Understanding the mechanisms linking childhood trauma, stress and suicide: the role of impulsivity and executive function

Olivia Louise Penrose Rogerson

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IPR statement

This thesis is an alternative style doctoral thesis and comprises jointly authored published manuscripts. The candidate confirms that the work submitted is her own, except where work which has formed part of jointly-authored publications has been included. The contribution of the candidate and the other authors to this work has been explicitly indicated below. The candidate confirms that appropriate credit has been given within the thesis where reference has been made to the work of others.

The work in chapter 2 is a journal publication:

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This large scale cross-sectional study was jointly developed by Daryl O'Connor (DOC) and I (OR). Sample size calculations were conducted by Thom Baguley (TB) and OR. Data analysis was solely performed by OR with supervision from both TB and DOC. OR wrote the first draft of the manuscript and all the authors provided comments and approved the final version.

The work in chapter 3 is a journal publication:

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OR and DOC designed and wrote the study protocol of the longitudinal study. Arianna Prudenzi (AP) and OR recruited the participants. OR conducted the analyses. OR wrote the first draft of the manuscript and all the authors provided comments and have approved the final version.

The work in chapter 4 is a journal publication:

Rogerson, O., O'Connor, R.C., & O'Connor, D.B. (2024). The effects of childhood trauma on stress-related vulnerability factors and daily indicators of suicide risk: an ecological momentary assessment investigation. *Journal of Affective Disorders.* doi: 10.1016/j.jad.2024.02.029.

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The work in chapter 5 is a journal publication:

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This systematic review and meta-analysis was jointly developed by DOC and OR. The data analysis was conducted by OR under the supervision by DOC, and each author contributed to the written publication. Sarah Wilding (SW) and AP, the second and third authors on this publication, acted as the second reviewers. OR wrote the first draft of the manuscript and all the authors provided comments and have approved the final version.

The thesis is constructed in this format in view of providing the examiner(s) optimum clarity over the variety of research findings derived from the PhD. There are six chapters within this thesis, encompassing: Chapter 1) a general introduction section containing the background, rationale and aims of the thesis; Chapter 2) study 1: a cross-sectional investigation of childhood trauma and suicide, a focus on impulsivity, executive function and stress; Chapter 3) study 2: a longitudinal analysis exploring the relationship between suicide vulnerability, impulsivity and executive functioning during COVID-19; Chapter 4) study 3: an ecological momentary assessment (EMA) investigation of the effects of childhood trauma on stress-related vulnerability factors and daily indicators of suicide risk; Chapter 5) study 4: a systematic review and meta-analysis of stress management interventions on cortisol levels in healthy adults; Chapter 6) a general discussion containing implications, future directions, strengths and limitations of the thesis and a conclusion.

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Abstract

Childhood trauma is experienced by a third of young people in the United Kingdom (UK) and is defined as the experience of traumatic, or negative, life events during childhood; including emotional abuse, emotional neglect, physical abuse, physical neglect, and sexual abuse. Importantly, childhood trauma is a key variable in the aetiology of poorer health outcomes, including suicidal behaviour. Of concern, suicidal behaviour, a term capturing both suicidal ideation and suicide attempts, is a major cause of death worldwide, responsible for 1.5% of all mortality, with an estimated 700,000 individuals that die each year by suicide. Moreover, childhood trauma has been implicated in increasing suicide risk, however, the specific pathways through which it influences suicidal behaviour remain unclear. Nonetheless, there is empirical research indicating a clear relationship between distal and proximal risk factors with suicidal behaviour: individuals with pre-existing vulnerabilities, such as childhood trauma and greater impulsive behaviours, are more likely to experience stressful life events and react to them with emotional and cognitive dysregulation which in turn is associated with greater suicidal ideation and suicide attempts.

There is a series of theoretical models mapping the pathways from childhood trauma to suicidal behaviour. Two dominant models being: 1) Lovallo's model, postulating childhood trauma to be associated with reduced stress reactivity, altered cognitive abilities and greater impulsive behaviours which in turn contribute to greater risk of experiencing poorer health behaviours, and 2) the Integrated Motivational-Volitional (IMV) model of suicidal behaviour, proposing three distinct phases in the transition to suicide attempts, highlighting a series of psychological vulnerability factors such as defeat, entrapment, stress and impulsivity that contribute to an increased risk of suicidal ideation and later suicide attempts. Informed by these models, the primary aim of this thesis was to address the evidence gap in understanding how childhood trauma increases the risk of later suicidal behaviour, by examining the role of risk and protective factors and to improve understanding of the nature of these associations.

The current research found childhood trauma and suicide significantly predicted greater impulsivity, poorer executive functioning and greater self-reported stress in a large cross-sectional survey of adults in the UK (chapter 2; study 1). In a longitudinal analysis, individuals with a history of suicidal behaviour appeared to have experienced poorer executive functioning, greater impulsivity and COVID-related stress in the initial phase

of the Coronavirus 2019 (COVID-19) pandemic (chapter 3; study 2). This evidence is corroborated by an ecological momentary assessment (EMA) study where childhood trauma was associated with greater daily stress and daily indicators of suicide risk. The research also uncovered key pathways whereby trauma had indirect effects on reasons for living, optimism, daily thoughts of suicide, defeat and entrapment through executive functioning, impulsivity, sleep quality and stress (chapter 4; study 3). Lastly, evidence was synthesised in a meta-analysis from 58 studies to assess the effectiveness of psychological interventions at improving cortisol levels, finding medium sized effects in positively influencing cortisol levels (chapter 5; study 4).

The findings from this thesis expand upon existing theoretical models and incorporate novel methodological approaches to highlight the complexity of childhood trauma and subsequent potential pathways which can lead to damaging impacts on stress-related vulnerability factors and poorer health outcomes. Greater understanding of pathways by which trauma may impact later health outcomes is essential for development of interventions. Future work is needed to elucidate the precise *causal* mechanisms between these factors and to determine effectiveness of interventions that can attenuate the relationship between childhood trauma and suicidal behaviour.

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List of Abbreviations

ADHD: Attention Deficit Hyperactivity Disorder

ANOVA: Analysis of Variance

APMS: Adult Psychiatric Morbidity Scale

BDI-II: Beck Depression Inventory-II

BIS: Barratt Impulsiveness Scale

BMI: Body Mass Index

CI: Confidence Interval

COVID-19: Coronavirus Disease 2019

CTQ: Childhood Trauma Questionnaire

DEX: Dysexecutive Questionnaire

EMA: Ecological Momentary Assessment

HLM: Hierarchical Linear Modelling

HPA: Hypothalamic-Pituitary-Adrenal

IMV: Integrated Motivational-Volitional

MANOVA: Multivariate Analysis of Variance

MCAR: Missing Completely At Random

MIS: Momentary Impulsivity Scale

NHS: National Health Service

OR: Odds Ratio

PSS: Perceived Stress Scale

SARS: Severe Acute Respiratory Syndrome

SSI: Scale for Suicidal Ideation

SWEMWBS: Short Warwick-Edinburgh Mental Wellbeing Scale

UK: United Kingdom

US: United States

Chapter 1 - Introduction

This introductory chapter outlines the psychological literature on childhood trauma, stress and suicide, alongside associated risk and protective factors considered to be mechanisms in understanding the trajectory between childhood trauma, stress and later suicidal behaviour. Various pathways between mechanisms will be considered, targets for interventions identified and the aims of the thesis will be outlined.

1.1 General introduction: the psychology of suicide

Suicide is a major cause of death worldwide, responsible for 1.5% of all mortality with an estimated 700,000 individuals that die each year by suicide (World Health Organisation, 2023). Although there have been major suicide prevention efforts, suicide remains a profound public health problem, with each case of suicide impacting a community of over 100 people (Cerel et al., 2019). The effects on this community are far-reaching with reports of feelings of grief, guilt, and even suicidal ideation (Cerel et al., 2016). For this reason, there has been a considerable amount of research to better understand the causes of suicidal behaviour (this term encompasses both suicidal ideation and suicide attempts), exploring factors with origins in biology, psychology (such as impulsivity, executive function, worry, rumination, feelings of defeat, entrapment, and stress), environment (housing, poverty), social environment (connectedness, support, and loneliness), and genetics. There have been a number of theoretical models developed, aiming to understand how a multitude of factors interact and lead to suicidal behaviour (Mann et al., 1999; O'Connor & Kirtley, 2018; O'Connor & Nock, 2014). Despite the research advances and the number of factors identified, predicting and preventing suicidal behaviour remains low, with predictive ability failing to improve over the past 50 years (Franklin et al., 2017; Zalsman et al., 2016).

