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The effects of nicotine on music-induced emotion and perception

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Abstract

This thesis investigates why nicotine is often consumed in the context of music. Nicotine and music both independently increase physiological and emotional indices of arousal and pleasure, however less is known about these responses when they occur together.

Study one tests the effects of nicotine on music-induced emotion in smokers and nonsmokers (n = 125) and overall finds trends indicative of additive effects (although nonsignificant) on the physiological and emotional responses of listeners. However, nonsmokers experienced negative side effects, such as a decrease in arousal and pleasure, due to their lack of tolerance for nicotine. To disassociate the effects of nicotine (e.g. increase in arousal, increase in pleasure) study two tests the effects of caffeine on music-induced emotion in smokers and nonsmokers (n = 120). Caffeine was predicted to only increase arousal without influencing pleasure, but increased both and had additive effects on the physiological and emotional responses to music. It is proposed that these additive effects occur through nicotine and caffeine's ability to increase the reward value of other stimuli and through excitation transfer, where increased physiological arousal from pharmacological substances amplifies the emotions experienced during music listening.

Following on from the above physiological studies, Study three examines how nicotine affects auditory information processing in nonsmokers (n = 36) using ERP (event related potentials) techniques. Nicotine decreases habituation, reflected by an increase in the P2 amplitude in the frontal region. Nicotine therefore reduces listeners' disengagement from repetition in music, thereby increasing familiarity and music-induced emotion.

These results agree with Dibben (2004) who found increased physiological arousal from exercise to intensify music-induced emotions and with Domino & Kishimoto (2002) who found nicotine to decrease habituation in nonsmokers during frequently occurring tones. Overall, this thesis suggests that music-induced emotion and musical engagement are enhanced as a result of nicotine consumption.

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List of abbreviations

Ach = Acetylcholine

AEP = Auditory evoked potentials

ANS = Autonomic nervous system

C = Central cortical area

CNS = Central nervous system

EEG = Electroencephalogram

ERP = Event related potentials

F = Frontal cortical area

HR = Heart rate

N1/N100 = First negative peak of an ERP wave

N2/N200 = Second negative peak of an ERP wave

NAcc = Nucleus accumbens

nAChR = Nicotinic acetylcholine receptor

O = Occipital cortical area

P = Parietal cortical area

P1/P100 = First positive peak of an ERP wave

P2/P200 = Second positive peak of an ERP wave

PAC = Primary auditory cortex

RT = Reaction time

SCL = Skin conductance level

SCR = Skin conductance response

STESS = Subjective Treatment Emergent Symptom Scale

z = Midline of scalp

1. Chapter one: Drug consumption in the context of music

1.1. Drug consumption in the context of music

Drugs are widely consumed in the context of music. Musicians are notorious for taking drugs while performing and creating music (Berridge, 1988) and many musicians have died of drug overdoses, including Jimmi Hendrix and Janis Joplin. However, it is not only performers that combine music and drugs, but also music-listeners. Students are renowned for drinking at dance clubs (Clapp et al., 2007) and illicit drugs are commonly self-administered in musical settings. Some obvious examples include ecstasy at dance-music events or raves (Forsyth, Barnard, & McKeganey, 1997; Saunders, 1995), where 96% of attendees self-reported using ecstasy (Winstock, Griffiths, & Stewart, 2001), and cannabis smoking at the 1969 Woodstock Festival (Musto, 1991), where 99% of attendees were speculated to have smoked marijuana (Sheehy, 2012).

Interestingly, nicotine is a psychoactive substance that is commonly consumed in the context of music. Cigarettes are prevalent among young adults (Conrad, Flay, & Hill, 1992) and college students (Wechsler, Rigotti, Gledhill-Hoyt, & Lee, 1998). This demographic is known to be most engaged with music (Hargreaves & North, 1997) and to attend music festivals (Packer & Ballantyne, 2010; Woodward, Taylor, & Bennett, 2014). Furthermore, nicotine products are consumed in musical settings, such as music festivals (Mackuľak et al., 2015). But why would music listening coincide with nicotine consumption? What characteristics do these activities share that encourage individuals to engage in both simultaneously?

It could be argued that music listening and cigarette smoking both contain a social aspect and are therefore highly likely to be consumed together in any social setting (including a musical one). Indeed, a review of past literature suggests this. That is, shared experiences during music listening have been regarded as highly positive experiences (Lamont, 2011). Likewise, there

are social smokers who only smoke cigarettes when with others (Gilpin, White, & Pierce, 2005; Moran, Wechsler, & Rigotti, 2004). College freshman describe this as 'party smoking' or 'weekend smoking' (Colder et al., 2006). This suggests that music listening co-occurs with cigarette smoking because these two activities commonly occur within a context or setting that is inherently social. For example, studies have found correlations between music listening and smoking cigarettes. For example, a preference for rap/hip-hop music was associated with an increase in smoking among adolescent girls compared to adults (Mulder et al., 2009) and a survey conducted at the Roskilde Festival in Denmark found that new onset of tobacco use was reported in 9.2% of never-smokers and resumption of tobacco use was reported by 24% of past year tobacco abstainers during the festival (Hesse, Tutenges, & Schliewe, 2010). Furthermore, many music videos portray smokers as successful and attractive (Gutschoven & Van den Bulck, 2004), which can lead to observational learning by teenagers, those most likely to watch music videos (Sun & Lull, 1986). For example, research has shown that even modest amounts of viewing music videos can result in substantial exposure to glamorized images of tobacco (DuRant et al., 1997). Indeed, adolescents who engaged in more risky behaviors, including smoking cigarettes, listened to the radio and watch music videos and television more frequently than those who in engaged in fewer risky behaviors (Klein et al., 1993) and past research has consistently found that watching positive images of others consume tobacco products on television were related to teenagers taking up smoking (Gidwani, Sobol, DeJong, Perrin, & Gortmaker, 2002; Pechmann & Shih, 1999; Sargent et al., 2001).

While no previous study has found a direct causal increase in smoking as a result of music listening, past research does suggest that music provides an ideal context for cigarette smoking. Additionally, this indicates a gap in the literature regarding the relationship between tobacco use and music consumption, indicating that that further research is needed to understand the reasons behind their co-consumption.

In addition to there being social reason why music listing and nicotine are consumed together, I suspect that there are also emotional and physiological reasons that help explain the co-occurrence of music with nicotine.

For instance, listening to music can affect one's emotions (Juslin & Västfjäll, 2008) and physiology (Khalfa, Peretz, Jean-Pierre, & Manon, 2002), especially when measured by arousal and pleasure (Blood & Zatorre, 2001; Salimpoor, Benovoy, Longo, Cooperstock, & Zatorre, 2009). The same is also true for nicotine (Benowitz, 2010; Nesbitt, 1973). Because cigarette smoking and music listening can independently increase arousal and pleasure, together they may be able to produce additive effects on these measurements in individuals who engaged in both activities simultaneously. That is, music listening and nicotine in combination may produce a total effect on pleasure, arousal, or both, that is equal to the effects that occur from both stimuli (music listening; nicotine) independently. Furthermore, there may be super additive or sub-additive effects, whereby the total effect on an individual's pleasure, arousal, or both is significantly greater than or less than the effects that occur from each stimuli independently. For example, it may be that nicotine consumption increases pleasure and arousal, and leads to an enhancement of music-induced emotion, again either by increasing pleasure, arousal, or both. Then, this enhancement of emotion may reinforce the co-consumption of nicotine and music, causing it to be repeated.

Understanding how music listening and nicotine affect emotion and physiological arousal, both alone and in combination, will help to improve the psychological and physiological health of smoking individuals as well discourage nonsmokers from taking up nicotine consumption. For example, for smokers, it is likely that music can be used as a non-nicotine replacement therapy. Although music would not replace any of the behavioral activities of smoking (e.g. hand/motor movements, oral/gustative sensations) it can be used as an emotional coping mechanism during the presence of withdrawal symptoms. More specifically, smokers in the acute stage of withdrawal typically experience stress and anxiety (Hughes, Higgins, & Bickel, 1994) and previous research has shown a dramatic decrease in feelings of reward during smoking abstinence (Al-Adawi & Powell, 1997; De Biasi & Dani, 2011). Importantly, one of the main reasons individuals report listening to music is for emotional manipulation, including stress reduction (Juslin & Sloboda, 2010). Furthermore, listening to some types of music has been shown to reduce stress and anxiety (Davis &

Thaut, 1989; Labbé, Schmidt, Babin, & Pharr, 2007) as well as increase feelings of reward and pleasure (Blood & Zatorre, 2001). Therefore, listening to music during smoking cessation may help decrease negative emotion and increase positive emotion in abstaining smokers. Additionally, when music listening is used in combination with other smoking interventions (e.g. nicotine replacement therapy) it may help to improve the low success rates of smoking cessation (~10-20% at 6-12 months) (Franklin et al., 2007). For example, it may be possible for smokers to replace smoking a cigarette with listening to certain types of music. Therefore, one goal of this thesis is to identify which emotional categories of music are best suited for non-nicotine replacement therapy.

The knowledge gained from thesis can also be used to teach individuals, particularly adolescents, about the detrimental consequences of smoking tobacco and how similar increases in physiological arousal can be obtained from music listening and nicotine. For example, if similar increases in physiological arousal and emotional responses are found between music listening and nicotine then it may be possible to deter young adults from taking up smoking and instead encourage them to listen to music. Furthermore, understanding why music and nicotine are consumed together can potentially help us explain why drug consumption in general is so prevalent in a musical context. It may be that in combination music and substances of abuse enhance emotional reactions and therefore encourage the use of one another. Lastly, because nicotine is a stimulant and increases arousal it can potentially facilitate cognitive processes. For example, it can enhance the speed and accuracy with which one can process incoming information by improving selective attention and divided attention (Heishman, Taylor, & Henningfield, 1994) as well as by preventing performance decrements (Frankenhaeuser, Myrsten, Post, & Johansson, 1971; Myrsten, Andersson, Frankenhaeuser, & Elgerot, 1975). Therefore, nicotine may also be able to facilitate the processing of auditory information, for example, to allow listeners to better understand fast and complex music or slower and simpler music if they are tired. Since the density of information in music can be carefully controlled in an experimental environment one can manipulate the

dose of nicotine to investigate the drug's effect on auditory information processing.

Based on the above premise that nicotine and music listening may be able to produce an additive effect on emotions and physiological arousal the rest of this chapter is aimed at explaining and discussing 1) the psychological constructs of arousal and pleasure and 2) describing how music and nicotine independently affect emotion and physiological arousal.

1.2. Emotion, arousal, and pleasure

The subjective feelings and associated physiological states termed emotions are key features of the human experience (Purves et al., 2008). Currently there is no agreed upon definition of emotion (Frijda, 2007; Russell & Barrett, 1999; Scherer, 2005), evidenced for example, by a study surveying thirty-three experts which found no consensus when asked to define emotion (Izard, 2007). However, there is some agreement that suggests emotions to have more than one psychological or behavioral manifestation. That is, in addition to subjective feeling, emotions also contain action tendencies, physiological arousal, cognitive appraisals, and expressive motor behavior (Niedenthal, Krauth-Gruber, & Ric, 2006; Scherer, 2005). Furthermore, the circumplex model of emotion suggests that there are two fundamental bi-polar dimensions of emotion, arousal and valence (Russell, 1980). This model constructs emotion (or core affect) on a two-dimensional circular structure with arousal representing the vertical (y) axis and valence represents the horizontal (x) axis. The center of the model represents a space where arousal and valence are neutral (e.g. neither high nor low levels of arousal; neither positive nor negative levels of valence). This model can therefore be used to represent emotions based on any combination of arousal and valence levels. Furthermore, these two dimensions represent core affect, the most elementary and raw affective feelings that are nonreflective and do not necessarily need to be consciously direct towards anything specific (Russell, 2003; Russell & Barrett, 1999). However, emotions that are elicited by a specific object (e.g. music; nicotine) are better termed emotional episodes and can be plotted on a circumplex model using the orthogonal dimensions of arousal and pleasure (Russell & Barrett, 1999).

Arousal can be defined as stimulation of the autonomic nervous system (ANS) and is therefore associated with physiological changes in heart rate, breathing, skin temperature, skin conductance and other bodily responses. Such changes have been observed in response to emotional stimuli (e.g. music) as well as non-emotional stimuli (e.g. exercise, drugs, sexual activity) and as a dimension of emotion it is ranges from low to high. The work of William James (1884) was the first to postulate the idea that the experience of emotion stems from the self-perception of visceral activities (Becker, 2010; Dibben, 2004). Current theories of emotion, such as the cognitive appraisal theory, have built on this, suggesting that when an event is cognitively appraised (evaluated) it leads to physiological changes in the body that facilitate action (e.g. running from a bear) and expressive behavior (e.g. screaming). In this way, emotions are rooted in our evaluations of events, which in turn lead to physiological changes/sensations that we then experience. These evaluations and resulting sensations are thought to be the experience of emotion (Scherer, 1999). Other research has suggested that the physiological changes experienced during an event can also influence emotion. That is, the arousal experienced in response to a cognitive appraisal can intensify the emotions experienced (Philippot, Chapelle, & Blairy, 2002). In this way, physiological arousal and emotion are coupled, but it has yet to be determined whether one precedes the other. It is beyond the scope of this thesis to test the chronological events of emotional episodes. However, the idea that physiological arousal is a key component of emotion and that it can amplify emotions suggests that extraneous increases in arousal (e.g. from stimulant drugs) may influence a subsequent emotional experience (e.g. music listening).

Pleasure is defined as the hedonic impact of a stimulus (Berridge & Robinson, 1998) and is a fundamental property of emotion (Titchener, 1908). Importantly, pleasure is viewed as a dimension of emotion, but is not an emotion in itself (Scherer, 2005). Pleasure is measured from unpleasant to pleasant. Unlike arousal, which can be measured with self-reports as well as physiological indices, pleasure is typically measured through self-reports. This is because self-reports are able to capture personal and subjective experiences

(Tiffany, Carter, & Singleton, 2000) better than physiological and behavioral measures.

Previous literature has suggested pleasure to be an immediate and automatic evaluation that precedes cognition (Zajonc, 1980). However, others suggest pleasure to be a function of two cognitive appraisals, whereby pleasure can be experienced either by attaining a goal that one wants or by avoiding an event or stimuli that one does not want (Roseman, 1984). Similarly, Scherer (1982) suggests that stimuli are intrinsically pleasant or unpleasant, but that our cognitive evaluation of them influence our experience of pleasure. That is, our evaluation of whether a stimuli in pleasurable will depend on their relevance to our current goals. In this way, pleasant stimuli that interrupt our goals will be evaluated negatively and therefore seen as unpleasant.

Interestingly, there are different categories of pleasure. Damasio (1999) suggested that pleasures arising from social and physical antecedents may stem from evolutionary goals. For example, the social pleasure gained from a strong family bond helps protect the family or group from foreign enemies, and as such enables survival, while the physical pleasure of sex encourages the activity and so helps perpetuate the species (Berridge & Kringelbach, 2008; Levitin, 2008). However, pleasures arising from intellectual and emotional antecedents may be less straightforward and as such may be characterized as convoluted 'pleasures of the mind' (Dube & La Bel, 2003). For example, emotional pleasures require complex appraisal and consist of negative emotions, such as sadness and guilt, as well as positive emotions. Furthermore, an experience of emotional pleasure is likely to begin with joyful anticipation before the antecedent is encountered (Dube & La Bel, 2003), a claim corroborated with musical stimuli (Salimpoor et al., 2011) and drugs of abuse (Blood & Zatorre, 2001). The notion that some stimuli contain an element of joyful anticipation may suggest that when music listening and nicotine are consumed together they be able to modulate the experience of pleasure in a cumulative fashion.

1.3. Nicotine: Mechanism of action

Nicotine is a naturally occurring substance found in the leaves of the tobacco plant *Nicotiana tabacum*. It is a legal and freely available drug that is commonly self-administered in many forms, including pulmonary inhalation through cigarettes, cigars, pipe tobacco, and electronic cigarettes (e-cigarettes), along with absorption through oral mucosa using snuff, chewing tobacco, gum, and lozenges, and absorption through the skin using transdermal patches (Benowitz, Porchet, Sheiner, & Jacob, 1988; Regan, Promoff, Dube, & Arrazola, 2013; Tro, 2009).

After inhaling smoke from a cigarette, nicotine is distilled from the tobacco and its smoke particles are carried into the alveoli of the lungs. From here it is absorbed quickly into the pulmonary venous circulation, after which it enters the arterial circulation and is rapidly transported to the brain (Benowitz, 2010). This process takes only 10-20 s (Benowitz, Hukkanen, & Jacob, 2009). Other methods of nicotine delivery, including smokeless tobacco, gum, and nicotine patches have slower absorption and decay rates, but nonetheless transport nicotine into the blood stream and across the blood brain barrier (Digard, Proctor, Kulasekaran, Malmqvist, & Richter, 2013; Schneider, Lunell, Olmstead, & Fagerström, 1996). Once nicotine enters the blood stream it interacts with nicotinic acetylcholine receptors (nAChRs). These receptors are found throughout the brain and body, including sites in the central nervous system, sensory nerve endings, neuromuscular junctions, and the adrenal medulla (Benowitz, 2010; Clarke, 1987). Several different subtypes of nAChRs exist and each has its own pharmacological and physiological profile along with its own distinct distribution in the brain (Paterson & Nordberg, 2000). This helps explain the multiple effects that nicotine has in humans (Benowitz, 1996). In general, the activation of nAChRs via nicotine increases physiological indices (Agué, 1974; Frankenhauser, Myrsten, & Post, 1970; Frankenhauser, Myrsten, Waszack, Neri, & Post, 1968) and causes the user to feel alert and attentive (Tro, 2009).

Neuronal nAChRs are ligand-gated cation channels with a pentameric structure and a central pore with a cation gate, which is necessary for ion selectivity and permeability. These receptors usually bind acetylcholine,

however, they also respond to nicotine. Binding of nicotine to its extracellular binding site leads to a conformational change of the central pore, which opens the ion channel and allows the entry of Na^+ or Ca^+ (Benowitz, 2010; Haass & Kübler, 1997). One effect of Ca^+ entering the neuron is the release of neurotransmitters (Benowitz, 2010; Dajas-Bailador & Wonnacott, 2004). Importantly, neuronal nAChRs modulate synaptic transmission by regulating the release of norepinephrine, acetylcholine, serotonin, γ-aminobutyric acid (GABA), glutamate, and endorphins. Nicotine also releases growth hormone, prolactin, adrenocorticotropic hormone (ACTH), and cortisol, all which mediate different behaviors (Benowitz, 2010; Rao, Correa, Adams, Santori, & Sacaan, 2003). Most important to nicotine addiction are the central nAChRs (Benowitz, 2010; Brody, 2006). Nicotine's stimulation of central nAChRs leads to the release of dopamine in the mesolimbic area, the corpus striatum, and the frontal cortex. Most notable within the mesolimbic area are the dopaminergic neurons of the ventral tegmental area (VTA) in the midbrain, and the release of dopamine in the shell of the nucleus accumbens (NAcc), which are strongly implicated in and critical for drug-induced reward a (Dani & De Biasi, 2001; Nestler, 2005). Nicotine also augments the release of glutamate and GABA, which facilitates and inhibits dopamine release respectively. With chronic exposure to nicotine, some nAChRs become desensitized, while others do not. Because of this GABA-mediated inhibitory tone diminishes, while glutamate-mediated excitation continues. This in turn increases the excitation of dopaminergic neurons and enhances the responsiveness to nicotine.

Other neurotransmitters released by nicotine, such as serotonin, result in reduced food consumption and may act as an antidepressant (Ribeiro, Bettiker, Bogdanov, & Wurtman, 1993). Nicotine also stimulates sympathetic neurotransmission, as it stimulates catecholamine release by activating nAChRs localized on peripheral postganglionic sympathetic nerve endings and the adrenal medulla. This leads to an increase in NE (norepinephrine) and results in cardiovascular effects, including an increased HR (Haass & Kübler, 1997). Lastly, nicotine has been shown to release β endorphins, which are at least partially implicated in the antinociceptive effects of the drug (Benowitz, 1996; Seyler, Pomerleau, Fertig, Hunt, & Parker, 1986).

1.4. Music affects emotion

Humans place such a high value on music because of its powerful ability to evoke emotion in listeners, making emotional manipulation one of the primary reasons behind listening to music (Sloboda, 1991). As we know, listening to music is a pleasurable experience, indicated by its ability to evoke such intense responses as thrills, tears, pleasure, and reward (Blood & Zatorre, 2001; Juslin & Västfjäll, 2008; Khalfa et al., 2002; Zentner, Grandjean, & Scherer, 2008). Furthermore, it has been shown to employ the same cerebral processing pathway for pleasure as biological pleasure antecedents (e.g. food, sex) (Gebauer, Kringelbach, & Vuust, 2012), such as the dopaminergic system implicated in reward and motivation (Berridge & Robinson, 1998; Menon & Levitin, 2005). Nicotine and music therefore share the mesolimbic pathway as both are rewarding stimuli, demonstrating their commonalities in eliciting reward for those who engage in their activities.

It may seem ironic that listening to music evokes pleasure because it has little in common with other reward stimuli. A strong emotional response such as pleasure typically exists either (1) with a clear biological purpose such as survival (e.g. eating) or species perpetuation (e.g. love, sex) (Kringelbach, 2005; Vuust & Kringelbach, 2010), (2) in response to tangible items that have a secondary reward, (e.g. money or other possessions), or (3) as a result of direct stimulation of the dopaminergic pathways in the mesolimbic system of the brain, such as those stimuli with addictive qualities (e.g. synthetic or pharmacological chemicals and gambling) (Salimpoor, Benovoy, Larcher, Dagher, & Zatorre, 2011; Salimpoor et al., 2009). Despite this, research has consistently shown music listening to be a pleasurable activity (Dubé & Le Bel, 2003) and to evoke a range of emotions within listeners (Zentner et al., 2008).

When asked to freely provide antecedents associated with pleasure music was found to be the 5th most mentioned concept (behind sports, sex, food, and friends) and in a follow up study the majority of participants classified music as an emotional pleasure compared to classifications of general, physical, social, and intellectual (Dubé & Le Bel, 2003). Furthermore, Zentner, Grandjean, and Scherer (2008) complied a list of music-induced emotions based on self-reports and studied the frequency with which these emotions were experienced.

From this they constructed a model that best accounts for music-induced emotions compared to other models (e.g. basic emotion model, dimensional emotion model). This demonstrates that music not only elicits emotion, but that it contains domain-specific emotions. That is, some emotions are more likely to be induced by music (e.g. happiness, nostalgic) than others (e.g. anger, sorrow). This suggests that music-induced emotions, as a domain, differ considerably from everyday emotions and therefore necessitate a domain-specific classification.

1.5. Nicotine affects emotion

Tomkin's (1966) model of smoking suggests that people smoke for a number of reasons, including regulating internal emotions, producing positive emotions, and minimizing negative emotions. In line with this, smokers report one motive for smoking is to increase pleasure and relaxation (Leventhal & Cleary, 1980). Furthermore, they frequently report feelings of tranquility and relaxation from nicotine use (Agué, 1973; Hatch, Bierner, & Fisher, 1983; Ikard, Green, & Horn, 1969; Silverstein, 1982) as well as tension reduction (Russell, Peto, & Patel, 1974). For example, in one study smokers used a checklist to indicate how they felt before and after smoking throughout the day under different puffing conditions. Pleasure was found to increase as nicotine increased (Agué, 1973). This study is supported by several other findings showing that intermediate doses of nicotine (0.74 to 1.5 mg) increase pleasure and enjoyment (Gilbert, Dibb, Plath, & Hiyane, 2000; Hasenfratz, Baldinger, & Bättig, 1993; Pomerleau & Pomerleau, 1992; West & Hack, 1991). In this light, it is not surprising that abstaining smokers rated feeling more pleasant and relaxed an hour after smoking high-nicotine cigarettes compared to low-nicotine and no-nicotine cigarettes (Agué, 1973). Although others have failed to demonstrate an effect of nicotine on pleasure (Gilbert, Meliska, Williams, & Jensen, 1992; Meliska & Gilbert, 1991) these findings suggest that under some conditions nicotine and pleasure are positively correlated.

Smokers report another motive for smoking is to reduce negative affect, such as stress, anxiety, and anger (Beckham et al., 2008; Gilbert, Robinson, Chamberlin, & Spielberger, 1989; Jamner, Shapiro, & Jarvik, 1999; Pomerleau,

1986). These emotions may begin to surface as nicotine withdrawal sets in, an experience characterized by irritability, anxiety, and depression (West & Hajek, 2004). Interestingly, rats have shown a dramatic decrease in brain reward function during nicotine withdrawal (Epping-Jordan, Watkins, Koob, & Markou, 1998), suggesting further that the reason nicotine can reduce negative affect is because it alleviates withdrawal symptoms. However, others have reported nicotine to reduce reports of anger and aggression (Cherek, Bennett, & Grabowski, 1991; Jamner et al., 1999), which may be mediated by nicotine's action as an agonist of cholinergic, dopaminergic, GABAergic, and serotonergic receptors (Benowitz, 1996; Damaj, Glennon, & Martin, 1994) via activation of nAChRs. Therefore, nicotine may help reduce negative affect in a way that is unrelated to the alleviation of withdrawal symptoms, by acting as an anxiolytic (Picciotto, Brunzell, & Caldarone, 2002).

1.6. Music and nicotine both affect physiology

Music is well known for evoking and modulating emotion (Juslin & Västfjäll, 2008) and emotional responses to music are often coupled with physiological changes (Rickard, 2004). Physiological changes have even occurred in response to musical features when they lack emotional connotation, including rhythm (Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006; Gomez & Danuser, 2007), tempo, accentuation (Gomez & Danuser, 2007; Khalfa, Roy, Rainville, Dalla Bella, & Peretz, 2008) and simple isochronous auditory pulses (Koelsch & Jäncke, 2015). This implies that music can consistently affect a listener's physiology. Previous studies examining such responses have found many trends, and although many inconsistencies exist, heart rate, skin conductance, respiration rate, and body temperature are the physiological changes found to be most affected by music (for a review see Hodges, 2010).

The impact of nicotine on the central nervous system is well known and has cascading effects on the physiology of tobacco users, most which result in peripheral nervous system changes (Pomerleau & Rosecrans, 1989). Both smokers and nonsmokers display similar physiological changes to nicotine (Foulds et al., 1997; Heishman, Snyder, & Henningfield, 1993). However, it is important to note that nonsmokers are more likely to experience adverse

effects, as they hold no tolerance for the drug (Foulds et al., 1997). These effects, associated with nicotine toxicity, include feelings of dysphoria, as well as the physiological responses of sweating, coldness of hands, palpitations, headache, arm pain, nausea, dizziness, indigestion, and upset stomach (Foulds et al., 1997; Guy, 1976a). There is some evidence that smokers experience adverse effects to nicotine similar to nonsmokers, but that they are interpreted as pleasurable. For example, the airway sensory effects of smoking are considered aversive for nonsmokers, but become pleasurable to smokers through repeated association with smoking (Rose & Levin, 1991). Smokers have also reported 'euphoriant' effect from smoking, which were described as a pleasurable high, buzz, or rush (Pomerleau & Pomerleau, 1992). However, it may be that smokers are unable to distinguish their 'high' from dizziness (Foulds et al., 1997). For example, in a study by Johnston (1942) smokers and nonsmokers were administered 1.3 mg of hypodermic injections of nicotine. While nonsmokers reported an unpleasant light-headedness, smokers reported the same experience, but described it as pleasant. As with music, the strongest physiological responses resulting from nicotine intake are changes in heart rate, skin conductance, respiration rate, and body temperature. Therefore, these four physiological responses are reviewed below.

1.6.1. Heart rate

Heart rate (HR), calculated by the number of beats per minute (Andreassi, 2007), is regulated by a number of circuits that are influenced by cortical forebrain structures involved in the processing of emotion, including the hypothalamus, amygdala, insular cortex, and orbitofrontal cortex (Armour & Ardell, 2004). Several studies have shown these structures to be activated during music-induced emotions (Blood & Zatorre, 2001; Koelsch, 2014; Koelsch & Skouras, 2014). Although other measurements of heart rate exist (e.g. heart rate variability, interbeat interval) a measurement based on beats per minute allows one to assess a change in physiology over a short time course. For example, heart rate variability requires a minimum stimulus duration of five min, while beats per minute requires only two min. As many experiments employ musical excerpts lasting less than 5 min, typically between 90 s and 4 min, (see Blood & Zatorre, 2001; Koelsch, 2014; Rickard, 2004) measuring beats per

minute is a valid method for assessing short-term changes in heart rate during music listening.

In general, research shows that high arousal or stimulating music, such as that which contains a fast tempo or staccato accentuation, increases HR (Edworthy & Waring, 2006; Gomez & Danuser, 2007). This holds true when comparing stimulating music to silence (Bernardi, Porta, & Sleight, 2006) as well as tranquilizing or sedative music, both of which decrease HR (Etzel et al., 2006; Guhn, Hamm, & Zentner, 2007; Koelsch & Jäncke, 2015). HR has also been observed to increase during pleasurable emotional responses to music, as was observed during music-induced chills (Blood & Zatorre, 2001). This suggests that positively valenced music (e.g. pleasant music) also increases HR and indeed this is what several studies have found (Orini et al., 2010; Salimpoor et al., 2009; Sammler, Grigutsch, Fritz, & Koelsch, 2007).

However, there are several inconsistencies found throughout the literature. For example, studies have found no change in HR during fast tempo music (Schwartz, Fernhall, & Plowman, 1990) or experimental rhythms (Shatin, 1957), while a decrease in HR has been observed during exciting music (Iwanaga & Moroki, 1999). Furthermore, some studies show no change in HR in response to emotionally powerful music (Rickard, 2004) or relaxing music (Davis & Thaut, 1989), while another study reported an increase in HR for both pleasant and unpleasant music (Krabs, Enk, Teich, & Koelsch, 2015). Other studies have shown either no difference in HR when comparing sad, fearful, happy, and displeasing music (Giovannelli et al., 2013) or have shown sad, fearful, and happy music to all decrease HR (Krumhansl, 1997). It may then be that HR is more affected by individual differences than by music (Ellis & Brighouse, 1952).

In contrast to the inconsistencies in studies with music, there is overwhelming and almost universal evidence that nicotine increases HR. This has been demonstrated across studies using different methodologies, including injected nicotine (Hopkins, Wood, & Sinclair, 1984; Lucchesi, Schuster, & Emley, 1967), nicotine gum (Parrott & Winder, 1989), and cigarette smoking (Gilbert & Hagen, 1980; Herxheimer, Griffiths, Hamilton, & Wakefield, 1967). In fact, nicotine's ability to increase HR has sometimes resulted in tachycardia

(Nyberg, Panfilov, Sivertsson, & Wilhelmsen, 1982; Schneider, Jarvik, & Forsythe, 1984). In one study 16 abstaining male smokers were administered 2 and 4 mg of nicotine through gum as well as through cigarettes. Dose-dependent effects of both methods of delivery (e.g. gum and cigarettes) were found on HR, although cigarette smoking was found to increase HR more than gum (Parrott & Winder, 1989). This is unsurprising given the rapid uptake of nicotine caused by inhalation compared to oral absorption (Benowitz, 1996; Parrott & Winder, 1989). Despite the paucity of literature with smokers, similar dose-dependent effects of nicotine on HR have been found for the nonsmoking population. For example, a study of four healthy nonsmokers found that compared to placebo 0.6 mg of nicotine via subcutaneous injections resulted in dose-dependent increases in HR (Foulds et al., 1997). In another study of six nonsmokers, which included 3 life-long never-smokers, participants were subject to injections of subcutaneous nicotine at either 13.25 μg/kg (nonsmokers) or 12.23 μg/kg (never-smokers). Throughout the study HR was measured and was shown to have a dose-dependent increase in line with nicotine administration. Interestingly, an acute tolerance to the drug was also observed. That is, over time the never-smokers' HR began to adapt to the nicotine, which led to a reduction in HR even as nicotine levels continued to increase (Russell, Jarvis, Jones, & Feyerabend, 1990). This suggests that like smokers, nonsmokers can experience acute physiological tolerance to nicotine (Perkins, Epstein, Stiller, Marks, & Jacob, 1989; Russell et al., 1990).

1.6.2. Skin conductance

Skin conductance, a method used to measure electrical resistance of the skin (Andreassi, 2007), has also been shown to change in response to music listening. Skin conductance is a sensitive measure of activation of the autonomic nervous system (ANS) (Quinlan et al., 2000), which occurs without voluntary control when sweat ducts fill with fluid in direct response to activation of the sweat gland via the sympathetic nervous system (Baumgartner, Lutz, Schmidt, & Jäncke, 2006; Dawson, Schell, Filion, & Berntson, 1990). There are two measures of skin conductance: 1) skin conductance level (SCL), which is the recording of background sweat-gland activity that provides information about the general activation of the ANS and 2) skin conductance response

(SCR), which is the recording of sweat-gland activity that occurs as a result of a specific event (e.g. a loud crash) (Agué, 1974; Lader & Wing, 1966; Lykken & Venables, 1971).

Arousal is strongly linked to increases in skin conductance (Hodges, 2010). Therefore, music of a highly arousing or stimulating nature increases SCR compared to music that is calm or neutral (Zimny & Weidenfellar, 1963). Indeed, several studies have found this pattern of response. For example, happy and fearful music produce higher SCRs compared to sad and peaceful music (Khalfa et al., 2002; Lundqvist, Carlsson, Hilmersson, & Juslin, 2008), presumably because of the higher levels of arousal produced by happy and fearful emotions. Similar results have been reported for joyful and horrific music (VanderArk & Ely, 1992, 1993) and studies examining music-induced chills have found it to increase SCRs compared to baseline or control conditions (Craig, 2005; Grewe, Nagel, Kopiez, & Altenmüller, 2005).

However, an increase in SCRs as a result of arousing music is not always consistently found. For example, skin conductance has been shown to increase for happy, sad, and fearful music all within a single experiment and without any significant differences between the conditions (Krumhansl, 1997). This demonstrates that sad music can modulate skin conductance despite its low arousal level and furthermore makes it difficult to distinguish whether there are idiosyncratic physiological responses between different musical emotions. Furthermore, many studies have found no reliable change in SCR during music listening (Blood & Zatorre, 2001; Davis, 1934; de Jong, Van Mourik, & Schellekens, 1973; Keller & Seraganian, 1984; Ries, 1969), even when subjective levels of anxiety decreased (Jellison, 1975).

Overall, nicotine's effect on skin conductance response and skin conductance level is also varied and inconsistent. When skin conductance level is examined in nicotine experiments results often show either an increase in this measurement or no change at all. For example, Agué (1974) found higher skin conductance levels immediately after smoking in 24 abstaining male smokers, and Frith and Agué (1969) found the same effect, which lasted ~30 min after the administration of nicotine via aerosol and cigarettes. In another study 30 abstaining smokers were subject to the stressful task of giving a speech. Before

the speech, they were assigned either to a no-smoking group, a low-nicotine cigarette-smoking group, or a high-nicotine cigarette-smoking group. Interestingly, mean skin conductance levels were not found to differ between the groups (Hatch et al., 1983). No significant effect of smoking on skin conductance level has also been reported in a study examining pain tolerance (Waller, Schalling, Levander, & Edman, 1983). Other studies have found no effect of nicotine on skin conductance response including one that compared nicotinized and denicotinized puffs from cigarettes (Naqvi & Bechara, 2006) and one examining pain tolerance in 33 male moderate smokers during placebo and smoking conditions (Waller et al., 1983).

In contrast to studies with music, some studies report nicotine to decrease skin conductance response. Because an increase in arousal is usually associated with an increase in skin conductance, it seems paradoxical that nicotine increases measures of arousal while simultaneously decreasing skin conductance response. However, this is inline with the known paradox whereby smoking has been associated with tranquilization and relaxation despite its arousing capabilities (Gilbert, 1979). For example, Gilbert & Hagen (1980) found that when minimally abstaining smokers viewed emotionally arousing scenes a high-nicotine cigarette resulted in a significantly lower skin conductance response than a low-nicotine cigarette. In another study smoking and nonsmoking subjects were given 1.1 mg of nicotine via a cigarette one day and no nicotine on the other day. Skin conductance responses were then recorded before and after an auditory task and during periods of rest. During tasks skin conductance responses were found to be smaller during smoking compared to during abstinence This may suggest that when arousal is increased by a task, nicotine can dampen skin conductance responses (Boyd & Maltzman, 1984) and potentially result in subjective relaxing effects (Gilbert & Gilbert, 1998).

The review of the literature shows inconsistent results regarding how nicotine affects skin conductance. For example, it may be that in tasks where arousal is increased nicotine is able to decrease skin conductance, as explained by Boyd & Maltzman (1984). However, it may also be that relaxing effects were felt by smokers during smoking compared during abstaining because they

experienced a relief of withdrawal symptoms- as nicotine withdrawals are known to be associated with anxiety and other negative mood states (Hughes et al., 1984). Furthermore, different methodologies were used between studies (e.g. no task, giving a speech, or auditory task), which may have influenced skin conductance responses differently and therefore resulting in discrepancies between studies. This suggests that further research is needed to better understand the effects of nicotine on skin conductance, especially during auditory tasks as there is limited research using auditory stimuli during nicotine consumption. Therefore, the current thesis will examine both smokers and nonsmoker in a single study using auditory/musical stimuli in order to better understand how nicotine affects skin conductance responses.

1.6.3. Respiration rate

Respiration rate is the number of breaths taken per minute and is measured by chest expansion while at rest (Sherwood, 2010). Respiration is strongly linked to emotional responses, a premise verified by numerous studies showing differences between conditions during music listening tasks using electroencephalogram (EEG) (Baumgartner, Esslen, & Jäncke, 2006), PET (Blood & Zatorre, 2001), and self-reports of emotion (Gomez & Danuser, 2004, 2007; Krumhansl, 1997). For example, breathing rates increased less for sad music compared to happy music in a study where participants listened to 3 min excerpts (Krumhansl, 1997). Breathing rates have also been found to entrain with music (Haas, Distenfeld, & Axen, 1986). One study found shorter breath lengths for happy music, intermediate for fearful music, and longer for sad music (Etzel et al., 2006). Not surprisingly, breathing rates are also faster with faster beats and slower with slower beats (Khalfa et al., 2008). Furthermore, increases in respiration rate frequently correspond with increases in HR (Bartlett, 1999), suggesting the two physiological parameters to be somewhat interrelated. Indeed, in several studies that measured both HR and respiration rate, both were found to be higher for exciting music compared to tranquilizing music (Bernardi et al., 2006; Etzel et al., 2006; Iwanaga, Ikeda, & Iwaki, 1996; Iwanaga & Moroki, 1999; Krumhansl, 1997). However, some studies have reported no change in respiration rate (Davis-Rollans & Cunningham, 1987). For example, no difference in respiration rate was found between women

listening to music and women listening to nothing while undergoing a medical procedure (Davis, 1992).

Although there is far less literature concerning nicotine's effect on human respiration, in smaller doses the drug is thought to increase respiration rate and in larger doses to paralyze it (Silvette, Hoff, Larson, & Haag, 1962). When nicotine increases respiration it does so by stimulating the chemoreceptors located near the carotid arteries and aorta. This is the dominant reflex mechanism responsible for ventilation (Heymans, Bouckaert, & Dautrebande, 1931; Najem et al., 2006; Wright, 1935). Furthermore, when nicotine is inhaled (e.g. via cigarette smoking) it stimulates the afferent nerve endings in the bronchial mucosa, which are mediated by the parasympathetic cholinergic pathways (Hansson, Choudry, Karlsson, & Fuller, 1994). In this way, nicotine can stimulate breathing by increasing the activity of muscles implicated in dilating the upper airway (Gothe, Strohl, Levin, & Cherniack, 1985). This in turn increases the supply of air that reaches the lungs (Najem et al., 2006). As with music, HR and respiration rate are linked. For example, Jones (1987) found that people who show an increase in HR within 1 minute of smoking also show an increase in respiratory rate, while those who exhibited little or no change in HR showed a decrease in respiratory rate.

Given the limited amount of research regarding nicotine and its effects on respiration it is clear that further investigation is needed in order to understand whether this drug can modulate respiration rate in smokers and nonsmokers. And given that respiration is strongly linked to emotional responses it seems plausible that respiration rate can be modulated by a combination of music listening and nicotine consumption. Therefore, this thesis will examine how respiration rate is affected by nicotine and music listening both independently and in combination.

1.6.4. Skin temperature

Skin temperature is related to blood flow in skin tissue and is a reflection of vasoconstriction and vasodilatation that occurs just below the skin's surface (Andreassi, 2007; Hodges, 2010; McFarland, 1985). Past research suggests that finger temperature corresponds to emotional valence, and to a lesser extent to arousal. Positively valenced music, such as soothing and soft music, increases

finger temperature (Hsu & Lai, 2004; Lai, 2004), as does sedative and relaxing music (Kibler & Rider, 1983; Peach, 1984). On the other hand, negatively valenced music, such as sad and fearful music, decrease finger temperature (Baumgartner, Esslen, et al., 2006; Krumhansl, 1997; Nater, Abbruzzese, Krebs, & Ehlert, 2006). Studies have also examined how arousal affects skin temperature. In one study McFarland (1985) examined how arousing and calming music would affect skin temperatures that were already increasing or decreasing. The study found that arousing music terminated increases in skin temperature and subsequently caused it to decrease, while the opposite was found for calming music, where it terminated a decrease in skin temperature and subsequently caused it to increase. This is inline with previous studies and suggests that music can predictably increase or decrease skin temperature depending on whether it is of low or high arousal, respectively.

A review of the literature, however, shows that not all research has found this predictable trend for skin temperature as it relates to valence and arousal. For example, one study reported sad music to decrease finger temperature, albeit happy music generated a lower finger temperature than sad music (Lundqvist et al., 2008) and another study examining how arousal affects skin temperature found that for sensation seekers heavy metal music resulted in a higher skin temperature than Renaissance/classical music (Nater et al., 2006). This suggests that arousal and valence do not always influence skin temperature in a predictable way and demonstrates how individual differences can influence physiological responses to music. Other studies have found no change in skin or body temperature during music-induced emotion (Blood & Zatorre, 2001; Craig, 2005; Rickard, 2004; Rider, Mickey, Weldin, & Hawkinson, 1991; Savan, 1999; Zimmerman, Pierson, & Marker, 1988), suggesting that many inconsistencies in this physiological response still exist.

Again, in contrast to music, the literature strongly and consistently suggests that nicotine decreases peripheral body temperature as demonstrated through studies showing a reduction in skin temperature (Agué, 1974; Frankenhauser et al., 1968; Stephens, 1977). This is because nicotine produces vasoconstriction, which results in a reduction of skin circulation and therefore causes a decrease in finger temperatures (Black et al., 2001; Roth, McDonald,

& Sheard, 1944). Cutaneous vasoconstriction following smoking or injected nicotine has been observed using skin temperature measures (Maddook & Coller, 1932; Roth et al., 1944; Wright, 1933), and through observation of capillary beds (Wright & Moffat, 1934), and plethysmograph (Bruce, Miller, & Hooker, 1909). These decreases have ranged from 0˚C to 4˚C for the finger and 0˚C to 2.8˚C for the toe in both smokers and nonsmokers (König & Classen, 1981; Larson, Haag, & Silvette, 1961). For example, when nicotine was infused into 14 male smokers at rates of 1.0 to 2.0 µg/kg/min the drug decreased fingertip skin temperature similar to that of cigarette smoking (Benowitz, Jacob, Jones, & Rosenberg, 1982). Another study using injected nicotine also found decreases in skin temperature (Rottenstein, Peirce, Russ, Felder, & Montgomery, 1960). In an experiment with cigarettes Agué (1974) asked 24 abstaining smokers to puff cigarettes containing 0, 0.75, 1.02, and 2.11 mg of nicotine at different times of the day and at fast and slow rates of inhalation. When participants smoked the nicotine cigarettes their mean skin temperature decreased by between 2.8 and 3.5˚C below base values. Similar results were found in a study that administered nicotinic cigarettes to abstaining (Moss, Hammer, & Sanders, 1984) and non-abstaining smokers (Frankenhauser et al., 1968). However, Agué (1974) suspected other factors besides nicotine contributed to the decrease as placebo cigarettes (lettuce-leaf cigarettes) also resulted in a decrease in skin temperature by 2˚C. Later studies wished to administer nicotine to participants without exposing them to the hazardous chemicals found in cigarettes, such as tar and carbon dioxide ($CO_{2)}$. This was accomplished by using 2 mg nicotine gum and interestingly resulted in elevated skin temperature (Usuki, Kanekura, Aradono, & Kanzaki, 1998) and observations of participants' hands becoming warm and sweaty (Kanekura & Kanzaki, 1995). However, one study by Heishman, Snyder, and Henningfield (1993) used nicotine gum (at 0, 2, and 4 mg) and found skin temperature to decrease in nonsmokers. Despite the inconsistencies, it is generally accepted that nicotine causes vasoconstriction (Rottenstein et al., 1960) and results in a decrease in skin temperatures for both smokers and nonsmokers.

1.7. Summary

In general, these physiological findings suggest that music and nicotine are capable of modulating bodily responses. Although there are numerous inconsistencies, there is evidence that music and nicotine have both similar and dissimilar effects on emotion and physiology. For example, both can increase pleasure (Blood & Zatorre, 2001; Leventhal & Cleary, 1980). Furthermore, both stimulating music and nicotine can increase HR (Gomez & Danuser, 2007; Parrott & Winder, 1989) and skin conductance (Agué, 1974; Khalfa et al., 2002), although somewhat inconsistently. They can both also increase respiration rate (Gothe et al., 1985; Krumhansl, 1997). However, while positively valenced music increases skin temperature (Kibler & Rider, 1983) and negatively valenced music decreases skin temperature (Baumgartner, Esslen, et al., 2006), there is strong evidence that nicotine actually decreases it (Frankenhauser et al., 1968).

The similarities between the effects of music and nicotine support the idea that these two interventions could have additive effects on an individual and therefore result in their frequent co-occurrence. However, the numerous inconsistencies in the studies using music are a problem that cannot be ignored and make comparisons between the effects of music and nicotine somewhat difficult. It could be that the inconsistencies are a result of the lack of standard methodologies between studies. For instance, there are different definitions of stimulating and sedative music and different genres are employed between studies. This may lead to differences in familiarity and liking for the listener and in turn cause more or less emotional and physiological responses. To combat this limitation it is necessary to incorporate music that is preferred by the listener. This will ensure adequately strong emotional responses, which may then lead to more robust physiological changes. Furthermore, a manipulation check is needed to confirm an emotional response from the listener. This can be accomplished by using self-reports of emotion during experimentation.

There is also great variation in the physiological responses to nicotine. This may be to due to the variation in smokers, abstaining smokers, and nonsmokers used throughout the literature. For example, whether a smoker is a heavy smoker or a light smoker will undoubtedly affect how they respond to

nicotine. That is, since heavy smokers hold a higher tolerance for nicotine they may be less physiologically responsive during experimentation or they may have faster rates of nicotine elimination (Pomerleau, 1995). There may also be individual differences in sensitivity to nicotine, causing more adverse effects in never smokers compared to nonsmokers (Pomerleau, 1995; Silverstein, Kelly, Swan, & Kozlowski, 1982). To account for these limitations it is necessary to use smoking and nonsmoking cohort with similar smoking habits. Therefore, this thesis will focus on a smoking cohort that consumes ~7 cigarettes per day for at least two years and who scored a minimum of 5 on the Fagerström Test for Nicotine Dependence (Heatherton, Kozlowski, Frecker, & Fagerström, 1991). This will ensure that 1) smokers are in a state of withdrawal when asked to abstain from nicotine, as smoking 5 or less cigarettes per day is indicative of a non-addicted or 'chipper' smoker (Frosch, Shoptaw, Nahom, & Jarvik, 2000; Shiffman & Paty, 2006) and 2) in order to control the level of addiction in smoking participants so that all participants have a similar level of addiction to nicotine. Furthermore, those who have smoked more than twenty cigarettes in a lifetime have experienced some level of craving and have shown signs of nicotine tolerance compared to those who have smoked less than twenty cigarettes. This too may affect the rate of nicotine elimination, as well as the physiological and subjective responses to nicotine (Pomerleau, Pomerleau, Snedecor, & Mehringer, 2004). In order to ensure accurate control measures with a nonsmoking population this thesis will focus on a nonsmoking cohort who has smoked fewer than 7 cigarettes in a lifetime and who scored a maximum of 2 on the Fagerström Test for Nicotine Dependence (Heatherton et al., 1991). Lastly, one of the hallmarks of experimental design is the use of a placebo control condition. This allows for a study to be executed under blind conditions. This means that control measures during drug administration reduces the likelihood of participants knowing the type of treatment they receive and therefore reduces demand characteristics from said participants. However, several studies use tobacco cigarettes for nicotine administration, and although this increases the ecological validity of the experiment, it unfortunately limits the use of a placebo condition. Although, some studies have administered herbal or lettuce leaf cigarettes as a placebo condition (West

& Hack, 1991) or denicotinized cigarettes, which have less than 0.1 mg of nicotine (Hasenfratz et al., 1993; Naqvi & Bechara, 2006), it is more common for studies to instead employ a repeated-measures design where smoking participants smoke cigarettes in one condition/session and abstain in another (Agué, 1974; Boyd & Maltzman, 1984; Moss et al., 1984; Waller et al., 1983). Therefore, the experiments in this thesis will include a placebo condition for both smokers and nonsmokers. More specifically, pharmaceuticals (both nicotine and caffeine) will be administered in gum and tablet form, respectively, so that single blind placebo controlled experiments can be conducted. This will ensure accurate control conditions and reduce the confound of smoking status on physiological, cognitive, and subjective responses.

Overall, previous research has demonstrated listeners' emotions and physiological arousal can be significantly affected by music and nicotine independently, but the effects on individuals during co-consumption of both stimuli have yet to be fully investigated. That is, to the best of my knowledge, this is the first to study to examine how nicotine and music consumption affect smokers and nonsmokers. The rest of this thesis therefore aims to explain why nicotine consumption and music listening often co-occur. More specifically, study 1 (chapter 2) will focus on how music and nicotine affect physiological arousal and emotion both independently and in combination. It will further test the explanation that nicotine and music are co-consumed because nicotine increases the reward value (e.g. pleasure) of other stimuli and increases peripheral feedback (e.g. physiological arousal) in smokers and nonsmokers, and in turn increases the emotions experienced during music listening. Study 2 (chapter 3) will follow on from study 1 with the aim of isolating the effects of physiological arousal on music-induced emotion in smokers and nonsmokers. In this way, the effects of peripheral feedback on music-induced emotion can be tested without the influence of pleasure/reward. This will be accomplished through the administration of caffeine, which is known to increase physiological arousal but not to increase the reward value of other stimuli (Herz, 1999). While study 1 and study 2 focus on the effects of nicotine/caffeine and music listening on physiological arousal and emotion, study 3 (chapter 4) examines whether there are cognitive explanations for the co-consumption of nicotine

and music. Therefore, through an ERP study with nonsmokers study 3 will investigate the cognitive effects of nicotine on auditory perception. This will test whether nicotine is able to enhance auditory perception and in turn increase music-induced emotion. Lastly, chapter 5 will compare and contrast the effects of nicotine and caffeine, alone and in combination with music, on physiological arousal and emotion. It will further explain the cognitive effects of nicotine on auditory perception, discuss explanations as to why nicotine and music are co-consumed, and provide suggestions for future research.

2. Chapter two: Effects of nicotine on music-induced emotion

2.1. Overview and rationale of study 1

As discussed in chapter one, nicotine and music can both independently affect pleasure and physiological arousal. In general, both can increase emotion and pleasure, as indicated by the motivations individuals self-report for engaging in smoking and music listening (Dubé & Le Bel, 2003; Leventhal & Cleary, 1980). Both activities can also heighten arousal, as indicated by an increase in HR, skin conductance, and respiration rate, as well as by a decrease in skin temperature (Agué, 1973, 1974; Hodges, 2009, 2010; Jones, 1987; Parrott & Winder, 1989). However, less is known about how nicotine and music in combination affect pleasure and arousal. I suggest that together nicotine and music can have an additive effect on an individual's pleasure, arousal, or both, and that this additive effect occurs because nicotine increases pleasure and physiological arousal, which in turn results in an enhancement of music-induced emotion. I suggest that nicotine can increase music-induced emotion through two mechanisms. The first is through the drug's ability to increase the pleasure derived from listening to music by releasing extracellular dopamine in the brain (Balfour, 2004). The second is through nicotine's ability to increase physiological arousal, which through sensory feedback leads to a heightened experience of felt emotion during music listening (Dibben, 2004). Therefore, the central focus of study one is to determine whether there is an additive effect on pleasure, arousal, or both as a result of the co-consumption of nicotine and music listening, and if so to identify the mechanisms through which this additive effect occurs. As an initial study examining the combined effects of music listening and nicotine I tested for an additive effect, as this will examine whether both stimuli are influencing arousal, pleasure, or both. Upon confirmation of such an effect further research may be conducted to determine the likelihood of super or sub-additive effects.

2.2. The relationship between arousal and pleasure

Research on emotion and mood has reached a general consensus regarding the most basic structure of the affective experience: a circumplex model of emotion (Barrett & Russell, 1999; Feldman, 1995; Russell, 1980, 2003). This model is made up of two fundamental dimensions (e.g. scales): valence (a unpleasant-pleasant continuum) and arousal (a deactivated – activated continuum). The circumplex model of emotion can be seen in Figure 2.1. Although some literature suggests otherwise, these scales are generally considered independent from one another and are sufficient at explain a high percentage of the variance of basic emotions (Barrett & Russell, 1999; Russell, 1980). In this way, the quality and intensity of all affective states can be defined in terms of valence (e.g. pleasantness) and aroused (e.g. energy) one feels, a concept known as core affect (Russell, 2003). For example, 'excited' would be high in valence/pleasure and high in arousal/energy, while 'depressed' would be low on both dimensions.

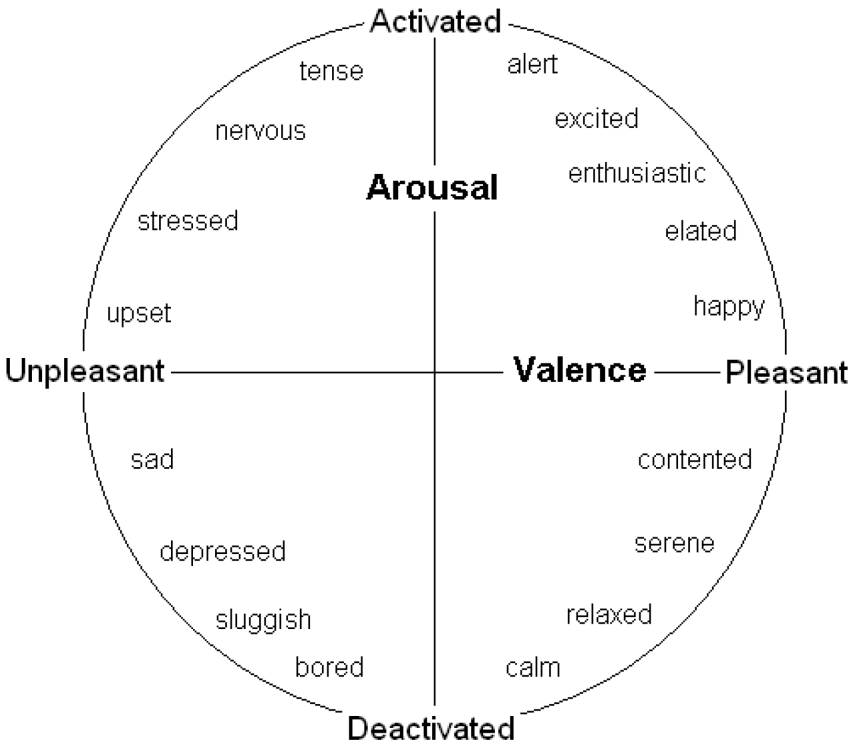

Figure 2.1. Circumplex Model of Emotion (Russell, 1980).

Although much of the evidence in support of the circumplex model has been based on responses to stimuli other than music, Bigand and colleagues

(2005) found these dimensions to explain the music categories created when participants were asked to group music excerpts based on their similar emotional meaning. The circumplex model of emotion has also gained support in music and emotion research (Gomez & Danuser, 2004; Schubert, 1999; Witvliet & Vrana, 2007), showing them to describe accurately the emotional experience of music. However, two-dimensional models have been noted for their limitations, for example, their inability to clearly distinguish between two emotions that are close together in the pleasure-arousal space, such as anger and fear (Tellegen, Watson, & Clark, 1999), and their difficultly in positioning complex emotions, such as nostalgia, within the model. This is particularly important, as nostalgia is a common emotion experienced in response to music (Zentner et al., 2008). Therefore, the two-dimensional model is not able to account for all the variance of music-induced emotions (Collier, 2007; Ilie & Thompson, 2006). Furthermore, some research has suggested that the relationship between arousal and pleasure is not orthogonal (Kuppens, 2008; Kuppens, Tuerlinckx, Russell, & Barrett, 2013). That is, within a single individual, individual differences can show a correlation between pleasure and arousal, showing that either pleasant/unpleasant feelings often co-occur with high arousal (reflecting joy/stress) or with low arousal (reflecting relaxation/sadness) (Kuppens, 2008). This means that the relationship between pleasure and arousal may vary depending on person and circumstances (Kuppens et al., 2013). However, this has yet to be demonstrated in a musical context as Bigand and colleagues (2005) showed that emotional responses to music were not subject to strong individual differences, and were reproducible within and between listeners. In this case, arousal and pleasure can still be considered independent dimensions used to measure emotion. However, it does suggest further research is needed and warrants the consideration of other emotion models when measuring music-induced emotion.

The discrete emotion model has also been used to measure music-induced emotion, where participants listen to music then rate predetermined affect terms to describe how they feel (Zentner et al., 2008). These terms reflect basic emotions, such as anger, fearfulness, surprised, happiness, and sadness (e.g. Baumgartner, Esslen, et al., 2006; Etzel et al., 2006; Kallinen,

2005; Krumhansl, 1997). Previous music research (e.g. Eerola & Vuoskoski, 2011) has shown that when unambiguous emotions are measured (e.g. happy, sad) the discrete emotion model produces results that correspond well to those measured on the circumplex model. In this way, it may be equally legitimate to measure arousal and pleasure on separate scales, and additionally measure basic emotions, such as happiness and sadness. The current study therefore measures self-reported responses of arousal, pleasure, happiness, and sadness in this way.

2.3. Nicotine increases the reward value of other stimuli

Research examining the emotional effects of nicotine have shown it to enhance the reward value of other stimuli, and in turn influence behavior (Donny et al., 2003). That is, nicotine has the ability to increase the pleasure derived from other activities or stimuli that occur in its presence (Attwood, Penton-Voak, & Munafò, 2009; Dawkins, Acaster, & Powell, 2007). It is suggested this occurs because the drug releases dopamine into the medial shell of the nucleus accumbens (NAcc) (Balfour, 2004; Donny et al., 2003). In turn, any activity or stimulus experienced during this overflow of dopamine can result in an increased hedonic impact (Balfour, 2004).

Evidence for this effect stems from animal studies. For example, following the administration of nicotine rats have shown a decreased threshold for brain reward stimulation (Kenny & Markou, 2006) as demonstrated by their increased response to food, alcohol, and cocaine (Bechtholt & Mark, 2002; Clark, Lindgren, Brooks, Watson, & Little, 2001; Popke, Mayorga, Fogle, & Paule, 2000). In studies with humans, nicotine has been found to increase ratings of facial attractiveness in nondependent smokers (Attwood et al., 2009) and to increase self-reports of pleasure in response to movie clips in abstaining smokers (Dawkins et al., 2007). This demonstrates that in humans nicotine is able to enhance the hedonic impact of other stimuli that occur in its presence and suggests that the drug could enhance music-induced pleasure.

2.4. Nicotine increases emotion via peripheral feedback

As previously mentioned, nicotine has the ability to increase physiological arousal (Nesbitt, 1973). Interestingly, heightened physiological arousal can

increase the physical sensations that accompany emotion and therefore lead to more intense emotional experiences (Konecni, 1975; Zillmann, 1978; Zillmann, Katcher, & Milavsky, 1972). Scherer and Zenter (2001) suggest this process to occur through peripheral feedback, which is the sensory feedback experienced as a result of physiological changes (Damasio, 1994). In general, each emotion tends to have its own distinguishable set of bodily changes (Philippot et al., 2002). For example, anger is associated with an increase in HR, breathing rate, and blood pressure (Kreibig, 2010). Therefore, activation of a particular set of body changes (e.g. an increase in HR, breathing rate, and blood pressure) may give rise to the emotion with which it is coupled (e.g. anger) (Damasio, 1994). In this way, peripheral feedback can influence the intensity of felt emotion.

Individuals have used peripheral feedback to inform them of their emotions in a number of experiments. In a seminal study Schachter and Singer (1962) injected either epinephrine (adrenaline) or a placebo into 184 university students. The epinephrine caused a rise in HR, blood pressure, blood flow, and respiration rate. Only one third of the participants were informed about the side effects of epinephrine, while the others were either deceived by being told the injection was used to test eyesight or by being left ignorant. The students were then placed into either a euphoric or angry social situation. Results showed that those students who were deceived or left ignorant about the injection and had been exposed to the euphoric social condition reported the most intense experiences of euphoria. This suggests that when no explanation for physiological arousal is apparent, individuals will label it based on their social situation, but most importantly this study demonstrates that under certain conditions physiological arousal can influence the intensity of an emotional experience.

In a more recent experiment involving music listening, Dibben (2004) demonstrated the ability of peripheral feedback to influence music-induced emotion. This was accomplished by inducing either physiological arousal via a short 5 min walk up hill or by inducing relaxation via a 5 min breathing exercise. Before and after arousal/relaxation was induced participants took their pulse rate. This was to check the effectiveness of exercise and relaxation on physiological arousal. The participants who had exercised showed an increase

in pulse rate after their walk relative to before. The exercise group also showed an increase in pulse rate after exercising compared to those in the relaxation group. Participants then listened to four music excerpts, which varied in valance (positive, negative) and arousal (high, low). They then rated the degree to which they perceived and felt 10 emotions in response to each piece of music. These 10 emotions included nostalgia, love, agitated-excitement, peacefulness, spirituality, triumph, happiness, sadness, anger, and anxiety.

As expected, the exercise group, those with higher physiological arousal, gave higher intensity ratings for their felt emotion when listening to music. More specifically, when compared to the relaxation group, the exercise group reported greater intensity of felt emotions that were congruent with the valance of the music. For example, when listening to a piece of music that was positively valenced the exercise group reported more intense felt emotion for happiness than did the relaxation group. This demonstrates that physiological arousal can influence a music listener's emotional experience in the context of music. It further suggests that physiological arousal intensifies the dominant valence of a musical experience. However, there was no difference in the intensity of felt emotion when the emotion was congruent with the arousal level/energy of the music. For example, when listening to a piece of music that was highly arousing (e.g. energetic) there was no difference in felt anger between the exercise and relaxation groups. These findings suggest that physiological arousal, and therefore peripheral feedback, help inform music listeners about the valence of a piece of music, but not about the emotional energy of it.

A follow up study using a similar design further tested the effects of increased physiological arousal on the emotions perceived and felt by music. Three groups, an exercise group, a delayed exercise group, and a control group were used. The exercise group rode an exercise bike for 2 min, while the delayed exercise group did the same, but then rested for 2 ½ min. The control group engaged in a puzzle task for 2 min. After this participants listened to four music excerpts, which again varied in valance (positive, negative) and arousal (high, low). After each excerpt they then either completed a question regarding emotions felt or expressed by the music by rating nine emotions: happiness,

exhilaration, tenderness, serenity, yearning, sadness, fear, anger, and frustration. Additionally, all participants' physiological measures of heart rate and skin temperature were taken before and after the exercise/puzzle tasks as well as during the music excerpts. Lastly, all participants also measured their mood and arousal levels at the beginning and end of the experiment.

Dibben (2004) found significant associations between the emotion ratings and both the self-reported and physiological measures of arousal. That is, those groups with increased arousal from exercise, as verified by an increase in heart rate and skin temperature, gave increased ratings of positive emotions expressed and felt by the music compared to the control group, particularly when the music excerpt was of a positive valence. More specifically, increased physiology intensified the valence of exercising participants' emotions during music listening. However, increased arousal did not affect the emotional energy (arousal dimension) of the excerpts. Furthermore, there was no difference found in mood state as a result of exercising, indicating that these effects were not due to a general increase in participants' mood states. These findings clearly demonstrate that heightened physiological arousal can influence the intensity of felt emotion and provide specific evidence that this kind of phenomenon can occur within a musical context.

Dibben (2004) provides clear evidence that increases in physiological arousal can enhance emotion in a musical context. This leads to the question of whether other forms of induced physiological arousal can increase emotional responses to music. Also, the physiological changes increased by exercise (e.g. heart rate, respiration rate) are those that have been previously associated with negative emotions, such as fear, anger, and sadness (Plutchik, 1994). Therefore, inducing physiological arousal through a stimulus strongly associated with pleasure (e.g. nicotine) may be more likely to enhance positive emotions. Furthermore, there are several indices of heightened physiology. That is, while increases in physiological arousal can be measured through heart rate and skin temperature, they can additionally be measured through respiration rate and skin conductance. Monitoring additional indices of physiology can provide more information about which type of physiological feedback influences musical emotions and which valance (e.g. positive, negative, both) is most affected.

The current study therefore aims to increase physiological arousal using nicotine instead of exercise. Nicotine has been associated with changes in all four of these measures (heart rate, skin conductance, respiration rate, and skin temperature). This drug will be administered to abstaining smokers in an attempt to increase arousal, pleasure, and emotion. Furthermore, nicotine will be administered to nonsmokers as a control to test whether nicotine absolutely increases physiology and enhances musical emotion or whether one must be in a state of withdrawal to feel its effects on emotion. There is also a lot of individual variability in terms of physical fitness, and this was not controlled for in Dibben (2004). Therefore having participants exercise for only 2 min may not consistently increase arousal across all subjects. Although individual variability also exists in terms of nicotine tolerance, it is expected that smokers with a similar level of cigarette consumption (7+ per day) will hold a similar level of tolerance. It is also expected that nonsmokers will have a similar lack of tolerance for nicotine, as all nonsmokers will have smoked less than 7 cigarettes in a lifetime. Lastly, nicotine dependence will be measured using the Fagerström Test for Nicotine Dependence (Heatherton et al., 1991) to ensure consistency across participants in each group.

A vast majority of music-psychology research uses musical stimuli from the Western classical genre (as opposed to popular music) (Västfjäll, 2002), and Dibben (2004) is no exception. Although classical music has been shown induce emotion in listeners in a number of seminal music and emotion studies (Krumhansl, 1997; Rickard, 2004; Sloboda, 1991), in order to increase the ecological validity of the current study popular music will be used. This is, nicotine is more likely to be consumed in a musical setting that contains popular music (e.g. pubs, clubs, and festivals) and this environmental detail should therefore be preserved in an experimental setting. Using popular music also provides a novel approach to studying music-induced emotion and the results of the current study could further legitimize its use, providing a platform for future research.

2.5. Summary and Overview

Nicotine and music are both independently known to increase pleasure and arousal (Benowitz et al., 1988; Dubé & Le Bel, 2003; Hodges, 2009; Leventhal & Cleary, 1980), however less is known about these responses when nicotine and music occur together. It is possible that in combination nicotine and music have an additive effect on pleasure and arousal. This additive effect may occur because nicotine is able to increase the hedonic impact of other stimuli (Attwood et al., 2009) as well as increase the intensity of felt emotion through peripheral feedback (Dibben, 2004; Nesbitt, 1973). Therefore, the aim of the current study was to determine if an additive effect on pleasure, arousal, or both occurred in response to the co-consumption of nicotine and music listening, and if so, to identify the mechanisms through which this additive effect transpired. These aims were accomplished by inducing a heightened physiological state in participants via nicotine administration then asking them to listen to four types of musical excerpts that varied in valence (positive, negative) and arousal (high, low). During the experiment self-reports of emotion and arousal, as well as physiological measurements indicative of arousal, were recorded. It was hypothesized that upon the intake of nicotine and subsequent action of music listening, two results would occur: (1) an individual would experience an increase in the intensity of felt emotion and (2) would experience an increase in arousal and/or pleasure in the context of the increase in emotional intensity.

2.6. Method

2.7. Pilot Study 1

First, 2 preliminary pilot studies were conducted in order to determine the musical material to be used in the main experiment. The two pilot studies identified the best excerpts for the main experiment by (1) verifying that each excerpt induced its intended emotion and (2) identifying excerpts that elicited the strongest emotion of their category (e.g. happy, sad, and neutral).

For the first survey (pilot study 1), 6 happy, 6 sad, and 6 neutral excerpts were presented. See Appendix O for excerpt list. Happy excerpts were

defined by a fast tempo and major key (tonality) and sad excerpts were defined by a slow tempo and minor key (Gagnon & Peretz, 2003). The songs were originally selected from the iTunes library with a music category associated with pop (e.g. rock, alternative, new releases). Although it has been argued that neutral music does not exist (Cooke, 1959; Krumhansl, 1997; Peretz, Gagnon, & Bouchard, 1998), neutral excerpts were defined by a moderate tempo and an ambiguous mode (e.g. no establishment of key, switching between major and minor key). The first survey was administered online to 98 volunteers with a mean age of 19.47 years (SD = 2.72) from The University of Sheffield. Because University of Sheffield students were the main target participants for this thesis, pilot study 1 and 2 were only administered to this population. Volunteers were requested to listen to 18 excerpts that were 1 min in length and to rate each of them on 3, 7-point scales: (1) pleasantness (unpleasant-very pleasant), (2) arousal (sleepy-energetic), and (3) liking (not at all-very much).

2.7.1. Results

First, mean and standard deviations were calculated for each of the 18 excerpts and for each rating (pleasure, arousal, and liking) in order to determine which excerpts from each emotion category (happy, sad, neutral) were rated the highest. Table 2.1 displays the mean and standard deviation for each excerpt and for each rating. It shows that 4 excerpts from the 'Happy' emotion category (Outside Villanova, Angel of Harlem, Hey Soul Sister, and She's Electric) are rated consistently higher in pleasure, arousal, and liking compared to all other excerpts.

Next, average ratings for pleasure, arousal, and liking were calculated for each excerpt type. Table 2.2 displays the mean and standard deviation for these averages. In order to test if pleasure, arousal, and liking ratings significantly differed between the emotion categories a repeated measures ANOVA was then performed- with an independent variable of excerpt with 3 levels (happy, sad, neutral) and a dependent variable of ratings with 3 levels (pleasure, arousal, and liking ratings). Where the assumption of sphericity was violated a Greenhouse-Geisser correction was used and for multiple comparisons a Bonferonni correction was applied.

Ratings were found to be significantly different between the excerpts, $F(6, 91) = 1382.85$, $p < .000$, n2 = .99. Arousal ratings significantly differed between the excerpts, $F(1.78, 170.49) = 1295.79$, $p < .000$, $\eta 2 = .93$. More specifically, happy excerpts were rated significantly higher than sad and neutral excerpts, $p < .000$. However, there was no significant difference in arousal ratings between sad and neutral excerpts, $p = .522$.

Pleasure ratings were significantly different between excerpts, $F(1.80, 172.49) = 1263.83$, $p < .000$, $\eta 2 = .93$. Happy excerpts were rated significantly higher in pleasure than sad and neutral excerpts, $p < .000$, but there was no significant difference in pleasure ratings between sad and neutral excerpts, $p = .622$.

Liking ratings significantly differed between excerpts, $F(1.87, 179.20) = 116.09$, $p < .000$, $\eta 2 = .76$. Happy excerpts were rated significantly higher in liking than sad and neutral excerpts, $p < .000$, but again there was no significant difference in liking ratings between sad and neutral excerpts, $p = .133$.

Table 2.1

Mean (SD) of each rating by excerpt

Excerpt	Emotion Category	Arousal	Pleasure	Liking
Ants Marching	Happy	5.32(1.10)	5.34(1.06)	2.78(1.44)
*Outside Villanova	Happy	6.13(.77)	6.52(.54)	5.96(1.00)
*Angel of Harlem	Happy	6.22(.77)	6.62(.49)	6.10(1.14)
*Hey Soul Sister	Happy	6.29(.77)	6.57(.54)	5.57(1.32)
*She's Electric	Happy	6.49(.69)	6.65(.48)	5.55(1.33)
Crosstown Traffic	Happy	4.66(1.09)	3.96(1.40)	2.90(1.30)
Brick	Sad	2.38(1.45)	3.61(1.25)	2.87(1.15)
Hopeless	Sad	2.82(1.40)	2.85(1.16)	2.54(2.37)
Hundred	Sad	2.23(1.32)	2.12(.92)	2.29(1.00)
Colorblind	Sad	3.23(1.32)	4.46(1.33)	4.58(1.42)
God of Wine	Sad	2.64(1.18)	2.10(1.53)	1.82(.77)
The Scientist	Sad	3.06(1.26)	4.19(1.33)	4.39(1.30)
Sweet and Low	Neutral	2.72(1.12)	2.55(1.13)	3.08(1.53)
Captain	Neutral	2.59(1.04)	2.52(1.55)	2.30(1.34)
Save Your Scissors	Neutral	2.18(1.16)	4.24(1.27)	3.20(1.83)
Death Defied by Will	Neutral	2.21(1.04)	2.73(1.18)	2.36(1.59)
Here is Gone	Neutral	3.89(1.37)	3.24(1.32)	3.08(1.35)
Without Reason	Neutral	3.40(1.23)	3.18(1.76)	3.35(1.34)

*Happy excerpts used in main experiment

Table 2.2

Mean (SD) for each emotion category by rating

Emotion Category	Arousal	Pleasure	Liking
Happy	5.86(.35)	5.94(.32)	4.08(.51)
Sad	2.72(.57)	3.17(.53)	3.08(.58)
Neutral	2.83(.51)	3.08(.59)	2.88(.62)

Based on these results showing that 1) 4 happy excerpts were rated consistently higher in all ratings compared to other excerpts and 2) the averaged rating for all happy excerpts were significantly higher in arousal, pleasure, and liking compared to the averaged ratings of sad and neutral

excerpts, the 4 Happy excerpts (marked with a * in Table 2.1) were chosen for use in the main experiment. However, because no significant differences were found between the averaged ratings for all sad excerpts compared to the averaged ratings of all neutral excerpts these results were considered inconclusive and a second pilot study was necessary in order to select excerpts from these 2 emotion categories.

