (Eds.), *The Oxford handbook of event-related potential components* (Chapter 16). New York, NY: Oxford University Press.

Hall, G. S. (1904). *Adolescence: Its psychology and its relations to physiology, anthropology, sociology, sex, crime, religion, and education* (Vols. I & II). New York, NY: D. Appleton & Co.

Harden, K. P., & Tucker-Drob, E. M. (2011). Individual differences in the development of sensation seeking and impulsivity during adolescence: Further evidence for a dual systems model. *Developmental Psychology*, *47*(3), 739-746.

Hare, T. A., Tottenham, N., Galvan, A., Voss, H. U., Glover, G. H., & Casey, B. J. (2008). Biological substrates of emotional reactivity and regulation in adolescence during an emotional go-nogo task. *Biological Psychiatry*, *63*(10), 927-934.

Hari, R., Salmelin, R., Mäkelä, J. P., Salenius, S., & Helle, M. (1997). Magnetoencephalographic cortical rhythms. *International Journal of Psychophysiology*, *26*(1-3), 51-62.

Harmon-Jones, E., Abramson, L. Y., Nusslock, R., Sigelman, J. D., Urosevic, S., Turonie, L. D., ... & Fearn, M. (2008). Effect of bipolar disorder on left frontal cortical responses to goals differing in valence and task difficulty. *Biological Psychiatry*, *63*(7), 693-698.

Harmon-Jones, E., & Allen, J. J. (1998). Anger and frontal brain activity: EEG asymmetry consistent with approach motivation despite negative affective valence. *Journal of Personality and Social Psychology*, *74*(5), 1310-1316.

Harmon-Jones, E., Gable, P. A., & Peterson, C. K. (2010). The role of asymmetric

frontal cortical activity in emotion-related phenomena: A review and update. *Biological Psychology*, *84*(3), 451-462.

Harry, B. B., Williams, M., Davis, C., & Kim, J. (2013). Emotional expressions evoke a differential response in the fusiform face area. *Frontiers in Human Neuroscience*, *7*, 692.

Hayward, C., Killen, J. D., Hammer, L. D., Litt, I. F., Wilson, D. M., Simmonds, B., & Taylor, C. B. (1992). Pubertal stage and panic attack history in sixth-and seventh-grade girls. *American Journal of Psychiatry*, *149*(9), 1239-1243.

Hegde, J., & Felleman, D. J. (2007). Reappraising the functional implications of the primate visual anatomical hierarchy. *The Neuroscientist*, *13*(5), 416-421.

Hileman, C. M., Henderson, H., Mundy, P., Newell, L., & Jaime, M. (2011). Developmental and individual differences on the P1 and N170 ERP components in children with and without autism. *Developmental Neuropsychology*, *36*(2), 214-236.

Hinojosa, J. A., Mercado, F., & Carretié, L. (2015). N170 sensitivity to facial expression: A meta-analysis. *Neuroscience and Biobehavioral Reviews*, *55*, 498-509.

Hirose, S., Chikazoe, J., Watanabe, T., Jimura, K., Kunimatsu, A., Abe, O., ... & Konishi, S. (2012). Efficiency of go/no-go task performance implemented in the left hemisphere. *Journal of Neuroscience*, *32*(26), 9059-9065.

Hirschfeld, R. M. (2001). The comorbidity of major depression and anxiety disorders: Recognition and management in primary care. *The Primary Care Companion to The Journal of Clinical Psychiatry*, *3*(6), 244-254.

Höhl-Abrahão, J. C., & Creutzfeldt, O. D. (1991). Topographical mapping of the thalamocortical projections in rodents and comparison with that in primates. *Experimental Brain Research*, *87*(2), 283-294.

Hollerman, J. R., Tremblay, L., & Schultz, W. (1998). Influence of reward expectation on behavior-related neuronal activity in primate striatum. *Journal of Neurophysiology*, *80*(2), 947-963.

Hoyle, R. H., Stephenson, M. T., Palmgreen, P., Lorch, E. P., & Donohew, R. L. (2002). Reliability and validity of a brief measure of sensation seeking. *Personality and Individual Differences*, *32*(3), 401-414.

Hua, M., Han, Z. R., Chen, S., Yang, M., Zhou, R., & Hu, S. (2014). Late positive potential (LPP) modulation during affective picture processing in preschoolers. *Biological Psychology*, *101*, 77-81.

Huttenlocher, P. R. (1979). Synaptic density in human frontal cortex—developmental changes and effects of aging. *Brain Research*, *163*(2), 195-205.

Irwin Jr, C. E. (1990). The theoretical concept of at-risk adolescents. *Adolescent Medicine (Philadelphia, Pa.)*, *1*(1), 1-14.

Jeffreys, D. A. (1989). A face-responsive potential recorded from the human scalp. *Experimental Brain Research*, *78*(1), 193-202.

Jensen, J., McIntosh, A. R., Crawley, A. P., Mikulis, D. J., Remington, G., & Kapur, S. (2003). Direct activation of the ventral striatum in anticipation of aversive stimuli. *Neuron*, *40*(6), 1251-1257.

Jensen, O., Bonnefond, M., & VanRullen, R. (2012). An oscillatory mechanism for prioritizing salient unattended stimuli. *Trends in Cognitive Sciences*, *16*(4), 200-206.

Jensen, O., & Mazaheri, A. (2010). Shaping functional architecture by oscillatory alpha activity: Gating by inhibition. *Frontiers in Human Neuroscience*, *4*, 186.

Johnstone, S. J., Pleffer, C. B., Barry, R. J., Clarke, A. R., & Smith, J. L. (2005). Development of inhibitory processing during the go/nogo task. *Journal of Psychophysiology*, *19*(1), 11-23.

Jung, T. P., Humphries, C., Lee, T. W., Makeig, S., McKeown, M. J., Iragui, V., & Sejnowski, T. J. (1998). Removing electroencephalographic artifacts: Comparison between ICA and PCA. *Neural Networks for Signal Processing*, *8*, 63–72.

Jung, T. P., Makeig, S., Westerfield, M., Townsend, J., Courchesne, E., & Sejnowski, T. J. (2000). Removal of eye activity artifacts from visual event-related potentials in normal and clinical subjects. *Clinical Neurophysiology*, *111*(10), 1745-1758.

Kamarajan, C., Pandey, A. K., Chorlian, D. B., & Porjesz, B. (2015). The use of current source density as electrophysiological correlates in neuropsychiatric disorders: A review of human studies. *International Journal of Psychophysiology*, *97*(3), 310-322.

Kann, L., Kinchen, S. A., Williams, B. I., Ross, J. G., Lowry, R., Grunbaum, J. A., & Kolbe, L. J. (2000). Youth risk behavior surveillance – United States, 1999. Youth risk behavior surveillance – United States, 1999. *Morbidity and Mortality Weekly Report Surveillance Summaries*, *49*(5), 1-96.

Katada, A., Ozaki, H., Suzuki, H., & Suhara, K. (1981). Developmental characteristics of normal and mentally retarded children's EEGs. *Electroencephalography and Clinical Neurophysiology*, *52*(2), 192-201.

Kayser, J., & Tenke, C.E. (2006). Principal components analysis of Laplacian waveforms as a generic method for identifying ERP generator patterns: I. Evaluation with auditory oddball tasks. *Clinical Neurophysiology*, *117*(2), 348-368.

Kayser, J., & Tenke, C. E. (2015a). On the benefits of using surface Laplacian (current source density) methodology in electrophysiology. *International journal of psychophysiology: Official Journal of the International Organization of Psychophysiology*, *97*(3), 171-173.

Kayser, J., & Tenke, C. E. (2015b). Issues and considerations for using the scalp surface Laplacian in EEG/ERP research: A tutorial review. *International Journal of Psychophysiology*, *97*(3), 189-209.

Keil, A., Bradley, M. M., Hauk, O., Rockstroh, B., Elbert, T., & Lang, P. J. (2002). Large-

scale neural correlates of affective picture processing. *Psychophysiology*, *39*(5), 641-649.

Keil, A., Sabatinelli, D., Ding, M., Lang, P. J., Ihssen, N., & Heim, S. (2009). Re-entrant projections modulate visual cortex in affective perception: Evidence from Granger causality analysis. *Human Brain Mapping*, *30*(2), 532-540.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593-602.

Klimesch, W. (1999). EEG alpha and theta oscillations reflect cognitive and memory performance: A review and analysis. *Brain Research Reviews*, *29*(2), 169-195.

Klimesch, W., Doppelmayr, M., Schimke, H., & Ripper, B. (1997). Theta synchronization and alpha desynchronization in a memory task. *Psychophysiology*, *34*(2), 169-176.

Klimesch, W., Sauseng, P., & Hanslmayr, S. (2007). EEG alpha oscillations: the inhibition–timing hypothesis. *Brain Research Reviews*, *53*(1), 63-88.

Klimesch, W., Schimke, H., Ladurner, G., & Pfurtscheller, G. (1990). Alpha frequency and memory performance. *Journal of Psychophysiology*, *4*(4), 381-390.

Klimesch, W., Schimke, H., & Pfurtscheller, G. (1993). Alpha frequency, cognitive load and memory performance. *Brain Topography*, *5*(3), 241-251.

Knutson, B., Fong, G. W., Adams, C. M., Varner, J. L., & Hommer, D. (2001). Dissociation of reward anticipation and outcome with event-related fMRI. *Neuroreport*, *12*(17), 3683-3687.

Knutson, B., Fong, G. W., Bennett, S. M., Adams, C. M., & Hommer, D. (2003). A region of mesial prefrontal cortex tracks monetarily rewarding outcomes: Characterization with rapid event-related fMRI. *Neuroimage*, *18*(2), 263-272.

Knyazev, G. G., Savostyanov, A. N., & Levin, E. A. (2004). Alpha oscillations as a correlate of trait anxiety. *International Journal of Psychophysiology*, *53*(2), 147-160.

Knyazev, G. G., Slobodskoj-Plusnin, J. Y., Bocharov, A. V., & Pylkova, L. V. (2011). The default mode network and EEG alpha oscillations: An independent component analysis. *Brain Research*, *1402*, 67-79.

Kujawa, A., Klein, D. N., & Hajcak, G. (2012). Electrocortical reactivity to emotional images and faces in middle childhood to early adolescence. *Developmental Cognitive Neuroscience*, *2*(4), 458-467.

Kujawa, A., Klein, D. N., & Proudfit, G. H. (2013). Two-year stability of the late positive potential across middle childhood and adolescence. *Biological Psychology*, *94*(2), 290-296.

Kujawa, A., Weinberg, A., Hajcak, G., & Klein, D. N. (2013). Differentiating event-related potential components sensitive to emotion in middle childhood: Evidence from temporal–spatial PCA. *Developmental Psychobiology*, *55*(5), 539-

550.

Ladouceur, R., Bouchard, C., Rhéaume, N., Jacques, C., Ferland, F., Leblond, J., & Walker, M. (2000). Is the SOGS an accurate measure of pathological gambling among children, adolescents and adults?. *Journal of Gambling Studies*, *16*(1), 1-24.

Ladouceur, C. D., Peper, J. S., Crone, E. A., & Dahl, R. E. (2012). White matter development in adolescence: The influence of puberty and implications for affective disorders. *Developmental Cognitive Neuroscience*, *2*(1), 36-54.

Lamme, V. A., & Roelfsema, P. R. (2000). The distinct modes of vision offered by feedforward and recurrent processing. *Trends in Neurosciences*, *23*(11), 571-579.

Lang, P. J., & Bradley, M. M. (2010). Emotion and the motivational brain. *Biological Psychology*, *84*(3), 437-450.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). Motivated attention: Affect, activation, and action. In P. J. Lang, R. F. Simons, & M. Balaban (Eds.), *Attention and orienting: Sensory and motivational processes* (Chapter 5). Mahwah, New Jersey: Lawrence Erlbaum Associates.

Lang, P. J., Bradley, M. M., Fitzsimmons, J. R., Cuthbert, B. N., Scott, J. D., Moulder, B., & Nangia, V. (1998). Emotional arousal and activation of the visual cortex: An fMRI analysis. *Psychophysiology*, *35*(2), 199-210.

Larson, R., & Richards, M. H. (1991). Daily companionship in late childhood and early adolescence: Changing developmental contexts. *Child Development*, *62*(2), 284-300.

Lau, J. Y., Britton, J. C., Nelson, E. E., Angold, A., Ernst, M., Goldwin, M., ... & Shiffrin, N. (2011). Distinct neural signatures of threat learning in adolescents and adults. *Proceedings of the National Academy of Sciences*, *108*(11), 4500-4505.

Laufs, H., Kleinschmidt, A., Beyerle, A., Eger, E., Salek-Haddadi, A., Preibisch, C., & Krakow, K. (2003). EEG-correlated fMRI of human alpha activity. *Neuroimage*, *19*(4), 1463-1476.

Laviola, G., Macrì, S., Morley-Fletcher, S., & Adriani, W. (2003). Risk-taking behavior in adolescent mice: Psychobiological determinants and early epigenetic influence. *Neuroscience and Biobehavioral Reviews*, *27*(1), 19-31.

Lejuez, C. W., Aklin, W., Daughters, S., Zvolensky, M., Kahler, C., & Gwadz, M. (2007). Reliability and validity of the youth version of the balloon analogue risk task (BART–Y) in the assessment of risk-taking behavior among inner-city adolescents. *Journal of Clinical Child and Adolescent Psychology*, *36*(1), 106-111.

Lejuez, C. W., Aklin, W. M., Zvolensky, M. J., & Pedulla, C. M. (2003). Evaluation of the Balloon Analogue Risk Task (BART) as a predictor of adolescent real-world risk-taking behaviours. *Journal of Adolescence*, *26*(4), 475-479.

Lejuez, C. W., Read, J. P., Kahler, C. W., Richards, J. B., Ramsey, S. E., Stuart, G. L., ... & Brown, R. A. (2002). Evaluation of a behavioral measure of risk taking: The Balloon Analogue Risk Task (BART). *Journal of Experimental Psychology:*

Applied, *8*(2), 75-84.

Lenroot, R. K., & Giedd, J. N. (2006). Brain development in children and adolescents: Insights from anatomical magnetic resonance imaging. *Neuroscience and Biobehavioral Reviews*, *30*(6), 718-729.

Lenroot, R. K., & Giedd, J. N. (2010). Sex differences in the adolescent brain. *Brain and Cognition*, *72*(1), 46-55.

Lenroot, R. K., Gogtay, N., Greenstein, D. K., Wells, E. M., Wallace, G. L., Clasen, L. S., Blumenthal, J.D., Lerch, J., Zijdenbos, A.P., Evans, A.C. & Thompson, P. M. (2007). Sexual dimorphism of brain developmental trajectories during childhood and adolescence. *Neuroimage*, *36*(4), 1065-1073.

Leuchter, A. F., Spar, J. E., Walter, D. O., & Weiner, H. (1987). Electroencephalographic spectra and coherence in the diagnosis of Alzheimer's-type and multi-infarct dementia: A pilot study. *Archives of General Psychiatry*, *44*(11), 993-998.

Levita, L., Hoskin, R., & Champi, S. (2012). Avoidance of harm and anxiety: A role for the nucleus accumbens. *Neuroimage*, *62*(1), 189-198.

Levita, L., Howsley, P., Jordan, J., & Johnston, P. (2014). Potentiation of the early visual response to learned danger signals in adults and adolescents. *Social Cognitive and Affective Neuroscience*, nsu048.

Lewin, K. (1935). *A dynamic theory of personality*. New York, NY: McGraw-Hill.

Lewinsohn, P. M., Gotlib, I. H., Lewinsohn, M., Seeley, J. R., & Allen, N. B. (1998). Gender differences in anxiety disorders and anxiety symptoms in adolescents. *Journal of Abnormal Psychology*, *107*(1), 109-117.

Li, X., Li, X., & Luo, Y. J. (2005). Anxiety and attentional bias for threat: An event-related potential study. *Neuroreport*, *16*(13), 1501-1505.

Li, D., Sun, F., & Jiao, Y. (1996). Frontal EEG characters in aging and the correlativity with some cognitive abilities. *Acta Psychologica Sinica*, *28*(1), 76-81.