Traditionally, suicide research has focussed on singular risk factors (such as pre-existing mental health conditions, gender, substance abuse; see Probert-Lindström et al., 2020 for a review), whilst this has increased the understanding of the number of different factors contributing to increased suicidal behaviour, it fails to recognise the complexity of suicidal behaviour being a result of the potential interaction between many of the risk and protective factors themselves. Given a recent network analysis found all the psychological risk factors for suicide were interrelated, it is argued that suicidal ideation is the result of the interplay of many different factors, some of which have direct or indirect relationships with suicidal ideation itself (De Beurs et al., 2019). Yet it is still unknown how the

complex interplay amongst factors relate to *both* suicidal ideation and suicide attempts. The thesis defines suicidal ideation as thoughts of ending one's own life which can be active, planned, or passive, with a wish to die but with no plan and suicide attempts are considered self-injurious behaviour with inferred or actual intent to die (Turecki et al., 2019).

To understand suicidal behaviour, it is important to consider the risk factors involved in the transition from ideation to attempt rather than considering risk factors for suicidal behaviour more generally. To add further complexity, most risk factors are found to predict suicidal ideation but not distinguish suicide attempts from suicidal ideation (May & Klonsky, 2016). This is supported by epidemiological research whereby risk factors accounted for approximately 60% of the variability in suicidal ideation but only 7% of attempts amongst individuals with a history of suicidal ideation (Glenn & Nock, 2014). It seems that we are understanding more about the contributors to increased suicidal ideation, but it is not as clear for suicide attempts. This is critical as most individuals that experience suicidal ideation do not go on to make attempts (May & Klonsky, 2016; Nock et al., 2008). Although there is likely a degree of overlap, research should explore the distinct risk factors for suicidal ideation and risk factors for suicide attempts (Klonsky et al., 2016).

There is a need for research to be based upon ideation-to-action theoretical models that acknowledge the complexity of the interaction of risk factors leading to both suicidal ideation and suicide attempts.

1.1.1 The Integrated Motivational Volitional model of suicidal behaviour

There are several theoretical models that attempt to explain the underpinnings of, and trajectories to, suicidal behaviour. A model of particular relevance and importance for the current thesis is the Integrated Motivational-Volitional (IMV) model, combining a plethora of constructs from existing models of suicidal behaviour into one theoretical framework to understand the complexity, and development of both suicidal ideation and suicide attempt, from biological, social and psychological origins (O'Connor & Kirtley, 2018).

The IMV acknowledges the complex interplay of factors from existing psychosocial models such as the diathesis-stress hypothesis (Schotte & Clum, 1987), the theory of planned behaviour (Ajzen, 1991) and Williams' arrested flight model (Williams, 2001). The IMV is a tripartite model with the premise that suicide is a behaviour that results

from a complex interplay of factors. There are specific moderators at each stage of the model which facilitate or prevent progress to the next stage, in addition to acknowledging that the prediction of suicidal ideation differs from the prediction of suicide attempts (O'Connor & Kirtley, 2018). The first stage of the model, the pre-motivational stage, recognises that when vulnerability factors (such as socioenvironmental factors) are combined with acute or chronic life stressors (defined as stress that lasts for a short period of time or consistent experience of stress over a long period of time, respectively) the combination increases the probability that an individual will experience an adverse psychological reaction to stress – forming the foundations for greater vulnerability to suicidal behaviour. An abundance of evidence links stressful life events, such as childhood trauma, various types of interpersonal stressors, such as financial, romantic or employment difficulties to an increased risk of suicidal behaviour but it is less well understood regarding the mechanisms through which childhood trauma increases the risk (O'Connor et al., 2018; O'Connor & Nock, 2014). The second stage of the IMV, the motivational phase, acknowledges feelings of defeat and humiliation triggering feelings of entrapment which in turn predict suicidal ideation as a solution to current circumstances. Throughout this process there are stage-specific moderators that facilitate or prevent progress to the next step within the motivational phase: threat-to-self moderators such as executive functioning, predict the transition from defeat to entrapment, whereas motivational moderators, such as resilience, social support, predict suicidal ideation. Lastly, the volitional stage, recognises that volitional moderators, such as impulsivity and past suicidal behaviour, predict the enactment on suicidal ideation. The IMV model outlines a clear link between negative life events, such as childhood trauma, stressors, and increased suicidal behaviour, yet the mechanisms by which this pathway interacts are not fully elucidated. Importantly, not all people who experience a negative life event such as childhood trauma develop suicidal behaviour, which suggests there are interacting mechanisms to produce suicidal behaviour. This emphasises the importance in using theoretical models as a framework on which to base suicide research. The IMV will be used as the foundation of the current research in this thesis to better understand the mechanisms linking childhood trauma, stress and suicidal behaviour.

A facet central to the IMV, as well as the aims of the current thesis, is to understand the interplay between distal and proximal suicide risk factors (Hawton & van Heeringen, 2009). Distal risk factors are pre-existing vulnerabilities that facilitate suicidal ideation, or behaviour, despite being years or months away from the event itself; including early

traumatic life events (Cohen et al., 2018) and personality characteristics (e.g. impulsivity). Proximal factors, on the other hand, pose an imminent suicide risk, from availability of means, current mental and physical health, and acute stress. Although distal risk factors can suggest who may be more likely to experience suicidal ideation and suicide attempt at a point in their lives, they do not help identify *when* individuals may be at greatest risk of an attempt. Whereas proximal risk factors point to individuals with greatest short-term intensity of suicidal ideation and probability of when individuals may act on their thoughts (Bagge et al., 2013). There is research indicating a clear relationship between distal and proximal risk factors with suicidal behaviour: individuals with pre-existing vulnerabilities, such as childhood trauma and greater impulsive behaviours, are more likely to experience stressful life events and react to them with emotional and cognitive dysregulation which in turn is associated with greater suicidal ideation and suicide attempts (Cohen et al., 2022). The current thesis plans to explore the mechanisms linking childhood trauma and suicidal behaviour that are both proximal and distal risk factors to ensure research begins to untangle the complex interplay between factors in increasing suicide risk, which arguably holds most utility in preventing suicide.

1.1.2 Our ability to predict suicide

Suicide, in particular, suicidal ideation, has been shown to fluctuate considerably (Franklin et al., 2017; Hallensleben et al., 2018). Asking someone to retrospectively report how strong their wish to die has been 'on average' over the past year, does not capture the true variation in suicidal ideation (Kleiman et al., 2017). Despite decades of research, little is known about the short-term variability in suicidal ideation which arguably has the greatest clinical utility in increasing understanding of someone's wish to die. Given research that retrospectively assessed suicidal ideation in the 24 hours prior to a suicide attempt found 45% of the variation in predicting suicidal ideation was due to within person short term changes, is evidence that suicidal ideation varies considerably over short periods of time (Bagge et al., 2014). Additionally for a majority of individuals in an ecological momentary assessment (EMA) study, suicidal ideation varied dramatically over the course of most days, more than over one quarter, supporting the narrative whereby assessing suicidal ideation and associated risk factors over long periods of time at infrequent intervals does not accurately depict the reality of how quickly, and frequently, suicidal ideation and its risk factors fluctuate over periods of minutes, hours or days (Kleiman et al., 2017). Moreover, the previous suicide risk factors identified using cross-sectional methodology, such as hopelessness, burdensomeness, and loneliness, are

correlated with suicidal ideation but may not have the predictive utility for the proximal changes in suicidal ideation across short time periods of hours (Kleiman et al., 2017). Therefore, there is a risk that extant methods are missing important fluctuations in suicidal ideation, missing critical insights into the predictors of suicidal ideation (Franklin et al., 2017).