2.8. Pilot Study 2

A second survey (pilot study 2) was then conducted. The second survey followed the same procedure as the first survey, but contained 14 excerpts (6 sad excerpts from a pop genre, 3 sad excerpts from a classical genre, and 5 neutral excerpts from a pop genre). See Appendix P for excerpt list. For this pilot study there were 3 sad excerpts from a classical genre chosen in order to compare ratings between established sad excerpts (classical excerpts) and between other sad excerpts (pop genre excerpts). The classical excerpts, *Adagio for String, Kol Nidre,* and *Schindler's List Theme* have all been previously used as sad musical stimuli in previous experiments (Krumhansl, 1997; Peretz et al., 1998; Vieillard et al., 2008).

There were 61 participants with a mean age of 21.79 years (SD = 3.07) who took part in the online survey. For each excerpt, participants were asked to rate 6, 7-point scales: (1) arousal, (2) pleasure, (3) happy, (4) sad, (5) familiar, and (6) liking. Although it is common for valence (happy/sad) to be rated on a single scale this survey used separate scales for happy and sad emotions in order to measure the intensity of each emotion. From the second survey sad excerpts from the pop genre were chosen based on those excerpts that 1) had an average sad rating of > 3.5, 2) had an average happy rating of < 3.5, 3) had the largest discrepancy between happy and sad ratings, and 4) had the lowest ratings of arousal for pop genre excerpts. Because 3.5 is the midpoint of all rating scales, sad ratings above this midpoint and happy ratings below this midpoint were considered to be indicative of a sad emotional response. Neutral excerpts were determined based on those excerpts that had 1) average sad and happy ratings at ~3.0 and 2) had the smallest discrepancy between happy and sad ratings. Average happy and sad ratings of ~3.0 were chosen as neutral

is thought to contain minimal emotional responses and therefore should have ratings below the midpoint of 3.5.

2.8.2. Results

First, mean and standard deviations were calculated for each of the 14 excerpts and for each rating (happy, sad, arousal, pleasure, familiar, liking) in order to determine which excerpts from each emotion category (sad, neutral) had the highest rating. Table 2.3 displays the mean and standard deviation for each excerpt and for each rating. Next, difference scores were calculated for each excerpt by subtracting sad ratings from happy ratings. This information is shown in the Sad-Happy Discrepancy column in Table 2.3.

Table 2.3

Mean (SD) of each rating by excerpt

Excerpt	Emotion Category	Happy	Sad	Sad - Happy Discrepancy	Arousal	Pleasure	Familiar	Liking
*Colorblind	Sad	2.41(1.54)	4.31(1.63)	1.90	2.10(1.61)	4.80(2.00)	2.93(2.35)	4.18(2.00)
*Everybody Hurts	Sad	2.52(1.26)	3.87(1.53)	1.35	2.37(1.08)	4.43(1.90)	5.13(2.38)	5.02(1.67)
Unchained Melody	Sad	3.55(1.71)	2.86(1.70)	-.69	2.71(1.34)	4.72(1.75)	6.47(1.17)	5.16(1.86)
*Do What You Have To Do	Sad	2.49(1.59)	3.89(1.77)	1.40	2.36(1.36)	3.98(1.89)	2.45(2.09)	4.37(1.84)
*Foolish Games	Sad	2.35(1.80)	3.77(1.69)	1.42	2.02(1.06)	4.31(2.13)	3.35(2.53)	4.46(2.06)
Someone Like You	Sad	2.50(1.48)	3.63(2.01)	1.13	2.79(1.46)	3.65(2.11)	6.03(1.85)	4.54(2.33)
Adagio for Strings	Sad	2.68(1.63)	3.70(2.15)	1.02	2.95(1.73)	4.97(1.86)	5.24(2.27)	4.97(2.22)
Schindler's List Theme	Sad	2.34(1.42)	4.30(1.96)	1.96	2.30(1.20)	4.45(1.92)	3.32(2.46)	5.26(1.93)
Kol Nidre	Sad	1.89(1.13)	4.02(1.84)	2.13	2.23(1.26)	3.55(1.87)	2.80(1.98)	4.20(1.76)
Fur Immer	Neutral	2.93(1.33)	2.11(1.21)	-.82	2.67(1.52)	2.76(1.54)	1.46(.99)	2.70(1.53)
Hallogallo	Neutral	3.15(1.32)	1.90(1.10)	-1.25	3.19(1.45)	3.08(1.37)	1.79(1.52)	3.08(1.46)
†Seeland	Neutral	2.05(1.20)	2.05(1.36)	0.00	2.32(1.53)	2.50(1.55)	1.50(1.22)	2.73(1.60)
†Negativland	Neutral	2.32(1.46)	1.80(1.31)	-.52	2.80(1.68)	2.62(1.63)	1.38(1.06)	2.68(1.87)
The Scientist	Neutral	3.45(1.77)	3.43(1.74)	-.02	3.13(1.65)	4.75(1.92)	5.15(2.34)	5.13(1.98)

*Sad excerpts used in main experiment; † Neutral excerpts used in the main experiment

Next, averages for each rating (happy, sad, arousal, pleasure, familiar, liking) were calculated for sad pop excerpts, sad classical excerpts, and neutral excerpts. A repeated measures ANOVA was then performed- with an independent variable of excerpt with 3 levels (sad pop excerpts, sad classical excerpts, and neutral excerpts) and a dependent variable of ratings with 6 levels (happy, sad, aroused, pleasure, familiar, and liking). Again, where the assumption of sphericity was violated a Greenhouse-Geisser correction was used and for multiple comparisons a Bonferonni correction was applied.

Results show a significant difference between excerpts and ratings, $F(12, 25) = 11.85$, $p < .000$, $\eta 2 = .85$. Happy ratings were not found to be significantly different between excerpts, $F(2, 72) = 2.61$, $p = .080$, $\eta 2 = .07$. Also, arousal ratings were not found to be significantly different between excerpts, $F(1.64, 59.12) = 2.34$, $p = .115$, $\eta 2 = .06$. However, sad ratings were significantly different between excerpts, $F(1.72, 61.76) = 48.07$, $p < .000$, $\eta 2 = .57$. More specifically, neutral excerpts were rated significantly less sad ($M = 2.15$, $SE = .15$) than sad pop excerpts (M = 3.63, SE .21) and sad classical excerpts ($M = 4.04$, $SE = .27$), $p < .000$. Importantly, there was no significant difference in sad ratings between pop and classical sad excerpts, $p = .190$.

There was also a significant difference in pleasure ratings between excerpts $F(2, 72) = 18.80$, $p < .000$, $\eta 2 = .34$, whereby neutral excerpts were rated significantly less pleasurable ($M = 3.04$, SE = .19) compared to sad classical excerpts ($M = 4.48$, $SE = .24$) and sad pop excerpts ($M = 3.98$, $SE = .20$), $p < .000$. Importantly, there was no significant different in pleasure ratings between sad classical and sad pop excerpts, $p = .104$.

There was also a significant difference in familiar ratings between excerpts, $F(1.72, 61.74) = 30.70$, $p < .000$, $\eta 2 = .46$. Neutral excerpts were significantly less familiar ($M = 2.25$, $SE = .16$) than sad classical excerpts ($M = 3.63$, $SE = .28$) and sad pop excerpts ($M = 4.33$, $SE = .21$), $p < .000$. However, there was no significant difference in ratings of familiarity between sad classical excerpts and sad pop excerpts, $p = .063$.

Lastly, there was a significant difference in liking ratings between excerpts, $F(2, 72) = 27.58$, $p < .000$, $\eta 2 = .43$. Neutral excerpts were rated significantly lower in liking ratings ($M = 3.24$, $SE = .20$) compared to sad

classical excerpts (*M* = 5.00, *SE* = .22) and sad pop excerpts (*M* = 4.51, *SE* = .18), $p < .000$. Again, there was no significant difference in ratings of liking between sad classical excerpts and sad pop excerpts, $p = .082$.

Importantly, the results from pilot study 2 show 1) no significant differences in any ratings between sad pop excerpts and sad classical excerpts and 2) sad pop excerpts to be significantly more sad, pleasurable, familiar, and liked compared to neutral excerpts. Therefore, sad pop excerpts with the highest ratings in sadness and the largest discrepancies between sad and happy ratings were chosen for use in the main experiment. These excerpts include, *Colorblind, Everybody Hurts, Foolish Games,* and *Do What You Have To Do* and are marked with a * in Table 2.3. Furthermore, neutral excerpts were found to be significantly lower in ratings of sadness, pleasure, familiarity, and liking compared to sad excerpts (both pop and classical) in. Although 3 neutral excerpts met the criteria for use in the main experiment (*Fur Immer, Seeland,* and *Negativland*) the excerpt, *Fur Immer*, was found to have a large discrepancy between sad and happy ratings (-.82) and was therefore omitted from the main experiment. Therefore, only 2 neutral excerpts were used in the main experiment, *Seeland* and *Negativland.* Overall, the main experiment included 4 happy, 4 sad, and 2 neutral excerpts. See Appendix E for the excerpt list.

2.9. Participants

For the main study, 125 participants living in England were recruited. Many were recruited with a flyer (Appendix A) as well as through a convenience sample. I recruited 61 smokers and 64 nonsmokers. Table 2.4 provides a summary of the age and gender for each group by smoking status (nonsmoking, smoking) and nicotine dose (0, 2, 4 mg). Furthermore, Table 2.5 and Table 2.6 display occupation as well as nicotine and music consumption information for smokers and nonsmokers, respectively. Smokers were defined as smoking at least seven cigarettes per day for at least two years and who scored a minimum of five on the Fagerström Test for Nicotine Dependence (Heatherton et al., 1991). Nonsmokers were defined as smoking less than seven cigarettes in a lifetime and who scored a maximum of two on the Fagerström Test for Nicotine Dependence (Heatherton et al., 1991). Although no participants were

professional musicians, 65% had musical performance experience to at least a high school level. Informed consent was obtained prior to experimentation and participants were paid £5 for their time. The research protocol met the ethical requirements of the University of Sheffield's Department of Psychology.

An independent *t*-test shows there was a significant difference in age between smokers and nonsmokers, $t(117) = 2.05$, $p = .042$, $d = .38$, where smokers were significantly older ($M = 25.54$, $SD = 7.92$) than nonsmokers ($M = 22.74$, $SD = 6.87$). An independent t-test shows there was no significant difference in the hours per week in which smokers and nonsmokers consumed music, $t(123) = .08$, $p = .936$, $d = .01$. In fact, smokers ($M = 18.95$, $SD = 16.87$) and nonsmokers ($M = 18.69$, $SD = 19.36$) consumed nearly equal amounts of music per week. Importantly, there was a significant difference in the number of cigarettes consumed per week by smokers compared to the number of cigarettes consumed in a lifetime by nonsmokers, $t(123) = 9.82$, $p < .000$, $d = 1.76$. Smokers consumed a significantly higher number of cigarettes per week ($M = 11.74$, $SD = 5.49$) than nonsmokers consumed in a lifetime ($M = 2.25$, $SD = 5.31$).

Table 2.4

Age and gender by smoking status and nicotine dose

	Smokers			Nonsmokers		
Nicotine Dose	*N*	Age	Gender	*N*	Age	Gender
0 mg	20	M = 27.85; SD = 10.56	10 M; 10 F	21	M = 23.30; SD = 7.62	9 M; 12 F
2 mg	20	M = 25.65; SD = 6.76	9 M; 11 F	22	M = 22.17; SD = 7.63	8 M; 14 F
4 mg	21	M = 23.50; SD = 5.29	10 M; 10 F	21	M = 22.70; SD = 5.57	7 M; 14 F

Table 2.5

Occupation and nicotine consumption for smokers by nicotine dose

	Smokers			
Nicotine Dose	Occupation	Average # of Cigarettes/day	Average Time Smoking (years)	Average Music Consumption (h/wk)
0 mg	Student UG 50% Student PG 25% *Non-student 25%	M = 10.25, SD = 4.63	8.80 years	M = 14.30, SD = 11.33
2 mg	Student UG 45% Student PG 40% *Non-student 15%	M = 14.30, SD = 7.27	9.53 years	M = 24.10, SD = 23.34
4 mg	Student UG 61.9% Student PG 28.6% *Non-student 9.5%	M = 10.71, SD = 3.13	6.47 years	M = 18.48, SD = 12.87

*Non-student employment included baker, civil servant, mental health advisor, theatre manager, administrator, care assistant, porter, and unemployed.

Table 2.6

Occupation and nicotine consumption for nonsmokers by nicotine dose

	Nonsmokers		
Nicotine Dose	Occupation	Average # of Cigarettes Smoked in Lifetime	Average Music Consumption (h/week)
0 mg	Student UG 52.4% Student PG 42.9% *Non-student 4.8%	M = 2.71, SD = 8.65	M =17.67, SD = 25.81
2 mg	Student UG 72.7% Student PG 9.1% *Non-student 18.2%	M = 2.00, SD = 2.18	M = 19.59, SD = 17.12
4 mg	Student UG 61.9% Student PG 38.1%	M = 2.05, SD = 2.94	M = 18.76, SD = 14.30

*Non-student employment included teacher, lecturer, researcher, and unemployed.

2.10. Material

2.10.1. Questionnaires

A musical background questionnaire (Appendix B) was administered to determine the extent of participants' musical knowledge and performance experience. A smoking history questionnaire (Appendix C) was also administered in order to determine participants' smoking status (e.g. smoker or nonsmoker) and eligibility. Eligible participants were required to complete a health screening survey (Appendix D) regarding their past and present physical and psychological health. This was to ensure that participants were healthy enough to receive nicotine and would not be endangering themselves or others by ingesting the drug. The health screening particularly asks if the participant has been diagnosed with a serious medical condition (e.g. angina, schizophrenia) or whether they are, or could be, pregnant. Participants also had their blood pressure measured to ensure that those with hypertension (a reading of 140/80 or higher) did not participate. Also, after receiving nicotine participants were administered the Subjective Treatment Emergent Symptom Scale (STESS) (Guy, 1976b) to assess their physical reactions to nicotine and the severity of any adverse side effects. Participants with a score of 50% or higher would have been discontinued from the study. However, no participants were discontinued for this reason.

2.10.2. Nicotine Gum

The nicotine polacrilex gum (2 mg and 4 mg) was Boots NicAssist ice mint flavored gum. For placebo Wrigley's Extra peppermint flavored chewing gum was chosen because of similar size, shape, and color to the nicotine gum. Polacrilex gum was chosen because it provides an administration method that can control the amount of nicotine given to each participant. This is especially true when a standardized chewing protocol is used, as it decreases individual response variability, and nicotine plasma levels are directly related to dose (Henningfield, London, & Benowitz, 1990). Furthermore, nicotine polacrilex gum has shown to have low dependence potential and toxicity (Heishman et al., 1993), which is particularly important for nonsmoking participants. For example, there have not been reports of any primary addictions that have developed in response to nicotine gum despite its widespread availability.

2.10.3. Musical excerpts

The musical material included 10 musical excerpts (4 happy, 4 sad, 2 neutral) that were 2 min in length. All excerpts were classified as 'pop' music (e.g. not classical music) and were chosen from music categories in iTunes associated with 'pop' (e.g. rock, alternative, new releases). See Appendix E for excerpt list. Each participant also self-selected a 2 min excerpt of chill-inducing music. Chill-inducing music was defined as any piece of music known to consistently and reliably bring one to chills, as based on the methods of Blood and Zatorre (2001). Because music preference is highly individualized, asking participants to self-select one piece of music was the most reliable way to ensure intense emotional responses with in participants (Thaut & Davis, 1993). Participants were allowed to select any music they liked without constrictions and were asked to email their song choice ahead of time to the experimenter.

2.10.4. Reading material

Two distraction tasks were administered during the experiment. The first was a 15 min reading task of chapter one of *Music: A Very Short Introduction* (Cook, 1998) and the second was a 10 min writing task consisting of open-ended essay questions regarding the reading material (Appendix F). I wanted to keep participants in a neutral state so that their affect was only influenced by music and nicotine. However, when chewing nicotine gum the taste and burning sensation from the nicotine may be a negative experience. Therefore I provided two neutral distraction tasks to keep participants' focused away from the taste of the gum. The distraction tasks also helped to pass the time as 25 min of chewing may become boring and subsequently cause participants to grow disinterested. Therefore, two neutral distractor tasks were administered and participants were told that they were used to assess the effects of nicotine on reading comprehension.

2.10.5. Rating scales

Self-reported ratings were used as a subjective measure of pleasure, arousal and emotion as well as a manipulation check in order to confirm that responses were elicited from the listener. Ratings were taken using 6, 7-point scales: (1) pleasantness (unpleasant-very pleasant), (2) arousal (sleepy-

energetic), (3) happy (happy- unhappy), (4) sad (sad – not sad), (5) liking (not at all- very much), and (6) familiar (very familiar- very unfamiliar).

2.10.6. Carbon monoxide testing

Using a Bedfont Micro Smokerlyzer carbon monoixide (CO) meter all participants underwent a CO reading immediately preceding experimentation to confirm that they had not recently smoked. If participants had an expired CO level greater than 5 ppm then they were not allowed to participate in the study. However, no participant was excluded for this reason.

2.10.7. Physiological equipment

Physiological measurements were recorded using the ProComp5 Inifiniti 5-channel system with Biograph Inifiniti software (Thought Technology Ltd Canada). I simultaneously recorded heart rate, skin conductance, respiration rate, and skin temperature. Heart rate and skin conductance were recorded via finger sensors that were attached to Velcro bands. These bands wrap around the fingertips of participants. Skin temperature was recorded via a thermistor that was taped to the palm of the hand. Respiration rate was recorded by stretching a Velcro belt around the chest of the participant with a sensor placed over the diaphragm. Figure 2.2 shows the physiological sensors and how they are placed on the body.

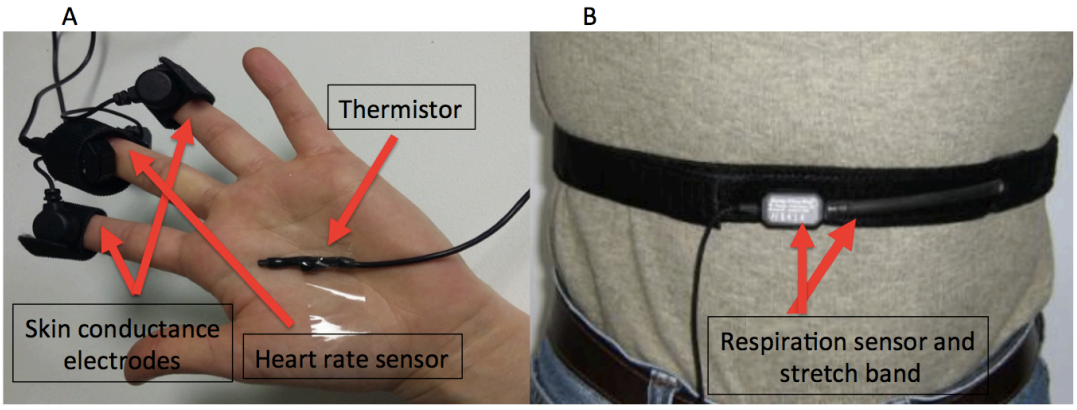

Figure 2.2. Body placement for heart rate, skin conductance, respiration rate, and skin temperature sensors, shown in section A and B respectively.

2.10.8. Physiological data acquisition

The Biograph Infiniti hardware includes 5 simultaneous feedback channels, which allows for real-time biophysical data acquisition processing and display. All sensors have a sampling rate of 256 samples/s.

Heart rate data was acquired through photoplethysmography, which bounces infra-red light against the surface of the skin to measure the amount of light reflected back. At a pulse/heart beat there is more blood in the skin and therefore more red light reflected. However, between pulses the amount of blood decreases and therefore more red light is absorbed and less is reflected.

Skin conductance data was acquired by applying a small electrical voltage through two electrodes. This establishes an electric circuit and allows the participant to act as the resistor. The sensors then measure resistance and from this calculate conductance, which is the inverse of resistance.

The respiration data was acquired through the stretch belt and diaphragm sensor, which records and converts expansion and contraction of the participant's chest to breaths/min for further analysis. Lastly, peripheral temperature (skin temperature) data is acquired through a thermistor. A thermistor is a resistor whose resistance is dependent upon temperature. In this way, the device converts changes in temperature to changes in electrical current.

2.11. Procedure

Before the experiment participants filled out a series of questionnaires regarding their musical background (Appendix B), smoking habits (Appendix C), and health (Appendix D). This information was necessary in order to screen participants and ensure that they were 1) non-musicians, 2) could be classified as either a smoker or nonsmoker and 3) healthy enough to ingest nicotine. Upon confirmation of eligibility an appointment for the study was scheduled and participants were requested to refrain from all products containing nicotine, caffeine, and alcohol for 24 h before their experiment.

Participants began the experiment by reading an information sheet (Appendix G) and providing informed consent. Next, the physiological sensors were attached. The respiration belt was stretched around the chest securely with the sensor placed over the diaphragm. The heart rate sensor was then wrapped around the fingertip of the middle finger of the dominant hand. Next, the skin conductance sensors were wrapped around the fingertip of the index and ring fingers. Lastly, the thermistor (temperature sensor) was tapped to the

palm with medical tape. Once all sensors were secure, participants were requested to leave their hand facing upwards and to move as little as possible throughout the experiment. A 2 min baseline recording of physiological arousal was then taken by having participants sit in silence. This was followed by baseline self-reports of arousal, pleasure, and emotion ratings.

After baseline readings participants were randomly assigned to either the placebo, 2, or 4 mg nicotine condition and administered the corresponding gum (regular chewing gum or nicotine gum). Participants then chewed the gum for 25 min. They used a chewing-resting cycle of 30 s, whereby they chewed for 10 s, then rested the gum inside their cheek for 20 s. By resting the gum inside the cheek this allows the nicotine to gradually be absorbed by the buccal mucosa (lining of the cheeks) and released into the blood stream, and allows it to stay in the blood stream for ~30-45 min. This yields nicotine levels comparable to smoking a commercial cigarette (4 mg) or a half a cigarette (2 mg) and is a chewing method used previously in nicotine research (Benowitz et al., 1988; Ernst, Heishman, Spurgeon, & London, 2001). To help participants remember the chewing-resting cycle an audio file was played that sounded a high bell tone when participants were to begin chewing and a low alarm tone when they were to rest. During the 25 min chewing task participants were engaged in two distraction tasks, a 15 min reading task (Cook, 1998), and a 10 min writing task consisting of open-ended essay questions regarding the reading material (Appendix F). After 25 min participants discarded the gum and were administered the STESS (Guy, 1976b) to check for adverse effects of nicotine. If participants scored 50% or higher on any of the four questions they were discontinued from the study; however, no participants were discontinued for this reason. In order to assess the effects of nicotine, physiological recordings and self-reports were then taken.

The main music listening task then began. Participants listened to four musical excerpts (happy, sad, neutral, self-selected/chill-inducing), which were presented in random order to account for order effects. During each listening physiological measurements were recorded and immediately after listening self-reports were taken.

2.12. Data analysis

In order to analyze physiological data an average score for each physiological measurement (HR, SCL, RR, and ST) was first calculated for each 2 min recording session (e.g. baseline pre-ingestation, baseline post-ingestation, happy excerpt, sad excerpt, neutral excerpt, and chill-inducing excerpt). That is, each physiological measurement had a sampling rate of 256 samples/s and at the end of each 2 min recording session these samples were averaged together to produce a temporal mean score for each physiological measurement and for each recording session. This yielded twenty-four temporal mean scores (4 physiological measurements x 6 recording sessions). Once these mean scores were calculated, post-ingestation baseline scores for each physiological measurement were subtracted from their subsequent and corresponding recording sessions that contained musical excerpts. For example, once calculated, the HR score recorded after nicotine ingestation was subtract from the HR score recorded during each of the four music conditions (happy music, sad music, neutral music, and chill-inducing music). In this way, I computed change scores for each physiological measurement by subtracting each participant's post-ingestation baseline score from his or her post-ingestation score during each of the four musical conditions. The same calculation was performed for self-reported data. Post-ingestation baseline ratings of arousal, pleasure, happiness, and sadness were subtracted from their subsequent and corresponding ratings for each of the four music categories (happy music, sad music, neutral music, and chill-inducing music). In total, for each cohort (smoker, nonsmoker) this yielded 16 change scores for the physiological variables and 16 change scores for the self-reported ratings.

The data were then analyzed to compare physiological (section 2.13-section 2.15) and self-reported (section 2.16 – section 2.18) response. First, in order to compare smokers' and nonsmokers' physiological responses to nicotine and music a repeated measures MANOVA was performed with between subjects variables of smoking status (two levels – smoking, nonsmoking) and nicotine condition (three levels – 0, 2, 4 mg), a within subjects variable of music (four levels – happy, sad, neutral, chill-inducing), and a dependent variable of physiological response (four levels – HR, SCL, RR, ST).

Two follow up repeated measures MANOVAs were then performed in order to examine the physiological responses of smokers and nonsmokers separately. Therefore, a repeated measures MANOVA was performed once for smokers and then again for nonsmokers. There was a between subjects variable of nicotine condition (three levels – 0, 2, 4 mg), a within subjects variable of music (four levels- happy, sad, neutral, chill-inducing), and a dependent variable of physiological response (four levels – HR, SCL, RR, ST).

Where relevant (if multivariate tests were statistically significant) the effects of nicotine across the music types were further examined by performing a series of one way univariate ANOVAs for each dependent measure (HR, SCL, RR, ST) and for each cohort (smoking, nonsmoking). If necessary, these were further followed up with *t*-tests. Due to the restricted number of comparisons (0 vs 2 mg; 2 mg vs 4 mg), follow up *t*-tests used a significance threshold (p value) of $p = .0125$.

The subsequent analysis involving self-reported responses follows the same structure as that of the physiological analysis. That is, in order to compare smokers' and nonsmokers' self-reported responses to nicotine and music a repeated measure MANOVA was performed with between subjects variables of smoking status (two levels – smoking, nonsmoking) and nicotine condition (three levels – 0, 2, 4 mg), as well as a within subjects variable of music (four levels – happy, sad, neutral, chill-inducing). The dependent variable was self-reported responses (four levels – arousal, pleasure, happiness, sadness).

Two follow up repeated measures MANOVAs were then performed in order to examine the self-reported responses of smokers and nonsmokers separately. A repeated measures MANOVA was performed once for smokers and then again for nonsmokers. There was a between subjects variable of nicotine condition (three levels – 0, 2, 4 mg), a within subjects variable of music (four levels- happy, sad, neutral, chill-inducing), and a dependent variable of self-reported responses (four levels – arousal, pleasure, happiness, sadness).

Where relevant, the effects of nicotine across the music types where examined further using a series of one-way univariate ANOVAs. These

univariate tests were performed separately for each dependent physiological measure (HR, SCL, RR, ST) and each self-reported measure (arousal, pleasure, happiness, sadness) and for each cohort (smoking, nonsmoking). These were followed up with *t*-tests when needed. Due to the restricted number of comparisons (0 vs 2 mg; 2 mg vs 4 mg), follow up *t*-tests used a significance threshold (*p* value) of $p = .0125$.

For all repeated measures MANOVAs variables found to violate the assumption of sphericity were corrected with a Greenhouse-Geisser correction and all post-hoc tests were corrected with a Bonferroni correction.

2.13. Results

The study examined the effects of music and nicotine on both the physiological and self-reported arousal/pleasure/emotional reactions of participants. Analyses were performed separately for physiological and self-reported data. The first set of results reports the physiological responses to nicotine (section 2.13 - 2.15). The second set of results reports the self-reported arousal/pleasure/emotional responses (section 2.16 – 2.18). To organize the results more clearly, the analysis involving physiological responses is divided into 3 sub-sections: first, a main effect of nicotine is presented (section 2.13), then a main effect of music (section 2.14), and finally an interaction effect of nicotine and music (section 2.15). The analysis involving self-reported responses is also divided into 3 sub-sections: first, a main effect of nicotine (section 2.16), then a main effect of music (section 2.17), then an interaction effect of nicotine and music (section 2.18).

After computing change scores for physiological arousal several variables were found to violate the assumption of normality. That is, several variables had an absolute value of skewness and kurtosis that were more than twice the standard error, indicating that the data was not symmetrical. Because each nicotine condition contained an equal number of participants ($N = 20$) and because the ANOVA test is robust to violations of the normality assumptions (Harwell, Rubinstein, Hayes, & Olds, 1992) the data was not transformed. Instead, I calculated the mean and standard deviation of each variable and then removed any scores that were more than three standard deviations away from the mean (Howitt & Cramer, 2005). Based on this criterion, in

measurements of heart rate I removed three outliers from happy music, two outliers each from sad and chill-inducing music, and four outliers from neutral music. In measurements of skin conductance level I removed one outlier each from happy, sad, and chill-inducing music, and three outliers from neutral music. In measurements of respiration rate I removed four outliers from happy music, two outliers from sad music, and three outliers each from neutral and chill-inducing music. In measures of skin temperature I removed one outlier each from happy and neutral music, three outliers from sad music, and two outliers from chill-inducing music. All subsequent analyses involving these variables were conducted with these outliers removed. The scores removed were also visually inspected using histograms and by referencing the raw data. This was done in order to confirm that the scores removed were indeed outliers. Most, but not all outliers removed were found to be values that were beyond the scope of human physiological responses (e.g. an increase in HR of 153.85 beats/min).

2.14. Effects of nicotine on physiological arousal

The following section reports the main effect of nicotine on the physiological response, first between smokers and nonsmokers (section 2.14.1), then on smokers (2.14.2), and finally on nonsmokers (2.14.3).

2.14.1. Effects of nicotine on physiological arousal between smokers and nonsmokers

A multivariate test showed a nonsignificant difference between smokers' and nonsmokers' physiological responses to nicotine, $F(4, 94) = 1.01$, $p = .408$, $\eta 2 = .04$. Although nonsignificant, cohort comparisons in Figure 2.2 and Figure 2.3 show smokers and nonsmokers to have similar HR and SCL responses. That is, for smokers and nonsmokers, nicotine increased HR above placebo and this increase was most pronounced at the low dose of 2 mg. For both cohorts as nicotine dose increased SCL systematically decreased. Respiration responses were different between cohorts. Showing that for smokers, as nicotine dose increase there was a systematic decrease in respiration. However, there was a negligible difference between the 2 and 4 mg conditions. For nonsmokers, there was a decrease in respiration at the low dose of 2 mg, but negligible changes in respiration in the placebo and 4 mg conditions. For skin temperature responses

both cohorts showed all conditions to decrease this measurement. However, the responses were somewhat different between cohorts. Smokers showed a further decrease in skin temperature in response to nicotine, with the largest decrease at the 2 mg dose. However, nonsmokers showed that as nicotine dose increased, skin temperature systematically increased compared to placebo. These physiological responses can be viewed in Figure 2.2 and Figure 2.3.

2.14.2. Effects of nicotine on physiological arousal in smokers

A multivariate test revealed a nonsignificant main effect of nicotine on physiological arousal in smokers, $F(8, 86) = 1.58$, $p = .144$, $\eta 2 = 13$.

Although these results were nonsignificant main effect of nicotine trends can be seen in the data in in Figure 2.2. and Figure 2.3. For example, it is clear that both doses of nicotine increased HR more than placebo and that the low dose of 2 mg increased HR the most. Furthermore, it is clear that as nicotine levels increased SCL systematically decreased. Trends in respiration rates show that both doses of nicotine increased respiration less than placebo. Lastly, trends in skin temperature show nicotine to decrease skin temperature more than placebo, with 2 mg decreasing it the most.

2.14.3. Effects of nicotine on physiological arousal in nonsmokers

A multivariate test revealed no significant effect of nicotine on physiological arousal in nonsmokers, $F(8, 96) = .94$, $p = .485$, $\eta 2 = .07$; however, some trends can be in Figure 2.2 and Figure 2.3. For example, as with smokers, it is clear that both doses of nicotine increased HR more than placebo and that 2 mg increased heart rate the most. Also similar to smokers, as nicotine levels increased SCL systematically decreased in nonsmokers. Trends in respiration rate are less clear, showing little change in the placebo and 4 mg conditions, but a marked decreased at the 2 mg dose. Lastly, skin temperature decreased for all conditions and as nicotine dose increased skin temperature systematically increased compared to placebo.

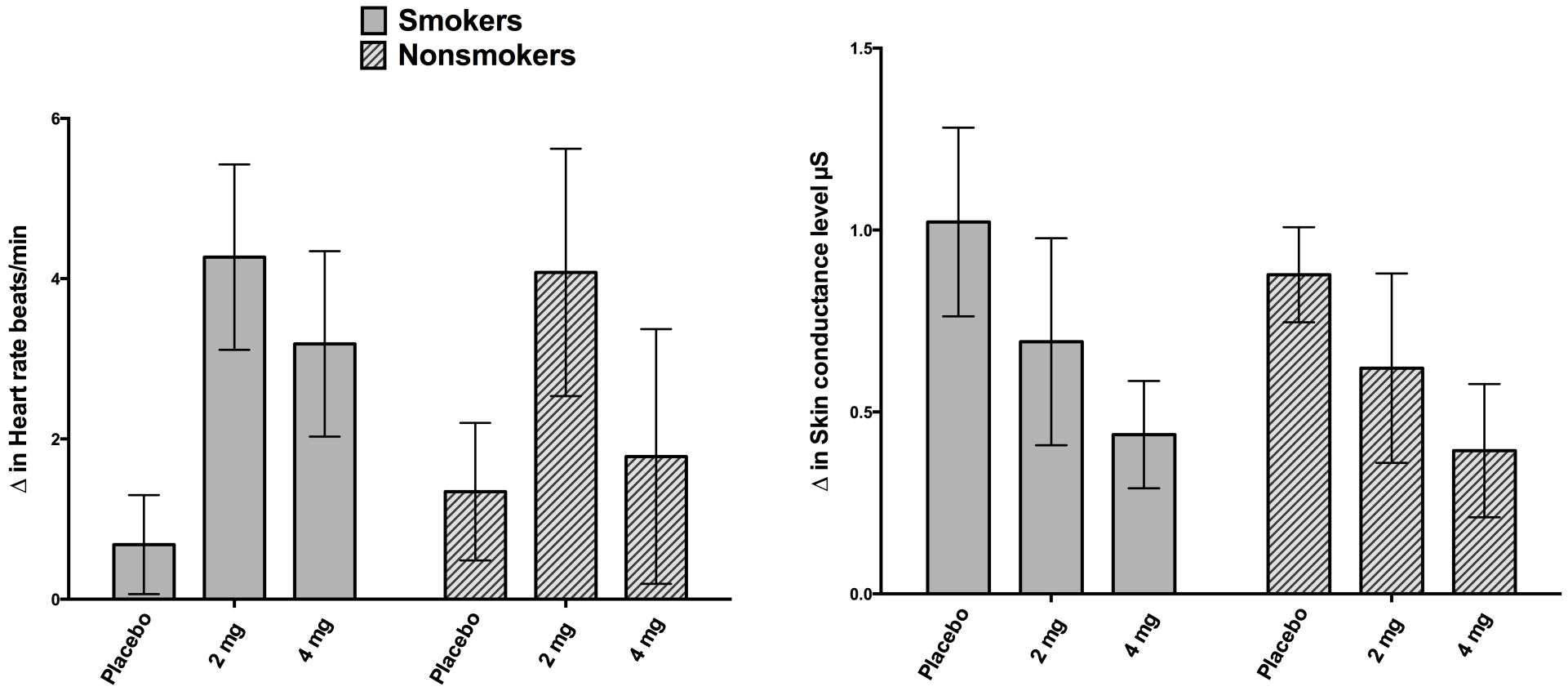

Figure 2.3. The mean and standard errors for smokers' and nonsmokers' heart rate and skin conductance level responses to each nicotine condition.

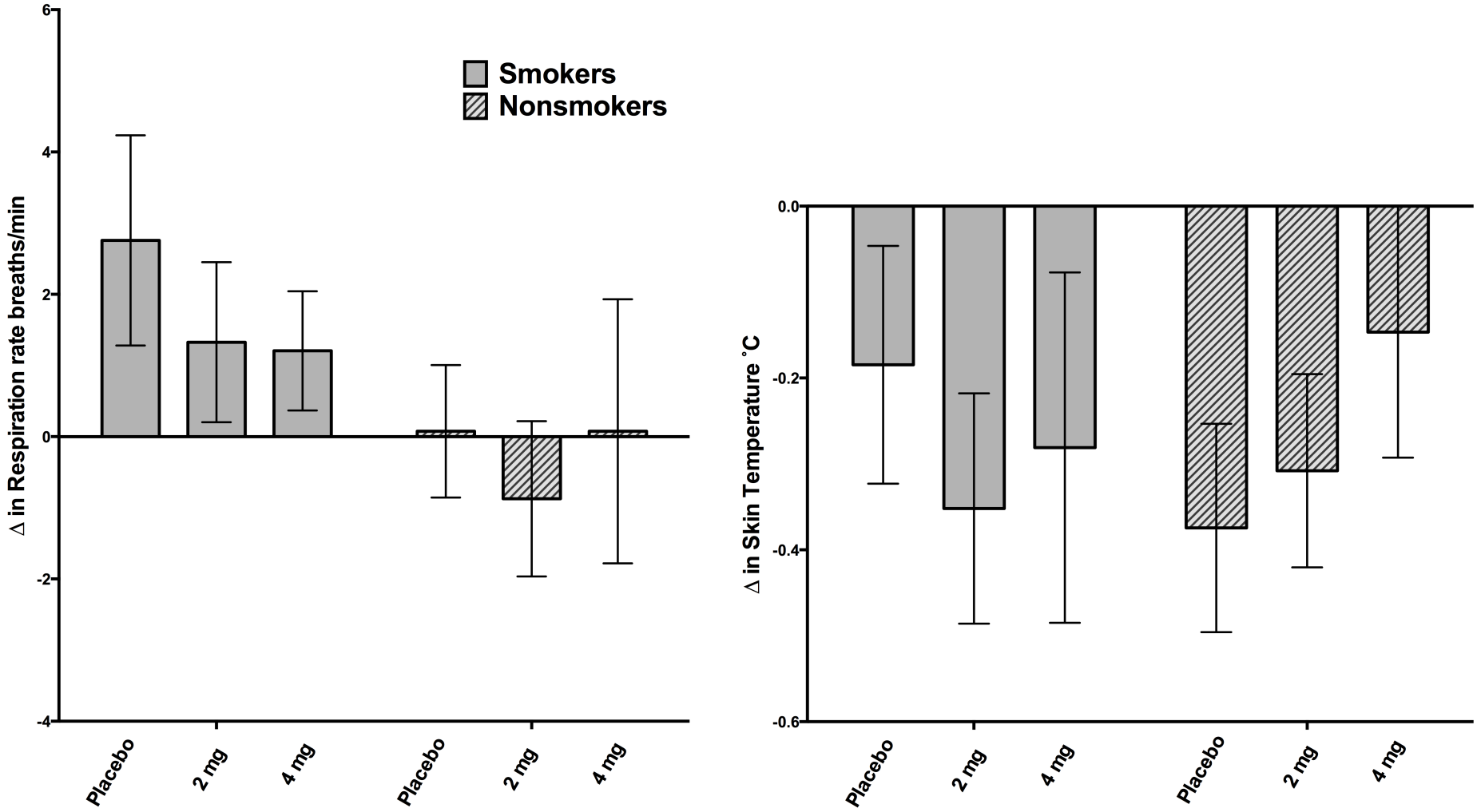

Figure 2.4. The mean and standard errors for smokers' and nonsmokers' respiration rate and skin temperature responses to each nicotine condition.

2.15. Effects of music on physiological arousal

The following section reports the main effect of music on the physiological response, first between smokers and nonsmokers (section 2.15.1), then on smokers (2.15.2), and finally on nonsmokers (2.15.3).

2.15.1. Effects of music on physiological arousal between smokers and nonsmokers

A multivariate test showed a nonsignificant difference between smokers' and nonsmokers' physiological responses to music, $F(12, 86) = 1.58$, $p = .112$, $\eta 2 = .18$. That is, in response to music there was no significant difference between smokers' and nonsmokers' physiology. Cohort comparisons shown in Figure 2.4 and Figure 2.5 reflect this as smokers and nonsmokers showed similar physiological response to music. For example, both cohorts showed a larger increase in HR during happy and chill-inducing music compared to during sad and neutral music, with chill-inducing music showing a markedly greater increase than the other music types. However, HR was lowest in smokers during neutral music, but lowest in nonsmokers during sad music. SCL showed the most similarities between cohorts. Happy and chill-inducing music increased in SCL more than sad and neutral music, with sad music increased SCL the least. Respiration rate was the most different between cohorts. Smokers showed happy and neutral music to increase in respiration rate more than sad and neutral music, while nonsmokers showed a decrease in respiration during all music conditions except happy music. Skin temperature was similar between cohorts, showing happy and neutral music to decrease the most in skin temperature. However, for smokers, chill-inducing music showed the smallest decrease in skin temperature, while for nonsmokers, sad music showed the smallest decrease in skin temperature.

2.15.2. Effects of music on physiological arousal in smokers

A multivariate test showed a significant effect of music on physiological arousal in smokers, $F(12, 35) = 3.47$, $p = .002$, $\eta 2 = .54$. Univariate tests revealed HR, $F(2.18, 100.44) = 6.46$, $p = .002$, $\eta 2 = .12$, SCL, $F(2.57, 117.99) = 10.66$, $p < .000$, $\eta 2 = .19$, and skin temperature, $F(3, 138) = 2.72$, $p = .047$, $\eta 2 = .06$ to significantly differ between music conditions in smokers. However,

respiration rate, $F(3, 138) = .28$, $p = .839$, $\eta 2 = .01$ did not significantly differ between music conditions.

Pairwise comparisons showed that HR was significantly higher during chill-inducing music compared to neutral music ($p = .003$). SCL was significantly higher during chill-inducing music compared to all other music types, include happy ($p = .010$), sad ($p = .001$), and neutral music ($p < .000$). Although a univariate test indicated a significant difference in skin temperature between music conditions pairwise comparisons show no significant differences. That is, there were no significant differences in respiration rate or skin temperature between music conditions. However, trends can be seen in these two physiological responses. For example, happy and neutral music were higher in respiration rate compared to sad and chill-inducing music. Chill-inducing music showed the lowest respiration rate across all music conditions. Happy and neutral music were also lower in skin temperature compared to sad and chill-inducing music. Chill-inducing music showed the highest skin temperature across the music types. Smokers' physiological responses to each music condition are shown in Figure 2.4 and Figure 2.5.

2.15.3. Effects of music on physiological arousal in nonsmokers

A multivariate test showed a significant effect of music on physiological arousal in nonsmokers, $F(12, 40) = 4.90$, $p < .000$, $\eta 2 = .60$. Univariate tests revealed HR, $F(1.91, 97.26) = 14.13$, $p < .000$, $\eta 2 = .22$ and SCL, $F(2.34, 119.18) = 10.46$, $p < .000$, $\eta 2 = .17$, to significantly differ between music conditions in smokers. However, respiration rate, $F(2.63, 134.02) = 2.55$, $p = .066$, $\eta 2 = .05$, and skin temperature, $F(3, 153) = 7.61$, $p = .518$, $\eta 2 = .02$ were not significantly different between music conditions.

Pairwise comparisons showed that HR was significantly higher during chill-inducing music compared to all other music types, including happy music ($p = .025$), sad music ($p < .000$), and neutral music ($p = .007$). Also, happy music was significantly higher in HR compared to sad music, ($p < .000$). SCL was significantly higher during chill-inducing music compared to sad and neutral music, ($p < .000$). Although there were no significant differences in respiration rate or skin temperature some trends can be seen. For example, respiration rate was decreased for all music conditions except happy music. Furthermore,

sad music decreased respiration rate the most. Skin temperature was decreased for all music conditions, with happy music decreasing skin temperature the most and sad music decreasing it the least. Nonsmokers' physiological responses to each music condition can be seen in Figure 2.4 and Figure 2.5.

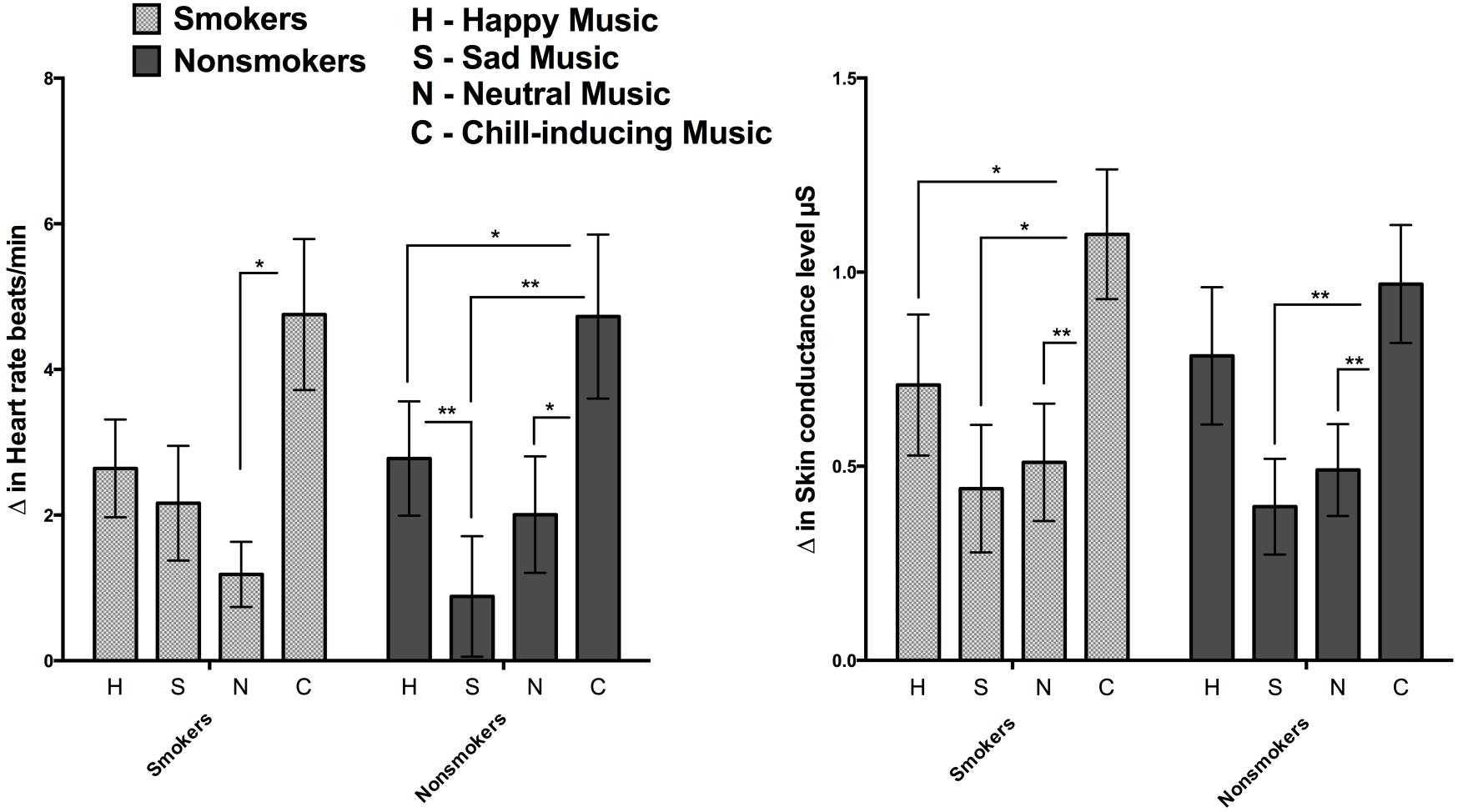

Figure 2.5. The mean and standard errors for smokers' and nonsmokers' heart rate and skin conductance level for each music condition.

* $p < .05$, ** $p < .001$.

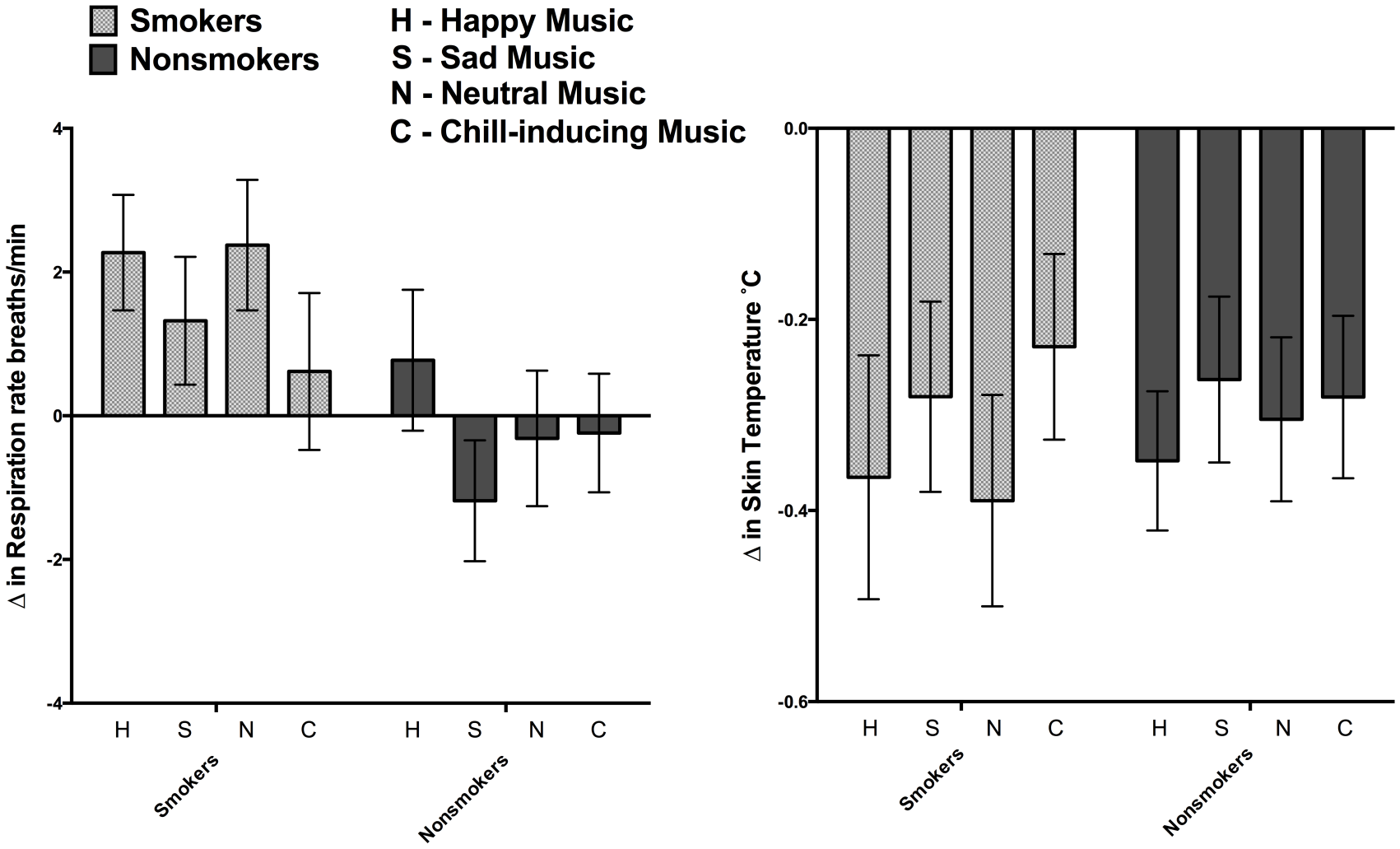

Figure 2.6. The mean and standard errors for smokers' and nonsmokers' respiration rate and skin temperature for each music condition.

2.16. Effects of nicotine and music together on physiological arousal

The following section reports the interaction effect of nicotine and music on physiological responses, first between smokers and nonsmokers (section 2.16.1), then on smokers (2.16.2), and finally on nonsmokers (2.16.3).

2.16.1. Effects of nicotine and music together on physiological arousal between smokers and nonsmokers

A multivariate test revealed a nonsignificant interaction effect between nicotine, music, and smoking status $F(24, 172) = .71$, $p = .837$, $\eta 2 = .09$, showing there to be no difference between smokers' and nonsmokers' physiological responses to the interaction effect of nicotine and music. Although nonsignificant, Figure 2.6 through Figure 2.9 show some similarities and differences between smokers' and nonsmokers' physiological responses to nicotine during each music type. For example, in general, both cohorts showed HR to increase in response to nicotine during all music types, with a pronounced increased at the low dose of 2 mg. While both cohorts also showed a decrease in SCL in response to nicotine, this decrease was systematic for smokers during all music types, but was systematic for nonsmokers only during sad and neutral music. Respiration rate showed the most variation in responses between smokers and nonsmokers. In general, for smokers, respiration rate decreased as nicotine dose increased (except for increases seen during happy music at 2 mg and neutral music at 4 mg). For nonsmokers, nicotine decreased respiration rate and mainly at the 2 mg dose. At the 4 mg dose happy and sad music showed a slight increase in respiration compared to the placebo condition and additionally, sad music showed a systematic increase in respiration at nicotine dose increased. Skin temperature showed the clearest difference in responses between smokers and nonsmokers. For smokers, as nicotine dose increase skin temperature decreased for all music types. However, for nonsmokers, as nicotine dose increased skin temperature increase for all music types.

2.16.2. Effects of nicotine and music together on physiological arousal in smokers

In regards to smokers, nicotine had broadly similar effects for each music type across the various physiological measures, as indicated by a

nonsignificant multivariate interaction ($F(24, 70) = .74$, $p = .789$, $\eta 2 = .20$). Although there was no significant interaction effect of nicotine and music on the physiological responses of smokers, some trends can be seen for HR (Figure 2.6), SCL (Figure 2.7), respiration rate (Figure 2.8), and skin temperature (Figure 2.9). For example, for all music types, HR was lowest in the placebo condition, while in general, it is highest in the 2 mg nicotine condition. SCL systematically decreases across the music types as nicotine levels increased. Less consistent trends were found in respiration rate, however respiration tended to be highest in the placebo condition and lowest in the 4 mg nicotine condition. However, for neutral music the 4 mg nicotine condition showed a marked increase in respiration, while for happy music the 2 mg nicotine condition was highest. In general, skin temperature shows a decreasing trend as nicotine levels increased.

2.16.3. Effects of nicotine and music together on physiological arousal in nonsmokers

In regards to nonsmokers, nicotine also had broadly similar effects for each music type across the various physiological measures, as indicated by a nonsignificant multivariate interaction ($F(24, 80) = .76$, $p = .779$, $\eta 2 = .19$. Although there was no significant interaction effect of nicotine and music on the physiological response of nonsmokers, some trends can be seen for HR (Figure 2.6), SCL (Figure 2.7), respiration rate (Figure 2.8), and skin temperature (Figure 2.9). For example, similar to smokers, HR is lowest in the placebo condition and highest in the 2 mg nicotine condition. Also similar to smokers, SCL tended to decrease across the music types as nicotine levels increased. Trends in respiration rate show this measurement to be lowest in the 2 mg nicotine condition across all music types except for sad music, which shows a systematic increase in respiration as nicotine dose increased. Interestingly, in opposition to smokers, skin temperature trended to increase across the music types as nicotine dose increased.

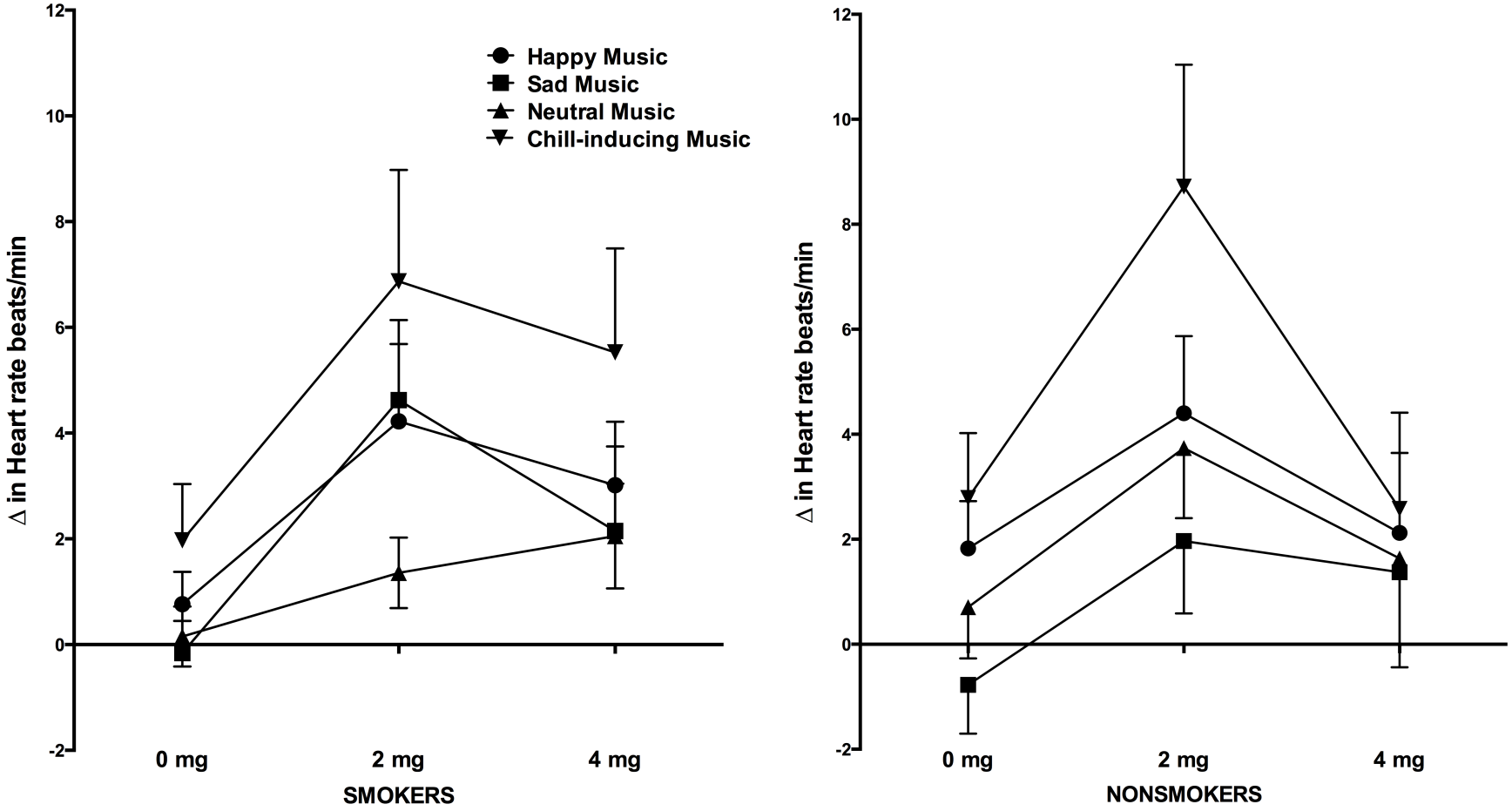

Figure 2.7. The mean and standard errors for smokers' heart rate responses to each nicotine condition for each music type. All comparisons are nonsignificant.

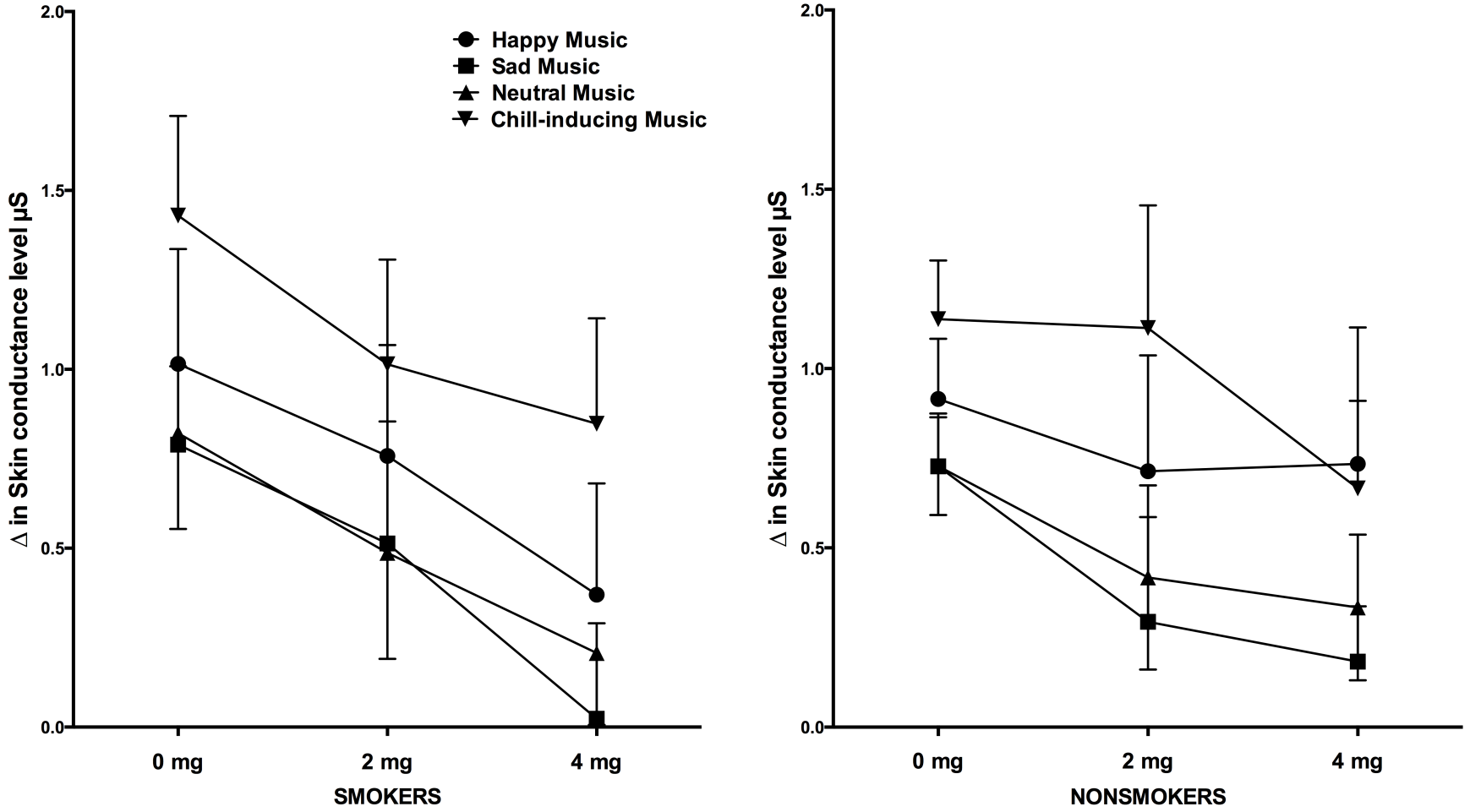

Figure 2.8. The mean and standard errors for smokers' and nonsmokers' skin conductance level responses to each nicotine condition for each music type. All comparisons are nonsignificant.

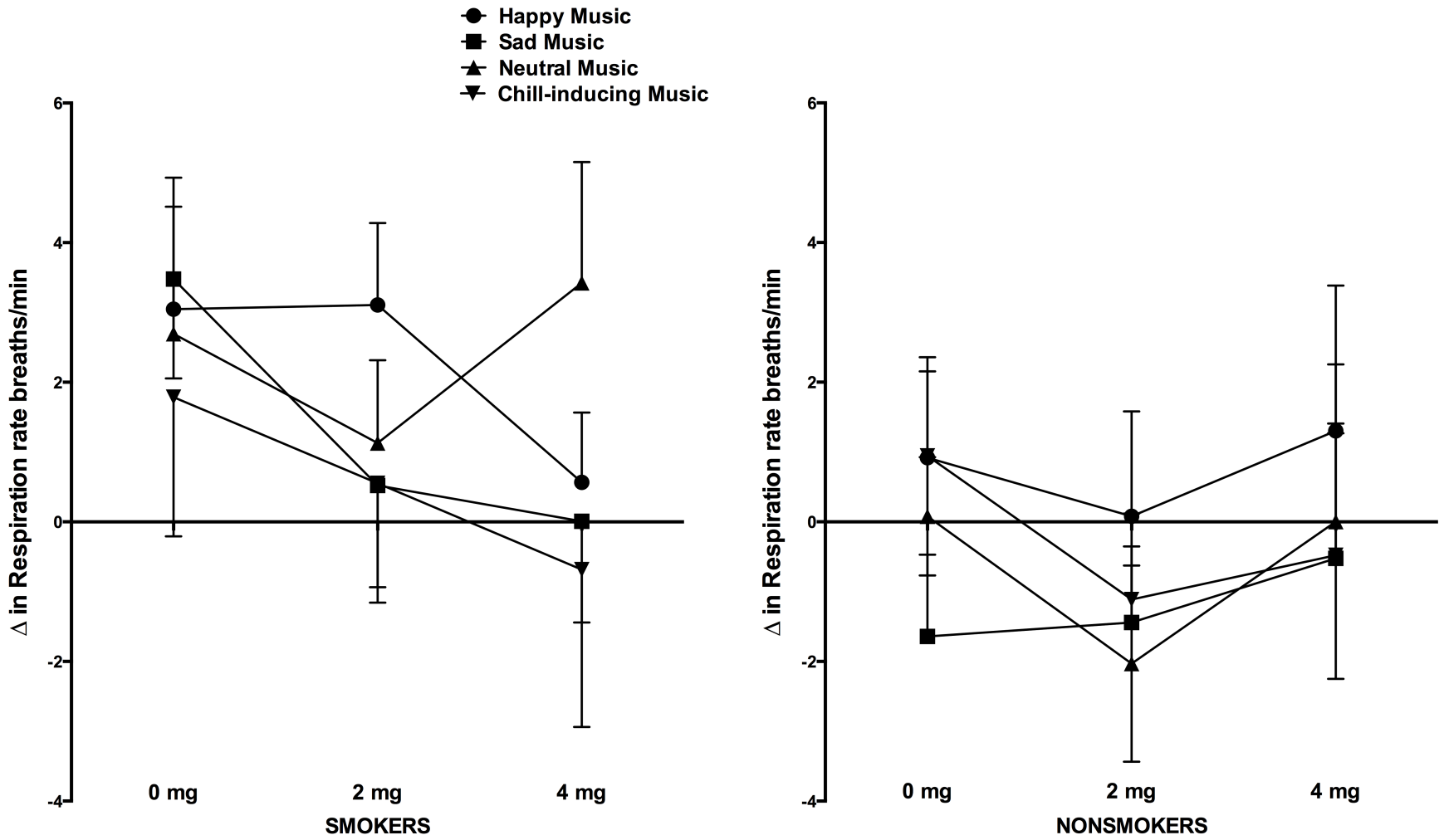

Figure 2.9. The mean and standard errors for smokers' and nonsmokers' respiration rate responses to each nicotine condition for each music type. All comparisons are nonsignificant.

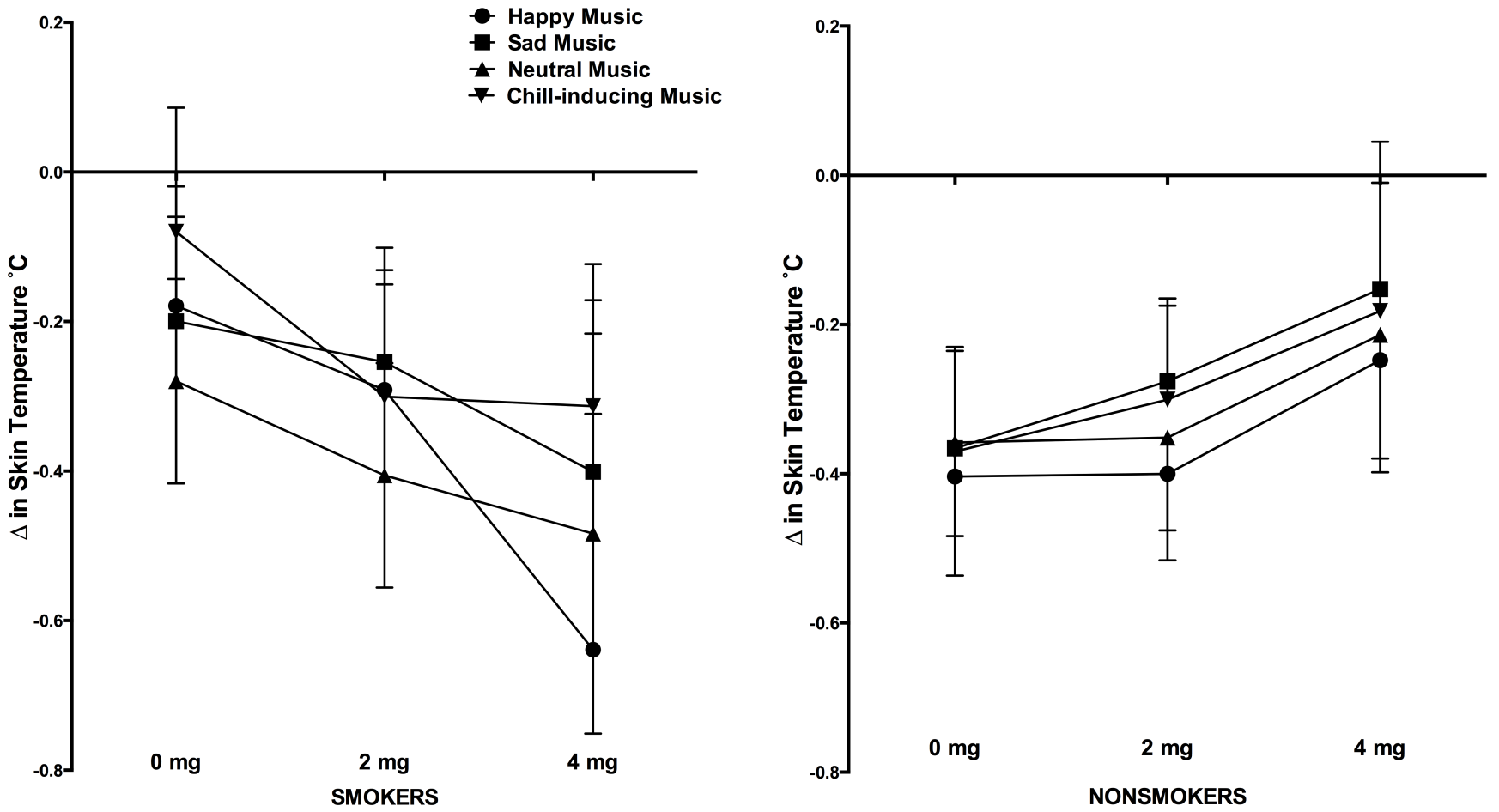

Figure 2.10. The mean and standard errors for smokers' skin temperature responses to each nicotine condition for each music type. All comparisons are nonsignificant.

2.17. Effects of nicotine on self-reported

The analysis involving self-reported responses is also divided into 3 sub-sections: a main effect of nicotine (section 2.17), a main effect of music (section 2.18), and an interaction effect of nicotine and music (section 2.19). After computing change scores some variables were skewed, therefore violating the assumption of normality. Therefore, in ratings of arousal I removed one outlier from chill-inducing music. From ratings of pleasure I removed one outlier each from happy and chill-inducing music. From ratings of happiness I removed one outlier from chill-inducing music. From ratings of sadness I removed one outlier each from sad and chill-inducing music. All subsequent analyses involving these variables were conducted with these outliers removed.

2.17.1. Effects of nicotine on self-reports between smokers and nonsmokers

A mulitivariate test indicated a nonsignificant difference between smokers' and nonsmokers' self-reported responses to nicotine, $F(4, 112) = 1.12$, $p = .353$, $\eta 2 = .04$. That is, in response to nicotine there was no significant difference between smokers' and nonsmokers ratings. Although nonsignificant, cohort comparisons can be seen in Figure 2.11 and Figure 2.12. They show ratings of arousal to decrease in response to nicotine for both smokers and nonsmokers. Contrastingly, as nicotine dose increased ratings of pleasure systematically increase for smokers, but systematically decrease for nonsmokers. Ratings of happiness were also different between smokers and nonsmokers. While both smokers and nonsmokers showed an increase in happiness in the placebo condition, smokers showed a decrease in happiness at the 2 mg dose, but a small increase at the 4 mg. Contrastingly, nonsmokers showed an increase in happiness at the 2 mg dose, but a decrease at the 4 mg dose. Ratings of sadness were also different between cohorts. As nicotine dose increased ratings of sadness systematically increased for smokers. However, for nonsmokers, 2 mg of nicotine increased ratings of sadness compared to placebo, while 4 mg decreased ratings of sadness compared to placebo.

2.17.2. Effects of nicotine on self-reports in smokers

A multivariate test indicated a nonsignificant effect of nicotine on self-reports in smokers, $F(8, 108) = .89$, $p = .532$, $\eta 2 = .06$. However, trends can

be seen for each rating in smokers, as shown in Figure 2.11 and Figure 2.12. For example, as nicotine dose increased, 1) arousal ratings systematically decreased, 2) pleasure and sad ratings systematically increased, and 3) happiness decreased, especially at the 2 mg nicotine dose.

2.17.3. Effects of nicotine on self-reports in nonsmokers

A multivariate test shows a nonsignificant effect of nicotine on self-reports in nonsmokers, $F(8, 110) = .29$, $p = .968$, $\eta 2 = .02$. However, trends for each rating in nonsmokers can also be seen in Figure 2.11 and Figure 2.12. As nicotine dose increased, 1) arousal ratings decreased, but there were negligible differences between the 2 and 4 mg conditions, 2) pleasure ratings systematically decreased, 3) happiness and sad ratings increased at the 2 mg dose, but decreased at the 4 mg dose.