Lighthall, N. R., Mather, M., & Gorlick, M. A. (2009). Acute stress increases sex differences in risk seeking in the balloon analogue risk task. *PLoS One*, *4*(7), e6002.

Lindsley, D. B. (1939). A longitudinal study of the occipital alpha rhythm in normal children: Frequency and amplitude standards. *Pedagogical Seminary and Journal of Genetic Psychology*, *55*(1), 197-213.

Liston, C., Watts, R., Tottenham, N., Davidson, M. C., Niogi, S., Ulug, A. M., & Casey, B. J. (2006). Frontostriatal microstructure modulates efficient recruitment of cognitive control. *Cerebral Cortex*, *16*(4), 553-560.

Liu, Y., Huang, H., McGinnis-Deweese, M., Keil, A., & Ding, M. (2012). Neural substrate of the late positive potential in emotional processing. *Journal of Neuroscience*, *32*(42), 14563-14572.

Lopez-Calderon, J., & Luck, S. J. (2014). ERPLAB: An open-source toolbox for the analysis of event-related potentials. *Frontiers in Human Neuroscience*, *8*, 213.

Lorian, C. N., & Grisham, J. R. (2010). The safety bias: Risk-avoidance and social anxiety pathology. *Behaviour Change*, *27*(01), 29-41.

Lüchinger, R., Michels, L., Martin, E., & Brandeis, D. (2011). EEG–BOLD correlations during (post-) adolescent brain maturation. *Neuroimage*, *56*(3), 1493-1505.

Luck, S. J. (2014). *An introduction to the event-related potential technique*. Cambridge, MA: MIT press.

Luck, S., Lopez-Calderon, J., Huang, S., & Foo, E. (2013, October 1). *Exporting and Importing EventLists to Combine Artifact Rejection and Artifact Correction*. Retrieved from: http://erpinfo.org/erplab/erplab-documentation/tutorial_4/Rejection_Plus_Correction.html?searchterm=artefact

Luna, B., Garver, K. E., Urban, T. A., Lazar, N. A., & Sweeney, J. A. (2004). Maturation of cognitive processes from late childhood to adulthood. *Child Development*, *75(*5), 1357-1372.

Luna, B., Thulborn, K. R., Munoz, D. P., Merriam, E. P., Garver, K. E., Minshew, N. J., ... & Sweeney, J. A. (2001). Maturation of widely distributed brain function subserves cognitive development. *Neuroimage*, *13*(5), 786-793.

Mackintosh, N. J. (1974). *The psychology of animal learning*. New York, NY: Academic Press.

MacLeod, C., & Mathews, A. (1988). Anxiety and the allocation of attention to threat. *The Quarterly Journal of Experimental Psychology*, *40*(4), 653-670.

MacNamara, A., Vergés, A., Kujawa, A., Fitzgerald, K. D., Monk, C. S., & Phan, K. L. (2016). Age-related changes in emotional face processing across childhood and into young adulthood: Evidence from event-related potentials. *Developmental Psychobiology*, *58*(1), 27-38.

MacPherson, L., Magidson, J. F., Reynolds, E. K., Kahler, C. W., & Lejuez, C. W. (2010). Changes in sensation seeking and risk-taking propensity predict increases in alcohol Use among early adolescents. *Alcoholism: Clinical and Experimental Research*, *34*(8), 1400-1408.

Maner, J. K., Richey, J. A., Cromer, K., Mallott, M., Lejuez, C. W., Joiner, T. E., & Schmidt, N. B. (2007). Dispositional anxiety and risk-avoidant decision-making. *Personality and Individual Differences*, *42*(4), 665-675.

Maner, J. K., & Schmidt, N. B. (2006). The role of risk avoidance in anxiety. *Behavior Therapy*, *37*(2), 181-189.

Marshall, P. J., Bar-Haim, Y., & Fox, N. A. (2002). Development of the EEG from 5 months to 4 years of age. *Clinical Neurophysiology*, *113*(8), 1199-1208.

Marshall, W. A., & Tanner, J. M. (1969). Variations in pattern of pubertal changes in girls. *Archives of Disease in Childhood*, *44*(235), 291-303.

Marshall, W. A., & Tanner, J. M. (1970). Variations in the pattern of pubertal changes in boys. *Archives of Disease in Childhood*, *45*(239), 13-23.

Martin, C. A., Kelly, T. H., Rayens, M. K., Brogli, B. R., Brenzel, A., Smith, W. J., &

Omar, H. A. (2002). Sensation seeking, puberty, and nicotine, alcohol, and marijuana use in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*(12), 1495-1502.

Martin, M., Williams, R. M., & Clark, D. M. (1991). Does anxiety lead to selective processing of threat-related information?. *Behaviour Research and Therapy*, *29*(2), 147-160.

Matsumoto, K., Suzuki, W., & Tanaka, K. (2003). Neuronal correlates of goal-based motor selection in the prefrontal cortex. *Science*, *301*(5630), 229-232.

McDonald, J. H. (2014). *Handbook of biological statistics* (3rd ed.). Baltimore, MD: Sparky House Publishing.

Merikangas, K. R., He, J. P., Burstein, M., Swanson, S. A., Avenevoli, S., Cui, L., ... & Swendsen, J. (2010). Lifetime prevalence of mental disorders in US adolescents: Results from the National Comorbidity Survey Replication – Adolescent Supplement (NCS-A). *Journal of the American Academy of Child and Adolescent Psychiatry*, *49*(10), 980-989.

Miller, D. C., & Byrnes, J. P. (1997). The role of contextual and personal factors in children's risk taking. *Developmental Psychology*, *33*(5), 814-823.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, *24*(1), 167-202.

Miller, G. A., Crocker, L. D., Spielberg, J. M., Infantolino, Z. P., & Heller, W. (2015). Issues in localization of brain function: The case of lateralized frontal cortex in cognition, emotion, and psychopathology. *Current Research and Emerging Directions in Emotion-Cognition Interactions*, *7*, 2.

Mills, K. L., Goddings, A. L., Clasen, L. S., Giedd, J. N., & Blakemore, S. J. (2014). The developmental mismatch in structural brain maturation during adolescence. *Developmental Neuroscience*, *36*(3-4), 147-160.

Mills, K. L., Goddings, A. L., Herting, M. M., Meuwese, R., Blakemore, S. J., Crone, E. A., ... & Tamnes, C. K. (2016). Structural brain development between childhood and adulthood: Convergence across four longitudinal samples. *Neuroimage*, *141*, 273-281.

Mineka, S., & Oehlberg, K. (2008). The relevance of recent developments in classical conditioning to understanding the etiology and maintenance of anxiety disorders. *Acta Psychologica*, *127*(3), 567-580.

Miniño A. M. (2010). *Mortality among teenagers aged 12-19 years: United States, 1999-2006. NCHS data brief, no 37*. Hyattsville, MD: National Center for Health Statistics.

Mobascher, A., Brinkmeyer, J., Warbrick, T., Musso, F., Wittsack, H. J., Stoermer, R., ... & Winterer, G. (2009). Fluctuations in electrodermal activity reveal variations in single trial brain responses to painful laser stimuli – a fMRI/EEG study. *Neuroimage*, *44*(3), 1081-1092.

Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., & Swann, A. C. (2001).

Psychiatric aspects of impulsivity. *American Journal of Psychiatry*, *158*(11), 1783-1793.

Monk, C. S., Grillon, C., Baas, J. M., McClure, E. B., Nelson, E. E., Zarahn, E., ... & Pine, D. S. (2003). A neuroimaging method for the study of threat in adolescents. *Developmental Psychobiology*, *43*(4), 359-366.

Moore, W. E., Pfeifer, J. H., Masten, C. L., Mazziotta, J. C., Iacoboni, M., & Dapretto, M. (2012). Facing puberty: Associations between pubertal development and neural responses to affective facial displays. *Social Cognitive and Affective Neuroscience*, *7*(1), 35-43.

Moosmann, M., Ritter, P., Krastel, I., Brink, A., Thees, S., Blankenburg, F., ... & Villringer, A. (2003). Correlates of alpha rhythm in functional magnetic resonance imaging and near infrared spectroscopy. *Neuroimage*, *20*(1), 145-158.

Moratti, S., Saugar, C., & Strange, B. A. (2011). Prefrontal-occipitoparietal coupling underlies late latency human neuronal responses to emotion. *Journal of Neuroscience*, *31*(47), 17278-17286.

Morell, P., & Quarles, R. H. (1999). Myelin formation, structure and biochemistry. In G. J. Siegel, B. W. Agranoff, R. W. Albers, S. K. Fisher, & M. D. Uhler (Eds.), *Basic neurochemistry: Molecular, cellular and medical aspects (*6th ed.) (Chapter 4). Philadelphia, PA: Lippincott-Ravens.

Mowrer, O. H. (1960). *Learning theory and behavior*. New York, NY: Wiley.

Natale, L. (2010). *Factsheet: Youth crime in England and Wales*. London, United Kingdom: CIVITAS Institute for the Study of Civil Society.

Nelson, B. D., Perlman, G., Hajcak, G., Klein, D. N., & Kotov, R. (2015). Familial risk for distress and fear disorders and emotional reactivity in adolescence: An event-related potential investigation. *Psychological Medicine*, *45*(12), 2545-2556.

Nelson, C. A., & McCleery, J. P. (2008). Use of event-related potentials in the study of typical and atypical development. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*(11), 1252-1261.

Nelson, E. E., Leibenluft, E., McClure, E. B., & Pine, D. S. (2005). The social re-orientation of adolescence: A neuroscience perspective on the process and its relation to psychopathology. *Psychological Medicine*, *35*(02), 163-174.

Neufang, S., Specht, K., Hausmann, M., Güntürkün, O., Herpertz-Dahlmann, B., Fink, G. R., & Konrad, K. (2009). Sex differences and the impact of steroid hormones on the developing human brain. *Cerebral Cortex*, *19*(2), 464-473.

Newbury-Birch, D., Gilvarry, E., McArdle, P., Ramesh, V., Stewart, S., Walker, J., ... & Kaner, E. (2009). *Impact of alcohol consumption on young people: A systematic review of published reviews* (DCSF research report 067). London, United Kingdom: Department for Children, Schools and Families.

Niedermeyer, E. (1997). Alpha rhythms as physiological and abnormal phenomena. *International Journal of Psychophysiology*, *26*(1), 31-49.

Obrist, W. D. (1979). Electroencephalographic changes in normal aging and dementia.

In F. Moffmeister & C. Müller (Eds.), *Brain function in old age, Bayer Symposium VIII* (pp. 102-111). Berlin, Germany: Springer-Verlag.

O'Doherty, J., Dayan, P., Schultz, J., Deichmann, R., Friston, K., & Dolan, R. J. (2004). Dissociable roles of ventral and dorsal striatum in instrumental conditioning. S*cience*, *304*(5669), 452-454.

Olbrich, S., Mulert, C., Karch, S., Trenner, M., Leicht, G., Pogarell, O., & Hegerl, U. (2009). EEG-vigilance and BOLD effect during simultaneous EEG/fMRI measurement. *Neuroimage*, *45*(2), 319-332.

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia*, *9*(1), 97-113.

Padmanabhan, A., Geier, C. F., Ordaz, S. J., Teslovich, T., & Luna, B. (2011). Developmental changes in brain function underlying the influence of reward processing on inhibitory control. *Developmental Cognitive Neuroscience*, *1*(4), 517-529.

Palmgreen, P., Donohew, L., Lorch, E. P., Hoyle, R. H., & Stephenson, M. T. (2001). Television campaigns and adolescent marijuana use: Tests of sensation seeking targeting. *American Journal of Public Health*, *91*(2), 292-296.

Palva, S., & Palva, J. M. (2007). New vistas for α-frequency band oscillations. *Trends in Neurosciences*, *30*(4), 150-158.

Pascual-Marqui, R. D. (2002). Standardized low-resolution brain electromagnetic tomography (sLORETA): Technical details. *Methods and Findings in Experimental and Clinical Pharmacology*, *24*(Suppl. D), 5-12.

Pastor, M. C., Rehbein, M. A., Junghöfer, M., Poy, R., López, R., & Moltó, J. (2015). Facing challenges in differential classical conditioning research: Benefits of a hybrid design for simultaneous electrodermal and electroencephalographic recording. *Frontiers in Human Neuroscience*, *9*, 336.

Patton, G. C., Hibbert, M., Rosier, M. J., Carlin, J. B., Caust, J., & Bowes, G. (1996). Is smoking associated with depression and anxiety in teenagers?. *American Journal of Public Health*, *86*(2), 225-230.

Patton, G. C., McMorris, B. J., Toumbourou, J. W., Hemphill, S. A., Donath, S., & Catalano, R. F. (2004). Puberty and the onset of substance use and abuse. *Pediatrics*, *114*(3), e300-e306.

Paus, T. (2005). Mapping brain maturation and cognitive development during adolescence. *Trends in Cognitive Sciences*, *9*(2), 60-68.

Peper, J. S., Schnack, H. G., Brouwer, R. M., Van Baal, G. C. M., Pjetri, E., Szekely, E., ... & Boomsma, D. I. (2009). Heritability of regional and global brain structure at the onset of puberty: A magnetic resonance imaging study in 9-year-old twin pairs. *Human brain mapping*, *30*(7), 2184-2196.

Perrin, F., Pernier, J., Bertrand, O., & Echallier, J. F. (1989). Spherical splines for scalp potential and current density mapping. *Electroencephalography and Clinical Neurophysiology*, *72*(2), 184-187.

Perrin, J. S., Hervé, P. Y., Leonard, G., Perron, M., Pike, G. B., Pitiot, A., ... & Paus, T. (2008). Growth of white matter in the adolescent brain: Role of testosterone and androgen receptor. *Journal of Neuroscience*, *28*(38), 9519-9524.

Petanjek, Z., Judaš, M., Šimić, G., Rašin, M. R., Uylings, H. B., Rakic, P., & Kostović, I. (2011). Extraordinary neoteny of synaptic spines in the human prefrontal cortex. *Proceedings of the National Academy of Sciences*, *108*(32), 13281-13286.

Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence*, *17*(2), 117-133.

Petry, N. M. (2001). Pathological gamblers, with and without substance abuse disorders, discount delayed rewards at high rates. *Journal of Abnormal Psychology*, *110*(3), 482-487.

Pfurtscheller, G., Stancak, A., & Neuper, C. (1996). Event-related synchronization (ERS) in the alpha band — an electrophysiological correlate of cortical idling: A review. *International Journal of Psychophysiology*, *24*(1), 39-46.

Pine, D. S., Cohen, P., Gurley, D., Brook, J., & Ma, Y. (1998). The risk for early-adulthood anxiety and depressive disorders in adolescents with anxiety and depressive disorders. *Archives of General Psychiatry*, *55*(1), 56-64.

Pinyerd, B., & Zipf, W. B. (2005). Puberty—Timing is everything!. *Journal of Pediatric Nursing*, *20*(2), 75-82.

Pizzagalli, D. A., Greischar, L. L., & Davidson, R. J. (2003). Spatio-temporal dynamics of brain mechanisms in aversive classical conditioning: High-density event-related potential and brain electrical tomography analyses. *Neuropsychologia*, *41*(2), 184-194.

Pohlack, S. T., Nees, F., Ruttorf, M., Schad, L. R., & Flor, H. (2012). Activation of the ventral striatum during aversive contextual conditioning in humans. *Biological Psychology*, *91*(1), 74-80.

Powell, N. J., & Redish, A. D. (2016). Representational changes of latent strategies in rat medial prefrontal cortex precede changes in behaviour. *Nature Communications*, *7*, 12830.

Quevedo, K. M., Benning, S. D., Gunnar, M. R., & Dahl, R. E. (2009). The onset of puberty: Effects on the psychophysiology of defensive and appetitive motivation. *Development and Psychopathology*, *21*(01), 27-45.

Reardon, L. E., Leen-Feldner, E. W., & Hayward, C. (2009). A critical review of the empirical literature on the relation between anxiety and puberty. *Clinical Psychology Review*, *29*(1), 1-23.

Rellecke, J., Sommer, W., & Schacht, A. (2013). Emotion effects on the N170: A question of reference?. *Brain Topography*, *26*(1), 62-71.