An outstanding question is what, and how do distal and proximal risk factors interact to increase the risk of suicide? What mechanisms are responsive to intervention? There is a gap in the current literature and a need to address these questions. There is also need to consider micro-longitudinal methods to establish the predictive ability of the mechanisms increasing suicide risk (Franklin et al., 2017). Micro-longitudinal methods offer an approach to study within-person associations between constructs that are not typically available in less time-intensive methods relying on repeated measurement of self-reported experiences in daily life (Czyz et al., 2023). Specifically EMAs will be used in the current thesis as an approach to examine the short-term predictors of suicidal ideation and suicide attempt, whilst considering distal factors such as childhood trauma. The reasoning for utilising this methodology is to improve our understanding of the short-term predictors of suicide instead of relying entirely on one point in time assessments, that a considerable amount of previous research has used (e.g. Cohen et al., 2022). Given the gap in the literature, the current thesis aims to gain a greater understanding of the pathways between distal risk factors, with more proximal risk factors, including stress, to better understand suicidal ideation and suicide attempt.

1.2 The relation between childhood trauma and suicide

There have been advances in identifying factors involved in suicide, a pivotal factor being the experience of childhood trauma. Childhood trauma is defined as traumatic, or negative life events such as emotional abuse, emotional neglect, physical abuse, physical neglect, and sexual abuse that happen during childhood and adolescence (Bernstein et al., 2003). Childhood trauma is an important variable in the aetiology of suicidal behaviour and is associated with poorer health outcomes in adulthood. Of concern, it is estimated that 6.3% of individuals in the United Kingdom (UK) reported experiences of sexual abuse and 14.8% reported physical abuse (Bellis et al., 2014). For young adolescents who have experienced childhood trauma, already more than 25% had at least one health problem (Flaherty et al., 2013). Research underlines the need to consider the influence that trauma exposure has on later health outcomes. There is a documented association between childhood trauma and suicide risk; a meta-analysis of longitudinal studies identified a

connection between sexual and emotional abuse, as well as physical neglect, and later suicide attempts (Zatti et al., 2017). Additional research indicates that approximately 80% of individuals with a history of suicide attempts have reported experiencing at least one moderate to severe form of childhood trauma (O'Connor et al., 2018). This was replicated in further empirical research again finding that approximately 80% of adults who had attempted to end their own life had experienced at least one moderate-to-severe childhood trauma (O'Connor et al., 2020a). Therefore, it is essential to examine which mechanisms may be responsible for driving this association between trauma and suicidal behaviour.

Consistent associations between negative early life events and later suicidal ideation and behaviour are reported across a series of meta-analytic investigations (Carr et al., 2013; Liu & Miller, 2014). A population-based study found childhood neglect, psychological abuse and physical abuse were strongly associated with the onset of new suicidal ideation and suicide attempts in a 3-year follow up period (Enns et al., 2006), in addition to the cumulative effect of the number of childhood adversities having a strong, positive relationship with suicidal behaviour in youth (Serafini et al., 2015). In a prospective cohort design, high and moderate trauma was found to be associated with later suicide attempts in drug users (Marshall et al., 2013). Too often though, studies in suicide prevention research focus on risk factors that have relatively low predictive power and perhaps more importantly do not account for *why* people die by suicide (O'Connor & Nock, 2014). There is also need to understand the pre-psychopathological processes prior to the onset of psychopathology so we can disentangle the mechanisms associated with suicidal behaviour and not as a *result* of psychopathologies.

The associations and precise mechanisms by which trauma links to poorer health outcomes, including suicide, is not well understood because extant literature has focussed predominantly on study samples which have already presented with physical and mental health difficulties. As a result, research comprised of individuals with pre-existing physical and mental health difficulties precludes the ability to determine mechanisms of suicide risk; associations reported in individuals who already show psychopathologies or chronic illness may instead result *from* illness itself (Tinajero et al., 2020). Despite a growing evidence base, a large number of the existing studies fail to explore the associations with other psychopathological factors and truly understand the micro-fluctuations of the associations, such as what is the extent to which childhood trauma may be linked to other important modifiable risk factors? A notable exception is a recent study by Tinajero et al., (2020) that utilised micro-longitudinal methodology. This study

conducted in healthy adults found childhood abuse and neglect were significantly associated with difficulties in emotion regulation, pre-sleep arousal, sleep quality, daily hassles, and executive function difficulties, whereby the associations reported could not be attributed to existing illness or psychopathology. Nevertheless, important questions remain unanswered. How do these aforementioned associations relate to suicide risk? How does childhood trauma have such serious consequences for mental and physical ill-health in adulthood? This thesis will build upon this work to understand the mechanisms linking trauma to later health outcomes.

1.3 Childhood trauma and other health outcomes

Traumatic experiences during childhood have the ability to induce significant biological changes, known as biological embedding, as well as modify the development and responsiveness of allostatic systems, exerting long-term negative effects on nervous, endocrine, and immune systems (Danese & McEwen, 2012). In fact, early life adversities links to negative health outcomes are well documented; individuals who have experienced early-life adversity report more physical and mental health difficulties in adulthood compared to those with no adversity (Bellis et al., 2014) including the development of psychopathological disorders such as depression (Nelson et al., 2017), anxiety (Li et al., 2016) and suicide (Angelakis et al., 2019). As well as those who have experienced childhood trauma having higher rates of chronic illness (Mock & Arai, 2011), chronic pain in later life (Davis et al., 2005), and poorer physical ill health in adulthood such as greater risk of cancer, diabetes and stroke (Bellis et al., 2014). Beyond the initial mental and physical health outcomes, the impact of trauma also has far reaching effects on additional health and life outcomes including unemployment (Egan et al., 2015), and overall mortality (Ploubidis et al., 2021).

Although it is well established that childhood trauma is associated with later health difficulties, it has not been clear exactly what mechanisms link trauma to later health outcomes or whether the relationship is causal. This is because maltreated children often have other psychiatric risk factors that could contribute to poorer health outcomes. A recent meta-analysis including 34 quasi-experimental studies found a *causal* contribution of childhood maltreatment on a broad range of mental health outcomes including suicide, in amongst other disorders such as anxiety, alcohol abuse, and attention deficit hyperactivity disorder (ADHD) (Baldwin et al., 2023). The possibility that childhood trauma may affect broad factors underlying multiple disorders, such as impaired cognitive function (e.g. poor executive functioning, impulsivity) and emotional dysregulation,

instead of suicide-specific or disorder-specific risk factors, is also supported by evidence from non-quasi experimental studies that show childhood trauma may affect a multitude of factors underlying multiple disorders (Cecil et al., 2017; McLaughlin & Lambert, 2017). Yet the mechanisms by which trauma impacts broad factors underlying mental health are still to be fully elucidated. This thesis aims to shed light on these mechanisms, specifically the role of executive function and impulsivity.

1.4 Lovallo's model

A theoretical model by Lovallo, (2013) suggests that experiencing childhood trauma can lead to modifications in frontolimbic brain functioning that may contribute to reduced stress reactivity, altered cognition (including more impulsive behaviours and poorer executive functioning), and unstable affect regulation (Lovallo, 2016). This trajectory of a more impulsive behavioural style could in turn increase the risk of addiction and the engagement in poor health behaviours. Evidence from a cohort study of healthy young adults found that early life adversity was associated with reduced cortisol reactivity to an acute stressor in adulthood (Lovallo, 2013). In line with this model, findings by O'Connor et al. (2018) suggest that blunted hypothalamic-pituitary-adrenal axis (HPA) axis activity in response to stress and during rest in individual's with high levels of childhood trauma provide evidence for one of the pathways suggested in Lovallo's model and extend his model to suicidal behaviour (Lovallo, 2013). However, to date, relatively little is known about the effects of childhood trauma on stress, and cortisol processes, in the context of suicide. Therefore, it is important to elucidate the mechanisms linking childhood trauma, stress, and suicide risk through Lovallo's framework – which this thesis seeks to accomplish. As highlighted in both the IMV and Lovallo's model, there are a selection of mechanisms that may underpin the association between childhood trauma, stress, and suicide, including many potential pathways by which the mechanisms may interact. The key mechanisms highlighted to have pivotal roles are executive function and impulsivity (Lovallo, 2013). These mechanisms are central components of the current thesis. In addition, there are other noteworthy mechanisms that have received further research attention and will be discussed as part of the thesis, including defeat and entrapment (O'Connor & Kirtley, 2018). Both models are presented in Figure 1 below.