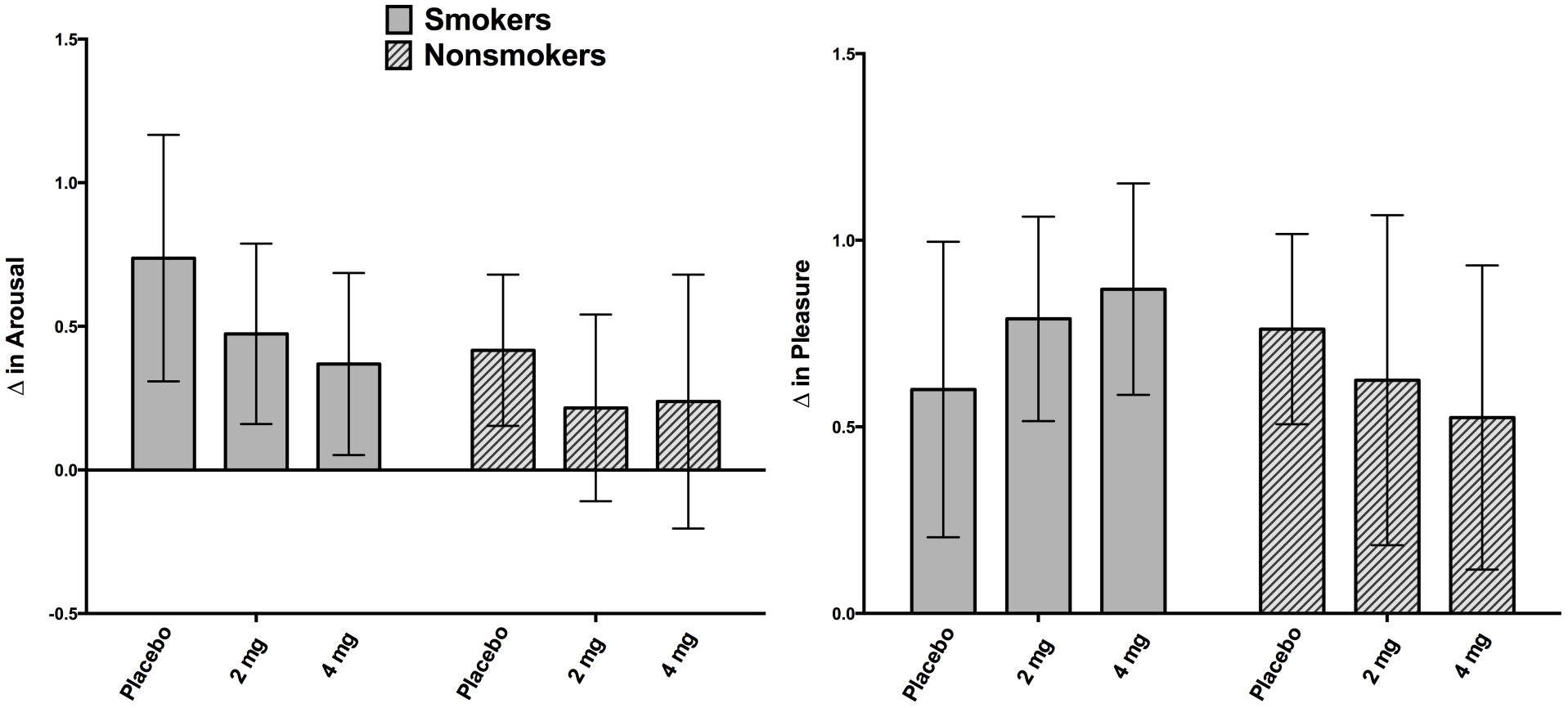

Figure 2.11. The mean and standard errors for smokers' and nonsmokers' ratings of arousal and pleasure to each nicotine condition.

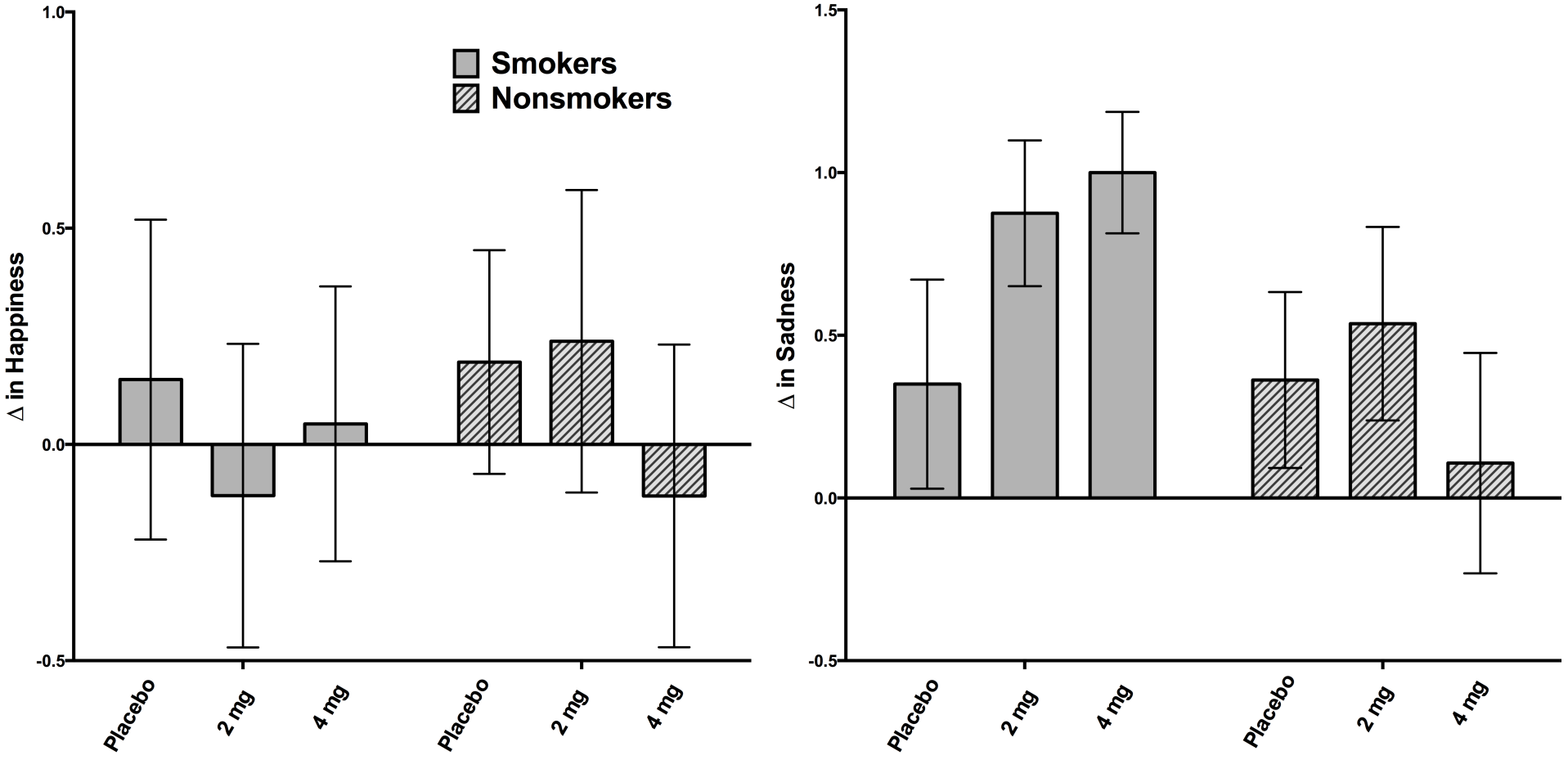

Figure 2.12. The mean and standard errors for smokers' and nonsmokers' ratings of happiness and sadness to each nicotine condition.

2.18. Effects of music on self-reports

The following section reports the main effect of music on self-reported ratings of arousal, pleasure, happiness, and sadness, first between smokers and nonsmokers (section 2.18.1), then on smokers (2.18.2), and lastly on nonsmokers (2.18.3).

2.18.1. Effects of music on self-reports between smokers and nonsmokers

A multivariate test showed a significant difference between smokers' and nonsmokers' self-reported responses to music, $F(12, 104) = 2.05$, $p = .027$, $\eta 2 = .19$. Univariate tests show arousal to significantly differ between smokers and nonsmokers, $F(3, 345) = 4.11$, $p = .007$, $\eta 2 = .04$. However, all other self-reported responses showed nonsignificant differences between smokers and nonsmokers, including pleasure, $F(3, 345) = 1.68$, $p = .172$, $\eta 2 = .01$, happiness, $F(3, 345) = 1.25$, $p = .290$, $\eta 2 = .01$, or sadness, $F(2.65, 293.98) = .12$, $p = .927$, $\eta 2 = .00$.

A follow up one-way ANOVA was then conducted to determine during which type of music self-reported arousal differed between smokers and nonsmokers. Results showed that during chill-inducing music ratings of arousal were significantly higher for smokers ($M = 1.88$, $SD = 1.82$) than nonsmokers ($M = 1.00$, $SD = 1.91$), $F(1, 123) = 6.92$, $p = .010$, $\eta 2 = .05$.

In general, smokers' and nonsmokers' self-reported responses during music followed similar trends. For example, for ratings of arousal, pleasure, and happiness happy and chill-inducing music increase in ratings, while sad and neutral music decreased in ratings (except for ratings of pleasure during sad music in smokers, which slightly increased). Ratings of sadness were also similar between cohorts. Ratings of sadness decreased during happy music, increased during all other music types, and were especially increased during sad music. Comparisons between smokers and nonsmokers can be seen in Figure 2.12 and Figure 2.13.

2.18.2. Effects of music on self-reports in smokers

A multivariate test indicated a significant effect of music on self-reports in smokers, $F(12, 46) = 25.17$, $p < .001$, $\eta 2 = .87$. Univariate tests showed all self-reported ratings to significantly differ between music conditions, including arousal, $F(3, 171) = 46.82$, $p < .001$, $\eta 2 = .45$, pleasure, $F(3, 171) = 26.25$, p

< .001, $\eta 2$ = .32, happiness, $F(3, 171) = 41.38$, $p < .001$, $\eta 2$ = .36, and sadness, $F(2.57, 146.20) = 31.63$, $p < .001$, $\eta 2$ = .36.

Pairwise comparisons showed that arousal was rated significantly higher during chill-inducing music compared to all other music types ($p < .001$). Arousal was also rated significantly higher during happy music compared to during sad and neutral music ($p < .001$). Pleasure was rated significantly higher during chill-inducing music compared to during happy (p = .004), sad and neutral music ($p < .001$) music. Also, pleasure was rated significantly higher during happy music compared to during sad (p = .014) and neutral music (p < .001). Happiness was rated significantly higher during chill-inducing and happy music compared to sad and neutral music ($p < .001$). Sadness was rated significantly higher during sad music compared to all other music conditions ($p < .001$). Sadness was also rated significantly lower during happy music compared to all other music conditions ($p < .001$). Smokers' self-reported responses to each music condition along with these pairwise comparisons are shown in Figure 2.13 and Figure 2.14.

2.18.3. Effects of music on self-reports in nonsmokers

A multivariate test indicated a significant effect of music on self-reports in nonsmokers, $F(12, 47) = 23.68$, $p < .001$, $\eta 2$ = .86. Univariate tests showed all self-reported ratings to significantly differ between music conditions, including arousal, $F(2.57, 149.25) = 29.76$, $p < .001$, $\eta 2$ = .34, pleasure, $F(3, 174) = 41.46$, $p < .001$, $\eta 2$ = .42, happiness, $F(3, 174) = 35.88$, $p < .001$, $\eta 2$ = .38, and sadness, $F(2.45, 142.14) = 37.88$, $p < .001$, $\eta 2$ = .40.

Pairwise comparisons showed ratings of arousal to be significantly higher for chill-inducing and happy music compared to sad and neutral music ($p < .001$). Similarly, ratings of pleasures were significantly higher for chill-inducing and happy music compared to sad and neutral music ($p < .001$). Again, ratings of happiness were significantly higher for chill-inducing and happy music compared to sad and neutral music ($p < .001$). Ratings of sadness were significantly higher for sad music compared to all other types of music ($p < .001$). Furthermore, happy music was rated significantly lower in sadness compared to all other types of music ($p < .001$). Nonsmokers' self-reported

responses to each music condition along with these pairwise comparisons are shown in Figure 2.13 and Figure 2.14.

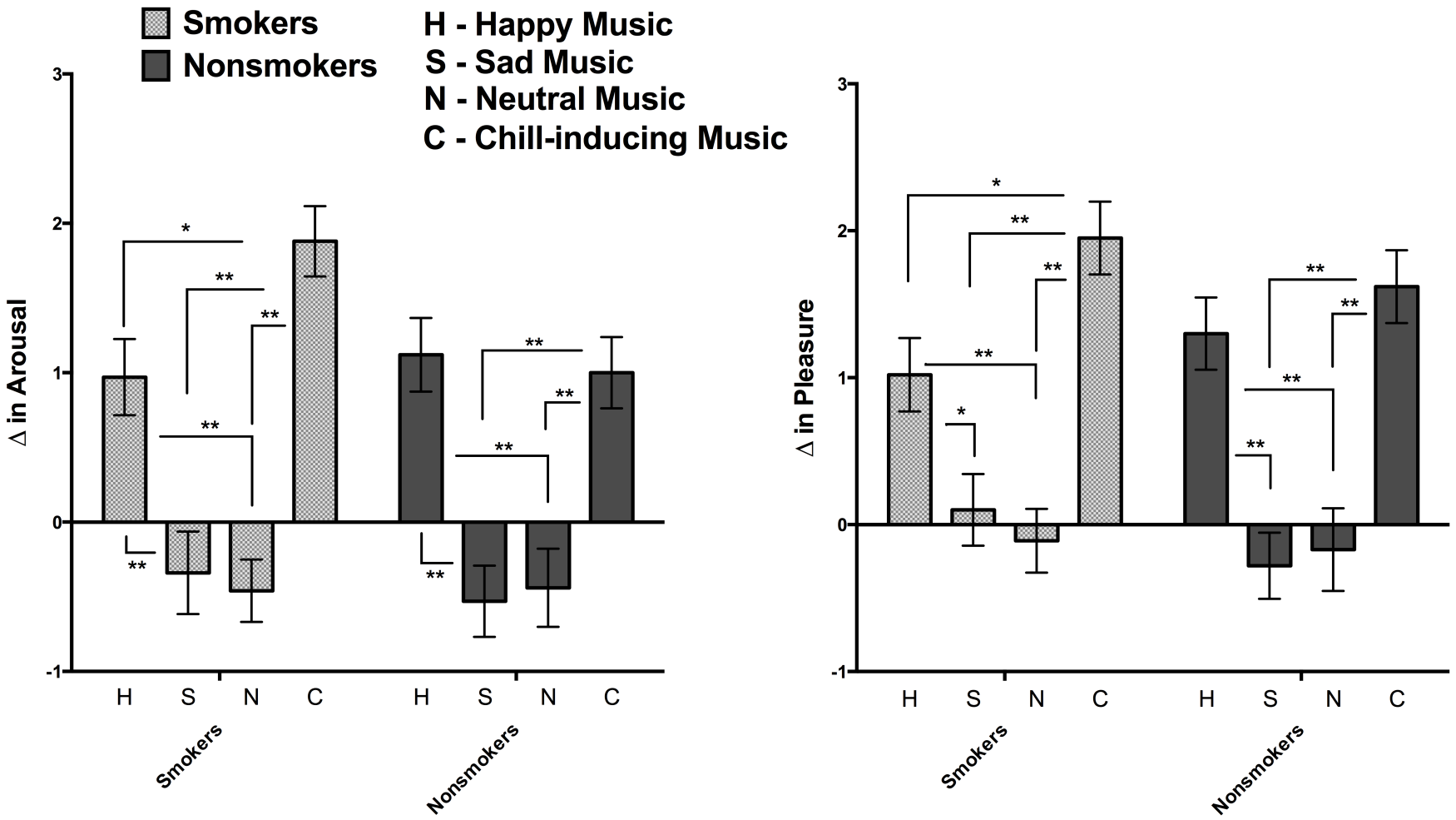

Figure 2.13. The mean and standard errors for smokers' and nonsmokers' ratings of arousal and pleasure for each music condition. * $p < .05$, ** $p < .001$.

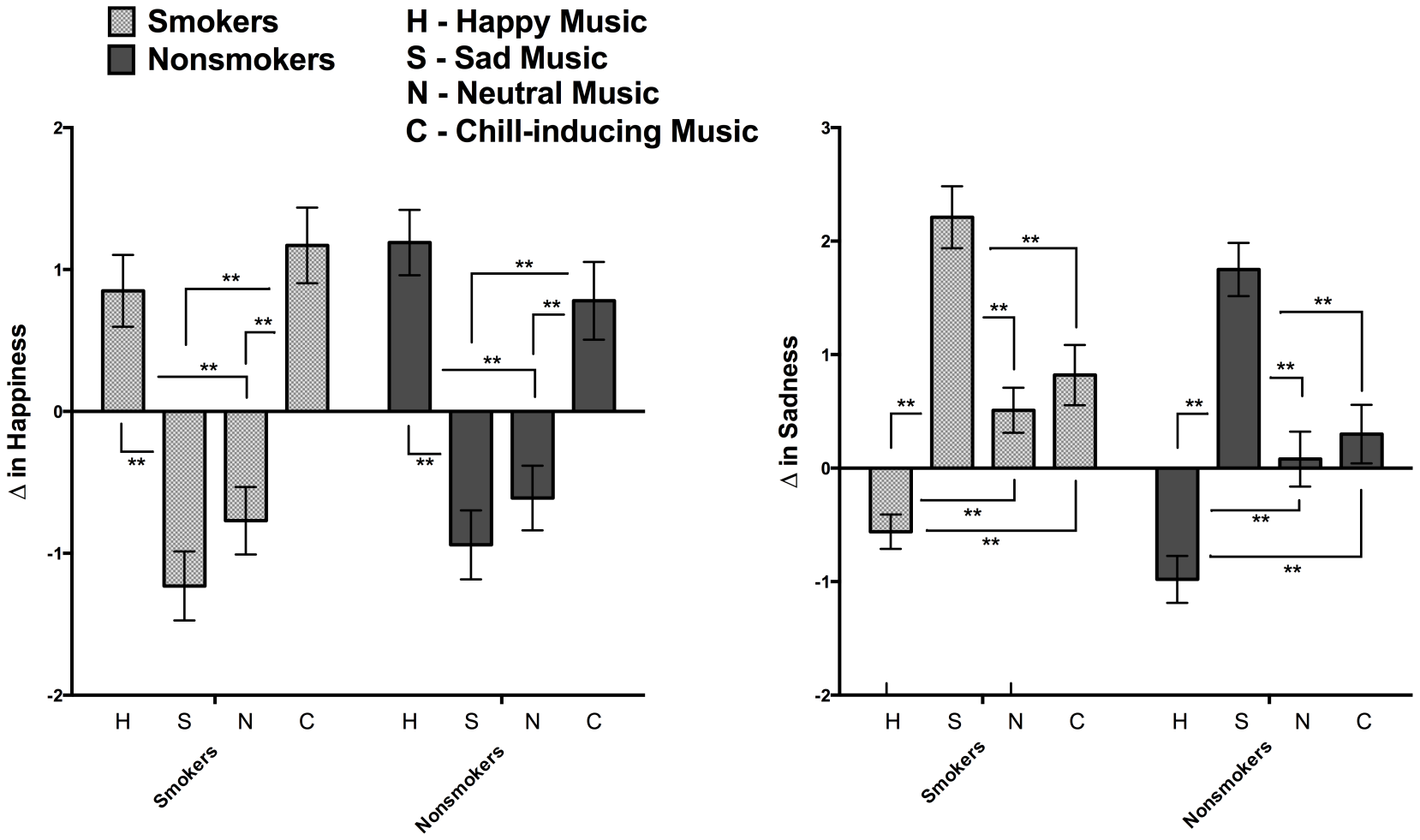

Figure 2.14. The mean and standard errors for smokers' and nonsmokers' ratings of happiness and sadness for each music condition. * p < .05, ** p < .001.

2.19. Effects of nicotine and music together on self-reports

The following section reports the interaction effect of nicotine and music on self-reported arousal, pleasure, and emotion, first between smokers and nonsmokers (2.19.1), then on smokers (2.19.2), and lastly on nonsmokers (2.19.3).

2.19.1. Effects of nicotine and music together on self-reports between smokers and nonsmokers

A multivariate tests indicated a nonsignificant interaction effect between nicotine, music, and smoking status, $F(24, 208) = .79$, $p = .762$, $\eta 2 = .08$. This indicated no difference between smokers' and nonsmokers' self-reports in response to the interaction of nicotine and music. Although nonsignificant, comparisons between cohorts can be seen in Figure 2.15 through 2.18. For ratings of arousal both cohorts showed chill-inducing music to increase in the placebo condition and to systematically increase as nicotine dose increased. However, for smokers, happy music showed a systematic decrease in arousal ratings as nicotine dose increased, while for nonsmokers happy music showing an increase in arousal ratings at the 2 mg dose, but a decrease at the 4 mg dose. In general, sad and neutral music for both cohorts show a decrease in arousal ratings during the nicotine conditions. For ratings of pleasure both cohorts showed happy and chill-inducing music to increase in the placebo condition. However, in general, ratings of pleasure were dissimilar between smokers and nonsmokers. For example, for smokers as nicotine dose increased so did ratings of pleasure during chill-inducing music. However, for nonsmokers, chill-inducing music showed negligible changes across the placebo/nicotine conditions. For smokers, sad and neutral music showed small, but systematic increases in ratings of pleasure, while in nonsmokers, sad and neutral music showed an overall decrease in pleasure. For ratings of happiness both cohorts showed that in the placebo condition happy and chill-inducing music increased and sad and neutral music decreased. However, the two cohorts showed different responses to nicotine during happy and chill-inducing music. Smokers showed an overall decrease in happiness during happy and a systematic increase in happiness during chill-inducing music. Nonsmokers showed an increase in happiness for these two music types at the 2 mg dose and a

decrease at the 4 mg dose. In general, both cohorts showed decreases in happiness as a result of nicotine during sad and neutral music. In general, for ratings of sadness both cohorts show sad music to increase in the placebo condition and to further increase in the nicotine conditions. However, for the other music conditions smokers and nonsmokers show dissimilar responses to nicotine. Smokers showed an increase in sadness in response to nicotine during happy and neutral music, while nonsmokers show a decrease in sadness during neutral music, but negligible changes during happy music. Lastly, both cohorts showed chill-inducing music to increase in the placebo condition, further increase at the 2 mg dose, and then decrease below placebo at the 4 mg dose.

2.19.2. Effects of nicotine and music together on self-reports in smokers

In general, nicotine has similar effects for each music condition across the four self-reported responses in smokers, as indicated by a nonsignificant multivariate interaction, $F(24, 92) = 1.37$, $p = .143$, $\eta 2 = .26$. Although there were no significant interaction effects of nicotine and music on smokers' self-reported ratings, trends can be seen for ratings of arousal (Figure 2.15), pleasure (Figure 2.16), happiness (Figure 2.17), and sadness (Figure 2.18). In the placebo condition, arousal ratings increased for all music types, except neutral music. As nicotine dose increased, arousal ratings tended to decrease in all music types except chill-inducing music, which systematically increased. In the placebo condition, pleasure ratings increased for all music types, except neutral music. As nicotine dose increased, ratings of pleasure systematically increased in all music types, except happy music, which decreased. In the placebo condition, happiness increased for happy and chill-inducing music, but decreased for sad and neutral music. As nicotine doses increased, happiness decreased for happy and sad music, increased for chill-inducing music, and had negligible effects for neutral music. In the placebo condition, sadness increased for sad and chill-inducing music, decreased for happy music, and did not change for neutral music. As nicotine dose increased sadness increased in all music types, except for chill-inducing music at the 4 mg dose, which showed a marked decreased in sadness.

2.19.3. Effects of nicotine and music together on self-reports in nonsmokers

Nicotine also has similar effects for each music condition across the four self-reported responses in nonsmokers, as indicated by a nonsignificant multivariate interaction, $F(24, 94) = .59$, $p = .932$, $\eta 2 = .13$. Although there were no significant interaction effects of nicotine and music on nonsmokers' self-reported ratings, trends can be seen for ratings of arousal (Figure 2.15), pleasure (Figure 2.16), happiness (Figure 2.17), and sadness (Figure 2.18). In the placebo condition, ratings of arousal increased for happy and chill-inducing music and decreased for neutral and sad music. As nicotine dose increased ratings of arousal systematically increased for chill-inducing music and showed negligible effects for sad music. For happy and neutral music arousal ratings were more varied. For the low dose of nicotine (2 mg) ratings of arousal increased for happy music, but decreased for neutral music. At the high dose of nicotine (4 mg) ratings of arousal decreased for happy music beyond placebo and increased for neutral music, but not above placebo levels. In the placebo condition, pleasure ratings increased for all music types except sad music, which decreased. As nicotine dose increased ratings of pleasure systematically decreased for neutral music and showed negligible effects for chill-inducing music. At the low dose of nicotine (2 mg) happy music negligibly increased, while sad music decreased. At the high dose of nicotine (4 mg), happy music decreased below placebo, while sad music increased slightly above placebo. In the placebo condition, happiness increased for happy and chill-inducing music and decreased for sad and neutral music. At the low dose of nicotine (2 mg), happy and chill-inducing music increased in happiness, while at the high dose (4 mg) they decreased. For both doses of nicotine, sad and neutral music showed a negligible decrease in happiness. In the placebo condition, sadness increased for all music types, except happy music, which decreased. In general, as nicotine dose increased chill-inducing and neutral music decreased in sadness, sad music increased in sadness, and happy music showed negligible changes.

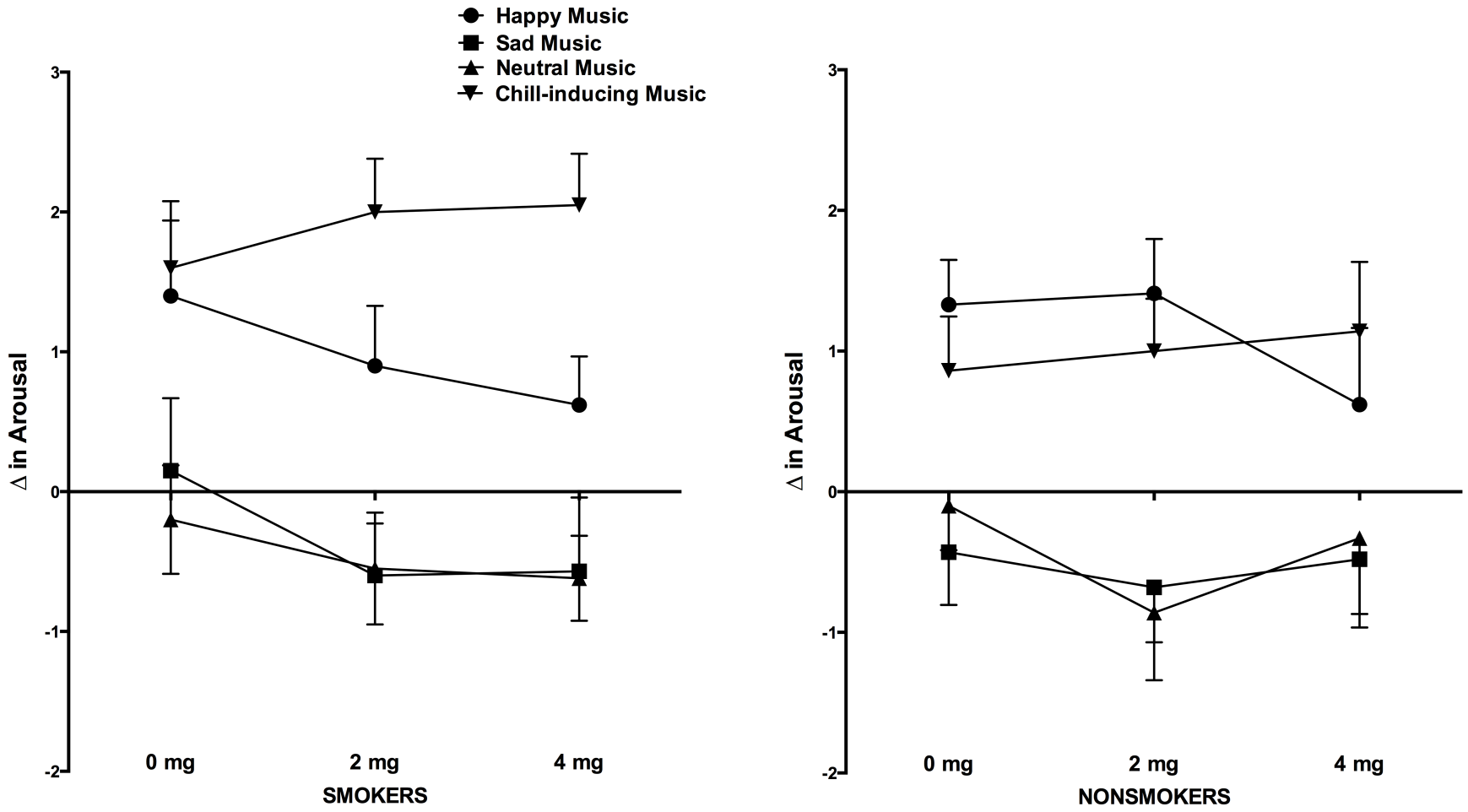

Figure 2.15. The mean and standard errors for smokers' and nonsmokers' ratings of arousal to each nicotine condition for each music type. All comparisons are nonsignificant.

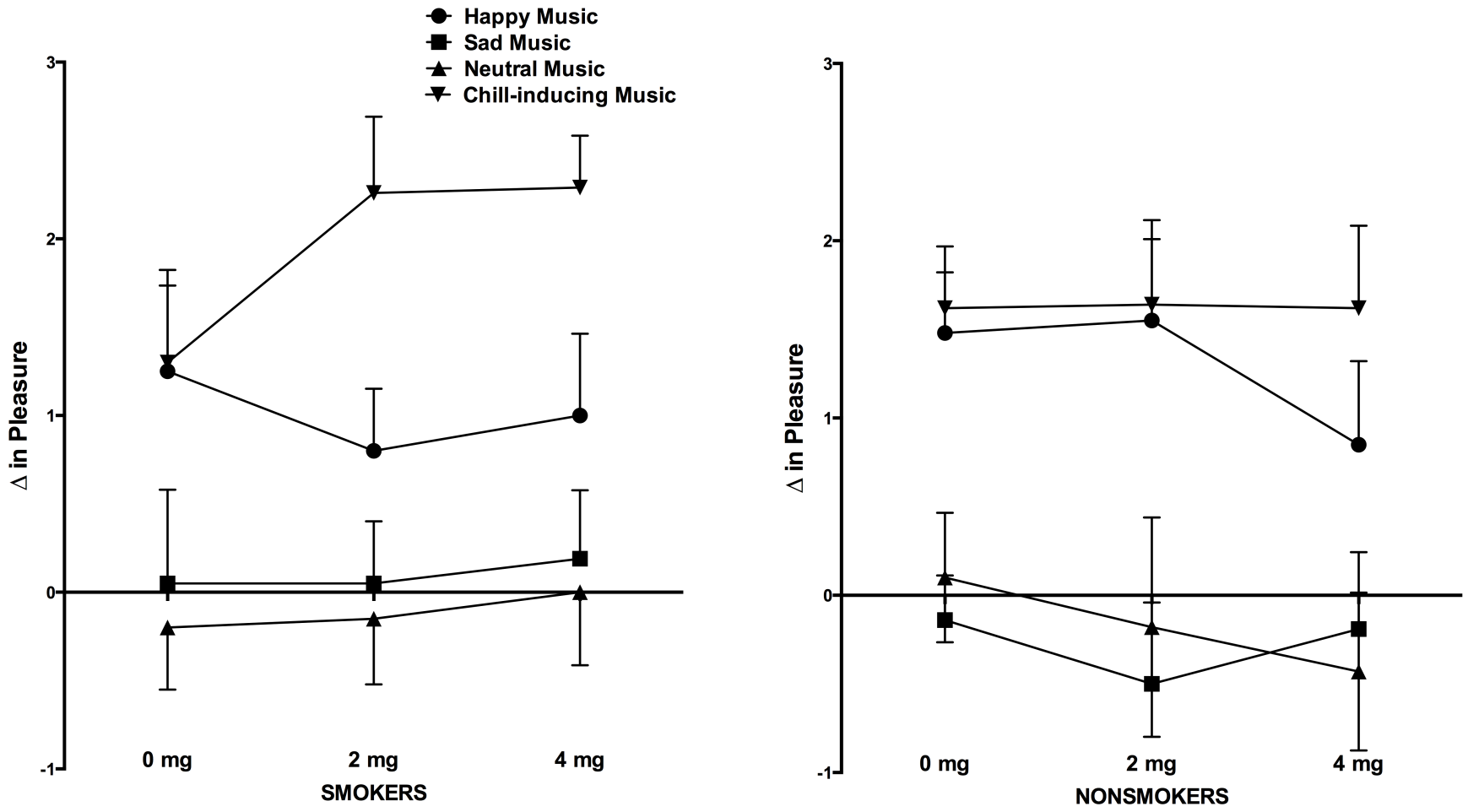

Figure 2.16. The mean and standard errors for smokers' ratings of pleasure to each nicotine condition for each music type. All comparisons are nonsignificant.

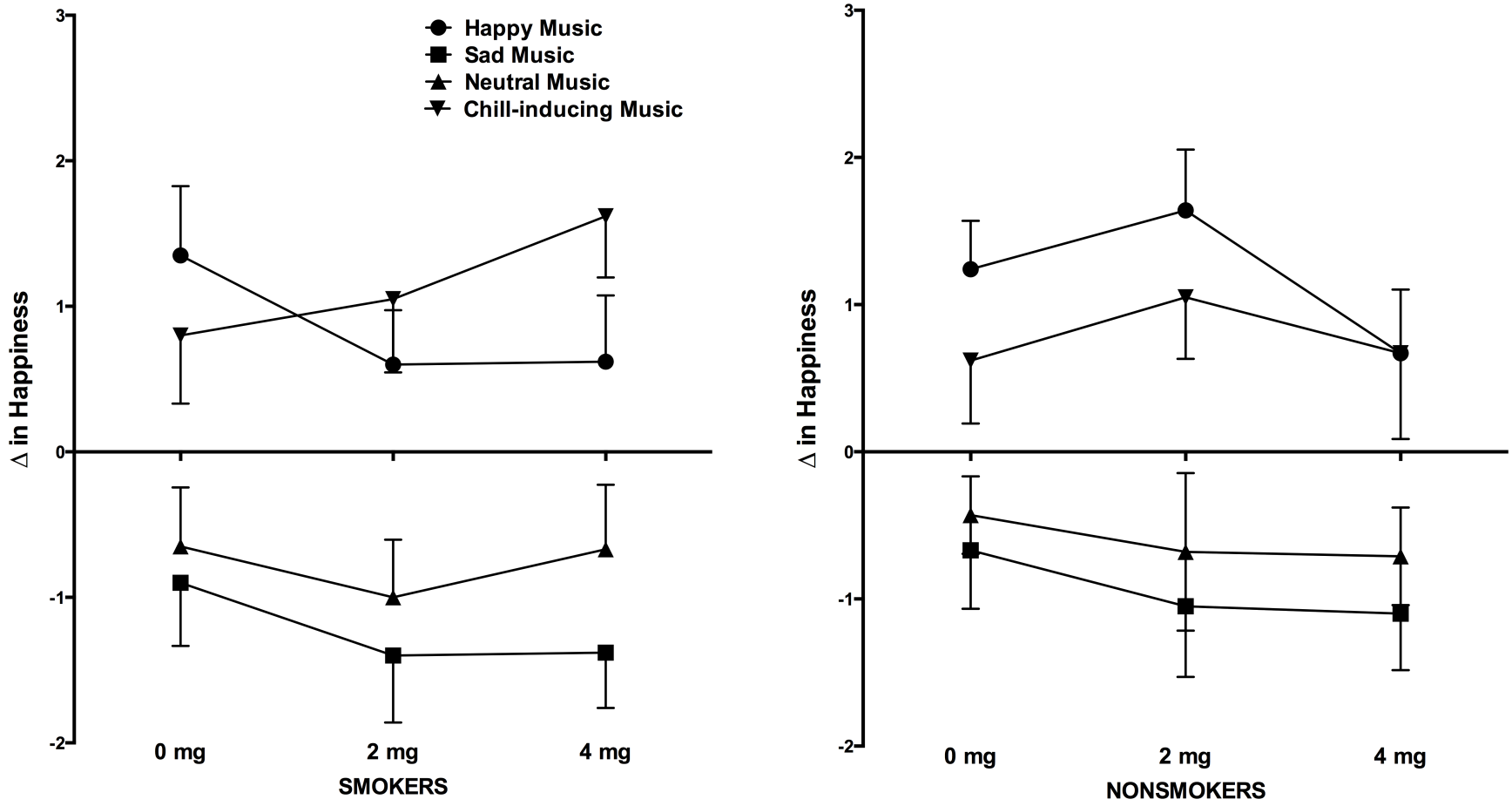

Figure 2.17. The mean and standard errors for smokers' ratings of happiness to each nicotine condition for each music type. All comparisons are nonsignificant.

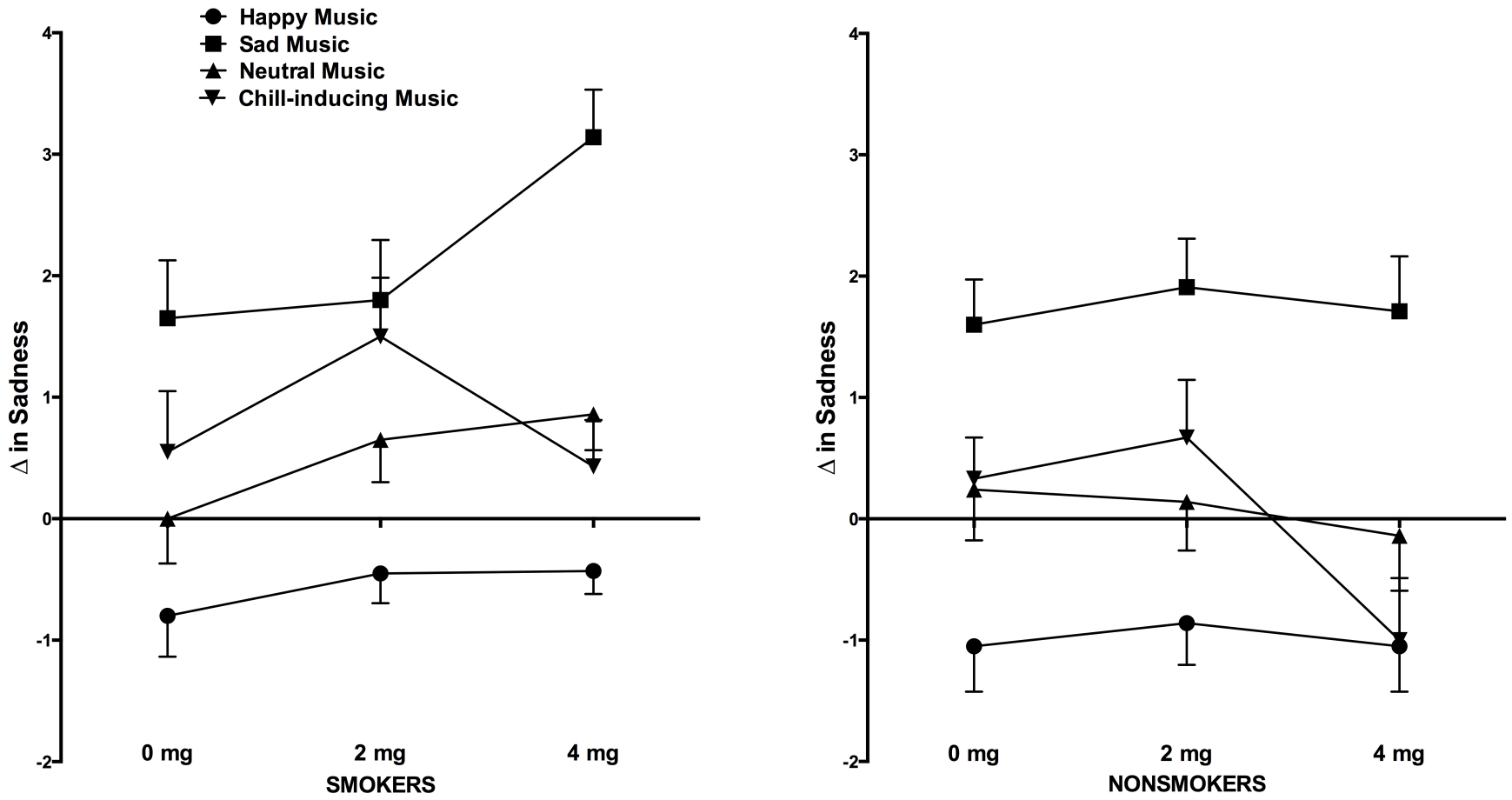

Figure 2.18. The mean and standard errors for smokers' ratings of sadness to each nicotine condition for each music type. All comparisons are nonsignificant.

2.20. Discussion

The aim of this study was to determine if an additive effect on pleasure, arousal, or both occurred in response to the co-consumption of nicotine and music listening, and if so, to identify the mechanisms that underlie this effect. This was investigated in order to help explain why nicotine and music listening often co-occur. I therefore examined the effects of nicotine on music-induced pleasure, arousal (measured through physiological and self-reported indices of arousal) and emotion in abstaining smokers and nonsmokers. I administered 0, 2, and 4 mg of nicotine to participants and asked them to listen to four musical excerpts varying in emotional quality (happy, sad, neutral, chill-inducing). I then compared their physiological, pleasure/arousal ratings, and emotional responses between the varying nicotine and music conditions.

Because both nicotine and music can independently increase participants' emotional and physiological responses, I hypothesized that their co-consumption would result in an additive effect on these responses. And I conjectured this effect to occur through nicotine's ability to increase positive affect (hedonia) and physiological arousal, and in turn increase music-induced emotions.

2.21. Effects of nicotine on physiological arousal

All results for the effects of nicotine on physiological arousal were nonsignificant. Furthermore, there was no significant difference found between smokers and nonsmokers in regards to the effects of nicotine on physiological arousal. However some trends can be seen in the data. Trends in HR show that for both cohorts both nicotine doses increased HR above placebo, which was more pronounced at the low 2 mg dose. Trends in SCL show that for both smokers and nonsmokers increases in nicotine resulted in a systematic decreased in SCL. Interestingly, trends in respiration rate were different between smokers and nonsmokers. For smokers, both doses of nicotine decreased respiration rate compared to placebo, with negligible differences between the 2 and 4 mg doses. For nonsmokers, nicotine decreased respiration, but only at the 2 mg dose, while there were negligible differences in respiration between the placebo and 4 mg conditions. Trends in skin temperature were

also different between smokers and nonsmokers. Smokers showed a decrease in skin temperature for both nicotine doses, with a more pronounced decrease at the 2 mg dose. However, nonsmokers showed a systematic increase in skin temperature as nicotine dose increased.

These results are somewhat consistent with past research, which shows nicotine to reliably increase heart rate (Parrott & Winder, 1989) and decrease skin conductance (Gilbert & Hagen, 1980). Although there is substantially less research on how respiration rate is affected by nicotine, past research has shown some doses of the drug to decrease respiration (Silvette et al., 1962; Wright, 1935). However, these studies did not focus on human subjects, but instead mammals and reptiles. On the other hand, a study with human participants has shown respiration rate to positively correlate with HR during nicotine consumption (Jones, 1987) and suggests nicotine to stimulate respiration (Gothe et al., 1985). Compared to placebo, both cohorts showed nicotine to result in a decreasing trend in respiration (except for nonsmokers at the 4 mg dose), which does not correlate with the responses found for HR. This may suggest that nicotine either depresses respiration rate irrespective of HR's responses to the drug or that respiration rate is not an accurate indicator of physiological arousal induced by nicotine.

Past research also reliably shows nicotine to decrease skin temperature (Agué, 1974), which reflects the trends found in smokers. However, nonsmokers showed nicotine to increase skin temperature relative to placebo. Only a few studies have reported an increase in skin temperature (Usuki et al., 1998) with participants' hands becoming warm and sweaty (Kanekura & Kanzaki, 1995). It may be that nonsmokers absorbed less nicotine, resulting in an increase in skin temperature relative to placebo. That is, nicotine gum is primarily absorbed across the mucous membranes (Benowitz et al., 1988). If nonsmokers, those unfamiliar with the effects of nicotine, did not enjoy the experience they have been less likely to chew the nicotine gum properly, resulting in less nicotine being absorbed. On the other hand, abstaining smokers were likely to enjoy the experience of nicotine as their body had been in a state of deprivation. This may help explain the discrepancy in skin temperature found between the two cohorts. It may also be that the time

course of the effects of nicotine on skin temperature is different between smokers and nonsmokers. For example, it could be that nonsmokers' skin temperature did not respond to the effects of nicotine within the short 2 min time course of the current study because nicotine had not yet been distributed across the body. However, further research is needed in order to test this speculation. Overall, these results somewhat support the claim that nicotine affects the physiological responses associated with arousal. However, because many of these results are trends, with no statistical significance, further research is needed in order to establish reliable physiological effects of nicotine on smokers and nonsmokers.

2.22. Effect of music on physiological arousal

Music significantly affected some physiological arousal responses in both smokers and nonsmokers. However, there was no significant difference found between smokers and nonsmokers in regards to the effects of music on physiological arousal. That is, similar patterns of physiological responses were observed between cohorts. For example, in both cohorts HR was higher for chill-inducing music compared to all other music conditions. This reached statistical significance for smokers' HR when comparing chill-inducing music to neutral music only. However, for nonsmokers, this reached statistical significance when comparing chill-inducing music to happy, sad, and neutral music. Additionally, for nonsmokers, HR was significantly higher for happy music compared to sad music. A similar trend was seen for SCL. That is, for both cohorts SCL was higher during chill-inducing music compared to all other music conditions. For smokers, this reached statistical significance when comparing chill-inducing music to happy, sad, and neutral music. For nonsmokers this reached statistical significance when comparing chill-inducing music to sad and neutral music. Although there were no statistically significant differences found for either cohort in respiration rate or in skin temperature there are trends. For example, for smokers, respiration rate was highest for happy and neutral music, while chill-inducing music was the lowest. For nonsmokers, only happy music showed an increase in respiration rate. The other music conditions showed a decrease, with sad music decreasing the most. Trends in skin temperature were similar between smokers and nonsmokers. All

music conditions showed a decrease in skin temperature, with happy and neutral music decreasing the most.

These results in general are consistent with previous literature, which has found happy and chill-inducing music to increase HR (Blood & Zatorre, 2001; Koelsch & Jäncke, 2015) and skin conductance (Hodges, 2010). Respiration rate has also been shown to increase as a result of happy and chill-inducing music, especially when compared to sad or control music (Blood & Zatorre, 2001; Krumhansl, 1997). In light of previous research, the results found here regarding how music affects respiration rate are surprising. That is, for smokers neutral music showed the highest increase in respiration, while chill-inducing music showed the lowest. Furthermore, other physiological measures show a correspondence between happy and chill-inducing music, which was not shown for respiration rate. Lastly, while other physiological measurements show similarities between smokers and nonsmokers respiration rate shows contrasting responses. For example, nonsmokers showed a decrease in respiration rate during chill-inducing music. In this case, respiration rate may be a more inconsistent measure of arousal and may be more affected by smoking status compared to other physiological responses.

Past music research has demonstrated that arousing music, including happy and sad music, decreases skin temperature (Baumgartner, Esslen, et al., 2006; Krumhansl, 1997; Lundqvist et al., 2008; McFarland, 1985). In light of this, the results of the current study suggest that all music was found to be arousing. However, the results are somewhat inconsistent with previous literature as neutral music was found to decrease skin temperature the most for smokers and the second most (after happy music) in nonsmokers. According to past research, neutral music would be expected to decrease skin temperature the least, as it is considered the least arousing compared to the other music conditions. It could be that skin temperature is more affected by musical preference than by valence and arousal, since sensation seekers have been shown to have higher skin temperatures during heavy metal music than during classical music (Nater et al., 2006). Furthermore, high sensation seekers have a higher preference for cigarette smoking than low sensation seekers (Zuckerman, Neary, & Brustman, 1970), which may help explain why skin temperature for

neutral music was lower for smokers than for nonsmokers, although this comparison was nonsignificant. Despite the inconsistencies between the current study's results and those of previous research, the results still demonstrate that music has a strong and consistent effect on physiological responses.

A summary table comparing how nicotine and music affect physiological arousal is shown in Table 2.7. In general, they show the drug to increase HR, but to decrease SCL, respiration rate, and skin temperature. On the other hand, music increased HR and SCL, while it decreased skin temperature. Music also increased respiration rate for smokers, but in general decreased it for nonsmokers. It seems then that both nicotine and music increased HR, while both decreased SCL and skin temperature. While for nonsmokers both nicotine and music decreased respiration rate, for smokers nicotine decreased respiration rate, but music increased it.

Table 2.7.

Summary table comparing the effects of nicotine and music on physiological arousal

Stimulus	Heart Rate		Skin Conductance Level		Respiration Rate		Skin Temperature	
Nicotine	↑ (S)	↑ (NS)	↓ (S)	↓ (NS)	↓ (S)	↓ (NS)	↓ (S)	↓ (NS)
Music	↑ *(S)	↑ *(NS)	↑ *(S)	↑ *(NS)	↑ *(S)	↑ *(NS)	↑ *(S)	↑ *(NS)

Note: Arrows are shown for smokers (S) (↑) and nonsmokers (NS) (↑). Direction of arrow indicates an increase or decrease in response.
** Indicates significant differences between conditions. All other conditions show nonsignificant trends.*

2.23. Effect of nicotine on self-reports

All results for the effects of nicotine on self-reported arousal were nonsignificant. Furthermore, there was no significant difference found between smokers and nonsmokers in regards to the effects of nicotine on self-reported arousal. However some trends can be seen in the data. For example, both smokers and nonsmokers showed a decrease in self-reported arousal. However, while nicotine increased pleasure for smokers, it decreased pleasure for nonsmokers. Nicotine's effect on happiness was varied. For smokers, nicotine decreased happiness, while for nonsmokers 2 mg increased happiness, but 4 mg decreased it. Nicotine's effect on sadness was also varied. While smokers showed an increase in sadness in response to nicotine, nonsmokers showed an increase at the 2 mg dose and a decrease at the 4 mg dose.

Past research has shown nicotine to increase positive affect and to play a role in smoking maintenance (Leventhal & Cleary, 1980; Tomkins, 1966). The results for smokers somewhat fit with previous research, as pleasure was shown to increase systematically as nicotine dose increased. However, in response to nicotine arousal and happiness ratings were shown to decrease and sadness ratings were shown to increase. Overall, this does not show that nicotine increased self-reported arousal in smokers and instead suggests an increase in negative affect.

It could be that for smokers nicotine induces a relaxing effect as tranquilizing and emotion-reducing effects of nicotine have been previously reported (Gilbert, 1979). That is, smokers have reported using nicotine for different reasons (Tomkins, 1966). While some smokers have reported using nicotine in order to increase arousal, for example when bored or tired, other have reported using the drug in order to relax or reduce their level of arousal, for example during highly arousing or stressful situations (Frith, 1971b; McKennell, 1970). More recent research (Kassel, Stroud, & Paronis, 2003; Spielberger, 1986) has shown that smokers report using nicotine, at least in part, for its anxiolytic and sedative properties. In this case, it may be that abstaining smokers were experiencing higher than normal levels of anxiety and restlessness due to nicotine withdrawal (West & Hajek, 2004). This in turn

resulted in higher levels of arousal when receiving placebo and lower levels of arousal (e.g. a relaxation effect) when receiving nicotine.

This relaxation effect, however, does not help explain why abstaining smokers receiving nicotine also reported a decrease in happiness and an increase in sadness compared to those receiving placebo. It may be that abstaining smokers who were administered nicotine did not receive enough of the drug for it to increase positive affect. This may be due to 1) the time course of nicotine gum compared to cigarette smoking and 2) the lower ecological validity of gum chewing, as nicotine gum does not provide the same oral sensations as smoking a cigarette. That is, the time course of nicotine delivery is much slower for nicotine gum (~30 min) compared to smoke inhalation (almost immediate) (Benowitz et al., 2009), which may have resulted in a less intense 'rush' of nicotine and therefore ales intense positive subjective experience for abstaining smokers. Furthermore, nicotine gum does not facilitate hand-to-mouth movements and stimulation as does smoking a cigarette. These oral sensations and sensorimotor behaviors associated with smoking have been found to be an important factor in the experience of smoking, as there is positive affect derived from lighting and handling cigarette, and even from watching the smoke curl upwards (Ikard et al., 1969). For these reason, abstaining smokers may have experienced less happiness and more sadness than otherwise predicted.

For nonsmokers it was apparent that as nicotine levels increased ratings of arousal and pleasure decreased, while ratings of happiness and sadness were less consistent across the doses. This strongly suggests that nonsmokers did not enjoy the experience of nicotine and that the drug negatively affected their emotions. Previous research supports these trends showing nonsmokers to experience adverse physiological and subjective effects from nicotine, such as headache, nausea, dizziness, indigestion, negative mood, anxiety, and nervousness (Foulds et al., 1997; Heishman & Henningfield, 2000). These trends further suggest that nicotine does not increase arousal and pleasure for everyone, especially not for those who hold no tolerance for the substance.

2.24. Effect of music on self-reports

Music significantly affected all self-reported responses in both smokers and nonsmokers. However, there was no significant difference found between smokers and nonsmokers in regards to the effects of music on self-reports. That is reflected in the similar patterns of self-reported responses observed between cohorts. For example, there was a trend for happy and chill-inducing music to dramatically increase ratings of arousal, pleasure, and happiness in both cohorts compared to sad and neutral music. More specifically, for both smokers and nonsmokers chill-inducing music significantly increase arousal ratings compared to sad and neutral music. Furthermore, in smokers, chill-inducing music significantly increased arousal ratings compared to happy music. Happy music was also rated significantly higher in arousal compared to sad and neutral music for both cohorts. Interesting, for both cohorts sad and neutral music decreased ratings of arousal. Pleasure ratings were similar. For both cohorts happy and chill-inducing music were rated significantly higher in pleasure compared to sad and neutral music. Additionally, in smokers, chill-inducing music was rated significantly higher in pleasure than happy music. Sad music for both cohorts and neutral music for nonsmokers decreased ratings of pleasure. Happiness ratings also mirrored those found for arousal and pleasure. For both cohorts happy and chill-inducing music significantly increased ratings of happiness compared to sad and neutral music. Sad and neutral music also decreased ratings of happiness for both cohorts. Ratings of sadness were different compared to arousal, pleasure, and happiness, but showed consistency across cohorts. Sad music was significantly more sad compared to all other music conditions and happy music was significantly less sad compared to all other music conditions. These results are consistent with previous research, showing music to strongly and reliably increase positive affect in listeners (Dubé & Le Bel, 2003; Zentner et al., 2008) and confirm that music can increase arousal and pleasure.

A summary table comparing how nicotine and music affect self-reports is shown in Table 2.8. It is clear that nicotine decreased arousal and increased sadness. Nicotine also decreased happiness in smokers, but resulted in mixed responses for nonsmokers. Nicotine further increased pleasure for smokers, but

decreased it for nonsmokers. The results for music are more straightforward. Happy and chill-inducing music increased arousal, pleasure, and happiness for both cohorts, as well as increased sadness for all music types except happy music.

Table 2.8.

Summary table comparing the effects of nicotine and music on self-reports

Stimulus	Arousal		Pleasure		Happiness		Sadness	
Nicotine	↓	↓	↑	↓	↓	↓	↑	↑
	(S)	(NS)	(S)	(NS)	(S)	†(NS)	(S)	(NS)
Music	↑	↑	↑	↑	↑	↑	↑	↑
	*(S)	*(NS)	*(S)	*(NS)	*(S)	*(NS)	*(S)	*(NS)

Note: Arrows are shown for smokers (S) (↑) and nonsmokers (NS) (↑). Direction of arrow indicates an increase or decrease in response.
** Indicates significant differences between conditions. All other conditions show nonsignificant trends.*
† Indicates that for NS, 2 mg increased happiness, 4 mg decreased happiness.

2.25. Effects of nicotine and music together on physiological arousal

How nicotine and music interact to affect each physiological response is central to the concerns of the present study. There was no significant difference in the interaction effect between smokers and nonsmokers and no significant interaction effects seen within smokers or nonsmokers. However trends can be seen in the data. For both cohorts, HR was elevated for all music types in the placebo condition except for sad music. Both cohorts showed HR to increase in response to nicotine and this increase was, in general, more pronounced at the 2 mg dose. Therefore, nicotine increased heart rate above

placebo for all music types in smokers and nonsmokers. Although nonsignificant, this is indicative of an additive effect.

For both cohorts, SCL was elevated for all music types in the placebo condition. Nicotine decreased SCL and this decrease was systematic for all music types and cohorts, except for nonsmokers during happy and chill-inducing music. As all music conditions for both cohorts showed a clear trend for SCL to decrease in the presence of nicotine, following an elevation in the placebo condition, these results are not indicative of an additive effect.

Respiration rate showed varying responses between cohorts and music conditions. For both cohorts, respiration rate was elevated for all music types in the placebo condition except for sad music in nonsmokers. For smokers, respiration rate decreased in response to nicotine, except for an increase in respiration for happy music at 2 mg and for neutral music at 4 mg. For nonsmokers, nicotine decreased respiration rate, particularly at the 2 mg dose. However, at 4 mg happy and sad music showed a small increase in respiration compared to placebo. Additionally, sad music showed a systematic increase in respiration as nicotine dose increased. As nicotine decreased respiration rate, following an increase in the placebo condition, these results are not indicative of an additive effect. Furthermore, although for nonsmokers sad music showed a decrease in respiration in the placebo condition, nicotine increased respiration for this music type. Therefore, this is also not indicative of an additive effect.

For both cohorts, skin temperature showed a reduction for all music types in the placebo condition. For smokers, nicotine further decreased skin temperature, which is indicative of an additive effect. However, for nonsmokers, nicotine increased skin temperature for all music types, which is not indicative of an additive effect.

2.26. Effects of nicotine and music together on self-reported emotions (happiness/sadness)

There was no significant difference in the interaction effect between smokers and nonsmokers and no significant interaction effects seen within smokers or nonsmokers. In the placebo condition, both cohorts showed happy and chill-inducing music to increase happiness, and sad and neutral music to decrease happiness. In smokers, as nicotine dose increased, ratings of

happiness systematically increased for chill-inducing music. However, during happy music nicotine caused a decrease in happiness, with negligible differences between the 2 and 4 mg doses. Although nonsignificant, there is a trend indicative of an additive effect of nicotine on happiness in smokers during chill-inducing music only. In nonsmokers, 2 mg of nicotine increased ratings of happiness in happy and chill-inducing music. However, 4 mg of nicotine only negligibly increased happiness above placebo during chill-inducing music and actually decreased happiness below placebo during happy music. Although nonsignificant, this is indicative of an additive effect of nicotine on happiness during happy and chill-inducing music in nonsmokers, but only at the 2 mg dose.

In smokers, 2 mg of nicotine decreased happiness below placebo during sad and neutral music. However, 4 mg of nicotine increased happiness above placebo during neutral music, but resulted in a negligible decrease in happiness during sad music. Although nonsignificant, this is indicative of an additive effect of nicotine on happiness during sad music in smokers, as happiness was decreased for sad music at placebo and nicotine further decreased happiness. In nonsmokers, nicotine at both doses slightly decreased happiness during sad and neutral music, but there were negligible differences in happiness between the nicotine conditions. Although nonsignificant, this too is indicative of an additive effect as nicotine further decreased happiness beyond placebo levels for sad and neutral music in nonsmokers.

In both cohorts, levels of sadness were increased for all music types in the placebo condition, except for happy music. In smokers, nicotine systematically increased sadness during sad, neutral, and happy music. During chill-inducing music 2 mg increased sadness above placebo, while 4 mg decreased it below placebo. Although nonsignificant, this is indicative of an additive effect of nicotine on sadness during sad and neutral music in smokers. For nonsmokers, nicotine slightly increased sadness during sad music, and this was slightly more pronounced at the 2 mg dose. Furthermore, 2 mg of nicotine increased sadness during happy and chill-inducing music. However, for happy music, 4 mg of nicotine returned ratings of sadness to placebo levels, and for chill-inducing music 4 mg of nicotine decreased sadness below placebo. For

neutral music, as nicotine dose increased, sadness ratings systematically decreased. Overall, this is indicative of an additive effect on sadness in nonsmokers during sad and chill-inducing music at the 2 mg dose.

2.27. Effects of nicotine and music together on self-reported arousal/pleasure

How nicotine and music interact to affect pleasure and arousal is also of interest to this study. However, there was no significant difference in the interaction effect between smokers and nonsmokers and no significant interaction effects seen within smokers or nonsmokers. For smokers, in the placebo condition, all music conditions increased in arousal except for neutral music, which decreased. As nicotine dose increased ratings of arousal systematically increased during chill-inducing music and systematically decreased during happy and neutral music. However, for sad music, nicotine decreased arousal, with negligible differences between the 2 and 4 mg doses. Although nonsignificant, this is indicative of an additive effect of nicotine on arousal for chill-inducing music in smokers.

For nonsmokers, in the placebo condition arousal ratings were increased during happy and chill-inducing music, and decreased for sad and neutral music. As nicotine dose increased ratings of arousal systematically increased for chill-inducing music. However, for happy music, 2 mg of nicotine slightly increased arousal above placebo, but 4 mg of nicotine decrease arousal below placebo. For sad and neutral music, nicotine decreased arousal further. While this decrease was negligible for sad music, this decrease was more noticeable for neutral music and more pronounced at the 2 mg dose. Although nonsignificant, this is indicative of an additive effect of nicotine on arousal for chill-inducing music in nonsmokers. Additionally, there is some indication of an additive effect of nicotine on arousal during neutral music, as placebo decreased arousal ratings and nicotine further decreased these ratings.

For smokers, pleasure was increased in the placebo condition for happy, sad, and chill-inducing music, while it was decreased for neutral music. As nicotine dose increased chill-inducing, sad, and neutral music systematically increased in pleasure. However, for happy music, both doses of nicotine decreased pleasure, and this was more pronounced at the 2 mg dose. Although

nonsignificant, this is indicative of an additive effect of nicotine on pleasure during sad and chill-inducing music in smokers.

For nonsmokers, pleasure was increased in the placebo condition for happy, neutral, and chill-inducing music. There were negligible changes in pleasure during chill-inducing music across the placebo and nicotine conditions. However, as nicotine dose increased ratings of pleasure decreased for neutral music. For sad music, 2 mg of nicotine decreased pleasure, but 4 mg increased it. Lastly, for happy music, 2 mg of nicotine increased pleasure, but 4 mg decreased it. This is not indicative of an additive effect of nicotine on pleasure for nonsmokers.

2.28. Summary

Although the results for interaction effects are nonsignificant there are some trends indicative of additive effects of nicotine and music on the physiological and self-reported responses. Physiological indices of arousal were clearly indicative of an additive effect of nicotine for HR in both cohorts, especially at the 2 mg dose. No other physiological responses showed trends of additive effects. Self-reports also showed some trends of additive effects, mainly during chill-inducing music and more for smokers than nonsmokers. Self-reported happiness showed trends of additive effects for smokers, but only during chill-inducing music. Happiness also showed trends indicative of additive effects for nonsmokers during happy and chill-inducing music, but only at the 2 mg dose. There were also trends indicative of additive effects for sad music in smokers, and sad and neutral music in nonsmokers, as these music conditions showed a decrease in happiness at the placebo level and a further decrease in happiness in response to nicotine. Trends indicative of additive effects in sadness were apparent for smokers during sad and neutral music and for nonsmokers during sad and chill-inducing music, but only at the 2 mg dose. In regards to arousal, there were trends indicative of an additive effect for both smokers and nonsmokers during chill-inducing music. There was also a trend indicative of an additive effect for nonsmokers during neutral music, as nicotine caused a further decrease in arousal. Lastly, there were trends indicative of an additive effect of nicotine on pleasure in smokers during sad and chill-inducing music.

2.29. Limitations and future research

The main limitation of this study was the small sample size (~20 participants per condition). A small sample size is a common problem for drug studies. It was difficult to recruit smokers who were willing to abstain from nicotine for 24 hours as well as nonsmokers who were willing to ingest the drug. Despite the difficultly in recruiting smoking participants who are willing to abstain from nicotine, it would have been beneficial to test participants who smoked more than 10 cigarettes per day for more than 10 years (as compared to 7+ cigarettes for a maximum of 10 years). This would have increased the level nicotine dependence, which would have increased nicotine craving during abstinence, and in turn could have increased physiological arousal and self-reports more when nicotine consumption was reinstated during the experiment. Future studies may wish to recruit heavy smokers who are thinking of quitting smoking in order to find participants who are both heavy smokers and willing to abstain from nicotine for 24 hours.

The surprising results for the self-reports of smokers (e.g. decrease in arousal and happiness, and an increase in sadness) need to be examined in future research in order to determine whether these results are genuine effects of nicotine on abstaining smokers or more related to methodological differences between the current study and previous literature. This will help to establish a clear explanation as to why smokers did not experience positive affect in response to nicotine. Future research may be interested in improving the ecological validity of experiments examining how nicotine affects abstaining smokers by decreasing the time course of nicotine delivery and increasing sensorimotor behaviors. This can be accomplished by using genuine cigarettes or e-cigarettes, both of which require the inhalation of smoke and hand-to-mouth movements.

Given that the precise role of arousal and pleasure in linking nicotine and music consumption is still somewhat unclear, it may be useful to use a substance other than nicotine to investigate the interaction – one that primarily affects arousal with a lesser effect on pleasure. A substance that is not as strongly associated with ill health, but that still increases physiological arousal is caffeine. Also, caffeine increases arousal, but has shown not to affect pleasure

(Herz, 1999). This makes caffeine an ideal substance for further investigations. Also, caffeine can be used to isolate the effects of physiological arousal from those of pleasure in order to examine how music-induced emotions are affected. And lastly, caffeine consumption is more widespread than nicotine (Ferré, 2008), meaning that nonsmokers may experience less adverse effects to the substance as they are likely to have been exposed to caffeine. Therefore, using caffeine may afford nonsmokers the opportunity to experience additive effects on their positive emotional responses to music, similar to smokers.

3. Chapter three: The effects of caffeine on music-induced emotion

3.1. Overview and rationale of study 2

In study one the effects of nicotine on music-induced emotion were investigated. The findings showed trends indicative of additive effects. Physiological changes in heart rate in both cohorts showed trends of additive effects. Self-reported changes in happiness for both cohorts, as well as sadness, arousal, and pleasure for smokers showed trends indicative of additive effects during some music types, mainly chill-inducing music. Although these trends were nonsignificant, they suggest that with the co-consumption of nicotine and music an additive effect on physiology and self-reports can be possible.

From these results it is not possible to determine to what extent nicotine's increase in arousal compared to pleasure influenced these additive effects. Furthermore, while nicotine increased physiological arousal via HR in both cohorts, it only increased self-reports of arousal for both smokers and nonsmokers (during chill-inducing music). However, pleasure was only increased for smokers, and actually decreased for nonsmokers. This may suggest that an increase in arousal was more influential on the additive effects seen in study one. However, in order to determine the validity of this conjecture further investigation is needed. Therefore, dissociating these effects of nicotine (e.g. increase in arousal, increase in pleasure) is necessary in order to better understand why nicotine and music are often co-consumed. Therefore, the aim of study two is to examine if an increase in only arousal (without an influence on pleasure) affects listeners' music-induced emotions. This manipulation can be accomplished using caffeine, which has been shown to induce arousal without influencing pleasure (Herz, 1999).

3.2. Caffeine: Mechanism of action

Caffeine is the single most prevalent psychoactive substance in the world (Ferré, 2008; Sawyer, Julia, & Turin, 1982), estimated to be consumed by at least 80% of the world (Heckman, Weil, Mejia, & Gonzalez, 2010). It is most commonly consumed as coffee, tea, soft drinks, and chocolate (Bonham &

Leaverton, 1979; Gokulakrishnan, Chandraraj, & Gummadi, 2005). Like nicotine, caffeine is a legal and freely available psychostimulant (Ferré, 2008).

Caffeine is characterized as a central nervous system (CNS) stimulant (Rall, 1980; Sawyer et al., 1982), which affects metabolic and cardiovascular functions. After consumption, caffeine is rapidly absorbed by the gastrointestinal tract (Blanchard & Sawers, 1983), diffused throughout the body, and penetrates through the blood-brain barrier (Axelrod & Reisenthal, 1953; McCall, Millington, & Wurtman, 1982). It reaches peak plasma concentration and exerts maximal pharmacological effects ~30-60 min post-consumption (Benowitz, 1990; Blanchard & Sawers, 1983).

Caffeine's main mechanism of action is through the antagonism of adenosine receptors. Adenosine is an endogenous hormone that exists throughout the body as a CNS inhibitor. It specifically inhibits the release of acetylcholine, norepinephrine, dopamine, GABA, and serotonin (Benowitz, 1990; Doré et al., 2011). It is also a potent vasodilator, which helps relax coronary muscles and regulate circulatory functions (Berne, 1980; Hori & Kitakaze, 1991).

Caffeine acts as a competitive inhibitor of adenosine by nonselectively binding to its receptors (Benowitz, 1990; Bünger, Haddy, & Gerlach, 1975). It thereby counteracts the inhibitory effects of adenosine and lowers the threshold for neuronal activation (Phillis, Edstrom, Kostopoulos, & Kirkpatrick, 1979). This causes a release of norepinephrine, dopamine, and serotonin (Benowitz, 1990; Fredholm, Bättig, Holmén, Nehlig, & Zvartau, 1999; Kenemans & Lorist, 1995). An increase in norepinephrine results in the increase firing of cortical neurons and the locus coeruleus, which regulate arousal and vigilance (Green & Suls, 1996; Grilly, 1994). This in turn temporarily increases the physiological responses under the control of the autonomic nervous system (ANS), including blood pressure, skin conductance, HR, and respiration rate (Cushney, 1913; Quinlan et al., 2000).

Caffeine also increases dopamine and glutamate in the shell of the nucleus accumbens (NAcc), one of the brain structures implicated in reward and motor-activation (Solinas et al., 2002). However, it has only mild reinforcing properties (Nehlig, 1999). Caffeine also increases serotonin, most

importantly in the raphe nuclei of the brainstem (Berkowitz & Spector, 1971; Stromberg & Waldeck, 1973). Caffeine's effect on serotonergic, as well as noradrenergic, neurons increases the self-sustained firing of motor neurons (Walton, Kalmar, & Cafarelli, 2002), leading to increased motor activity. Lastly, caffeine has an antagonistic action on blood circulation (Sawyer et al., 1982), which causes the smooth muscles of coronary, pulmonary, and general systemic blood vessel walls to dilate, while simultaneously stimulating the medullary vasomotor center in the brain stem, which causes these vessels to constrict (Ritchie, 1975).

3.3. Caffeine effects emotion

The effects of caffeine on mood and emotion can typically be categorized by the size of the dose consumed (Smith, Osborne, Mann, Jones, & White, 2004). Low to moderate doses of caffeine range from 20-200 mg, medium to high doses from 200-800 mg, and high to extreme doses from 1000-1500 mg (Herz, 1999; Hughes, 1996; Loke, 1988).

There is strong evidence that low doses of caffeine (20-200 mg) induce positive subjective effects (Fredholm et al., 1999; Smith, Sturgess, & Gallagher, 1999). Doses of 100 mg and below have resulted in increased ratings of alertness, well-being, social disposition, motivation for work, concentration, energy, self-confidence and euphoria as well as decreased ratings of anxiety, headache and sleepiness in a number of studies (Griffiths et al., 1990; Quinlan, Lane, & Aspinall, 1997; Silverman & Griffiths, 1992; Smith, Sturgess, et al., 1999). These effects are thought to occur in as little as 30 min post-consumption (Quinlan et al., 1997). However, not all cohorts have shown such a positive response. For example, only younger (age 18-37), but not older (age 65-75) subjects, reported 200 mg of caffeine to make them feel more alert and calmer (Swift & Tiplady, 1988). Furthermore, Lieberman, Wurtman, Emde, Roberts, and Coviella (1987) reported no effect of low doses of caffeine (32, 64, 128, 256 mg) on self-reported mood. This suggests that although the effects of caffeine on mood are consistent at low doses, they be somewhat complicated by dose, age, and individual differences (Smith et al., 2004).

Higher doses of caffeine (200-800 mg), on the other hand, often produce negative affect. This has been demonstrated in caffeine-deprived, non-

abstaining, and non-caffeine consumers (Griffiths & Mumford, 1995; Totten & France, 1995; Warburton, 1995). The most common negative affect reported is anxiety (Hughes, 1996 #427;Smith, 2004 #416). However, other feelings include tense arousal (Penetar et al., 1993), jitteriness, nervousness, (Evans & Griffiths, 1991; Green & Suls, 1996), shakiness, and trembling (Bruce, Scott, Lader, & Marks, 1986), as well as gastrointestinal disturbances (Greden, 1974), and appetite suppression (Sours, 1983). In general, these responses are thought to increase in severity as the dose of caffeine increases (Bruce et al., 1986).

Regular consumption of extreme levels of caffeine (1000-1500 mg) can lead to caffeinism, a condition that produces symptoms similar to anxiety neurosis, including nervousness, irritability, muscle twitching, insomnia, sensory disturbances, and flushing, among others (Greden, 1974). Recent research refers to this as caffeine intoxication and suggests only 500-600 mg of caffeine are needed to produce its anxiogenic effects (James & Stirling, 1983). Additionally, caffeine seems to exacerbate existing symptoms in those with anxiety disorders (Smith et al., 2004). This suggests that at low doses the substance results in a mild stimulant perceived as positive, but the substance can easily cause feelings of anxiety at moderate doses, which tend to increase in severity as dose increases.