Reynolds, E. K., Schreiber, W. M., Geisel, K., MacPherson, L., Ernst, M., & Lejuez, C. W. (2013). Influence of social stress on risk-taking behavior in adolescents. *Journal of Anxiety Disorders*, *27*(3), 272-277.

Richards, J. M., Patel, N., Daniele-Zegarelli, T., MacPherson, L., Lejuez, C. W., & Ernst, M. (2015). Social anxiety, acute social stress, and reward parameters interact to predict risky decision-making among adolescents. *Journal of Anxiety Disorders*, *29*, 25-34.

Ridderinkhof, K. R., Van Den Wildenberg, W. P., Segalowitz, S. J., & Carter, C. S. (2004). Neurocognitive mechanisms of cognitive control: The role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. *Brain and Cognition*, *56*(2), 129-140.

Robinson, O. J., Charney, D. R., Overstreet, C., Vytal, K., & Grillon, C. (2012). The adaptive threat bias in anxiety: amygdala–dorsomedial prefrontal cortex coupling and aversive amplification. *Neuroimage*, *60*(1), 523-529.

Romer, D., Betancourt, L., Giannetta, J. M., Brodsky, N. L., Farah, M., & Hurt, H. (2009). Executive cognitive functions and impulsivity as correlates of risk taking and problem behavior in preadolescents. *Neuropsychologia*, *47*(13), 2916-2926.

Rossion, B., & Caharel, S. (2011). ERP evidence for the speed of face categorization in the human brain: Disentangling the contribution of low-level visual cues from face perception. *Vision Research*, *51*(12), 1297-1311.

Rossion, B., Gauthier, I., Goffaux, V., Tarr, M. J., & Crommelinck, M. (2002). Expertise training with novel objects leads to left-lateralized facelike electrophysiological responses. *Psychological Science*, *13*(3), 250-257.

Rossion, B., Gauthier, I., Tarr, M. J., Despland, P., Bruyer, R., Linotte, S., & Crommelinck, M. (2000). The N170 occipito-temporal component is delayed and enhanced to inverted faces but not to inverted objects: an electrophysiological account of face-specific processes in the human brain. *Neuroreport*, *11*(1), 69-72.

Rossion, B., & Jacques, C. (2012). The N170: Understanding the time course of face perception in the human brain. In S. J. Luck & E. S. Kappenman (Eds.), *The Oxford handbook of event-related potential components* (pp. 115-141). New York, NY: Oxford University Press.

Rubia, K., Overmeyer, S., Taylor, E., Brammer, M., Williams, S. C. R., Simmons, A., ... & Bullmore, E. T. (2000). Functional frontalisation with age: Mapping neurodevelopmental trajectories with fMRI. *Neuroscience and Biobehavioral Reviews*, *24*(1), 13-19.

Sabatinelli, D., Keil, A., Frank, D. W., & Lang, P. J. (2013). Emotional perception: Correspondence of early and late event-related potentials with cortical and subcortical functional MRI. *Biological Psychology*, *92*(3), 513-519.

Sabatinelli, D., Lang, P. J., Bradley, M. M., Costa, V. D., & Keil, A. (2009). The timing of emotional discrimination in human amygdala and ventral visual cortex. J*ournal of Neuroscience*, *29*(47), 14864-14868.

Sabatinelli, D., Lang, P. J., Keil, A., & Bradley, M. M. (2007). Emotional perception:

Correlation of functional MRI and event-related potentials. *Cerebral Cortex*, *17*(5), 1085-1091.

Sadato, N., Nakamura, S., Oohashi, T., Nishina, E., Fuwamoto, Y., Waki, A., & Yonekura, Y. (1998). Neural networks for generation and suppression of alpha rhythm: A PET study. *Neuroreport*, *9*(5), 893-897.

Sadeh, B., Podlipsky, I., Zhdanov, A., & Yovel, G. (2010). Event-related potential and functional MRI measures of face-selectivity are highly correlated: A simultaneous ERP-fMRI investigation. *Human Brain Mapping*, *31*(10), 1490-1501.

Salkovskis, P. M. (1991). The importance of behaviour in the maintenance of anxiety and panic: A cognitive account. *Behavioural Psychotherapy*, *19*(1), 6-19.

Santesso, D. L., Bogdan, R., Birk, J. L., Goetz, E. L., Holmes, A. J., & Pizzagalli, D. A. (2012). Neural responses to negative feedback are related to negative emotionality in healthy adults. *Social Cognitive and Affective Neuroscience*, *7*(7), 794-803.

Santesso, D. L., Segalowitz, S. J., Ashbaugh, A. R., Antony, M. M., McCabe, R. E., & Schmidt, L. A. (2008). Frontal EEG asymmetry and sensation seeking in young adults. *Biological Psychology*, *78*(2), 164-172.

Schneider, S., Peters, J., Bromberg, U., Brassen, S., Miedl, S. F., Banaschewski, T., ... & Heinz, A. (2012). Risk taking and the adolescent reward system: A potential common link to substance abuse. *American Journal of Psychiatry*, *169*(1), 39-46.

Schneirla, T. C. (1959). An evolutionary and developmental theory of biphasic processes underlying approach and withdrawal. In M. R. Jones (Ed.) *Nebraska symposium on motivation* (pp. 1-42). Lincoln, NE: University of Nebraska Press.

Schreckenberger, M., Lange-Asschenfeld, C., Lochmann, M., Mann, K., Siessmeier, T., Buchholz, H. G., ... & Gründer, G. (2004). The thalamus as the generator and modulator of EEG alpha rhythm: A combined PET/EEG study with lorazepam challenge in humans. *Neuroimage*, *22*(2), 637-644.

Schultz, W., Apicella, P., Scarnati, E., & Ljungberg, T. (1992). Neuronal activity in monkey ventral striatum related to the expectation of reward. *Journal of Neuroscience*, *12*(12), 4595-4610.

Schupp, H. T., Cuthbert, B. N., Bradley, M. M., Cacioppo, J. T., Ito, T., & Lang, P. J. (2000). Affective picture processing: The late positive potential is modulated by motivational relevance. *Psychophysiology*, *37*(2), 257-261.

Schupp, H. T., Flaisch, T., Stockburger, J., & Junghöfer, M. (2006). Emotion and attention: Event-related brain potential studies. *Progress in Brain Research*, *156*, 31-51.

Schupp, H. T., Junghöfer, M., Weike, A. I., & Hamm, A. O. (2004). The selective processing of briefly presented affective pictures: An ERP analysis. *Psychophysiology*, *41*(3), 441-449.

Schutter, D. J., De Haan, E. H., & Van Honk, J. (2004). Anterior asymmetrical alpha

activity predicts Iowa gambling performance: Distinctly but reversed. *Neuropsychologia*, *42*(7), 939-943.

Schyns, P. G., Petro, L. S., & Smith, M. L. (2007). Dynamics of visual information integration in the brain for categorizing facial expressions. *Current Biology*, *17*(18), 1580-1585.

Sebastian, C. L., Fontaine, N. M., Bird, G., Blakemore, S. J., De Brito, S. A., McCrory, E. J., & Viding, E. (2011). Neural processing associated with cognitive and affective Theory of Mind in adolescents and adults. *Social Cognitive and Affective Neuroscience*, nsr023.

Segalowitz, S. J., Santesso, D. L., & Jetha, M. K. (2010). Electrophysiological changes during adolescence: A review. *Brain and Cognition*, *72*(1), 86-100.

Sekihara, K., Sahani, M., & Nagarajan, S. S. (2005). Localization bias and spatial resolution of adaptive and non-adaptive spatial filters for MEG source reconstruction. *Neuroimage, 25*(4), 1056-1067.

Seymour, B., Daw, N., Dayan, P., Singer, T., & Dolan, R. (2007). Differential encoding of losses and gains in the human striatum. *Journal of Neuroscience*, *27*(18), 4826-4831.

Shagass, C. (1972). Electrical activity of the brain. In N. S. Greenfield & R. A. Sternbach (Eds.), *Handbook of psychophysiology* (pp. 263–328). New York, NY: Holt, Rinehart & Winston.

Shirtcliff, E. A., Dahl, R. E., & Pollak, S. D. (2009). Pubertal development: Correspondence between hormonal and physical development. *Child Development*, *80*(2), 327-337.

Shulman, E. P., Harden, K. P., Chein, J. M., & Steinberg, L. (2014). The Development of Impulse Control and Sensation-Seeking in Adolescence: Independent or Interdependent Processes?. *Journal of Research on Adolescence*, *26*(1), 37-44.

Shulman, E. P., Harden, K. P., Chein, J. M., & Steinberg, L. (2015). Sex differences in the developmental trajectories of impulse control and sensation-seeking from early adolescence to early adulthood. *Journal of Youth and Adolescence*, *44*(1), 1-17.

Simons-Morton, B., Lerner, N., & Singer, J. (2005). The observed effects of teenage passengers on the risky driving behavior of teenage drivers. *Accident Analysis and Prevention*, *37*(6), 973-982.

Sisk, C. L., & Foster, D. L. (2004). The neural basis of puberty and adolescence. *Nature Neuroscience*, *7*(10), 1040-1047.

Sisk, C. L., & Zehr, J. L. (2005). Pubertal hormones organize the adolescent brain and behavior. *Frontiers in Neuroendocrinology*, *26*(3), 163-174.

Skinner, B. F. (1948). Superstition' in the pigeon. *Journal of Experimental Psychology*, *38*, 168-172.

Smit, D. J. A., Posthuma, D., Boomsma, D. I., & De Geus, E. J. C. (2007). The relation between frontal EEG asymmetry and the risk for anxiety and

depression. *Biological Psychology*, *74*(1), 26-33.

Smith, A. R., Steinberg, L., Strang, N., & Chein, J. (2015). Age differences in the impact of peers on adolescents' and adults' neural response to reward. *Developmental Cognitive Neuroscience*, *11*, 75-82.

Smith, D. G., Xiao, L., & Bechara, A. (2012). Decision making in children and adolescents: Impaired Iowa Gambling Task performance in early adolescence. *Developmental Psychology*, *48*(4), 1180-1187.

Solomon, B., DeCicco, J. M., & Dennis, T. A. (2012). Emotional picture processing in children: An ERP study. *Developmental Cognitive Neuroscience*, *2*(1), 110-119.

Somerville, L. H., Jones, R. M., & Casey, B. J. (2010). A time of change: Behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues. *Brain and Cognition*, *72*(1), 124-133.

Somerville, L. H., Van Den Bulk, B. G., & Skwara, A. C. (2014). Response to: ''The triadic model perspective for the study of adolescent motivated behavior''. *Brain and Cognition*, *89*, 112-113.

Somsen, R. J., Van't Klooster, B. J., Van Der Molen, M. W., Van Leeuwen, H. M., & Licht, R. (1997). Growth spurts in brain maturation during middle childhood as indexed by EEG power spectra. *Biological Psychology*, *44*(3), 187-209.

Sørensen, K., Juul, A., Christensen, K., Skytthe, A., Scheike, T., & Jensen, T. K. (2013). Birth size and age at menarche: A twin perspective. *Human Reproduction*, det283.

South, M., Dana, J., White, S. E., & Crowley, M. J. (2011). Failure is not an option: Risk-taking is moderated by anxiety and also by cognitive ability in children and adolescents diagnosed with an autism spectrum disorder. *Journal of Autism and Developmental Disorders*, *41*(1), 55-65.

Sowell, E. R., Thompson, P. M., Holmes, C. J., Jernigan, T. L., & Toga, A. W. (1999). In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. *Nature Neuroscience*, *2*(10), 859-861.

Sowell, E. R., Thompson, P. M., Tessner, K. D., & Toga, A. W. (2001). Mapping continued brain growth and gray matter density reduction in dorsal frontal cortex: Inverse relationships during postadolescent brain maturation. *Journal of Neuroscience*, *21*(22), 8819-8829.

Sowell, E. R., Trauner, D. A., Gamst, A., & Jernigan, T. L. (2002). Development of cortical and subcortical brain structures in childhood and adolescence: A structural MRI study. *Developmental Medicine and Child Neurology*, *44*(1), 4-16.

Spear, L. P. (2000). The adolescent brain and age-related behavioral manifestations. *Neuroscience and Biobehavioral Reviews*, *24*(4), 417-463.

Spear, L. P. (2009). The biology of adolescence. Paper presented at IOM Committee on the Science of Adolescence Workshop, Washington, DC.

Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., & Jacobs, G. A. (1983). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting

Psychologists Press.

Steimer, T. (2002). The biology of fear-and anxiety-related behaviors. *Dialogues in Clinical Neuroscience*, *4*(3), 231-249.

Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review*, *28*(1), 78-106.

Steinberg, L. (2010). A dual systems model of adolescent risk-taking. *Developmental Psychobiology*, *52*(3), 216-224.

Steinberg, L., Albert, D., Cauffman, E., Banich, M., Graham, S., & Woolard, J. (2008). Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: Evidence for a dual systems model. *Developmental Psychology*, *44*(6), 1764-1778.

Steinberg, L., & Morris, A. S. (2001). Adolescent development. *Journal of Cognitive Education and Psychology*, *2*(1), 55-87.

Steriade, M., Gloor, P. L. R. R., Llinas, R. R., Da Silva, F. L., & Mesulam, M. M. (1990). Basic mechanisms of cerebral rhythmic activities. *Electroencephalography and Clinical Neurophysiology*, *76*(6), 481-508.

Stroganova, T. A., Orekhova, E. V., & Posikera, I. N. (1999). EEG alpha rhythm in infants. *Clinical Neurophysiology*, *110*(6), 997-1012.

Sugase, Y., Yamane, S., Ueno, S., & Kawano, K. (1999). Global and fine information coded by single neurons in the temporal visual cortex. *Nature*, *400*(6747), 869-873.

Suldo, S. M., Olson, L. A., & Evans, J. R. (2002). Quantitative EEG evidence of increased alpha peak frequency in children with precocious reading ability. *Journal of Neurotherapy*, *5*(3), 39-50.

Surwillo, W. W. (1963). The relation of simple response time to brain-wave frequency and the effects of age. *Electroencephalography and Clinical Neurophysiology*, *15*(1), 105-114.

Surwillo, W. W. (1971). Frequency of the EEG during acquisition in short-term memory. *Psychophysiology*, *8*(5), 588-593.

Sutton, S. K., & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science*, *8*(3), 204-210.

Tamm, L., Menon, V., & Reiss, A. L. (2002). Maturation of brain function associated with response inhibition. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*(10), 1231-1238.

Taylor, M. J., Edmonds, G. E., McCarthy, G., & Allison, T. (2001). Eyes first! Eye processing develops before face processing in children. *Neuroreport*, *12*(8), 1671-1676.

Taylor, M. J., McCarthy, G., Saliba, E., & Degiovanni, E. (1999). ERP evidence of developmental changes in processing of faces. *Clinical Neurophysiology*, *110*(5),

910-915.

Tenke, C. E., & Kayser, J. (2005). Reference-free quantification of EEG spectra: Combining current source density (CSD) and frequency principal components analysis (fPCA). *Clinical Neurophysiology*, *116*(12), 2826-2846.

Thibodeau, R., Jorgensen, R. S., & Kim, S. (2006). Depression, anxiety, and resting frontal EEG asymmetry: A meta-analytic review. *Journal of Abnormal Psychology*, *115*(4), 715-729.

Thompson, P.M., Giedd, J.N., Woods, R.P., MacDonald, D., Evans, A.C., & Toga, A.W. (2000). Growth patterns in the developing brain detected by using continuum mechanical tensor maps. *Nature*, *404*, 190–193.

Thorndike, E. L. (1905). *The elements of psychology*. New York, NY: A. G. Seiler.

Tiemeier, H., Lenroot, R. K., Greenstein, D. K., Tran, L., Pierson, R., & Giedd, J. N. (2010). Cerebellum development during childhood and adolescence: A longitudinal morphometric MRI study. *Neuroimage*, *49*(1), 63-70.

Tomarken, A. J., Davidson, R. J., & Henriques, J. B. (1990). Resting frontal brain asymmetry predicts affective responses to films. *Journal of Personality and Social Psychology*, *59*(4), 791-801.