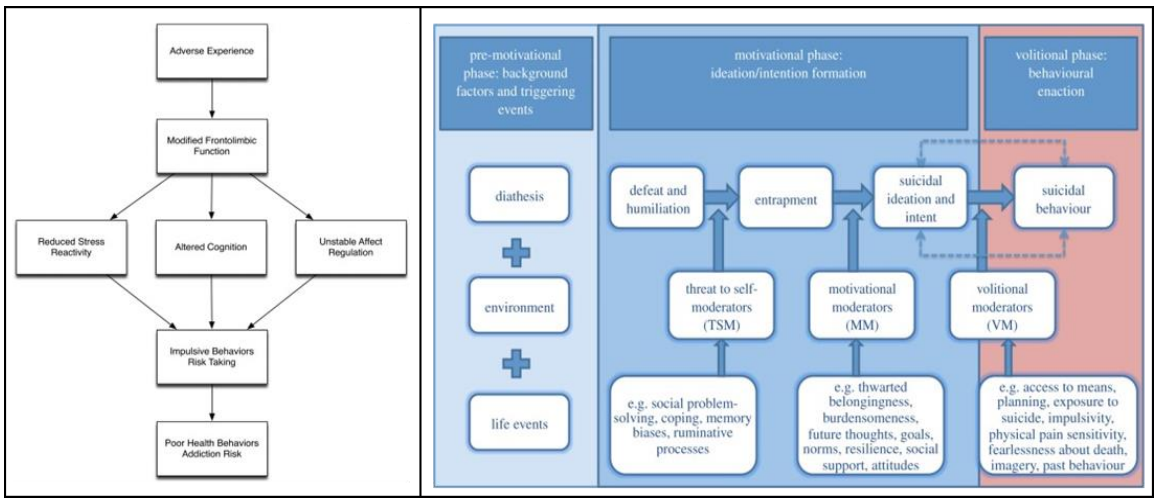


Figure 1. Lovallo's model from adverse life experience to risky health behaviours (Lovallo, 2013); the IMV model of suicidal behaviour (O'Connor, 2011; O'Connor & Kirtley, 2018).

1.4.1 The role of executive function and impulsivity

Executive function is put forward as a candidate mechanism linking childhood trauma, stress, and suicide. Executive function is a broad term referring to an array of higher cognitive processes which manage and control thoughts, emotions, and actions, required to shift attention or achieve goals. The term largely refers to three, interrelated core skills; inhibitory control, working memory and cognitive flexibility (Diamond, 2013; Miyake et al., 2000). The pathways between childhood trauma, executive functioning, and suicide, are not completely established. Findings from clinical and non-clinical settings suggest neurological changes during development, instigated by childhood trauma, give rise to subsequent difficulties in executive function shown by poor co-ordination of thoughts and actions (Gould et al., 2012). This pathway is well described in the theoretical model by Lovallo and colleagues where experiencing childhood trauma can result in neuroanatomical changes in the frontolimbic brain areas which affect individuals functioning and subsequently reduce stress activity, alter cognition through a focus on shorter term goals with greater impulsive behavioural styles and unstable affect regulation (Lovallo, 2013). Collectively, the negative consequences listed contribute to poorer working memory (a facet of executive function) and an overall impulsive behavioural style, shown through poor decision making that can risk a greater likelihood of engaging in poorer health behaviours.

Similarly, in a study of young adults, childhood trauma was linked to a greater risk for cognitive impairments and impaired executive functioning (Lu et al., 2017). As well as, a meta-analysis finding youth who had experienced childhood trauma had impaired

executive function compared to controls (Kavanaugh et al., 2017). Across ages, it appears that individuals who have experienced childhood trauma often demonstrate neuropsychological deficits in executive function that persist into adulthood (Gould et al., 2012; Zelazny et al., 2019). To add to the trauma – executive functioning relationship, stress has been shown to negatively impact executive functioning, which, if already dysregulated from the experience of childhood trauma, could have further damaging effects on behaviour and health. As recent evidence found working memory and cognitive flexibility were impaired under high stress situations (Shields et al., 2016).

Additionally, poorer executive functioning has been recently associated with greater levels of suicidal behaviour (Zelazny et al., 2019). Moreover, a study by McGirr et al. (2010) demonstrated a sample of first-degree relatives of those who died by suicide not only had a blunted cortisol response to stress but also had impaired executive functioning. McGirr et al. (2010) has argued that poorer executive function in response to real-life stressors (such as financial, health or relationship stressors) could be a risk factor that increases the likelihood of suicidal behaviour. Taken together, it appears that poorer executive function is associated with vulnerability to suicide. However, it is important to elucidate how poor executive functioning may contribute to increased suicide risk (Bredemeier & Miller, 2015). Is it that vulnerable individuals with impaired inhibition may be unable to resist enactment on their suicidal thoughts? Is it that executive functioning becomes most compromised following exposure to acutely stressful events (c.f., Diamond, 2013)? A review by Bredemeier and Miller (2015) has suggested that stressful life events, such as childhood trauma, may exacerbate the effects of executive dysfunction on suicidal behaviour. However, these relationships are yet to be fully understood in the general population. Little is known about how collectively childhood trauma, stress, impulsivity, and executive functioning interact particularly in the context of increasing risk of suicidal behaviour. Therefore, this thesis will use a variety of methodologies to explore the role of stress and executive functioning, in explaining the association between childhood trauma and suicide.

It is noteworthy that the experience of childhood trauma is elevated amongst individuals who have a previous suicide attempt and researchers have suggested a mediated pathway exists where early stressful experiences alter cognitive function which contribute to altered thought processes that can increase the occurrence of suicidal behaviour later in life (O'Connor et al., 2018). Therefore, it seems imperative, that methodologically, to also explore executive function in relation to suicidal behaviour in individuals who have

experienced childhood trauma, but not suicidal ideation, prior to symptomology developing. This investigation will form part of the current thesis research to help identify mechanisms and associations that link childhood trauma and negative health outcomes and to obtain evidence to inform later prevention and intervention.

Another important variable that has been linked to suicidal behaviour is impulsivity. It is defined as one's inability to effectively regulate behaviours, such as inappropriate pre-emptive responses and risk taking with little forethought for the consequences of the actions being taken (Chamberlain & Sahakian, 2007). Impulsivity is related to executive function as it is characterised by behaviours which reflect impaired self-regulation including acting without considering consequences and risk taking. Impulsivity is a key factor implicated in both suicide models (the IMV; O'Connor & Kirtley, 2018) and stress-diathesis models (Mann et al., 1999). Impulsivity is considered to be both involved in a volitional phase factor in the IMV as well as in the trait like diathesis, and when combined with a stressor, it increases the risk of suicidal behaviour. Impulsivity is a mechanism therefore considered to be a link between childhood trauma and suicide (Braquehais et al., 2010; Roy, 2005). Greater early adverse life experiences have been shown to be associated with both poorer executive function (Narvaez et al., 2012) and to predict greater impulsivity (Lovallo, 2013).

Previous meta-analytic investigations have found evidence of significant associations between impulsivity and suicidal behaviour but these have been found to be small in magnitude, suggesting impulsivity's relationship with suicidal behaviour is likely to be indirect rather than causal (Anestis et al., 2014). In addition, a cross-sectional study in patients with major depressive disorder by Onat et al. (2018) found there was a positive correlation between greater impulsivity scores and poorer executive function. Therefore, it is likely that there are both direct and indirect pathways between childhood trauma and suicidal behaviour. Yet it is not clear how impulsivity relates to childhood trauma, stress, executive function, and suicidal behaviour collectively. From this research, it is clear that not only impulsivity is associated with distal risk factors such as childhood trauma, but it is also linked to proximal risk factors such as stress and executive function, as well as suicidal behaviour itself.

1.4.2 Executive function, stress and cortisol

Stress is well documented to have clear yet complex effects on executive function, with the effects driven mostly through the stress hormone cortisol (McEwen, 2012). Stress can be operationalised in a number of ways and executive function is also a broad, multi-dimensional construct involving several high-level cognitive processes, each depending upon distinct areas within the pre-frontal cortex (Girotti et al., 2018). Indeed, it is unsurprising that empirical studies investigating the relationship between executive function, stress, and cortisol have therefore been inconsistent in their findings (Shields et al., 2015). Previous studies have found positive associations between the cortisol awakening response and specific facets of executive function, such as working memory (Almela et al., 2012) and attention switching (Evans et al., 2012). Conversely, other studies report a negative association between the cortisol awakening response and planning and problem-solving (Butler 2017), while some studies show no association at all with working memory, attention switching and cognitive inhibition (e.g. Ennis et al., 2016). Additionally, some studies suggest that, in certain conditions, stress can improve working memory performance (Schoofs et al., 2013). Overall, this suggests that cortisol has differential impacts on facets of executive function. Confirming this notion, a recent meta-analysis found cortisol to impair working memory, but enhance inhibition (Shields et al., 2015). Stress should thus be investigated as a crucial mechanism in the study of executive function, given that its influence on cognition is closely tied to cortisol dynamics (Lovallo et al., 2013). However, there remains a gap in our understanding as to how this relationship is related to childhood trauma and later suicide risk. Consequently, clarifying the pathways connecting childhood trauma, dysregulated cortisol levels, stress, executive function and suicide risk is essential for identifying at-risk individuals.