3.4. Caffeine effects physiological arousal

Based on pharmacodynamics and subjective reports it is quite clear that the CNS, gastrointestinal system, and cardiovascular system are affected by caffeine consumption (Smith et al., 2004). This can result in many physiological responses including changes in heart rate, skin conductance, respiration rate, and body temperature, all of which are discussed below.

3.4.1. Heart rate

Caffeine's actions on HR are complex as it acts through several different mechanisms at multiple sites within the central and peripheral nervous systems. Caffeine can act at sympathetic nerve terminals as an adenosine antagonist, which releases norepinephrine and causes an increase in HR and contractility (Dunwiddie & Haas, 1985; Green, Kirby, & Suls, 1996). This effect is further augmented by an increase in the sympathetic drive to the heart via an increase

in activity in the locus coeruleus (Elam, Svensson, & Thorén, 1986). However, caffeine can also stimulate the medullary vagal nuclei either directly or via baroreceptor reflex mechanisms, which can lead to a decrease in HR (Green et al., 1996; Sawyer et al., 1982).

A review of the literature shows some studies to report an increase in HR due to caffeine in both coffee drinkers (Green & Suls, 1996) and caffeine-naïve subjects (Robertson, Wade, Workman, Woosley, & Oates, 1981). For example, moderate to high doses of caffeine (3 and 10 mg/kg; 500 mg) have significantly increased HR in abstaining men during cognitive tasks (Pincomb, Lovallo, Passey, & Wilson, 1988; Smith, Clark, & Gallagher, 1999) and when consumed at work (Lane, Phillips-Bute, & Pieper, 1998). When testing the effects of multiple oral doses of caffeine, Passmore, Kondowe, and Johnston (1987) found the highest dose of 360 mg to show a late increase in HR at 3 and 4 h post-consumption. Higher doses of caffeine can also lead to tachycardia and arrhythmias (Dobmeyer, Stine, Leier, Greenberg, & Schaal, 1983).

Despite the results of the aforementioned studies, there is overwhelming evidence that caffeine actually decreases HR. This result has been found for moderate doses of caffeine (3.3 mg/kg) (Pincomb et al., 1985) and for studies using coffee (Smits, Thien, & van't Laar, 1985; Whitsett, Manion, & Christensen, 1984). Caffeine has been shown to decrease HR when standing and sitting (Charney, Galloway, & Heninger, 1984), and even for one study which incorporated exercise into the design (Pincomb, Wilson, Sung, Passey, & Lovallo, 1991). Some suggest that this decrease in HR progressively declines as caffeine dose increases (Quinlan et al., 2000).

The inconsistencies found in the literature may be partially explained by studies that show HR to have a diphasic response to caffeine. For example, caffeinated beverages between 37.5- 150 mg have resulted in an immediate increase, followed by a decrease in HR 10-30 min post-consumption, an effect shown to persist 60-105 min post-consumption (Quinlan et al., 2000). Another study found caffeine to decrease HR, but not until 30-90 min post-consumption, after which HR began to increase (Astrup et al., 1990). Overall, this diphasic response is thought to result in a decrease in HR that reaches a minimum ~45 min post-consumption (Robertson et al., 1978), a time window similar to when

caffeine reaches peak plasma level (e.g. ~30-60 min post-consumption) (Benowitz, 1990; Blanchard & Sawers, 1983). This suggests that caffeine decreases HR, but as the substance is metabolized the physiological effect fades.

3.4.2. Skin conductance

One of the most consistent physiological effects of caffeine is an increase in skin conductance, both for tonic (SCL) and phasic (SCR) measures (Bruce et al., 1986; Davidson & Smith, 1991; Totten & France, 1995; Zahn & Rapoport, 1987a, 1987b). Studies show 100 mg of caffeine to increase SCR 3-30 min post-consumption and SCL 30-57 min post-consumption (Quinlan et al., 1997). Similar results were obtained for a later study where caffeinated tea (37.5 and 75 mg) and coffee (75 and 150 mg) increased SCR more so than hot water, but this effect only lasted for 10-30 min post-consumption. Higher doses of caffeine over longer time courses have yielded similar results. For example, a study administering 250 and 500 mg of caffeine showed a dose-related increase in SCL that persisted over the 5 h testing period (Bruce et al., 1986).

Increases in SCL and SCR have also been found for caffeine during task performances. In a study administering 3.3 mg/kg of caffeine, participants' physiological measurements were taken during resting baseline as well as during stressor tasks, including during a cold pressor, mental arithmetic, and an anxiety-provoking film. Caffeine elicited significant increases in resting SCL and further increased SCL during the stressor tasks (Totten & France, 1995). In an auditory experiment, caffeine resulted in dose dependent increases in SCL and SCR during a listening task (Smith, Wilson, & Davidson, 1984). However, two studies administered 3 and 10 mg/kg to high and low caffeine consumers during rest, a series of tones, and a RT task (Zahn & Rapoport, 1987a, 1987b). Although SCR was found to increase during the RT task, an effect that was larger for low consumers, SCL only increased during the non-task periods.

Interestingly, Zahn and Rapoport (1987b) also found caffeine to slow the rate at which SCL returned to normal during the resting period and higher doses of caffeine slowed the rate of skin conductance orienting responses more so than lower doses during the task performance. This additionally suggests that caffeine may have a habituation effect on arousal, keeping arousal

elevated where it would otherwise decrease. Although further analyses by the researchers purposed this effect to be somewhat equivocal and measure-dependent, other studies have found caffeine to maintain arousal. For example, using 20 identical auditory tones, one study found 300 and 600 mg of caffeine to reduce the rate of diminution for SCR amplitude (Lader, 1969). Another study suggests caffeine to both slow and smooth habituation, demonstrated by 300 mg of caffeine during a digit-span stimulus task (Davidson & Smith, 1989). This suggests that caffeine, in addition to increasing arousal, can help maintain it. In a later study, Davidson and Smith (1991) administered either 300 mg of caffeine or placebo to 48 participants. They were then subject to two backwards recall tasks, one that was novel and one that was repetitive. Caffeine produced and maintained higher SCL during both tasks, demonstrating that caffeine can slow and smooth habituation, as well as enhance the arousal effects of novel stimulation.

3.4.3. Respiration rate

Overall, caffeine is thought to increase measures of respiration. This is due to caffeine stimulating the medullary respiratory center, which causes an increase in respiration rate, oxygen consumption, and the elimination of CO^2 (Sawyer et al., 1982). Caffeine is thought to increase respiration rate by ~20% (D'urzo et al., 1990) and is particularly found to enhance maximal oxygen consumption (VO_2) (Toner et al., 1982). The earliest work showed that the increase in respiration rate was greater after caffeine consumption compared to before (Cushney, 1913). Since then, studies have focused on how caffeine affects apnea and how it enhances exercising capabilities.

In premature infants, caffeine has been helpful in treating apnea (Larsen, Brendstrup, Skov, & Flachs, 1995). For example, in preterm infants, caffeine reduced the number of days needed for respiratory support, supplemental oxygen therapy, and assisted ventilation (Davis et al., 2010; Schmidt et al., 2006). Furthermore, loading doses of 20 mg/kg followed by maintenance doses of 5 or 10 mg/kg of caffeine resulted in an increase in respiration rate in newborn infants with apnea (Aranda, Gorman, Bergsteinsson, & Gunn, 1977).

Caffeine's effect as a respiratory stimulant also has implications for adults. For example, 250 mg of caffeine give to 9 non-coffee drinkers increased

respiration rate over time (Robertson et al., 1978). Also, caffeine's effect as a respiratory stimulant enhances oxygen uptake, helping performance during sustained exercise. This has been demonstrated with 3 g of caffeine during high-intensity running (Wiles, Bird, Hopkins, & Riley, 1992). It has also been found during cycling exercises, as was found for 500 mg during isokinetic cycling (Ivy, Costill, Fink, & Lower, 1979) and 330 mg during a bicycle exercise where participants cycled until exhaustion (Costill, Dalsky, & Fink, 1978).

However, some studies find caffeine to exert no effect on respiratory measures, suggesting that caffeine's respiratory effects are inconsistent or subject to individual differences. For instance, 6 males with impaired responses to epinephrine (e.g. tetrapalegics) were tested with 6 mg/kg of caffeine and were found to have no change in respiratory exchange ratio (RER), the ratio between the amount of O_2 consumed and the amount of CO_2 produced from a single breath (Van Soeren, Mohr, Kjaer, & Graham, 1996). Another study found no effect of caffeine when examining a healthy population during exercise. That is, using 9 mg/kg of caffeine, Spriet and colleagues (1992) examined runners during cycling and running, but found no effect of caffeine on RER.

3.4.4. Skin temperature

Caffeine is thought to increase resting metabolic rate through thermogenesis, which can increase internal body temperature in both physically trained and sedentary individuals (Armstrong, Casa, Maresh, & Ganio, 2007). However, caffeine is also suggested to decreases peripheral skin temperature as a result of a rise in plasma catecholamines (Smits, Hoffmann, Thien, Houben, & Van't Laar, 1983), which lead to peripheral vasoconstriction (Quinlan et al., 1997). This suggests that caffeine acts on different mechanisms to cause an increase and a decrease in skin temperature and a review of the literature reflects this dichotomy.

There is an increase in skin temperature in a number of studies using low to moderate doses of caffeine. Such studies suggest a significant increase in skin temperature 90-120 min post-consumption (Koot & Deurenberg, 1995; Tagliabue et al., 1994). However, these studies often report no information about the initial bodily responses to caffeine and ignore the effects that hot beverages can have on skin temperature. Therefore, Quinlan, Lane, and

Aspinall (1997) examined individuals who were administered hot water, as well as 100 mg of caffeine infused in tea and coffee. All hot beverages were rapidly increase peripheral skin temperature by ~1.7˚C, with a peak at ~15 min post-consumption. This was followed by a decline in temperature that returned to baseline ~1 h later. Interestingly, when compared to water, the caffeine in both tea and coffee decreased skin temperature by ~0.7˚C. This occurred 30-60 min post-consumption, although tea maintained a higher skin temperature than coffee. Similar reductions in peripheral skin temperature have been reported for 3 and 10 mg/kg of caffeine (Zahn & Rapoport, 1987b).

Dose dependent responses to caffeine have also been investigated using tea (37.5 and 75 mg) and coffee (75 and 150 mg), along with water and no-drink control conditions. Results show that in the first 10 min, hot beverage ingestion rapidly increased skin temperature by ~1˚C, followed by an increase of ~1.8˚C at the 10-30 min time point. However, no dose dependent effects and no significant differences between tea and coffee on skin temperature were found. In a follow up study, participants were subject to various caffeine conditions: hot water, 5, 30, 55, 105, and 205 mg of caffeine in tea. Again, hot beverages were associated with an increase in skin temperature of ~1.5˚. This effect also occurred 10 min post-consumption, but then declined to baseline or below ~40 min post-consumption. This increase in skin temperature is thought to be largely due to the effects of hot water as caffeine overall decreased skin temperature and did so with a dose dependent response. That is, 5 mg tea maintained the highest skin temperature, where as 205 mg of caffeine decreased skin temperature by 1.39˚C. This decrease is likely a reflection of peripheral vasoconstriction and an increase in vascular resistance (Quinlan et al., 2000).

3.5. Caffeine increases emotion via misattribution and excitation transfer

As mentioned in Chapter 2, heightened physiological arousal can increase the physical sensations that accompany emotions and lead to an intensification of emotional experiences (Zillmann, 1978). This occurs through peripheral feedback, where individuals use the bodily sensations they experience from physiological changes in order to inform them of their

emotions (Schachter & Singer, 1962). These processes have been demonstrated in a musical context, where physiological arousal was induced through exercise and in turn led to an increase in the intensity of music-induced emotions (Dibben, 2004). Therefore, caffeine may be able to similarly increase physiological arousal and in turn increase music-induced emotion. There are two other mechanisms that may help to explain this process further, misattribution and excitation transfer.

A concept similar to peripheral feedback is misattribution, where individuals mistakenly link their increase in physiological arousal, and therefore an induction of emotion, to the wrong stimulus. This occurs when a stimulus that induces arousal is not identified or is ambiguous and causes an individual to attribute their arousal to their current environment (Schachter, 1964; Schachter & Wheeler, 1962). For example, Nisbett and Schachter (1966) administered placebo pills and found that those who had been told the pill would induce symptoms of arousal (e.g. heart palpitations, tremors, and increases in breathing) tolerated higher levels of pain when electrically shocked. Subjects tolerated these higher pain levels because they determined that the cause of their arousal stemmed from the pill and not from the shocks. Misattribution has also been associated with positive emotion. Dutton & Aron (1974) demonstrated the effect of anxiety on heightened sexual attraction. After crossing either a wobbly "fear-arousing" suspension bridge or a stable wooden bridge, male passers-by were approached and asked to complete a Thematic Apperception Test (TAT). At the end of the survey the female experimenter wrote down her phone number. She invited participants to call and discuss the details of the study with her if they wished. The men who had crossed the fear-arousing bridge experienced an increase in physiological arousal due to vertigo. In turn, they interpreted their arousal as infatuation, which led to greater sexual content in their TAT stories and a greater likelihood of them phoning the experimenter. In this way, misattribution can help explain why an increase in physiological arousal, induced by caffeine, may increase music-induced emotion. However, misattribution is thought to influence individuals mainly when their arousal is unexplained or ambiguous. Therefore, it may not be able to explain completely how caffeine's influence on arousal can

increase music-induced emotion, especially if individuals are aware of their caffeine consumption. Therefore, the related mechanism of excitation transfer may also help explain this circumstance.

Excitation transfer is a misattribution of excitation, where residual arousal from one experience amplifies the emotional reactions of an immediate and unrelated subsequent experience (Zillmann, 1971, 1983). Excitation transfer has been demonstrated using a number of paradigms, including those with exercise and caffeine (Cantor, Zillmann, & Bryant, 1975; Miller, Murphy, & Buss, 1981; Zillmann et al., 1972). For example, males aroused with 350 mg of caffeine were more aggressive towards a confederate (Taylor, O'Neal, Langley, & Butcher, 1991). This demonstrates that caffeine is able to increase physiological arousal and intensify a subsequent emotional experience. Excitation transfer has also been demonstrated in a musical setting. Cantor and Zillmann (1973) presented one of four film segments to individuals that varied in valence (positive, negative) and arousal (high, low), then asked participants to rate three musical excerpts. Excitation transfer was found for the highly arousing film, which intensified the positive responses to the music. Also, in Dibben (2004) participants who were induced with arousal through exercise prior to giving emotional judgments of musical excerpts provided increased ratings of emotions compared to a relaxation control group. This demonstrates that heightened physiological arousal can influence musical emotion. It further suggests that if arousal is induced by caffeine then it too may lead to an increase in music-induced emotion through the process of excitation transfer.

3.6. Summary and overview

Previously, nicotine administration was shown to result in patterns of physiological responses and self-reports indicative of an additive effect on music-induced emotion in smokers. However, it is not well understood to what extent these additive effects stemmed from nicotine's ability to increase arousal compared to its ability to increase pleasure. Furthermore, nicotine increased arousal in both smokers and nonsmokers, it only increased pleasure in smokers. This suggests that an increase in arousal may have played a larger role in increasing music-induced emotions, but further investigation in needed to confirm this. Therefore, the aim of the current study is to use caffeine to

disassociate the effects of nicotine by increasing physiological arousal without increasing pleasure (Herz, 1999). This will help to identify the role that increased physiological arousal has on the amplification of music-induced emotion. In turn, this may help to explain why nicotine and music often co-exist.

Caffeine is known to increase arousal based on its ability to manipulate heart rate, skin conductance, respiration rate, and skin temperature (Cushney, 1913; Quinlan et al., 2000). Since caffeine can increase arousal, and in turn arousal can intensify emotion through misattribution and excitation transfer (Schachter & Wheeler, 1962; Zillmann, 1971), it may be that caffeine can amplify music-induced emotions.

The aim of this study was to examine the effects of caffeine on music-induced emotion. This was accomplished by inducing a heightened physiological state in participants via caffeine administration, then asking them to listen to the same musical excerpts used in study one. As with the previous study, self-reports of arousal, pleasure and, emotion, and physiological measurements, were recorded. It was hypothesized that upon the intake of caffeine and subsequent action of music listening, two results would occur: (1) an individual would experience an increase in the intensity of felt emotion and (2) would experience an increase in physiological and self-reported arousal.

3.7. Methods

3.8. Participants

For this study I recruited a total of 120 participants living in England. As with the nicotine study, many participants were recruited with a flyer (Appendix H) as well as through a convenience sample. Table 3.1 provides a summary of the age and gender for each group by smoking status (nonsmoking, smoking) and caffeine dose (0, 200, 400 mg). Furthermore, Table 3.2 and Table 3.3 display occupation as well as caffeine and music consumption information for smokers and nonsmokers, respectively. Smokers and nonsmokers were defined using the same criteria as study one. No participants were professional musicians, but 62% had musical performance experience to at least a high school level. Informed consent was obtained prior to experimentation and participants received £5 for their time. The research protocol met the ethical requirements of the University of Sheffield's Department of Psychology.

Table 3.1

Table report of age and gender by smoking status and caffeine dose

	Smokers			Nonsmokers		
Caffeine Dose	*N*	Age	Gender	*N*	Age	Gender
0 mg	21	M = 26.05; SD = 10.46	9 M; 12 F	20	M = 23.20; SD = 4.90	10 M; 10 F
200 mg	19	M = 23.84; SD = 8.62	8 M; 11 F	20	M = 23.10; SD = 3.29	9 M; 11 F
400 mg	20	M = 22.25; SD = 4.79	9 M; 11 F	20	M = 24.80; SD = 8.49	8 M; 12 F

Table 3.2

Table report of occupation and nicotine consumption by smoking status and nicotine dose

	Smokers				
Caffeine Dose	Occupation	Average # of Cigarettes/day	Average Time Smoking	Average Music Consumption (h/wk)	Average Caffeine Beverages/wk
0 mg	UG 52.4% PG 23.8% *Non-student 23.8%	M = 13.29, SD = 5.19	8.80 years	M = 23.10, SD = 18.46	M = 26.67, SD = 23.67
200 mg	UG 73.7% PG 10.5% *Non-student 15.8%	M = 10.26, SD = 3.26	9.53 years	M = 16.63, SD = 10.88	M = 36.21, SD = 12.74
400 mg	UG 65% PG 20% *Non-student 15%	M = 11.35, SD = 4.67	6.47 years	M = 22.05, SD = 17.57	M = 17.55, SD = 8.90

Note: UG = undergraduate student; PG = postgraduate student
*Non-student employment included administrator, construction worker, museum educator, personal assistant, civil servant, photographer, cleaner, office worker, and waiter.

Table 3.3

Table report of occupation and nicotine consumption by smoking status and nicotine dose

Nonsmokers				
Caffeine Dose	Occupation	Average # of Cigarettes Smoked in Lifetime	Average Music Consumption (h/wk)	Average Caffeine Beverages/ week
0 mg	Student UG 60% Student PG 30% *Non-student 10%	M = 4.55, SD = 13.31	M =23.30, SD = 22.96	M = 16.35, SD = 8.50
200 mg	Student UG 60% Student PG 25% *Non-student 15%	M = 3.85, SD = 10.02	M = 18.70, SD = 17.62	M = 15.60, SD = 7.98
400 mg	Student UG 60% Student PG 20% *Non-student 20%	M = 2.75, SD = 8.84	M = 23.70 SD = 24.87	M = 18.75, SD = 10. 79

*Non-student employment included cleaner, data archiver, art administrator, lawyer, writer, manager, nurse, teacher, and waiter.

3.9. Material

3.9.1. Caffeine tablets

The caffeine was administered in 200 and 400 mg doses in tablet form. For placebo, 15 mg of zinc tablets were chosen because they closely resembled the caffeine tablets; both were small, round, and white. Tablets were chosen because it is an easy and effective method of administration that can control the amount of caffeine ingested by each participant. Doses of 200 and 400 mg were chosen because previous research shows these to be moderate doses of caffeine that effect mood (Loke, 1988; Quinlan et al., 2000).

3.9.2. Other material

All other materials used were identical to that of study one, including the musical background questionnaire (Appendix B), smoking history questionnaire (Appendix C), health screening survey (Appendix D), Subjective Treatment

Emergent Symptom Scale (STESS) (Guy, 1976b), musical excerpts (Appendix E), and reading material (Appendix F). Additionally, caffeine consumption questions were added to the smoking history questionnaire (Appendix I). Participants were also CO tested before the experiment and were administered the same rating scales and subject to the same physiological measurements. These materials were purposefully kept consistent across studies to ensure accurate comparisons between the effects of nicotine and caffeine.

3.10. Procedure

Again, in order to draw accurate comparisons between nicotine and caffeine, the procedure for study two was identical to study one. That is, after confirming eligibility, participants refrained from nicotine, caffeine, and alcohol for 24 h prior to experimentation. The experiment then began with an information sheet (Appendix J) and participants provided informed consent. Next, participants were attached to the physiological recording equipment and a 2 min baseline recording was taken. This was followed by baseline self-reports of arousal, pleasure, and emotion ratings. Participants were then administered placebo, 200, or 400 mg of caffeine and asked to engage in a reading (Cook, 1998) and writing distraction task (Appendix F). The STESS (Guy, 1976b) was then administered to check for any adverse effects of caffeine, where a score of 50% or higher discontinued the participants from the study. No participants were discontinued for this reason. The music listening task then began, where participants listened to four musical excerpts (happy, sad, neutral, self-selected/chill-inducing) presented in random order. During each musical excerpt physiological measurements were taken and afterwards self-reports were provided.

3.11. Data analysis

Data were analyzed using the same method as study one. Each physiological measurement (HR, SCL, RR, ST) was first averaged over each 2 min recording session to produce temporal mean scores. Then, change scores were computed by subtracting each participant's post-ingestation baseline score for HR, SCL, RR, and ST from his or her subsequent and corresponding post-ingestation scores during each of the four musical conditions. For self-reported data post-ingestation baseline ratings for arousal, pleasure, happiness, and

sadness were subtracted from their subsequent and corresponding ratings for each of the four music categories (happy music, sad music, neutral music, and chill-inducing music).

The data were then analyzed to compare physiological (section 3.12 – section 3.15) and self-reported (section 3.16- section 3.18) response. First, comparisons between smokers' and nonsmokers' physiological responses to nicotine and music were conducted using a repeated measures MANOVA with between subjects variables of smoking status (two levels- smoking, nonsmoking) and caffeine condition (three levels – 0, 2, 4 mg), a within subjects variable of music (four levels – happy, sad, neutral, chill-inducng), and a dependent variable of physiological response (four levels – HR, SCL, RR, ST).

Two follow up repeated measures MANOVAs were then performed to examine the physiological response of smokers and nonsmokers separately. More specifically, a repeated measures MANOVA was performed once for smokers and then again for nonsmokers. Each MANOVA had a between subjects variable of caffeine condition (three levels – 0, 200, 400 mg), a within subjects variable of music (four levels – happy, sad, neutral, chill-inducing), and a dependent variable of physiological response (four levels – HR, SCL, RR, ST).

To further examine the effects of caffeine across the music types, a series of one way univariate ANOVAs were performed separately for each dependent measure (HR, SCL, RR, ST) and for each cohort (smoking, nonsmoking), where relevant (if multivariate tests were statistically significant). These were further followed up with *t*-tests where relevant. Due to the restricted number of comparisons (0 vs 2 mg; 2 mg vs 4 mg), follow up *t*-tests used a significance threshold (p value) of $p = .0125$.

The analysis of the self-reported responses follows the same structure as that of the physiological analysis. That is, smokers' and nonsmokers' self-reported responses to caffeine and music were examined using a repeated measure MANOVA with between subjects variables of smoking status (two levels – smoking, nonsmoking) and caffeine condition (three levels – 0, 200, 400 mg), as well as a within subjects variable of music (four levels – happy, sad, neutral, chill-inducing). The dependent variable was self-reported responses (four levels – arousal, pleasure, happiness, sadness).

Two follow up repeated measures MANOVAs were then performed in order to examine the self-reported responses of smokers and nonsmokers separately. A repeated measures MANOVA was performed once for smokers and then again for nonsmokers. There was a between subjects variable of caffeine condition (three levels – 0, 200, 400 mg), a within subjects variable of music (four levels- happy, sad, neutral, chill-inducing), and a dependent variable of self-reported responses (four levels – arousal, pleasure, happiness, sadness).

Where relevant, the effects of nicotine across the music types where examined further using a series of one-way univariate ANOVAs. These univariate tests were performed separately for each dependent physiological measure (HR, SCL, RR, ST) and each self-reported measure (arousal, pleasure, happiness, sadness) and for each cohort (smoking, nonsmoking). These were followed up with *t*-tests where relevant. Due to the restricted number of comparisons (0 vs 200 mg; 200 mg vs 400 mg), follow up *t*-tests used a significance threshold (*p* value) of $p = .0125$.

For all repeated measures MANOVAs variables found to violate the assumption of sphericity were corrected with a Greenhouse-Geisser correction and all post-hoc tests were corrected with a Bonferroni correction.

3.12. Results

The study investigated the effects of caffeine and music on the physiological and self-reported arousal/pleasure/emotional responses of participants. Analyses were performed separately for physiological and self-reported data. The first set of results reports the physiological responses to nicotine (section 3.13-3.15). The second set of results reports the self-reported arousal/pleasure/emotional response (section 3.16-3.18). To organize the results more clearly, the analysis involving physiological responses is divided into 3 sub-sections: first, a main effect caffeine (section 3.13), then a main effect of music (section 3.14), then an interaction effect of caffeine and music (section 3.15). The analysis involving self-reported responses is also divided into 3 sub-sections: first, a main effect of caffeine (section 3.16), then a main effect of music (section 3.17), then an interaction effect of caffeine and music (section 3.18).

After computing change scores for physiological arousal several variables were found to violate the assumption of normality with an absolute value of skewness and kurtosis that were more than twice the standard error. Because each caffeine condition contained an equal number of participants (N = 20) and because the ANOVA test is robust to violations of the normality assumptions (Harwell et al., 1992) the data was not transformed. Instead, I calculated the mean and standard deviation of each variable and then removed any scores that were more than three standard deviations away from the mean (Howitt & Cramer, 2005). Based on this criterion, for heart rate I removed four outlier each from happy and sad music, one outlier from neutral music, and five outliers from chill-inducing music. From skin conductance level I removed one outlier each from happy and chill-inducing music, two from sad music, and three from neutral music. From Respiration rate I removed one outlier each from happy, sad, neutral and chill-inducing music. From Skin temperature I removed one outlier each from happy and sad music, and two outliers from neutral music. All subsequent analyses involving these variables were conducted with these outliers removed. Outliers were also visually inspected using histograms and by referencing the raw data. This was done in order to confirm that the scores removed were outliers. Many of the values removed were found to be outliers, but were not beyond the scope of human physiological responses (as was seen with nicotine data in Chapter 2).

3.13. Effects of caffeine on physiological arousal

The following section reports the main effect of caffeine on physiological responses, first between smokers and nonsmokers (section 3.13.1), then on smokers (3.13.2), and lastly on nonsmokers (3.13.3).

3.13.1. Effects of caffeine on physiological arousal between smokers and nonsmokers

A Multivariate test revealed a nonsignificant difference between smokers' and nonsmokers' physiological response to caffeine, $F(4, 89) = .37$, $p = .830$, $\eta 2 = .02$. Figure 3.1 and Figure 3.2 reflect this as both cohorts had some similar physiological responses. In response to caffeine HR was found to systematically decrease for smokers, but systematically increase for nonsmokers. However, SCL systematically increased in response to caffeine for

both cohorts, although smokers showed a great response than nonsmokers. For both cohorts caffeine increased respiration rate above placebo, with a greater response at the lower 200 mg dose. For both cohorts caffeine decreased skin temperature. However, this response was greater for smokers at the 200 mg dose, while for nonsmokers it was greater at the 400 mg dose.

3.13.2. Effects of caffeine on physiological arousal in smokers

A multivariate test revealed a nonsignificant main effect of caffeine on physiological arousal in smokers, $F(8, 98) = .57$, $p = .798$, $\eta 2 = .05$. However, there are trends observable in the data. For example, in Figure 3.1 and Figure 3.2 it clear that in response to caffeine 1) HR systematically decreased, 2) SCL systematically increased, 3) respiration rate increased, more so at the 200 mg dose, and 4) skin temperature decrease, more so at the 200 mg dose.

3.13.3. Effects of caffeine on physiological arousal in nonsmokers

A multivariate test revealed a nonsignificant main effect of caffeine on physiological arousal in nonsmokers, $F(8, 74) = .76$, $p = .641$, $\eta 2 = .08$. However, there are trends observable for nonsmokers in Figure 3.1 and Figure 3.2. For example in response to caffeine, 1) HR and SCL systematically increase, 2) respiration rate increase, more so at the 200 mg dose, and 3) skin temperature systematically decreased.

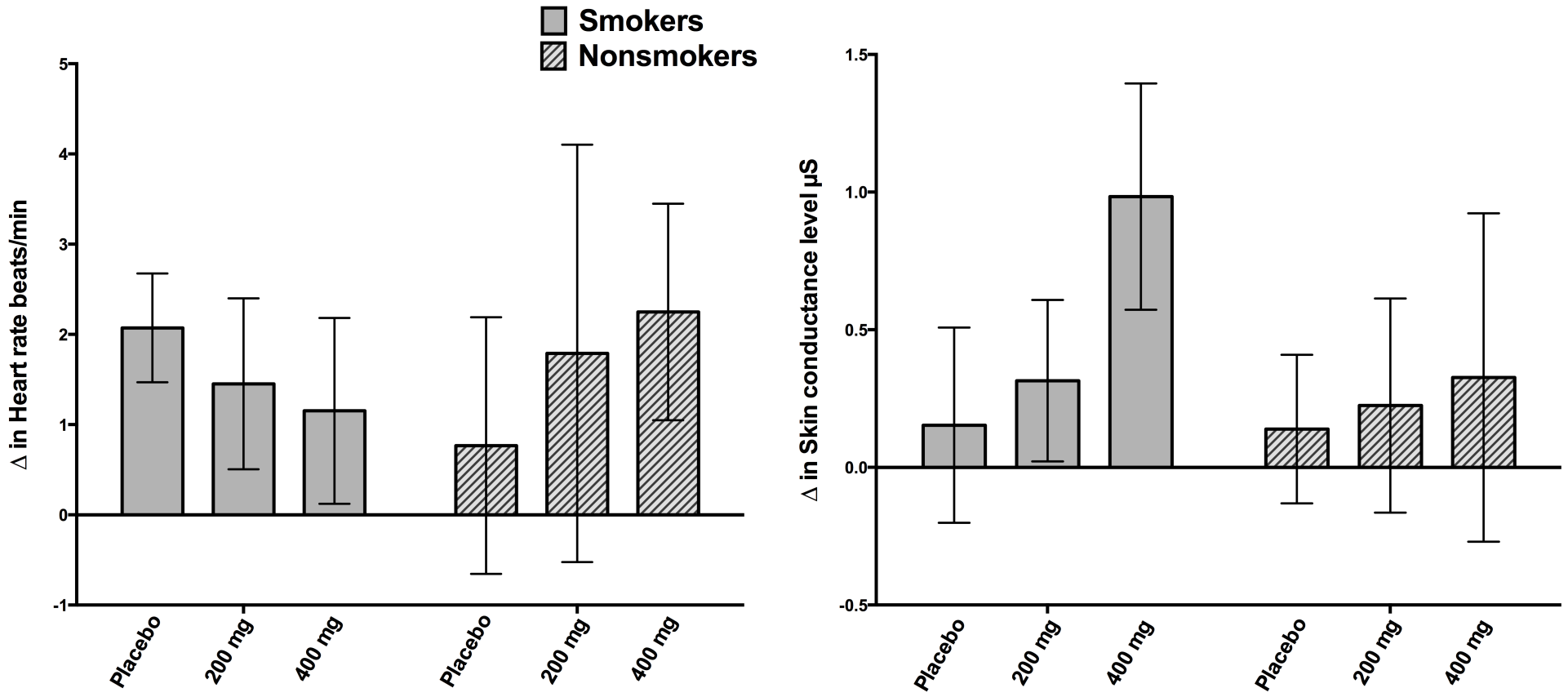

Figure 3.1. The mean and standard errors for smokers' and nonsmokers' heart rate and skin conductance level responses to each caffeine condition.

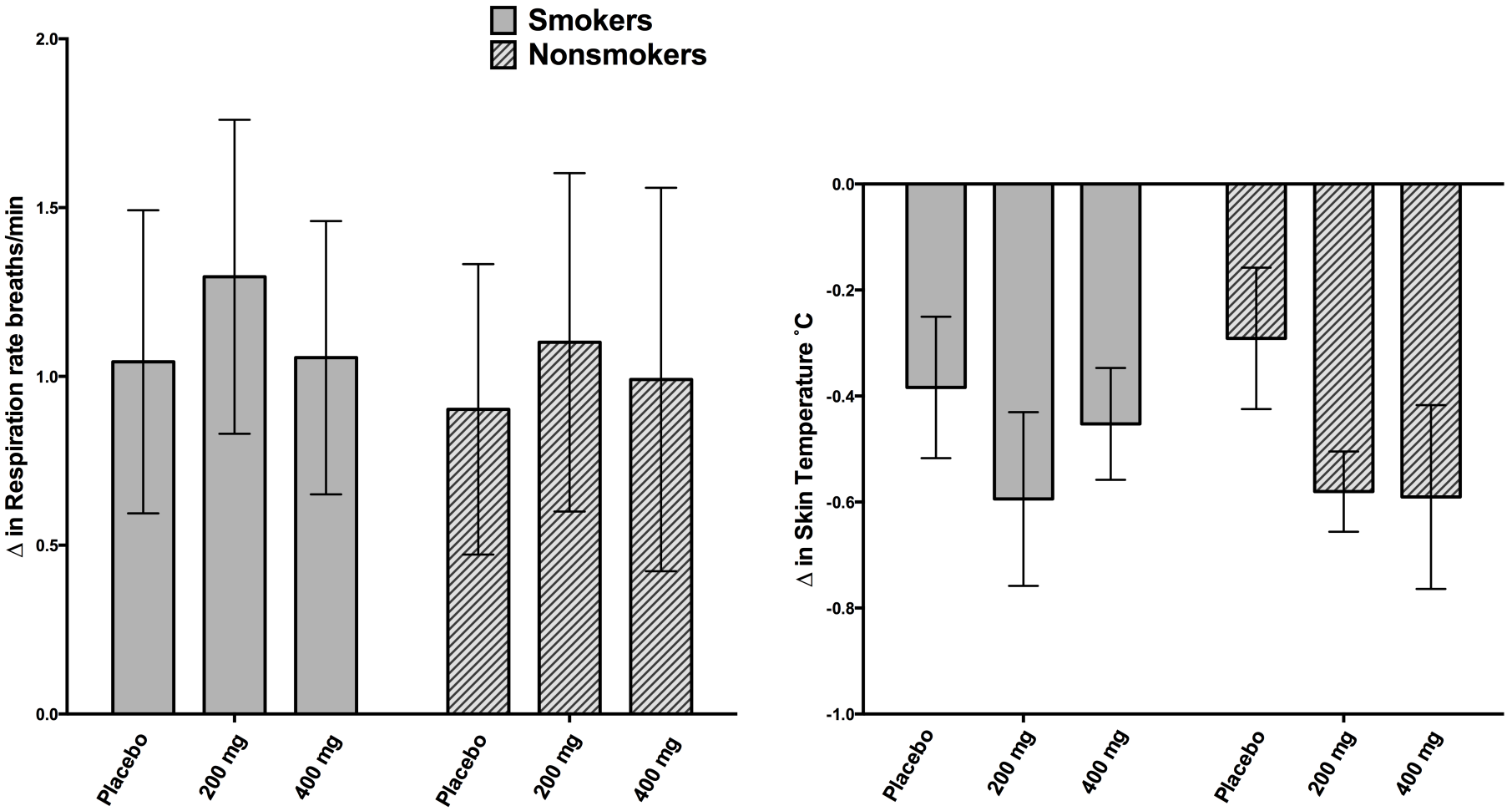

Figure 3.2.The mean and standard errors for smokers' and nonsmokers' respiration rate and skin temperature responses to each caffeine condition.

3.14. Effects of music on physiological arousal

The following section reports the main effect of music on the physiological response, first between smokers and nonsmokers (section 3.14.1), then on smokers (3.14.2), and finally on nonsmokers (3.14.3).

3.14.1. Effects of music on physiological arousal between smokers and nonsmokers

A Multivariate test revealed a nonsignificant difference between smokers' and nonsmokers' physiological response to music, $F(12, 81) = 1.37$, $p = .197$, $\eta 2 = .17$. More specifically, in response to music there was no significant difference between smokers' and nonsmokers' physiological responses. Figure 3.3 and Figure 3.4 reflect this as both cohorts show happy and chill-inducing music to result in larger increases in HR, SCL, and respiration rate, and smaller decreases in skin temperature, compared to sad and neutral music.

3.14.2. Effects of music on physiological arousal in smokers

A multivariate test showed a significant main effect of music on physiological arousal in smokers, $F(12, 41) = 3.95$, $p < .001$, $\eta 2 = .54$. Further analysis showed HR, $F(2.09, 108.64) = 8.92$, $p < .001$, $\eta 2 = .15$ and SCL, $F(1.33, 69.17) = 16.15$, $p < .001$, $\eta 2 = .24$, to significantly differ between music conditions, but not respiration rate, $F(2.11, 109.51) = 1.23$, $p = .298$, $\eta 2 = .02$, or skin temperature, $F(2.58, 133.97) = .99$, $p = .389$, $\eta 2 = .02$.

Pairwise comparisons show HR to be significantly higher for chill-inducing music compared to all other music types, including happy ($p = .041$), sad ($p = .002$), and neutral music ($p = .010$). SCL was significantly higher for chill-inducing music compared to all other music types ($p < .001$). Additionally, SCL was significantly higher during happy music compared to sad ($p = .025$) and neutral music ($p = .020$). Although respiration rate and skin temperature showed no significant differences between the music conditions trends existed. Happy and chill-inducing music were higher in respiration rate compared to sad and neutral music, with sad music showing the lowest respiration rate. Happy and chill-inducing music also showed a higher skin temperature compared to sad and neutral music, which were nearly equal in skin temperature. These results can be viewed in in Figure 3.3 and Figure 3.4.

3.14.3. Effects of music on physiological arousal in nonsmokers

A multivariate test showed a significant main effect of music on physiological arousal in nonsmokers, $F(12, 29) = 4.95$, $p < .001$, $\eta 2 = .67$. Further analysis showed HR, $F(2.25, 89.80) = 11.99$, $p < .001$, $\eta 2 = .23$, SCL, $F(1.81, 72.04) = 10.66$, $p < .001$, $\eta 2 = .21$, and respiration rate, $F(3, 120) = 5.51$, $p = .001$, $\eta 2 = .12$ to significantly differ between music conditions, but not skin temperature, $F(3, 120) = .56$, $p = .643$, $\eta 2 = .01$.

Pairwise comparisons show HR to be significantly higher during chill-inducing music compared to sad ($p = .001$) and neutral music ($p = .002$). Also, HR was significantly higher during happy music compared to sad ($p < .001$) and neutral music ($p = .004$). Similar findings are shown for SCL, which was significantly higher during chill-inducing music compared to sad ($p = .025$) and neutral music ($p = .001$). Additionally, SCL was significantly higher during happy music compared to sad ($p = .029$) and neutral music ($p = .001$). Respiration rate was significantly higher during chill-inducing music compared to sad ($p = .033$) and neutral music ($p = .011$). Although skin temperature showed no significant differences between the music conditions trends show chill-inducing music to have the highest skin temperature and sad music to have the lowest skin temperature. These responses can be viewed in Figure 3.3 and Figure 3.4.

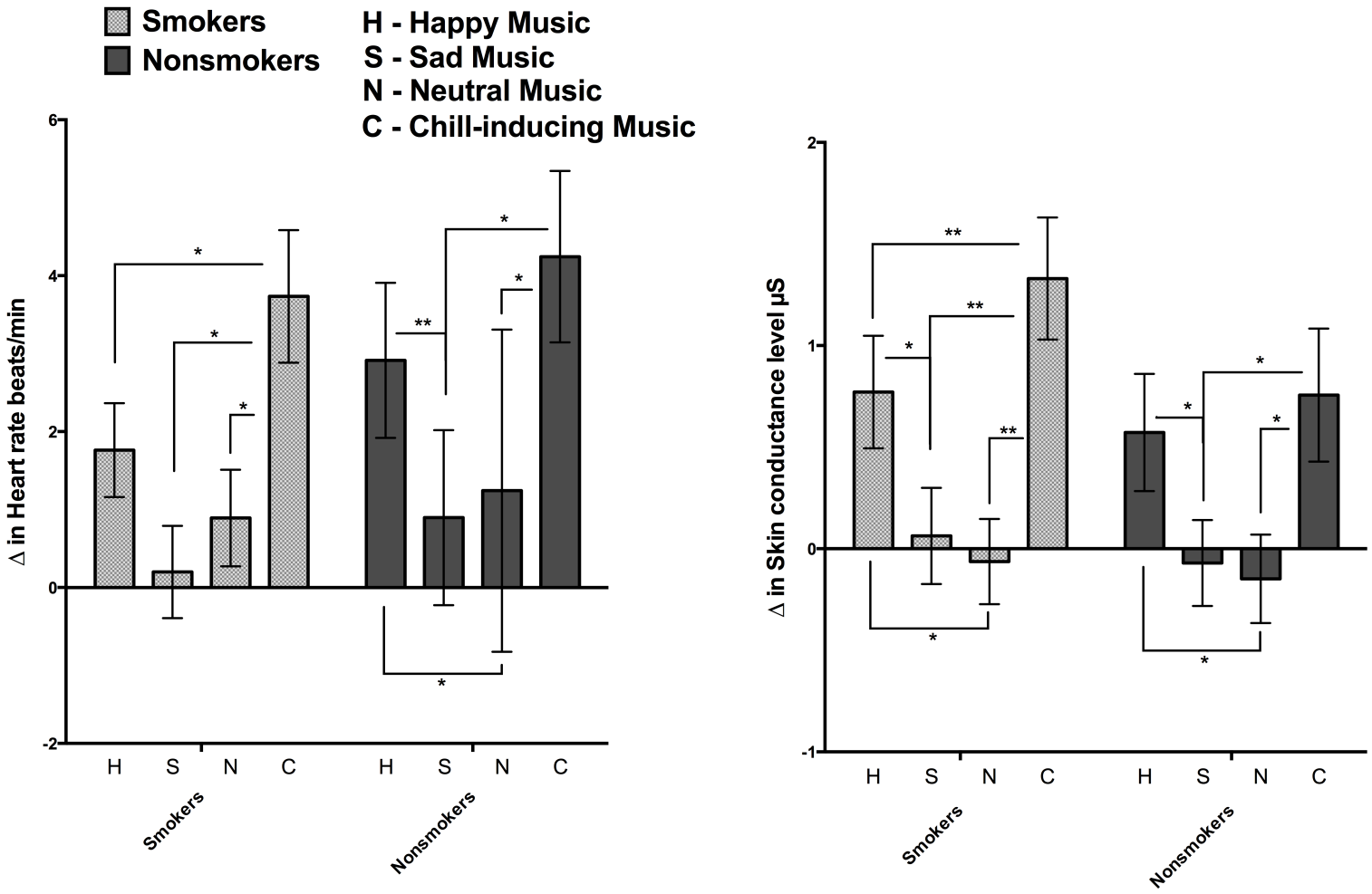

Figure 3.3. The mean and standard errors for smokers' and nonsmokers' heart rate and skin conductance level responses to each music condition. * $p < .05$, ** $p < .001$.

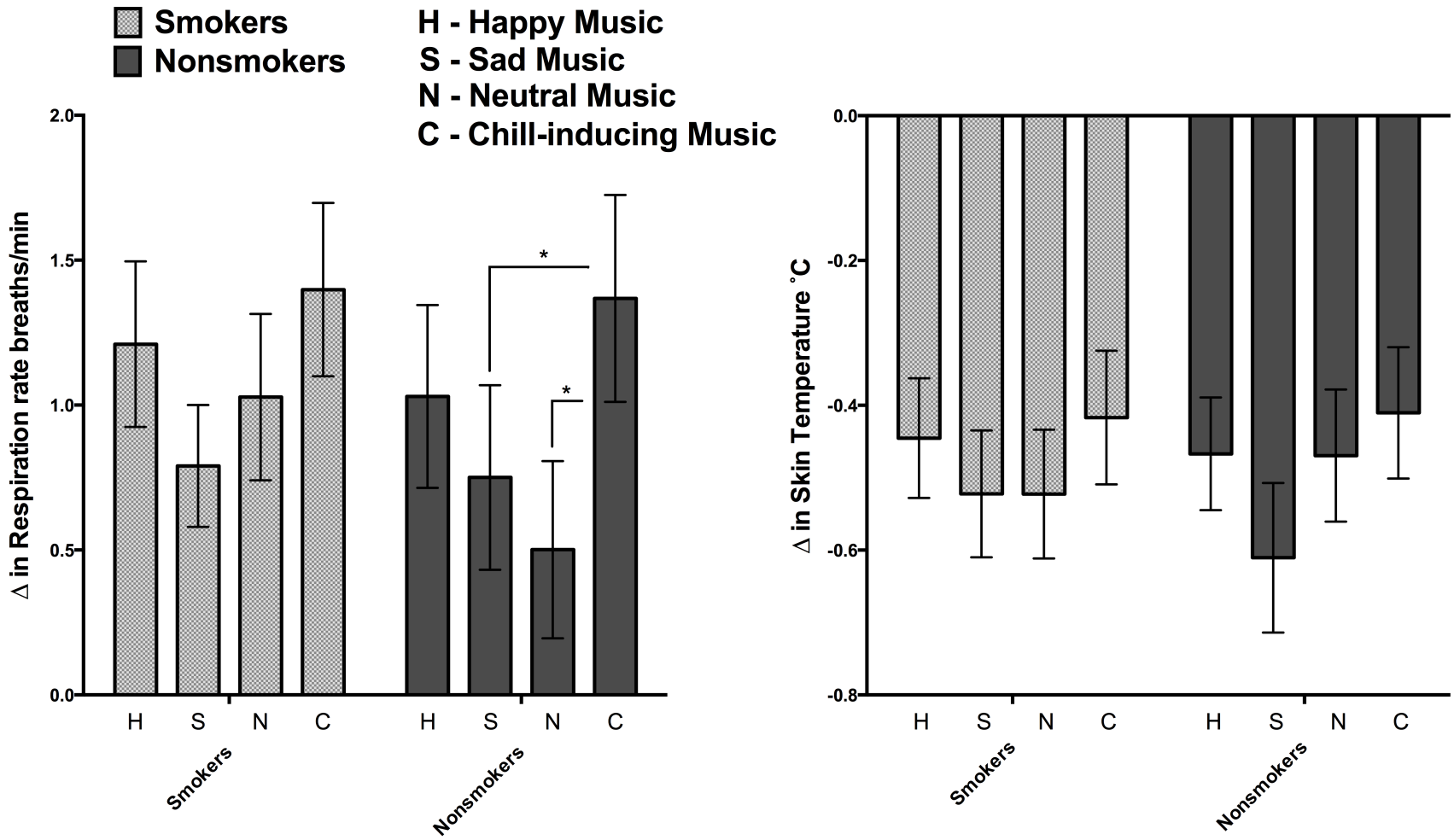

Figure 3.4. The mean and standard errors for smokers' and nonsmokers' respiration rate and skin temperature responses to each music condition. * $p < .05$, ** $p < .001$.

3.15. Effects of caffeine and music together on physiological arousal

The following section reports the interaction effect of caffeine and music on physiological responses, first between smokers and nonsmokers (section 3.15.1), then on smokers (3.15.2), and finally on nonsmokers (3.15.3).

3.15.1. Effects of caffeine and music together on physiological arousal between smokers and nonsmokers

A multivariate test revealed a nonsignificant interaction effect between caffeine, music, and smoking status on physiological arousal, $F(24, 162) = .51$, $p = .972$, $\eta 2 = .07$. Therefore, there was no difference between smokers' and nonsmokers' physiological responses to the interaction effect of caffeine and music. Although nonsignificant, Figure 3.5 through Figure 3.8 show different physiological effects of caffeine and music between smokers and nonsmokers. For example, in smokers, HR only increased as a result of nicotine for chill-inducing music, while systematic decreases were found for all other music conditions. However, in nonsmokers, nicotine resulted in an increase in HR for all music types, which was systematic for neutral and sad music. For SCL responses, both cohorts showed an overall increase during happy and chill-inducing music in response to nicotine. This increase was systematic for smokers only. SCL responses during sad and neutral music were more varied. Smokers showed a systematic increase in SCL during sad music, while nonsmokers showed a general decrease. In smokers, SCL showed a decrease at the 200 mg nicotine dose during neutral music, but an increase at the 400 mg dose. In nonsmokers, SCL decreased during neutral music. Fewer similarities can be seen between cohorts in respiration rate responses. However, both cohorts show nicotine to increase in respiration during chill-inducing music. In smokers, when compared to placebo, sad and neutral music show an increase in respiration at the 200 mg dose, but a negligible decrease at the 400 mg dose. In nonsmokers, nicotine resulted in a decrease in respiration during sad music and an increase during neutral music. In smokers, nicotine showed a negligible decrease in respiration during happy music, while smokers showed a systematic increase. For skin temperature, in general, both cohorts showed a decrease in response to nicotine. This effect was stronger at the 200 mg dose, with a few

exceptions. In smokers, sad music showed a systematic increase in response to nicotine and in nonsmokers, sad and neutral music showed a systematic decrease.

3.15.2. Effects of caffeine and music together on physiological arousal in smokers

Caffeine had broadly similar effects for each music type across the various physiological measures in smokers, as indicated by a nonsignificant multivariate interaction, $F(24, 82) = 1.33$, $p = .173$, $\eta 2 = .28$. Although there was no significant interaction effect of caffeine and music on the physiological responses of smokers, some trends can be seen for HR (Figure 3.5), SCL (Figure 3.6), respiration rate (Figure 3.7), and skin temperature (Figure 3.8). For example, all music types increased in HR in the placebo condition. Caffeine systematically increased HR for chill-inducing music, but systematically decreased it for all other music conditions. In the placebo condition SCL was increased for happy and chill-inducing music, but decrease for sad and neutral music. Caffeine systematically increased SCL for happy, sad, and chill-inducing music. However, 200 mg of caffeine decreased SCL during neutral music, but increased it during 400 mg. Respiration rate was increased for all music types in the placebo condition. Caffeine increased respiration rate during chill-inducing music and systematically decreased it during happy music. Furthermore, 200 mg of caffeine increased respiration rate for sad and neutral music, but 400 mg decreased it. Skin temperature was decreased for all types in the placebo condition. In general, caffeine further decreased skin temperature for all music types except sad music, which showed a systematic increase in skin temperature as caffeine dose increased.

3.15.3. Effects of caffeine and music together on physiological arousal in nonsmokers

Caffeine had broadly similar effects for each music type across the various physiological measures in nonsmokers, as indicated by a nonsignificant multivariate interaction, $F(24, 58) = .90$, $p = .607$, $\eta 2 = .27$. Although there was no significant interaction effect of caffeine and music on the physiological responses of nonsmokers, some trends can be seen for HR (Figure 3.5), SCL (Figure 3.6), respiration rate (Figure 3.7), and skin temperature (Figure 3.8).

For example, in the placebo condition HR increased during happy and chill-inducing music, but decreased during sad and neutral music. Caffeine increased HR for all music types. This increase was systematic for sad and neutral music, while happy and chill-inducing music saw a greater increase in HR at the 200 mg dose. For SCL, all music types were slightly increased in the placebo condition. Caffeine continued to increase SCL for happy and chill-inducing music, with a greater increase at 200 mg. However, for sad and neutral music caffeine decreased SCL, with a greater decrease at 200 mg. For respiration rate, all music types saw an increase in the placebo condition. Caffeine showed respiration to continue to increase during happy, sad, and neutral music. This increase was systematic for happy and neutral music, but not for chill-inducing music, which showed a greater increase in respiration at the 200 mg dose. Lastly, sad music showed a systematic decrease in respiration in response to caffeine. For skin temperature, all music types decreased at the placebo condition. Caffeine continued to decrease skin temperature. This decrease was systematic for sad music, but showed a greater decrease in skin temperature at the 200 mg dose during happy, neutral, and chill-inducing music.

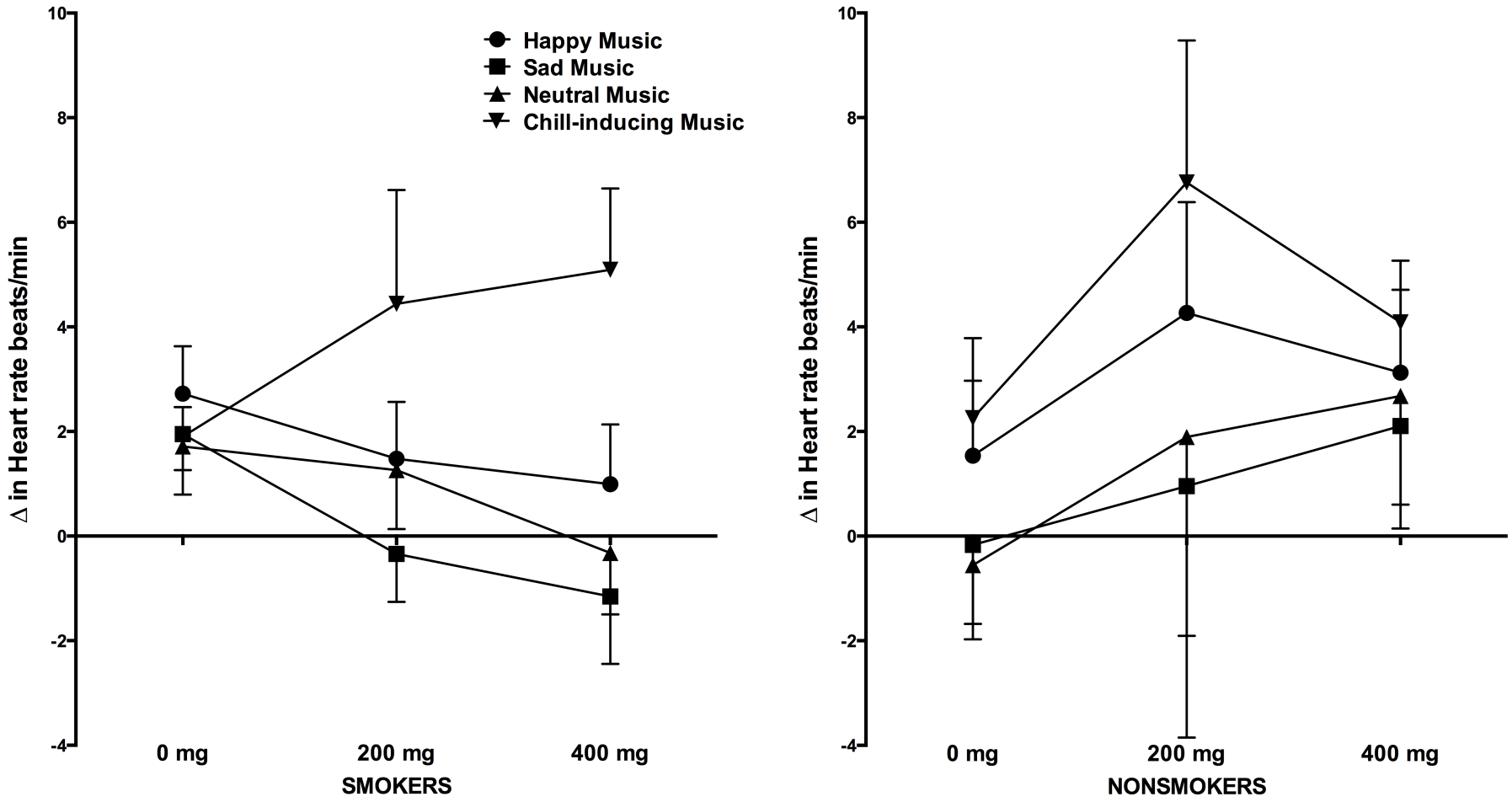

Figure 3.5. The mean and standard errors for smokers' and nonsmokers' heart rate responses to each caffeine condition for each music type. All comparisons are nonsignificant.

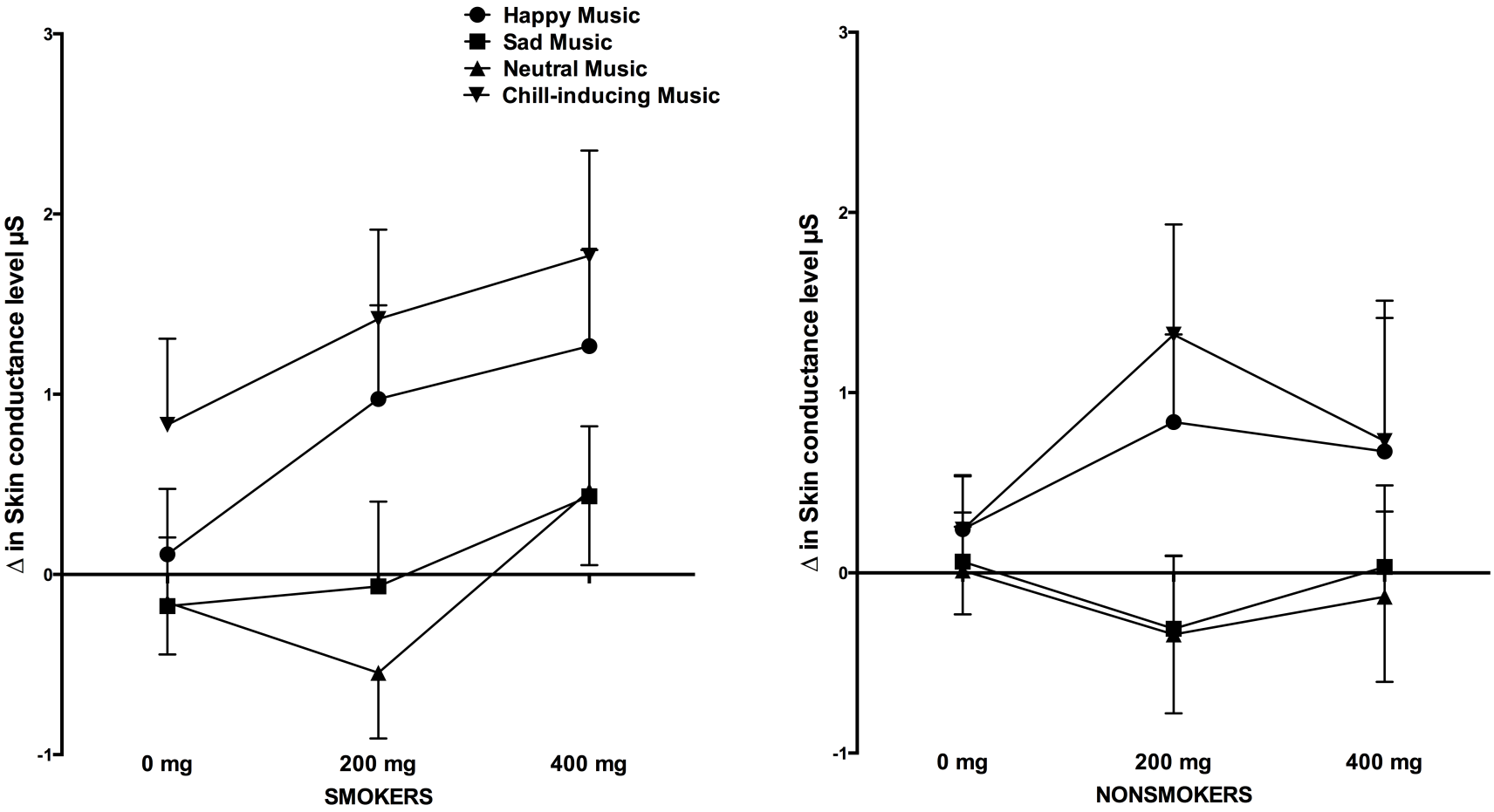

Figure 3.6. The mean and standard errors for smokers' and nonsmokers' skin conductance level responses to each caffeine condition for each music type. All comparisons are nonsignificant.

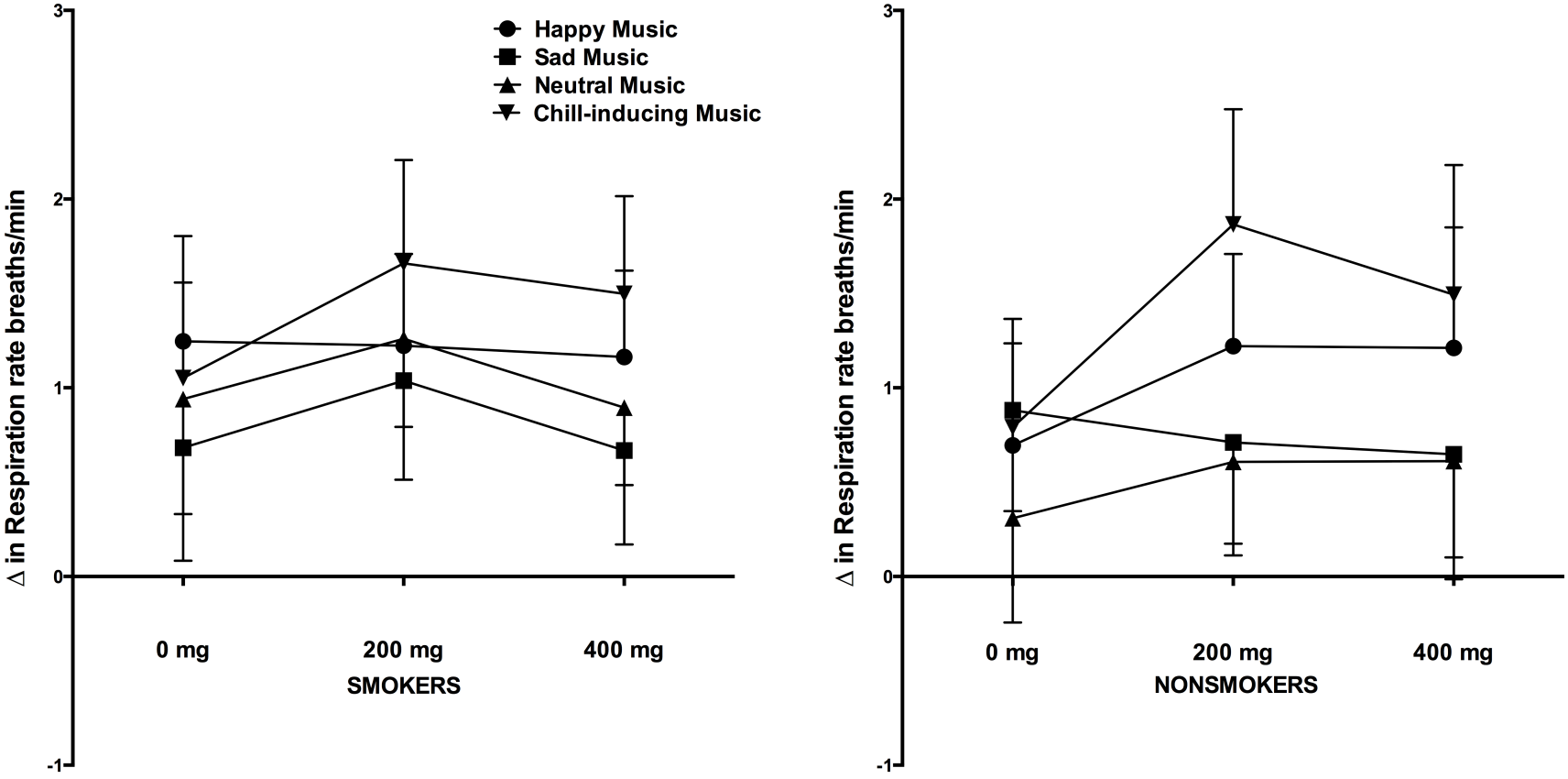

Figure 3.7. The mean and standard errors for smokers' and nonsmokers' respiration rate responses to each caffeine condition for each music type. All comparisons are nonsignificant.

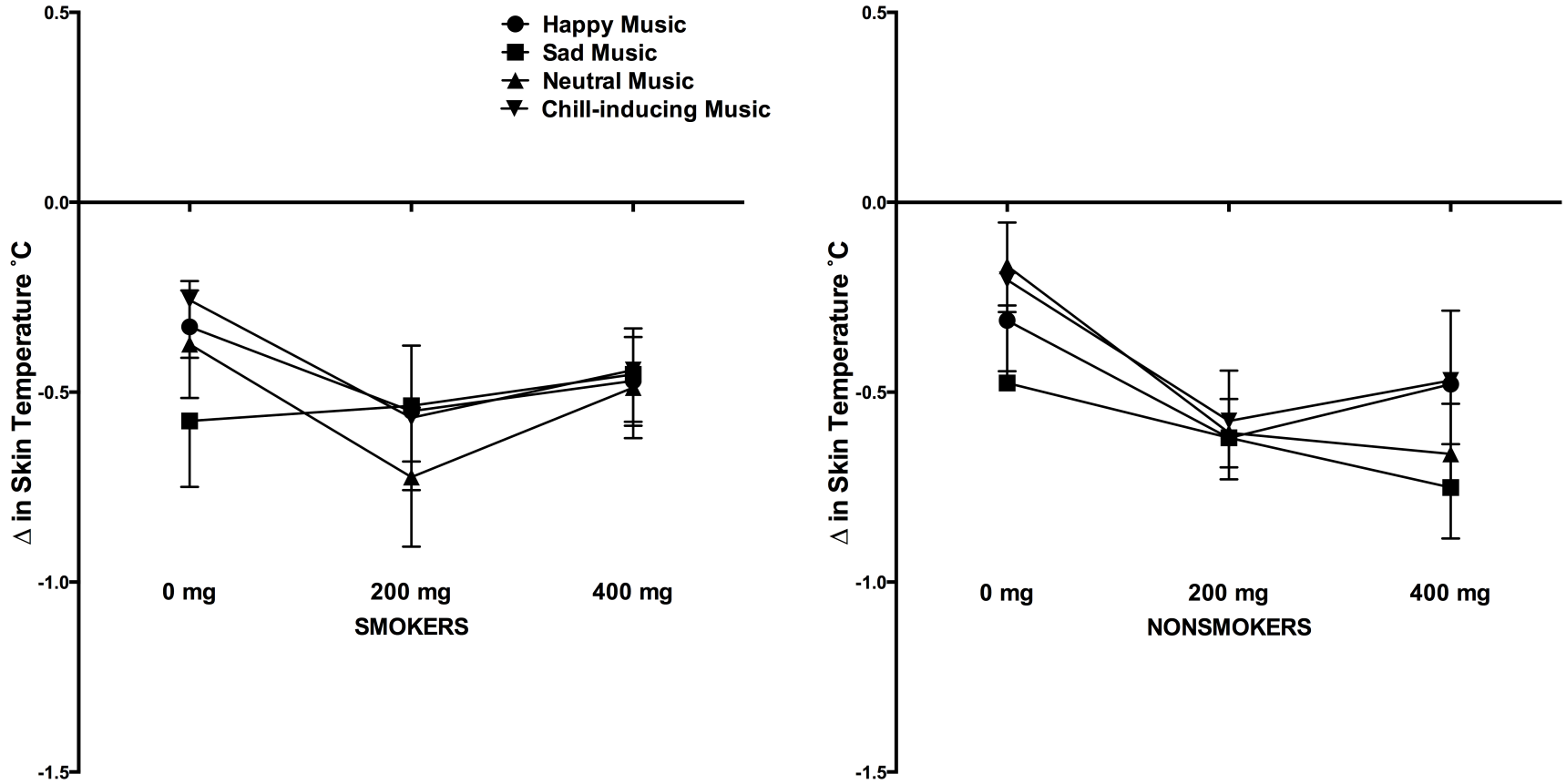

Figure 3.8. The mean and standard errors for smokers' and nonsmokers' skin temperature responses to each caffeine condition for each music type. All comparisons are nonsignificant.

3.16. Effects of caffeine on self-reported pleasure, arousal, and emotion

The analysis involving self-reported responses is also divided into 3 sub-sections: a main effect of nicotine (section 3.16), a main effect of music (section 3.17), and an interaction effect of nicotine and music (section 3.18). After computing change scores for smokers' self-reports, some variables were skewed and kurtotic. Therefore, in ratings of arousal I removed one outlier from neutral music. From ratings of pleasure I removed one outlier each from happy, neutral, and chill-inducing music. From ratings of sadness I removed three outliers from sad music. All subsequent analyses involving this variable were conducted with this outlier removed.

3.16.1. Effects of caffeine on self-reports between smokers and nonsmokers

A multivariate test indicated a nonsignificant difference between smokers' and nonsmokers' self-reported responses to caffeine, $F(4, 104) = .62$, $p = .652$, $\eta 2 = .02$. That is, in response to caffeine there was no significant difference between smokers' and nonsmokers ratings. Although nonsignificant, cohort comparisons can be seen in Figure 3.9 and Figure 3.10, which show some differences between smokers and nonsmokers. For example, in ratings of arousal and pleasure smokers showed a systematic increase, while nonsmokers showed a systematic decrease. However, caffeine resulted in a decrease in ratings of sadness for both cohorts.

3.16.2. Effects of caffeine on self-reports in smokers

A multivariate test indicated a nonsignificant effect of caffeine on self-reports in smokers, $F(8, 100) = 1.12$, $p = .358$, $\eta 2 = .08$. That is, for smokers, there was no significant difference between caffeine conditions in regards to self-reported ratings. However, trends can be seen for each rating in smokers, as shown in Figure 3.9 and Figure 3.10. For example, as caffeine dose increased, 1) arousal, pleasure, and happiness ratings systematically increased and 2) sadness ratings decreased, with negligible differences between the 200 and 400 mg conditions.

3.16.3. Effects of caffeine on self-reports in nonsmokers

A multivariate test shows a nonsignificant effect of caffeine on self-reports in nonsmokers, $F(8, 102) = 1.28$, $p = .264$, $\eta 2 = .09$. That is, for smokers, there was no significant difference between caffeine conditions in regards to self-reported ratings. However, trends can be seen for each rating in smokers, as shown in Figure 3.9 and Figure 3.10. For example, as caffeine dose increased, 1) arousal and pleasure ratings systematically decreased, 2) happiness ratings decreased at the 200 mg dose, but increased at the 400 mg dose and 3) sadness decreased, and more so at the 2 mg caffeine dose.

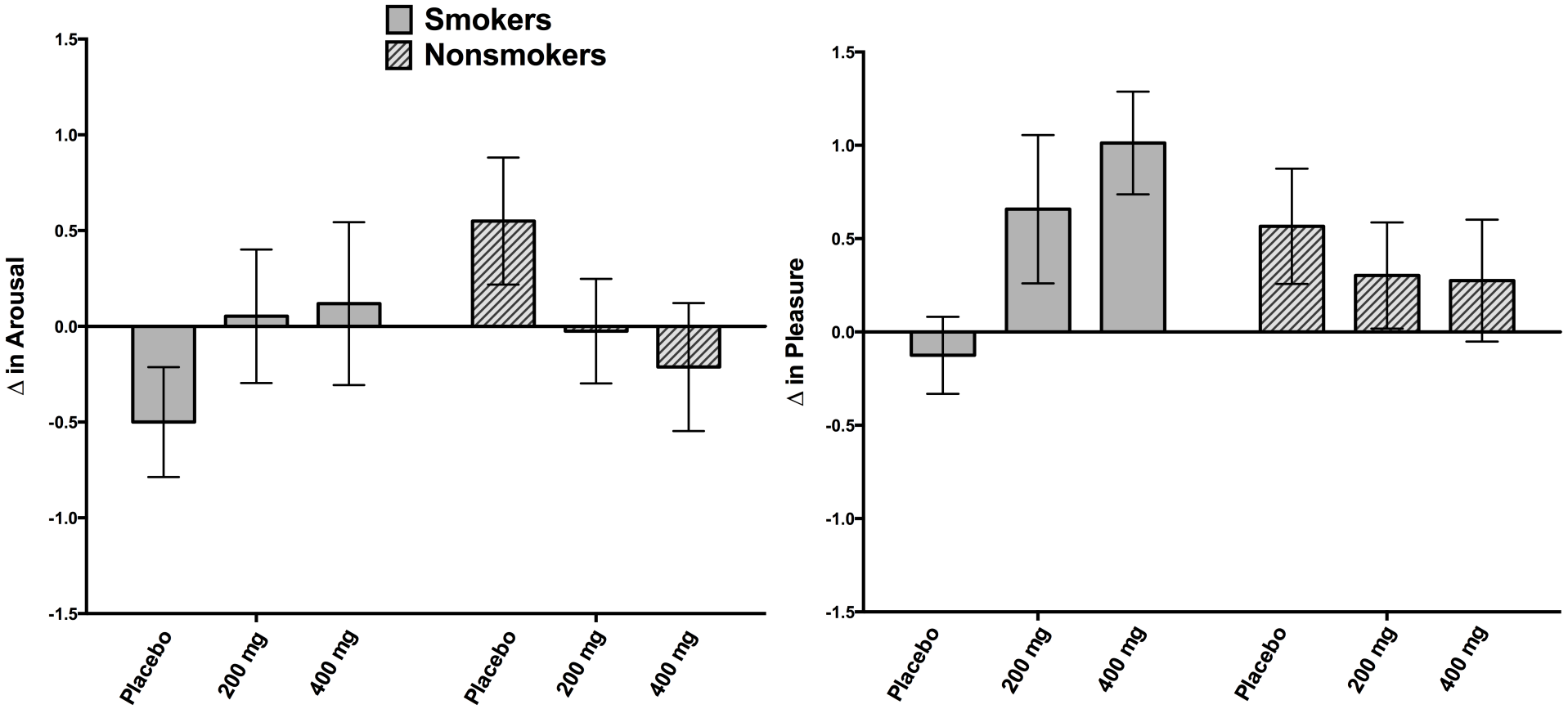

Figure 3.9. The mean and standard errors for smokers' and nonsmokers' ratings of arousal and pleasure to each caffeine condition.