Tomarken, A. J., Davidson, R. J., Wheeler, R. E., & Doss, R. C. (1992). Individual differences in anterior brain asymmetry and fundamental dimensions of emotion. *Journal of Personality and Social Psychology*, *62*(4), 676-687.

Turner, C., & McClure, R. (2003). Age and gender differences in risk-taking behaviour as an explanation for high incidence of motor vehicle crashes as a driver in young males. *Injury Control and Safety Promotion*, *10*(3), 123-130.

Tversky, A., & Kahneman, D. (1992). Advances in prospect theory: Cumulative representation of uncertainty. *Journal of Risk and Uncertainty*, *5*(4), 297-323.

Ulmer, S., & Jansen, O. (2010). *fMRI: Basics and clinical applications*. New York, NY: Oxford University Press.

Vaca, F. E., Walthall, J. M., Ryan, S., Moriarty-Daley, A., Riera, A., Crowley, M. J., & Mayes, L. C. (2013). Adolescent Balloon Analog Risk Task and behaviors that influence risk of motor vehicle crash injury. *Annals of Advances in Automotive Medicine*, *57*, 77-88.

Van Duijvenvoorde, A. C., Peters, S., Braams, B. R., & Crone, E. A. (2016). What motivates adolescents? Neural responses to rewards and their influence on adolescents' risk taking, learning, and cognitive control. *Neuroscience and Biobehavioral Reviews*, *70*, 135-147.

Van Leijenhorst, L., Westenberg, P. M., & Crone, E. A. (2008). A developmental study of risky decisions on the cake gambling task: Age and gender analyses of probability estimation and reward evaluation. *Developmental Neuropsychology*, *33*(2), 179-196.

Van Leijenhorst, L., Zanolie, K., Van Meel, C. S., Westenberg, P. M., Rombouts, S. A., & Crone, E. A. (2010). What motivates the adolescent? Brain regions mediating

reward sensitivity across adolescence. *Cerebral Cortex*, *20*(1), 61-69.

Van Oort, F. V. A., Greaves-Lord, K., Verhulst, F. C., Ormel, J., & Huizink, A. C. (2009). The developmental course of anxiety symptoms during adolescence: The TRAILS study. *Journal of Child Psychology and Psychiatry*, *50*(10), 1209-1217.

Van Winsun, W., Sergeant, J., & Geuze, R. (1984). The functional significance of event-related desynchronization of alpha rhythm in attentional and activating tasks. *Electroencephalography and Clinical Neurophysiology*, *58*(6), 519-524.

Velo, J. R., Stewart, J. L., Hasler, B. P., Towers, D. N., & Allen, J. J. (2012). Should it matter when we record? Time of year and time of day as factors influencing frontal EEG asymmetry. *Biological Psychology*, *91*(2), 283-291.

Vigil-Colet, A. (2007). Impulsivity and decision making in the balloon analogue risk-taking task. *Personality and Individual Differences*, *43*(1), 37-45.

Viner, R. (2012). Life stage: Adolescence. In S. C. Davies (Ed.), *Annual report of the Chief Medical Officer. Our children deserve better: Prevention pays* (Chapter 8). Retrieved from: https://www.gov.uk/government/publications/chief-medical-officers-annual-report-2012-our-children-deserve-better-prevention-pays

Vuilleumier, P. (2005). How brains beware: Neural mechanisms of emotional attention. *Trends in Cognitive Sciences*, *9*(12), 585-594.

Wechsler D. (2011). *Wechsler Abbreviated Scale of Intelligence–Second Edition (WASI-II)*. San Antonio, TX: NCS Pearson.

Wheeler, R. E., Davidson, R. J., & Tomarken, A. J. (1993). Frontal brain asymmetry and emotional reactivity: A biological substrate of affective style. *Psychophysiology*, *30*(1), 82-89.

White, D., Leach, C., Sims, R., Atkinson, M., & Cottrell, D. (1999). Validation of the Hospital Anxiety and Depression Scale for use with adolescents. *British Journal of Psychiatry*, *175*(5), 452-454.

Whitford, T. J., Rennie, C. J., Grieve, S. M., Clark, C. R., Gordon, E., & Williams, L. M. (2007). Brain maturation in adolescence: concurrent changes in neuroanatomy and neurophysiology. *Human Brain Mapping*, *28*(3), 228-237.

Wiedemann, G., Pauli, P., Dengler, W., Lutzenberger, W., Birbaumer, N., & Buchkremer, G. (1999). Frontal brain asymmetry as a biological substrate of emotions in patients with panic disorders. *Archives of General Psychiatry*, *56*(1), 78-84.

Windle, M., Spear, L. P., Fuligni, A. J., Angold, A., Brown, J. D., Pine, D., ... & Dahl, R. E. (2008). Transitions into underage and problem drinking: Developmental processes and mechanisms between 10 and 15 years of age. *Pediatrics*, *121*(Suppl. 4), S273-S289.

Woodman, G. F. (2010). A brief introduction to the use of event-related potentials in studies of perception and attention. *Attention, Perception, & Psychophysics*, *72*(8), 2031-2046.

Woodward, L. J., & Fergusson, D. M. (2001). Life course outcomes of young people

with anxiety disorders in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*(9), 1086-1093.

World Health Organization (2003). *Strategic directions for improving the health and development of children and adolescents*. Geneva, Switzerland: World Health Organization.

Wu, L., Eichele, T., & Calhoun, V. D. (2010). Reactivity of hemodynamic responses and functional connectivity to different states of alpha synchrony: A concurrent EEG-fMRI study. *Neuroimage*, *52*(4), 1252-1260.

Yordanova, J., & Kolev, V. (1997). Alpha response system in children: changes with age. *International Journal of Psychophysiology*, *26*(1), 411-430.

Yurgelun-Todd, D. (2007). Emotional and cognitive changes during adolescence. *Current Opinion in Neurobiology*, *17*(2), 251-257.

Zigmond, A. S., & Snaith, R. P. (1983). The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavica*, *67*(6), 361-370.

Zimring, F. E. (1981). Kids, groups and crime: Some implications of a well-known secret. *Journal of Criminal Law and Criminology*, *72*(3), 867-885.

Zuckerman, M. (1994). *Behavioral expressions and biosocial bases of sensation seeking*. New York, NY: Cambridge University Press.

Zuckerman, M. (1996). The psychobiological model for impulsive unsocialized sensation seeking: A comparative approach. *Neuropsychobiology*, *34*(3), 125-129.

Zuckerman, M., Eysenck, S. B., & Eysenck, H. J. (1978). Sensation seeking in England and America: cross-cultural, age, and sex comparisons. *Journal of Consulting and Clinical Psychology*, *46*(1), 139-149.

Zuckerman, M., & Kuhlman, D. M. (2000). Personality and risk-taking: Common biosocial factors. *Journal of Personality*, *68*(6), 999-1029.

Appendix 1

Chapter 3 supplementary tables

Supplementary Table 3.1

Descriptive statistics for risk-taking (BART, YRBSS)

		Risk-taking								
		BART Pumps		BART Points		BART Balloons		YRBSS		
Age Group	Gender	*M*	95% CI	*M*	95% CI	*M*	95% CI	*M*	95% CI	α
Preadolescents	Females	37.72	31.95, 43.92	5514.74	4634.20, 6441.94	7.00	5.28, 9.17	0.83	0.40, 1.27	0.43
	Males	41.57	36.53, 46.18	6236.22	5501.10, 7027.70	8.78	7.14, 10.81	1.01	0.57, 1.54	0.41
	All	39.65	35.93, 43.29	5875.48	5284.82, 6476.63	7.89	6.67, 9.28	0.92	0.55, 1.28	0.40
Mid-adolescents	Females	38.76	32.12, 45.30	5748.82	4648.51, 6875.20	8.39	6.72, 9.97	2.09	1.31, 2.95	0.74
	Males	42.24	34.85, 49.20	6083.22	5217.09, 6981.17	9.16	7.03, 11.38	3.92	2.62, 5.22	0.74
	All	40.50	35.26, 45.45	5916.02	5210.14, 6600.04	8.77	7.34, 10.06	3.01	2.28, 3.74	0.77
Late Adolescents	Females	47.49	41.20, 53.46	7047.97	6126.16, 7928.95	9.65	8.17, 11.35	4.15	3.36, 4.98	0.64
	Males	48.62	42.12, 54.96	6960.28	6158.82, 7746.38	10.41	8.31, 12.67	3.30	2.21, 4.39	0.69
	All	48.06	43.59, 52.25	7004.12	6433.53, 7566.42	10.03	8.68, 11.64	3.72	3.06, 4.40	0.67

Note. BART Pumps = BART number of adjusted pumps; BART Points = BART total points won; BART Balloons = BART number of popped balloons; YRBSS = Youth Risk Behaviour Surveillance Survey; 95% CI = bootstrapped 95% confidence intervals; α = Cronbach's alpha. Means are adjusted for IQ scores.

Supplementary Table 3.2

Descriptive statistics for sensation seeking (BSSS) and impulsivity (Go/NoGo)

		Sensation seeking			Impulsivity					
		BSSS			Go Accuracy (%)		NoGo Accuracy (%)		Go RT (ms)	
Age Group	Gender	*M*	95% CI	α	*M*	95% CI	*M*	95% CI	*M*	95% CI
Preadolescents	Females	26.68	24.67, 28.89	0.62	96.61	95.19, 97.78	78.70	70.37, 86.67	449	424, 473
	Males	26.36	24.61, 28.13	0.38	94.56	91.11, 97.19	75.26	71.42, 78.93	457	436, 479
	All	26.52	25.19, 27.80	0.52	95.56	93.73, 97.14	76.94	72.34, 81.53	453	437, 469
Mid-adolescents	Females	30.78	28.20, 32.74	0.65	98.96	98.27, 99.58	81.46	75.21, 87.08	385	366, 406
	Males	27.10	23.71, 30.28	0.79	99.58	99.11, 99.93	80.21	73.96, 86.25	378	355, 403
	All	28.94	26.83, 30.85	0.77	99.27	98.82, 99.65	80.83	76.67, 84.79	382	367, 399
Late Adolescents	Females	28.30	25.40, 31.07	0.81	99.61	99.39, 99.78	83.50	77.17, 89.00	370	354, 386
	Males	26.56	23.90, 29.22	0.75	98.68	97.71, 99.51	89.58	82.92, 94.58	386	365, 409
	All	27.43	25.55, 29.31	0.78	99.20	98.61, 99.66	86.20	81.20, 89.91	378	365, 391

Note. BSSS = Brief Sensation Seeking Scale; RT = reaction time; 95% CI = bootstrapped 95% confidence intervals; α = Cronbach's alpha. Means are adjusted for IQ scores.

Supplementary Table 3.3

Descriptive statistics for anxiety (STAI), depression (HADS-D), and anxiety controlling for depression (STAI HADS-D)

		Anxiety						Depression			Anxiety cont. for depression			
		STAI-S			STAI-T			HADS-D			STAI-S		STAI-T	
Age Group	Gender	*M*	95% CI	α	*M*	95% CI	α	*M*	95% CI	α	*M*	95% CI	*M*	95% CI
Preadolescents	Females	47.58	44.15, 51.52	0.90	57.28	52.76, 62.50	0.46	2.96	2.12, 4.06	0.46	49.03	45.02, 53.61	60.45	55.00, 66.81
	Males	48.33	45.30, 51.37	0.74	57.77	52.39, 63.68	0.35	3.75	2.81, 4.79	0.35	48.42	45.54, 51.17	57.96	53.62, 62.99
	All	47.96	45.76, 50.50	0.86	57.52	54.06, 61.41	0.42	3.36	2.74, 4.20	0.42	48.73	46.23, 51.28	59.17	55.65, 62.67
Mid-adolescents	Females	48.28	43.77, 52.94	0.87	56.50	50.05, 64.11	0.59	4.01	3.03, 5.17	0.59	47.92	43.34, 52.50	55.72	50.38, 61.07
	Males	37.93	34.96, 41.00	0.90	49.20	42.60, 56.43	0.69	4.37	3.05, 5.75	0.69	36.95	33.27, 40.23	47.05	41.09, 52.87
	All	43.10	40.37, 45.97	0.87	52.85	48.00, 58.54	0.62	4.19	3.27, 5.16	0.62	42.39	39.20, 45.86	51.37	47.12, 56.22
Late Adolescents	Females	44.75	38.72, 51.79	0.96	53.00	45.16, 60.36	0.67	3.70	2.65, 4.74	0.67	44.92	38.92, 51.36	53.38	47.81, 58.31
	Males	42.43	36.71, 49.41	0.94	53.62	46.78 60.92	0.39	4.16	2.93, 5.47	0.39	41.81	36.08, 48.28	52.27	45.85, 59.80
	All	43.59	39.47, 48.25	0.95	53.31	48.20, 58.75	0.56	3.93	3.14, 4.77	0.56	43.57	39.51, 48.00	52.91	48.57, 57.27

Note. STAI-S = State Trait Anxiety Inventory-State Anxiety; STAI-T = State Trait Anxiety Inventory-Trait Anxiety; HADS-D = Hospital Anxiety and Depression Scale-Depression Scale; 95% CI = bootstrapped 95% confidence intervals; α = Cronbach's alpha. Means are adjusted for IQ scores.

Supplementary Table 3.4

Pearson correlation coefficients between risk-taking (BART, YRBSS) and anxiety (STAI), and between risk-taking (BART, YRBSS) and anxiety controlling for depression (STAI HADS-D)

			Anxiety		Anxiety controlling for depression	
Age Group	Gender	Risk-taking measure	STAI-S	STAI-T	STAI-S	STAI-T
Preadolescents	Females	BART Pumps	0.61**	0.23	0.62*	0.25
		BART Points	0.53*	0.15	0.53	0.16
		BART Balloons	0.50*	0.17	0.55*	0.27
		YRBSS	0.06	0.68**	0.04	0.65**
	Males	BART Pumps	-0.17	-0.56*	-0.06	-0.44
		BART Points	-0.08	-0.46	0.01	-0.39
		BART Balloons	-0.26	-0.27	-0.15	0.08
		YRBSS	0.39	0.44	0.30	0.23
Mid-adolescents	Females	BART Pumps	0.19	0.10	0.07	-0.21
		BART Points	0.18	0.09	0.06	-0.24
		BART Balloons	-0.00	0.02	-0.14	-0.28
		YRBSS	-0.09	-0.07	-0.19	-0.33
	Males	BART Pumps	0.04	-0.18	0.05	-0.20
		BART Points	0.10	-0.08	0.10	-0.08
		BART Balloons	0.14	-0.15	0.13	-0.22
		YRBSS	0.08	0.20	0.02	-0.01
Late Adolescents	Females	BART Pumps	-0.50*	-0.30	-0.48*	-0.17
		BART Points	-0.54*	-0.32	-0.43	-0.06
		BART Balloons	-0.50*	-0.46	-0.39	-0.33
		YRBSS	-0.22	-0.21	-0.03	-0.02
	Males	BART Pumps	-0.17	-0.18	-0.16	-0.17
		BART Points	-0.20	-0.30	-0.16	-0.24
		BART Balloons	-0.11	-0.16	-0.09	-0.14
		YRBSS	0.20	0.22	0.19	0.21

Note. STAI-S = State Trait Anxiety Inventory-State Anxiety; STAI-T = State Trait Anxiety Inventory-Trait Anxiety; HADS-D = Hospital Anxiety and Depression Scale-Depression Scale; BART Pumps = BART number of adjusted pumps; BART Points = BART total points won; BART Balloons = BART number of popped balloons; YRBSS = Youth Risk Behaviour Surveillance Survey. IQ was included as a covariate. Correlational analyses were bootstrapped using 1000 samples. The B-H procedure was used to correct for multiple comparisons and had a FDR of 0.1. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Supplementary Table 3.5

Intercorrelations between chronological age, pubertal development (PDS), and measures of risk-taking (BART, YRBSS), sensation seeking (BSSS), impulsivity (Go/NoGo), anxiety (STAI), and anxiety controlling for depression (STAI HADS-D) for females and males aged 9-17 years