1.4.3 Other mechanisms of importance: defeat and entrapment

As outlined above, the relationship between childhood trauma, stress and suicide is complex and influenced by other risk and protective factors. It is not surprising that impulsivity and executive function are therefore not mutually exclusive in their association with suicidal behaviour. In fact, there are a number of other factors considered to be mechanisms that may explain the links between childhood trauma and later suicidal behaviour. In particular, research attention has now turned to investigating defeat and entrapment. Defeat is the overwhelming feeling of powerlessness from damage to social position, identity, or goals (Sloman & Gilbert, 2000). Entrapment is the experience of feeling trapped in a situation with no foreseeable escape, a situation could be one's own thoughts and feelings (internal entrapment) or a situation (external entrapment) (De Beurs

et al., 2020; Gilbert & Allan, 1998). The IMV model proposes that feelings of defeat can trigger feelings of entrapment, which in turn predict suicidal ideation as a solution to one's current situation (Branley-Bell et al., 2019; O'Connor & Kirtley, 2018). Previous research shows that entrapment is key mechanistically in pathways to suicidal ideation, and suggests that entrapment may also account for the emergence of suicidal ideation across time periods of 3 to 12 hours (van Ballegooijen et al., 2022). It is notable that, when measured at three hours, entrapment had two-way temporal association with defeat and suicidal ideation and could therefore constitute a mediated pathway between defeat and suicidal ideation. The mediated pathway between defeat and suicidal ideation is supported by cross-sectional evidence in both meta-analytic investigations and systematic reviews (Siddaway et al., 2015; Taylor et al., 2011). Additional longitudinal evidence provides further support for this association that entrapment mediates the association between defeat and suicidal ideation (Branley-Bell et al., 2019). Given their fundamental role in the suicide risk trajectory, defeat and entrapment are important risk factors to consider when elucidating the pathways between childhood trauma, stress, and suicide, as well as considering for relevant clinical assessment and being a potential intervention target.

1.5 Psychological interventions for cortisol

Given the prevalence of suicide, preventing it ought to be a priority. Yet the exact intervention targets and timing for *how* to prevent suicide are complex; intervening *after* suicidal ideation has been reported may not be the most effective. It is imperative to determine the pre-psychopathological factors, prior to the onset of suicidal ideation, to tailor intervention prior to ideation. Especially in light of a meta-analytic investigation finding childhood maltreatment to be associated with 2.5-fold greater odds for suicidal ideation in adulthood (Angelakis et al., 2019). Exploring the mechanisms by which childhood trauma is associated with suicide risk factors, prior to the development of mental and physical health problems may help further elucidate the pathways that increase future suicide vulnerability and may identify key targets for intervention.

Childhood trauma has been clearly linked to altered functioning of the HPA axis (D. B. O'Connor et al., 2021a). Recent work has found that higher levels of childhood trauma are associated with lower resting cortisol, and blunted cortisol reactivity to stress, in adults who also have a previous suicide attempt (O'Connor et al., 2018). These findings suggest that experiencing childhood trauma may predispose individuals to vulnerability to suicide by leading to blunted HPA axis activity to stress later in life. In line with these findings,

that childhood trauma leads to altered functioning of the HPA axis, individuals with moderate to severe childhood trauma, exhibited lower cortisol reactivity to a psychosocial stressor, relative to no trauma controls (Carpenter et al., 2007). Indeed, dysregulation of cortisol is well documented across a variety of studies, with evidence synthesised in a meta-analysis to find cortisol dysregulation significantly worsened outcomes including depression, fatigue, immune and inflammatory outcomes, obesity, cancer, other physical and mental health outcomes, and mortality (Adam et al., 2017).

Given our ability to identify at risk individuals who have experienced trauma, and the damaging effects of trauma on cortisol dysregulation, there is the need for intervention through targeting the stress response system prior to the onset of serious mental and physical health difficulties appear, including suicidal behaviour. Previous interventions targeting the stress hormone, cortisol, are well documented; cognitive behavioural stress management reduced serum cortisol and increased relaxation in women who suffer from breast cancer (Phillips et al., 2008). As there are a multitude of pathways between childhood trauma, stress, and suicide, there is a need to understand whether the effects of childhood trauma on cortisol levels are amendable to psychological intervention. The ability to influence cortisol with psychological interventions is important for individuals at high-risk of suicidal behaviour; individuals who had made a suicide attempt in the past year had a blunted cortisol response to stress, although individuals with a more historical suicide attempt had cortisol responses closer to that of the group with no history of suicidal behaviour (O'Connor et al., 2017). This finding questions whether psychological, or pharmacological, intervention could produce benefits in the time following a suicide attempt through partial facilitating the recovery of the HPA axis stress response system (O'Connor et al., 2018). There is an urgent need for research to investigate the effectiveness of psychological interventions, particularly therapies focused on childhood trauma. To comprehensively comprehend the connections between early adversity and suicidal tendencies, it is essential to examine not only psychological assessments but also endocrine evaluations.

1.6 The impact of COVID-19

The thesis research was conducted throughout the coronavirus disease 2019 (COVID-19) pandemic. This presented as an opportunity to understand how previously identified suicide risk and protective factors, originating from the IMV, interacted longitudinally across the COVID-19 UK lockdown. In particular, it allowed for an exploration of how naturally occurring stress impacts already vulnerable individuals, such as those who have

experienced previous suicidal ideation or attempts. Infectious disease related public health emergencies are notable in that they simultaneously increase the presence and severity of multiple risk factors; potentially having a knock-on effect on suicide rates (Zortea et al., 2021). Notably, previous epidemics such as the severe acute respiratory syndrome (SARS) epidemic have been associated with a rise in death by suicide (Zortea et al., 2021).

COVID-19 had a profound effect on all aspects of society, especially on physical and mental health; the government enforced lockdown periods provided the basis to exacerbate known risk factors for suicide (O'Connor et al., 2020b). In particular, risk factors for suicide such as stress, isolation, mental health difficulties, economic hardship, were likely to be intensified by COVID-19 (Que et al., 2020). Stress in particular can lead to other physiological changes and negative consequences for health that can be important predictors of suicidal risk (O'Connor et al., 2020a). Moreover, increased stress is known to impair executive functioning, including cognitive flexibility, and working memory, which is argued to indirectly impact later suicidal behaviour (Shields et al., 2016). Previous research alludes to limited social connectedness leading to impulsive behaviours, with social support moderating the relationship between impulsivity and suicide risk (Kleiman et al., 2012) therefore, it is likely that this observed pattern of association was only intensified throughout COVID-19. In addition, there were periods of great uncertainty felt by the UK population throughout the pandemic, due to the unprecedented speed at which COVID-19 was transmitted. It is known that uncertainty is considered more stressful as a state than knowing that something bad is going to occur (de Berker et al., 2016) and it was reported that fear of illness being the most significant risk factor for increased stress (Qiu et al., 2020; Tang et al., 2020). Therefore, there was an opportunity for this thesis to explore how naturally occurring stress may impact known suicide risk factors and later suicidal ideation and suicide attempts.

Specifically, it was reported that vulnerable individuals faced an increased risk of negative health outcomes due to COVID-19 – including women, young individuals, and those with pre-existing mental and physical health conditions (O'Connor et al., 2022). A meta-analytic investigation suggested that while there was an overall increase in mental health symptoms during the pandemic, this increase was more pronounced in individuals with pre-existing physical health conditions (Robinson et al., 2022). Moreover, individuals with a history of childhood trauma faced additional challenges during the pandemic. The lack of social interaction and connection, resulting from the restrictions,

provided the foundations to potentially increase feelings of entrapment, reflecting past traumatic experiences and re-traumatisation (Taggart et al., 2021). Indeed, an indirect relationship was found between childhood trauma and poorer mental health through feelings of loneliness and lack of social support throughout the pandemic (Seitz et al., 2021; Shreffler et al., 2021).