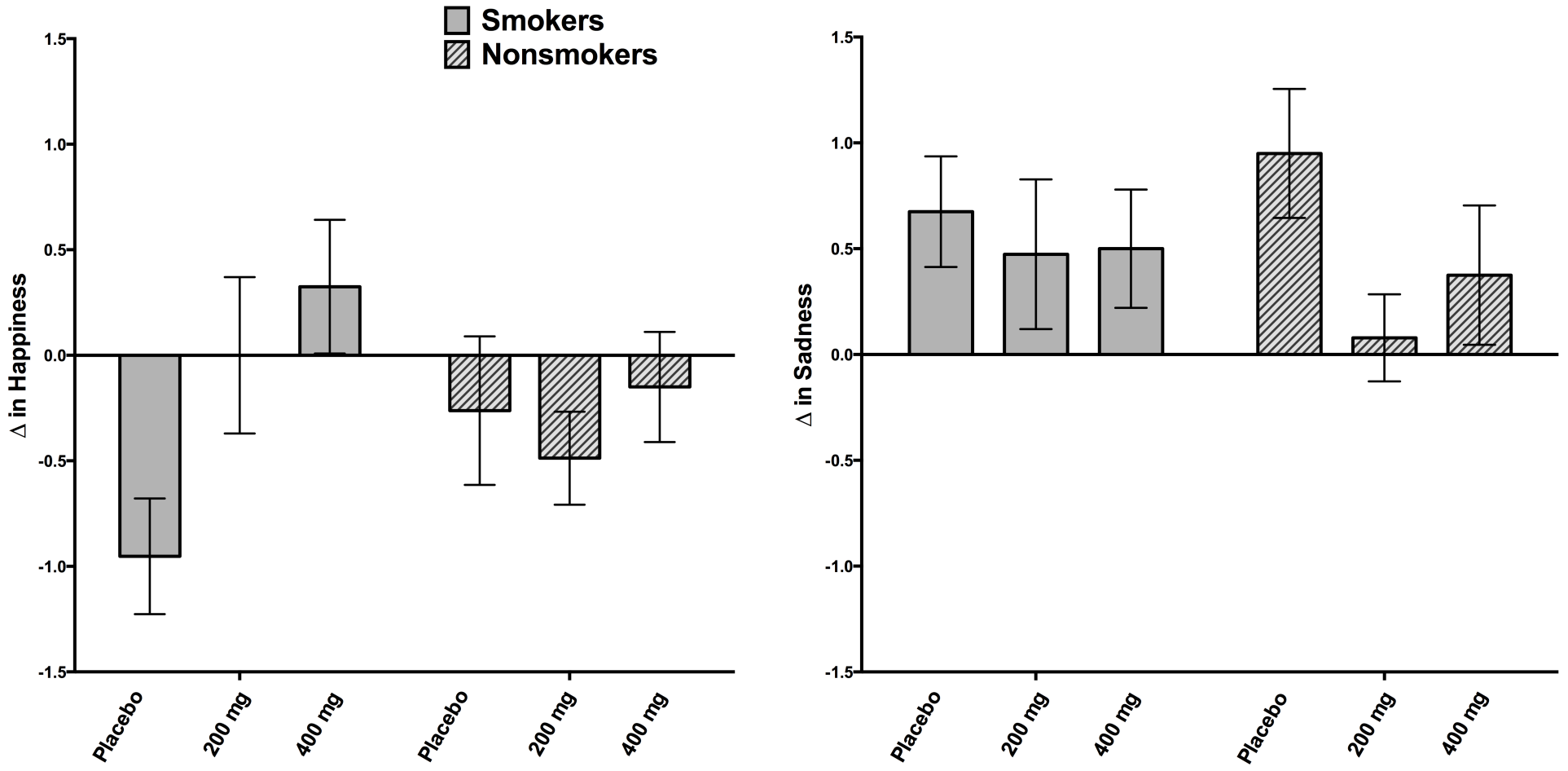

Figure 3.10. The mean and standard errors for smokers' and nonsmokers' ratings of happiness and sadness to each caffeine condition.

3.17. Effects of music on self-reports

The following section reports the main effect of music on self-reported ratings of arousal, pleasure, happiness, and sadness, first between smokers and nonsmokers (section 3.17.1), then on smokers (3.17.2), and lastly on nonsmokers (3.17.3).

3.17.1. Effects of music on self-reports between smokers and nonsmokers

A multivariate test showed a nonsignificant difference between smokers' and nonsmokers' self-reported responses to music, $F(12, 96) = .62$, $p = .825$, $\eta 2 = .07$. This is reflected by the similarities seen between cohorts in Figure 3.11 and Figure 3.12. For example, in both cohorts, ratings of arousal, pleasure, and happiness increased during happy and chill-inducing music, but decreased during sad and neutral music. Ratings of sadness also showed similar responses between smokers and nonsmokers. Happy music decreased in ratings of sadness, while all other music types increased. Furthermore, both cohorts show a pronounced increase in sadness during sad music.

3.17.2. Effects of music on self-reports in smokers

A multivariate test indicated a significant effect of music on self-reports in smokers, $F(12, 42) = 32.16$, $p < .001$, $\eta 2 = .91$. Univariate tests showed all self-reported ratings to significantly differ between music conditions, including arousal, $F(3, 159) = 33.86$, $p < .001$, $\eta 2 = .39$, pleasure, $F(3, 159) = 26.34$, $p < .001$, $\eta 2 = .33$, happiness, $F(3, 159) = 37.76$, $p < .001$, $\eta 2 = .38$, and sadness, $F(2.52, 133.44) = 23.57$, $p < .001$, $\eta 2 = .31$.

Pairwise comparisons showed that arousal was rated significantly higher during happy and chill-inducing music compared to sad and neutral music ($p < .001$). Similarly, pleasure was rated significantly higher during chill-inducing music compared to sad and neutral music ($p < .001$). Happy music was also rated significantly higher in pleasure compared to sad ($p = .001$) and neutral music ($p < .001$). Happiness was rated significantly higher during happy and chill-inducing music compared to sad and neutral music ($p < .001$). Sadness was rated significantly lower during happy music compared to during all other music conditions ($p < .001$). Furthermore, sadness was rated significantly higher during sad music compared to during neutral music ($p < .001$). Smokers'

self-reported responses to each music condition along with these pairwise comparisons are shown in Figure 3.11 and Figure 3.12.

3.17.3. Effects of music on self-reports in nonsmokers

A multivariate test indicated a significant effect of music on self-reports in smokers, $F(12, 43) = 27.51$, $p < .001$, $\eta 2 = .89$. Univariate tests showed all self-reported ratings to significantly differ between music conditions, including arousal, $F(3, 162) = 57.86$, $p < .001$, $\eta 2 = .52$, pleasure, $F(3, 162) = 30.18$, $p < .001$, $\eta 2 = .36$, happiness, $F(3, 162) = 38.42$, $p < .001$, $\eta 2 = .42$, and sadness, $F(3, 162) = 38.73$, $p < .001$, $\eta 2 = .42$.

Pairwise comparisons showed that arousal was rated significantly higher during happy and chill-inducing music compared to sad and neutral music ($p < .001$). Similarly, pleasure was rated significantly higher during happy and chill-inducing music compared to during sad and neutral music ($p < .001$). Again, happiness was rated significantly higher during happy and chill-inducing music compared to during sad and neutral music ($p < .001$). Sadness was rated significantly lower during happy music compared to during all other music conditions ($p < .001$). Furthermore, sadness was rated significantly higher during sad music compared to during all other music conditions ($p < .001$). Nonsmokers' self-reported responses to each music condition along with these pairwise comparisons are shown in Figure 3.11 and Figure 3.12.

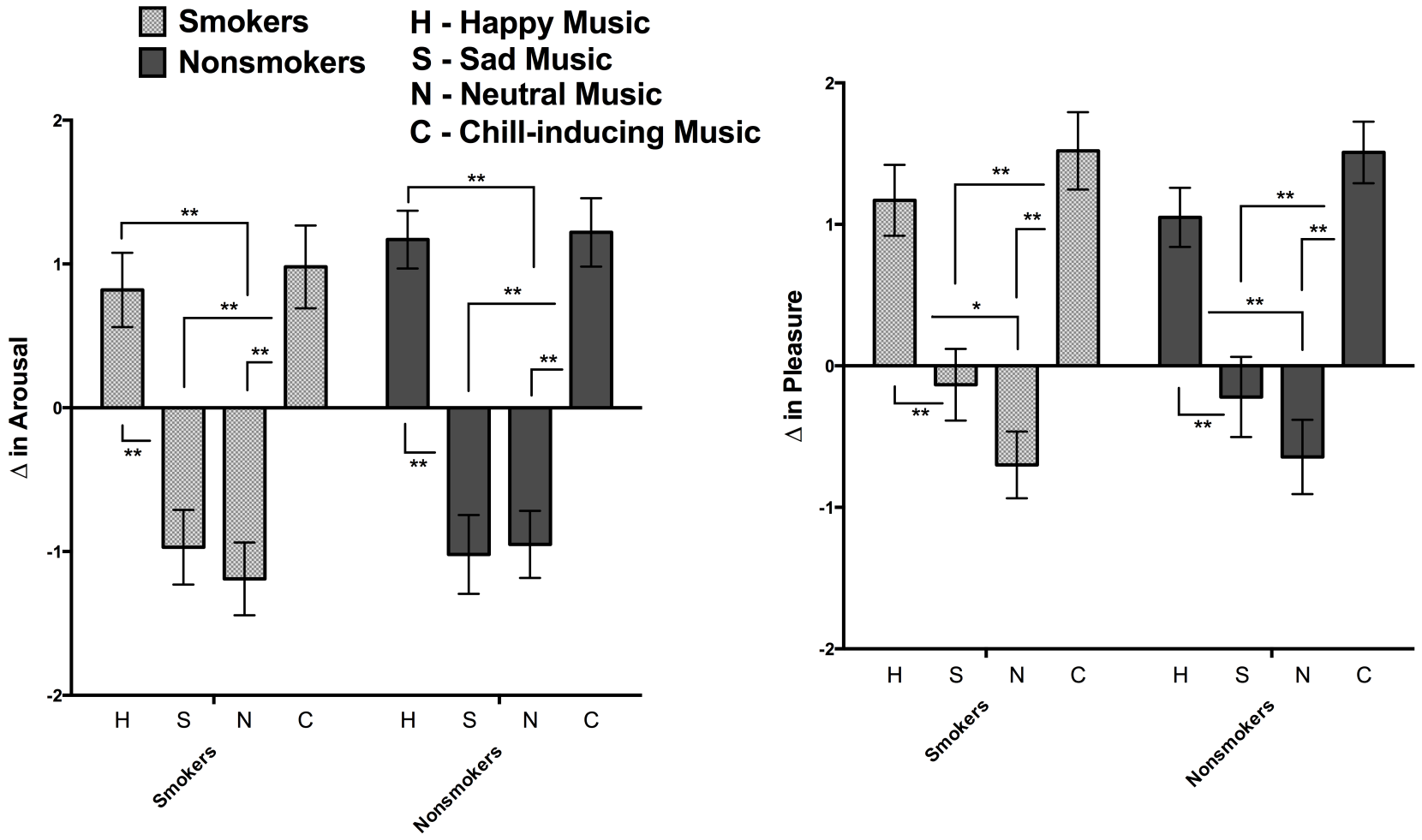

Figure 3.11. The mean and standard errors for smokers' and nonsmokers' ratings of arousal and pleasure for each music condition. * p < .05, ** p < .001.

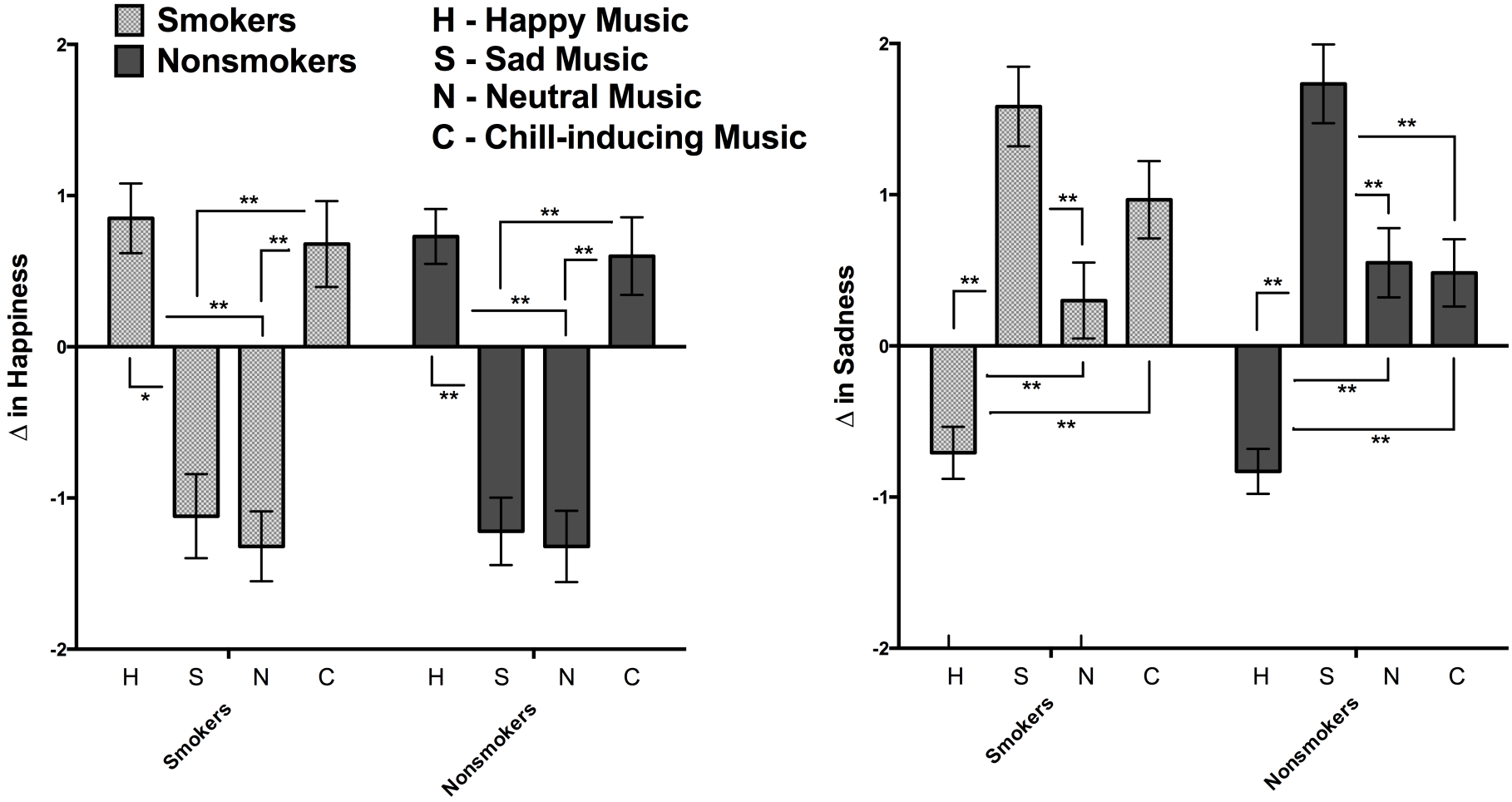

Figure 3.12. The mean and standard errors for smokers' and nonsmokers' ratings of happiness and sadness for each music condition. * $p < .05$, ** $p < .001$.

3.18. Effects of caffeine and music together on self-reports

The following section reports the interaction effect of nicotine and music on self-reported arousal, pleasure, and emotion, first between smokers and nonsmokers (3.18.1), then on smokers (3.18.2), and lastly on nonsmokers (3.18.3).

3.18.1. Effects of caffeine and music together on self-reports between smokers and nonsmokers

A multivariate tests revealed a nonsignificant interaction effect between caffeine, music, and smoking status, $F(24, 192) = 1.16$, $p = .286$, $\eta 2 = .13$. This indicated no difference between smokers' and nonsmokers' self-reports in response to the interaction of caffeine and music. This is reflected in Figure 3.13 through Figure 3.16, which shows that for smokers, caffeine systematically increased ratings of arousal, except for neutral music, which showed a greater increase at the 200 mg dose. However, in nonsmokers, caffeine generally decreased ratings of arousal. This was systematic for neutral music, while happy and chill-inducing music showed a greater decrease at the 200 mg dose. Sad music showed a negligible increase in arousal at 200 mg, but a decrease below placebo at 400 mg. For smokers, caffeine systematically increased ratings of pleasure during all music types except neutral music, which showed a greater increase at 200 mg. However, nonsmokers showed a systematic decrease in pleasure during neutral and chill-inducing music. Happy music showed a systematic increase in pleasure, while sad music showed negligible changes. For smokers, caffeine also systematically increased ratings of happiness during all music types except neutral music, which again showed a greater increase at 200 mg. For nonsmokers, again the responses were more varied. There was a systematic decrease in happiness during sad music and a systematic increase in happiness during chill-inducing music. However, neutral music showed a decrease in happiness, with a greater decrease at the 200 mg dose, while happy music showed a decrease at the 200 mg dose, but an increase above placebo at the 400 mg dose. For smokers, caffeine showed an overall decrease in sadness. This decrease was systematic only during sad music. Contrastingly, neutral music showed negligible changes at 200 mg and an increase at the 400 mg dose, while happy music showed an increase at 200 mg and decrease at 400 mg. While nonsmokers also showed a general

decrease in ratings of sadness across the music types, this effect was systematic for happy and chill-inducing music, but was most pronounced at the 200 mg dose for sad and neutral music.

3.18.2. Effects of caffeine and music together on self-reports in smokers

In general, caffeine has similar effects for each music condition across the four self-reported responses in smokers, as indicated by a nonsignificant multivariate interaction, $F(24, 84) = 1.26$, $p = .217$, $\eta 2 = .27$. Although there were no significant interaction effects of caffeine and music on smokers' self-reported ratings, trends can be seen for ratings of arousal (Figure 3.14), pleasure (Figure 3.15), happiness (Figure 3.16), and sadness (Figure 3.17). In the placebo condition, arousal ratings increased for happy and chill-inducing music, but decreased for sad and neutral music. Caffeine increased arousal ratings for all music types. This increase was systematic for sad and chill-inducing music, but was more pronounced at the 200 mg dose for happy and neutral music. In the placebo condition, pleasure ratings increased for happy and chill-inducing music, but decreased for sad and neutral music. Caffeine increase arousal and this increase was systematic for happy, sad, and chill-inducing music, but was more pronounced at the 200 mg dose for neutral music. In the placebo condition, happiness increased for happy and chill-inducing music, but decreased for sad and neutral music. Caffeine increased happiness and this increase was again, systematic for happy, sad, and chill-inducing music, but was more pronounced at the 200 mg dose for neutral music. Ratings of sadness showed little consistency between music types. For example, In the placebo condition, sadness increased for all music types except happy music, which decreased. As caffeine dose increased, ratings of sadness systematically decreased for sad music. For chill-inducing music caffeine decreased ratings of sadness, with negligible differences between the 200 nd 400 mg doses. Neutral music showed negligible differences in sadness between placebo and 200 mg of caffeine, but showed an increase in sadness at the 400 mg dose. Lastly, Happy music showed a slight increase in skin temperature at the 200 mg dose compared to placebo, but showed negligible changes in this measurement between the placebo and 400 mg conditions.

3.18.3. Effects of caffeine and music together on self-reports in nonsmokers

Caffeine also has similar effects for each music condition across the four self-reported in nonsmokers, as indicated by a nonsignificant multivariate interaction, $F(24, 86) = .88$, $p = .630$, $\eta 2 = .20$. Although there were no significant interaction effects of nicotine and music on nonsmokers' self-reported ratings, trends can be seen for ratings of arousal (Figure 3.13), pleasure (Figure 3.14), happiness (Figure 3.15), and sadness (Figure 3.16). In the placebo condition, ratings of arousal increased for happy and chill-inducing music and decreased for neutral and sad music. As nicotine dose increased ratings of arousal decreased. For happy and chill-inducing music there were negligible differences between the 200 and 400 mg. However, neutral music showed a systematic decrease in arousal as caffeine dose increased. Lastly, sad music showed a slight increase at the 200 mg dose compared to placebo, and a slight decrease at the 400 mg compared to placebo. In the placebo condition, pleasure ratings increased for happy and chill-inducing music, but decreased for sad and neutral music. As caffeine dose increased systematic decreases in pleasure were found for chill-inducing and neutral music. Contrastingly, happy music systematically increased. Lastly, sad music showed a slight increase in happiness at the 200 mg dose compared to placebo, and a slight decrease at the 400 mg compared to placebo. In the placebo condition, happiness increased for happy and chill-inducing music and decreased for sad and neutral music. As caffeine dose increased chill-inducing music systematically increased and neutral music systematically decreased. Furthermore, caffeine decreased happiness during neutral music, with a greater decrease at the 200 mg dose. For happy music, 200 mg of caffeine decreased happiness below placebo, while 400 mg increased happiness above placebo. In the placebo condition, sadness increased for all music types, except for happy music, which decreased. In general, caffeine decreased sadness ratings. This decrease was systematic for neutral and happy music. While for sad and chill-inducing music there was a greater decrease in sadness at the 200 mg dose.

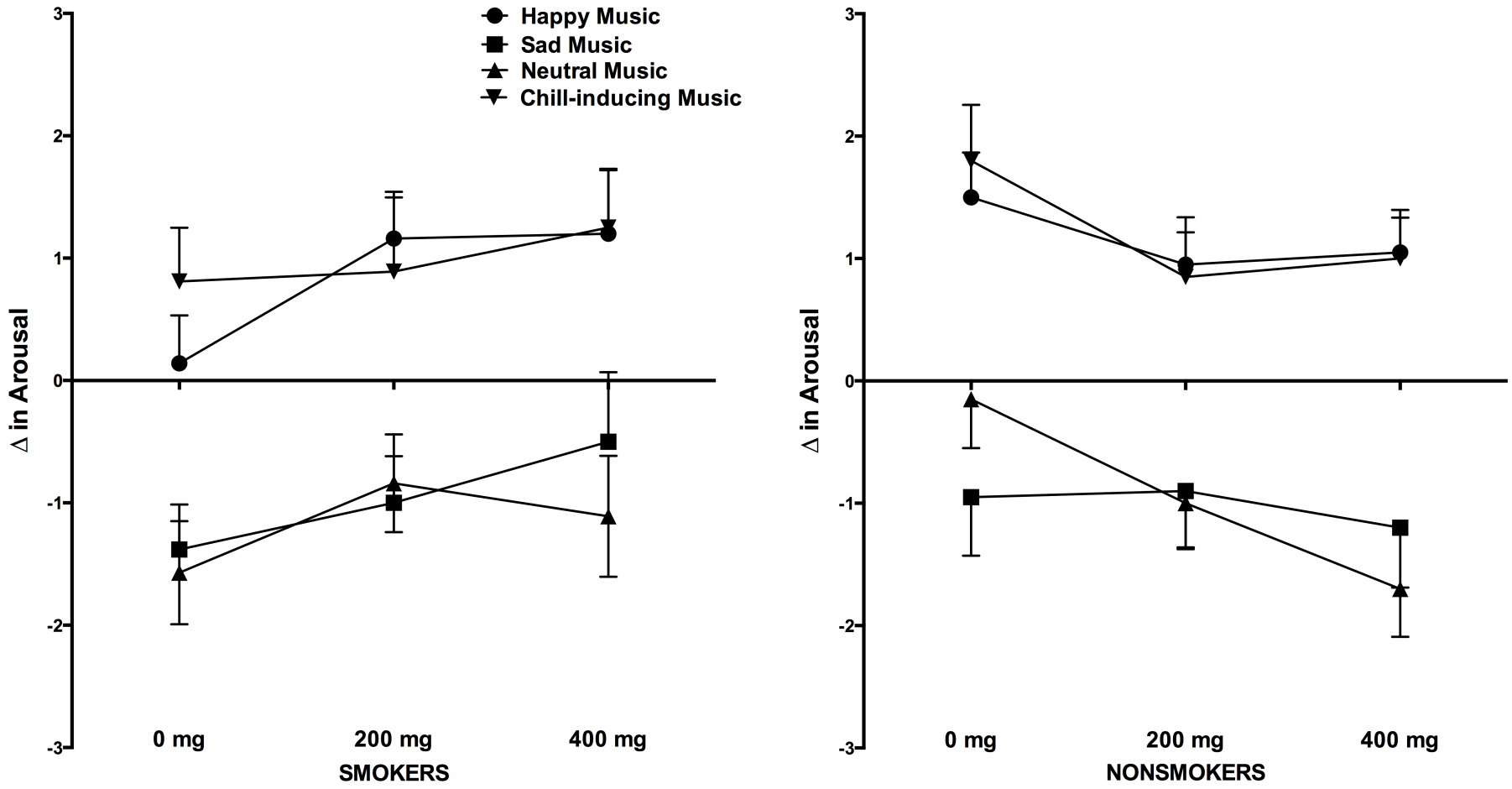

Figure 3.13. The mean and standard errors for smokers' and nonsmokers ratings of arousal to each caffeine condition for each music type. All comparisons are nonsignificant.

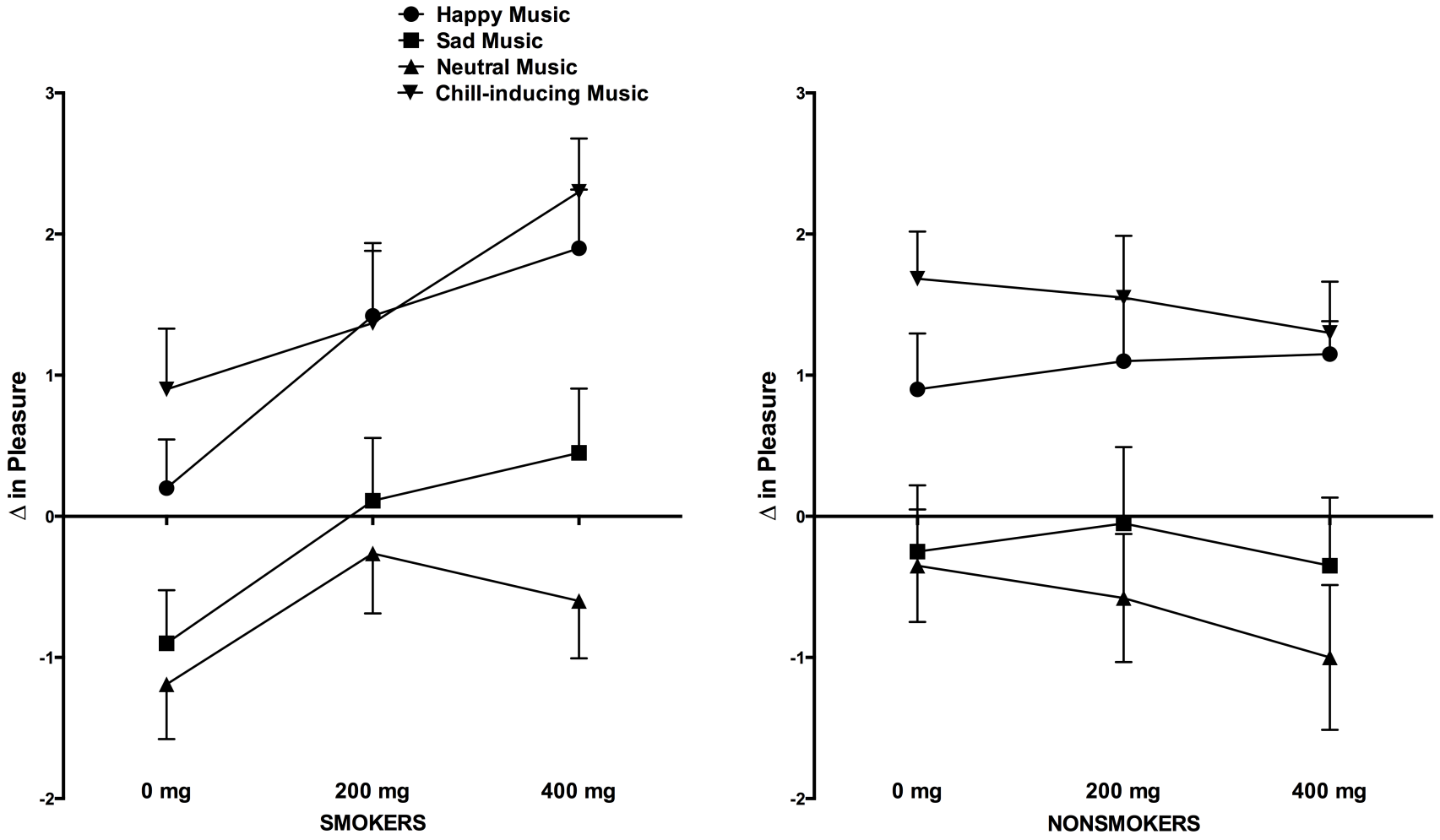

Figure 3.14. The mean and standard errors for smokers' and nonsmokers ratings of pleasure to each caffeine condition for each music type. All comparisons are nonsignificant.

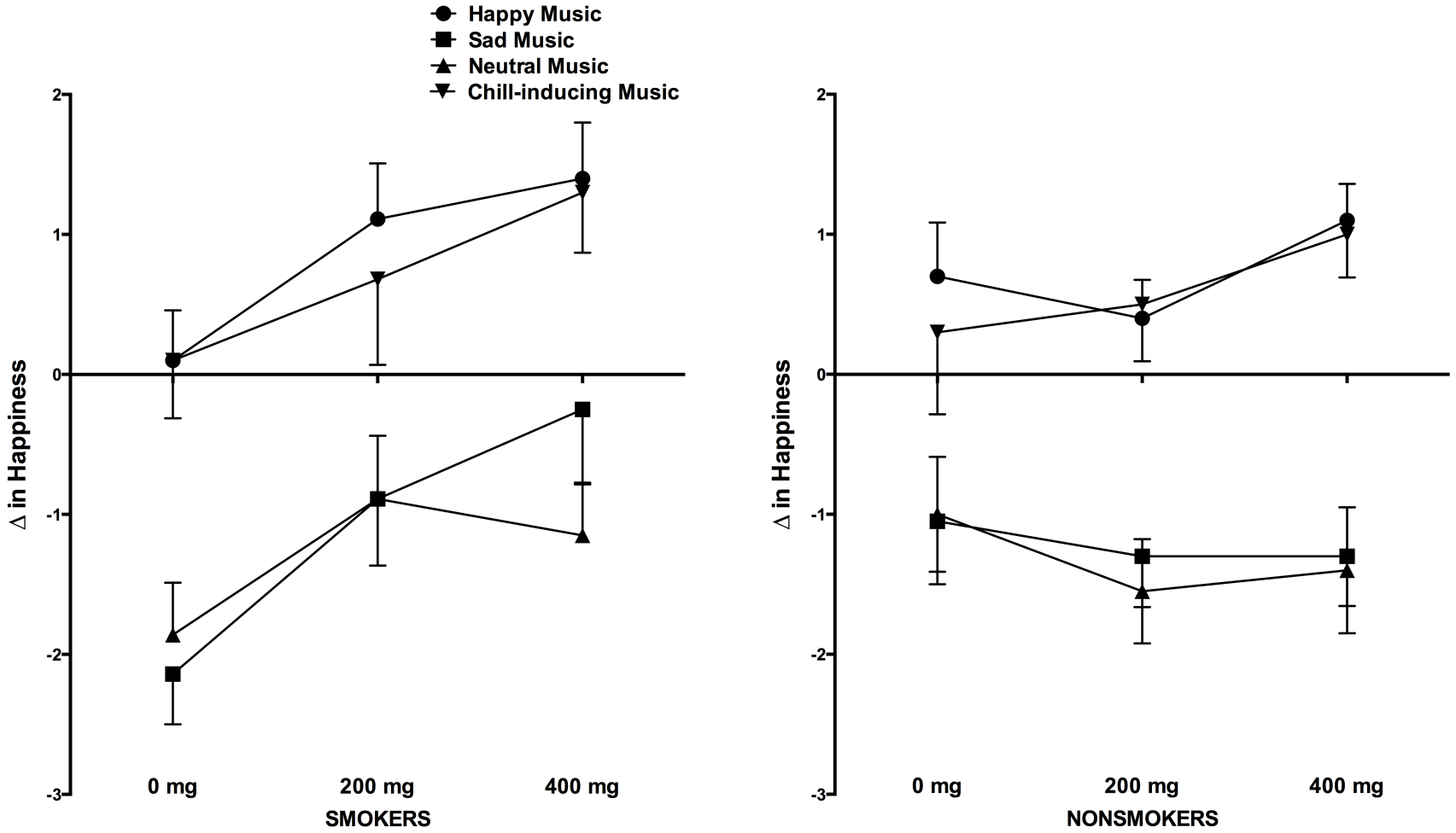

Figure 3.15. The mean and standard errors for smokers' and nonsmokers ratings of happiness to each caffeine condition for each music type. All comparisons are nonsignificant.

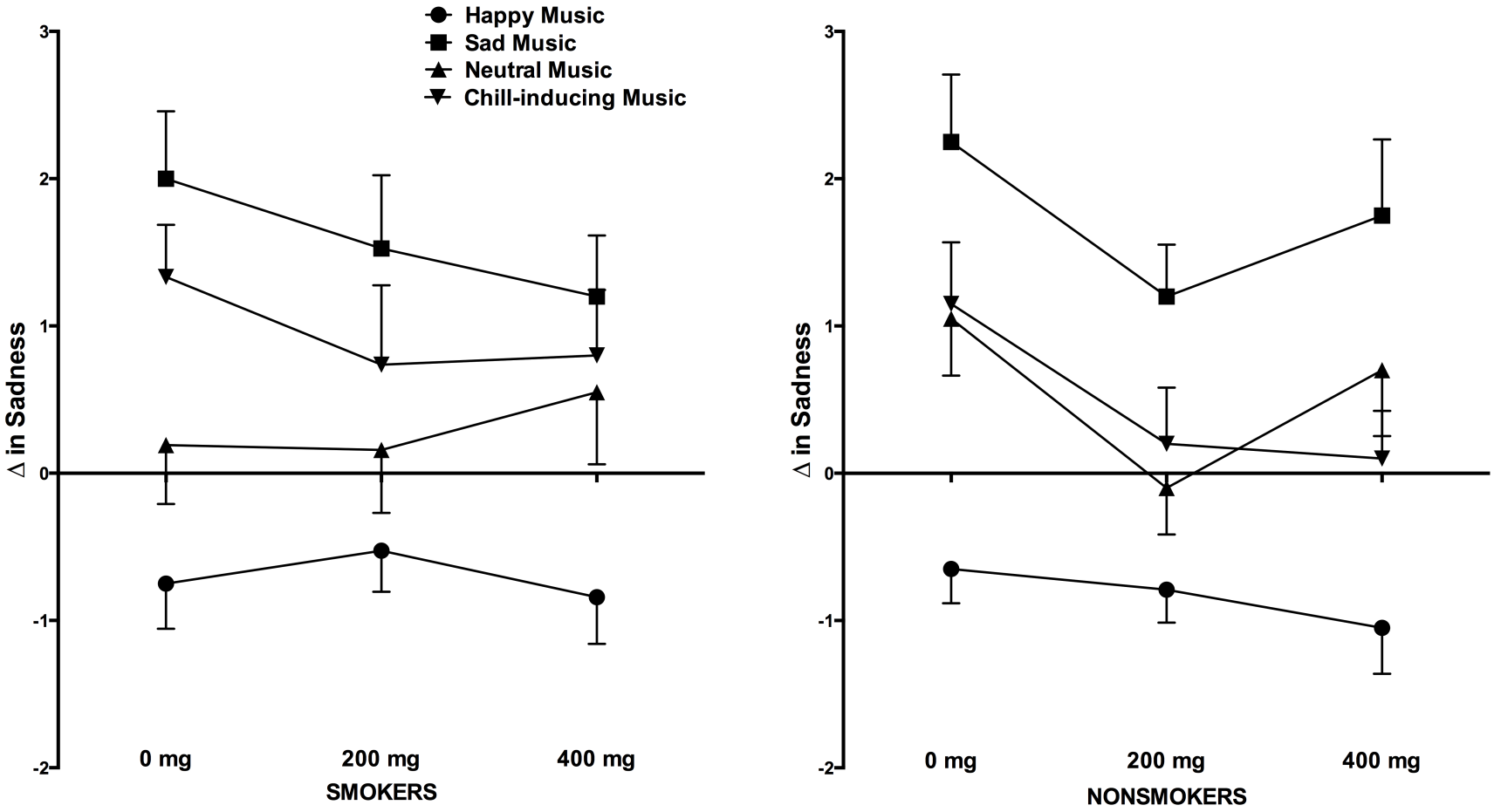

Figure 3.16. The mean and standard errors for smokers' and nonsmokers ratings of sadness to each caffeine condition for each music type. All comparisons are nonsignificant.

3.19. Discussion

The aim of this study was to determine if an additive effect on pleasure, arousal, or both occurred in response to the co-consumption of caffeine and music listening, and if so, to determine how much this effect can be explained by the phenomena of misattribution and excitation transfer. This was examined in order to determine how an increase in physiological arousal, without a manipulation of pleasure, affected music-induced emotion. Better understanding arousal's role in this exchange may help explain why nicotine and music listening often co-exist. I therefore examined the effects of caffeine on music-induced pleasure, arousal, (measured through physiological and self-reported indices of arousal) and emotion in abstaining smokers and nonsmokers. I administered 0, 200, and 400 mg of caffeine to participants and asked them to listen to the same four musical excerpts used in study one (see Appendix E for excerpt list). I then compared their physiological, pleasure/arousal ratings, and emotional responses between the varying caffeine and music conditions.

Because caffeine can increase physiological arousal, without influencing pleasure (Herz, 1999), I hypothesized that caffeine would result in an additive effect on these responses. I further hypothesized this effect to occur through caffeine's ability to increase physiological arousal, which, through the processes of misattribution and excitation transfer, would increase music-induced emotions.

3.20. Effects of caffeine on physiological arousal

All results for the effects of caffeine on physiological arousal were nonsignificant. Furthermore, there was no significant difference found between smokers and nonsmokers in regards to the effects of caffeine on physiology. However, some trends can be seen in the data. For example, as caffeine dose increased HR systematically decreased for smokers, but systematically increased for nonsmokers. However, as caffeine dose increased both cohorts showed a systematic increase in SCL. This increase was larger for smokers, particularly at the 400 mg dose compared to nonsmokers. Respiration rate also showed similar responses from smokers and nonsmokers, where by caffeine increased respiration compared to placebo, with a more pronounced effect at

the 200 mg dose. Caffeine resulted in a decrease in skin temperature compared to placebo for both cohorts. However, this decrease was more pronounced at the 200 mg dose for smokers and was systematic for nonsmokers.

These results are in general consistent with previous literature. Studies examining heart rate have found caffeine to both increase and decrease heart rate (Green et al., 1996), as well as increase for skin conductance (Zahn & Rapoport, 1987a, 1987b) and respiration rate (Sawyer et al., 1982), and decrease skin temperature (Quinlan et al., 2000), which help to explain the trends found in the current study. However, there is an inconsistency in HR responses between smokers and nonsmokers. There are also inconsistencies in the literature regarding how HR is affected by caffeine, suggesting this physiological response to be influenced by other factors than caffeine dose. For example, it may be that abstaining smokers and nonsmoker have different baseline HR, resulting in different HR responses to caffeine. This seems probably as the placebo conditions shows a higher HR for smokers than for nonsmokers. Overall, these results support the idea that caffeine can affect the physiological indices typical of arousal.

3.21. Effects of music on physiological arousal

Music significantly affected many, but not all physiological responses in smokers and nonsmokers. However, there were no significant difference found between smokers and nonsmokers in regards to the effects of music on physiology. This is reflected in the similar patterns of responses found between cohorts. For example, for both cohorts Happy and chill-inducing music increased HR, SCL, and respiration rate more than sad and neutral music. More specifically, for smokers, chill-inducing music significantly increased HR more than all other music conditions. For nonsmokers, chill-inducing and happy music significantly increased HR more than sad and neutral music. Similar results were found for SCL, showing that for smokers, chill-inducing music significantly increased SCL more than all other music conditions, and furthermore, happy music significantly increased SCL more than sad and neutral music. For nonsmokers, happy and chill-inducing music significantly increased SCL more than sad and neutral music. Respiration rate showed less significant results, but still had clear trends in the data. For nonsmokers, chill-inducing music

significantly increased respiration rate more than sad and neutral music. Although nonsignificant, there was also a trend showing happy music to be higher in respiration rate than sad and neutral music. For smokers there were no significant differences in the respiration rate responses, but there is a clear trend showing chill-inducing and happy music to be higher in respiration than sad and neutral music. For skin temperature there were no significant differences between music conditions, however, trends can be seen in the data. For smokers, sad and neutral music decreased skin temperature more than happy and chill-inducing music. For nonsmokers, sad and neutral music also decreased skin temperature more than happy and chill-inducing music, and this decrease was particularly pronounced for sad music.

These results are consistent with past research, which show happy and chill-inducing music to increase heart rate (Blood & Zatorre, 2001; Koelsch & Jäncke, 2015), skin conductance (Hodges, 2010), and respiration rate (Blood & Zatorre, 2001; Krumhansl, 1997), and decrease skin temperature (Krumhansl, 1997). These results demonstrate that music has a strong and consistent effect on the physiological indices of arousal.

A summary table comparing the effects of caffeine and music on physiological arousal is shown in Table 3.4. The results show music, as well as caffeine for nonsmokers to increase HR, while caffeine for smokers decreased HR. It also shows music and caffeine to both increase SCL and respiration rate, and to decreased skin temperature.

Table 3.4.

Summary table comparing the effects of caffeine and music on physiological arousal

Stimulus	Heart Rate		Skin Conductance Level		Respiration Rate		Skin Temperature	
Caffeine	↓ (S)	↑ (NS)	↑ (S)	↑ (NS)	↑ (S)	↑ (NS)	↓ (S)	↓ (NS)
Music	↑ *(S)	↑ *(NS)	↑ *(S)	↑ *(NS)	↑ (S)	↑ *(NS)	↓ (S)	↓ (NS)

Note: Arrows are shown for smokers (S) (↑) and nonsmokers (NS) (↑). Direction of arrow indicates an increase or decrease in response.
** Indicates significant differences between conditions. All other conditions show nonsignificant trends.*

3.22. Effects of caffeine on self-reports

All results for the effects of caffeine on self-reports were nonsignificant. Furthermore, there was no significant difference found between smokers and nonsmokers in regards to the effects of caffeine on self-reports. However, some trends can be seen in the data. For example, as caffeine dose increased ratings of arousal systematically increased for smokers, but systematically decreased for nonsmokers. Similarly, as caffeine dose increased ratings of pleasure systematically increased for smokers, but systematically decreased for nonsmokers. As caffeine dose increased ratings of happiness also systematically increased for smokers. However, for nonsmokers, 200 mg of caffeine decreased happiness compared to placebo, while 400 mg increased it compared to placebo. Sadness again showed similarities between cohorts. That is, caffeine decreased ratings of sadness for smokers and nonsmokers. However, for smokers, there were negligible differences in this decrease between the 200

and 400 mg conditions, while for nonsmokers, 200 mg of caffeine decreased sadness more than 400 mg of caffeine.

The trends found for smokers, although nonsignificant, are supported by previous research, showing caffeine to increase alertness and energy as well as positive subjective effects (Fredholm et al., 1999; Silverman & Griffiths, 1992). However, the trends found for nonsmokers, although also nonsignificant, suggest that caffeine decreases arousal and positive affect. Higher doses of caffeine (e.g. 200-800 mg) have been shown to produce negative effects, such as tense arousal (Penetar et al., 1993) and nervousness (Green & Suls, 1996). This may help explain why nonsmokers experienced a decreased in arousal, pleasure, and happiness. However, it does not explain the discrepancies found between cohorts. These discrepancies may have resulted from the fact that nonsmokers were not in a state of withdrawal and therefore did not experience a similar increase in self-reports to smokers. That is, because smokers were nicotine deprived they were in a state of withdrawal, characterized by low levels of alertness, a lack of hedonia, and an increase in anxiety and irritation (Epping-Jordan et al., 1998; West & Hajek, 2004). Therefore, stimulation experienced from caffeine may have had a more positive effect on the subjective state of smokers, causing them to regain alertness, hedonic capacity, and positive affect (e.g. the ability to feel aroused, pleasant, and happy despite their state of withdrawal) more than nonsmokers. Because abstaining smokers are in an abnormal state/mood it might also be that they are hypersensitive to their body state and as such more aware of the effects of caffeine on their arousal. If correct, this result might suggest that conscious awareness of arousal influences subjective emotional experiences.

3.23. Effects of music on self-reports

Music significantly affected all self-reported responses in smokers and nonsmokers. However, there were no significant difference found between smokers and nonsmokers in regards to the effects of music on these self-reports. This is reflected in the similar patterns of responses found between cohorts. For example, for both cohorts arousal, pleasure, and happiness were significantly increased for happy and chill-inducing music compared to sad and

neutral music. For both cohorts sadness was significantly higher for sad music and significantly lower for happy music compared to all other music conditions.

These results are corroborated with past studies that show music to strongly and reliably increase positive affect in listeners (Dubé & Le Bel, 2003; Zentner et al., 2008) and confirm music's ability to increase arousal and pleasure (Blood & Zatorre, 2001). Past research also shows sadness to be the most salient emotion experienced in response to sad music (Vuoskoski, Thompson, McIlwain, & Eerola, 2012).

A summary table comparing the effects of caffeine and music on self-reports is shown in Table 3.5. Smokers and nonsmokers showed opposing responses to caffeine in almost all self-reports. For example, smokers showed an increase in arousal, pleasure, and happiness, while nonsmokers showed a decrease in these measurements. However, both cohorts showed a decrease in sadness in response to caffeine. The cohorts showed more consistency in their self-reported responses to music. That is, both smokers and nonsmokers showed an increase in all self-reported measurements in response to music.

Table 3.5.

Summary table comparing the effects of caffeine and music on physiological arousal

Stimulus	Arousal		Pleasure		Happiness		Sadness	
Caffeine	↑	↓	↑	↓	↑	↓	↓	↓
	(S)	(NS)	(S)	(NS)	(S)	(NS)	(S)	(NS)
Music	↑	↑	↑	↑	↑	↑	↑	↑
	*(S)	*(NS)	*(S)	*(NS)	*(S)	*(NS)	*(S)	*(NS)

Note: Arrows are shown for smokers (S) (↑) and nonsmokers (NS) (↑). Direction of arrow indicates an increase or decrease in response.
** Indicates significant differences between conditions. All other conditions show nonsignificant trends.*

3.24. Effects of caffeine and music together on physiological arousal

HR was elevated for all music types in the placebo condition for smokers, while for nonsmokers HR was elevated for happy and chill-inducing music and decreased for sad and neutral music. For smokers, as caffeine dose increased HR systematically increased for chill-inducing music, but systematically decreased for all other music conditions. However, for nonsmokers, caffeine increased HR for all music conditions. This increase was most pronounced at the 200 mg dose for happy and chill-inducing music and was systematic for sad and neutral music. Although nonsignificant, this is indicative of an additive effect for smokers, but only during chill-inducing music. Although nonsignificant, this is also indicative of an additive effect for happy and chill-inducing music for nonsmokers, as these two music conditions increased at placebo, then further increased in response to caffeine.

For smokers, SCL was increased in the placebo condition for happy and chill-inducing music and decreased in the placebo condition for sad and neutral

music. As caffeine dose increased so did SCL for happy, sad and chill-inducing music. However, neutral music showed a decrease in SCL at the 200 mg dose and an increase at the 400 mg dose. For nonsmokers, SCL was increased in the placebo condition for all music conditions. Caffeine increased SCL for happy and chill-inducing music and was most pronounced at the 200 mg dose. However, caffeine decreased SCL for sad and neutral music and this was more pronounced at the 200 mg dose. Although nonsignificant, this is indicative of an additive effect for smokers and nonsmokers during happy and chill-inducing music.

For both cohorts, respiration rate was increased in the placebo condition for all music conditions. For smokers, caffeine increased respiration rate for chill-inducing music, more so at the 200 mg dose than at the 400 mg dose. As caffeine dose increased respiration rate systematically decreased during happy music, however respiration rate negligibly differed between placebo and caffeine conditions. For sad and neutral music, 200 mg of caffeine increased respiration rate, but 400 mg returned respiration rate to nearly the same levels seen in the placebo condition. For nonsmokers, caffeine increased respiration rate for all music types, except sad music. While this increase in respiration was systematic for neutral music, happy and chill-inducing music showed a greater increase in respiration rate at the 200 mg dose. As caffeine dose increased sad music showed a systematic decrease in respiration. Although nonsignificant, this is indicative of an additive effect for smokers during chill-inducing music, as well as for sad and neutral music, but only at the 200 mg dose. Although nonsignificant, this is indicative of an additive effect for nonsmokers during all music conditions except sad music.

Skin temperature was decreased for all music types in the placebo condition for both smokers and nonsmokers. Except for sad music, caffeine further reduced skin temperature in all music types for both cohorts, especially at the 200 mg dose. As caffeine dose increased, sad music showed a systematic increase in skin temperature for smokers, and a systematic decreased in skin temperature for nonsmokers. Although nonsignificant, this is indicative of an additive effect for all music conditions, except sad music, in both cohorts.

3.25. Effects of caffeine and music together on self-reported emotions (happiness/sadness)

In both cohorts, happiness was increased for happy and chill-inducing music and decreased for sad and neutral music in the placebo condition. For smokers, as caffeine dose increased, happiness systematically increased for all music types, except neutral music. Caffeine increased happiness in neutral music, but this was more pronounced at the 200 mg dose. For nonsmokers, as caffeine dose increased, happiness systematically increased during chill-inducing music, and systematically decreased during sad music. For happy music, 200 mg of caffeine decreased happiness below placebo levels, while 400 mg increased happiness above placebo levels. For neutral music caffeine decreased happiness, and this was more pronounced at the 200 mg dose. Although nonsignificant, this is indicative of an additive effect for happy and chill-inducing music in smokers, and for chill-inducing music in nonsmokers.

In both cohorts, sadness was increased in the placebo condition for all music types, except happy music. For smokers, as caffeine dose increased, ratings of sadness systematically decreased for sad music. Caffeine also decreased sadness during chill-inducing music, but this was slightly more pronounced at the 200 mg dose. For neutral music, 200 mg of caffeine negligibly decreased sadness, while 400 mg increased it above placebo. For happy music, 200 mg of caffeine increased sadness, while 400 mg decreased it. For nonsmokers, caffeine decreased sadness in all music types. This decrease was more pronounced at the 200 mg dose during sad and neutral music, and this decrease was systematic for chill-inducing and happy music. This is only indicative of an additive effect on sadness for nonsmokers during happy music.

3.26. Effects of caffeine and music together on self-reported arousal/pleasure

For both cohorts, in the placebo condition arousal was increased for happy and chill-inducing music and decreased for sad and neutral music. For smokers, as caffeine dose increased, sad and chill-inducing music systematically increased in arousal ratings. Caffeine also increased arousal ratings in happy and neutral music, but this was more pronounced at the 200 mg dose. For nonsmokers, caffeine decreased ratings of arousal for almost all music conditions. For neutral music this decrease was systematic. For happy and chill-

inducing music there were negligible differences in this decrease between the 200 and 400 mg doses. Lastly, for sad music, 200 mg of caffeine negligibly increased arousal ratings, while 400 mg decreased it. Although nonsignificant, this is indicative of an additive effect for smokers during happy and chill-inducing music. This is also indicative of an additive effect for neutral music in nonsmokers, as nicotine further decreased arousal.

For both cohorts, in the placebo condition pleasure was increased for happy and chill-inducing music and decreased for sad and neutral music. For smokers, as caffeine dose increased pleasure systematically increased during all music conditions except neutral music. For neutral music, caffeine increased pleasure, but this was more pronounced at the 200 mg dose. For nonsmokers, as caffeine dose increased there was a systematic decrease in pleasure for chill-inducing and neutral music. There was also a systematic increase in pleasure during happy music, but these increases were negligible. For sad music, 200 mg of caffeine increased pleasure above placebo, while 400 mg slightly decreased pleasure below placebo. Although nonsignificant, this is indicative of an additive effect for smokers during happy and chill-inducing music. Also, this is indicative of an additive effect for nonsmokers during neutral music, as caffeine further decreased pleasure compared to placebo.

3.27. Summary

Although the results for interaction effects are nonsignificant there are some trends indicative of additive effects of caffeine and music on the physiological and self-reported responses. Physiological indices of arousal were clearly indicative of an additive effect of caffeine on HR for both cohorts, but only during chill-inducing music for smokers and only during happy and chill-inducing music in nonsmokers. There were also indications of an additive effect of caffeine on SCL during happy and chill-inducing music for both smokers and nonsmokers. Respiration rate shows trends of additive effects for smokers during chill-inducing music, as well as at the 200 mg dose for sad and neutral music. Respiration rate also showed trends of additive effects for nonsmokers during happy, neutral, and chill-inducing music. For skin temperature there were trends of additive effects for both cohorts during happy, neutral, and chill-inducing music.

Self-reports also showed some trends of additive effects, mainly for happy and chill-inducing music. Self-reports were indicative of an additive effect of caffeine on ratings of happiness for smokers during happy and chill-inducing music, as well as for nonsmokers during chill-inducing music. Sadness showed a trend indicative of an additive effect for nonsmokers during happy music. Furthermore, arousal showed trends of an additive effect of caffeine for smokers during happy and chill-inducing music. Arousal also showed trends indicative of an additive effect of caffeine for nonsmokers during neutral music, as caffeine further decreased arousal ratings compared to placebo. Lastly, pleasure showed trends of an additive effect of caffeine for smokers during happy and chill-inducing music. Also, pleasure showed trends of an additive effect of caffeine for nonsmokers during neutral music, as caffeine further decreased pleasure compared to placebo.

3.28. Misattribution and excitation transfer

In regards to misattribution and excitation transfer, it seems that the first step necessary to induce these phenomena is an increase in physiology via caffeine administration. Although nonsignificant, this was clearly the case as both smokers and nonsmokers shows trends indicative of additive effects in all four physiological measures, particularly for happy and chill-inducing music, and less frequently for sad and neutral music. The second step necessary to confirm misattribution and excitation transfer is a subsequent increase in self-reported responses. There were trends indicative of additive effects on self-reported responses in both cohorts (e.g. happiness ratings), however, these trends were much more apparent for smokers than nonsmokers (e.g. happiness, arousal, and pleasure ratings). This suggests that smokers experienced excitation transfer from caffeine to music-induced emotion more so that nonsmokers. This may be attributed to the nicotine-abstaining state experienced by smokers. That is, it is common for those in a state of nicotine withdrawal to experience an increase in negative emotion (Hughes et al., 1994; West & Hajek, 2004) and a decrease in positive emotion (Epping-Jordan et al., 1998; Tomkins, 1966). Therefore, it is likely that smokers were experiencing sub-baseline measures of arousal, pleasure, and emotion. In turn, caffeine increased the physiological arousal of both cohorts, but this increase in physiology only resulted in an

excitation transfer in music-induced emotion for those who started at sub-baseline levels (e.g. smokers). Perhaps then, smokers misattributed their increase in physiological arousal to their emotional responses to music, while nonsmokers attributed this to caffeine, and in turn this resulted in an increase in music-induced emotions for smokers, but not nonsmokers.

Interestingly, trends indicative of additive effects on self-reports were only seen in positively valenced measures (e.g. ratings of happiness and pleasure). More specifically, sadness did not show any trends of additive effects for smokers or nonsmokers. These results are similar to previous findings by Cantor and Zillmann (1973), who found a highly arousing film to intensify positive responses to music. However, Dibben (2004) found in study one that exercise increased the dominant valence of the emotional response, while study two found exercise to intensify positive emotions for musical excerpts that were positive in valence. This study adds to previous literature by suggesting that an increase in physiology induced by caffeine can potentially cause positive emotions to be amplified by positively valenced music, and that negatively valenced emotions are unaffected by this increase in physiology. It could be that the negatively valenced response monitored in this study (sadness) is an emotion that is more expressed, than felt in music. More specifically, it could be that the intensity at which sadness is expressed by music is greater than that felt by listeners (Kawakami, Furukawa, Katahira, & Okanoya, 2013) or it could be that some form of positive emotions (e.g. enjoyment, pleasure) are experienced in response to sad music (Huron, 2011). In fact, as caffeine increased, smokers' ratings of pleasure increased during sad music, and an increase in pleasure was also found for nonsmokers during sad music, but only at the 200 mg dose. However, the additive effects found in this study are trends only (nonsignificant) and this study did not measure expressed emotion. Therefore, further research is needed to confirm these additive effects on positively valenced self-reports, both statistically and empirically.

3.29. Limitations and future research

Although this study also had a small sample size (~20 participants per condition), as did study 1, the number of participants was not changed compared to study 1 in order to keep the design exactly the same. This made

the results between study 1 and study 2 directly comparable. Therefore, the main limitation to this study was that caffeine status was not controlled for in participants. Although all participants did abstain from caffeine, as well as nicotine and alcohol for 24 hours, caffeine consumption was not controlled for in order to make the results of the current study directly comparable to the previous study. Participants' varying tolerance and dependence levels associated with frequent caffeine use may have influenced the results (Smith, 2002). Future research should account for caffeine consumption in participants as varying levels of tolerance can potentially result in less physiological and self-reported responses.

The discrepancy in HR responses to caffeine found between smokers and nonsmokers, as well as the inconsistencies found throughout the literature, warrants further research as to the effects of caffeine on physiology. This is needed in order to determine whether smoking status influences HR at baseline or only during caffeine consumption. Future research may be interested in account for caffeine consumption in order to examine how HR response different between smoking cohorts.

Future studies may also be interested in the cognitive mechanisms that influence the enhancement of arousal on music-induced emotion. That is, stimulants such as caffeine and nicotine affect the CNS and the auditory pathway (Crawford, McClain-Furmanski, Castagnoli, & Castagnoli, 2002; Dixit, Vaney, & Tandon, 2006). This may enhance auditory perception through the excitation of the auditory pathway. In turn, this may lead to an enhancement of music-induced emotion. Therefore, better understanding the cognitive mechanisms underlying nicotine's ability to enhance music-induced emotion may help explain why nicotine and music are often co-consumed. This could be accomplished through an electrophysiological study, which is able to test the speed at which auditory information in processed and therefore confirm whether nicotine is able to enhance auditory perception.

4. Chapter four: Effects of nicotine on auditory perception

4.1. Overview and rationale of study 3

In studies one and two the effects of nicotine and caffeine on music-induced emotions were assessed through physiological measurements and self-reports. In general, findings revealed that nicotine and caffeine in combination with music had additive effects on physiological arousal, as well as self-reports of arousal, pleasure, and emotion. However, we do not know which cognitive mechanism(s) are responsible for this enhancement. Previous research has established that cholinergic systems are important for cognitive functioning and that nicotine is a potent cholinergic stimulant that affects many of the central nervous system (CNS) pathways, including the auditory pathway (Crawford et al., 2002). This means that the receptors of the auditory pathway are cholinergic and therefore activated by acetylcholine (Ach). Because nicotine mimics the actions of Ach it can therefore excite the auditory pathway. This suggests that nicotine is somehow able to increase arousal, and in addition, may be able to enhance auditory perception through the excitation of the auditory pathway. This in turn could potentially enhance listeners' music-induced emotions.

Previous research examining the effects of nicotine commonly measure task performance and have reported improvements in attention, learning, reaction time (RT), problem solving, and stimulus evaluation and discrimination (Heishman et al., 1994; Le Houezec & Benowitz, 1991; Wesnes & Warburton, 1983). However, some studies have found dose-related decreases in performance and attention tasks, such as visual scanning in nonsmokers (Heishman & Henningfield, 2000) or have found no effect of nicotine on attentional switching in smokers (Mancuso, Warburton, Mélen, Sherwood, & Tirelli, 1999). Although nicotine-enhanced arousal and attentional functions are thought to underlie behavioral improvements (Knott et al., 2011) these inconsistent results suggest that further research is needed to clarify under

which circumstances nicotine can increase cognitive and behavioral performance.

Several neuroscientific studies investigating nicotine's effect on auditory perception have confirmed nicotine's ability to enhance arousal and attention using functional magnetic resonance imagining (fMRI) (Smucny, Olincy, Eichman, & Tregellas, 2015; Thiel & Fink, 2007) and magnetencephalography (MEG) (Otsuru et al., 2012). These methods have helped identify and localize the brain structures and neural-networks involved in arousal and attention. Electrophysiological techniques (e.g. EEG, ERP) have also supported nicotine's role as an enhancer of arousal and attention (Harkrider & Champlin, 2001). Such studies are particularly useful as they can assess with high temporal resolution the neural effects of nicotine on auditory information processing. Therefore, study 3 is an event-related potential (ERP) study examining the effects of nicotine on auditory perception.

The cognitive enhancements of nicotine are well disputed and are typically explained by either a primary effect of nicotine or by a reversal effect of a nicotine-induced abstinence deficit. Therefore, in order to avoid the influence of withdrawal affects those without a nicotine dependence, nonsmokers, will be examined in the current study. This will allow us to test whether nicotine has a primary effect on cognition/attention.

The aim is to test whether nicotine enhances auditory information processing, and if so, to identify which cognitive mechanisms are responsible for this enhancement. This will help provide a neurological explanation for why nicotine is consumed in the context of music. Information explaining and describing the EEG technique and how ERPs are derived from this method are provided in Appendix K.

4.2. Hypotheses and components of interest

For the current study I am interested in identifying which cognitive mechanisms underlie nicotine's ability to enhance music-induced emotion. To determine this I will test the effects of nicotine on auditory pitch perception in healthy nonsmokers. I have chosen four ERP components to examine, P1, N1, P2, and N2, as these are implicated in arousal and attention. Furthermore, ERP

studies using auditory stimuli have shown these components to be particularly affected by nicotine.

For auditory stimuli, P1 occurs approximately 50 ms after the onset of a stimulus. For this reason it may be referred to as the P50 (Key, Dove, & Maguire, 2005). It is strongly affected by stimulus factors, such as intensity (Kaskey, Salzman, Klorman, & Pass, 1980), as well as arousal (Harkrider & Champlin, 2001). The P1 has maximal amplitude over the frontal and central regions of the scalp (Key et al., 2005) and is thought to be partially generated by the cholinergic pedunculopontine nucleus (PPN) neurons that give rise to the ascending reticular activating system (RAS) (Buchwald et al., 1992). Its source is also the primary auditory cortex (PAC), superior temporal gyrus (Huotilainen et al., 1998; Thoma et al., 2003) and the medial frontal cortex (Weisser et al., 2001).

The N1 component is one of the easiest auditory components to identify and occurs approximately 100 ms after stimulus onset. It is affected by arousal (Harkrider & Champlin, 2001). It is also enhanced by increased selective attention to basic stimulus characteristics (Hillyard, Hink, Schwent, & Picton, 1973). It has maximal amplitude over frontocentral areas (Vaughan & Ritter, 1970) and the vertex (Picton, Hillyard, Krausz, & Galambos, 1974). Its source is the primary auditory cortex in the temporal lobe (Vaughan & Ritter, 1970), although some have suggested additional sources in the frontal lobe (Giard et al., 1994).

For auditory stimuli the P2 component occurs approximately 180-250 ms after stimulus onset (Friedman & Meares, 1980). This component shares many characteristics with N1 and as such they are often examined together as the N1P2 complex. For example, the P2 is also implicated in arousal and attention (Harkrider & Champlin, 2001) and is sensitive to physical characteristics of stimuli, including pitch (Novak, Ritter, & Vaughan, 1992). Furthermore, it is sensitive to habituation processes (Rust, 1977) and decreases as an indication of more efficient stimulus filtering (Knott, 1989). It has maximal amplitude over the central region (Holcomb, Ackerman, & Dykman, 1986; Iragui, Kutas, Mitchiner, & Hillyard, 1993) and its source is the PAC and the secondary auditory cortex (Zouridakis, Simos, & Papanicolaou, 1998).

The N2 is evoked between 180-325 ms following the onset of auditory stimuli (Patel & Azzam, 2005). It is modulated by arousal and attention (Harkrider & Champlin, 2001) and is also associated with response inhibition (Jodo & Kayama, 1992; Kaiser et al., 2006). It has maximal amplitude over the central parietal (Simson, Vaughan, & Ritter, 1977) and the frontal-central (Kaiser et al., 2006) regions. It has bilateral sources in the supratemporal auditory cortex (Bruneau & Gomot, 1998). Additionally, its neural generators may include the anterior cingulate cortex (ACC) (Gemba & Sasaki, 1989) and the right inferior frontal gyrus (IFG) (Lavric, Pizzagalli, & Forstmeier, 2004).

4.3. P1

Many of nicotine's performance-enhancing properties can be explained through its ability to shift brain-state arousal (Heishman, Kleykamp, & Singleton, 2010; Wesnes & Warburton, 1983). That is, many of the cognitive improvements seen with nicotine are thought to be indirectly mediated by its mood-elevating and physiological arousal properties (Newhouse, Potter, & Singh, 2004; Waters & Sutton, 2000) and indeed smokers have self-reported that arousal control is one motive for nicotine use (Gilbert, 1979).

P1, the component implicated in arousal and known to be sensitive to stimulus factors (Harkrider & Champlin, 2001; Kaskey et al., 1980), has been shown to increase in amplitude in studies examining nicotine's effect on smokers and nonsmokers using auditory stimuli. Knott (1985b) examined 16 abstinent female smokers who were tested under smoking and nonsmoking conditions. They were presented with distracting tones in their left ear that were either of high (100 dB) or low (60 dB) intensity. For passive and active tasks participants were told to ignore the distracting stimuli. In the active task participants completed a choice reaction time (CRT) task as well as an auditory digit detection (ADD) task, which was presented in their right ear. Results showed a P1 amplitude increase during non-task (passive) conditions, irrespective of intensity. This suggests that nicotine enhances initial sensory level intake of surrounding stimuli, irrespective of their relevance. These results are partially corroborated by a study that examined nonsmokers who were stimulated with electrical nerve pulses under transdermal nicotine administration and placebo conditions. The P1-N1 amplitude was found to

increase with nicotine in the right hemisphere, although it was also found to decrease in the left hemisphere. Furthermore, there was no significant effect of nicotine on P1 latency (Harkrider & Champlin, 2001). These results provide moderate evidence supporting nicotine's ability to affect cortical activity and the transmission of acoustic information. It is particularly important that these effects were found in nonsmokers, which suggests that the cognitive effects of nicotine are evident even for those not regularly exposed to nicotine. This supports the idea that nicotine's effects are not due to the reversal of withdrawal symptoms.

The aforementioned studies have found P1 amplitude to increase as a result of nicotine in both smokers and nonsmokers. However, this has only occurred during passive listening tasks using auditory clicks or electrical pulses. To the best of my knowledge no study has found significant results when testing nonsmokers using an active-listening task to examine the effects of nicotine on the P1 component. Furthermore, the results found for passive tasks have not been demonstrated consistently. For example, a study by Friedman and Meares (1980) found no effect of nicotine on smokers who listened to auditory clicks. Participants were tested over varying abstaining periods and before and after the administration of two cigarettes or a waiting period where no nicotine was administered. Results showed no effect of nicotine on the amplitude of the P1N1 component.

Taken together, the results of these studies suggest that although nicotine can increase P1 amplitude further research is needed to discern under which conditions nicotine exerts its cognitive effects on arousal and whether passive and active listening plays a role in these effects. They further suggest that while nicotine can affect physiological arousal, the cognitive consequences of this are equivocal. They indicate, at best, a weak enhancement of cortical registration of auditory stimuli regardless of the stimuli's relevance. This may be a result of the different methodologies employed. For example, different delivery methods of nicotine (e.g. transdermal patches, nicotine gum), different cohorts (e.g. smokers, nonsmokers), and different task paradigms (e.g. task or non-task responses) may have resulted in variations in metabolic rates and therefore nicotine plasma concentrations. This may have consequently led to

inconsistent affects on arousal and the P1 component or it may have resulted in cognitive responses that are not replicable.

There is also a gap in the literature regarding how nicotine affects nonsmokers' arousal and P1 component as most studies examine a smoking population. However, smokers are likely in a state of withdrawal when beginning experimentation as they are often requested to abstain from nicotine before testing. In this way nicotine only serves to normalize smokers, returning them from cognitive and physiological deficits to baseline levels. Therefore, it is of interest to investigate nonsmokers, who begin experimentation at baseline levels, in order to test the true effects of nicotine.

With the exception of Knott (1985b) all of the aforementioned studies used sound stimuli (e.g. pulses, clicks), which lack the physical dimensions of music (e.g. loudness, pitch). However, music is more likely to be purposefully encountered and attended to in everyday life. Therefore, sound stimuli that incorporate a musical dimension may facilitate auditory perception better than pulses and clicks, and may therefore be more sensitive to the effects of nicotine. Furthermore, the study by Knott (1985b) manipulated loudness, one dimension of music. Another dimension of music, which has yet to be examined, is pitch. The basic perceptual mechanisms involved in pitch processing and how pitch is analyzed by the auditory system is well established (McDermott & Oxenham, 2008). For example, we know that variations in pitch (e.g. high pitch, low pitch) are easy to perceive and discriminate (McAdams, 1989). Therefore, the current study will use one high-pitched and one low-pitched tone to investigate how nicotine affects auditory perception. Additionally, the results of study one from this thesis found a (nonsignificant) trend for nicotine to increase heart rate in nonsmokers, suggesting that an increase in arousal may be possible for nonsmokers receiving nicotine. Therefore, I predict that using pitch stimuli will increase P1 amplitude after the administration of nicotine in nonsmokers (Harkrider & Champlin, 2001; Knott, 1985b). Furthermore, the current study predicts this increase in P1 amplitude to occur in the frontal and central scalp regions (Key et al., 2005), and although there is limited research regarding how nicotine affects P1 latency, I predict it to decrease as nicotine has been shown

to increase the speed of information processing in nonsmokers (Le Houezec et al., 1994).

4.4. N1- P2

The results of nicotine studies examining the P1 component suggest that the effects of nicotine on the processing of auditory information may be less related to arousal and more related to attention, and indeed, the most consistently affected cognitive function of nicotine is attention (Newhouse et al., 2004; Stolerman, Mirza, & Shoaib, 1995). Attention, although related to arousal, is a separate mechanism that enables cognitive resources to be selectively directed to the processing of one stimulus over others, which are thought to be either partially or completely rejected from perception, experience, entry into long-term memory, and control over behavior (Knudsen, 2007). One aspect of attention that is particularly influenced by nicotine is selective attention. This can be explained by the stimulus-filter hypothesis that suggests nicotine to contain attentional narrowing properties by gating out irrelevant or distracting stimuli and/or gating in relevant stimuli. This helps narrow the range of stimuli that enters conscious awareness and requires cognitive processing (Broadbent, 1958; Friedman, Horvath, & Meares, 1974; Knott, 1978). Kassel (1997) later expanded this hypothesis with the 2-factor model, proposing that additionally, nicotine contains attentional broadening properties. These properties increase one's perceptual capacity by enhancing attentional focus to relevant stimuli. That is, because nicotine screens out irrelevant stimuli, cognitive resources are freed up and allocated to task-relevant stimuli.

N1, the component strongly associated with attention, consistently increases in amplitude during auditory tasks of selective attention (Hillyard et al., 1973). In general, this effect is further enhanced by nicotine (Knott, 1985b, 1986), reflecting the drug's ability to improve attentional processes (Hillyard & Picton, 1979). P2, the component implicated in habituation processes (Rust, 1977), consistently decreases as a result of nicotine in auditory tasks of selective attention (Friedman, Horvath, et al., 1974; Knott & Harr, 1995). This reflects a more efficient filtering process and an enhanced ability to disengage from irrelevant stimuli (Knott, 1985a, 1989).

Research suggests that modulations of the N1 and P2 reflect an enhancement of two steps in the chain of auditory information processing: first an attentional focus, and then an attentional switching. To examine this process Knott (1985a) tested female smokers under sham-smoking and real-smoking conditions during an S_1-S_2 RT task. Participants were subject to an initial auditory warning signal (S_1) consisting of a binaurally presented tone. This was followed by a visual imperative (green light) signal (S_2). The RT task required participants to respond to S_2 under two conditions, with or without an auditory distraction task. There was no effect of nicotine or task on N1 amplitude, but there was a reduction of P2 amplitude during the auditory signal (S_1) following smoke intake. Similar findings have been reported by others using male smokers (Friedman, Goldberg, Horvath, & Meares, 1974; Friedman & Meares, 1980) and may reflect tobacco's ability to facilitate a more efficient cognitive disengagement or switching of attentional resources from redundant stimuli (S_1) to processes that prepare responses to relevant or imperative stimuli (S_2) (Knott, 1984). A follow up study that added two levels of complexity to the RT task corroborates this as it also found a decrease in P2 amplitude (Knott, 1986). Additionally this follow up study found an increase in N1 amplitude. These two findings (an increase in N1 amplitude and a decrease in P2 amplitude), along with the results of Knott (1985a), suggest an enhancement of two sequential cognitive actions as a result of nicotine: an initial enhancement in attentional focus on S_1, then a disengagement or attentional switch from S_1 and the auditory distraction task to the future-oriented, perceptual/cognitive/motor processing of the visual signal (Knott, 1989). Tobacco's ability to initially enhance and then disengage an individual's attention may be reflected by smokers who self report smoking to help with thinking and concentration (Wesnes & Warburton, 1983).