		Risk-taking				Sensation seeking	Impulsivity			Anxiety		Anxiety controlling for depression	
Gender	Term	BART Pumps	BART Points	BART Balloons	YRBSS	BSSS	Go Accuracy	NoGo Accuracy	Go RT	STAI-S	STAI-T	STAI-S	STAI-T
Females	Age	0.00	-0.03	0.10	0.46**	0.23	0.42*	0.10	-0.56**	0.11	0.12	0.07	0.05
	PDS	-0.11	-0.11	0.02	0.56**	0.26	0.23	0.02	-0.51**	0.17	0.16	0.11	0.03
Males	Age	-0.10	-0.11	-0.17	0.70***	0.09	0.54**	0.29	-0.69***	-0.55**	-0.15	-0.56	-0.27
	PDS	0.02	0.01	-0.09	0.79***	0.27	0.40*	0.10	-0.53**	-0.36*	-0.06	-0.40	-0.25
Females	Age^2	0.01	-0.01	0.08	0.47	0.24	0.42*	0.10	-0.56**	0.10	0.13	0.06	0.04
	PDS^2	-0.14	-0.13	-0.02	0.60	0.25	0.20	0.03	-0.48**	0.18	0.17	0.10	0.03
Males	Age^2	-0.09	-0.10	-0.17	0.72	0.11	0.52**	0.30	-0.66***	-0.53**	-0.12	-0.55	-0.25
	PDS^2	-0.03	-0.05	-0.12	0.81	0.26	0.40*	0.13	-0.54**	-0.39*	-0.09	-0.41	-0.26

Note. BART Pumps = BART number of adjusted pumps; BART Points = BART total points won; BART Balloons = BART number of popped balloons; YRBSS = Youth Risk Behaviour Surveillance Survey; BSSS = Brief Sensation Seeking Scale; RT = reaction time; STAI-S = State Trait Anxiety Inventory-State Anxiety; STAI-T = State Trait Anxiety Inventory-Trait Anxiety; HADS-D = Hospital Anxiety and Depression Scale-Depression Scale. Correlational analyses were bootstrapped using 1000 samples. The B-H procedure was used to correct for multiple comparisons and had a FDR of 0.1. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Supplementary Table 3.6

Linear and quadratic regression coefficients for the relationships between chronological age, pubertal development (PDS), and measures of risk-taking (BART, YRBSS), sensation seeking (BSSS), impulsivity (Go/NoGo), anxiety (STAI), and anxiety controlling for depression (STAI HADS-D) for females and males aged 9-17 years

			Linear					Quadratic				
					Bootstrap					Bootstrap		
			β	*B*	*B SE*	*p*	*B* 95% CI	*β*	*B*	*B SE*	*p*	*B* 95% CI
DV: BART Pumps												
Females	Block 1	Age	0.00	-0.01	0.85	0.991	-1.49, 1.57	0.01	0.00	0.03	0.955	-0.07, 0.07
	Block 2	Age	0.24	1.31	1.58	0.402	-1.91, 4.27	0.28	0.06	0.06	0.306	-0.06, 0.15
		PDS	-0.31	-4.63	4.36	0.303	-13.63, 4.43	-0.36	-1.03	0.78	0.201	-2.55, 0.64
Males	Block 1	Age	-0.10	-0.45	0.75	0.565	-1.75, 0.71	-0.09	-0.02	0.03	0.567	-0.08, 0.04
	Block 2	Age	-0.28	-1.29	1.43	0.387	-3.80, 1.48	-0.20	-0.04	0.06	0.594	-0.16, 0.08
		PDS	0.24	3.74	5.37	0.479	-7.88, 12.72	0.13	0.43	1.37	0.777	-2.36, 3.49
DV: BART Points												
Females	Block 1	Age	-0.03	-24.69	148.96	0.859	-318.97, 271.58	-0.01	-0.31	5.49	0.968	-11.05, 9.32
	Block 2	Age	0.17	138.66	244.66	0.588	-357.96, 523.67	0.21	6.85	9.11	0.459	-13.46, 22.61
		PDS	-0.24	-570.25	708.37	0.431	-1913.44, 867.80	-0.29	-129.97	127.75	0.329	-351.04, 118.33
Males	Block 1	Age	-0.11	-70.40	104.20	0.502	-276.22, 118.12	-0.10	-2.38	4.08	0.560	-11.47, 5.60
	Block 2	Age	-0.29	-185.56	207.86	0.406	-568.46, 299.17	-0.17	-4.23	9.36	0.665	-21.76, 12.64
		PDS	0.23	513.99	759.97	0.505	-1052.42, 1726.53	0.09	41.49	189.41	0.824	-319.67, 420.02
DV: BART Balloons												
Females	Block 1	Age	0.10	0.16	0.24	0.506	-0.33, 0.64	0.08	0.01	0.01	0.597	-0.02, 0.02
	Block 2	Age	0.23	0.36	0.47	0.414	-0.77, 1.23	0.22	0.01	0.02	0.438	-0.03, 0.04
		PDS	-0.16	-0.72	1.22	0.550	-2.72, 1.74	-0.18	-0.16	0.23	0.482	-0.59, 0.37
Males	Block 1	Age	-0.17	-0.25	0.28	0.388	-0.81, 0.29	-0.17	-0.01	0.01	0.364	-0.03, 0.01
	Block 2	Age	-0.24	-0.37	0.40	0.374	-1.26, 0.54	-0.22	-0.01	0.02	0.458	-0.05, 0.03
		PDS	0.10	0.50	1.46	0.732	-2.50, 2.92	0.07	0.07	0.34	0.825	-0.55, 0.71
DV: YRBSS												
Females	Block 1	Age	0.46	0.31	0.09	0.007	0.14, 0.50	0.47	0.01	0.00	0.002	0.01, 0.02
	Block 2	Age	0.03	0.02	0.15	0.876	-0.34, 0.34	0.03	0.00	0.01	0.867	-0.01, 0.01
		PDS	0.53	1.02	0.46	0.049	0.19, 1.79	0.57	0.21	0.09	0.031	0.02, 0.37
Males	Block 1	Age	0.70	0.61	0.10	0.001	0.41, 0.80	0.72	0.02	0.00	0.001	0.02, 0.03
	Block 2	Age	0.22	0.19	0.12	0.102	-0.02, 0.39	0.16	0.01	0.01	0.355	-0.01, 0.02
		PDS	0.62	1.85	0.41	0.001	1.00, 2.77	0.68	0.42	0.12	0.002	0.20, 0.69

Supplementary Table 3.6 (continued)

Linear and quadratic regression coefficients for the relationships between chronological age, pubertal development (PDS), and measures of risk-taking (BART, YRBSS), sensation seeking (BSSS), impulsivity (Go/NoGo), anxiety (STAI), and anxiety controlling for depression (STAI HADS-D) for females and males aged 9-17 years

			Linear					Quadratic				
					Bootstrap					Bootstrap		
			β	*B*	*B SE*	*p*	*B* 95% CI	*β*	*B*	*B SE*	*p*	*B* 95% CI
DV: BSSS												
Females	Block 1	Age	0.23	0.49	0.42	0.255	-0.36, 1.24	0.24	0.02	0.02	0.283	-0.02, 0.05
	Block 2	Age	0.07	0.14	0.64	0.836	-1.51, 1.12	0.11	0.01	0.02	0.702	-0.05, 0.04
		PDS	0.21	1.22	1.24	0.299	-0.84, 3.88	0.17	0.19	0.24	0.425	-0.27, 0.84
Males	Block 1	Age	0.09	0.17	0.31	0.573	-0.49, 0.75	0.11	0.01	0.01	0.546	-0.02, 0.03
	Block 2	Age	-0.30	-0.54	0.60	0.381	-1.70, 0.92	-0.33	-0.02	0.03	0.389	-0.07, 0.03
		PDS	0.50	3.14	2.25	0.190	-1.25, 6.82	0.53	0.69	0.52	0.203	-0.19, 1.67
DV: Go accuracy												
Females	Block 1	Age	0.42	0.45	0.15	0.011	0.14, 0.78	0.42	0.02	0.01	0.004	0.01, 0.03
	Block 2	Age	0.67	0.70	0.31	0.035	0.15, 1.28	0.65	0.03	0.01	0.026	0.01, 0.05
		PDS	-0.31	-0.90	0.90	0.376	-2.94, 0.96	-0.29	-0.16	0.17	0.376	-0.54, 0.17
Males	Block 1	Age	0.54	1.05	0.34	0.018	0.44, 1.88	0.52	0.04	0.01	0.015	0.02, 0.07
	Block 2	Age	0.59	1.13	0.47	0.037	0.24, 2.03	0.58	0.04	0.02	0.047	0.01, 0.08
		PDS	-0.05	-0.36	1.31	0.752	-2.39, 3.06	-0.07	-0.10	0.28	0.712	-0.55, 0.47
DV: NoGo accuracy												
Females	Block 1	Age	0.10	0.63	1.14	0.583	-1.54, 2.87	0.10	0.03	0.04	0.566	-0.06, 0.12
	Block 2	Age	0.22	1.45	1.79	0.435	-1.82, 4.57	0.19	0.05	0.06	0.459	-0.08, 0.19
		PDS	-0.16	-2.86	4.94	0.569	-11.58, 8.00	-0.11	-0.39	0.89	0.671	-2.07, 1.23
Males	Block 1	Age	0.29	1.27	0.74	0.085	-0.17, 2.62	0.30	0.05	0.03	0.094	0.00, 0.10
	Block 2	Age	0.54	2.35	1.06	0.036	0.41, 4.61	0.57	0.10	0.05	0.055	0.01, 0.21
		PDS	-0.32	-4.81	3.80	0.199	-13.54, 2.19	-0.33	-1.03	0.90	0.241	-2.75, 0.29
DV: Go RT												
Females	Block 1	Age	-0.56	-12.80	3.41	0.001	-20.50, -5.70	-0.56	-0.50	0.13	0.001	-0.76, -0.29
	Block 2	Age	-0.41	-9.56	5.78	0.107	-21.57, 3.37	-0.46	-0.41	0.20	0.041	-0.75, -0.01
		PDS	-0.18	-11.32	17.09	0.516	-41.01, 21.30	-0.13	-1.65	2.85	0.557	-7.30, 3.61
Males	Block 1	Age	-0.69	-15.15	2.89	0.001	-21.01, -10.54	-0.66	-0.57	0.12	0.001	-0.83, -0.40
	Block 2	Age	-0.69	-15.24	3.51	0.001	-22.31, -7.83	-0.66	-0.57	0.17	0.007	-0.90, -0.30
		PDS	0.01	0.42	10.64	0.973	-23.50, 18.15	0.00	-0.07	2.83	0.985	-8.01, 6.39

Supplementary Table 3.6 (continued)

Linear and quadratic regression coefficients for the relationships between chronological age, pubertal development (PDS), and measures of risk-taking (BART, YRBSS), sensation seeking (BSSS), impulsivity (Go/NoGo), anxiety (STAI), and anxiety controlling for depression (STAI HADS-D) for females and males aged 9-17 years

			Linear					Quadratic				
					Bootstrap					Bootstrap		
			β	*B*	*B SE*	*p*	*B* 95% CI	*β*	*B*	*B SE*	*p*	*B* 95% CI
DV: STAI-S												
Females	Block 1	Age	0.11	0.42	0.65	0.516	-0.78, 1.71	0.10	0.02	0.03	0.530	-0.03, 0.06
	Block 2	Age	-0.07	-0.26	1.30	0.831	-2.83, 1.84	-0.08	-0.01	0.05	0.780	-0.11, 0.07
		PDS	0.23	2.40	3.48	0.476	-5.04, 11.08	0.24	0.48	0.62	0.409	-0.86, 1.97
Males	Block 1	Age	-0.55	-1.62	0.46	0.001	-2.57, -0.79	-0.53	-0.06	0.02	0.005	-0.10, -0.03
	Block 2	Age	-0.66	-1.96	0.75	0.017	-3.26, -0.43	-0.65	-0.08	0.03	0.038	-0.14, -0.01
		PDS	0.15	1.51	2.69	0.576	-4.29, 6.00	0.15	0.32	0.60	0.610	-0.85, 1.33
DV: STAI-T												
Females	Block 1	Age	0.12	0.65	0.87	0.446	-0.92, 2.30	0.13	0.03	0.03	0.442	-0.04, 0.09
	Block 2	Age	-0.01	-0.06	1.73	0.971	-3.64, 3.16	-0.02	0.00	0.07	0.969	-0.15, 0.11
		PDS	0.17	2.49	5.08	0.604	-7.22, 13.15	0.18	0.52	0.97	0.581	-1.22, 2.54
Males	Block 1	Age	-0.15	-0.77	0.89	0.399	-2.39, 0.58	-0.12	-0.02	0.04	0.497	-0.10, 0.03
	Block 2	Age	-0.28	-1.39	1.46	0.355	-4.13, 1.99	-0.15	-0.03	0.07	0.661	-0.15, 0.09
		PDS	0.16	2.76	5.23	0.603	-7.93, 11.40	0.03	0.12	1.19	0.925	-2.17, 2.73
DV: STAI-S HADS-D												
Females	Block 1	HADS-D	0.33	1.24	0.63	0.044	0.16, 2.60	0.33	1.24	0.65	0.045	-0.07, 2.75
	Block 2	HADS-D	0.32	1.20	0.64	0.062	0.06, 2.57	0.32	1.20	0.65	0.057	-0.10, 2.76
		Age	0.07	0.26	0.62	0.689	-1.00, 1.33	0.05	0.01	0.02	0.727	-0.04, 0.05
	Block 3	HADS-D	0.30	1.14	0.66	0.074	0.01, 2.57	0.30	1.13	0.66	0.075	-0.12, 2.61
		Age	-0.04	-0.14	1.21	0.896	-2.65, 1.86	-0.05	-0.01	0.04	0.834	-0.11, 0.07
		PDS	0.14	1.42	3.31	0.669	-4.67, 8.49	0.14	0.29	0.58	0.591	-0.80, 1.58
Males	Block 1	HADS-D	0.08	0.26	0.56	0.661	-0.73, 1.61	0.08	0.26	0.57	0.638	-0.81, 1.64
	Block 2	HADS-D	0.14	0.47	0.48	0.333	-0.49, 1.63	0.15	0.50	0.50	0.320	-0.47, 1.63
		Age	-0.56	-1.67	0.49	0.001	-2.63, -0.84	-0.55	-0.06	0.02	0.008	-0.11, -0.03
	Block 3	HADS-D	0.12	0.42	0.50	0.402	-0.66, 1.56	0.14	0.46	0.52	0.366	-0.59, 1.65
		Age	-0.64	-1.88	0.80	0.024	-3.33, -0.28	-0.64	-0.07	0.03	0.040	-0.13, -0.01
		PDS	0.10	0.99	2.75	0.739	-4.61, 5.37	0.11	0.23	0.62	0.718	-1.13, 1.38

Supplementary Table 3.6 (continued)

Linear and quadratic regression coefficients for the relationships between chronological age, pubertal development (PDS), and measures of risk-taking (BART, YRBSS), sensation seeking (BSSS), impulsivity (Go/NoGo), anxiety (STAI), and anxiety controlling for depression (STAI HADS-D) for females and males aged 9-17 years

			Linear					Quadratic				
					Bootstrap					Bootstrap		
			β	*B*	*B SE*	*p*	*B* 95% CI	*β*	*B*	*B SE*	*p*	*B* 95% CI
DV: STAI-T HADS-D												
Females	Block 1	HADS-D	0.59	3.19	1.09	0.009	0.62, 5.02	0.59	3.19	1.03	0.006	0.33, 5.00
	Block 2	HADS-D	0.58	3.15	1.15	0.013	0.65, 5.33	0.58	3.16	1.11	0.013	0.43, 5.39
		Age	0.04	0.23	0.93	0.787	-1.48, 1.61	0.03	0.01	0.03	0.834	-0.05, 0.05
	Block 3	HADS-D	0.59	3.16	1.22	0.017	0.65, 5.40	0.58	3.16	1.20	0.020	0.59, 5.46
		Age	0.06	0.29	1.37	0.846	-2.25, 2.48	0.04	0.01	0.05	0.875	-0.09, 0.08
		PDS	-0.02	-0.24	3.98	0.959	-8.12, 8.05	0.00	-0.01	0.75	0.988	-1.34, 1.59
Males	Block 1	HADS-D	0.58	3.32	1.12	0.008	0.95, 5.58	0.58	3.32	1.11	0.013	1.25, 5.24
	Block 2	HADS-D	0.60	3.46	1.14	0.007	1.11, 5.75	0.61	3.47	1.13	0.012	1.26, 5.42
		Age	-0.22	-1.11	0.61	0.092	-2.41, -0.16	-0.20	-0.04	0.03	0.145	-0.10, 0.00
	Block 3	HADS-D	0.62	3.55	1.12	0.008	1.06, 5.92	0.62	3.56	1.09	0.010	1.54, 5.43
		Age	-0.15	-0.75	1.25	0.577	-3.12, 1.70	-0.07	-0.01	0.06	0.796	-0.11, 0.09
		PDS	-0.10	-1.66	4.88	0.741	-11.77, 6.51	-0.16	-0.58	1.09	0.615	-2.95, 1.43

Note. PDS = Pubertal Developmental Scale; BART Pumps = BART number of adjusted pumps; BART Points = BART total points won; BART Balloons = BART number of popped balloons; YRBSS = Youth Risk Behaviour Surveillance Survey; BSSS = Brief Sensation Seeking Scale; RT = reaction time; STAI-S = State Trait Anxiety Inventory-State Anxiety; STAI-T = State Trait Anxiety Inventory-Trait Anxiety; HADS-D = Hospital Anxiety and Depression Scale-Depression Scale; β = standardised regression coefficient; *B* = unstandardised regression coefficient; *B SE* = standard error for the unstandardised regression coefficient; *B* 95% CI = bootstrapped 95% confidence intervals for the unstandardised regression coefficient. Regression analyses were bootstrapped using 1000 samples.