A previous suicide attempt is a robust predictor for repeated attempt and later death by suicide (O'Connor et al., 2013). Additionally, suicidal ideation can transcend into enactment in the presence of other risk factors (Gunnell et al., 2020). It is possible that risk factors for suicide were exacerbated during the current pandemic, such that when known risk factors are combined with pandemic induced feelings of distress or entrapment from the lockdown restrictions, it acts as a catalyst for thoughts to develop into suicide attempts (O'Connor & Kirtley, 2018). For instance, there were reports of an association between COVID-19 distress and past month suicidal ideation and suicide attempts (Ammerman et al., 2021). Although, it is likely that some protective factors were at play during the pandemic whereby individuals felt a sense of belonging, or social connectedness that could buffer against the aforementioned negative effects of the COVID-19 pandemic and the hypothesised increase in suicide risk (O'Connor & Kirtley, 2018; Reger et al., 2020). Therefore, for these reasons, it was felt there was an unique opportunity to understand how known risk and protective factors interact during a naturally occurring, stressful period to later impact suicidal behaviour.

Stress can be understood in multiple ways: physiologically, through cortisol measurement, and psychologically, through self-reported perceived stress. Both cortisol levels and perceived stress are well-documented to correlate with childhood trauma and subsequent suicide risk (O'Connor et al., 2018), yet few studies have empirically connected these elements to identify a specific pathway of risk. The current PhD research aimed to investigate the relationship between childhood trauma, stress, and suicide risk, with a particular focus on cortisol as a biomarker of stress. Building upon recent research by O'Connor et al. (2018), this PhD initially aimed to employ both cortisol and perceived stress as measures to explore this pathway comprehensively. The goal was to understand not only the relationship between measures of stress themselves but also in relation to childhood trauma, suicide and associated risk factors.

However, the onset of COVID-19 disrupted the original aim and approach of the PhD, making the collection of cortisol data no longer feasible and prompting a shift to relying

solely on self-reported measures of perceived stress. Although cortisol was omitted from the empirical studies, it remained central to the systematic review, underscoring the significance of cortisol in understanding how childhood trauma impacts later health outcomes and suicide risk as a reliable and potentially modifiable mechanism for intervention.

In this way, COVID-19 led to an unexpected deviation in methodology, yet the research continues to emphasise the critical role of stress, and by proxy cortisol, in understanding the impacts of childhood trauma. Despite the pivot, the empirical research findings provide crucial insights to how perceived stress should be considered a key mechanism in pathway from childhood trauma to suicide. Additionally, the systematic review provides robust evidence that stress, as measured through cortisol, may be responsive to interventions, offering a promising avenue for mitigating the effects of trauma on long-term health outcomes.

1.7 Thesis rationale and aims

The literature highlighted in this chapter has identified important gaps in this research area and outlines key pathways that need further research to better understand how childhood trauma can lead to poorer health outcomes, particularly suicidal behaviour.

The thesis aims to elucidate the mechanisms between childhood trauma, stress and suicide and does so by first, conducting a large-scale online survey to investigate the cross-sectional associations between childhood trauma and history of suicidal ideation or attempt, and the relation with self-reported stress, executive control, and impulsivity. A large-scale survey has never included executive function measures, in relation to childhood trauma, suicide and impulsivity, thus provides cross sectional inferences into the possible associations between the risk factors and important foundations for the remainder of the thesis. Second, exploring the longitudinal associations of suicide risk factors, impulsivity, executive function, and suicidal ideation and attempts under naturally occurring stressful conditions – COVID-19. Third, utilising alternative methodology, EMA, to understand the effects of childhood trauma on daily fluctuations in stress-related vulnerability factors whilst observing if known suicide risk or protective factors impacted these associations. Lastly, to understand the most effective psychological interventions for stress, specifically cortisol, through a meta-analytic investigation, to fully elucidate targets for intervention. This thesis utilises a range of methodologies used to complement one another to maximise the usefulness of the

findings of each study to explore the general aim of the thesis to gain a more comprehensive understanding of the mechanisms linking childhood trauma, stress, and suicide. The thesis has four specific aims (and respective sub-aims), each detailed below.

i. To explore the role of executive functioning and impulsivity in explaining the association between childhood trauma and suicidal ideation.
    a. To understand whether the relationships between childhood trauma and impulsivity, executive functioning, and suicidal ideation were moderated by recent stress.

ii. To examine fluctuations in key suicide risk factors (stress, worry, rumination, impulsivity, and executive functioning) during the initial phases of the UK COVID-19 lockdown.
    a. To examine whether suicide vulnerability was associated with greater impulsivity levels and poorer executive functioning during lockdown.
    b. To explore whether COVID-related stress, rumination and worry were associated with greater impulsivity levels and poorer executive functioning, and whether these relationships were moderated by suicide vulnerability.

iii. To investigate the effects of childhood trauma on daily stress-related vulnerability factors (daily stress, hassles, impulsivity, executive functioning, and sleep) over a period of 7 days and to test whether any observed relationships were moderated by protective factors (resilience, social support, and social connectedness) or risk factors (loneliness and suicide history).
    a. To explore the indirect effects of childhood trauma on reasons for living, optimism, daily suicidal ideation, defeat, and entrapment through the daily stress-related vulnerability factors.

iv. To evaluate the effectiveness of psychological interventions to improve cortisol levels in healthy adults in studies that used randomised controlled trial designs.
    a. To investigate the heterogeneity of any observed effects in terms of the type of cortisol measurement (blood, hair, or saliva), control group, (active, inactive, or passive) and intervention type whilst exploring the moderating effects of sample size, study quality and risk of bias.

Chapter 2 - Childhood Trauma & Suicide: associations between impulsivity, executive functioning and stress

2.1 Abstract

Suicide is a leading cause of death worldwide and childhood trauma has been found to be an important risk factor. However, the mechanisms linking trauma to suicide risk remain unclear. The current registered report sought to: i) investigate whether childhood trauma (and its subtypes) were related to suicide risk in adulthood and, ii) explore the potential mechanisms associating childhood trauma with suicide and wellbeing; specifically executive functioning, impulsivity, and stress. A cross-sectional survey of 457 individuals who reported experiencing suicidal ideation in the past 12 months. Childhood trauma and its subtypes were associated with an increased risk of reporting recent suicidal ideation, COVID-related suicide attempts and recent suicide attempts. There were also significant indirect effects of childhood trauma on recent suicidal ideation and wellbeing through executive functioning and impulsivity. These findings show that childhood trauma is associated with suicide risk in adulthood and suggest that poorer executive functioning and higher levels of impulsivity contribute to this increased risk. These results have implications for the development of future interventions to reduce suicide vulnerability.

2.2 Introduction

It is estimated that each year approximately 700,000 individuals worldwide die by suicide and that between 10-20 million more individuals make an attempt to die by suicide (World Health Organisation, 2023). For this reason, there have been continual efforts to elucidate the precise risk factors for suicidal behaviour. As a result, a plethora of risk factors have been identified, with roots in psychological, neurobiological and social domains (Franklin et al., 2017; O'Connor et al., 2016). However, predicting and preventing suicidal behaviour remains low, with predictive ability not improving in the past 50 years (Franklin et al., 2017; Zalsman et al., 2016). In addition, numerous theoretical models of suicidal behaviour have highlighted the complexity of the interaction of risk factors leading to suicidal behaviour (O'Connor & Kirtley, 2018). For example, the Integrated Motivational-Volitional (IMV) model (O'Connor & Kirtley, 2018) recognises the importance of understanding both proximal and distal risk factors, as well as the need to distinguish between suicidal ideation and suicide attempt (Mann et al., 1999; O'Connor & Kirtley, 2018; Oken et al., 2010).