Research with smokers has used other paradigms to test the effects of nicotine on the N1 and P2 components. In general, they provide support for an increase in N1 amplitude and a decrease in P2 amplitude. Such paradigms include the dichotic listening task and the auditory oddball task. These paradigms are advantageous because they demand no motor response from participants and therefore isolate the cognitive mechanisms of attention. In a

dichotic listening task participants are asked to attend to and detect target deviant stimuli in one ear while simultaneously ignoring similar stimuli in the other ear. However, in one such study using smokers with overnight abstinence, 4 mg of nicotine gum failed to affect the amplitude of N1 (Knott et al., 2006). In an auditory oddball task participants are asked to count rare low-pitched tones (considered relevant) compared to frequent high-pitched tones (considered irrelevant). In one study this task was performed with abstaining smokers and in general, supports the trend that nicotine increases N1 amplitude and decreases P2 amplitude. More specifically, N1 amplitude was found to increase for the irrelevant tones (high-pitched tones), but was decreased for the relevant tones (low-pitched tones). N1 latency was also reduced as a result of smoking, and was more reduced for irrelevant than relevant tones. Lastly, P2 amplitude showed a reduction for both relevant and irrelevant tones (Domino & Kishimoto, 2002).

The results of the aforementioned studies using different task paradigms demonstrate that after periods of abstinence, smoking/nicotine increases N1 amplitude and decreases P2 amplitude for smokers. However, not much is known about the effects of nicotine on N1 and P2 in nonsmokers and none of the studies use a decision-making task to assess these effects. A decision-making paradigm would allow the opportunity to examine selective attention, response inhibition, and habituation in a single study, making it an ideal paradigm for studying the effects of nicotine on attention and related processes. Furthermore, although some of these studies have used tones as part of their auditory stimuli (Friedman, Goldberg, et al., 1974; Friedman & Meares, 1980; Knott, 1985a), as opposed to pulses and clicks, the tones were only used as target and distractor stimuli and so were not manipulated to test how pitch perception is affected by nicotine intake. The exception to this is Domino (2002) who used high and low-pitched tones and found N1 amplitude to be increased for high-pitched, but not low-pitched tones, as well as N1 latency to be decreased more for high-pitched than low-pitched tones. However, these results are confounded by the fact that the high-pitched tones occurred more frequently than the low-pitched tones and so make it difficult to discern if it was pitch or the relevance of tone that influenced the N1 and P2 components. This

suggests that further research is needed to determine if nicotine can affect the auditory processing of high-pitched and low-pitched tones, and if so how. Therefore, the current study will use an equal number of high and low-pitched tones to assess how attentional mechanisms and pitch perception, via the N1 and P2 components, are affected by nicotine in nonsmokers.

Research examining the effects of nicotine on auditory processing with nonsmokers is small and equivocal. For example, in a study using an auditory distraction paradigm Knott and colleagues (2009) asked nonsmokers to discriminate between task-relevant stimuli (standard tones of long and short duration at 1,000 Hz) with and without distractors (deviant tones at 900 Hz or 1,100 Hz). Overall, 6 mg of nicotine gum was found to diminish the automatic processing of deviant stimuli. This corroborates the results found in smoking populations and suggests nicotine to enhance early pre-attentive stages of deviant detection in nonsmokers by rendering them less distracting. Although no effect of nicotine was found on N1 amplitude or latency, there was an effect of deviant stimuli, which caused N1 amplitude to increase in the frontal, central, and occipital regions of the scalp (Knott, Bolton, et al., 2009). Other paradigms using nonsmokers have found similar results. In an experiment using a dichotic listening task no effect of nicotine was found on N1 amplitude or latency. However, there was a trend for nicotine to increase N1 amplitude in the frontal region during attended stimuli (Knott, Shah, et al., 2009). These non-significant results are further supported by Harkrider and Champlin (2001) who found no effect of nicotine on N1-P2 and P2-N2 amplitude as well as the P2 latency in nonsmokers during monaural electrical pulses. The aforementioned auditory oddball experiment by Domino and Kishimoto (2002) also tested nonsmokers. Similar to other studies, they found no effect of nicotine on the N1 component. However, they did find an increase in P2 amplitude to irrelevant stimuli (frequent, high-pitched tones), but not to relevant stimuli.

The modest and inconsistent findings of nicotine's effect on nonsmokers suggests further research is needed on this population in order to determine if nicotine's cognitive enhancing effects are a reflection of the normalization of withdrawal-induced decrements in abstaining smokers or whether nicotine's effects are absolute regardless of smoking status. Therefore the current study

will examine nonsmokers with the following hypotheses. However, because there is minimal research on the electrophysiological effects of nicotine on nonsmokers the following hypotheses take into account the findings from both smoking and nonsmoking experiments. Furthermore, although the current study does not contain relevant and irrelevant stimuli, which is often used to assess habituation, as reflected by the P2 component, habituation can still be examined in the current study. That is, habituation is an adaptation to the same sounds presented repeated many hundreds of times. In this way, habituation can be considered an overlearning of repeated stimuli that results in an increase in processing efficiency and is reflected by a reduction in P2 amplitude and latency (Baldeweg, Wong, & Stephan, 2006). Therefore, the current study hypothesizes that nicotine will increase N1 amplitude as well as decrease P2 amplitude in the frontal and central scalp regions (Knott, 1986; Knott, Shah, et al., 2009; Pritchard, Sokhadze, & Houlihan, 2004) despite the non-significant findings from nonsmoking studies with nicotine. Furthermore, because nicotine is able to enhance selective attention and improve the efficiency of processing auditory stimuli it may be able to reduce the speed at which these processes take place (Domino & Kishimoto, 2002; Friedman, Horvath, et al., 1974). Therefore, I predict the latency for both the N1 and P2 components to be reduced as a result of nicotine.

4.5. N2

The N2 component occurs in response to attended and unattended deviants and can reflect disparity between a deviant stimulus and a sensory-memory representation of the target stimulus (Patel & Azzam, 2005). The N2 is also implicated in response inhibition in go/nogo tasks (Jodo & Kayama, 1992). Early research with this component (Picton & Hillyard, 1974; Picton et al., 1974) suggests the amplitude of the auditory N2 to be inversely related to behavioral arousal and therefore to be significantly smaller during high activation states (Knott, 1989). Initial reports examining the influence of nicotine on ERPs using auditory stimuli reported a reduction of the P2-N2 wave. For example, Friedman, Goldberg, and colleagues (1974) examined 10 male smokers' passive response to monoaurally presented clicks. Participants were tested over three sessions under either 12 h abstaining or non-abstaining conditions. They were tested

before and after no smoking, placebo smoking, and smoking two cigarettes. Results suggest a reduction for N2 amplitude after 12 h of nicotine abstinence. Similar results were obtained for the aforementioned study by Friedman and Meares (1980), who tested smokers' responses to auditory clicks. They found a decrease in the amplitude of the P2-N2 complex during smoking conditions compared to nonsmoking conditions. Furthermore, this reduction in amplitude was found to be more dramatic when smokers abstained from smoking for 12 h compared to only 1 hr. This suggests that while nicotine stimulates the central nervous system, it also triggers an inhibitory mechanism that facilitates cognitive focus without distraction of extraneous elements (Friedman, Horvath, et al., 1974). Furthermore, since N2 is associated with activity of the cortico-thalamic efferent auditory pathway the reductions found in N2 may reflect more efficient gating of irrelevant or disruptive stimuli by the efferent auditory system. This in turn allows relevant auditory stimuli to ascend to higher levels of cortical processing (Harkrider & Champlin, 2001). Later studies have found no effect of nicotine on the amplitude of the N2 component and P2-N2 complex (Knott, 1985b; Knott, Kerr, Hooper, & Lusk-Mikkelsen, 1995). Furthermore, few studies have examined the effects of nicotine on the N2 component using a nonsmoking population. However, Harkrider and Champlin (2001) found that after electric nerve pulses nicotine decreased N2 amplitude as well as reduced N2 latency in nonsmokers.

As is the case for other ERP components, the literature regarding the effects of nicotine on N2 is mixed. While there is some evidence for a reduction of the N2 amplitude and latency, the results overall necessitate further research in order to clarify the pattern of electrophysiological responses to nicotine. Many of these studies used a passive listening paradigm and although there is a decision-making element to go/nogo tasks, stimuli in this paradigm are not represented equally. Also, the stimuli used were not representative of music (e.g. auditory clicks), with the exception of Knott (1985b) and Jodo (1992) who used a 1000 Hz tone, but did not vary pitch. Furthermore, the research using nonsmokers is significantly smaller than that with smokers, emphasizing the need for future research to focus on this cohort to establish whether nicotine exerts its effects through withdrawal reversal or absolute enhancement. Lastly,

based on the above findings that suggest N2 to be inversely related to arousal (Picton & Hillyard, 1974; Picton et al., 1974) and based on the nonsmoker findings of Harkrider and Champlin (2001) who found a reduction for N2 amplitude and latency I hypothesize a decrease in both the amplitude and latency of the N2 component.

4.6. Task performance and reaction time

Much research has focused on understanding the cognitive influences of nicotine, perhaps using nicotine's enhancement to partially justify its widespread use. Most of these studies, which have led to a variety of conclusive and inconclusive findings, employ a paradigm involving task performance in order to test sensory ability, motor ability, attention, learning and memory, problem-solving, as well as other skills.

Importantly, many nicotine studies use a variety of populations, including smokers, abstinent smokers, and nonsmokers. A clear distinction between these populations must be made as the effects of nicotine can substantially differ between those with and without a tolerance to the drug (Heishman et al., 1994). Experiments with nonsmokers have found nicotine administration to enhance performance in a few areas. These experiments indicate that nicotine administration reliably enhances finger-tapping rates when administered through nasal spray or subcutaneous injection (Jones, Sahakian, Levy, Warburton, & Gray, 1992; Perkins, Stiller, Sexton, Debski, & Jacob, 1990; West & Jarvis, 1986) and produces modest, but limited, improvements in tests of divided attention. For example, modest nicotine-induced enhancement was reported in tracking tasks (Heishman et al., 1994). While some studies show no effect of nicotine on reaction times in nonsmokers during tasks of psychomotor performance (Hindmarch, Kerr, & Sherwood, 1990; Kerr, Sherwood, & Hindmarch, 1991), others have shown the drug to decrease reaction times in nonsmokers during working memory tasks (Ernst, Heishman, et al., 2001). Other evidence suggesting that nicotine enhances behavioral and cognitive tasks is weak to inconclusive, including studies which examine sensory abilities, varying types of attention, learning, and memory (Hindmarch et al., 1990; Jones et al., 1992; Kerr et al., 1991), as well as for reasoning and problem solving (Dunne, MacDonald, & Hartley, 1986; Heishman et al., 1993). This

suggests that nonsmokers can potentially benefit from nicotine administration, but that more research is needed to understand under which tasks this enhancement occurs. Based on the research of Ernest and colleagues (2001) and the review from Heishman and colleagues (1994), which showed nicotine-induced enhancement on reaction time during tracking and working memory tasks, I hypothesized that nicotine would result in a decrease in reaction time during the decision-making task.

4.7. Current Study

In order to extend previous research the current study aims to better understand if and how nicotine is able to enhance information processing in the auditory pathway. That is, does nicotine affect the neural responses implicated in pitch perception and if so, which cognitive mechanisms are responsible for this enhancement? This will help explain the co-consumption of nicotine and music listening.

This study builds on previous research in a number of ways:

1. From the electrophysiological studies described above not much is known about how nicotine affects nonsmokers. Furthermore, it is still unclear whether nicotine's cognitive enhancing effects are a result of withdrawal reversal or an enhancement of some aspect of auditory perception and cognition. Therefore the current study examines nonsmokers. Furthermore, the nonsmoking studies described above provide mixed results regarding the effects of nicotine on the ERP components P1, N1, P2, and N2. These inconsistencies may be a result of the different nicotine delivery methods (transdermal, gum) and task paradigms (passive, active) employed across studies. Different nicotine delivery methods result in different pharmacodynamics and so can result in different cognitive effects. Furthermore, different tasks require different cognitive functions (e.g. arousal, selective attention, sustained attention), making it difficult to compare results across studies. Therefore, the current study will examine nonsmokers during a decision-making task in order to examine how these ERP components are affected by nicotine during auditory perception.

2. Research has mainly employed auditory stimuli that vary in intensity (volume) and to a lesser extent in duration. However, pitch is also an important fundamental element of music (Spencer & Temko, 1988). Furthermore, the tonotopical organization of the PAC mirrors the distribution of receptors in the cochlea, which contains a gradient of neurons that preferentially responds to high and low frequencies (Humphries, Liebenthal, & Binder, 2010; Talavage et al., 2004). Because nicotine is able to activate receptors in the auditory pathway (Crawford et al., 2002) it may be that pitch perception is affected by the administration of nicotine. Therefore, the current study will use pitch in order to investigate how nicotine affects auditory perception and cognition.

3. The aforementioned studies employ several different methods for nicotine administration including smoking, transdermal patches, and nicotine gum. This may explain the variation in findings regarding how nicotine affects cognition. Two studies that used nonsmokers (Knott, Bolton, et al., 2009; Knott, Shah, et al., 2009) administered nicotine via pilocrilex gum, as did the first study of this thesis. Therefore, the current study will administer 4 mg of pilocrilex nicotine gum to nonsmokers in order to mirror the methodology of similar past studies as well to remain consistent across the studies of this thesis.

4. Several different paradigms have been used to test the effects of nicotine on auditory perception in nonsmokers, including passive listening (Harkrider & Champlin, 2001), a discrimination task (Knott, Bolton, et al., 2009) and a dichotic listening task (Knott, Shah, et al., 2009). Again, this variation may account for the different ERP results found across these studies. With this mind, a simple and repetitive task was employed for the current study where participants were asked to make a decision based on the combination of auditory and visual stimuli presented. A decision-making paradigm requires attention and response inhibition, while repetitive stimuli is conducive to habituation. This allows us to test these cognitive mechanisms during nicotine and placebo conditions.

5. Previous neuroscientific literature suggests that there is an association between the anterior cingulate cortex (ACC) and nicotine's acute effects and nicotine addiction (Brody, 2006; Brody et al., 2004; Ernst, Matochik, et al., 2001; Giessing, Fink, Rösler, & Thiel, 2007; Grünwald, Schröck, & Kuschinsky, 1987; Nybäck et al., 1989; Stein et al., 1998). The ACC, located on the medial surface of the frontal lobes forms a ring around the rostrum of the corpus callosum. It makes critical contributions to the neural systems involved in the executive control of cognition and emotion (Carter, Botvinick, & Cohen, 1999; Fallgatter, Bartsch, & Herrmann, 2002). Three theories have been developed in regards to the role of the ACC, 1) motivated attention, emphasizing the connections between the ACC and the limbic system (e.g. amygdala), 2) attention allocation, emphasizing activation of the ACC during tasks that elicit incompatible response tendencies, which require thought for correct performances, and 3) error detection, emphasizing the negative scalp potentials that occur during incorrect responses and which appear to have a medial frontal generator (Carter et al., 1999).

Interestingly, fMRI studies have found nicotine-induced activation of the ACC (Kumari et al., 2003; Stein et al., 1998), which helps to regulate the cognitive and emotional processes implicated in attentional, sensory, and motor responses (Allman, Hakeem, Erwin, Nimchinsky, & Hof, 2001; Bush, Luu, & Posner, 2000). As previously mentioned, ERP studies have found nicotine to enhance arousal and attentional processes, such as selective attention and stimulus filtering, amongst others (Knott, 1985b, 1989; Knott, Bolton, et al., 2009; Kumari et al., 2003; Newhouse et al., 2004; Stein et al., 1998; Stolerman et al., 1995; Warbrick et al., 2011). Because nicotine has been shown to increase activation of the ACC, and because the ACC is involved in arousal and attentional processes relevant to auditory information processing, the regions of interest in this study are those cortical areas associated with the ACC.

The cortical location of the ACC is suggested to be on or near the midline of the prefrontal cortex (Bush et al., 2000; Dehaene, Posner, & Tucker, 1994). Additionally, ERP studies investigating the effects of nicotine on arousal and attention consistently examine scalp electrical activity at the midline in the frontal and central lobes, as this is where amplitudes peaked in response

nicotine administration (Houlihan, Pritchard, & Robinson, 2001; Hummel, Livermore, Hummel, & Kobal, 1992; Knott et al., 2006; Knott, Shah, et al., 2009; Nieuwenhuis, Yeung, Van Den Wildenberg, & Ridderinkhof, 2003). These areas correspond to F_z and C_z of the 10-20 system (Jasper, 1958). In addition, nicotine ERP studies have also examined scalp electrical activity adjacent to the frontal and central midlines (Fehr, Wiedenmann, & Herrmann, 2006; Inami, Kirino, Inoue, & Arai, 2005; Knott, Bosman, Mahoney, Ilivitsky, & Quirt, 1999), which corresponds to F_3 and F_4, and C_3 and C_4, respectively. Therefore, these regions were of particular interest in the current study.

In the current study we predicted nicotine to enhance arousal and attention, reflected by an increase in P1 and N1 amplitudes, as well diminish habituation and response inhibition, reflect by a decrease in P2 and N2 amplitudes. Furthermore, we predicted nicotine to enhance auditory information processing, reflected by a decrease in latency for all ERP components. Lastly, based on previous research showing the frontal and central regions of the scalp to be most affected by nicotine we examined these regions for modulations in ERPs. I tested these predictions using a simple decision-making paradigm in a nonsmoking healthy population.

Nonsmoking participants were administered placebo or 4 mg of nicotine gum. They then heard either a high-pitched or low-pitched tone, followed by an image containing both an up arrow and down arrow. If participants heard the high-pitched tone they were to concentrate on the position of the up arrow and if they heard the low-pitched tone they were to concentrate on the position of the down arrow. Participants were then engaged in a decision-making task regarding their 'target arrow'. If their target arrow was position on the left-side of the image then they were to press '1' on a keypad and if their target arrow was position on the right-ride of the image then they were to press '4' on the keypad. There were 400 trials total.

4.8. Methods

4.9. Participant

I recruited 36 participants living in England. There were 18 males and 18 females with a mean age of 21.33 years, ranging from 18 to 29 (*SD* = 3.25). The age, gender, and number of participants per condition can be viewed in

Table 4.1. There was no significant difference in age between the participants of the placebo and nicotine conditions, $t(34) = -.74$, $p = .467$, $d = -.25$. All participants were undergraduate and postgraduate students at the University of Sheffield. Four participants ERP data was excluded either because there were no patterns found in the waveforms or because too much noise existed in the frontal electrodes. The ERP data presented below is the result of the remaining 32 participants. Informed consent was obtained prior to experimentation and participants either received participatory credits as undergraduate psychology students or were paid £10 for one hour and fifteen minutes of their time. The research protocol met the ethical requirements of the University of Sheffield's Department of Psychology.

Stringent criteria for participation was necessary in order to control for a number of confounds, including neurological health, language, handedness, musicianship, and smoking status. All participants were free of neurological and psychiatric illnesses based on self-reports and none were pregnant or breastfeeding, all contraindications against the use of nicotine gum (Baldeweg et al., 2006).

All participants were native English speakers with minimal exposure to secondary languages. Language background was controlled because it is known to strongly influence auditory processing (Salmelin et al., 1999; Vihla, Kiviniemi, & Salmelin, 2002) and exposure to a tonal language is particularly known to increase pitch perception (Krishnan, Xu, Gandour, & Cariani, 2005). Therefore, competency of secondary languages was assessed through self-reports of listening, speaking, reading, and writing (Appendix L). Those who scored a 4 or higher (on a 7-point scale) on any of the language subscales were excluded from participation. Volunteers were also excluded if they reported any experience with a tonal language, such as Mandarin or Vietnamese.

In order to control for hemispheric specialization (Alexander & Polich, 1997) and to conform to previous research methods (Wioland, Rudolf, Metz-Lutz, Mutschler, & Marescaux, 1999) all participants were right-handed, as defined by a score of 80-100% on the Edinburgh laterality test (Oldfield, 1971).

Because musical training has repeatedly shown to improve pitch processing (Besson, Schön, Moreno, Santos, & Magne, 2007) and musicians in

particular are thought to have superior pre-attentive auditory processing (Koelsch, Schröger, & Tervaniemi, 1999) musicians were excluded from the study. All participants were non-musicians defined as having no regular experience with playing a musical instrument and no musical training beyond mandatory music lessons in primary and secondary school.

Lastly, nonsmokers were recruited to control for participants' pre-drug state (Edwards & Warburton, 1982). Furthermore, in studies using abstaining smokers it is difficult to determine whether the results are due to the attention-enhancing effects of nicotine, withdrawal relief, or an alleviation of pre-existing attentional deficits that smoking self-medicates (Gilbert & Gilbert, 1995; Kassel, 1997). Therefore, participants were required to be entirely nicotine free for at least one year. This included habitual as well as occasional use, such as social smoking and shisha.

Table 4.1

Age and gender by nicotine dose

Nicotine Dose	*N*	Age	Age Range (years)	Gender
0 mg	18	M = 20.94; SD = 3.24	18-28	8 M; 10 F
4 mg	18	M = 21.72; SD = 3.10	18-29	8 M; 10 F

4.10. Material

4.10.1. Nicotine gum

The 4 mg nicotine polacrilex gum was Boots NicAssist ice mint flavored gum. 4 mg of nicotine gum was used in the experiment, as opposed to the lower dose of 2 mg, because this higher dose was shown to have a larger effect on SCL and skin temperature. Also, 4 mg of nicotine decreased sadness more than 2 mg. For placebo, Wrigley's Extra peppermint flavored chewing gum was chosen because of similar size, shape, and color to the nicotine gum.

4.10.2. Auditory stimuli

Sound stimuli were constructed based on previous research examining auditory perception using event-related potentials (ERP) and mismatch negativity (MMN) (Baldeweg et al., 2006; Tervaniemi, Just, Koelsch, Widmann, & Schröger, 2005). Sound stimuli consisted of two spectrally complex tones, one high and one low. The high-pitched tone consisted of its fundamental frequency, 523.25Hz (C5 on the Western scale) and its following four overtones of the harmonic series: 1046.50Hz, 1567.98 Hz, 2093.00Hz, and 2637.02Hz (C6, G6, C7, and E7 respectively on the Western scale). The low-pitched tone consisted of its fundamental frequency, 130.81Hz (C3) and its following four overtones of the harmonic series: 261.63 Hz, 392.00 Hz, 523.25Hz, and 659.25 Hz (C4, G4, C5, and E5 respectively). A single pitch-class was used (pitch C) so that the high and low-pitched tones were only distinguishable based on pitch height. This stopped participants from recognizing each tone based on pitch chroma (e.g. different pitches) and instead required them to recognize each pitch based solely on how high/low the tones were relative to each other. The tones contained harmonics as previous behavioral and neural research have shown complex tones to better facilitate pitch processing compared to fundamental frequencies only (Tervaniemi, Ilvonen, et al., 2000; Tervaniemi, Schröger, Saher, & Näätänen, 2000). The stimuli were synthesized using Ableton Live 9.1 Suite, a software music sequencer, on a Macbook Pro, 2014. All sounds had a presentation time of 300 ms with a 5 ms rise and fall time, similar to previous research methods (Koelsch et al., 1999). Sounds were presented binaurally via insert earbuds at ~80dB SPL.

4.10.3. Visual stimuli

Two images of upward and downward facing arrows were constructed. The first image presented an upward arrow on the left side and a downward arrow on the right side. The second image consisted of these same two arrows, but placed in reverse order, so that the downward arrow was on the left side while the upward arrow was on the right. The arrows and their two different arrangements can be viewed in Figure 4.1.

Figure 4.1. Visual stimuli of arrows

4.10.4. Pure tone audiometry

A Pure Tone Audiometry (PTA) hearing test was used to check for any signs of hearing loss and to confirm that participants could detect stimuli. The PTA hearing test was used based on previous research by Light and colleagues (2010). The test was performed at ~80dB SPL and consisted of tones at 125Hz, 250Hz, 500Hz, 1000Hz, 2000Hz, 4000Hz, 8000Hz, and 10,000Hz. The test was performed twice, once in each ear. Participants would have been excluded if they were unable to detect any tones in either ear or if they had gross abnormalities or asymmetries in their hearing between ears. No participants were excluded for this reason.

4.11. Procedure

Prior to the experiment participants took part in a screening questionnaire to determine their eligibility based on their health (Appendix D), language background (Appendix L), handedness (Oldfield, 1971), musicianship (Appendix B), and smoking history (Appendix C). Upon approval of eligibility an appointment for the EEG study was scheduled and participants were asked to refrain from all products containing nicotine, caffeine, and alcohol for 24 h before their experiment. The experiment lasted ~1 h. At the start of the EEG study, participants read an information sheet (Appendix M), gave informed consent, and were subject to a pure tone audiometry hearing test to confirm self-reports of normal hearing. Participants were then randomly assigned to

either the nicotine or placebo condition and were given the appropriate piece of gum (either nicotine or regular gum) to chew based on this assignment. Participants were instructed to chew the gum on a chewing-resting cycle of 30 s. That is, they chewed the gum for 10 s, then rested the gum on the inside of the cheek for 20 s. This cycle repeated for 25 min.

To help participants stay on task during the chewing-resting cycle a video was played that mirrored the action of chewing or resting. When the subject was to chew gum a high tone bell rang and an image of a mouth chewing gum appeared. When the subject was to rest, a low alarm tone sounded and an image of a stop sign with a halt hand in the center appeared. While participants were engaged in the chewing-resting cycle their head was measured and fitted with the EEG net and the sensors were checked for impedance levels. At the end of the 25 min a final image of a chewed piece of gum appeared and a message overtop read "Please spit out gum." At this time participants discarded the gum into a trash can and prepared to begin the auditory perception task. They did this by centering themselves 50 cm in front of the computer screen and by having earbuds fitted into their ears and checked for sound.

For safety reasons adverse effects were also monitored through self-report. Upon completion of the chewing-resting cycle participants were administered the Subjective Treatment Emergent Symptom Scale (STESS) that assess the physical reactions to nicotine and the severity of these reactions(Guy, 1976b). Participants with scores of 50% or more on any of the four subscales were discontinued from the study. Two participants were discontinued for this reason.

Before beginning the task participants were introduced to the general procedure of the experiment. Participants were told that on the computer screen a fixation cross would appear, followed by a sound. After this an image of two arrows facing in opposite directions (one up, one down) would appear and that based on the arrangement of these arrows they would be asked to indicate a response on a keypad using their index fingers. They were also told that after their response the procedure would repeat.

Participants were then introduced to the auditory and visual stimuli used in the experiment, which was presented using E-prime 2.0 software (Psychology Software Tools, Inc., Pittsburgh, PA). Participants first listened to the high-pitched and low-pitched tones separately. Because high and low are relative terms it would have been difficult for subjects to determine which tone was higher or lower without hearing both prior to experimentation. Next, participants were shown both arrow images and further details of the procedure were explained. They would hear a tone (either high-pitched or low-pitched) followed by one of the arrow images. If they heard a high-pitched sound they were to focus on the position of the up arrow. If the up arrow was positioned on the left-side of the image, then they were to press '1' on the keypad; if the up arrow was positioned on the right-side of the image, then they were to press '4'. Alternatively, if they heard a low-pitched sound, they were to focus on the position of the down arrow. If the down arrow was positioned on the left-side of the image, then they were to press '1'; if the down arrow was positioned on the right side of the image, then they were to press '4'. Figure 4.2 illustrates this procedure and displays the duration (in milliseconds) of each event.

In order to record the highest quality of EEG data participants were requested to refrain from blinking as best they could during presentation of the fixation cross and sound and to instead try to blink during the arrow images or while responding with the keypad. After these verbal instructions were given the lights were turned off and participants were left alone in the room. In order to reiterate the experimental instructions the procedure of the experiment was written out on the computer and participants were given practice trials consisting of two blocks of 8 trials each. After practicing, the experiment began, which consisted of 4 blocks of 100 trials each. In between each block participants were allowed to rest for as long as they liked. Rest periods were employed in order to maximize concentration during the experiment. At the end of the experiment participants were detached from the EEG net and debriefed.

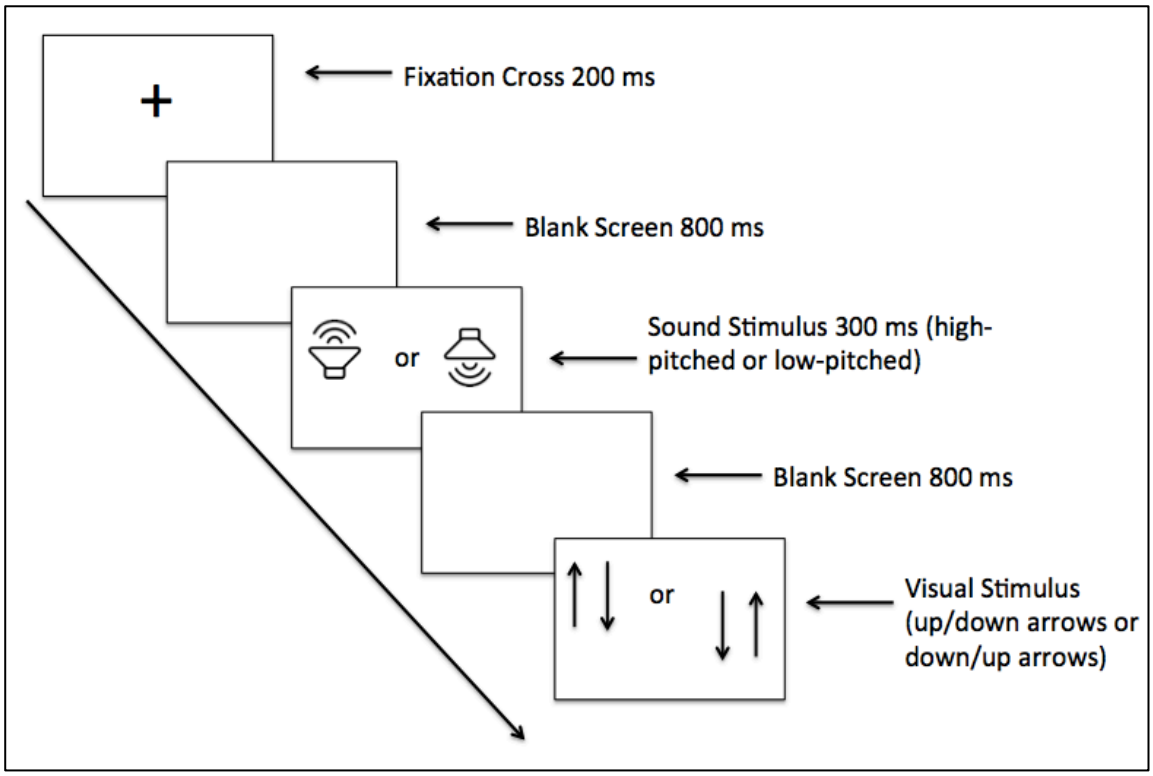

Figure 4.2. The Experimental Procedure for each of the 400 trials

4.12. Data acquisition

Electrophysiological data was time-locked to the auditory stimuli by recording a trigger at the same time as stimulus presentation. This data was recorded continuously from the scalp using a high-density array of 128-channel Geodesic Sensor Net (GSN) (Tucker, 1993) from Electrical Geodesics, Inc. (EGI) (Eugene, Oregon). The GSN is a lightweight knitted network of elastic threads that house electrodes in small plastic pedestals. Inside each pedestal is a Ag/AgCl synthetic sponge sensor that serves to detect and record the electrophysiological data. The sponges are soaked in a solution of potassium chloride (KCl) in order to render them conductive (Casanova et al., 2012). The GSN has an even inter-electrode distance of 2.7 cm and a C_z reference at the vertex of the scalp (Sabbagh, Moulson, & Harkness, 2004). The 6 most anterior electrodes of the GSN record the horizontal and vertical electroculogram (EOG) that monitors eye movements and eye blinks. These electrodes were located at the outer canthi and above and below the left and right eyes. The GSN connects to the EGI high-input impedance amplifier (200 MOhm, Net Amps) with an in-line finite impulse response (FIR) bandpass filter of .1 Hz – 400 Hz.

Individual electrodes were adjusted in order to keep impedance below 50 kΩ, as recommended by the manufacturer. Channel signals were amplified (1000x) and digitized with a 12-bit A/D converter at a sampling rate of 1000 Hz (1 ms samples). The EEG data, as well as event onset times, were collected and digitally stored on a Macintosh G4 (10.2.8) power PC using EGI Net Station 4.1.2 for further analysis. Simultaneously with the electrophysiological data, trial specific information, such as condition type (e.g. combination of visual and auditory stimuli), accuracy of response and reaction times were collected through E-prime2 on a PC and stored for further use in data analysis.

4.13. Data Analysis

Subsequent processing and analyses were performed offline using the EGI Net station 4.1.2 software (Electrical Geodesics, Inc.) for the ERP data and E-prime2 for the reaction time data. All statistical tests were performed using SPSS version 23.0 for Mac. Data was digitally filtered offline with a bandpass of 1-50 Hz. A highpass, first order filter of 1 Hz was used in order to exclude any slow direct current shift, while a lowpass-filter of 50 Hz was used in order to remove any mains interference. This finite impulse response-filter had a pass band gain of 99.0%, a stop band gain of 1.0%, and a roll off rate of 2.00 Hz. Segmentation of the continuous EEG data was performed using an epoch that began 100 ms prior to the onset of the sound stimulus and ended 400 ms after. Next, artifacts were removed from the epochs. This was first done automatically by employing Net Station's artifact detection routine. That is, individual channels within each epoch were marked bad if they contained either zero variance, a fast average amplitude exceeding 200 μV, or a differential average amplitude exceeding 200 μV. Channels were also marked as bad for the entire recording if they were bad for more than 20% of the segments. Furthermore, individual epochs were rejected if they contained eye movements, identified by a maximum to minimum differential of 70 μV, or eye blinks, identified by a maximum to minimum differential of 100 μV. All segments were then subjected to a visual inspection in order to identify and remove any remaining artifacts that did not exceed the threshold values (e.g. noisy channels and noisy segments of data). Individual segments were rejected if they contained more than 10 bad channels (e.g. > 13 channels). For the remaining segments,

individual bad channels were replaced with a spherical spline algorithm (Srinivasan, Nunez, Tucker, Silberstein, & Cadusch, 1996), which interpolates data for bad channels using data of the surrounding channels. Overall, 74% of the segments were retained. A summary of the retained epochs for each condition by group is shown in Table 4.2. A summary of the bad channels that were interpolated for each condition by group is shown in Table 4.3.

Table 4.2

M(SD) and range of segments remained, number of segments eliminated, and % of segments retained d for each condition by group

Group	**High Pitch**	**Low Pitch**
Placebo		
M(SD) of segments remained	142.00(31.51)	141.27(30.46)
Range of segments remained	95-193	99-184
% of segments remained	71%	71%
Segments eliminated	58.00	58.73
Nicotine		
M(SD) of segments remained	151.19(27.22)	151.81(26.54)
Range of segments remained	109-192	05-195
% of segments remained	76%	76%
Segments eliminated	48.19	48.19

Table 4.3

M(SD) channels interpolated for each condition by group

Group	**High Pitch**	**Low Pitch**
Placebo	14.53(13.34)	13.20(14.29)
Nicotine	12.31(10.22)	13.31(11.45)

The remaining trials were then segregated by condition (high pitch; low pitch) and averaged for each participant. For ERP analysis the conditions of the visual stimuli were collapsed over high pitch and low pitch. That is, up/down

arrow images and down/up arrow images were combined when paired with high pitch, and combined when paired with low pitch. The ERPs obtained for both high pitch and low pitch stimuli were taken regardless of whether the correct keypad response was given by the participant. This is because the decision-making aspect of the experiment and the subsequent response was used to keep participants focused on listening to the auditory stimuli as well as to conceal the true nature of the experiment.

Next, all ERPs were baseline-corrected. This was performed for each channel by taking the average of all the samples within the 100 ms of pre-stimulus data and subtracting it from all the remaining samples (stimulus onset to 400 ms post-stimulus). Finally, the individual participants' ERPs were re-referenced in order to correct for the polar average reference effect (PARE). That is, voltage measurements from EEG are actually differentials. They are a measurement of the difference in potential between the site being measured (a specific electrode) and the reference site (C_z), which is assumed to have a voltage of zero. However, in order for C_z to have a true voltage of zero the GSN would need to have full coverage of the head's surface, which is not the case. Instead, the surface of the scalp is unevenly sampled because electrodes are concentrated on the top of the head. This causes the average reference to be biased towards the top of the head and results in differences in the average to be smaller at the vertex than at the periphery. This bias is known as the polar average reference effect (PARE) and requires a PARE-corrected average reference (Junghöfer, Elbert, Tucker, & Braun, 1999). After the data was re-referenced group averages of ERPs were calculated separately for the nicotine and placebo groups for both the high pitch and low pitch conditions.

The ERP components of interest were P1, N1, P2, and N2. They were identified through visual inspection of group averages and individual data. Furthermore, they were found to be most distinct and of largest absolute amplitude in the frontal and central regions of the scalp. The time windows chosen for each component were based on previous literature (Key et al., 2005; Picton & Hillyard, 1974) as well as visual inspection of the data. Table 4.4 specifies these time windows for each component.

Table 4.4.

Time window for each ERP component

Component	Time Window
P1	30-70 ms
N1	80-120 ms
P2	140-200 ms
N2	240-300 ms

4.14. Statistical analysis

Visual inspection of the ERP data showed the grand average waveforms to be most clearly defined, as well as maximal in amplitude, in the frontal and central regions of the scalp. This is in agreement with past nicotine-based ERP studies (Fehr et al., 2006; Inami et al., 2005; Knott et al., 1999), and possibly suggests that this source of cortical activation could originate from the subcortical structure, the ACC (Kumari et al., 2003). Therefore, these regions were further investigated. The mean amplitude and latency of the P1, N1, P2, and N2 components from the frontal and central regions of the scalp were statistically analyzed for the left, central, and right areas. For these regions a group of channels (electrodes) was averaged together for the left and right areas. That is, groups of neighboring electrodes (e.g. those surrounding F_3) were shown to have nearly identical amplitude and latency values for each component of interest and were therefore averaged together. Averaging a group of neighboring electrodes is a standard approach taken in ERP analyses (Baruth, Casanova, Sears, & Sokhadze, 2010; Picton et al., 2000) and is done in order to improve the signal to noise ratio, thereby increasing the statistical power of the data (Oken & Chiappa, 1986). Averaging was performed for F_3, F_4, C_3, and C_4. These channel groups and their relation to the 10-20 International System are presented in Figure 4.3. The channel groups for the left, central, and right areas of the frontal region (those areas circled in Figure 4.3) as well as for the left, central, and right areas of the central region (those areas squared in Figure 4.3) were formed based on previous research investigating ERPs in response to nicotine (Buzzell, Fedota, Roberts, & McDonald, 2014). However, most studies examining auditory ERPs in response to nicotine analyze

single electrodes. Therefore, studies using auditory ERPs to investigate attentional processes were also used to form electrode channel groups (e.g. Beer & Röder, 2004; Gamble & Luck, 2011; Hötting, Rösler, & Röder, 2003) as was visual inspection of the grand average waveforms. These frontal groups correspond to F_3, F_z, and F_4 of the 10-20 International System and are therefore given these names in the current study. However, compared to previous literature the current study's grand average waveforms show the left and right areas of the central region to have maximal activation closer to the vertex. Therefore, the channel groups used for the central areas have been moved inward compared. For this reason, C_3 and C_4 of the 10-20 International System are not contained within the central region's left and right channel groups, respectively. However, because these groups approximately correspond to C_3 and C_4 they are given these names. C_z in the current study corresponds to C_z of the 10-20 International System and therefore is given this name. Figure 4.4 displays the grand average waveforms for all recording sites and delineates the channel groups used in this study. Furthermore, Figure 4.5 shows only those waveforms used in the current analysis and contains expanded waveforms on the periphery. These expanded waveforms are representative of each channel group and show the characteristic ERP components P1, N1, P2, and N2. However, because there are 4 conditions shown on each waveform, individual waveforms are also displayed on a large scale (see Figure 4.6 - Figure 4.11). This allows for close visual inspection before statistical analysis in order to identify differences between conditions.

Peak amplitudes in individual subject ERPs were found within the time window, which was defined by the group averaged ERPs and measured relative to the pre-stimulus baseline. Peak latency was calculated relative to the stimulus onset. The peak amplitude and latency from all electrodes in a channel group were averaged. Although the number of channels within channel groups vary (F_3, F_4, C_3, and C_4 are comprised of four channels each, while F_z and C_z consist of only one channel each) this did not affect the variance of the between-subjects condition (nicotine and placebo). This is illustrated in Table 4.7 – Table 4.10, showing that the standard deviation for amplitude and latency were similar between the nicotine and placebo groups.

For each individual participant the average amplitude was calculated for each component (P1, N1, P2, N2) in each region of interest (frontal/central; left, center, right). The mean and standard deviation of each variable was then calculated and any values that were more than three standard deviations away from the mean were removed (Howitt & Cramer, 2005). In total, 27 outliers were removed. The data was then analyzed by means of a repeated-measures analysis of variance (ANOVA). This same procedure was repeated for latency information. This led to 16 separate ANOVAs: 4 components X 2 ERP measurements X 2 scalp regions. For each ANOVA there were two within-subjects factors: 1) sound (high-pitched and low-pitched) and 2) area (left, right, and central). There was also one between-subject factor, nicotine condition (placebo or nicotine).

ERP differences were also analyzed in order to assess whether frequency range affected cortical responses. First, the low-pitched amplitude was subtracted from the high-pitched amplitude for each component and for each scalp region. The mean and standard deviation of each variable was then calculated and values more than three standard deviations away from the mean were removed (Howitt & Cramer, 2005). In total, 6 outliers were removed. These same calculations were performed for latency data. Then, the difference waveforms were analyzed using a repeated-measures ANOVA. This also led to 16 separate ANOVA, one performed for each component (4) X each ERP measurement (2) X each scalp region (2). For each ANOVA there was a within-subjects factor of area (left, right, and central) and a between-subject factor of nicotine condition (placebo or nicotine).

For all statistical analyses where variables were found to violate the assumption of sphericity a Greenhouse-Geisser correction was used. For post-hoc analyses a Bonferroni correction was employed. Where appropriate, one-way ANOVA tests and *t*-tests followed significant ($p < .05$) interactions and site effects.

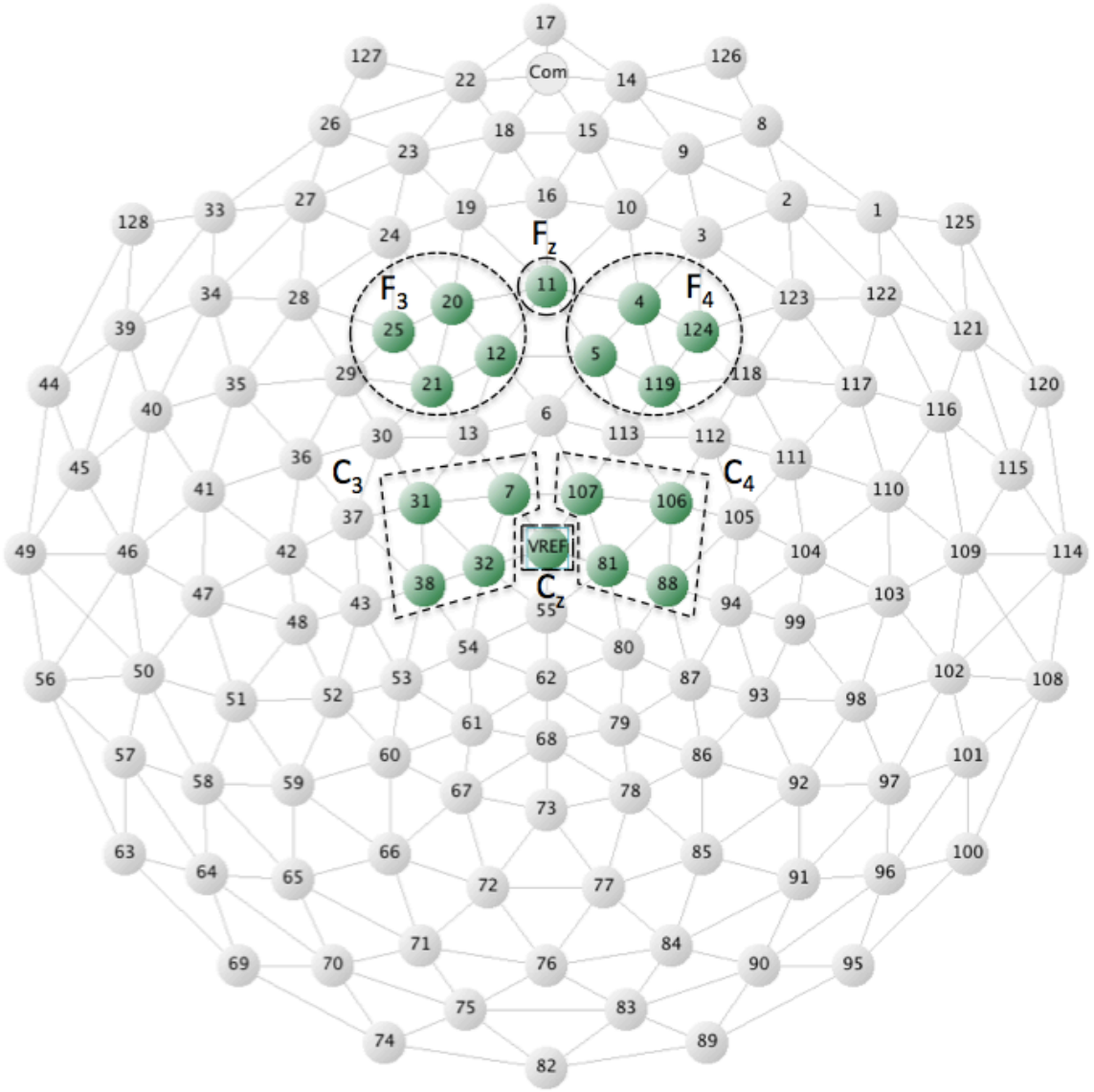

Figure 4.3. Channel groups selected from montage for averaging ERPs. A representation of the electrodes grouped together in the frontal region (upper circled channel groups) and central region (lower squared channel groups). These channel groups are further divided by hemisphere and midline. Their approximate locations that correspond to the 10-20 International System (Jasper, 1958) are labeled (e.g. F_3, F_z, F_4; C_3, C_z, C_4) next to each channel group.

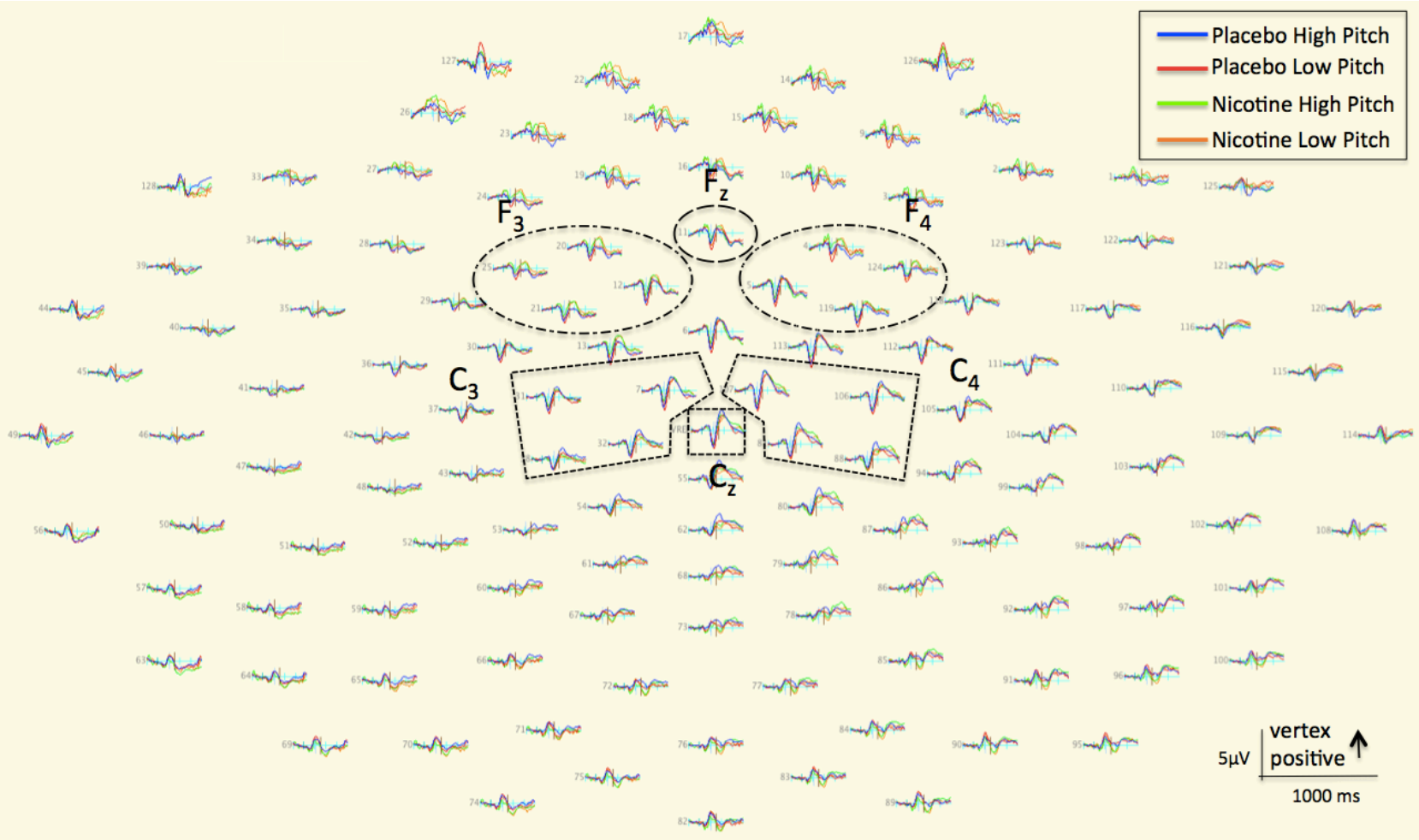

Figure 4.4. Illustration of grand average waveforms chosen and grouped together in the frontal region (circled channel groups) and central region (squared channel groups). Names of the 10-20 International System (Jasper, 1958) that correspond to these channel groups are also displayed (e.g. F_3, F_z, F_4; C_3, C_z, C_4).

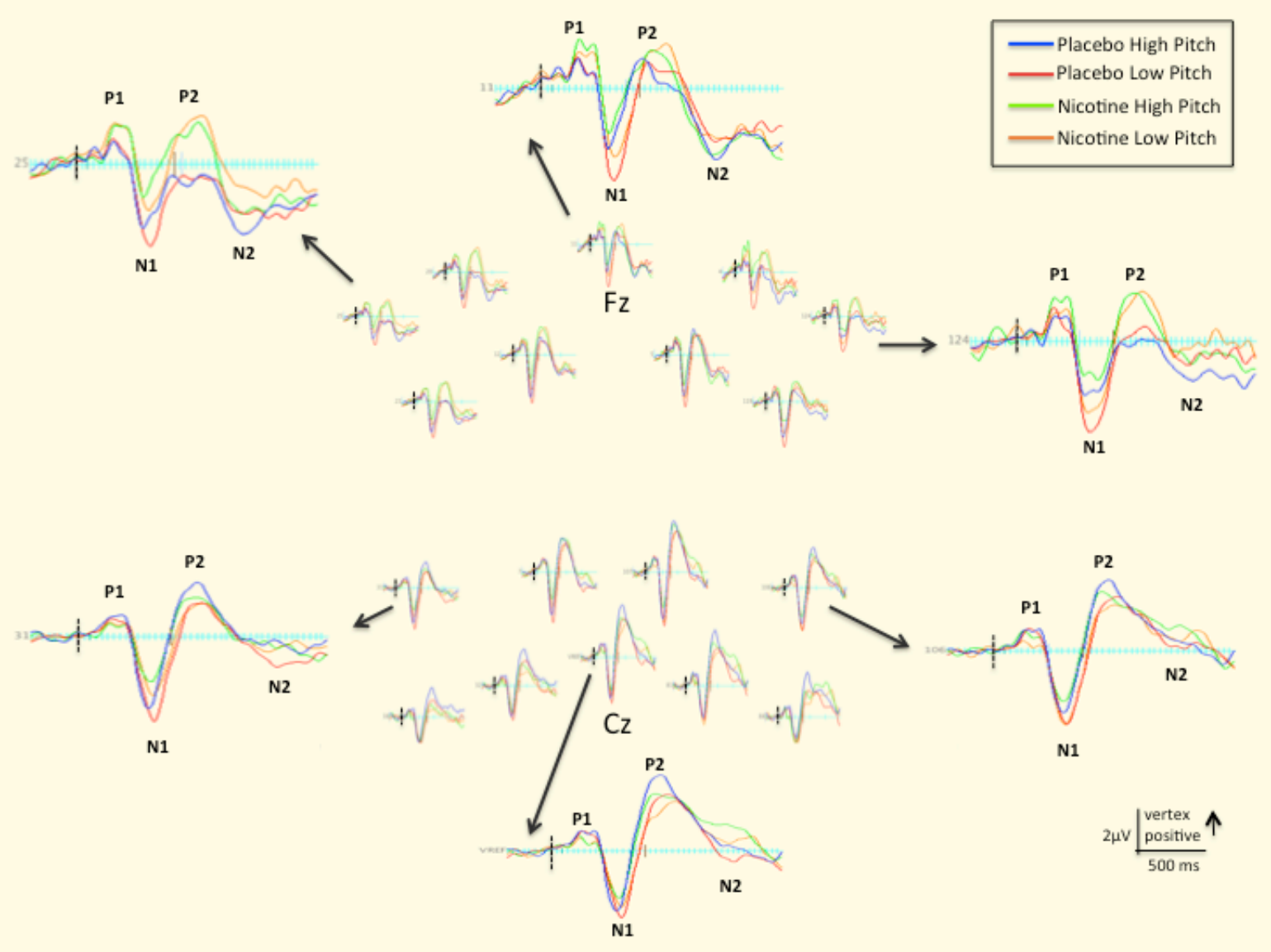

Figure 4.5. Figure of analysis montage showing channels/ channel groups used in analysis. The larger scale waveforms shown are representative waveforms for each channel/channel group. F_z and C_z are provided for reference.

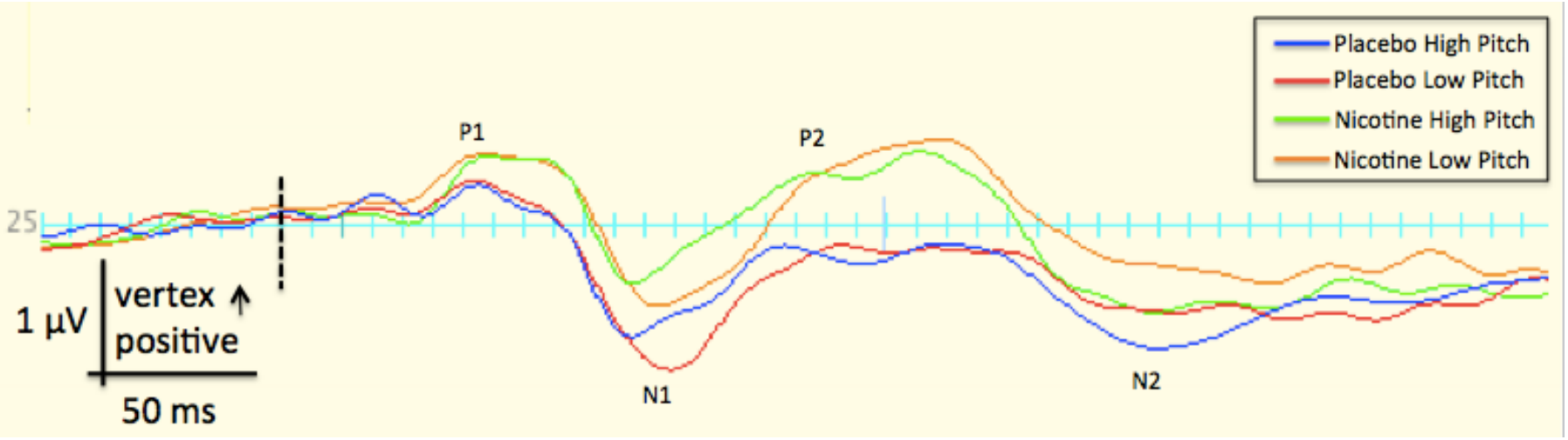

Figure 4.6. Waveform representative for F_3 channel group.

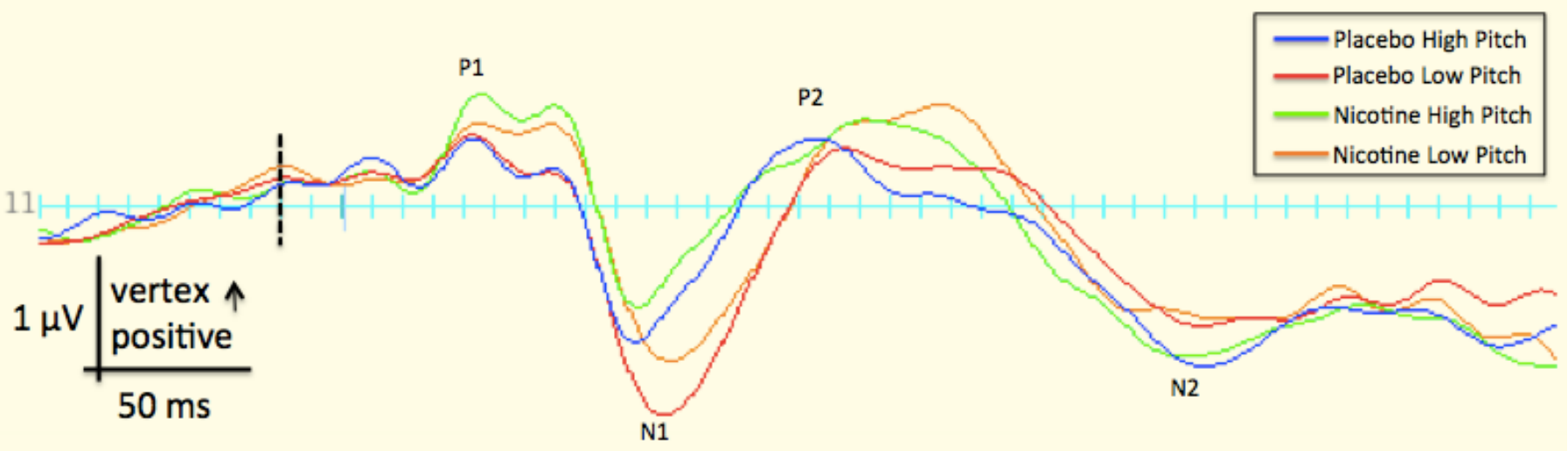

Figure 4.7. Waveform representative of F_z.

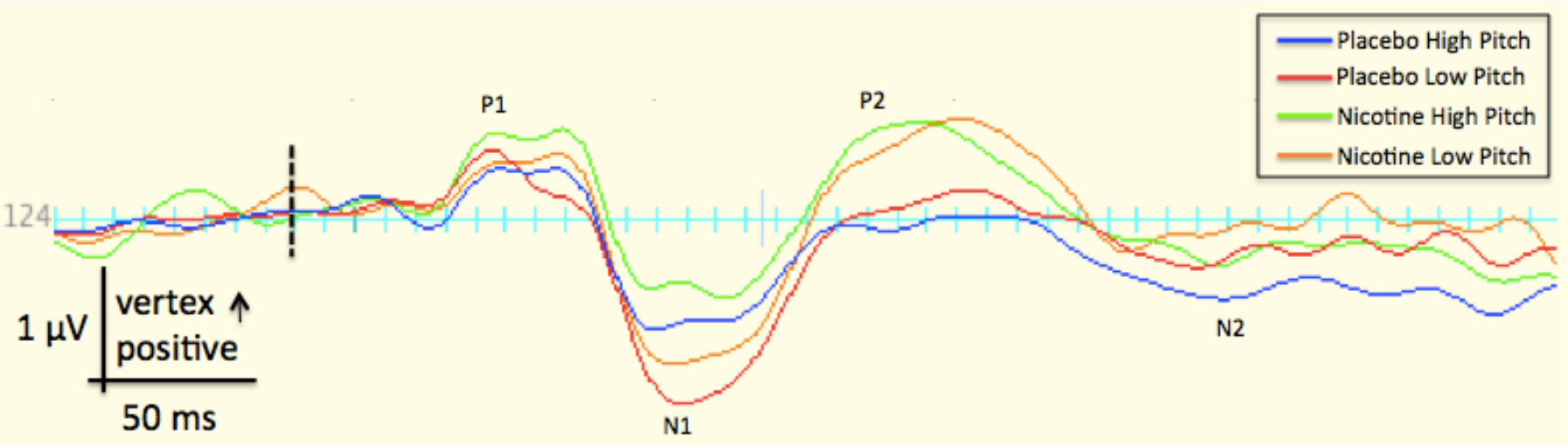

Figure 4.8. Waveform representative for F_4 channel group.

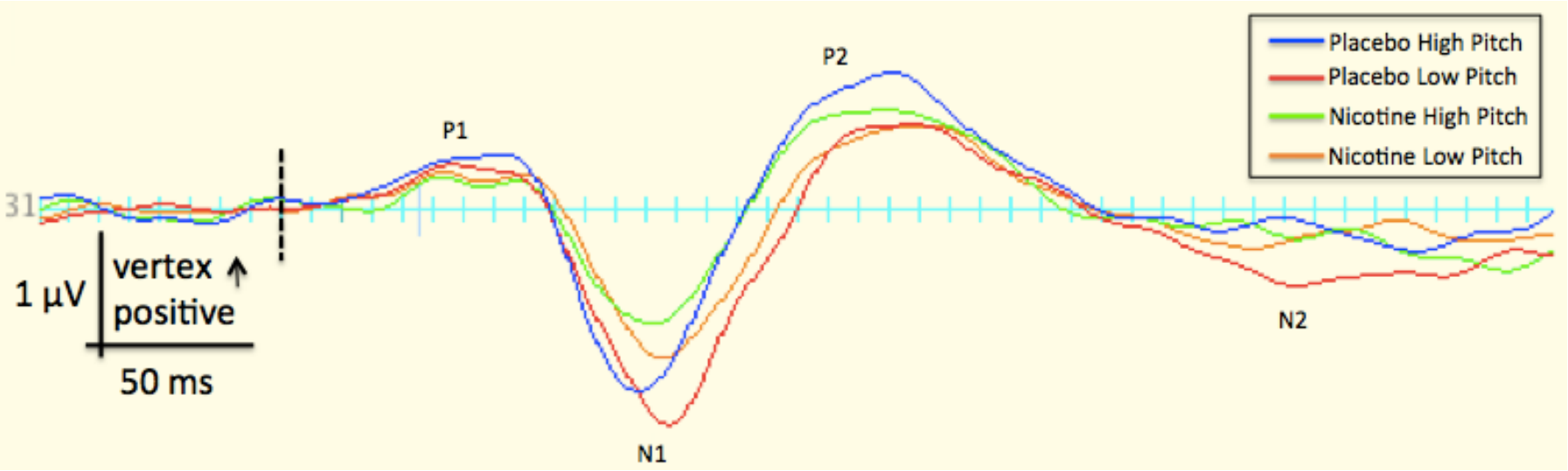

Figure 4.9. Waveform representative for C_3 channel group.

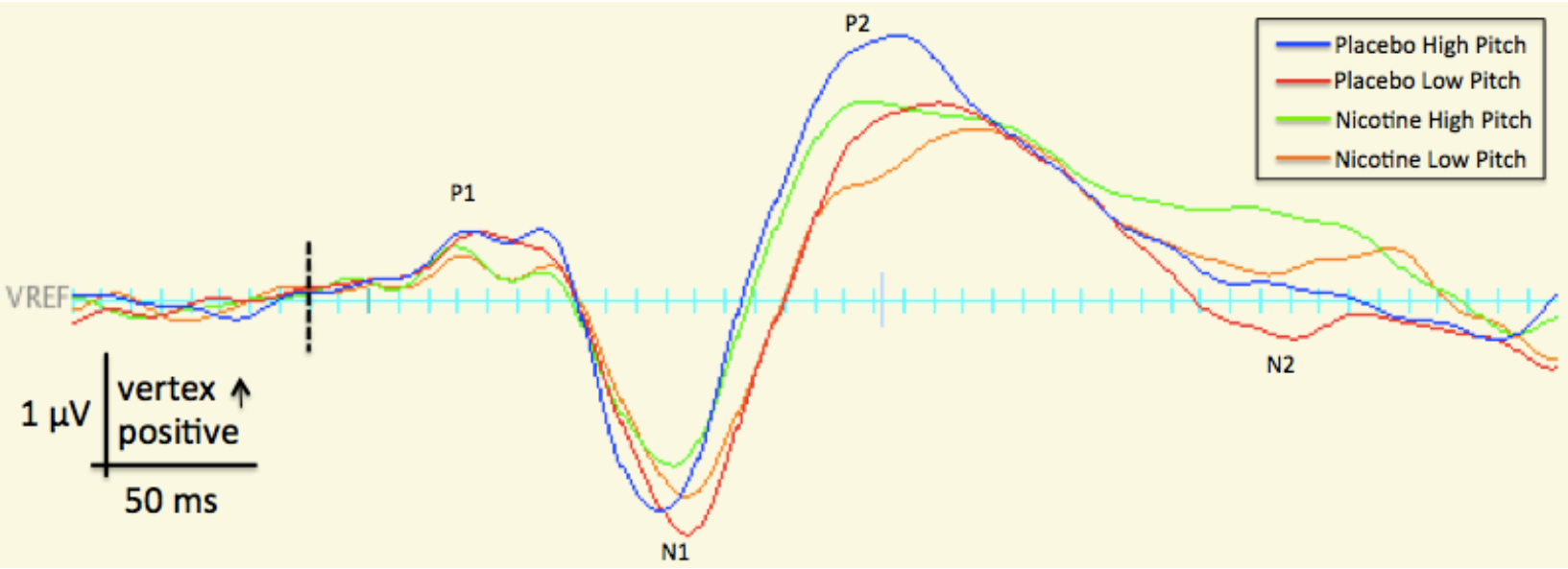

Figure 4.10. Waveform representative of C_z.

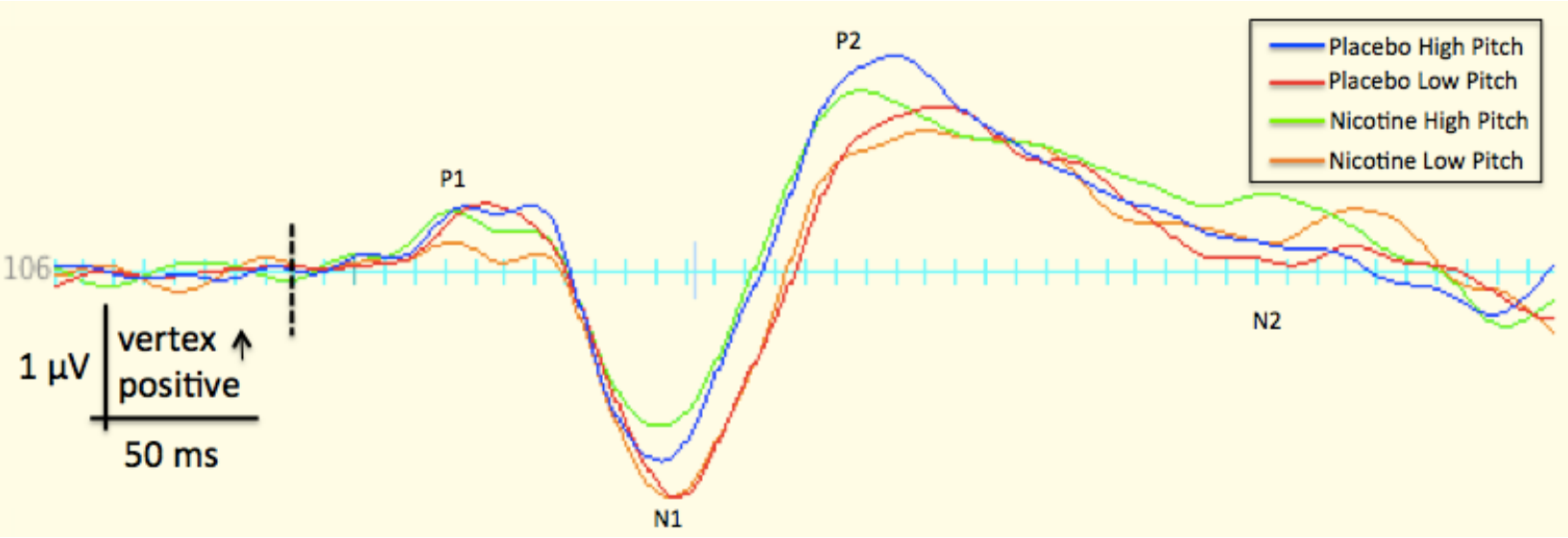

Figure 4.11. Waveform representative of C_4.

4.15. Results

4.16. Behavioral data

The mean reaction times for each group (e.g. placebo, nicotine) were compared across the four visual+audio conditions. These four conditions are show below in Table 4.5 and indicate a corresponding numeric label.

Table 4.5.

Four within subjects conditions and corresponding number label

Within Subjects Condition	Condition Label
↑↓ +	1
↑↓ +	2
↓↑ +	3
↓↑ +	4

Note: Indicates high-pitched tone and Indicates low-pitched tone

Indicates an image with an up arrow followed by a down arrow

Indicates an image with a down arrow followed by an up arrow

For each participant, responses representing errors (e.g., a response for an up arrow was pressed when a down arrow was shown), and outliers (e.g., responses greater than three standard deviations from each participant's mean) were removed from the analyses (Howitt & Cramer, 2005). One participant's behavioral data was not included due to equipment malfunction (e.g. data did not record). In general, the error rate was low for both groups. On average, those receiving placebo had an accuracy rate of 96%, while those receiving nicotine had an accuracy rate of 98%. The mean and standard deviations of correct reaction times were then calculated for each of the four visual+audio conditions. This information is shown in Table 4.3. Condition 1 in the placebo group was found to be kurtoic and therefore required the removal of one outlier

(e.g. a mean that was greater than 3 standard deviations from the mean). Next, a repeated measure ANOVA was performed in order to examine if there were any differences in reaction times between each group and between each condition. The between subjects variable was group (2 levels – placebo, nicotine) and the within subjects variable was visual+audio condition (4 levels – see Table 4.5 for levels). A multivariate test showed no significant difference in reaction time between the nicotine conditions, $F(3, 30) = 1.54$, $p = .224$, $\eta 2 = .13$. However, there was a clear trend showing that the reaction time was less for those receiving nicotine compared to those receiving placebo. This trend is shown in Figure 4.12.

Table 4.6.

Mean and standard deviation reaction times for each visual+audio condition by each nicotine condition

Condition	Placebo *M(SD)*	Nicotine *M(SD)*
1	642.95(220.08)	542.23(125.93)
2	635.77(183.80)	528.59(135.20)
3	635.95(200.78)	544.57(153.81)
4	655.91(222.65)	549.84(149.34)

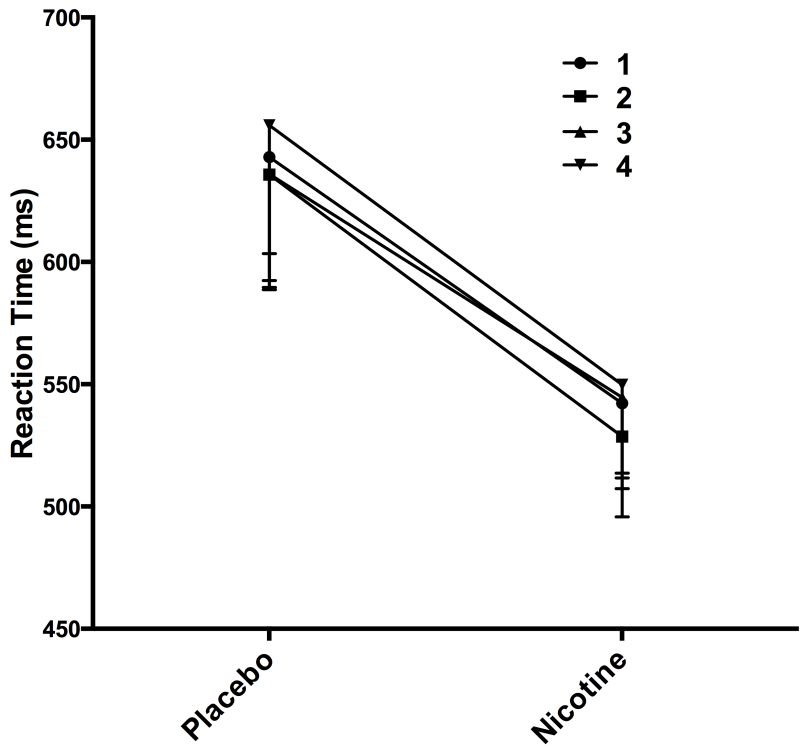

Figure 4.12. Mean and standard error for each of the visual+audio conditions by each nicotine condition

4.17. ERP data

The grand average waveforms for the high-pitched and low-pitched conditions at all recording sites are presented for the nicotine and control group in Figure 4.4. An expanded representative waveform can be viewed for the frontal areas, F3 (Figure 4.6), Fz (Figure 4.7), and F4 (Figure 4.8), as well as for the central areas, C3 (Figure 4.9), Cz (Figure 4.10), and C4 (Figure 4.11). Furthermore, the mean amplitude values for each component are displayed by condition and area. This is presented separately for the frontal (Table 4.7) and central (Table 4.8) regions. The latency values for each component are also displayed by condition and area, and again are presented separately for the frontal (Table 4.9) and central (Table 4.10) regions.

4.18. P1

The amplitude and latency of the ERP components P1, N1, P2, and N2 was selected for statistical analysis as previously described. The repeated measures ANOVA with 3 factors (pitch condition, area, and nicotine group) were performed separately for the frontal and the central regions, and revealed some significant findings. For P1 amplitude in the frontal region there was a main effect of area, $F(2, 52) = 6.21$, $p = .004$, $\eta^2 = .19$, whereby F_z was significantly larger ($M = .95$, $SE = .12$) than F_3 ($M = .73$, $SE = .10$), $p = .010$. For the P1 amplitude in the central region there was also a main effect of area, $F(2, 58) = 5.23$, $p = .008$, $\eta^2 = .15$, whereby C_z was also significantly larger ($M = .76$, $SE = .10$) than C_3 ($M = .62$, $SE = .08$), $p = .019$.