Supplementary Table 3.7

Pearson correlation coefficients between risk-taking (BART, YRBSS), sensation seeking (BSSS), and impulsivity (Go/NoGo)

			Risk-taking				Sensation seeking	Impulsivity		
Age Group	Gender	Measure	BART Pumps	BART Points	BART Balloons	YRBSS	BSSS	Go Accuracy	NoGo Accuracy	Go RT
Preadolescents	Females	BART Pumps	1.00	0.92***	0.86***	0.24	-0.08	-0.01	-0.28	-0.38
		BART Points		1.00	0.65**	0.28	-0.13	-0.13	-0.20	-0.25
		BART Balloons			1.00	0.19	0.10	0.14	-0.10	-0.22
		YRBSS				1.00	0.21	-0.33	0.05	0.06
		BSSS					1.00	-0.29	0.12	0.23
		Go Accuracy						1.00	0.30	-0.15
		NoGo Accuracy							1.00	0.73**
		Go RT								1.00
	Males	BART Pumps	1.00	0.94***	0.59*	0.01	0.03	-0.43	-0.10	0.41
		BART Points		1.00	0.39	0.10	0.01	-0.26	-0.12	0.35
		BART Balloons			1.00	0.00	0.30	-0.37	-0.45	0.23
		YRBSS				1.00	0.09	0.28	0.06	-0.39
		BSSS					1.00	-0.15	-0.34	-0.07
		Go Accuracy						1.00	0.22	-0.62**
		NoGo Accuracy							1.00	-0.01
		Go RT								1.00
Mid-adolescents	Females	BART Pumps	1.00	0.98***	0.69**	0.06	0.10	-0.05	-0.37	0.21
		BART Points		1.00	0.70**	0.12	0.08	-0.07	-0.34	0.27
		BART Balloons			1.00	0.23	0.29	-0.02	-0.33	0.49
		YRBSS				1.00	0.24	-0.04	-0.12	0.28
		BSSS					1.00	0.09	-0.19	0.03
		Go Accuracy						1.00	0.75**	0.23
		NoGo Accuracy							1.00	0.23
		Go RT								1.00
	Males	BART Pumps	1.00	0.96***	0.72**	0.03	0.37	0.09	-0.16	-0.35
		BART Points		1.00	0.54*	0.05	0.39	0.02	-0.11	-0.24
		BART Balloons			1.00	-0.05	0.28	0.14	-0.06	-0.35
		YRBSS				1.00	0.73**	0.13	-0.19	-0.23
		BSSS					1.00	0.33	-0.19	-0.33
		Go Accuracy						1.00	-0.12	-0.63
		NoGo Accuracy							1.00	0.63*
		Go RT								1.00

Supplementary Table 3.7 (continued)

Pearson correlation coefficients between risk-taking (BART, YRBSS), sensation seeking (BSSS), and impulsivity (Go/NoGo)

			Risk-taking				Sensation seeking	Impulsivity		
Age Group	Gender	Measure	BART Pumps	BART Points	BART Balloons	YRBSS	BSSS	Go Accuracy	NoGo Accuracy	Go RT
Late Adolescents	Females	BART Pumps	1.00	0.93***	0.78***	-0.26	-0.02	0.42	0.04	-0.40
		BART Points		1.00	0.60**	-0.28	0.05	0.39	0.05	-0.30
		BART Balloons			1.00	-0.06	0.16	0.52	-0.11	-0.42
		YRBSS				1.00	0.78***	0.00	-0.38	-0.13
		BSSS					1.00	0.29	-0.40	-0.34
		Go Accuracy						1.00	-0.11	-0.63**
		NoGo Accuracy							1.00	0.47
		Go RT								1.00
	Males	BART Pumps	1.00	0.86***	0.82***	0.33	0.28	-0.01	-0.27	-0.09
		BART Points		1.00	0.64*	0.47	0.21	0.30	-0.46	-0.33
		BART Balloons			1.00	0.40	0.21	0.15	0.04	-0.23
		YRBSS				1.00	0.37	0.20	-0.28	-0.30
		BSSS					1.00	-0.12	-0.34	0.20
		Go Accuracy						1.00	-0.25	-0.74**
		NoGo Accuracy							1.00	0.33
		Go RT								1.00

Note. BART Pumps = BART number of adjusted pumps; BART Points = BART total points won; BART Balloons = BART number of popped balloons; YRBSS = Youth Risk Behaviour Surveillance Survey; BSSS = Brief Sensation Seeking Scale; RT = reaction time. IQ was included as a covariate. Correlational analyses were bootstrapped using 1000 samples. The B-H procedure was used to correct for multiple comparisons and had a FDR of 0.1. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Appendix 2

Chapter 4 supplementary tables

Supplementary Table 4.1

Instrumental task performance

		Accuracy (% correct)								Reaction Time (ms)			
		Reward Block *M* [95% CI]				Avoidance Block *M* [95% CI]				Reward Block *M* [95% CI]		Avoidance Block *M* [95% CI]	
Age Group	Gender	RG	RN	CG	CN	AG	AN	CG	CN	RG	CG	AG	CG
Preadolescents	Females	95.88 [94.01, 97.74]	98.24 [97.09, 99.40]	88.51 [82.11, 94.91]	96.93 [94.80, 99.06]	95.70 [92.78, 98.62]	98.07 [96.57, 99.58]	88.25 [80.47, 96.02]	98.42 [97.54, 99.30]	416 [377, 456]	422 [389, 454]	401 [361, 441]	404 [363, 446]
	Males	95.17 [92.86, 97.49]	94.65 [91.48, 97.82]	86.49 [80.70, 92.28]	94.65 [91.27, 98.03]	95.53 [92.29, 98.76]	98.33 [97.25, 99.41]	89.65 [83.06, 96.24]	98.42 [96.49, 100.35]	448 [406, 490]	450 [415, 484]	428 [380, 475]	425 [380, 471]
	All	95.53 [93.99, 96.97]	96.45 [94.30, 98.06]	87.50 [82.85, 91.36]	95.79 [93.81, 97.54]	95.61 [93.37, 97.45]	98.20 [97.20, 98.99]	88.95 [83.66, 93.33]	98.42 [97.32, 99.18]	432 [406, 459]	436 [413, 458]	414 [389, 444]	415 [389, 442]
Mid-adolescents	Females	96.14 [94.16, 98.12]	99.03 [98.19, 99.88]	94.82 [92.01, 97.64]	99.03 [97.99, 100.08]	97.19 [95.48, 98.91]	99.47 [99.01, 99.93]	96.75 [94.57, 98.94]	99.74 [99.33, 100.15]	353 [323, 384]	361 [331, 391]	368 [341, 396]	364 [338, 390]
	Males	97.72 [96.25, 99.19]	98.60 [97.41, 99.78]	95.35 [92.22, 98.48]	98.07 [96.42, 99.72]	98.68 [97.05, 100.31]	99.47 [98.81, 100.14]	96.93 [94.95, 98.91]	99.82 [99.57, 100.08]	394 [366, 423]	390 [355, 424]	373 [335, 411]	376 [332, 419]
	All	96.93 [95.66, 98.07]	98.82 [98.12, 99.47]	95.09 [93.00, 97.20]	98.55 [97.41, 99.60]	97.94 [96.75, 98.97]	99.47 [99.03, 99.82]	96.84 [95.04, 98.23]	99.78 [99.52, 99.96]	374 [354, 392]	375 [355, 392]	371 [351, 388]	370 [348, 389]
Late Adolescents	Females	98.83 [98.02, 99.64]	99.05 [98.38, 99.71]	97.44 [96.00, 98.89]	98.10 [95.96, 100.24]	97.95 [96.74, 99.17]	99.34 [98.78, 99.90]	97.37 [95.77, 98.97]	99.49 [98.98, 100.00]	345 [325, 365]	349 [331, 368]	325 [299, 350]	321 [294, 348]
	Males	97.04 [95.11, 98.96]	99.26 [98.63, 99.88]	96.22 [93.70, 98.74]	99.26 [98.41, 100.11]	98.85 [98.23, 99.47]	99.51 [99.07, 99.94]	98.03 [97.03, 99.02]	99.59 [99.17, 100.01]	372 [342, 403]	372 [342, 403]	341 [306, 377]	352 [312, 391]
	All	97.99 [96.90, 98.88]	99.15 [98.68, 99.53]	96.87 [95.47, 98.03]	98.65 [97.25, 99.54]	98.37 [97.72, 98.99]	99.42 [99.07, 99.69]	97.68 [96.87, 98.45]	99.53 [99.15, 99.81]	358 [341, 375	360 [344, 377]	333 [314, 351]	335 [315, 356]

Note. RG = Reward^{Go}; RN = Reward^{NoGo}; AG = Avoidance^{Go}; AN = Avoidance^{NoGo}; CG = Control^{Go}; CN = Control^{NoGo}; 95% CI = bootstrapped 95% confidence intervals.

Supplementary Table 4.2

Pearson correlation coefficients between LPP potentiation and reaction times to Go trials in the reward and avoidance blocks

			Reward Block		Avoidance Block	
Age Group	Gender	Measure	Reward^{Go} RT	Control^{Go} RT	Avoidance^{Go} RT	Control^{Go} RT
Preadolescents	Females	LPP Reward^{Go}-Control^{Go}	-0.31	-0.20		
		LPP Avoidance^{Go}-Control^{Go}			-0.41	-0.54
	Males	LPP Reward^{Go}-Control^{Go}	-0.50	-0.34		
		LPP Avoidance^{Go}-Control^{Go}			-0.20	-0.16
Mid-adolescents	Females	LPP Reward^{Go}-Control^{Go}	0.46	0.38		
		LPP Avoidance^{Go}-Control^{Go}			-0.47	-0.30
	Males	LPP Reward^{Go}-Control^{Go}	0.21	0.22		
		LPP Avoidance^{Go}-Control^{Go}			0.14	0.26
Late Adolescents	Females	LPP Reward^{Go}-Control^{Go}	0.28	0.25		
		LPP Avoidance^{Go}-Control^{Go}			0.33	0.26
	Males	LPP Reward^{Go}-Control^{Go}	-0.25	-0.31		
		LPP Avoidance^{Go}-Control^{Go}			-0.17	-0.15

Note. LPP = late positive potential; RT = reaction time. Correlational analyses were bootstrapped using 1000 samples. The B-H procedure was used to correct for multiple comparisons and had a FDR of 0.1. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Supplementary Table 4.3

Pearson correlation coefficients between LPP potentiation in the reward and avoidance blocks and measures of risk-taking (BART, YRBSS), anxiety (STAI), and anxiety controlling for depression (STAI HADS-D)

			Risk-taking				Anxiety		Anxiety cont. depression	
Age Group	Gender	Measure	BART Pumps	BART Points	BART Balloons	YRBSS	STAI-S	STAI-T	STAI-S	STAI-T
Preadolescents	Females	LPP Reward^{Go}-Control^{Go}	0.41	0.32	0.47	-0.07				
		LPP Reward^{NoGo}-Control^{NoGo}	0.13	-0.06	0.32	0.36				
		LPP Avoidance^{Go}-Control^{Go}					-0.32	-0.36	-0.31	-0.37
		LPP Avoidance^{NoGo}-Control^{NoGo}					-0.03	0.14	0.09	0.26
	Males	LPP Reward^{Go}-Control^{Go}	0.10	0.29	0.06	-0.03				
		LPP Reward^{NoGo}-Control^{NoGo}	-0.50	-0.47	-0.06	0.25				
		LPP Avoidance^{Go}-Control^{Go}					-0.06	0.48	-0.24	0.19
		LPP Avoidance^{NoGo}-Control^{NoGo}					0.55	0.25	0.56	0.35
Mid-adolescents	Females	LPP Reward^{Go}-Control^{Go}	0.15	0.18	0.02	0.14				
		LPP Reward^{NoGo}-Control^{NoGo}	-0.22	-0.22	0.01	0.15				
		LPP Avoidance^{Go}-Control^{Go}					-0.03	-0.28	0.18	-0.12
		LPP Avoidance^{NoGo}-Control^{NoGo}					0.41	0.32	0.37	0.27
	Males	LPP Reward^{Go}-Control^{Go}	0.13	0.25	-0.10	-0.01				
		LPP Reward^{NoGo}-Control^{NoGo}	-0.37	-0.37	-0.34	-0.10				
		LPP Avoidance^{Go}-Control^{Go}					0.14	-0.04	0.12	-0.19
		LPP Avoidance^{NoGo}-Control^{NoGo}					0.25	-0.28	0.30	-0.17
Late Adolescents	Females	LPP Reward^{Go}-Control^{Go}	-0.21	-0.15	-0.05	0.07				
		LPP Reward^{NoGo}-Control^{NoGo}	0.26	0.19	0.20	0.18				
		LPP Avoidance^{Go}-Control^{Go}					0.24	0.10	0.37	0.17
		LPP Avoidance^{NoGo}-Control^{NoGo}					0.32	0.18	0.33	0.13
	Males	LPP Reward^{Go}-Control^{Go}	-0.29	-0.18	-0.30	-0.34				
		LPP Reward^{NoGo}-Control^{NoGo}	-0.32	-0.32	-0.32	-0.53				
		LPP Avoidance^{Go}-Control^{Go}					-0.02	0.01	-0.13	-0.29
		LPP Avoidance^{NoGo}-Control^{NoGo}					-0.10	-0.04	-0.13	-0.11

Note. LPP = late positive potential; BART Pumps = BART number of adjusted pumps; BART Points = BART total points won; BART Balloons = BART number of popped balloons; YRBSS = Youth Risk Behaviour Surveillance Survey; STAI-S = State Trait Anxiety Inventory-State Anxiety; STAI-T = State Trait Anxiety Inventory-Trait Anxiety; HADS-D = Hospital Anxiety and Depression Scale-Depression Scale. Correlational analyses were bootstrapped using 1000 samples. The B-H procedure was used to correct for multiple comparisons and had a FDR of 0.1. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Supplementary Table 4.4

Intercorrelations between chronological age, pubertal development (PDS), and N170 and LPP potentiation for the reward and avoidance blocks for females and males aged 9-17 years