Recent research has shown that childhood trauma is an important risk factor associated with suicidal behaviour. O'Connor et al. (2018) found that approximately 80% of individuals who had attempted suicide in adulthood had reported experience of childhood trauma. Additionally, a meta-analysis by Angelakis et al. (2019) found all types of childhood maltreatment increased the risk for suicide attempts and ideation in adults. These authors suggested that one of the main outstanding challenges was to better understand the mechanisms which underpin the development of suicidal behaviour in individuals exposed to childhood trauma. Previous research and statistical techniques have focussed on identifying risk factors for suicidal behaviour but have ignored the potential relationships between risk factors (De Beurs et al., 2019). Consequently the mechanisms by which childhood trauma may lead to the emergence of suicidal behaviour are unclear and multiple risk factors may interact to produce suicidal behaviour. Moreover, there are a number of theoretical models that suggest childhood trauma has the capacity to modify behaviour patterns that can lead to negative health outcomes (e.g., Lovallo, 2013). Therefore, the central aim of the current study was to investigate the potential mechanisms associating childhood trauma and suicide; namely the role of executive functioning, impulsivity and stress. In addition, this study examined the relationships between childhood trauma and mental wellbeing, as a secondary outcome, alongside the aforementioned potential mechanisms (McElroy & Hevey, 2014).

A study by Lovallo et al. (2013) demonstrated that early adversity (including childhood trauma) was related to a reduced stress response, poorer working memory (a facet of executive function) and increased impulsive behavioural style, all factors linked to suicidal behaviour, in a sample of young adults with and without a family history of alcoholism. The relationship between childhood trauma, executive functioning, impulsivity and negative health outcomes is conceptualised in a model proposed by Lovallo (2013). The model posits that childhood trauma can cause modifications in frontolimbic brain function which may have the capacity to lead directly to reduced stress reactivity and altered cognition, impulsive behaviours and a focus on short term goals. Consistent with Lovallo's theorising, O'Connor et al. (2018) found evidence of blunted hypothalamic pituitary adrenal (HPA) axis activity in response to stress in individuals vulnerable to suicide who also had high levels of childhood trauma, thereby, providing evidence for the proposed reduced stress responsivity pathway. More recently, another study found that childhood trauma was associated with suicide vulnerability in adulthood and that this relationship was, in part, mediated by lower cortisol levels following

awakening (O'Connor et al., 2020a). However, in the broader context, much less work has investigated the precise mechanisms that link childhood trauma to suicide. Therefore researchers have argued that Lovallo's model should be extended to suicidal behaviour to help understand how childhood trauma may lead to suicidal behaviour. For example, is childhood trauma associated with having a more disinhibited lifestyle or impulsive behavioural style in adulthood? What is the relationship between childhood trauma, impaired executive function and suicidal behaviour? McGirr et al. (2010) found that first degree relatives of individuals who had died by suicide had a blunted cortisol reactivity to stress compared to matched controls, suggesting that stress reactivity, as marked by blunted cortisol, could be a trait marker of suicidal behaviour risk. However, to the best of our knowledge, no research has investigated whether, collectively, these variables, impulsivity and executive function, are mechanisms linking childhood trauma and suicidal behaviour. Likewise, whether the effects of specific forms of childhood trauma influence the relationships between risk factors and suicidal behaviour differently is unknown. For example, Angelakis et al. (2019) found that all types of childhood trauma conferred risk of suicidal behaviour but sexual abuse produced the greatest risk followed by physical abuse and emotional abuse. Therefore, the current study aimed to further extend Lovallo's (2013) model and to examine the precise relationships between childhood trauma, its sub-types, impulsivity and executive functioning within the context of suicidal behaviour.

Stress-diathesis models have a long history in the field of suicide research (O'Connor, et al., 2020c). An early example was introduced by Schotte and Clum (1987) in the context of their diathesis-stress-hopelessness model of suicidal behaviour. These authors found evidence that impaired social problem-solving, a specific cognitive vulnerability factor, acted as a diathesis and it was associated with suicide risk in the presence of stress. Another influential diathesis-stress model, developed by Mann and colleagues, was the clinical model of suicidal behaviour (Mann et al., 1999). In this model, risk was postulated to change as a function of the interaction between psychiatric disorder (recent stressor) and a trait-like diathesis. Diatheses are biological, others are cognitive in nature, and others still are personality factors, however, they are all important. Therefore, a secondary aim of the current study was to investigate whether the relationships between childhood trauma and impulsivity/executive functioning, and childhood trauma and suicidal ideation, were moderated by recent stress.

Finally, the coronavirus disease 2019 (COVID-19) pandemic represents the greatest international biopsychosocial emergency the world has faced for a century (O'Connor et al., 2020b). This pandemic has fundamentally changed how societies function, affecting how we work, educate, parent, socialise, shop, communicate and travel. Evidence is emerging to suggest that COVID-19 is increasing the severity of mental health challenges faced by many individuals. A recent national study has shown that the mental health and wellbeing of the UK adult population appears to have been substantially affected in the initial phase of the COVID-19 pandemic, especially for women, young adults, the socially disadvantaged and those with pre-existing mental health problems (R. C. O'Connor et al., 2021). Moreover, a national study also found concerning increased rates of suicidal ideation especially among young adults, as well as changes in mental health and wellbeing outcomes. As a result, given the global reach, virulence and the on-going and longer-term impact of COVID-19, the current study operationalised suicidal behaviour in three ways by assessing: 1) recent suicidal ideation and attempt, 2) lifetime suicidal ideation and attempt and 3) COVID-related suicidal ideation and attempt, as well as including a measure of mental wellbeing.

To summarise, the primary aim of this study was to explore the role of executive functioning and impulsivity in explaining the association between childhood trauma and suicidal ideation (including COVID-related suicide measures). The secondary aims were to investigate whether the relationships between childhood trauma and impulsivity/executive functioning, childhood trauma and suicidal ideation were moderated by recent stress.

The hypotheses were:

H1: Childhood trauma (and sub-types) will be associated with both recent and lifetime suicidal ideation and attempt (including COVID-related suicide measures).

H2: The effects of childhood trauma (and sub-types) on recent suicidal ideation and wellbeing will be mediated by executive functioning and impulsivity (Figure 2; Panel A).

H3: The relationship between childhood trauma (and sub-types) and impulsivity/executive functioning will be moderated by recent stress (Figure 2; Panel B).

H4: The relationship between childhood trauma (and sub-types) and recent suicidal ideation and wellbeing will be moderated by recent stress (Figure 2; Panel C).