For the P1 latency in the frontal region there was a main effect of area, $F(2, 52) = 3.39$, $p = .041$, $\eta^2 = .12$. However, post-hoc tests reveal no significant differences between F_3 ($M = 46.85$, $SE = 2.00$) and F_z ($M = 49.01$, $SE = 1.82$), $p = .480$, between F_3 and F_4 ($M = 50.87$, $SE = 1.78$), $p = .106$, or between F_z and F_4, $p = .482$. For the P1 latency in the central region no significant effects were found.

In summary, P1 amplitude was larger at the midline (F_z and C_z) compared to the left and right hemispheres. However, these effects are not a result of nicotine or pitch as both variables resulted in nonsignificant findings. The main effect of area can be viewed in Table 4.7 and Table 4.8, which show P1 amplitude to be largest for F_z and C_z. They can also be view in topographic

form in Figure 4.13, which shows P1 amplitude to have maximal activation at F_z and C_z sites. P1 latency showed no significant differences in the frontal or central regions as a result of nicotine or pitch.

4.19. N1

For the N1 amplitude in the frontal region there was a main effect of pitch, $F(1, 28) = 6.20$, $p = .019$, $\eta^2 = .18$, whereby low pitch had a significantly larger amplitude ($M = -1.85$, $SE = .25$) than high pitch ($M = -1.47$, $SE = .15$). For the N1 amplitude in the central region there was a main effect of area, $F(2, 58) = 4.34$, $p = .018$, $\eta^2 = .13$, whereby C_z was significantly larger ($M = -1.92$, $SE = .22$) than C_3 ($M = -1.62$, $SE = .17$), $p = .027$. There was also an interaction effect of area and group, $F(2, 58) = 3.59$, $p = .034$, $\eta^2 = .11$. However, post-hoc tests revealed no significant differences between groups in C_3, $F(1, 30) = 1.43$, $p = .241$, C_z, $F(1, 30) = .52$, $p = .477$, or C_4, $F(1, 30) = .00$, $p = 1.00$.

For the N1 latency in the frontal region there was a main effect of pitch, $F(1, 28) = 18.81$, $p = .000$, $\eta^2 = .40$. High pitch had a significantly shorter latency ($M = 97.96$, $SE = 1.67$) than low pitch ($M = -104.04$, $SE = 1.29$). There was also a main effect of area, $F(2, 56) = 4.22$, $p = .020$, $\eta^2 = .13$, whereby F_4 was marginally significantly longer in latency ($M = 102.94$, $SE = 1.47$) than both F_z ($M = 100.60$, $SE = 1.52$), $p = .052$, and F_3 ($M = 99.47$, $SE = 1.49$), $p = .052$. For the N1 latency in the central region there was a main effect of pitch, $F(1, 29) = 24.59$, $p = .000$, $\eta^2 = .46$. High pitch ($M = 96.97$, $SE = 1.48$) was significantly shorter in latency than low pitch ($M = 103.52$, $SE = 1.08$). There was also a main effect of area, $F(1.34, 38.98) = 4.66$, $p = .027$, $\eta^2 = .14$, whereby C_3 was significantly shorter in latency ($M = 98.10$, $SE = 1.20$) than C_z ($M = 100.77$, $SE = 1.24$), $p = .024$. Lastly, there was a significant interaction effect of pitch and area, $F(2, 58) = 3.27$, $p = .045$, $\eta^2 = .10$. For all areas high pitch had a shorter latency than low pitch. Specifically, for C_3 high pitch had a shorter latency ($M = 95.48$, $SE = 1.37$) compared to low pitch ($M = 101.69$, $SE = 1.38$), $p = .000$. For C_z high pitch had a shorter latency ($M = 96.03$, $SE = 2.04$) compared to low pitch ($M = 105.48$, $SE = 1.22$), $p = .000$. For C_4 high pitch had a shorter latency ($M = 99.69$, $SE = 1.79$) compared to low pitch ($M = 104.18$, $SE = 1.56$), $p = .009$.

In summary, for N1 amplitude the frontal and central regions did not correspond on main effects. The frontal region showed a main effect of pitch. Table 4.7 shows N1 amplitude to be larger in the frontal region during low pitch for almost all conditions. The central region showed a main effect of area. Figure 4.14 topographically shows larger activation of N1 over the C_z site. For N1 latency both the frontal and central regions showed a main effect of pitch. Table 4.9 and Table 4.10, for the frontal and central regions respectively, show high pitch to have a shorter latency than low pitch for all areas.

4.20. P2

For the P2 amplitude in the frontal region there was a main effect of group, $F(1, 23) = 4.46$, $p = .046$, $\eta^2 = .16$. Nicotine had a significantly larger amplitude ($M = 1.68$, $SE = .25$) compared to placebo ($M = .97$, $SE = .23$). For the P2 amplitude in the central region there was a main effect of pitch, $F(1, 28) = 10.46$, $p = .003$, $\eta^2 = .27$. High pitch had a significantly larger amplitude ($M = 2.03$, $SE = .17$) compared to low pitch ($M = 1.62$, $SE = .15$). There was also a main effect of area, $F(1.49, 41.69) = 10.17$, $p = .001$, $\eta^2 = .27$, whereby C_3 ($M = 1.47$, $SE = .17$) was significantly smaller in amplitude compared to both C_z ($M = 2.07$, $SE = .17$), $p = .000$, and C_4 ($M = 1.94$, $SE = .16$), $p = .037$.

For the P2 latency in the frontal region there was a main effect of pitch, $F(1, 23) = 14.42$, $p = .001$, $\eta^2 = .39$, whereby high pitch had a significantly shorter latency ($M = 168.49$, $SE = 3.43$) than low pitch ($M = 178.87$, $SE = 3.30$). For P2 latency in the central region there was a main effect of pitch, $F(1, 28) = 8.26$, $p = .008$, $\eta^2 = .23$, where by high pitch also had a significantly shorter latency ($M = 174.04$, $SE = 2.76$) than low pitch ($M = 180.39$, $SE = 2.35$).

In summary, for P2 amplitude the frontal and central regions did not correspond on main effects. The frontal region showed a main effect of group. Table 4.7 shows P2 amplitude to be higher in the nicotine group for all conditions and areas compared to placebo. The central region showed a main effect of pitch and area. Table 4.8 shows P2 amplitude to be larger in the central region during high pitch for both nicotine and placebo groups. Figure 4.15 shows a smaller activation of P2 over the C_3 area. For P2 latency both the frontal and central regions showed a main effect of pitch. Table 4.9 and Table

4.10, for the frontal and central regions respectively, show high pitch to have a shorter latency than low pitch for all areas.

4.21. N2

For N2 amplitude in the frontal region there was a main effect of area, $F(1.39, 24.93) = 6.69$, $p = .010$, $\eta^2 = .27$, whereby F_z was significantly larger ($M = -1.61$, $SE = .25$) compared to F_4 ($M = -1.14$, $SE = .18$), $p = .000$. For N2 amplitude in the central region there was a main effect of area, $F(1.49, 40.30) = 10.08$, $p = .001$, $\eta^2 = .27$, whereby C_3 was significantly larger ($M = -0.40$, $SE = .11$) compared to both C_z ($M = -.10$, $SE = .16$), $p = .018$, and C_4 ($M = -0.80$, $SE = .17$), $p = .004$.

For N2 latency in the frontal region there was a main effect of area, $F(2, 34) = 3.36$, $p = .047$, $\eta^2 = .17$, whereby F_4 was significantly shorter in latency ($M = 266.56$, $SE = 3.08$) compared to F_z ($M = 272.41$, $SE = 3.64$), $p = .031$. For N2 latency in the central region there were no significant findings.

In summary, for N2 amplitude the frontal and central region both showed main effects of area, but did not correspond on the affected areas. Figure 4.16 displays this topographically with larger activation in F_z and C_3. For N2 latency only the frontal region showed significant effects. Table 4.9 shows overall F_4 to be shorter in latency.

Table 4.7.

Amplitude of ERP peaks in frontal region. Group mean values (mean ± SE) in μV

Group		High Pitch			Low Pitch	
ERP Peak	F_3	F_z	F_4	F_3	F_z	F_4
Placebo						
P1	.72 ± .22	.94 ± .23	.90 ± .17	.77 ± .16	.94 ± .19	.94 ± .18
N1	-1.79 ± .25	-1.62 ± .23	-1.74 ± .27	-2.01 ± .32	-2.22 ± .33	-2.22 ± .34
P2	.75 ± .23	1.31 ± .36	1.08 ± .34	.67 ± .23	1.12 ± .38	.99 ± .32
N2	-1.39 ± .29	-1.72 ± .38	-1.26 ± .29	-1.22 ± .25	-1.39 ± .38	-.97 ± .31
Nicotine						
P1	1.04 ± .22	1.43 ± .25	1.42 ± .28	.94 ± .22	1.11 ± .23	1.00 ± .23
N1	-1.15 ± .27	-1.50 ± .39	-1.32 ± .27	-1.34 ± .41	-1.72 ± .51	-1.73 ± .42
P2	1.40 ± .37	1.49 ± .50	1.46 ± .39	1.46 ± .40	1.40 ± .49	1.26 ± .35
N2	-1.20 ± .29	-1.95 ± .54	-.99 ± .36	-.93 ± .33	-1.68 ± .58	-.88 ± .37

Note: P1, N1, P2, and N2 are ERP components. F_3, F_z, F_4 and C_3, C_z, C_4 are electrode groups

Table 4.8.

Amplitude of ERP peaks in central region. Group mean values (mean ± SE) in μV

Group		High Pitch			Low Pitch	
ERP Peak	C_3	C_z	C_4	C_3	C_z	C_4
Placebo						
P1	.80 ± .19	.92 ± .22	.88 ± .16	.70 ± .13	.88 ± .16	.81 ± .13
N1	-1.80 ± .27	-2.23 ± .36	-1.85 ± .30	-2.01 ± .24	-2.30 ± .28	-2.01 ± .24
P2	1.82 ± .29	2.80 ± .39	2.56 ± .35	1.36 ± .27	2.16 ± .30	1.92 ± .26
N2	-.39 ± .18	-.13 ± .24	-.12 ± .21	-.59 ± .20	-.41 ± .26	-.10 ± .28
Nicotine						
P1	.60 ± .11	.76 ± .15	.83 ± .12	.59 ± .12	.68 ± .16	.63 ± .15
N1	-1.42 ± .24	-1.78 ± .36	-1.75 ± .30	-1.55 ± .31	-1.99 ± .52	-2.11 ± .43
P2	1.40 ± .23	2.36 ± .40	2.26 ± .32	1.17 ± .22	1.89 ± .28	1.81 ± .27
N2	-.38 ± .16	.42 ± .31	.56 ± .35	-.55 ± .23	.08 ± .27	.24 ± .30

Note: P1, N1, P2, and N2 are ERP components. F_3, F_z, F_4 and C_3, C_z, C_4 are electrode groups

Table 4.9.

Latency of ERP peaks in frontal region. Group mean values (mean ± SE) in μV

Group		High Pitch			Low Pitch	
ERP Peak	F_3	F_z	F_4	F_3	F_z	F_4
Placebo						
P1	42.97 ± 2.79	44.75 ± 3.19	50.30 ± 2.96	45.14 ± 2.88	48.75 ± 3.14	46.72 ± 2.55
N1	96.27 ± 2.68	97.44 ± 2.63	99.58 ± 2.75	105.20 ± 2.12	103.25 ± 2.02	106.13 ± 2.03
P2	164.55 ± 4.89	158.88 ± 5.19	168.61 ± 4.45	172.31 ± 4.90	173.94 ± 5.13	178.06 ± 4.22
N2	271.23 ± 4.61	277.81 ± 4.32	272.92 ± 3.92	274.27 ± 4.43	279.25 ± 4.63	274.17 ± 4.01
Nicotine						
P1	50.34 ± 2.93	52.13 ± 3.22	51.20 ± 2.42	50.19 ± 3.39	54.81 ± 3.09	57.66 ± 2.80
N1	96.27 ± 2.44	100.06 ± 2.73	102.14 ± 2.35	102.86 ± 2.21	103.81 ± 2.27	105.45 ± 1.85
P2	172.22 ± 4.61	172.25 ± 5.63	172.98 ± 4.24	175.36 ± 4.66	177.25 ± 5.53	182.94 ± 3.08
N2	271.13 ± 4.59	271.75 ± 4.92	270.59 ± 4.48	276.88 ± 4.00	274.69 ± 4.83	268.94 ± 4.75

Note: P1, N1, P2, and N2 are ERP components. F_3, F_z, F_4 and C_3, C_z, C_4 are electrode groups

Table 4.10.

Latency of ERP peaks in central region. Group mean values (mean ± SE) in μV

Group		High Pitch			Low Pitch	
ERP Peak	C_3	C_z	C_4	C_3	C_z	C_4
Placebo						
P1	44.72 ± 2.65	44.13 ± 3.49	48.47 ± 2.72	44.47 ± 3.25	42.19 ± 3.02	43.53 ± 2.78
N1	94.86 ± 1.63	95.31 ± 2.77	100.00 ± 2.34	101.61 ± 1.97	105.31 ± 1.69	106.09 ± 1.81
P2	170.88 ± 4.01	172.63 ± 4.56	176.13 ± 3.11	178.31 ± 3.78	180.13 ± 3.85	180.84 ± 3.31
N2	274.03 ± 4.85	283.63 ± 4.72	280.89 ± 5.02	279.78 ± 4.12	285.63 ± 4.58	283.38 ± 3.77
Nicotine						
P1	41.72 ± 3.17	37.06 ± 3.05	40.41 ± 3.16	46.28 ± 3.37	40.56 ± 3.45	41.23 ± 3.24
N1	96.09 ± 2.24	97.06 ± 2.90	99.38 ± 2.78	101.77 ± 2.00	106.31 ± 1.83	102.27 ± 2.50
P2	174.30 ± 4.39	178.19 ± 5.87	174.27 ± 3.51	176.92 ± 4.18	186.19 ± 3.93	180.38 ± 3.78
N2	270.19 ± 4.61	267.06 ± 5.32	272.33 ± 4.70	274.02 ± 5.13	277.00 ± 4.55	275.69 ± 4.29

Note: P1, N1, P2, and N2 are ERP components. F_3, F_z, F_4 and C_3, C_z, C_4 are electrode groups

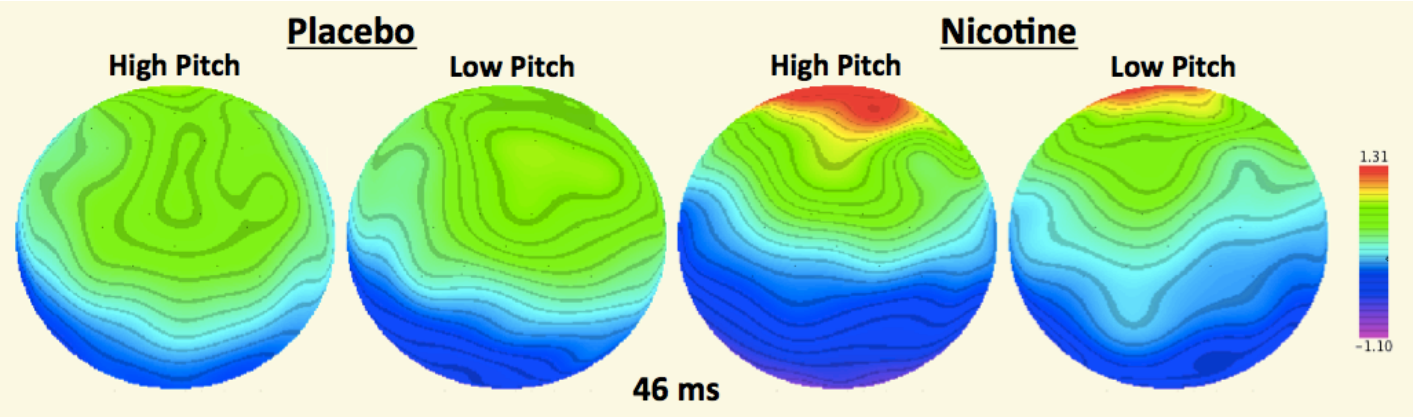

Figure 4.13. Topographic ERP for P1 peak. Activation map captured at 46 ms for all pitch and nicotine conditions. All images are shown from a top viewpoint.

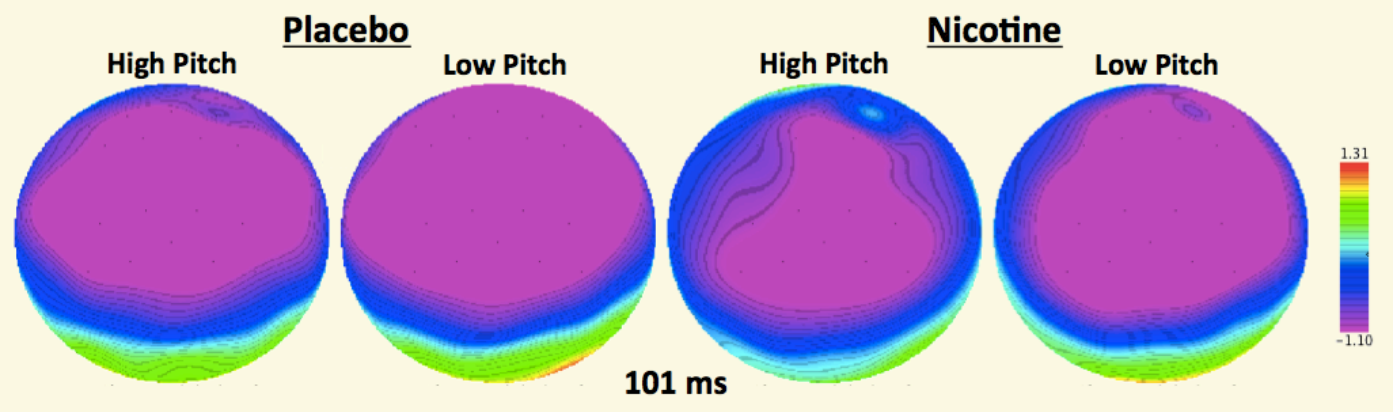

Figure 4.14. Topographic ERP map for N1 peak. Activation map captured at 101 ms for all pitch and nicotine conditions. All images are shown from a top viewpoint.

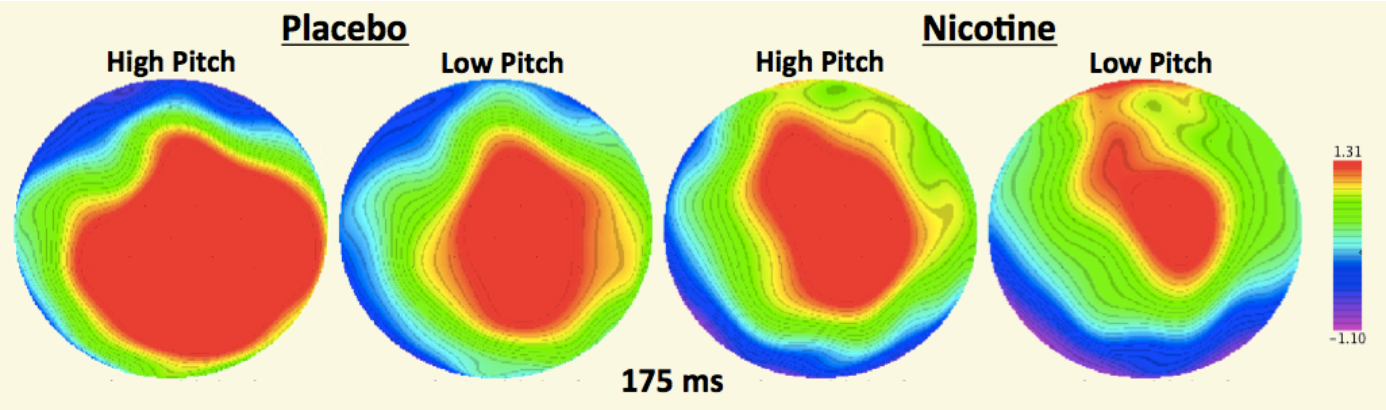

Figure 4.15. Topographic ERP map for P2 peak. Activation map captured at 175 ms for all pitch and nicotine conditions. All images are shown from a top viewpoint.

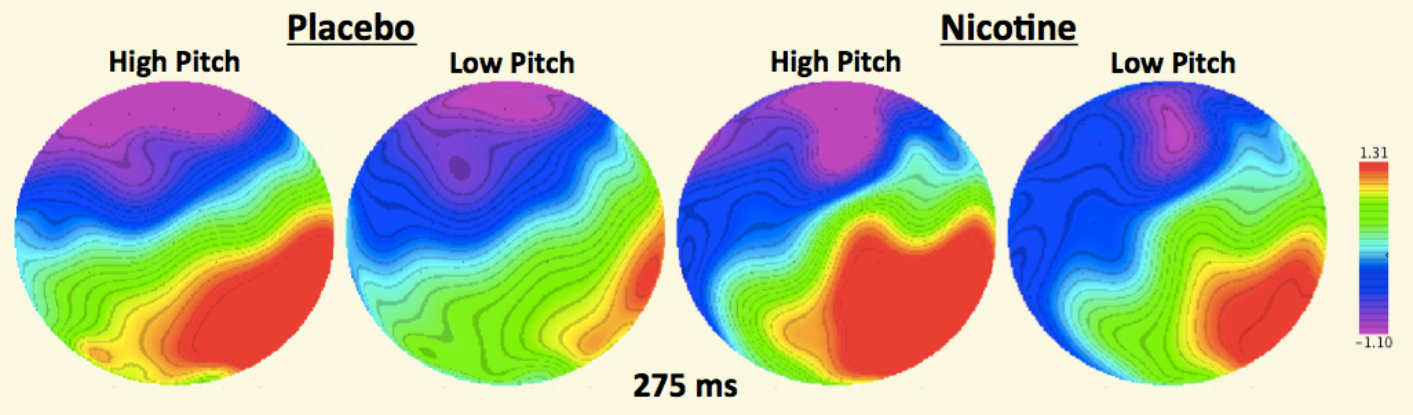

Figure 4.16. Topographic ERP map for N2 peak. Activation map captured at 275 ms for all pitch and nicotine conditions. All images are shown from a top viewpoint.

4.22. ERP differences

The mean amplitude and latency values for the ERP differences for each component are displayed by condition and area. This is presented separately for the frontal (Table 4.11) and central (Table 4.12) regions.

4.23. P1

For P1 amplitude in the frontal region there was a main effect of nicotine condition, $F(1, 29) = 5.63$, $p = .025$, $\eta^2 = .16$, whereby nicotine ($M = .29$, $SE = .13$) had a significantly larger amplitude than placebo ($M = -.17$, $SE = .14$). No significant interaction effect of area and nicotine condition was found. For P1 amplitude in the central region there were no significant findings.

For P1 latency in the frontal region there were no significant findings. From P1 latency in the central region there were also no significant findings.

In summary, only P1 amplitude in the frontal region showed a significant difference in ERP waves, with the nicotine group showing a significantly larger difference in amplitude between the frequency ranges (e.g. high-pitch and low-pitch) than placebo.

4.24. N1

For N1 amplitude in the frontal region there was a main effect of area, $F(2, 60) = 4.15$, $p = .021$, $\eta^2 = .12$, whereby F_4 ($M = .45$, $SE = .15$) was significantly larger in amplitude compared to F_z ($M = .17$, $SE = .13$), $p = .049$. No significant interaction effect was found. For N1 amplitude in the central region there were no significant findings.

For N1 latency in the frontal region there were no significant findings. For N1 latency in the central region there was a main effect of area, $F(2, 60) = 3.28$, $p = .045$, $\eta^2 = .10$, whereby C_z ($M = -9.63$, $SE = 2.27$) was significantly shorter in latency compared to C_4 ($M = -4.50$, $SE = 1.61$), $p = .045$. No significant interaction effect of area and nicotine condition was found.

In summary, ERP differences for the N1 waveform did not correspond between the frontal and central regions. That is, the difference in N1 amplitude between the frequency ranges was significantly larger in the frontal area for F_4 compared to F_z, while N1 latency difference between the frequency ranges was significantly shorter in the central area for C_z compared to C_4.

4.25. P2

For P2 amplitude in the frontal region there were no significant findings. For P2 amplitude in the central region there were also no significant findings.

For P2 latency in the frontal region there were no significant findings. From P2 latency in the central region there were also no significant findings.

4.26. N2

For N2 amplitude in the frontal region there were no significant findings. or N2 amplitude in the central region there were also no significant findings. For N2 latency in the frontal region there were no significant findings. From N2 latency in the central region there were also no significant findings.

Table 4.11.

Amplitude and latency of ERP differences in frontal region. Group mean values (mean ± SE) in μV

Group		Differences Amplitude			Differences Latency	
ERP Peak	F_3	F_z	F_4	F_3	F_z	F_4
Placebo						
P1	-.17 ± .14	.01 ± .24	-.03 ± .19	-1.90 ± 2.34	-4.00 ± 4.10	4.67 ± 3.38
N1	.20 ± .16	.60 ± .22	.51 ± .20	-8.73 ± 2.68	-5.81 ± 2.62	-6.17 ± 2.33
P2	.06 ± .21	.19 ± .21	.05 ± .24	-5.96 ± 3.64	-15.06 ± 5.12	-8.77 ± 3.68
N2	-.23 ± .28	-.16 ± .18	-.32 ± .20	-2.10 ± 3.12	-1.44 ± 5.73	.88 ± 3.96l
Nicotine						
P1	.09 ± .14	.32 ± .16	.45 ± .15	.83 ± 2.69	-2.69 ± 3.81	-4.77 ± 2.69
N1	.13 ± .21	.22 ± .30	.39 ± .22	-6.79 ± 2.29	-3.75 ± 2.24	-3.31 ± 1.89
P2	-.08 ± .22	.09 ± .26	.22 ± .16	-5.60 ± 3.67	-5.00 ± 6.35	-9.33 ± 4.21
N2	-.27 ± .15	-.127 ± .17	-.11 ± .20	-4.35 ± 4.21	-2.94 ± 5.08	3.85 ± 2.87

Note: P1, N1, P2, and N2 are ERP components; F_3, F_z, F_4 are electrode groups

Table 4.12.

Amplitude and latency of ERP peaks in central region. Group mean values (mean ± SE) in μV

Group		Difference Amplitude			Difference Latency	
ERP Peak	C_3	C_z	C_4	C_3	C_z	C_4
Placebo						
P1	.10 ± .13	.05 ± .17	.07 ± .13	.25 ± 3.51	1.94 ± 3.97	4.94 ± 3.46
N1	.20 ± .16	.07 ± .23	.16 ± .18	-6.75 ± 1.93	-10.00 ± 3.38	-6.09 ± 2.32
P2	.46 ± .20	.63 ± .23	.64 ± .20	-7.44 ± 2.56	-7.50 ± 3.76	-4.72 ± 3.43
N2	.19 ± .18	.28 ± .19	.22 ± .18	-5.75 ± 4.14	-2.00 ± 5.32	-2.48 ± 5.69
Nicotine						
P1	.01 ± .11	.09 ± .17	.09 ± .14	-4.56 ± 4.65	-3.50 ± 3.43	-.83 ± 2.28
N1	-.04 ± .14	.21 ± .27	.37 ± .21	-5.67 ± 1.48	-9.25 ± 3.03	-2.89 ± 2.23
P2	.23 ± .16	.47 ± .21	.46 ± .19	-2.63 ± 4.35	-8.00 ± 5.10	-3.18 ± 2.40
N2	.18 ± .16	.35 ± .18	.32 ± .15	-3.83 ± 3.75	-9.94 ± 5.03	-3.36 ± 4.65

Note: P1, N1, P2, and N2 are ERP components; C_3, C_z, C_4 are electrode groups

4.27. Discussion

The purpose of this study was to determine if nicotine was able to enhance auditory information processing, and if so, to identify which cognitive mechanisms were responsible for this enhancement. This was investigated in order to help explain the co-consumption of nicotine and music listening. I therefore tested the effects of nicotine on the neural responses implicated in pitch perception. To test this, electrophysiological responses to high-pitched and low-pitched auditory stimuli were compared between those receiving nicotine and placebo. In some cases differences between the groups provided evidence that nicotine can affect pitch perception in nonsmokers. However, pitch was found to influence electrophysiology more consistently, typically showing high-pitched sounds to elicit larger and faster responses than low-pitched sounds.

4.28. Reaction time

In line with the hypothesis that nicotine would result in enhanced arousal and attention, and therefore an improvement in auditory information processing, and based on previous behavioral experiments showing nicotine-induced enhancement on reaction time during tracking and working memory tasks (Ernst, Heishman, et al., 2001; Heishman et al., 1994), behavioral performance/reaction time was expected to decrease during the decision-making task in those receiving nicotine. Although the findings were not statistically significant and therefore did not support the hypothesis, there is a trend showing a shorter reaction time during task performance for those receiving nicotine. Previous research has shown no difference in reaction time when comparing nicotine and placebo conditions in nonsmokers (Hindmarch et al., 1990; Kerr et al., 1991), however the trend found in the current suggests that with more statistical power these results may become significant. This may suggest that nonsmokers can experience improvement in auditory information processing during a decision-making task when receiving nicotine, but that nicotine only provides mild improvements.

4.29. P1

An increase in P1 amplitude as well as a decrease in P1 latency is thought to be indicative of enhanced arousal, which leads to improved primary

auditory pathway transmission (Harkrider & Champlin, 2001; Le Houezec et al., 1994) and increased sensitivity to sensory input (Knott, 1985b). Therefore, the current study predicted that P1 would increase in amplitude and decrease in latency as a result of nicotine. The findings did not support these hypotheses and showed no effect of nicotine on P1 amplitude or latency. However, there was an overall increase in P1 amplitude in the midline areas of the frontal and central regions of the scalp (F_z and C_z), regardless of nicotine or pitch. This is consistent with previous research showing the auditory P1 to have maximal amplitude over these areas (Key et al., 2005).

Although some previous research has found nicotine to increase P1 amplitude in smokers (Knott, 1985b) and nonsmokers (Harkrider & Champlin, 2001) the effect overall has been weak and inconsistent across studies (Friedman & Meares, 1980). The findings of the current study suggest that nicotine does not enhance arousal, auditory transmission, or sensitivity to auditory stimuli in nonsmokers. It may be that nonsmokers do not experience these improvements from nicotine because they are not in a state of nicotine withdrawal, as are abstaining smokers. This is turn means that nonsmokers are not experiencing a decrease in arousal and cognition before receiving nicotine and so do not benefit from the arousing effects of the drug. Furthermore, the results of this study run parallel to those found in study one, where nonsmokers reported a decrease in self-reported arousal as well as a decrease in positive affect (e.g. decrease in happiness and pleasure). This further suggests that for nonsmokers nicotine does not result in an increase in arousal. However, study one did show a (nonsignificant) trend for nicotine to increase heart rate in nonsmokers. This suggests that there may be a potential for nicotine to increase physiological arousal in nonsmokers. Overall, the current study's findings, in conjunction with the findings from study one, suggest that, at least for nonsmokers, nicotine does not increase arousal enough to enhance auditory perception.

4.30. N1

An increase in N1 amplitude is indicative of an enhancement of selective attention (Hillyard et al., 1973), while a decrease in N1 latency is related to more efficient information processing of stimuli (Domino & Kishimoto, 2002;

Friedman, Horvath, et al., 1974). Therefore, the current study predicted N1 to increase in amplitude and decrease in latency as a result of nicotine. The findings do not support these hypotheses as no effect of nicotine was found on N1 amplitude or latency. No changes in N1 have been reported in other nicotine studies examining nonsmokers (Harkrider & Champlin, 2001; Knott, Bolton, et al., 2009; Knott, Shah, et al., 2009). This suggests that for nonsmoking populations nicotine may not affect selective attention. However, pitch was shown to affect N1 amplitude and latency. That is, low pitch resulted in a larger N1 amplitude compared to high pitch in the frontal area. This is in contrast to previous research showing the N1 amplitude to increase for high pitch (Domino & Kishimoto, 2002) and high intensity sounds (Knott, 1985b). However, the results of these studies were observed with abstaining smokers, which further suggest that nonsmokers react to nicotine and auditory stimuli differently than smokers. Furthermore, high pitch was shown to have a shorter N1 latency compared to low pitch in both the frontal and central regions. Interestingly, a previous study has found a similar result for abstaining smokers, but not for nonsmokers (Domino & Kishimoto, 2002). This result suggests there to be a more efficient processing for high-pitched sounds compared to low-pitched sounds.

4.31. P2

A decrease in P2 amplitude and latency is related to habituation processes (Rust, 1977) and therefore indicative of more efficient processing (Domino & Kishimoto, 2002) and an enhanced ability to disengage from irrelevant stimuli (Knott, 1985a, 1989). Therefore, the current study predicted P2 to decrease in both amplitude and latency as a result of nicotine. The findings did not support these hypotheses and instead contradicted previous research by showing nicotine to increase P2 amplitude in the frontal region as well as showing no effect of nicotine on P2 latency. The increase in P2 amplitude in the frontal region suggests that in nonsmokers nicotine may cause a lack of habituation, resulting in nonsmokers being unable to adapt to repeated stimuli. Alternatively, it may suggest that nicotine results in a less efficient processing of information in nonsmokers. This may suggest that nicotine actually results in a cognitive impairment for nonsmokers.

In the central region P2 amplitude was found to be larger for high-pitched compared to low-pitched stimuli. This is a similar to the findings of Harkrider and Champlin (2001) who found the P2-N2 amplitude to increase for nonsmokers with high-intensity stimuli compared to low-intensity stimuli. This suggests high-intensity stimuli to increase cortical responsiveness. It may be that high-pitched sounds are difficult to habituate to. This is based on similar findings from Knott (1985b), who suggested high intensity sounds to be difficult to ignore because they override selective mechanisms (Picton, Campbell, Baribeau-Braun, & Proulx, 1978). Since high-pitched sounds are physically louder than low-pitched sounds (Contours, 2003), they may also override other attentional processes as well, such as habituation, and therefore increase P2 amplitude.

For P2 latency both the frontal and central regions resulted in a shorter latency for high-pitched compared to low-pitched sounds. Similar findings were reported by Domino and Kishimoto (2002), who found an increase in P2 amplitude as a result of irrelevant, high-pitched tones. These results may suggest that high-pitched sounds are processed faster than low-pitched sounds, and therefore processed more efficiently.

With no effect of nicotine on N1 amplitude, N1 latency, and P2 latency, and with an increase in P2 amplitude in the frontal region the results of this study contradict the most consistent findings of past research, which is that nicotine enhances selective attention and habituation processes. These results suggest that nonsmokers experience no change in selective attention and experience decrements in stimulus filtering and habituation processes as a result of nicotine intake. From these results it may be that the effects of nicotine on selective attention are more a reflection of withdrawal reversal, which returns abstaining smokers' cognition to baseline, than genuine and absolute cognitive enhancement. However, this study did not test abstaining smokers, so this statement can only be speculative.

4.32. N2

The N2 component is inversely related to arousal (Picton & Hillyard, 1974; Picton et al., 1974) and therefore reduced during states of high activation (Knott, 1989). For this reason the N2 component was predicted to decrease in

amplitude and latency in response to nicotine administration. The findings did not support these hypotheses, showing no effect of nicotine on N2 amplitude or latency. Past research examining nonsmokers found similar results (Knott, 1985b; Knott et al., 1995). This may suggest that the N2 is more associated with response inhibition, as proposed by go/nogo tasks (Jodo & Kayama, 1992). However, since this experimental paradigm was not used in the current study, response inhibition could not be tested. The results of the current study may further suggest that in nonsmokers, nicotine does not affect response inhibition.

Given the increase seen in P2 amplitude, indicating a decrease in habituation as a result of nicotine, people may smoke and listen to music because they do not experience a drop in emotional responses music when consuming nicotine. That is, music is repetitive by nature (Huron, 2006; Margulis, 2012) and past research has shown that familiarity with music, which is achieved through repetition, is a critical factor for emotional engagement with music (Pereira et al., 2011). Therefore, nicotine may help stop smokers or other nicotine consumers from disengaging with music's repetitive elements by decreasing habituation. This is turn, may lead to more emotional engagement with music during nicotine consumption.

4.33. ERP differences

There was a greater difference in P1 amplitude between frequency ranges for those receiving nicotine compared to those receiving placebo. From Table 4.11 it is clear that for the placebo group low-pitched tones were higher in P1 amplitude than high-pitched tones (indicated by a negative value). Interestingly, this relationship was inversed for the nicotine group, which showed high-pitched tones to be larger in P1 amplitude than low-pitched tones. However, this difference was not significant, as there was no main effect of pitch on the P1 amplitude for either group in the main ERP analysis. As P1 is implicated in arousal, this result may suggest that in nonsmokers nicotine is able to enhance arousal at high frequency ranges, but not at low frequency ranges. Nicotine was not shown to affect any other ERP components in terms of amplitude or latency.

4.34. Limitations and future research

Future research may be interested in expanding on this study's findings in two important ways. First, in order to better understand whether nicotine's effects on cognition are due to withdrawal reversal or due to a true enhancement future studies should compare the effects of nicotine on smokers, abstaining smokers, and nonsmokers within a single experiment. This would help to directly compare the behavioral and electrophysiological effects of nicotine on each cohort within a single study that uses the same methodology. That is, in the current study claims cannot be made as to how well nonsmokers perform on a task compared to smokers because both populations were not studied together. Therefore, any conclusions comparing these populations are speculative because these comparison is made between two or studies, which have used different methodologies to study the effects of nicotine.

Second, cigarettes are falling out of fashion thanks to the popularized e-cigarette (Loughead, 2015). E-cigarettes work by inhaling a heated liquid that usually contains nicotine and flavoring, as well as propylene glycol and glycerol (McRobbie, Bullen, Hartmann-Boyce, & Hajek, 2014). In this case, future experiments may be interested in using this method of nicotine administration because it most accurately imitates the act of smoking a real cigarette. This would increase the ecological validity for nicotine studies using a cigarette-smoking population. Furthermore, the growing popularity of e-cigarettes means that there is a part of the smoking population using this method of delivery in everyday life. For this reason, future experiments may also be interested in examining the cohort of smokers who use e-cigarettes compared to those who use tobacco products. E-cigarettes users may respond differently to nicotine since e-cigarettes deliver the drug at a much slower and lower rate than regular cigarettes, which can result in lower absorption of the drug (Farsalinos et al., 2014; Schroeder & Hoffman, 2014). This difference in delivery and absorption may lead to different cognitive and electrophysiological responses and may ultimately affect consumers' preferences for certain nicotine products.

The use of 2 mg nicotine gum on nonsmokers must also be considered. Study one clearly showed that for nonsmokers, 4 mg of nicotine decreased happiness and pleasure ratings more than 2 mg of nicotine. Additionally, 2 mg of nicotine increased HR more than 4 mg of nicotine. This suggests that for

nonsmokers, 2 mg of nicotine may increase physiological arousal and self-reports of happiness and pleasure more than 4 mg of nicotine. This in turn may suggest that nonsmokers' electrophysiological responses to auditory stimuli may be more enhanced by a low dose of nicotine. In light of this, future research may be interested in examining both high and low doses of nicotine, as well as placebo, in order to gain a more comprehensive understanding of how nicotine affects auditory perception in nonsmokers.

5. Chapter five: Discussion

5.1. Summary of studies

In this thesis a series of studies was conducted in order to better understand why nicotine and music are often consumed together. I conjectured that nicotine is consumed in the context of music because arousal, pleasure, or both are significantly increased by their co-consumption and that this increase was beyond that which would be experienced independently by either stimulus.

Understanding the relationship between nicotine and music consumption may help devise a non-nicotine replacement therapy for those wishing to stop smoking or help dissuade those who are considering the habit. It may be that music in general or music of a specific emotional category can decrease stress and increase pleasure, and therefore help with nicotine withdrawal symptoms. Also, if similar physiological changes are found to occur in response to nicotine and music this information could be used to teach adolescents, those most enticed by nicotine products, that listening to music is equally as arousing as nicotine. This may dissuade them from taking up smoking.

Understanding why nicotine and music are co-consumed can also potentially help explain why drug consumption in general is so prevalent in a musical setting. It may be that drugs and music enhance emotional reactions and therefore encourage their co-consumption. It may also be that drugs, including nicotine, facilitate the processing of auditory information. This in turn may allow listeners to better understand music and enhance their emotional reactions to music.

To test the relationship between nicotine and music, study one attempted to induce physiological arousal/pleasure via nicotine administration, then asked participants to listen to four types of music that varied in valence (positive, negative) and arousal (high, low). I hypothesized that with the ingestion of nicotine and subsequent action of music listening that there would be an additive effect on the physiological indices and self-reported responses of arousal, pleasure, and music-induced emotion.

There were no statistically significant additive effects of nicotine and music on physiology or self-reports. However, there were trends indicative of

additive effects on HR for both cohorts, and on self-reports mainly during chill-inducing music and more for smokers than nonsmokers. In addition, the results surprisingly showed that nicotine's effect on smokers resulted in a decrease in arousal and happiness, and an increase in sadness. One possible explanation for the lack of additive effects for both cohorts and the surprising results for smokers (decrease in arousal and happiness; increase in sadness) is the low ecological validity of nicotine gum, which may have produced spurious or nonsignificant effects. The use of genuine cigarettes or e-cigarettes may have been more appropriate for smokers who were in a state of withdrawal, as these would have recreated a more natural smoking environment.

Another possibility is that not enough nicotine was administered to smokers, perhaps either due to the doses of nicotine administered or due to the time course of nicotine gum. That is, nicotine is released more slowly into the bloodstream with nicotine gum (~30 min)(Benowitz et al., 2009), which could have resulted in less intense physiological effects and therefore less intense self-reports. However, the time course of nicotine gum was accounted for by having participants adhere to a standardized chewing protocol as well as wait ~30 min post-ingestion before beginning experimentation. Furthermore, nicotine dependence/tolerance was controlled for, as smoking participants were operationally defined based on their cigarette consumption (7+ cigarettes per day) and nicotine dependence (scoring a minimum of five on the Fagerström Test for Nicotine Dependence). Therefore, individual differences in tolerance would not have affected the results of this study. However, it may be that all smoking participants were not heavy enough smokers and therefore were not in an intense enough state of withdrawal. This may resulted in nicotine gum not modulating physiology and self-reports intensely enough to affect music-induced emotion.

Moreover, this study did not elucidate the role of arousal and pleasure in linking nicotine and music consumption and therefore disassociating these two dimensions was necessary in order to better understand how nicotine affects music-induced emotion. This was achieved through the use of caffeine instead of nicotine in study two.

In study two, caffeine was used to disassociate the effects of nicotine (e.g. increase in arousal, increase in pleasure) in order to determine to what extend an increase in arousal, without an increase in pleasure, would result in additive effects on the physiological indices and self-reported responses of arousal, pleasure, and music-induced emotion. In order to directly compare the effects of nicotine and caffeine, this study's procedure was the same as study one. That is, arousal was induced via caffeine administration, then participants listened to the same four types of music as used in study one. I hypothesized that with the ingestion of caffeine and subsequent action of music listening there would be an additive effect on the physiological indices and self-reported responses of arousal and music-induced emotion.

Similar to study one, there were no statistically significant additive effects of caffeine and music on physiology or self-reports. However, there were trends indicative of additive effects on all four physiological measures (HR, SCL, and respiration rate, and skin temperature) for both cohorts, mainly during happy and chill-inducing music. Self-reports also showed some trends of additive effects on happiness for both cohorts, mainly during chill-inducing music. The trends of additive effects on arousal and pleasure were only seen for smokers and mainly during chill-inducing music. Although these trends are nonsignificant, they suggest that with more statistical power (e.g. more participants) an effect of caffeine on music-induced emotion could be demonstrated, especially in abstaining smokers. In this way, abstaining smokers and nonsmokers may experience excitation transfer whereby they misattribute their increase in arousal from caffeine to their music-induced emotions.

One possible explanation as to why trends indicative of additive effects were mainly seen in abstaining smokers may be because of smokers' sub-baseline levels of arousal, pleasure, and positive emotion, which were experienced due to nicotine withdrawal. The physiological effects of caffeine may have then been misattributed to music-induced emotion more in smokers, than nonsmokers. However, assuming that abstaining smokers started with sub-baseline levels of arousal is in direct opposition to the elevated HR responses seen for smokers receiving placebo. That is, smokers who received

placebo were found to have a larger increase in HR compared to those receiving nicotine (see Figure 3.1 for reference). This may suggest that abstaining smokers have an increase in arousal, perhaps due to the anxiety felt during nicotine withdrawals, and that caffeine actually helps to relieve this arousal/anxiety. It is clear from the literature that there are inconsistent effects of caffeine on HR. While this study adds to the research that suggests caffeine to decrease HR, it also demonstrates that caffeine can have different effects on HR in different cohorts (e.g. abstaining smokers, nonsmokers). These results, although nonsignificant, suggest that the inconsistencies in the literature are a reflection of oversimplification. That is, past research has not investigated thoroughly other factors that may influence how caffeine affects HR (e.g. smoking status).

Although nonsignificant, the trends indicative of additive effects on self-reports were only seen in positively valenced measures (e.g. happiness and pleasure). This is in line with previous music and emotion research (Cantor & Zillmann, 1973; Dibben, 2004) showing that music-induced emotions that are positively valenced can be amplified by an increase in physiological arousal. One explanation for this result may be that the sadness is an emotion more intensely expressed than felt by music. It may also be that participants are less willing to admit to feeling a negative emotion. That is, it might be more socially acceptable to admit to feeling positive emotions than negative emotions. However, further research would be needed to verify this claim.

While the above studies investigated the physiological and emotional mechanisms that help explain the co-consumption of nicotine and music, study three aimed to examine the mechanisms involved in this phenomenon. Few studies have examined auditory information processing using nicotine in nonsmokers and this is the first study to specifically examine this using high and low-pitched tones. Because nicotine is a cholinergic stimulant it can excite the auditory pathway, which may facilitate auditory information processing and in turn enhance arousal, pleasure, and music-induced emotion. To test nicotine's ability to enhance auditory information processing an event-related potential (ERP) study was conducted where nonsmoking participants were administered nicotine, then asked to engage in a decision-making task

concerning high-pitched and low-pitched sounds. It was hypothesized that nicotine would enhance pitch perception indicated by the ERP components implicated in arousal, selective attention, and habituation.

The results showed nicotine to only affect P2 amplitude, which increased in the frontal region in response to the drug. Although this was contradictory to the hypothesis, it suggests that in nonsmokers nicotine can result in cognitive impairments. That is, nicotine may either induce a lack of habituation or less efficient information processing in nonsmokers. This could potentially mean that nonsmokers receiving nicotine are unable to adapt to repeated stimuli, such as music, but could alternatively suggest that nicotine is detrimental to those unfamiliar with its effects. Furthermore, there was a greater difference in P1 amplitude between frequency ranges for those receiving nicotine compared to those receiving placebo. This may suggest that in nonsmokers nicotine is able to enhance arousal at frequency ranges, but not at lower ones.

The effects of nicotine on P2 amplitude and the lack of effects observed for the other ERP components suggest that in nonsmokers nicotine either has no effect on auditory information processing or reduces it. This may be a result of the high dose of nicotine used (4 mg), as study one gave some indication that nonsmokers experience adverse effects in response to this dose. Examining how auditory information is processed in nonsmokers under a smaller dose of nicotine may show that the drug is capable of enhancing cognition in this cohort, or it may confirm the results of this study. Furthermore, a single study comparing how nicotine affects auditory information processing in smokers and nonsmokers would provide a more holistic view on the relationship between nicotine consumption and music listening and would help account for any adverse effects experienced by nonsmokers.

The remainder of this chapter will compare how music, nicotine, and caffeine affected physiological and self-reported responses independently and in combination, as well as address the underlying mechanisms responsible for these changes. For reference, study one's main effects of nicotine and music on physiological arousal and self-reports can be viewed in Table 2.7 and Table 2.8, respectively. Similarly, study two's main effects of nicotine and music on physiological arousal and self-reports can be viewed in Table 3.4 and Table 3.5,

respectively. Furthermore, Table 5.1, Table 5.2, Table 5.3 provide a summary of how these stimuli (e.g. nicotine + music; caffeine + music) affect these responses in combination. This chapter will then go on to discuss the cognitive mechanisms that may underlie and therefore help explain these physiological and self-reported changes based on the results of study three, which is an ERP study. Lastly, this chapter will address the wider question of why nicotine and music listening may be consumed together. Alternative explanations will be considered and future research and limitations will be discussed.

5.2. Music, emotion, arousal, and pleasure

Music's effects on happiness and sadness were consistent across studies. See Figure 2.13 and Figure 2.14 for music's effect on self-reported response in the nicotine study, and Figure 3.11 and Figure 3.12 for music's effect on self-reported response in the caffeine study. Happiness was consistently increased during happy and chill-inducing music, while it was consistently decreased during sad and neutral music. Sadness was increased for all music types except happy music. It was distinctly increased during sad music and distinctly decreased during happy music, while neutral and chill-inducing music showed relatively smaller increases in sadness across studies.

Music's effects on self-reported arousal and pleasure were also consistent across studies. Happy and chill-inducing music greatly increased arousal, while sad and neutral music decreased. The decreases in arousal seen for sad and neutral music were slightly larger in the caffeine study than in the nicotine study. Similar results were found for pleasure. Happy and chill-inducing music greatly increased pleasure, while sad and neutral music decreased it. However, there was one exception, sad music slightly increased pleasure in the nicotine study.

In general, music's effects on physiology were consistent across studies. See Figure 2.5 and Figure 2.6 for music's effect on physiology in the nicotine study, and Figure 3.3 and Figure 3.4 for music effect on physiology in the caffeine study. HR was increased for both studies and was more increased for happy and chill-inducing music than for sad and neutral music. SCL was increased for all music types in the nicotine study, with happy and chill-inducing music increasing it more than sad and neutral music. However, in the caffeine

study happy and chill-inducing music increased SCL, while sad and neutral music decreased. However, there was one exception in the caffeine study. For smokers, sad music slightly increased SCL. For respiration rate, similar results were found for smokers across studies. All music types increased respiration rate. IN the nicotine study respiration rate was increased the most for happy and neutral music, while in the caffeine study it was increased the most for happy and chill-inducing music. However, results differed across the studies for nonsmokers. In the nicotine study respiration rate was found to decrease for all music types, expect happy music, while in the caffeine study all music types increased respiration rate, with happy and chill-inducing music increasing it the most. Skin temperature had similar results across studies. All music types decreased skin conductance. However, in the nicotine study happy and neutral music decreased it the most, while in the caffeine study smokers saw the greatest decrease during sad and neutral music, while nonsmokers saw the greatest decrease during sad music only. These results overall are corroborated with past research (Blood & Zatorre, 2001; Hodges, 2010; Koelsch & Jäncke, 2015; Krumhansl, 1997).

The results of study one and two provide clear evidence that music can evoke emotion, as indicated by self-reported responses (and changes in physiology). These findings are corroborate with past research showing similar changes in responses during exposure to music (Hodges, 2009; Ritossa & Rickard, 2004). Emotional responses in general are often coupled with arousal and pleasure, both in a physiological and subjective sense (Russell, 1980; Salimpoor et al., 2009). Furthermore, arousal and pleasure are consistent features of models that measure and classify emotion (Russell, 1980; Smith & Ellsworth, 1985) and have been used to measure music-induced emotion in several lines of research (Egermann & McAdams, 2013; Egermann, Nagel, Altenmüller, & Kopiez, 2009; Nagel, Kopiez, Grewe, & Altenmüller, 2007; Schubert, 1999, 2001).

Emotions are coupled with physiological responses of arousal via the autonomic nervous system (ANS), which functions to activate bodily systems to support action (Rickard, 2004; Schmidt & Thews, 1989). Emotion, by definition, has a physiological component (Damasio, 1999; James, 1884; Schachter &

Singer, 1962), and therefore the ANS plays a critical role in emotion, producing visceral sensations that shape subjective emotional experiences. As previously mentioned, arousal is associated with an increase in heart rate, skin conductance, and respiration rate, as well as a decrease in skin temperature (Rickard, 2004). Furthermore, arousal is a primary component in theories of emotional responses to music (Berlyne, 1974; Bever, 1988; Meyer, 1956; North & Hargreaves, 1997; Thaut, 1990) and suggest that while cognitive information and context help determine the type of emotion experienced, it is physiological arousal that helps determine the intensity or strength of that emotion (Rickard, 2004; Schachter & Singer, 1962). Empirical evidence linking music and emotional arousal have shown emotions induced by music to result in physiological changes in the body (Khalfa et al., 2002; Krumhansl, 1997), as well as peripheral feedback from physiological arousal to modulate the strength of an emotion after it has been generated in the brain (Damasio, 1994; LeDoux, 1996) and after exercise (Dibben, 2004).

While physiological arousal is a reliable indicator of emotional arousal, it is controversial in regards to detecting the valence or pleasure dimension of emotion. However, previous research has demonstrated a strong and positive relationship between physiological arousal and subjective ratings of pleasure (Salimpoor et al., 2009) and a prominent theory of music-induced emotion suggests that the emotion experienced during music listening is in itself rewarding and pleasurable (Huron, 2006; Meyer, 1956; Sloboda & Juslin, 2001). That is, music is a source of pleasure because it evokes emotion. This suggests that pleasure, whether influenced by arousal or experienced independently, is a valid component of music-induced emotion and can be used to measure and explain such emotion. Empirical evidence linking pleasure and music-induced emotion has shown music to be consistently rated as one of the top ten most pleasurable activities (Dubé & Le Bel, 2003) and brain-based studies haven found music listening to modulate the dopaminergic system and to activate the limbic and paralimbic regions of the brain (Blood & Zatorre, 2001; Menon & Levitin, 2005), areas well established for being implicated in reward and motivation (Rodríguez de Fonseca & Navarro, 1998).

In this thesis it is clear that music listening evoked emotion, as revealed by self-reported responses and also – insofar as physiological changes link to emotion- physiological responses.

There physiological and self-reported responses to music were consistent across studies, especially in regards to the self-reported measures. Happy and chill-inducing music increased self-reports, while sad and neutral music decreased them. Chill-inducing music increased physiology more than any other type of music, while sad and neutral music increased physiology the least or even decreased it. This suggests that when examining arousal, pleasure, and basic emotions (e.g. happiness; sadness) measuring multiple emotion categories of music may not be necessary. Similar results could be obtained from comparing chill-inducing music, which is positively valenced and shows the most intense changes in responses, to either a control (e.g. neutral) piece of music or to a negatively valenced (sad) piece of music.

The physiological responses to music also indicate that some measures are better at reflecting arousal and pleasure than others. For example, in the nicotine study measures of respiration rate did not show similar patterns of responses for each music type between smokers and nonsmokers. Therefore, respiration rate might be more influenced by individual differences or other confounds to be a reliable measure of music-induced emotion. Furthermore, for both studies skin temperature showed only minor changes in responses between the music conditions, suggesting that skin temperature may not reflect different emotional responses to music. Overall, this suggests that HR and SCL are better measures of music-induced emotion. Indeed this is reflected in the literature, as these two measures are the most commonly measured physiological indices of emotion in music research. How this emotion is modulated due to an increase in arousal and pleasure from nicotine and caffeine, will be considered next.

5.3. Nicotine, emotion, arousal, and pleasure

See Figure 2.11 and 2.12 for nicotine's effect on self-reported response. It is important to note that these effects of nicotine were nonsignificant and therefore only reflect trends seen in the data. The effects of nicotine on happiness and sadness were different between smokers and nonsmokers. For

smokers, both doses of nicotine decreased happiness compared to placebo. However, for nonsmokers, 2 mg of nicotine increased happiness, while 4 mg decreased it. In smokers, nicotine systematically increased sadness, while for nonsmokers, 2 mg of nicotine increased sadness, but 4 mg decreased it.

The effects of nicotine on self-reported arousal and pleasure were also somewhat different between cohorts. For both cohorts nicotine decreased arousal. This decrease was systematic for smokers, but for nonsmokers the difference in arousal was negligible between the 2 and 4 mg doses. For smokers, nicotine systematically increased pleasure, while for nonsmokers nicotine systematically decreased pleasure.

See Figure 2.3 and 2.4 for nicotine's effect on physiology. Again, it must be emphasized that these effects were nonsignificant and therefore only reflect trends. In general, nicotine typically resulted in similar changes in physiology between cohorts. Nicotine increased HR in both cohorts and this increase was greater for 2 mg compared to 4 mg of nicotine. Nicotine systematically decreased skin conductance level for both cohorts. For smokers, nicotine decreased respiration rate, with negligible differences seen between the 2 and 4 mg doses. For nonsmokers, 2 mg of nicotine decreased respiration rate, while 4 mg of nicotine showed a negligible difference compared to placebo. For smokers, nicotine decreased skin temperature, with a greater effect for 2 mg of nicotine. For nonsmokers, nicotine systematically increased skin temperature.

The physiological and self-reported responses arising from nicotine demonstrate this substance's ability to modulate emotion, arousal, and pleasure, albeit somewhat differently between smokers and nonsmokers. These findings are somewhat consistent with previous research (Agué, 1974; Gilbert, 1979; Gilbert & Hagen, 1980; Leventhal & Cleary, 1980; Parrott & Winder, 1989; Silvette et al., 1962; Usuki et al., 1998; Wright, 1935), but suggest that nicotine did not consistently increase physiological and self-reported indices of arousal as expected. For example, nicotine's effect on respiration in both cohorts (e.g. a decrease in respiration) was unexpected, suggesting a decrease in arousal, and requiring replication. Although the effects of nicotine on respiration were unexpected they mirror the decreases found for self-reported arousal and happiness for both cohorts, as well as the increases in sadness. This suggests

that nicotine was unable to induce physiological arousal as intended and was only able to increase pleasure as intended for smokers. In light of these results, nicotine, especially nicotine gum, may not be best suited for inducing physiological arousal and emotion.

Nicotine may not have increased physiological arousal and self-reports in nonsmokers because the adverse effects experienced due to a lack of tolerance for the drug (Foulds et al., 1997). However, the lack of increased physiological and self-reported responses for smokers is more surprising. This may have been for methodological reasons, such as using nicotine gum instead of genuine cigarettes. The time course of nicotine gum delivers nicotine more slowly to the body over a longer period of time compared to genuine cigarettes (Benowitz et al., 2009). This may have resulted in a less intense 'rush' of nicotine and therefore less intense physiological and self-reported responses in smokers. Also, nicotine gum lacks ecological validity, as smoking a cigarette involves hand-to-mouth movement as well as oral and sensations. This may have diminished smokers' responses to nicotine.

Examining Figure 2.3 and Figure 2.4 for nicotine and Figure 2.5 and Figure 2.6 for music comparisons can be seen concerning how these stimuli affected the physiology of smokers and nonsmokers. However, it is important to note that these comparisons were not statistically analyzed and these comparisons were made through visual inspection of the data. An increase in HR was the most salient similarity found across music and nicotine. The most salient difference observed between music and nicotine concerns skin conductance, which increased for music, but decreased for nicotine. Although nicotine's decrease in skin conductance does not imply arousal, this response is most likely explained by nicotine's vasoconstriction properties, which inhibit blood flow and therefore reduce skin conductance (Agué, 1974). Respiration rate and skin temperature showed more complex comparisons. For example, another similarity between nicotine and music was a decrease in respiration. However, while nicotine decreased respiration rate for both cohorts, music only decreased respiration rate for nonsmokers during sad, neutral, and chill-inducing music. Another similarity between nicotine and music was a decrease

in skin temperature. However, nicotine only decreased skin temperature for smokers.

Examining Figure 2.11 and Figure 2.12 for nicotine and Figure 2.13 and Figure 2.14 for music comparisons can be seen concerning how these stimuli affected the self-reports of smokers and nonsmokers. Again, these comparisons were not statistically analyzed and comparisons were made through visual inspection. Similarities and differences in self-reports between music and nicotine can also be seen, however the results are more difficult to compare because of the variation seen within music types and nicotine doses. The most surprising difference between music and nicotine were ratings of arousal, which were consistently increased during happy and chill-inducing music, but decreased for smokers and nonsmokers in response to nicotine. Happiness was also different between music and nicotine. Happiness was consistently increased during happy and chill-inducing music, but decreased for both cohorts in response to nicotine (except for nonsmokers in response to 2 mg of nicotine). An increase in arousal and happiness during happy and chill-inducing music is expected as these music types are known to strongly correlate with positive affect due to emotional contagion (Juslin & Västfjäll, 2008), liking, and familiarity factors (Ritossa & Rickard, 2004). However, a decrease in arousal and happiness for smokers is surprising. Nicotine would be predicted to increase these responses in smokers due to the relief of withdrawal symptoms (Hughes et al., 1984). A similarity between music and nicotine was seen for rating of sadness. Sadness was consistently increased only during sad music, again an effect likely due to emotional contagion (Juslin & Västfjäll, 2008). Sadness also increased during neutral and chill-inducing music, but to a lesser extent than during sad music. Also, in response to nicotine sadness systematically increased for smokers and increase for nonsmokers at the 2 mg dose. These responses to nicotine suggest that the drug did not increase subjective arousal as intended and further suggest that the drug actually increased negative affect.

Another similarity between music and nicotine was seen for ratings of pleasure. Pleasure was consistently increased during happy and chill-inducing music, an expected response due again to emotional contagion, liking and

familiarity factors known to underpin musical emotion (Juslin & Västfjäll, 2008; Ritossa & Rickard, 2004). Nicotine systematically increased pleasure in smokers, and systematically decreased it in nonsmokers. Nicotine likely increased pleasure in smokers due to the relief of withdrawal symptoms (Hughes et al., 1984) and decreased pleasure in nonsmokers due to adverse effects (Foulds et al., 1997).

The many differences seen in physiological and self-reported responses between music and nicotine suggest that these two stimuli are not equally robust in influencing arousal and pleasure. From the above trends it seems that music is better able to increase the physiological and self-reported responses associated with arousal and pleasure. Furthermore, responses that differed between music and nicotine may negate the potential for additive effects. That is, if music increases a response (e.g. arousal), but nicotine decreases it then there is less likelihood for their co-consumption to result in additive effects on individuals. However, some responses were similar between music and nicotine, such as an increase in HR and pleasure. For this reason HR and pleasure ratings may be more accurate and reliable indicators of arousal (both physiological and subjective), pleasure, and emotion compared to other measures, such as respiration rate or sadness, which showed inconsistent findings both between and within music and nicotine conditions. Furthermore, these measures may be more robust at reflecting any additive effects found on arousal and pleasure during the co-consumption of music and nicotine.

5.4. Caffeine, emotion, arousal, and pleasure

See Figure 3.9 and 3.10 for caffeine's effect on self-reported responses. These effects of caffeine were nonsignificant and therefore only reflect trends seen in the data. The effects of caffeine on happiness and sadness were somewhat difference between cohorts. Caffeine systematically increased happiness for smokers, while for nonsmokers 200 mg decreased happiness and 400 mg increased happiness. For ratings of sadness, caffeine decreased sadness for both cohorts.

The effects of caffeine were different between smokers and nonsmokers in regards to self-reported arousal and pleasure. In response to caffeine smokers showed a systematic increase in arousal and pleasure, while

nonsmokers showed a systematic decrease in these ratings. In this case, it is clear that caffeine did not isolate the effects of self-reported arousal. That is, caffeine was used with the intention of disassociating the effects of nicotine (e.g. increase in arousal, increase in pleasure) on music-induced emotion and to therefore determine to what extent an increase in arousal was responsible for the effects of nicotine on music-induced emotion. However, the above findings show that caffeine increased both arousal and pleasure in smokers. If arousal had been isolated from pleasure as intended then pleasure would have been unaffected by caffeine. However, it seems that in smokers, pleasure increased in response to caffeine and did so more than arousal.

See Figure 3.1 and 3.2 for caffeine's effect on physiology. Again, it must be emphasized that these effects were nonsignificant and therefore only reflect trends. Caffeine typically resulted in systematic changes in physiology, although respiration rate showed greater responses at the lower dose of 200 mg. More specifically, caffeine systematically decreased HR in smokers and systematically increased it in nonsmokers. Caffeine also systematically increased SCL for both cohorts and this effect was more dramatic for smokers than nonsmokers. Caffeine increased respiration rate for both cohorts, with a larger increase at the 200 mg dose. Skin temperature responses to caffeine varied between cohorts. For smokers, caffeine decreased skin temperature with a greater decrease at the 200 mg dose. For nonsmokers, caffeine systematically decreased skin conductance.

Caffeine's effect on physiology and self-reported responses demonstrate its ability to influence emotion, arousal, and pleasure, and these results run parallel to previous literature (Green et al., 1996; Green & Suls, 1996; Quinlan et al., 2000; Sawyer et al., 1982; Silverman, Mumford, & Griffiths, 1994; Zahn & Rapoport, 1987a, 1987b). The increasing trends in physiology are likely due to caffeine's stimulatory effects on the central nervous system (Leavitt, 1974; Stroebel, 1972) and likely influenced the increasing trends seen in the self-reported responses.

The trends found here in regards to how caffeine affects physiology and self-reports also suggest that caffeine did not isolate arousal from pleasure. Some research has suggested that caffeine increases arousal without affecting

pleasure (Herz, 1999) and therefore this stimulant can be used to disassociate arousal from pleasure. However, many others have found caffeine to be associated with liking (Lieberman et al., 1987) and reinforcing effects (Juliano & Griffiths, 2004), which are feelings strongly associated with pleasure. There is also previous literature demonstrating caffeine to increase both pleasure and arousal (Quinlan et al., 2000), and caffeine has been shown to release dopamine in the nucleus accumbens (NAcc) (Solinas et al., 2002), a brain region implicated in reward and motivation (Bardo, 1998; Blood & Zatorre, 2001). Caffeine has been shown to release dopamine in quantities comparable to nicotine in rats (Di Chiara & Imperato, 1988; Pontieri, Tanda, & Di Chiara, 1995) and has been shown to have mildly addictive qualities (Satel, 2006). Furthermore, there is some evidence suggesting that pleasure is somewhat dependent on arousal, both in a physiological and self-reported sense (Kuppens et al., 2013; Salimpoor et al., 2009). This strongly suggests that caffeine increases both arousal and pleasure and helps explains why caffeine was not able to isolate arousal from pleasure in study two. The trends found here in regards to how caffeine affects physiology and self-reports also provides supporting evidence that arousal and pleasure are not independent from each other. Lastly, these trends suggest that caffeine enhances physiology and self-reports of arousal, pleasure, and emotion in abstaining smokers and nonsmokers more than nicotine. This has strong implication for research examining how an increase in physiology affects emotion.

Many similarities can also be seen between caffeine, nicotine, and music in regards to physiological and self-reported responses. These results are again difficult to compare because of the variation seen within music types and nicotine and caffeine doses. Furthermore, these comparisons were not statistically analyzed and comparisons were made through visual inspection. Caffeine increased HR in nonsmokers and similarly nicotine and music increased HR for both cohorts. HR was increased the most for chill-inducing music and the least for caffeine. Also, HR increased more during study one than study two. Although nicotine decreased SCL, caffeine and music increased it, with chill-inducing music increasing SCL slightly more than either caffeine dose. Respiration responses differed between the nicotine and caffeine studies. In the

caffeine study, caffeine and music increased respiration for all conditions, with music increases respiration more than caffeine. In the nicotine study, nicotine decreased respiration for both cohorts. Music increased respiration for smokers during all music conditions, but decreased it for nonsmokers in all music conditions except happy music. Skin temperature also differed between studies. In the caffeine study, caffeine and music decreased skin temperature for all conditions. In the nicotine study, nicotine decreased skin temperature for smokers, but increased it for nonsmokers. Music decreased skin temperature for both cohorts.

These trends suggest that while nicotine somewhat increased physiological arousal, indicated by an increase in HR and a decrease in skin temperature, caffeine and music were more robust in increasing physiology, indicating by an increase in HR, SCL, and respiration rate, and a decrease in skin temperature. Furthermore, music modulated physiological responses more than caffeine or nicotine, indicated by greater increases in HR, SCL, and respiration rate compared to either psychostimulant.

There are also similarities and differences in self-reports between caffeine, nicotine, and music. Happy and chill-inducing music increased happiness. Nicotine and caffeine did not consistently increase happiness across cohorts. That is, an increase in happiness can only be seen for smokers receiving caffeine and nonsmokers receiving 2 mg of nicotine. Furthermore, an increase in happiness was clearly larger for music than for caffeine (for smokers) or nicotine (nonsmokers receiving 2 mg of nicotine). Increases in sadness were seen for music, especially during sad music. Nicotine also increased sadness for smokers as well as for nonsmokers at the 2 mg dose. Caffeine decreased sadness. Sadness was rated higher for sad music than for nicotine. Ratings of arousal were clearly increased during happy and chill-inducing music, however only smokers receiving caffeine showed a similar, but less intense, increase. Nicotine, for both cohorts, decreased arousal. Pleasure was also strongly increased for happy and chill-inducing music. Similar increases were seen for smokers receiving both nicotine and caffeine. This increase in pleasure was larger for music than for nicotine or caffeine. Nonsmokers showed a decrease in pleasure for both nicotine and caffeine.

These trends show that nicotine only increased self-reports of pleasure and only for smokers. They further show that caffeine increased self-reports of happiness, arousal, and pleasure, but also only for smokers. However, music increased all measures of self-reports and did consistently across both cohorts. Happy and chill-inducing music increased ratings of arousal, pleasure, and happiness, while sad music increased ratings of sadness. Similar to the trends found in for physiological responses, music increased arousal, pleasure, and emotion more than either psychostimulant. Furthermore, caffeine was better able to modulate physiological and self-reported responses of arousal and pleasure than nicotine. It may be that abstaining smokers were not administered enough nicotine to relieve withdrawal symptoms, while simultaneously too much nicotine was administered to nonsmokers, making them feel ill. It could also be that because caffeine consumption was not controlled for those participants receiving this psychostimulant had a low daily consumption of caffeine. This in turn resulted in strong pharmacological actions on participants, greatly increasing their physiological and self-reported responses.

Nonsignificant differences were found between the nicotine conditions as well as between the caffeine conditions. However, based on visual inspection it is clear that differences between doses of each psychostimulant existed. These nonsignificant differences may have been due to this study being underpowered. One possible solution to this would have been to collapse the self-reported measures into global scores. Previous research has suggested that arousal and pleasure may be inextricably linked (Kuppens,2008), suggesting that measuring these two dimensions of emotion together is a more ecologically valid method for measuring self-reported responses. As such, in the present study it may have been possible to collapse happiness and pleasure into a global pleasure score or a global positive emotion scores. However, upon visual inspection of Figures 2.11 – 2.12 and Figures 3.9-3.10 for the self-reports of nicotine and caffeine, respectively, there was no consistent relationship between happiness and pleasure ratings as such there was no statistical basis for combining the two measures. Similarly, happiness and sadness (reverse coded) could have been collapsed to indicate positive emotion. However, visual

inspection of the data also revealed no consistent relationship between happiness and sadness ratings.

5.5. Effects of music and nicotine, and music and caffeine, on physiology and self-reports

Although nonsignificant, nicotine and caffeine interacted with music to produce some similarities and differences in trends indicative of additive effects on physiology and self-reports. These results are summarized in Table 5.1 for physiological responses, Table 5.2 for happy and sad responses, and Table 5.3 for arousal and pleasure responses. That is, the combination of nicotine + music and of caffeine + music resulted in (nonsignificant) trends indicative of additive effects on physiology and self-reports. These trends are directly compared between study one and study two in order to understand 1) if each psychostimulant (e.g. nicotine; caffeine) interacted with music in different ways and 2) to understand if these interactions affected music-induced emotion in ways that may help explain why nicotine and music are often co-consumed. While this information will be discussed in detail in the following sections, the tables aim organize and summarize this information in a concise manner. Each table displays at which dose and during which music condition trends indicative of additive effects were seen for each of measurement (physiology; self-reports). These tables are further categorized by psychostimulant and smoking cohort. It must be noted that the comparisons made between study one (nicotine+ music) and study two (caffeine + music) were not performed statistical and are based solely on visual inspection of the data.

5.5.1. Effects of music and nicotine, and music and caffeine, on physiology

HR showed results with trends indicative of additive effects for nicotine and caffeine. For both doses of nicotine and for both cohorts trends indicative of additive effects on HR were seen during happy, neutral, and chill-inducing music. For both doses of caffeine and for both cohorts trends indicative of additive effects on happiness were seen during chill-inducing music. Additionally, in nonsmokers, both doses of caffeine showed trends indicative of additive effects on happiness during happy music. SCL showed trends indicative of additive effects, but only for caffeine. In both smokers and nonsmokers caffeine

showed trends indicative of additive effects on SCL during happy and chill-inducing music. Respiration rate showed trends indicative of additive effects, but again only for caffeine. For smokers caffeine showed trends indicative of additive effects on respiration for both doses during chill-inducing music and additionally for 200 mg during sad and neutral music. For nonsmokers caffeine showed trends indicative of additive effects on respiration for both doses during happy, neutral, and chill-inducing music. Skin temperature showed trends indicative of additive effects for both nicotine and caffeine. For smokers, nicotine showed trends indicative of additive effects on skin temperature at both doses for all music types. For nonsmokers nicotine showed no additive effects. For both cohorts caffeine showed trends indicative of additive effects on skin temperature at both doses for happy, neutral, and chill-inducing music. These results are in agreement with past research showing music, nicotine, and caffeine to independently increase physiology (Agué, 1974; Hodges, 2010; Jones, 1987; Smith et al., 2004) and demonstrate their ability to combined with music in order to modulate physiological responses associated with arousal.