		N170								LPP			
		$Reward^{Go}$ - $Control^{Go}$		$Reward^{NoGo}$ - $Control^{NoGo}$		$Avoidance^{Go}$ - $Control^{Go}$		$Avoidance^{NoGo}$ - $Control^{NoGo}$		$Reward^{Go}$ - $Control^{Go}$	$Reward^{NoGo}$ - $Control^{NoGo}$	$Avoidance^{Go}$ - $Control^{Go}$	$Avoidance^{NoGo}$ - $Control^{NoGo}$
Gender	Term	L	R	L	R	L	R	L	R				
Females	Age	-0.37*	-0.40*	-0.04	0.06	0.17	0.12	-0.37*	-0.30	-0.23	-0.12	-0.26	-0.08
	PDS	-0.36*	-0.40*	0.10	-0.10	0.07	-0.03	-0.26	-0.43*	-0.19	-0.14	-0.11	-0.07
Males	Age	-0.08	0.02	0.09	0.29	-0.27	-0.15	-0.04	0.10	-0.26	0.05	-0.15	-0.27
	PDS	-0.19	-0.09	0.23	0.21	-0.26	-0.20	-0.07	0.20	-0.27	0.20	-0.15	-0.14
Females	Age^2	-0.35	-0.38*	-0.05	0.05	0.16	0.09	-0.35	-0.29	-0.24	-0.12	-0.25	-0.02
	PDS^2	-0.35	-0.39*	0.08	-0.11	0.07	-0.04	-0.24	-0.43*	-0.19	-0.15	-0.11	-0.05
Males	Age^2	-0.09	0.04	0.08	0.26	-0.26	-0.14	-0.04	0.11	-0.26	0.05	-0.15	-0.28
	PDS^2	-0.18	-0.06	0.21	0.19	-0.27	-0.22	-0.09	0.14	-0.30	0.20	-0.16	-0.16

Note. LPP = late positive potential; PDS = Pubertal Developmental Scale; L = left occipitotemporal cluster; R = right occipitotemporal cluster. Correlational analyses were bootstrapped using 1000 samples. The B-H procedure was used to correct for multiple comparisons and had a FDR of 0.1. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Supplementary Table 4.5

Linear and quadratic regression coefficients for the relationships between chronological age, pubertal development (PDS), and N170 potentiation for the reward and avoidance blocks for females and males aged 9-17 years

			Linear					Quadratic				
					Bootstrap					Bootstrap		
			β	*B*	*B SE*	*p*	*B* 95% CI	β	*B*	*B SE*	*p*	*B* 95% CI
Reward Block												
DV: N170 Reward^{Go} – Control^{Go} Left												
Females	Block 1	Age	-0.37	-17.21	8.60	0.066	-38.28, -0.30	-0.35	-0.63	0.32	0.073	-1.35, -0.02
	Block 2	Age	-0.21	-9.76	9.56	0.332	-31.48, 8.56	-0.20	-0.36	0.34	0.315	-1.12, 0.12
		PDS	-0.20	-27.15	43.58	0.533	-109.75, 68.15	-0.20	-5.15	7.28	0.488	-19.57, 14.30
Males	Block 1	Age	-0.08	-3.46	9.73	0.716	-22.59, 16.65	-0.09	-0.16	0.34	0.649	-0.87, 0.49
	Block 2	Age	0.19	8.34	14.80	0.574	-21.19, 41.10	0.16	0.29	0.55	0.605	-0.82, 1.38
		PDS	-0.34	-48.44	38.85	0.211	-118.01, 23.89	-0.31	-9.26	7.86	0.201	-23.52, 7.08
DV: N170 Reward^{Go} – Control^{Go} Right												
Females	Block 1	Age	-0.40	-17.85	7.73	0.035	-31.73, -2.77	-0.38	-0.66	0.28	0.024	-1.18, -0.14
	Block 2	Age	-0.23	-9.98	9.28	0.290	-29.87, 7.76	-0.22	-0.37	0.32	0.235	-1.02, 0.15
		PDS	-0.22	-28.66	31.32	0.368	-88.21, 35.29	-0.22	-5.42	5.70	0.338	-16.80, 8.21
Males	Block 1	Age	0.02	0.55	6.62	0.930	-10.92, 11.93	0.04	0.05	0.25	0.838	-0.43, 0.48
	Block 2	Age	0.22	7.04	9.91	0.495	-9.90, 25.20	0.26	0.33	0.39	0.398	-0.46, 1.05
		PDS	-0.26	-26.62	24.87	0.280	-75.42, 24.72	-0.27	-5.90	5.06	0.213	-15.31, 5.40
DV: N170 Reward^{NoGo} – Control^{NoGo} Left												
Females	Block 1	Age	-0.04	-1.25	5.50	0.820	-12.97, 10.17	-0.05	-0.07	0.20	0.721	-0.48, 0.34
	Block 2	Age	-0.32	-10.94	11.26	0.318	-34.02, 6.71	-0.27	-0.36	0.39	0.324	-1.38, 0.31
		PDS	0.35	35.30	32.38	0.268	-20.49, 116.19	0.28	5.49	5.72	0.331	-3.11, 18.24
Males	Block 1	Age	0.09	2.58	5.64	0.651	-8.47, 12.05	0.08	0.09	0.21	0.678	-0.41, 0.58
	Block 2	Age	-0.23	-6.89	10.55	0.519	-27.55, 12.01	-0.28	-0.33	0.40	0.413	-1.21, 0.47
		PDS	0.40	38.87	30.23	0.189	-26.38, 99.37	0.44	8.79	6.21	0.138	-1.86, 21.08
DV: N170 Reward^{NoGo} – Control^{NoGo} Right												
Females	Block 1	Age	0.06	2.82	8.09	0.733	-11.36, 18.36	0.05	0.09	0.29	0.781	-0.42, 0.64
	Block 2	Age	0.37	18.68	14.01	0.198	-6.93, 51.47	0.31	0.61	0.52	0.229	-0.14, 1.87
		PDS	-0.39	-57.78	37.63	0.123	-137.25, -0.45	-0.35	-9.85	7.15	0.173	-24.45, 0.41
Males	Block 1	Age	0.29	12.43	6.56	0.076	-2.22, 26.44	0.26	0.43	0.26	0.114	-0.15, 0.98
	Block 2	Age	0.33	14.06	10.13	0.156	-7.41, 37.68	0.31	0.52	0.42	0.201	-0.21, 1.50
		PDS	-0.05	-6.69	33.47	0.813	-76.58, 49.24	-0.06	-1.78	6.90	0.778	-17.17, 9.40

Supplementary Table 4.5 (continued)

Linear and quadratic regression coefficients for the relationships between chronological age, pubertal development (PDS), and N170 potentiation for the reward and avoidance blocks for females and males aged 9-17 years

			Linear					Quadratic				
					Bootstrap					Bootstrap		
			β	*B*	*B SE*	*p*	*B* 95% CI	β	*B*	*B SE*	*p*	*B* 95% CI
Avoidance Block												
DV: N170 Avoidance^{Go} – Control^{Go} Left												
Females	Block 1	Age	0.17	8.70	9.58	0.377	-13.42, 28.93	0.16	0.31	0.36	0.381	-0.50, 1.09
	Block 2	Age	0.33	16.47	10.89	0.123	-4.15, 33.55	0.26	0.50	0.36	0.127	-0.19, 1.18
		PDS	-0.19	-28.28	40.66	0.470	-100.21, 75.18	-0.13	-3.57	6.86	0.595	-15.59, 10.83
Males	Block 1	Age	-0.27	-12.05	8.20	0.155	-28.49, 2.06	-0.26	-0.44	0.32	0.184	-1.09, 0.15
	Block 2	Age	-0.19	-8.22	13.73	0.560	-34.69, 20.76	-0.11	-0.20	0.55	0.742	-1.30, 0.79
		PDS	-0.11	-15.75	40.90	0.682	-108.63, 53.58	-0.18	-5.20	9.41	0.602	-23.74, 16.33
DV: N170 Avoidance^{Go} – Control^{Go} Right												
Females	Block 1	Age	0.12	6.29	9.71	0.516	-12.44, 24.49	0.09	0.19	0.38	0.596	-0.54, 1.08
	Block 2	Age	0.39	20.73	12.53	0.102	-0.46, 42.51	0.30	0.62	0.47	0.137	-0.21, 1.72
		PDS	-0.34	-52.59	35.15	0.137	-123.16, 29.53	-0.27	-8.13	6.59	0.218	-22.55, 5.26
Males	Block 1	Age	-0.15	-6.49	8.99	0.488	-26.89, 8.10	-0.14	-0.23	0.35	0.530	-1.04, 0.40
	Block 2	Age	0.01	0.54	17.36	0.970	-37.00, 36.50	0.13	0.22	0.68	0.753	-1.28, 1.45
		PDS	-0.21	-28.88	48.75	0.566	-136.02, 60.55	-0.33	-9.46	10.61	0.432	-28.40, 14.14
DV: N170 Avoidance^{NoGo} – Control^{NoGo} Left												
Females	Block 1	Age	-0.37	-14.46	5.56	0.012	-24.46, -5.06	-0.35	-0.53	0.20	0.012	-0.95, -0.16
	Block 2	Age	-0.44	-17.57	10.67	0.095	-40.49, 2.43	-0.40	-0.61	0.37	0.078	-1.42, 0.00
		PDS	0.10	11.35	35.40	0.756	-60.43, 78.60	0.07	1.48	6.37	0.805	-11.25, 14.88
Males	Block 1	Age	-0.04	-1.40	5.88	0.803	-12.02, 11.61	-0.04	-0.07	0.22	0.774	-0.43, 0.40
	Block 2	Age	0.04	1.65	9.33	0.868	-18.73, 21.28	0.09	0.13	0.37	0.721	-0.61, 0.89
		PDS	-0.10	-12.49	36.78	0.713	-72.06, 63.42	-0.16	-4.12	7.41	0.550	-15.35, 12.86
DV: N170 Avoidance^{NoGo} – Control^{NoGo} Right												
Females	Block 1	Age	-0.30	-16.34	12.21	0.195	-39.28, 8.07	-0.29	-0.60	0.44	0.187	-1.51, 0.21
	Block 2	Age	0.13	7.26	15.19	0.620	-24.10, 45.08	0.11	0.22	0.49	0.629	-0.73, 1.29
		PDS	-0.54	-85.95	37.41	0.032	-166.10, -22.70	-0.51	-15.56	6.03	0.021	-28.01, -4.14
Males	Block 1	Age	0.10	4.25	7.96	0.605	-11.23, 21.67	0.11	0.17	0.29	0.544	-0.43, 0.74
	Block 2	Age	-0.13	-5.33	15.62	0.745	-34.47, 35.88	-0.01	-0.02	0.63	0.969	-1.35, 1.39
		PDS	0.30	39.34	50.96	0.504	-56.28, 115.62	0.15	4.09	10.87	0.736	-13.75, 23.66

Note. PDS = Pubertal Developmental Scale; Left = left occipitotemporal cluster; Right = right occipitotemporal cluster; β = standardised regression coefficient; *B* = unstandardised regression coefficient; *B SE* = standard error for the unstandardised regression coefficient; *B* 95% CI = 95% confidence intervals for the unstandardised regression coefficient. Regression analyses were bootstrapped using 1000 samples.

Supplementary Table 4.6

Linear and quadratic regression coefficients for the relationships between chronological age, pubertal development (PDS), and LPP potentiation for the reward and avoidance blocks for females and males aged 9-17 years

			Linear					Quadratic				
					Bootstrap					Bootstrap		
			β	*B*	*B SE*	*p*	*B* 95% CI	β	*B*	*B SE*	*p*	*B* 95% CI
Reward Block												
DV: LPP Reward^{Go} – Control^{Go}												
Females	Block 1	Age	-0.23	-33.94	22.59	0.141	-85.50, 5.85	-0.24	-1.36	0.87	0.130	-3.05, 0.30
	Block 2	Age	-0.20	-30.72	38.07	0.401	-99.87, 67.97	-0.22	-1.30	1.36	0.323	-3.37, 2.03
		PDS	-0.03	-11.72	127.33	0.924	-276.24, 168.38	-0.02	-1.24	24.07	0.965	-54.25, 33.82
Males	Block 1	Age	-0.26	-48.38	39.12	0.237	-125.66, 31.91	-0.26	-1.94	1.46	0.207	-4.65, 0.72
	Block 2	Age	-0.11	-19.88	59.98	0.762	-136.38, 122.84	-0.05	-0.34	2.39	0.874	-4.48, 3.77
		PDS	-0.19	-117.02	154.46	0.422	-395.37, 139.77	-0.26	-33.22	33.97	0.312	-102.33, 60.51
DV: LPP Reward^{NoGo} – Control^{NoGo}												
Females	Block 1	Age	-0.12	-19.66	32.25	0.542	-77.72, 27.63	-0.12	-0.71	1.17	0.543	-2.83, 1.29
	Block 2	Age	-0.02	-3.45	40.75	0.937	-80.35, 56.18	0.00	0.02	1.37	0.984	-2.90, 2.32
		PDS	-0.13	-59.04	127.20	0.633	-296.14, 233.70	-0.15	-13.94	21.72	0.500	-58.16, 33.75
Males	Block 1	Age	0.05	6.59	29.04	0.796	-49.24, 65.96	0.05	0.24	1.11	0.815	-1.78, 2.43
	Block 2	Age	-0.28	-37.52	46.68	0.430	-134.94, 63.30	-0.35	-1.88	1.93	0.342	-5.77, 2.43
		PDS	0.42	181.07	125.84	0.155	-39.13, 410.15	0.49	44.20	27.28	0.118	-12.38, 90.04
Avoidance Block												
DV: LPP Avoidance^{Go} – Control^{Go}												
Females	Block 1	Age	-0.26	-53.14	39.95	0.200	-127.81, 38.69	-0.25	-1.93	1.47	0.190	-4.50, 0.88
	Block 2	Age	-0.49	-99.29	56.97	0.096	-216.90, 41.74	-0.39	-3.04	1.90	0.110	-7.00, 1.54
		PDS	0.29	168.08	150.98	0.252	-147.31, 447.12	0.19	21.16	27.31	0.416	-32.35, 70.56
Males	Block 1	Age	-0.15	-23.62	25.75	0.361	-74.16, 27.49	-0.15	-0.98	0.98	0.296	-3.28, 1.05
	Block 2	Age	-0.08	-12.12	49.91	0.816	-102.66, 120.60	-0.07	-0.41	1.84	0.820	-3.74, 3.75
		PDS	-0.09	-47.21	156.58	0.749	-468.08, 191.40	-0.11	-11.75	30.12	0.669	-81.65, 42.12
DV: LPP Avoidance^{NoGo} – Control^{NoGo}												
Females	Block 1	Age	-0.08	-14.37	45.36	0.765	-101.25, 80.65	-0.02	-0.15	1.73	0.927	-3.40, 2.93
	Block 2	Age	-0.06	-11.83	63.12	0.877	-167.40, 91.43	0.04	0.30	2.16	0.881	-4.95, 3.32
		PDS	-0.02	-9.26	166.82	0.954	-371.56, 336.18	-0.08	-8.67	27.49	0.759	-63.65, 50.43
Males	Block 1	Age	-0.27	-39.55	29.87	0.203	-99.22, 22.95	-0.28	-1.60	1.09	0.162	-3.75, 0.62
	Block 2	Age	-0.42	-60.56	56.63	0.304	-164.73, 73.43	-0.45	-2.55	2.18	0.242	-6.97, 3.00
		PDS	0.19	86.22	139.69	0.539	-219.30, 295.08	0.20	19.76	28.88	0.467	-44.76, 64.12

Note. LPP = late positive potential; PDS = Pubertal Developmental Scale; β = standardised regression coefficient; *B* = unstandardised regression coefficient; *B SE* = standard error for the unstandardised regression coefficient; *B* 95% CI = 95% confidence intervals for the unstandardised regression coefficient. Regression analyses were bootstrapped using 1000

Appendix 3

Chapter 5 supplementary tables

Supplementary Table 5.1

Intercorrelations between chronological age, pubertal development (PDS), and alpha CSD in the sLORETA ROIs during the eyes-open and eyes-closed conditions for females and males aged 9-17 years

		Eyes-open				Eyes-closed			
Gender	Term	mPFC	DLPFC	OFC	Occipital	mPFC	DLPFC	OFC	Occipital
Females	Age	-0.60**	-0.60**	-0.59**	-0.46*	-0.54**	-0.52**	-0.48*	-0.40*
	PDS	-0.53**	-0.58**	-0.56**	-0.57**	-0.58**	-0.58**	-0.51**	-0.48**
Males	Age	-0.50**	-0.48**	-0.43*	-0.15	-0.35	-0.38*	-0.24	-0.15
	PDS	-0.53**	-0.53**	-0.54**	-0.44*	-0.52**	-0.56**	-0.46*	-0.38*
Females	Age^2	-0.57**	-0.56**	-0.56**	-0.44*	-0.51**	-0.48**	-0.44*	-0.38*
	PDS^2	-0.49**	-0.54**	-0.51**	-0.55**	-0.54**	-0.54**	-0.47*	-0.45*
Males	Age^2	-0.48**	-0.47**	-0.42*	-0.15	-0.36	-0.39*	-0.25	-0.15
	PDS^2	-0.51**	-0.52**	-0.51**	-0.39*	-0.52**	-0.56**	-0.46*	-0.36*

Note. mPFC = medial prefrontal cortex; DLPFC = dorsolateral prefrontal cortex; OFC = orbitofrontal cortex; Occipital = occipital cortex. Correlational analyses were bootstrapped using 1000 samples. The B-H procedure was used to correct for multiple comparisons and had a FDR of 0.1. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Supplementary Table 5.2

Linear and quadratic regression coefficients for the relationships between chronological age, pubertal development (PDS), and alpha CSD in the sLORETA ROIs (mPFC, DLPFC, OFC, occipital cortex) during the eyes-open and eyes-closed conditions for females aged 9-17 years

			Linear					Quadratic				
					Bootstrap					Bootstrap		
			β	*B*	*B SE*	*p*	*B* 95% CI	*β*	*B*	*B SE*	*p*	*B* 95% CI
DV: mPFC												
Eyes-open	Block 1	Age	-0.60	-0.02	0.01	0.004	-0.03, -0.01	-0.57	0.00	0.00	0.007	0.00, 0.00
	Block 2	Age	-0.47	-0.01	0.01	0.026	-0.03, 0.00	-0.46	0.00	0.00	0.021	0.00, 0.00
		PDS	-0.16	-0.01	0.02	0.424	-0.05, 0.04	-0.16	0.00	0.00	0.442	-0.01, 0.00
Eyes-closed	Block 1	Age	-0.54	-0.02	0.01	0.095	-0.03, -0.01	-0.51	0.00	0.00	0.084	0.00, 0.00
	Block 2	Age	-0.23	-0.01	0.01	0.187	-0.02, 0.01	-0.24	0.00	0.00	0.121	0.00, 0.00
		PDS	-0.41	-0.04	0.03	0.252	-0.10, 0.00	-0.36	-0.01	0.00	0.206	-0.02, 0.00
DV: DLPFC												
Eyes-open	Block 1	Age	-0.60	-0.02	0.01	0.009	-0.03, -0.01	-0.56	0.00	0.00	0.021	0.00, 0.00
	Block 2	Age	-0.37	-0.01	0.01	0.065	-0.03, 0.00	-0.36	0.00	0.00	0.067	0.00, 0.00
		PDS	-0.29	-0.03	0.02	0.155	-0.06, 0.02	-0.27	-0.01	0.00	0.176	-0.01, 0.00
Eyes-closed	Block 1	Age	-0.52	-0.02	0.01	0.125	-0.03, -0.01	-0.48	0.00	0.00	0.115	0.00, 0.00
	Block 2	Age	-0.16	-0.01	0.01	0.341	-0.01, 0.01	-0.19	0.00	0.00	0.211	0.00, 0.00
		PDS	-0.46	-0.04	0.03	0.263	-0.12, 0.00	-0.41	-0.01	0.00	0.200	-0.02, 0.00
DV: OFC												
Eyes-open	Block 1	Age	-0.59	-0.02	0.01	0.006	-0.04, -0.01	-0.56	0.00	0.00	0.009	0.00, 0.00
	Block 2	Age	-0.40	-0.02	0.01	0.049	-0.03, 0.00	-0.39	0.00	0.00	0.024	0.00, 0.00
		PDS	-0.25	-0.03	0.02	0.278	-0.07, 0.02	-0.23	-0.01	0.00	0.249	-0.01, 0.00
Eyes-closed	Block 1	Age	-0.48	-0.03	0.01	0.096	-0.05, -0.01	-0.44	0.00	0.00	0.109	0.00, 0.00
	Block 2	Age	-0.20	-0.01	0.01	0.326	-0.03, 0.01	-0.22	0.00	0.00	0.221	0.00, 0.00
		PDS	-0.36	-0.06	0.04	0.259	-0.17, 0.02	-0.31	-0.01	0.01	0.255	-0.03, 0.00
DV: Occipital												
Eyes-open	Block 1	Age	-0.46	-0.05	0.02	0.071	-0.08, -0.02	-0.44	0.00	0.01	0.089	0.00, 0.00
	Block 2	Age	-0.05	-0.01	0.02	0.697	-0.02, 0.04	-0.09	0.00	0.01	0.430	0.00, 0.00
		PDS	-0.53	-0.15	0.08	0.154	-0.33, -0.05	-0.48	-0.03	0.02	0.155	-0.06, -0.01
Eyes-closed	Block 1	Age	-0.40	-0.21	0.10	0.084	-0.42, -0.03	-0.38	-0.01	0.01	0.111	-0.02, 0.00
	Block 2	Age	-0.07	-0.04	0.10	0.732	-0.23, 0.19	-0.10	0.00	0.01	0.553	-0.01, 0.00
		PDS	-0.43	-0.61	0.36	0.126	-1.47, -0.03	-0.38	-0.11	0.06	0.138	-0.24, -0.01

Note. PDS = Pubertal Developmental Scale; mPFC = medial prefrontal cortex; DLPFC = dorsolateral prefrontal cortex; OFC = orbitofrontal cortex; Occipital = occipital cortex; *β* = standardised regression coefficient; *B* = unstandardised regression coefficient; *B SE* = standard error for the unstandardised regression coefficient; *B* 95% CI = 95% confidence intervals for the unstandardised regression coefficient. Regression analyses were bootstrapped using 1000 samples.

Supplementary Table 5.3

Linear and quadratic regression coefficients for the relationships between chronological age, pubertal development (PDS), and alpha CSD in the sLORETA ROIs (mPFC, DLPFC, OFC, occipital cortex) during the eyes-open and eyes-closed conditions for males aged 9-17 years

			Linear					Quadratic				
					Bootstrap					Bootstrap		
			β	*B*	*B SE*	*p*	*B* 95% CI	*β*	*B*	*B SE*	*p*	*B* 95% CI
DV: mPFC												
Eyes-open	Block 1	Age	-0.50	-0.01	0.00	0.020	-0.02, 0.00	-0.48	0.00	0.00	0.022	0.00, 0.00
	Block 2	Age	-0.21	-0.01	0.01	0.399	-0.02, 0.00	-0.20	0.00	0.00	0.461	0.00, 0.00
		PDS	-0.37	-0.03	0.01	0.081	-0.05, 0.00	-0.35	-0.01	0.00	0.155	-0.01, 0.00
Eyes-closed	Block 1	Age	-0.35	-0.01	0.00	0.027	-0.01, 0.00	-0.36	0.00	0.00	0.016	0.00, 0.00
	Block 2	Age	0.12	0.00	0.01	0.688	-0.01, 0.01	0.19	0.00	0.00	0.524	0.00, 0.00
		PDS	-0.61	-0.04	0.02	0.090	-0.09, 0.00	-0.67	-0.01	0.00	0.059	-0.02, 0.00
DV: DLPFC										0.01		
Eyes-open	Block 1	Age	-0.48	-0.01	0.01	0.026	-0.02, -0.01	-0.47	0.00	0.00	0.029	0.00, 0.00
	Block 2	Age	-0.18	-0.01	0.01	0.474	-0.02, 0.01	-0.15	0.00	0.00	0.579	0.00, 0.00
		PDS	-0.39	-0.03	0.02	0.048	-0.06, 0.00	-0.39	-0.01	0.00	0.082	-0.02, 0.00
Eyes-closed	Block 1	Age	-0.38	-0.01	0.00	0.021	-0.02, 0.00	-0.39	0.00	0.00	0.014	0.00, 0.00
	Block 2	Age	0.14	0.00	0.01	0.569	-0.01, 0.02	0.20	0.00	0.00	0.430	0.00, 0.00
		PDS	-0.67	-0.05	0.02	0.012	-0.09, -0.01	-0.72	-0.01	0.00	0.011	-0.02, 0.00
DV: OFC												
Eyes-open	Block 1	Age	-0.43	-0.01	0.01	0.050	-0.02, 0.00	-0.42	0.00	0.00	0.041	0.00, 0.00
	Block 2	Age	-0.02	0.00	0.01	0.933	-0.02, 0.01	0.00	0.00	0.00	0.987	0.00, 0.00
		PDS	-0.52	-0.05	0.02	0.043	-0.08, 0.00	-0.51	-0.01	0.01	0.068	-0.02, 0.00
Eyes-closed	Block 1	Age	-0.24	-0.01	0.01	0.124	-0.02, 0.00	-0.25	0.00	0.00	0.122	0.00, 0.00
	Block 2	Age	0.29	0.01	0.01	0.211	-0.01, 0.03	0.38	0.00	0.00	0.164	0.00, 0.00
		PDS	-0.69	-0.08	0.03	0.015	-0.14, -0.01	-0.77	-0.02	0.01	0.011	-0.03, -0.01
DV: Occipital												
Eyes-open	Block 1	Age	-0.15	-0.01	0.01	0.266	-0.03, 0.01	-0.15	0.00	0.00	0.253	0.00, 0.00
	Block 2	Age	0.49	0.04	0.03	0.437	-0.02, 0.09	0.49	0.00	0.00	0.447	0.00, 0.00
		PDS	-0.82	-0.20	0.13	0.345	-0.45, 0.03	-0.79	-0.04	0.03	0.329	-0.09, 0.00
Eyes-closed	Block 1	Age	-0.15	-0.08	0.07	0.338	-0.22, 0.06	-0.15	0.00	0.00	0.349	-0.01, 0.00
	Block 2	Age	0.37	0.18	0.17	0.271	-0.16, 0.51	0.43	0.01	0.01	0.226	0.00, 0.02
		PDS	-0.67	-1.05	0.58	0.076	-2.26, 0.17	-0.71	-0.23	0.12	0.051	-0.47, -0.03

Note. PDS = Pubertal Developmental Scale; mPFC = medial prefrontal cortex; DLPFC = dorsolateral prefrontal cortex; OFC = orbitofrontal cortex; Occipital = occipital cortex; *β* = standardised regression coefficient; *B* = unstandardised regression coefficient; *B SE* = standard error for the unstandardised regression coefficient; *B* 95% CI = 95% confidence intervals for the unstandardised regression coefficient. Regression analyses were bootstrapped using 1000 samples.

Supplementary Table 5.4

Pearson correlation coefficients for the relationships between sLORETA ROI alpha asymmetry scores (mPFC, DLPFC, OFC) and measures of risk-taking (BART, YRBSS) during the eyes-open and eyes-closed conditions

			Eyes-open			Eyes-closed		
Age Group	Gender	Measure	mPFC	DLPFC	OFC	mPFC	DLPFC	OFC
Preadolescents	Females	BART Pumps	-0.03	-0.15	-0.04	0.08	0.18	0.19
		BART Points	0.04	-0.10	0.01	0.07	0.13	0.18
		BART Balloons	-0.18	-0.31	-0.26	0.03	0.07	0.09
		YRBSS	0.29	0.31	0.16	0.25	0.39	0.22
	Males	BART Pumps	0.08	0.00	0.11	-0.31	-0.34	-0.24
		BART Points	-0.01	0.00	0.13	-0.25	-0.32	-0.24
		BART Balloons	0.22	0.04	0.14	-0.27	-0.16	0.15
		YRBSS	-0.06	0.07	0.29	0.07	0.37	0.39
Mid-adolescents	Females	BART Pumps	0.12	0.39	-0.31	0.05	0.52	0.41
		BART Points	0.10	0.41	-0.29	0.09	0.57*	0.45
		BART Balloons	0.18	0.29	-0.05	0.14	0.44	0.44
		YRBSS	0.38	0.63*	0.38	0.15	0.30	0.37
	Males	BART Pumps	0.43	0.28	0.43	-0.14	-0.16	0.20
		BART Points	0.51	0.42	0.57*	-0.14	-0.15	0.28
		BART Balloons	0.08	-0.06	0.12	-0.16	-0.22	-0.05
		YRBSS	0.40	0.39	0.21	0.52	0.51	0.47
Late Adolescents	Females	BART Pumps	0.20	0.18	0.08	-0.18	0.00	-0.16
		BART Points	0.18	0.19	0.06	-0.22	-0.01	-0.11
		BART Balloons	-0.06	-0.13	-0.08	-0.13	-0.17	-0.39
		YRBSS	-0.28	-0.25	-0.19	0.18	-0.10	-0.07
	Males	BART Pumps	-0.05	-0.13	-0.22	-0.11	0.30	0.03
		BART Points	-0.07	-0.11	-0.39	-0.05	0.20	0.03
		BART Balloons	0.11	-0.20	-0.04	-0.08	0.06	0.00
		YRBSS	0.08	-0.57*	-0.04	0.18	-0.03	0.24

Note. mPFC = medial prefrontal cortex; DLPFC = dorsolateral prefrontal cortex; OFC = orbitofrontal cortex; BART Pumps = BART number of adjusted pumps; BART Points = BART total points won; BART Balloons = BART number of popped balloons; YRBSS = Youth Risk Behaviour Surveillance Survey. Correlational analyses were bootstrapped using 1000 samples. The B-H procedure was used to correct for multiple comparisons and had a FDR of 0.1. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Supplementary Table 5.5

Pearson correlation coefficients for the relationships between scalp alpha asymmetry scores (F4-F3, F8-F7) and measures of risk-taking (BART, YRBSS) during the eyes-open and eyes-closed conditions

			Eyes-open		Eyes-closed	
Age Group	Gender	Measure	F4-F3	F8-F7	F4-F3	F8-F7
Preadolescents	Females	BART Pumps	-0.13	0.29	-0.10	0.38
		BART Points	-0.14	0.29	-0.14	0.43
		BART Balloons	-0.17	0.20	-0.04	0.18
		YRBSS	0.46	0.30	0.54	0.17
	Males	BART Pumps	0.08	0.23	-0.01	-0.06
		BART Points	0.06	0.19	-0.02	0.06
		BART Balloons	0.43	0.40	0.16	-0.09
		YRBSS	0.17	-0.04	0.04	0.42
Mid-adolescents	Females	BART Pumps	0.33	0.46	0.31	0.53
		BART Points	0.33	0.43	0.28	0.50
		BART Balloons	0.12	0.18	0.02	0.18
		YRBSS	0.35	0.21	0.28	0.36
	Males	BART Pumps	-0.20	-0.08	-0.24	0.08
		BART Points	-0.13	0.09	-0.20	0.20
		BART Balloons	-0.38	-0.38	-0.40	-0.24
		YRBSS	0.34	0.06	0.31	0.07
Late Adolescents	Females	BART Pumps	0.33	0.10	0.29	-0.10
		BART Points	0.32	0.15	0.31	-0.06
		BART Balloons	-0.11	-0.17	-0.26	-0.33
		YRBSS	-0.44	-0.07	-0.36	0.12
	Male	BART Pumps	-0.19	-0.32	-0.19	-0.32
		BART Points	-0.15	-0.23	-0.15	-0.23
		BART Balloons	-0.17	-0.28	-0.17	-0.28
		YRBSS	0.09	0.33	0.09	0.33

Note. BART Pumps = BART number of adjusted pumps; BART Points = BART total points won; BART Balloons = BART number of popped balloons; YRBSS = Youth Risk Behaviour Surveillance Survey. Correlational analyses were bootstrapped using 1000 samples. The B-H procedure was used to correct for multiple comparisons and had a FDR of 0.1. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.