Differences in trends indicative of additive effects on physiology can be seen between study one (nicotine + music) and study two (caffeine + music). For example, it is clear that in study two there were more trends indicative of additive effects on physiology than in study one. Caffeine showed trends indicative of additive effects in both cohorts for all four physiological measures. Contrastingly, nicotine only showed trends indicative of additive effects for HR in both cohorts and for skin temperature in smokers. By examining the main effects of caffeine and nicotine trends can be seen indicating that caffeine increased physiological arousal more than nicotine. For example, the main effects of nicotine on physiology were often in the opposite direction compared to the main effects of music (Figure 2.7), this may have decreased the potential for additive effects to occur when nicotine and music were combined, as one may possibly negated the other. However, this was not the case for the main effects of caffeine and music (Figure 3.4), as both almost always modulated each physiological measurement in the same direction. This raises the potential for additive effects to occur when caffeine and music are combined. Furthermore, caffeine and music were consistent in producing trends indicative

of additive effects on all physiological measurements and these trends were consistent across cohorts. While nicotine and music consistently produced trends indicative of additive effects on HR for both smokers and nonsmokers, the trends indicative of additive effects on skin temperature were only seen for smokers. Therefore, it may be that caffeine and music together are also more consistent in increasing physiology than nicotine and music.

A few differences in trends indicative of additive effects on physiology can also be seen between smokers and nonsmokers. For example, nicotine and music produced trends indicative of additive effects on skin temperature in smokers, but not in nonsmokers. This suggests that smokers' skin temperature was more affected by nicotine and music than nonsmokers. This may be due to the differences in tolerance level for nicotine or the state of nicotine deprivation experienced by smokers. However, caffeine and music affected smokers and nonsmokers similarly. This may be because all participants in study two were caffeine consumers. However, because caffeine consumption was not controlled for is it impossible to tell how caffeine tolerance and consumption affected these results. Future research should control for caffeine consumption by examining high, low, and non-consumers of caffeine. This could be accomplished through a pre-screening survey.

Differences in trends indicative of additive effects on physiology can also be seen between the various music conditions (e.g. happy, sad, neutral, chill-inducing music). More specifically, trends indicative of additive effects on physiology were seen more during happy and chill-inducing music. The main effect of music showed the most robust (and significant) effects on physiological responses compared to the main effects of nicotine and caffeine. The main effects of music also show happy and chill-inducing music to modulate physiological responses significantly more than sad and neutral music (see Figures 2.5, 2.6, 3.3, and 3.4). This suggests that happy and chill-inducing music were better able to produce trends indicative of additive effects on physiological responses when combined with nicotine/caffeine. As previously mentioned, further research should take note of this as it likely possible to reduce the number of music conditions (e.g. compare only chill-inducing music to sad/neutral music).

Table 5.1.

Trends indicative of additive effects on physiology

Measurement	Cohort	Nicotine + Music	Caffeine + Music
Heart Rate	Smokers	2 + 4 mg, happy, neutral, chill-inducing music	200 + 400 mg, chill-inducing music
	Nonsmokers	2 + 4 mg, happy, neutral, chill-inducing music	200 + 400 mg, happy and chill-inducing music
Skin Conductance Level	Smokers	No additive effects	200 + 400 mg, happy and chill-inducing music
	Nonsmokers	No additive effects	200 + 400 mg, happy and chill-inducing music
Respiration Rate	Smokers	No additive effects	200 + 400 mg, chill-inducing music; 200 mg, sad and neutral music
	Nonsmokers	No additive effects	200 + 400 mg, happy, neutral, chill-inducing music
Skin Temperature	Smokers	2 + 4 mg, all music	200 + 400 mg, happy, neutral, chill-inducing music
	Nonsmokers	No additive effects	200 + 400 mg, happy, neutral, chill-inducing music

NB: All results are nonsignificant trends

All trends show an increasing trend indicative of additive effects

5.5.2. Effects of music and nicotine, and music and caffeine, on self-reported emotion (happiness/sadness)

Nicotine and caffeine also interacted with music to produce some similarities and differences in their trends indicative of additive effects on emotional responses. These results are summarized in Table 5.2. Happiness showed trends indicative of additive effects for nicotine and caffeine. For smokers, trends indicative of additive effects on happiness were seen at both doses of nicotine during chill-inducing music. Happiness also showed trends indicative of additive effects for nonsmokers during happy and chill-inducing

music, but only at the 2 mg dose. There were also trends indicative of additive effects on happiness for sad music in smokers, and sad and neutral music in nonsmokers, as these music conditions showed a decrease in happiness at the placebo level and a further decrease in happiness in response to nicotine. For smokers caffeine showed trends indicative of additive effects on happiness at both doses during happy and chill-inducing music. For nonsmokers caffeine showed trends indicative of additive effect on happiness at both doses during chill-inducing music only. Sadness also showed trends indicative of additive effects. For smokers, nicotine showed trends indicative of additive effects on sadness at both doses during sad and neutral music and for nonsmokers during sad and chill-inducing music, but only at the 2 mg dose. Lastly, caffeine showed trends indicative of additive effects on sadness, but only for nonsmokers during happy music, as happy music showed a decrease in happiness at the placebo level and a further decrease in happiness in response to caffeine.

Findings from Dibben (2004) showed an increase in the intensity of positive emotion (study two) as well as an increase in the dominant emotion felt in response to music (study one) as a result of increased physiological arousal from exercise. Other emotional situations have also increased emotional intensity as a result of increased physiological arousal, such as sexual attraction and emotional responses to film (Dutton & Aron, 1974; Schachter & Singer, 1962). This mirrors the results found in this thesis for trends indicative of additive effects on happiness and sadness by showing that listeners are influenced by their body state when experiencing emotions induced by music. Furthermore, the trends found in this thesis, although nonsignificant, support the current thesis's hypothesis that increased physiological arousal can lead to an intensification of felt emotion during music listening.

Similarities in trends indicative of additive effects on emotion can be seen between study one (nicotine + music) and study two (caffeine + music). For example, both studies showed trends indicative of additive effects on happiness during happy and chill-inducing music. This runs parallel to the trends seen in physiological responses, where trends indicative of additive effects on physiology occurred more during happy and chill-inducing music than during sad and neutral music. This suggests that happy and chill-inducing music

were better able to produce trends indicative of additive effects on emotion when combined with nicotine/caffeine and that subsequent research can reduce the number of music conditions used when examining how increases in physiology affect music-induced emotion.

Differences in trends indicative of additive effects on emotion were also seen between study one (nicotine + music) and study two (caffeine + music). For example, study one showed trends indicative of additive effects on sadness during sad, neutral, and chill-inducing music. However, study two did not show any increasing trends indicative of additive effects on sadness. Furthermore, in study one, trends indicative of additive effects on sadness varied between smokers and nonsmokers. Sadness showed trends indicative of additive effects for smokers during sad and neutral music, and for nonsmokers during sad and chill-inducing music. In this case, it may be that negative emotion is less affected by increases in physiological arousal, and when it is affected, it is only consistently done so during negatively valenced music (e.g. sad music). This may suggest that increases in physiological arousal do not consistently affect negative emotion during music listening. However, further research would need to test this claim by examining other negative emotions induced by music, including anger and fear, as well as sadness.

Although nicotine and music did not result in many trends indicative of additive effects on physiology (e.g. only on HR and skin temperature) it seems that together they are able to produce trends indicative of additive effects on emotion. Furthermore, a main effect of nicotine showed a trend of decreasing happiness, while music showed a significant increase in happiness (during happy and chill-inducing music compared to sad and neutral music). These opposing main effects would seem to negate any trends indicative of additive effects on positive emotion. This may suggest that when consumed together nicotine and music may be able to affect positive emotion more so than when consumed individually. Subsequent research may be interested in examining this further to determine why nicotine alone does not increase happiness, but when combined with music listening it is able to do so.

There were also decreasing trends indicative of additive effects on happiness and sadness. That is, for study one sad music for both cohorts, as

well as neutral music for nonsmokers, showed a decrease in happiness in the placebo condition and a further decrease in response to nicotine. This shows that for these music conditions (e.g. sad and neutral music) nicotine further decreased happiness ratings. A similar trend was found for caffeine, where a further decrease in sadness ratings occurred during happy music in nonsmokers. These trends may further suggest that increases in physiological arousal can modulate emotion by making sad and neutral music less happy and by making happy music less sad. Although these trends were not specifically predicted, they are not completely unexpected, considering that happiness and sadness are opposite emotions. Previous research has not examined how increases in physiological arousal can affect music-induced emotions that are opposite to the emotions expressed by music (e.g. how happiness is affected by sad music; how sadness is affected by happy music). Further research may therefore be able to provide further insight into the effects of peripheral feedback on positive and negative emotion by investigating such relationships.

Table 5.2.

Trends indicative of additive effects on emotion

Measurement	Cohort	Nicotine + Music	Caffeine + Music
Happiness	Smokers	2 + 4 mg, *sad, chill-inducing music	200 + 400 mg, happy and chill-inducing music
	Nonsmokers	2+4 mg, *sad and *neutral music 2 mg, happy and chill-inducing music	200 + 400 mg, chill-inducing music
Sadness	Smokers	2+4 mg, sad and neutral music	No additive effects
	Nonsmokers	2 mg, sad and chill-inducing music	200 + 400 mg, *happy music

NB: All results are nonsignificant trends

** Indicates a decreasing trend indicative of additive effects, all others are increasing trends*

5.5.3. Effects of music and nicotine, and music and caffeine, on self-reported arousal and pleasure

Nicotine and caffeine interacted with music to produce some similarities and differences in trends indicative of additive effects on arousal and pleasure. These results are summarized in Table 5.3. Arousal showed results with trends indicative of additive effects for nicotine and caffeine. For smokers and nonsmokers, nicotine showed trends indicative of additive effects at both doses during chill-inducing music. There was also a trend indicative of an additive effect on arousal for neutral music in nonsmokers, which showed a decrease in arousal at the placebo level and a further decrease in response to nicotine. For smokers, caffeine showed trends indicative of additive effects on arousal at both doses for chill-inducing music. For nonsmokers caffeine showed an additive effect on arousal for neutral music in nonsmokers, which showed a decrease in arousal at the placebo level and a further decrease in response to caffeine. Pleasure showed results with trends indicative of additive effects for nicotine and caffeine. For smokers, nicotine showed trends indicative of additive effects on pleasure at both doses for sad and chill-inducing music. For smokers, caffeine showed trends indicative of additive effects on pleasure at both doses for happy and chill-inducing music. For nonsmokers caffeine showed an additive effect on pleasure for neutral music in nonsmokers, which showed a decrease in arousal at the placebo level and a further decrease in response to caffeine.

Similarities in trends indicative of additive effects on self-reported arousal and pleasure were also seen between study one (nicotine + music) and study two (caffeine + music). For example, although caffeine and music showed more trends indicative of additive effects on physiological arousal than did nicotine and music, both studies resulted in similar findings in smokers. That is, for both studies, smokers showed trends indicative of additive effects on self-reported arousal and pleasure, especially during chill-inducing music. The similarities between study one and two, show nicotine and music, as well as caffeine and music, to produce trends indicative of additive effects on HR and skin temperature. This may suggest that these two physiological responses can provide enough peripheral feedback in individuals to enhance self-reports of arousal and pleasure during music listening.

Compared to other music conditions, chill-inducing music showed more trends indicative of additive effects on self-reported arousal and pleasure when combined with nicotine/caffeine. This is a pattern shown throughout studies one and two and suggests that physiological arousal as well as self-reported arousal, pleasure, and emotion can be more enhanced when nicotine and caffeine are consumed in combination with chill-inducing music. This also suggests that future research needs to strongly consider the use of chill-inducing/self-selected music when investigating music-induced emotion.

Similarities and differences in trends indicative of additive effects on self-reported arousal can also be seen between smokers and nonsmokers. For example, trends indicative of additive effects on arousal were seen for smokers and nonsmokers in study one (e.g. nicotine + music), but only for smokers in study two (caffeine + music). Furthermore, nonsmokers showed a decreasing trend indicative of additive effects on arousal during neutral music in both study one and two.

Similarities and differences in trends indicative of additive effects on self-reported pleasure can also be seen between smokers and nonsmokers. In study one and two, trends indicative of additive effects on pleasure were only seen for smokers. Nonsmokers showed a decreasing trend indicative of additive effects on pleasure during neutral music in study two and showed no trends in study one. Furthermore, the trends seen for the main effects of nicotine and caffeine showed a decrease in pleasure and arousal for nonsmokers. However, for study one, in nonsmokers, ratings of happiness increased during happy and chill-inducing music at the 2 mg dose (and decreased at the 4 mg dose), while ratings of happiness systematically increased during chill-inducing music and 400 mg of caffeine increased ratings of happiness during happy music. Such differences between the trends indicative of additive effects between study one and two may highlight the importance of nicotine deprivation on self-reported pleasure, as it may be that because nonsmokers were not in a state of withdrawal they did not experience an increase in pleasure as a result of caffeine or nicotine consumption during music listening. This trend (or lack of) in pleasure has implications for the emotions experienced during music listening.

Showing that some music conditions (e.g. happy and chill-inducing music) can still increase in emotion despite the main effects of nicotine and caffeine.

The differences seen between self-reported arousal and pleasure between smokers and nonsmokers is surprising, considering that caffeine alone, as well as in combination with music showed more trends of increased physiology compared to nicotine (both alone and in combination with music). This may suggest that while changes in physiology are coupled with the experience of emotion, increases in physiological arousal do not necessarily lead to increases in self-reported arousal and pleasure during the consumption of psychostimulants for all individuals. Further investigation would need to be carried out to explain why caffeine in combination with music can affect emotion (e.g. happiness; sadness) in nonsmokers, but not affect self-reported arousal or pleasure. Therefore, these results only partially support the hypothesis that an increase in physiological arousal can lead to an increase in self-reported arousal and pleasure.

In summary, both study one (nicotine + music) and study two (caffeine + music) showed trends indicative of additive effects on HR and skin temperature. Additionally, study two showed trends indicative of additive effects on SCL and respiration rate. Although study two showed more trends indicative of additive effects on physiology compared to study one, both showed similar trends indicative of additive effects on arousal and pleasure for smokers, as well as similar trends indicative of additive effects on happiness on both smokers and nonsmokers. However, only study one showed trends indicative of additive effects on sadness. Overall, this suggests that increases in physiological arousal can enhance the effects of music-induced emotion, especially for smokers and especially during happy and chill-inducing music.

Table 5.3.

Trends indicative of additive effects on self-reported arousal and pleasure

Measurement	Cohort	Nicotine + Music	Caffeine + Music
Arousal	Smokers	2 + 4 mg, chill-inducing music	200 + 400 mg, happy and chill-inducing music
	Nonsmokers	2 + 4 mg, chill-inducing and *neutral music	200 + 400 mg, *neutral music
Pleasure	Smokers	2+ 4 mg, sad and chill-inducing music	200 + 400 mg, happy and chill-inducing music
	Nonsmokers	No additive effects	200 + 400 mg, *neutral music

NB: All results are nonsignificant trends

** Indicates a decreasing trend indicative of additive effects, all others are increasing trends*

5.6. Mechanisms underlying additive effects of physiological and self-reported responses

The above results demonstrate that nicotine and caffeine in combination with music affected both arousal and pleasure, albeit somewhat differently between smokers and nonsmokers. Therefore, caffeine was unable to disassociate arousal from pleasure and allow a 'pure' form of the former to be investigated. This leaves open the possibility that an increase in reward value of other stimuli and excitation transfer underpin the additive effects seen as a result of nicotine and caffeine administration and subsequent action of music listening.

Nicotine has previously been shown to increase the reward value of other stimuli (Attwood et al., 2009; Dawkins et al., 2007) because of its ability to increase dopamine in the NAcc (Balfour, 2004; Donny et al., 2003). Previous research has shown caffeine to also increase dopamine in the NAcc (Solinas et

al., 2002), suggesting that it too can increase the reward value of stimuli. This suggests both nicotine and caffeine in combination with music are able to increase pleasure and therefore emotion in listeners.

Peripheral feedback also played a role in the additive effects seen in study one and two. Nicotine and caffeine both showed trends of increased physiological arousal, results that are corroborated with past research (Konecni, 1975; Scherer & Zentner, 2001). This increase in physiology may have resulted in sensory feedback, which led music listeners to misattribute their physiological arousal to the emotions they experienced during music listening, and in turn led them to experience more intense emotion. In this way, excitation (arousal) from nicotine and caffeine may have amplified the emotions experienced during music listening.

Zillmann (1971) suggests that excitation transfer occurs when there is a lack of information about the source of arousal. In this way, excitation from one source is misattributed to another. This idea suggests that there is residual excitation from a prior emotional event, which is then transferred to an immediate and subsequent stimulus by intensifying the subsequent emotional reaction (Zillmann, 1971, 2006). Previous research on excitation transfer has examine the effects of known and unknown sources of arousal on emotion. For example, after physical exercise participants were exposed to an erotic film. Those who were unaware of the source of their arousal, which stemmed from exercise, felt more sexually aroused and evaluated the film more positively than those who were aware of their physiological state. However, some studies have demonstrated that excitation transfer can occur when information about the source of arousal in available. For example, (Taylor et al., 1991) found that angered individuals showed increased aggression when they were able to use the information that they had ingested an arousing drug (e.g. 350 mg of caffeine) as justification for their aggression. Similarly, this study may have also demonstrated that the excitation transfer process can occur when the source of arousal is known, as participants were aware that they might have received a psychostimulant substance (e.g. nicotine; caffeine) as compared to placebo. Furthermore, all participants were knowledgeable of the effects of nicotine (see Appendix G for nicotine information sheet, which explains the

effects of nicotine) and caffeine, as all participants were caffeine consumers and therefore had experienced the effects of caffeine on physiology prior to the study. However, participants' knowledge of arousal was not confirmed in experiments one and two of this thesis as they were not question on whether they knew the source of their arousal and they were not directly told whether they were receiving a psychostimulant or a placebo. To confirm this, participants would have needed to be overtly informed of which condition they had been allocated to (e.g. 0, 2, 4 mg of nicotine/ 0, 200, 400 mg of caffeine) and a manipulation check would be needed.

5.7. Summary of ERP findings

Study three was an initial attempt to clarify the cognitive mechanisms that may be responsible for the co-consumption of nicotine and music. Past research has provided mixed results concerning the cognitive effects of nicotine on nonsmokers. For example, nicotine's ability to facilitate auditory perception in nonsmokers has been reported in some EPR studies. This includes an increase in P1 amplitude, suggesting an increase in arousal and initial sensory intake (Harkrider & Champlin, 2001; Knott, 1985b), an increase in N1 amplitude, suggesting enhanced selective attention (Knott, 1985b, 1986; Knott, Bolton, et al., 2009), an increase in P2 amplitude, suggesting a decrease in habituation (Domino & Kishimoto, 2002), and a decrease in N2 amplitude and latency, also suggesting an increase in arousal (Harkrider & Champlin, 2001; Knott, 1989). However, nicotine has also been reported to not enhance auditory information processing in nonsmokers. For example, no effect of nicotine on N1 amplitude or latency in nonsmokers was found during a dichotic listening task (Knott, Shah, et al., 2009) and no effect of nicotine on the N1-P2 and P2-N2 amplitude as well as the P2 latency was found in nonsmokers listening to monaural pulses (Harkrider & Champlin, 2001).

Interestingly nicotine did cause a decrement in habituation, reflected by an increase in the P2 amplitude in the frontal region of the scalp. This is similar to the results of Domino and Kishimoto (2002), who found nicotine in nonsmokers to increase the P2 amplitude during irrelevant frequent tones. This may suggest that nicotine results in cognitive impairments in nonsmokers. However, an alternative interpretation of these results may suggest that

nicotine impairs listeners' ability to habituate to music, and in turn leads to an increase in emotional engagement.

Habituation typically occurs when stimuli becomes repetitive or too familiar and in turn causes disengagement (Rankin et al., 2009). Music is known to be a very repetitive stimulus, which helps communicate to the listener that a musical feature or passage is important or salient (Margulis, 2012, 2013). Examples of repetition in music include earworms (e.g. songs that get 'stuck' in your head) (Williamson et al., 2012) and the beat or pulse of music (Huron, 2006). In this case, repetition may cause listeners to disengage with music or consider the stimuli as 'background music'. However, nicotine may decrease a listener's ability to habituate to the receptiveness of music and instead allow listeners to become familiar with it.

Familiarity with music is known to underpin emotional engagement (Pereira et al., 2011). For example, familiarity with music significantly influences the chills response, a pleasurable and emotionally rewarding physiological response to music (Grewe, Kopiez, & Altenmüller, 2009) and highly pleasant and familiar music can enhance the connectivity between the ventral tegmental area (VTA) and nucleus accumbens (NAcc), two brain areas implicated in reward and music-induced emotion (Blood & Zatorre, 2001). Familiarity also plays a role in less intense emotional responses to music. The *mere exposure effect* (Zajonc, 1968) suggests that enjoyment and related liking can increase AS exposure to stimuli increases (Brattico & Pearce, 2013). For example, North and Hargreaves (1997) reported a positive linear relationship between liking and familiarity for pop music and Pereira and colleagues (2011) reported activation of the limbic and paralimbic areas, including the NAcc, to familiar music compared to unfamiliar music. In this way, nicotine may help listeners to stay engaged with music despite its repetitive features, in turn affording familiarity with the music and causing an enhanced emotional reaction. This effect of nicotine on music-induced emotion may be stronger in smokers, those who do not experience ill effects to the drug. However, further research is needed to determine to what extent nicotine reduces habituation in smokers during auditory perception.

In contrast to the effect on the amplitude of P2, the effects of nicotine were nonsignificant for the amplitude and latency of the P1, N1, and N2 ERP components. That is, nicotine did not affect arousal or selective attention in nonsmokers. These findings may be due to nonsmokers' unfamiliarity with nicotine, which causes this cohort to absorb the drug faster, metabolize it slower, and respond with greater sensitively to its effects (Benowitz & Jacob, 1993; Srivastava, Russell, Feyerabend, Masterson, & Rhodes, 1991). This in turn may lead to no enhancement in cognition, which has been found in several past studies examining cognitive information processing (Dunne et al., 1986; Heishman et al., 1993; Hindmarch et al., 1990; Wesnes & Warburton, 1984).

These negative findings concerning P1, N1, and N2 ERP components are corroborated by the self-reports of nonsmokers found in study one. That is, in response to nicotine, nonsmokers reported a decrease in arousal, pleasure, and happiness, and an increase in sadness. Furthermore, no additive effect on pleasure was found for nonsmokers in response to nicotine and music. This suggests that while nicotine can increase physiological arousal in nonsmokers, it results in a decrease in subjective self-reports and cognition. It may potentially even result in cognitive impairments. Similar results have been reported in other studies showing that in nonsmokers, nicotine increased heart rate and blood pressure, as well as decreased skin temperature, but nicotine also increased negative affect and the desire to repeat nicotine ingestion (Heishman et al., 1993).

5.8. Future research and limitations

One main limitation of this thesis was the method of administering nicotine and caffeine to participants. That is, nicotine was administered as nicotine gum, while caffeine was administered as a tablet. Because the dose of nicotine and caffeine can vary widely when consumed in a natural setting (Frith, 1971a; Mandel, 2002) administration of these stimulants was heavily controlled. While this helps to control the dose of each psychostimulant it decreases the ecological validity of these studies. More specifically, when nicotine and caffeine are consumed in the context of music they are either inhaled through the lunged (e.g. cigarettes, e-cigarettes) or are drunk (e.g. cup of coffee). These more 'natural' methods of delivery therefore have different time courses

compared to gum and tablets forms. For example, smoking a cigarette allows for almost instantaneous delivery of nicotine to the brain and smokers are able to control how much nicotine they consume based on puffing strength, rate of puffs, and how long they choose to hold the smoke in their lungs before exhaling. Similarly with caffeine, consumers are able to decide how quickly they drink coffee/other caffeinated beverages and the amount of caffeine varies widely between cups of coffee. There are also other factors involved in nicotine and caffeine consumption that are not present in a laboratory setting. For cigarettes, the hand-to-mouth movements and oral sensations of smoking are important aspects of the experience (Ikard et al., 1969). Likewise for caffeine, the warm sensations of a hot cup of coffee/tea play a role in the psychological effects of the psychostimulant (Quinlan et al., 1997; Quinlan et al., 2000) and there are many other substances in coffee and tea besides caffeine (Mandel, 2002). The absence of these factors in a laboratory setting may have influenced the findings in this thesis, resulting in less additive effects being elucidated when these stimulants were co-consumed during music listening.

Furthering on from this thesis, an important future study examining the effects of nicotine and caffeine on music-induced emotion will use different delivery methods for the administration of nicotine and caffeine. For ethical reasons it is unlikely that nicotine will be able to be administered through cigarettes, due to the carcinogens present in the dug. However, nicotine may be able to be administered through the use of e-cigarettes. This allows for sensorimotor and oral sensations that more closely resemble the smoking of a cigarette. Furthermore, research with nicotine may recruit e-cigarettes users instead of cigarette smokers, to further increase the ecological validity of a laboratory setting for nicotine-consuming participants. Caffeine may be administered in a drink, for example, by adding caffeine tablets to a cup of decaf coffee in order to control the amount of caffeine administered, while also increasing the ecological validity of the experiment. Previous experiment have administered caffeine in a similar way (Quinlan et al., 1997; Quinlan et al., 2000).

Another main limitation of this thesis is that in study three the effects of nicotine on auditory perception were not tested on a population of abstaining

smokers. This made it impossible to compare the cognitive effects of nicotine across smokers and nonsmokers and left many unanswered questions regarding whether tolerance to nicotine plays a role in the drug's cognitive enhancing effects. A future ERP study should examine nonsmokers, deprived smokers, and non-deprived smokers to determine how nicotine affects auditory perception and information processing. This will help elucidate further whether the effects of nicotine are due to withdrawal reversal or whether they are absolute. It may also help explain the co-consumption of nicotine and music. For example, if arousal and selective attention are enhanced due to nicotine, then this may suggest that nicotine helps listeners focus on music listening and disengage with irrelevant background noise. However, further research is needed to test this premise.

5.9. Conclusion

Two novel results are reported in this thesis. First, nicotine and caffeine increase physiological arousal, which leads to an increase in self-reported arousal, pleasure, and emotion during music listening. Second, that in nonsmokers nicotine causes a reduction in habituation, which reduces disengagement from music listening and increases familiarity, and in turn leads to an increase in music-induced emotion. Taken together, these findings suggest that nicotine and music listening are likely co-consumed because nicotine is able to enhance or intensify music-induced emotion.

Considering these findings in the context of previous work, the results of this thesis are in line with Dibben (2004) who found an increase in physiological arousal as a result of exercise to result in more intense emotional experiences during music listening. They are also in line with Domino & Kishimoto (2002) who found habituation to decrease as a result of nicotine in nonsmokers during frequently occurring tones. This suggests that nicotine and caffeine may enhance music-induced emotion by increasing the reward value of other stimuli (e.g. music) and through excitation transfer, where those ingesting nicotine misattribute their increase in physiological arousal to their music-induced emotions. Additionally, it may suggest that nicotine stops listeners from disengaging with the repetitive features of music and allows them to become familiar with it and in turn experience an increase in music-induced emotion.

Furthermore, the results of this thesis overall suggest that nonsmokers receiving nicotine experienced negative subjective effects in response to the drug. This suggests that the effects of nicotine are more likely to occur in those who hold a tolerance for the drug, such as smokers.

One goal of this thesis was to identify which emotional categories of music are best suited for non-nicotine replacement therapy. The results show that chill-inducing music, and to a lesser extend, happy music may be useful for therapy. Happy and chill-inducing music showed the most consistent trends indicative of additive effects on physiological and self-reported responses, suggesting that for those trying to quit smoking, listening to chill-inducing music may help lower the negative emotions experienced during abstinence.

Another implication of this thesis was the potential to information individuals, particularly adolescents, that similar physiological responses could be obtained from nicotine and music, in hopes of deterring them from taking up a smoking habit. This was partially fulfilled, as certain types of music were found to have similar physiological responses as nicotine in nonsmokers. For example, all music types and nicotine doses similarly increased HR and decreased skin temperature in nonsmokers (as well as smokers). However, while nicotine consistently decreased SCL all music types increased SCL. Furthermore, nicotine either decreased or has no effect on respiration rate, while sad, neutral, and chill-inducing music decreased respiration rate. Based on these results listening to sad, neutral, or chill-inducing music may result in similar physiological changes as nicotine when consumed by nonsmokers.

Furthermore, understanding why music and nicotine are consumed together can potentially help us explain why drug consumption in general is so prevalent in a musical context. From this thesis nicotine and music, as well as caffeine and music, showed (nonsignificant) trends indicative of additive effects on arousal, pleasure, and emotion. This may suggest that in combination, music and substances which increase physiological arousal (e.g. substances of abuse, nicotine, caffeine) can enhance emotional reactions and therefore encourage the co-consumption of substances and music.

Lastly, nicotine was also suggested to potentially facilitate the processing of auditory information, for example, to allow listeners to better understand fast

and complex music or slower and simpler music if they are tired. However, based on the findings of this thesis, this was not found to be the case. That is, nicotine was found to decrease habituation in nonsmokers. This may be interpreted as nicotine resulting in a cognitive deficit in nonsmokers. However, there may be a positive consequence to a decrease in habituation, whereby listeners consuming nicotine may not habituation to repetitive stimuli in music and therefore be able to become familiar with music and enjoy it more. However, further research would need to be conducted to test this claim, as this effect of nicotine on familiarity and liking of music was not directly tested.

Overall, the results of this thesis suggest that when nicotine is consumed, especially by smokers, this can lead to an increase in physiological arousal. This increase in physiology can in turn lead to an enhancement of music-induced emotion, especially during happy and chill-inducing music.

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Appendix A

Are you a smoker? Would you like to earn £5?

- We are looking for (1) smokers who smoke at least 7 cigarettes a day and have been smoking for at least 2 years and (2) nonsmokers.

- Participants are asked to refrain from nicotine, caffeine, and alcohol for 24 hours. They will then be given nicotine gum and asked to rate their emotional responses to music.

- The experiment will take 1 hour and upon completion you will receive £5

For more detail please visit:
www.psychologyofmusic.co.uk/MusicandNicotine.html
or contact Theresa Veltri:
TMVeltri2@sheffield.ac.uk

Appendix B

Musical Background

Please answer the following questions:

1. Have you ever played an instrument or sung? If yes, when, for how long, and on which instrument/voice?

_____

_____

_____

2. Have you ever taken regular music lessons? If yes, when, for how long, on what instrument/voice, and to what grade?

_____

_____

_____

3. How many hours per week do you listen to music?

_____

4. What are your three favorite musical genres?

1. _____

2. _____

3. _____

Appendix C

Smoking History

Please answer the following questions:

1. Do you currently smoke cigarettes? □ Yes (go to Q3) □ No

2. How many cigarettes have you smoked in your life? _____ (end survey)

3. How many cigarettes do you smoke per day? _____

4. How long have you been smoking cigarettes? (months & years) _____

5. Do you ever smoke alone? □ Yes □ No

6. Do you smoke throughout the day? □ Yes □ No

7. Do you purchase cigarettes? □ Yes □ No

8. Are you familiar with nicotine gum? □ Yes □ No

8. Have you had caffeine in the past hour? □ Yes □ No

9. Have you smoked in the past 24 hours? □ Yes □ No

Appendix D

Participant Health Screening

Before the study begins we must obtain information about psychological and physical well being. Please answer the following questions.

1. What is your age _____ yrs

2. What is your gender? □ Male □ Female

3. What is your height? _____ m , _____ ft _____ in

4. What is your weight? _____ kg, _____ stones _____ lbs

3. Please read the following list and tick any statements that apply to you:

I am currently taking medication □ Yes □ No

Please list the medication and say what it is for:

_____

I am pregnant and/or breastfeeding □ Yes □ No

I have been diagnosed with one of the following conditions:
stroke, thyroid problems, persistent indigestion, stomach ulcers, angina, liver/kidney disease, or heart disease.

□ Yes □ No

If yes, please explain.

_____

My blood pressure is above 190/40

□ Yes □ No □ Don't Know

(Don't worry if you don't know your blood pressure, it will be taken later).

4. Please read the following list and tick any conditions which you have been diagnosed with *and recently suffered from* in the last year:

□ Senile and presenile dementias
□ Schizophrenic disorders
□ Major depression
□ Bipolar disorder
□ Agoraphobia
□ Simple phobia
□ Obsessive-compulsive disorder
□ Dysthymic disorder
□ Somatisation disorder
□ Antisocial personality disorder
□ Drug abuse/dependence
□ None of the above
□ Any other psychological disorder. Please specify
_____

Appendix E

Table E1.

Musical excerpt list

Emotion Category	Title	Artist
Happy	Angel of Harlem	U2
Happy	Hey Soul Sister	Train
Happy	Outside Villanova	Eric Hutchinson
Happy	She's Electric	Oasis
Sad	Everybody Hurts	R.E.M.
Sad	Colorblind	Counting Crows
Sad	Foolish Games	Jewel
Sad	Do What You Have To Do	Sarah McLachlan
Neutral	Negativland	Neu!
Neutral	Seeland	Neu!

Appendix F

Reading Comprehension Questions

Please stop reading now and answer the following questions in essay format. Try to answer in as much detail as possible. It is more important to give detail than it is to complete all the questions.

1. Describe and explain the Prudential commercial first depicted at the beginning of the chapter. What was the dream about? Why did the author write about the commercial? Please give as much detail as possible.

2. What do you think Rock music represents. How would you contrast it with classical music? Please give as much detail as possible.

3. How is the rhythm 'n' blues (white music) similar and different to blues (black music)? Please give as much detail as possible.

4. Describe at least one example where authenticity of music ahs been violated. You ay use an example from the book or from your own experience. Please give as much detail as possible.

5. What is the difference between Rock music and Pop music? Please give as much detail as possible.

6. How are the performers of classical music similar to pop artists? How do you think they are different? Please give as much detail as possible.

7. Compare and contrast the music industry's actions of Production, Distribution, and Consumption with the artistic actions of Composing, Performing, and Appraising. Please give as much detail as possible.

Appendix G

The Effects of Nicotine on Music-induced Emotions Participant Information Sheet

Invitation paragraph

You are invited to participate in a research project. Before deciding please understand the research and what it involves. Read the following information to decide if you wish to take part. This study has met the ethical requirements of the University of Sheffield's Department of Psychology.

What is the purpose of the project?

This research wishes to examine if nicotine has an effect on emotional reactions to happy, sad, and chill-inducing music. It will run from September 2012 to December 2012. You are asked to participate in this study because you are a student at the University of Sheffield. A total of 90 participants will participate.

What will happen to me if I take part?

You will be asked to chew nicotine gum for 30 minutes. You will then listen to musical excerpts, which includes 3 predetermined songs and 1 self-selected song. You will then rate how you feel in response to each excerpt. Throughout the experiment your heart rate, respiration rate, skin conductance and body temperature will be measured.

What are the possible disadvantages and risks of taking part?

During this experiment you may experience effects of nicotine, which are temporary. These effects may include some increases in heart rate, blood pressure, or breathing rate. You may also feel nervous, a loss of appetite, and a later feeling of relaxation. ***See second sheet for more detail. If you have high blood pressure you will not be allowed to participate in this study.***

What happens if I wish to discontinue my participation?

If you wish to stop participating during the experiment you are free to do so without any consequences. Your participation is voluntary. You are free to refuse participation or to withdraw at any time. If you decide to take part, this information sheet and a consent form will be provided to you. If you have any questions or complaints about the study please direct them to Theresa Veltri at TMVeltri2@sheffield.ac.uk or Dr. Paul Overton at p.g.overton@sheffield.ac.uk. If needed, please direct complaints to the Registrar@sheffield.ac.uk, +44 (0) 114 222 1101.

Will my taking part in this project be kept confidential?

All information collected will be kept confidential and any information disseminated will not contain your name.

What will happen to the results of the research project?

Research results become part of a PhD thesis. Results will also be presented at conferences and submitted to a peer reviewed journal. To obtain a copy of these results contact Theresa Veltri.

Contact for further information
For more information contact Theresa Veltri: TMVeltri2@sheffield.ac.uk or Paul Overton, p.g.overton@sheffield.ac.uk., +44 (0) 114 222 6624. Thank you for participating! You will receive this Participant Information Sheet and a signed Participant Consent Form to keep for your records.

Nicotine Information Sheet
PLEASE READ THE FOLLOWING PARAGRAPHS VERY CAREFULLY:

Nicotine is a naturally occurring substance found in the tobacco plant. It has two potential effects on the body 1) it activates the sympathetic nervous system and so may causes a rapid release of adrenaline, which can lead to an increase in your heart rate and blood pressure, as well as a change in your breathing. **For precautionary reasons if you have high blood pressure you will not be allowed to participate in this study,** 2) it may effect the brain by influencing your reaction time and your ability to pay attention, making you feel like you can work better. Pathways mediating reward, memory and arousal are also activated by the drug, hence our interest in how nicotine affects the emotional response to music. Nicotine by itself, and in recreational dosages, is not known to be damaging or toxic. Dangerous doses of nicotine exceed recreational doses by thirty or more times.

Nicotine can be self administered through cigarettes, cigars, pipe tobacco, chewing tobacco, oral snuff, nicotine patches, or nicotine gum. The average strength of a cigarette deposits about 1.9 mg of nicotine into the bloodstream, which is equal to the amount deposited by a piece of nicotine gum containing 4 mg of nicotine. In this study we will use nicotine gum at two doses, 4 mg and 2 mg (the latter being equivalent to half a cigarette). These are the most commonly used doses in human nicotine research, especially research looking at performance and cognitive function. These doses have been used safely in smokers, abstinent smokers, and people who have never smoked. However, in a small number of people nicotine gum can cause unpleasant feelings in the stomach. Although these symptoms will be monitored, if you report marked side effects testing will be discontinued.

Please remember that you do not have to participate in this study and you have the right to withdraw at any time without any explanation or negative consequences.

I have read and understood this nicotine information sheet

□ Yes □ No

I understand that my participation in completely voluntary and that I have the right to withdrawal from this study at any time with out negative consequences.

□ Yes □ No

Appendix H

Are you a smoker? Do you listen to music?

- We are looking for smokers who smoke at least 7 cigarettes a day and have been smoking for at least 2 years.

- Participants will be asked to refrain from nicotine and caffeine for 24 hours. They will then be given caffeine tablets and asked to rate their emotional responses to music.

- The experiment will take no more than 1 hour.

For more detail please visit
www.psychologyofmusic.co.uk/MusicandCaffeine.html
or contact Theresa Veltri:
TMVeltri2@sheffield.ac.uk

Appendix I

Caffeine Consumption Questionnaire

Please answer the following questions:

Do you consume caffeine regularly (at least 3 times per week)?

□ Yes □ No

Please indicate for how long you have been consuming caffeine

_____ years and _____ months

How many cups of tea do you consume per week? _____

How many cups of coffee do you consume per week (instant coffee, filter coffee, espresso)? _____

How many energy drinks do you consume per week? _____

How many cola drinks do you consume per week (coke, pepsi, dr. pepper)? _____

Appendix J

The Effects of Caffeine on Music-induced Emotions

Participant Information Sheet

You are invited to participate in a research project. Before deciding please understand the research and what it involves. Read the following information to decide if you wish to take part. This study has been approved by The Department of Psychology Ethics Committee.

The aim of this research is to examine the effects of caffeine on emotional reactions to music. The study will run from February 2014 to July 2014. We are looking for a total of 30 volunteers who are smokers that generally enjoy listening to music.

What will happen to me if I take part?
You will be asked to take either caffeine pills or a placebo vitamin pill. While waiting for your body to metabolize the pills you will engage in a reading comprehension and writing task. These tasks will take 30 minutes. Next, you will listen to four music excerpts, which include 3 predetermined songs and 1 self-selected song. After listening to each excerpt you will report your felt emotions using 6 rating scales. Throughout the experiment your heart rate, respiration rate, skin conductance and body temperature will be measured.

What are the possible disadvantages and risks of taking part?
During this experiment you may experience effects of caffeine, which are temporary. These effects may include an increase in heart rate, blood pressure, or sweating. You may also feel nervous, shaky, a loss of appetite, or stomach disturbances. See second sheet (Caffeine Information Sheet) for more detail. If you have high blood pressure you will not be allowed to participate in this study.

What happens if I wish to discontinue my participation?
If you wish to stop participating during the experiment you are free to do so without any consequences. Your participation is voluntary. You are free to refuse participation or to withdraw at any time. If you decide to take part, this information sheet and a consent form will be provided to you. If you have any questions or complaints about the study please direct them to Theresa Veltri at TMVeltri2@sheffield.ac.uk or Prof Paul Overton at P.Overton@sheffield.ac.uk. If needed you can direct complaints to the Registrar@sheffield.ac.uk, +44 (0) 114 222 1101.

Will my taking part in this project be kept confidential?
All information collected will be kept confidential and any information disseminated will not contain your name.

What will happen to the results of the research project?
Research results become part of a PhD thesis. Results will also be presented at conferences and submitted for publication in a peer-reviewed journal. To obtain

a copy of these results contact Theresa Veltri.

Contacts for further information
For more information contact Theresa Veltri, TMVeltri2@sheffield.ac.uk or Prof. Paul Overton, P.Overton@sheffield.ac.uk., +44 (0) 114 222 6624.

Your participation is greatly appreciated! You will receive this Participant Information Sheet and a signed Participant Consent Form to keep for your records.

Caffeine Information Sheet
PLEASE READ THE FOLLOWING PARAGRAPHS VERY CAREFULLY:
Caffeine is a naturally occurring substance found in seeds, leaves, and plants. It is most commonly extracted from the seeds of the coffee plant and the leaves of the tea bush. Caffeine has 2 potential effects on the body 1) it activates the central nervous system which can cause an increase in your heart rate and blood pressure, and may cause you to sweat. For precautionary reasons if you have high blood pressure you will not be allowed to participate in this study. 2) it may affect the brain by influencing reaction time and your ability to pay attention, making you feel like you can work better. Caffeine by itself, and in recreational dosages, is not known to be damaging or toxic. A daily intake of 1000 mg of caffeine (over 8 cups of brewed coffee) is considered to be potentially harmful.

Caffeine can be found in a variety of products including coffee, tea, soft drinks, energy drinks, and cocoa products, such as chocolate. The average strength of one cup of coffee is about 100 mg. In this study we will use caffeine at two doses, 200 mg and 400 mg. These are the most commonly used doses in human caffeine research, especially research looking at performance and cognitive function. These doses have been used safely in smokers, abstinent smokers, and people who have never smoked. However, in some people caffeine can cause unpleasant feelings, especially in the stomach. These symptoms will be monitored, and if you report marked side effects, testing will be discontinued.

Remember, you do not have to participate in this study and you have the right to withdrawal at any time without any explanation or negative consequences.

I have read and understood this caffeine information sheet

□ Yes □ No

I understand that my participation is completely voluntary and that I have the right to withdrawal from this study at any time without negative consequences.

□ Yes □ No

Appendix K

History of EEG and ERP

Richard (1875) was the first to observe and record the spontaneous electrical activity of the brain. He reported using a mirror galvanometer with non-polarizable electrodes to observe electrical impulses from the exposed brains of live animals (Collura, 1993). He placed unipolar electrodes on the surface of each hemisphere or placed one electrode on the grey matter of the cerebral cortex and the other on the skull. From this he found distinct increases in electrical currents of the grey matter, especially when shining light into the animals' eyes, during sleep, and during the onset of their death. He further found that after death these currents decreased until they disappeared completely (Berger, 1929). While EEG experiments continued on animals, most notable by Beck (Beck, 1890a, 1890b), it was not until the 20th century that they were performed on humans.

In 1924 Hans Berger was experimenting with blood flow changes in a patient with a surgical skull defect. He placed two clay electrodes 4 cm apart in the patient's surgical holes within the skull. Because the skull was not obstructing the electrical signal Berger was able to observe continuous oscillations of the galvanometer. By 1928, through experimentation and technological advances in galvanometers, Berger was able to produce high quality electroencephalograms (EEGs) that changed based on the psychological state of his patients. He found that by placing an electrode on the scalp, amplifying the signal, and plotting the voltage changes over time, he could then measure the electrical activity of the human brain (Luck, 2005; Millett, 2001). Although Berger's observations were first met with skepticism, Adrian and Matthews (1934) were able to replicate and promulgate Berger's discoveries. Later, Berger's findings were further confirmed by others (Gibbs, Davis, & Lennox, 1935; Jasper & Carmichael, 1935), which led to an acceptance of EEG as a genuine method of electrophysiological research (Luck, 2005).

Neurophysiological basis of EEG

The slow acceptance of EEG as a legitimate research method was due to the lack of understanding in its underlying system of neuronal generation, particularly because of the complexity in the transfer of electrical signals from

the cortex to the scalp (Lopes da Silva, 2010). It is therefore important to understand the neurophysiological basis of EEG.

EEG records the summed electrical activity of hundreds of neurons. Neurons are excitable brain cells that contain intrinsic electrical properties and their activity produces electrical fields that can then be measured by electrodes placed on the scalp. When activated, neurons generate electrical currents. These currents flow across the cellular membrane of a neuron and originate from one of two types of activations. The first type of activation is a fast depolarization of a neuronal membrane that creates an action potential (Lopes da Silva, 2010; Lopes da Silva & van Rotterdam, 2005). Neurons have a high concentration of potassium (K^+) and chloride (Cl^-) ions inside the cell, and a high concentration of sodium (Na^+) and calcium (Ca^{2+}) ions outside the cell. This arrangement results in a voltage difference of approximately -60 mV to -70 mV inside the cell compared to its outside surrounding environment. This voltage difference is the membrane potential and can be modified by the flux of ions depending on the opening and closing of ion channels. An influx of positively charged ions into the cell usually results in a positive membrane potential (e.g. when the influx is large enough) and is termed depolarization (Bucci & Galderisi, 2011).

When depolarization occurs it triggers the transmission of an action potential. The action potential is mediated by a rapid influx of Na^+ ions across the cell membrane and results in an intracellular potential jump from a negative to a positive charge, approximately from -70 mV to +40 mV. The action potential then travels down the axon to the axon terminal. This usually occurs within ~1 ms (Bucci & Galderisi, 2011; Lopes da Silva, 2010), but is dependent upon distance, diameter, and myelination variability. Once reaching the axon terminal action potentials trigger the release of neurotransmitters from the presynaptic cell across the synaptic cleft to the postsynaptic neuron, thereby propagating the electrical signal.

When action potentials trigger the release of neurotransmitters they induce either an excitatory or inhibitory postsynaptic potential in the postsynaptic neuron (Bucci & Galderisi, 2011). These postsynaptic potentials are the second type of activation that occurs in the cellular membrane of

neurons. They are much slower than action potentials and last approximately 10-250 ms. As previously mentioned, there are two main types of postsynaptic potentials: excitatory (EPSPs) and inhibitory (IPSPs) postsynaptic potentials.

When an EPSP occurs there is an active current sink generated in the extracellular medium at the excitatory synapse, whereby positive ions flow into the cell to depolarize the membrane. This also produces electrical negativity in the immediately surrounding extracellular space. Since there is no accumulation of charge in the extracellular medium, the positive transmembrane current that flows into the neuron will have compensatory currents that flow through the neuron and exit back into the extracellular space. Therefore, an EPSP will have simultaneous passive current sources at a more distal portion of the cell.

In the case of an IPSP, the inside of the cell is hyperpolarized. This means that there is an active current source, whereby a positive ionic current flows from the inside of the postsynaptic neuron outward or a negative ionic current from outside the cell flows inward. This produces electrical positivity in the immediately surrounding extracellular space (sink). Furthermore, this is coupled with a negatively charged passive current source. In this way, an active synapse causes a dipole sink-source arrangement (Lopes da Silva, 2010; Nelson & Monk, 2001).

These active and passive currents produced by synaptic activity pass through extracellular and intracellular space and create a potential field around the cell. When they reach the scalp through the process of volume condition, they interact with the metal of the EEG electrodes and the difference in voltage that arises over time is the EEG signal. However, the electrical signal of a single neuron is too small to be recorded by an electrode and therefore the measured activity must originate from a summation of the synchronous electrical activity generated by hundreds of neurons with similar spatial orientation.

Importantly, it is the pyramidal neurons located in the cortical layers of III, V, and VI that produce most of the EEG signal (Ebersole, 2003). This is because of their physical properties and synchronous activity. The apical dendrites of pyramidal cells are aligned together in an orientation that is perpendicular to the surface of the cortex (Lopes da Silva, 2010). This means that their sources and sinks correspond to a ‘dipole current’ that is also

perpendicular to the cortical surface (Lopes da Silva & van Rotterdam, 2005). Furthermore, postsynaptic potentials last much longer (10-250 ms) compared to action potentials (1-2 ms). This longer time course permits the summation across neurons. This is especially true when they are activated synchronously, meaning that the voltage fields generated on the dendrites of pyramidal cells can be summated to produce a potential large enough to be recorded with little attenuation at the scalp's surface (Näätänen, 1992). In this way, many neurons aligned in parallel and simultaneously producing electrical activity leads to a summation of current in the same direction. This creates an open field that allows current to be volume-conducted through extracellular space up to the scalp's surface. With this in mind, the EEG signal can be more accurately defined as the sum of extracellular electrical field potentials produced by synchronized postsynaptic currents on cortical pyramidal neurons (Nelson & Monk, 2001). From this a graphic representation of the difference in voltage between two cerebral locations can be plotted over time to create a two-dimensional waveform (Olejniczak, 2006).

Recording of EEG

EEG is a non-invasive and relatively inexpensive research method used to investigate the neural correlates of cognitive function (Light et al., 2010; Nelson & Monk, 2001). Its high temporal resolution, in the order of milliseconds, makes it an ideal research method for investigating the early stages of information processing as well as the transition from sensory-based perceptual processes to the higher-order cognitive functions (Light et al., 2010).

To record EEG a minimum of two electrodes must be used. The most common types of electrodes are Ag/AgCl because of their low resistance for direct current and low frequency potentials. Furthermore, they produce stable electrode potentials that are resistant to electrode movement artifacts (Kamp, Pfurtschneller, Edlinger, & Lopes da Silva, 2005). An active electrode is position over a site with neuronal activity while a reference electrode is positioned away from this site. The electrodes measure the potential difference between an active and reference electrode. The reference electrode is usually positioned at a strategic location on the scalp (e.g. the vertex). It needs to be located in a place that is not likely to pick up neuronal activity, but that is still affected by

noise picked up by active electrodes, such as eye blinks, muscle artifacts, and electrical mains activity. Likely locations for reference electrodes include the vertex, chin, neck, earlobes, mastoids, and tip of the nose (Luck, 2005).

Most EEG recordings consist of a number of active electrodes, which are positioned over the top and side of the scalp, according to the 10-20 International System (Jasper, 1958), which can be viewed in Figure K1. This system consists of 21 electrodes with an interelectrode distance of 6 cm (Gevins et al., 1994). It specifies the standard positioning of the electrodes in relation to a fixed point on the head, usually the nasion located above the nose and the inion located at the back of the skull above the neck. The electrodes are labeled with abbreviations that correspond to cortical areas: (F) frontal, (C) central, (T) temporal, (P) parietal, and (O) occipital. The electrodes placed in overlying areas are given subscripts so that odd numbers correspond to the left hemisphere, while even numbers correspond to the right hemisphere. The electrodes placed on the midline, between the nose and putamen magnum, are given the subscript 'z'.

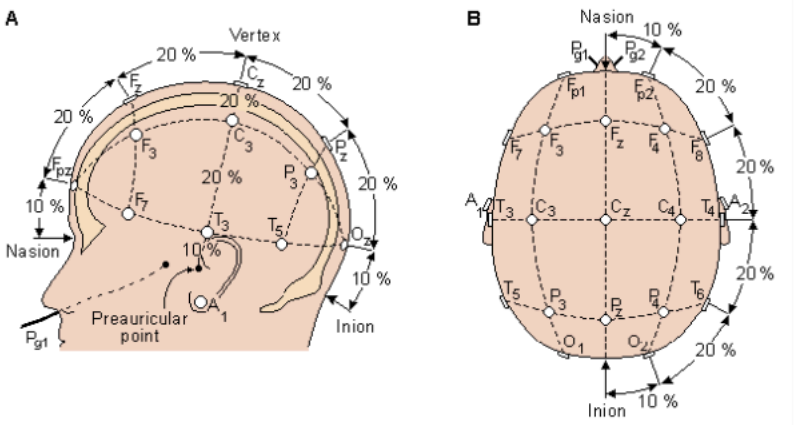

Figure K1. 10-20 International System
This figure diagrams the 10-20 international system and shows the standard placement of EEG electrodes.

Although the 10-20 International System is used to standardized electrode placement, advancements in technology have produced EEG systems with higher electrode counts. The most common systems use between 32 and 256 electrodes. These electrodes can be embedded into caps or nets, which allows for fast, easy, and standardized measurements of EEG data for most

head sizes (Niedermeyer & Lopes da Silva, 2005). An example of one of these is a high density net, created by Electrical Geodesic Inc. (EGI), called the Geodesic Sensor Net (GSN) and is shown in Figure K2. It contains 128 electrodes with an interelectrode distance of 28-30 mm (Tucker, 1993). High density EEG nets record EEG data as well as eye movements related to stimulus activation. For instance, the net shown in Figure K2 contains six electrodes, grouped in pairs, for recording eye movements. Two pairs of electrodes are positioned vertically above and below the eyes in order to record horizontal eye movements, while the last pair of electrodes is positioned on the outer side of the eye in order to record vertical eye movements.

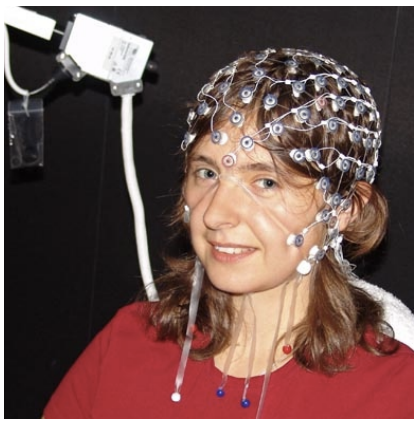

Figure K2. Geodesic Sensor Net (GSN) with 128 electrodes
The 128 electrode GSN used for recording EEG, eye movements, as well as muscular and electrical activity.

Deriving ERPs from EEG

ERPs are voltage fluctuations within the EEG signal that are time-locked to a specific event, usually to the onset of a stimulus or a behavioral response (Kappenman & Luck, 2012). The change in voltage observed is related to the brain activity that is (Kappenman & Luck, 2012)required in order to process the time-locked event (Picton & Hillyard, 1988). Therefore, they reflect the successive stages of information processing (Knott, Bolton, et al., 2009). Furthermore, because postsynaptic potentials reach the scalp almost instantaneously and because ERPs are time-locked, they allow researchers to investigate sensory, perceptual, and cognitive processing with millisecond precision (Light et al., 2010).

ERPs that are specific to auditory stimuli are known as auditory evoked potentials (AEP) (Kraus & Nicol, 2009; Picton & Hillyard, 1988). AEPs can reflect activation of the auditory pathway. This begins with the transduction of auditory stimuli via vibrations of the inner ear, this in turn causes displacement of cochlear fluid and hair cells in the organ of Corti. The auditory signal then travels to spiral ganglion cells and the VIIIth nerve, synapsing in the dorsal cochlear nucleus through to the superior olive in the brain stem. From here all ascending fibers decussate in the lateral lemniscus, then stop in the inferior colliculus in the midbrain and the medial geniculate nucleus in the thalamus. Lastly, the auditory signal travels to the superior temporal gyrus (also known as the primary auditory cortex or Herschel's gyrus), from which the signal diverges to other cortical processing areas, such as the secondary and association cortices, Wernicke's, Broca's area (Goldstein, 2009).

AEPs can be classified based on their response latency, into early, middle and late-latency potentials. The early-latency AEPs are also known as the auditory brainstem response (ABR). They are represented in Figure K3 as auditory components I-V. They are typically recorded in the first 10 ms after stimulus presentation and have very short latencies, lasting the length of the stimulus. They represent activation of the auditory nerve and low midbrain structures (brainstem). They are termed exogenous because they are largely dependent on the physical properties of stimuli, such as modality and intensity (Kraus & Nicol, 2009). Furthermore, their amplitude and latency are dependent on the intensity and rate of presentation of the auditory stimuli (usually abrupt broadband clicks). For this reason they are said to parallel the automatic, data-driven, sensory-analysis processes (Knott, 1989).

The middle-latency AEPs follow the ABR up to ~80 ms. Their neuronal generators are less specific, but reflect activation of the thalamus (for P_0 and N_a) and cortex (for P_a, N_b, and P1). Unlike ABR components, middle and late-latency response are indexed with a 'P' or 'N' to reflect their polarity. This is discussed in the following section, 'ERP components'.

Lastly, the late-latency AEPs are cortical in origin and considerably larger and lower in frequency compared to early and middle-latency potentials. Furthermore, they are highly dependent on stimulus type and recording location,

and may overlap with one another. Late-latency AEPs are usually categorized and described as either exogenous or endogenous potentials.

Exogenous potentials are considered obligatory responses to (sound) stimuli and reflect the physical parameters of the stimuli. For this reason they are also known as sensory evoked potentials. Exogenous potentials describe early and middle-latency potentials, as well as some late-latency potentials. On the other hand, endogenous responses are more cognitive, and as such are sensitive to attentional and cognitive states. Therefore, endogenous potentials reflect information processing and the subjective evaluation of stimuli.

The main late-latency exogenous potentials are P1 (although sometimes P1 is classified as a middle-latency potential), N1, P2, and N2. They have a latency range of approximately 80- 250 ms. They are cortical in origin and are maximal in amplitude at the central top of the scalp. For this reason they may be referred to as the 'vertex potentials'. These components are also sometimes referred to as mesogeneous potentials because they lie inbetween purely exogenous and endogenous components (Picton, 1980). The P1, N1, P2, and N2 are all affected differently by experimental manipulations and have different scalp topographies, suggesting that they are functionally independent with different intra-cranial generators.

Late-latency AEPs that are considered endogenous occur approximately 200 ms post-stimulus (Picton, 1980). These AEPs are still induced by external stimuli, but are not considered obligatory responses. Instead, they are related to high-level cognitive processes such as information processing (Sur & Sinha, 2009) and conscious attention (Kraus & Nicol, 2009). However, wtih auditory stimuli, endogenous components may still be affected by physical properties of the stimuli, including intensity and location (Näätänen & Picton, 1987). Examples of AEPs that are endogenous include the P300 and N400. The P300 is thought to reflect a form of stimulus evaluation and classification, while the N400 is involved in linguist and musical concepts (Daltrozzo & Schön, 2009).

Measuring ERPs

ERPs are measured by repeating a large number of time-locked trials in a single experiment then averaging the data from these trials together. This averaging technique can be achieved because the EEG signal is recorded by sampling neural activation slightly before, during, and after the onset of a

stimulus. Because ERPs are considerably small, ranging from less than a microvolt to tens of microvolts, compared to background EEG activity (which is approximately 50 mV) this averaging procedure helps to improve the signal-to-noise ratio. Therefore, random, nonsystematic noise will be minimized when many signals are averaged together. In this way, the ERP signal becomes salient, while the random activity (noise) averages out and therefore fails to contribute to the ERP. Other methods used to improve the signal-to-noise ratio include filtering, whereby artifacts from non-neuronal electrical activity are removed. For example, the amplifiers used to record ERP data use an in-line filter to truncate electrical activity that occurs above and below certain frequencies. This helps to rid the ERP signal of unwanted, nonsystematic noise, such as muscle activity, movement, electrocardiographic activity, skin potentials, equipment-related artifact, and electrical noise in the environment (Clayson, Baldwin, & Larson, 2013).

ERPs are measured over three properties, amplitude, latency, and scalp distribution. Amplitude indicates the degree of neural activity that occurs in response to stimuli. One way this is measured is through peak amplitude. This is achieved by selecting a time window that surrounds the peak (or trough) in a waveform, then finding the largest peak within the window. In this way, the amplitude represents the magnitude of the component. Latency measures the time point of the peak amplitude and is a measure of processing speed. The temporal resolution of ERPs makes them excellent for investigating the time course of a neural or psychological process. This is achieved by measuring the latency of a specific peak between two different conditions then using this information as a measure of the time needed to process the stimuli (Kappenman & Luck, 2012). Lastly, scalp distribution is shown through topographic images that display amplitude values over the entire surface of the head at a given point in time. In this way, a two-dimensional graphical representation of the amplitude for a specific component can be presented. The amplitude in these images is represented by different colors, for example, red for positive values and blue for negative values (Electrical Geodesics, Inc, 2006).

ERP components

The ERP signal is expressed as a series of positive and negative deflections that occur over time (Nelson & Monk, 2001), which can be seen in

Figure K3. The deflections, termed peaks, waves, or components, are dependent on the orientation of the dipole. As such, their polarity is labeled 'P' for positive going and 'N' for negative going waves. The deflections are also numerically labeled. The numbers are either assigned by the average time, in milliseconds, the deflection occurs after stimulus onset (e.g. P100; N100) or assigned with ordinal numbers with respect to their placement in the series of deflections (e.g. P1; N1). When ordinal numbers are used they are assumed to correspond to the millisecond-labeling method so that P1, the first positive deflection, occurs approximately 100 ms after stimulus onset. In this way, P1 is synonymous with P100 (Luck, 2005). Sometimes the deflections are assigned numbers using a specific time, in millisecond, of when they occur (e.g. N125) or specify a time window (e.g. N20-50), but these are used less often. Lastly, it is common to plot ERP waveforms with negative voltages upward and positive voltages downward. However, this approach is not universal and therefore it is necessary to indicate the polarity of the waveform. This can be indicated with a '+' or '-' sign on the y-axis, where amplitude is plotted, or noted within the waveform figure with an upward or downward-facing arrow, as is done in Figure K3.

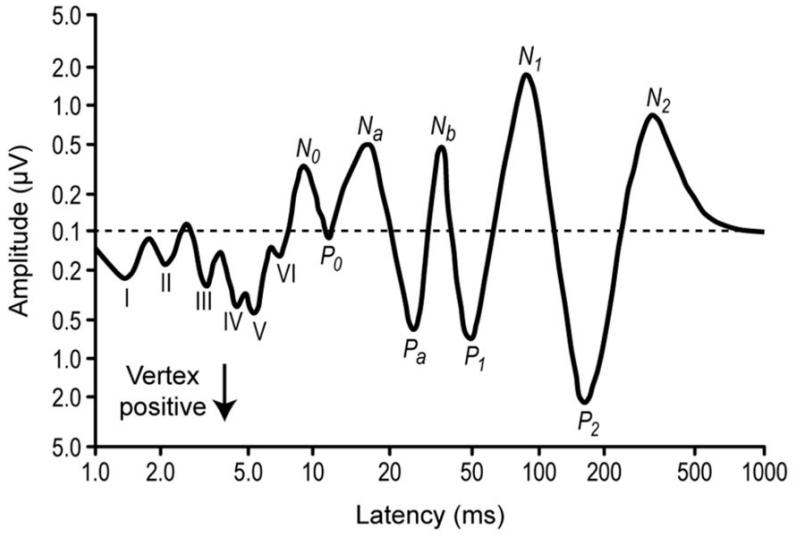

Figure K3. The components of an auditory ERP
The source localization of the early components (I-VI) is the cochlea and auditory brainstem nuclei. The source localization of the middle latency components (N_0, P_0, N_a, P_a, N_b) is the thalamus and auditory cortex, while the source localization of the later latency components (P1, N1, P2, N2) is the auditory cortices as well as the frontal cortex (Key et al., 2005; Picton et al., 1974).

ERP components definition and measurement

ERP components are an important tool for studying the neural correlates of sensory, attentional, and cognitive processes. Investigating these components provides useful information regarding the sequence of perceptual and cognitive operations involved in the processing of stimuli or in the generation of responses. For example, early components reflect mostly sensory and early attentional processes (Pratt, 2011). When processing an auditory event, early ERP components, such as the N1, indicates activity in the first cortical areas that receive sensory input (e.g. auditory cortex). However, a subsequent deflection, such as P2, reflects early stimulus evaluation and feature detection (Luck & Hillyard, 1994) in the temporal cortex. Later ERP components, such as the P_3, are thought to process information at higher cognitive levels, such as during the shifting of attention or updating mental representations in working memory (Donchin, Karis, Bashore, Coles, & Gratton, 1986). In this way, the P_3 is regarded as a 'cognitive' neuroelectric phenomenon because it is produced in psychological tasks that require attention and discrimination of stimulus events that differ from one another (Polich & Kok, 1995). Even later components can reflect responses to violations

of semantic (N400) or syntactic (P600) expectancy (Osterhout, Holcomb, & Swinney, 1994).

Appendix L

Language Background

1. What is your native language?

□ English □ Other (required) _____

2. Do you have a learning/language disorder? (e.g. Dyslexia, ADHD)

□ Yes □ No

3. If yes, please state/explain the learning/language disorder:

_____

_____

4. Do you speak or have you ever studied another language besides your native language?

□ Yes □ No

If yes, please list any other languages that you speak or have studied:

2nd Language _____

3rd Language _____

4th Language _____

5. Please rate your current ability in terms of listening, speaking, reading, and writing in each of your non-native languages: *Very Poor* =1, *Poor* = 2, *Fair* = 3, *Functional* = 4, *Good* = 5, *Very Good* = 6, *Native-Like* = 7

	Language	Listening	Speaking	Reading	Writing
2nd Language					
3rd Language					
4th Language					

Appendix M

Effects of Nicotine on Auditory Perception
Participant Information Sheet and Nicotine Information Sheet

You are invited to participate in a research project. Before deciding please understand the research and what it involves. Read the following information to decide if you wish to take part. This study has been approved by The Department of Psychology Ethics Sub-committee. If there is anything that you are unsure of, or if you would like any more information, please contact Paul Overton at P.G.Overton@Sheffield.ac.uk

The aim of this research is to examine the effects of nicotine on auditory sensory perception using EEG techniques. The study will run from February 2015 to July 2015. We are looking for participants who are smokers and nonsmokers.

What happens to me if I take part?
You will be asked to chew either nicotine gum or regular chewing gum. Whilst doing so you will engage in a reading comprehension and writing task, which will take 25 minutes total. After this, you will be given ear buds and will hear either a high or low-pitched sound. You will then be asked to complete a simple computer task based on the pitch you heard. Throughout the experiment your brain activity will be recorded using standard EEG equipment.

What is EEG?
EEG stands for Electroencephalography, which is a non-invasive technique used to record changes in electrical fields caused by the brain's neural activity. Neural activity in the brain is associated with tiny electric currents that are recorded outside the human head by a number of electrodes attached to the scalp. The procedure of electrode attachment is painless, though it might occasionally leads to discomfort. EEG recording involves wearing a specialized net with attached sponges, which are soaked in a potassium chloride solution and attached to individual electrodes. In this lab we use an adjustable net that is specifically designed to maximize comfort during the fitting process. After the recording session the potassium chloride solution may need to be removed, which is easily done by washing the hair. You will be provided with shampoo, a washbasin, a towel, and a bathroom should you wish to use them. This EEG experiment will require about 30 minutes to apply the electrodes, followed by the experiment, which will take about 45 minutes. In total, the study will take no more than one hour and a half.

What are the possible disadvantages and risks of taking part?
During this experiment you may experience effects of nicotine, which are temporary. These effects may include increases in heart rate, blood pressure,

or breathing rate. You may also feel nervous, a loss of appetite, and later a feeling of relaxation. ***Please see Nicotine Information Sheet for more detail.***

What happens if I wish to discontinue my participation?
If you wish to stop participating during the experiment you are free to do so without any consequences. Your participation is completely voluntary. You are free to refuse participation or to withdraw at any time. If you decide to take part, this information sheet and a consent form will be provided to you. If you have any questions please contact Theresa Veltri at tmveltri2@sheffield.ac.uk. If needed complaints can be directed to registrar@sheffield.ac.uk, +44 (0) 114 222 1101.

Will my taking part in this project be kept confidential?
All information collected will be kept confidential and any information disseminated will not contain your name.

What will happen to the results of the research project?
Research results become part of a PhD thesis. Results will also be presented at conferences and submitted for publication in a peer-reviewed journal. To obtain a copy of these results contact Theresa Veltri.

Contacts for further information
For more information please contact Theresa Veltri: tmveltri2@sheffield.ac.uk, Prof. Paul Overton: p.g.overton@sheffield.ac.uk, +44 (0) 114 222 6624, or Dr. Yanjing Wu: yanjing.wu@sheffield.ac.uk, +44 (0) 114 222 6515.

Nicotine Information Sheet:
PLEASE READ THE FOLLOWING PARAGRAPHS VERY CAREFULLY:

Nicotine is a naturally occurring substance found in the tobacco plant. It has two potential effects on the body 1) it activates the sympathetic nervous system and so may causes a rapid release of adrenaline, which can lead to an increase in your heart rate and blood pressure, as well as a change in your breathing. **For precautionary reasons if you have high blood pressure you will not be allowed to participate in this study.** 2) it may effect the brain by influencing your reaction time and your ability to pay attention, making you feel like you can work better. Pathways mediating reward, memory and arousal are also activated by the drug, hence our interest in how nicotine affects the emotional response to music. Nicotine by itself, and in recreational dosages, is not known to be damaging or toxic. Dangerous doses of nicotine exceed recreational doses by thirty or more times.

Nicotine can be self administered through cigarettes, cigars, pipe tobacco, chewing tobacco, oral snuff, nicotine patches, or nicotine gum. The average strength of cigarettes deposit about 1.9 mg of nicotine into the bloodstream, which is equal to the amount deposited by a piece of nicotine gum containing 4 mg of nicotine. In this study we will use 4 mg nicotine gum (equivalent to one cigarette). This is one of the most commonly used doses in human nicotine research, especially research looking at performance and cognitive function.

This dose has been used safely in smokers, abstinent smokers and people who have never smoked. However, in a small number of people nicotine gum can cause unpleasant feelings in the stomach. These symptoms will be monitored, and if you report marked side effects, testing will be discontinued.

Please remember that you do not have to participate in this study and you have the right to withdraw at any time without any explanation or negative consequences.

I have read and understood this nicotine information sheet

□ Yes □ No

I understand that my participation is completely voluntary and that I have the right to withdrawal from this study at any time without negative consequences.

□ Yes □ No

Appendix N

Identifying Happy, Sad, and Neutral Music

Participant Information Sheet

Invitation paragraph
You are invited to participate in a research project. Before deciding please understand the research and what it involves. Read the following information to decide if you wish to take part.

What is the purpose of the project?
This research wishes to identify music which is experienced as sad, happy, and neutral. It will run from June 2012 to July 2012. You are asked to participate in this study because you listen to music.

What happens to me if I take part?
If you decide to take part you will complete a 25-minute online music survey where you will listen to musical excerpts and will be asked to rate them on their emotional quality. Furthermore, you may be asked to suggest music that you find sad, happy, and neutral.

What are the possible disadvantages and risks of taking part?
There is no known harm of physical or psychological nature to participants involved in this study. However, participants may feel sad, happy, or may experience chills from listening to the musical excerpts.

What happens if I wish to discontinue my participation?
If you wish to stop participating during the experiment you are free to do so without any consequences. Your participation is voluntary. You are free to refuse participation or to withdraw at any time. If you decide to take part, this information sheet and a consent form will be provided to you. If you have any questions or complaints about the study please direct them to Theresa Veltri at muq11tmv@sheffield.ac.uk, Dr. Renee Timmers at R.Timmers@Sheffield.ac.uk, or Dr. Paul Overton at P.Overton@sheffield.ac.uk. If needed, you can direct complaints to the Registrar@sheffield.ac.uk, +44 (0) 114 222 1101.

Will my taking part in this project be kept confidential?
All information collected will be kept confidential and any information disseminated will not contain your name.

What will happen to the results of the research project?
Research results become part of a second experiment where musical stimuli are needed. In this second experiment participants will be administered nicotine, then listen to happy, sad, and chill inducing music. Furthermore, the results from the current survey and from the second experiment will become part of a PhD thesis and may be used for subsequent academic publications and conference presentations. To obtain a copy of this thesis (upon its completion) please contact Theresa Veltri at muq11tmv@sheffield.ac.uk.

Who has reviewed the project?
Depart of Music Ethics Committee

Contact for further information
For more information contact Theresa Veltri: muq11tmv@sheffield.ac.uk, Dr. Renee Timmers: R.Timmers@sheffield.ac.uk, 0114 222 0477, or Dr. Paul Overton: P.Overton@sheffield.ac.uk, 0114 222 6624. Thank you for participating!

Appendix O

Table O1.

Musical excerpt list for pilot study 1

Emotion Category	Title	Artist
Happy	Ants Marching	Dave Matthews Band
Happy	*Outside Villanova	Eric Hutchinson
Happy	*Angel of Harlem	U2
Happy	*Hey Soul Sister	Train
Happy	*She's Electric	Oasis
Happy	Crosstown Traffic	Jimmy Hendrix
Sad	Brick	Ben Folds Five
Sad	Hopeless	Train
Sad	Hundred	The Fray
Sad	*Colorblind	Counting Crows
Sad	God of Wine	Third Eye Blind
Sad	The Scientist	Coldplay
Neutral	Sweet and Low	Augustana
Neutral	Captain	Dave Matthews Band
Neutral	Save Your Scissors	City and Colour
Neutral	Death Defied by Will	Eagle Eye Cherry
Neutral	Here is Gone	Goo Goo Dolls
Neutral	Without Reason	The Fray

Note: * Indicates excerpts used in Study 1 and Study 2

Appendix P

Table P1.

Musical excerpt list for pilot study 2

Emotion Category	Title	Artist
Sad	*Colorblind	Counting Crows
Sad	Everybody Hurts	REM
Sad	Unchained Melody	Righteous Brothers
Sad	Do What you Have To Do	Sarah McLachlan
Sad	Foolish Games	Jewel
Sad	Someone Like You	Adele
Sad (classical)	Adagio for Strings	Barber
Sad (classical)	Schindler's List Theme	John Williams
Sad (classical)	Kol Nidre	Max Bruch
Neutral	Fur Immer	Neu!
Neutral	Hallogallo	Neu!
Neutral	Seeland	Neu!
Neutral	Negativland	Neu!
Neutral	The Scientist	ColdPlay

Note: * Indicates excerpts used in Study 1 and Study 2