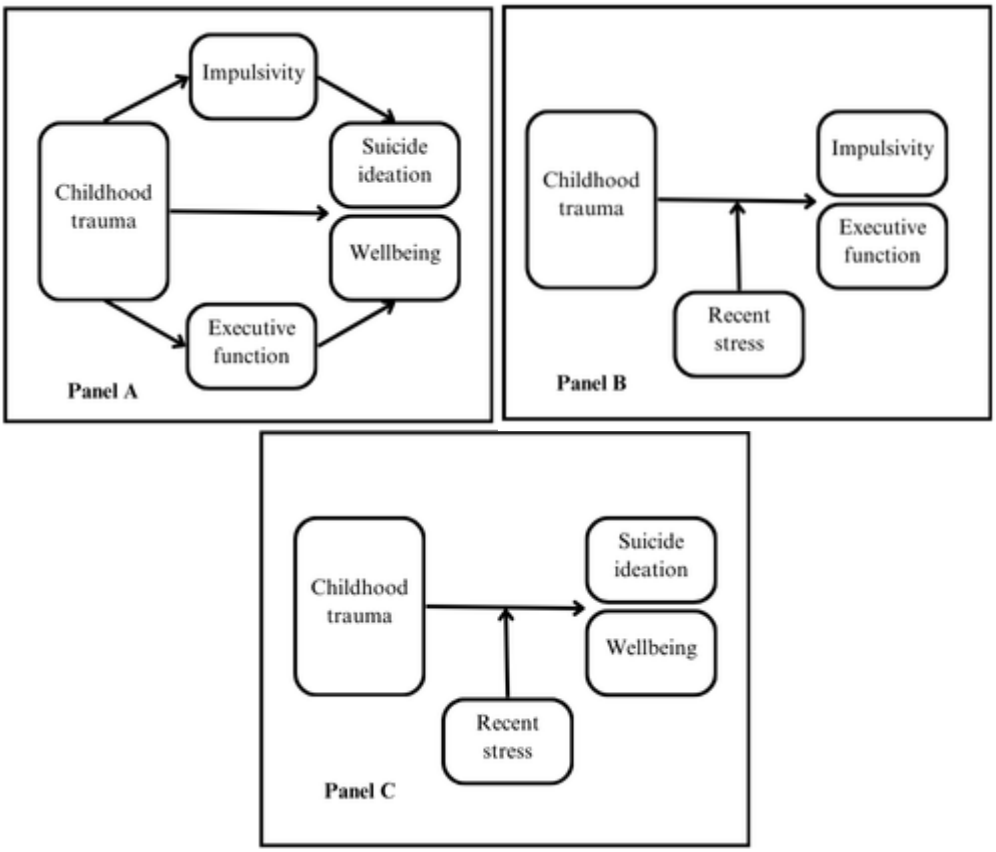


Figure 2. A path diagram to illustrate the proposed study hypotheses

2.3 Methods

2.3.1 Design and Participants

An online cross-sectional survey with individuals at risk of suicide ideation (see Stage 1 registered report: https://doi.org/10.17605/OSF.IO/GXU67). The inclusion criteria for participants were: individuals aged 18 years or older, understanding English language and having reported experiencing suicide ideation in the past 12 months. Understanding suicide risk was the primary concern of the current study, therefore, history of suicide ideation was the main inclusion criterion. However, it is important to note that previous research has established that there are high levels of exposure to childhood trauma in individuals with a recent history of suicide ideation (e.g., 56.7% in recent study by O'Connor et al., 2020). Therefore, adopting this approach ensured we had a good range of scores on the Childhood Trauma Questionnaire, as well as for suicide ideation, thereby allowing us to robustly test our study hypotheses. Participants were recruited through advertisements on social media, Prolific, the University Psychology department

participant pool and university emailing lists. Ethical approval for the study was granted (PSYC-150).

To estimate the sample size required for the current study a priori power analysis was conducted. The general approach adopted for the power analysis was to start with reasonable values of the parameters (e.g., effect size, correlations between predictors, base rates of outcomes) and estimate power as a function of *n*. As the parameters are not known with any degree of certainty, the values have been varied slightly around those reasonable starting points to gauge sensitivity to the key parameters and presented graphically (Hughes et al., 2017; see 7.6.5, see Appendix). For complex analyses the values for power are simulated and all analyses were undertaken in R 4.03 (R Core Team, 2020). All analyses assume *alpha* = .05 unless otherwise stated. In summary, the aim was not to arrive at a single number for each test but arrive at an overall sample size that will have good power (e.g., approximately 80% or more) for a wide range of plausible effect sizes. The desired sample size following the calculations was in the region of $n = 400$. However, to allow for missing data and any technical issues that may lower the power, the study aimed to recruit 500 participants.

2.3.2 Measures

Childhood Trauma

Childhood trauma was measured with the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003). A 28-item self-report inventory assessing history of abuse and neglect in childhood. The CTQ asks people about their experiences growing up as a child and a teenager. Individuals are required to indicate how true each item is, an example being ‘I felt loved’, to be rated from 1 - 'never true’ to 5 - ‘very often true’. The total and sub-scale scores were calculated following the recommendations by Bernstein et al. (2003). Cronbach’s $\alpha = 0.64$.

Impulsivity

Impulsivity was assessed with the Barratt Impulsiveness Scale-11 (BIS-11; Patton et al., 1995). A 30-item self-report questionnaire assessing impulsive behaviour. Individuals rate each item, such as ‘I do things without thinking’, from 1 - ‘never’ to 4 - ‘almost always/always’. Greater the total score, greater impulsive behaviours. Cronbach’s $\alpha = 0.66$.

Executive Function

Executive function measured via the Dysexecutive Questionnaire (DEX; Wilson et al., 1996). A 20-item scale to identify executive difficulties whereby each statement, such as 'I have difficulty thinking ahead or planning for the future', had to be rated from 0 - 'never' to 4 - 'often'. It is part of a larger test battery – the Behavioural Assessment of the Dysexecutive Syndrome (BADS; Wilson et al., 1996) and can be administered in a self-report format, taking around 10 minutes to complete. The higher the score, greater impairment of executive functioning (Shaw et al., 2015). Cronbach's $\alpha = 0.88$.

Stress

Stress was measured with the Perceived Stress Scale (PSS-Brief; Cohen, Kamarck & Mermelstein, 1983). A 4-item self-report measure for perception of stress, individuals are required to indicate how little or often they have felt or thought the items over the past 4 weeks, such as the extent to which they are unable to control the important things in their life, ranking each item from 0 - 'never' to 4 - 'very often'. Items 2 and 3 are reverse scored. Cronbach's $\alpha = 0.72$.

Mental Wellbeing

Participant's mental wellbeing was assessed with the Short Warwick-Edinburgh Mental Wellbeing Scale (SWEMWBS; Stewart-Brown et al., 2009). A 7-item measure to determine wellbeing of individuals over the past 4 weeks (modified from 2 weeks). An example item asks individuals to consider whether 'I've been thinking clearly' from 1 - 'None of the time' to 5 - 'All of the time'. Cronbach's $\alpha = 0.81$.

Depressive symptoms

Participant's depressive symptoms were assessed with the Beck Depression Inventory-II (BDI-II; Beck et al. 1996). A 21-item measure established to determine a range of depressive symptoms over the past 4 weeks (modified from 2 weeks). An example item in the measure is for sadness where individuals choose one of the following responses to indicate the way they have been feeling in the past four weeks: 'I do not feel sad', 'I feel sad much of the time', 'I am sad all the time', 'I am so sad or unhappy that I can't stand it'. It has been shown to yield reliable, internally consistent and valid scores in in adult (Beck et al., 1996) and adolescent populations (Osman et al., 2008). Cronbach's $\alpha = 0.90$.

2.3.2.1 Suicidal behaviour measures

Lifetime suicidal behaviour

Two items were used from the Adult Psychiatric Morbidity Scale (APMS) “Have you ever seriously thought of taking your life, but not actually attempted to do so?” and “Have you ever made an attempt to take your life, by taking an overdose of tablets or in some other way?” Responses to these questions allowed participants to be categorised: 1. Experience of suicidal ideation but not an attempt; 2. Experience of a suicide attempt.

Recent suicidal behaviour

The Scale for Suicidal Ideation (SSI, Beck et al., 1979) was used to determine the presence of suicidal ideation over the previous 4 weeks (modified from the previous 7 days), a 21-item measure to determine individual thoughts towards thinking about suicide. Each of the items has three responses, an example being; ‘I have no wish to die’, ‘I have a weak wish to die’, ‘I have a moderate to strong wish to die.

COVID-related suicidal behaviour

Given the current developments in COVID-19, two questions were added “In the past 12 months, have you had any thoughts of taking your life as a consequence of the COVID-19 pandemic?” and “In the past 12 months, have you attempted to end your life as a consequence of the COVID-19 pandemic?”. For both questions individuals indicated ‘Yes’ or ‘No’, and “if yes, how many times?”.

2.4 Results

2.4.1 Descriptive statistics

A total of 502 individuals were recruited. 457 out of the 502 participants reported suicidal ideation in the last 12 months and a lifetime history of suicidal ideation. 45 participants were excluded due to inconsistent reporting whereby they reported suicidal ideation in the past 12 months *but* no lifetime history of suicidal ideation. The number of individuals reporting lifetime history of suicidal ideation (n = 238) and suicide attempts (n = 219) resulted in similarly distributed groups. In addition, 80% (n = 368) of the sample reported experience of childhood trauma.

Table 10 (see Appendix) shows the means and standard deviations for outcomes for the total sample as well as by suicide history group. All study variables were significantly associated with one another apart from perceived stress, Pearson’s *r* correlation is reported in Table 11 (see Appendix).

2.4.2 Inferential statistics

2.4.2.1 Hypothesis 1: Childhood trauma (and sub-types) will be associated with both recent and lifetime suicidal ideation and suicide attempt.

For the outcome recent suicidal ideation, a hierarchical linear regression was conducted. As outlined in Table 12, see Appendix, childhood trauma was significantly associated with recent suicidal ideation, in both the unadjusted model and in the adjusted model (which controlled for gender, age and depressive symptoms). Each subscale of the CTQ was significantly associated with recent suicidal ideation, in both unadjusted and adjusted models. The model for the emotional neglect subscale appeared to account for the greatest proportion of variance. For recent suicide attempt, an ordinal logistic regression was conducted. In both the adjusted and unadjusted models childhood trauma was associated with a greater likelihood of reporting a recent suicide attempt in the past month (OR = 1.57, 95% CI [1.33, 1.75]), that is a meaningful unit change in CTQ score (14.3 units) was associated with 57% increased likelihood of reporting a recent suicide attempt in the past month.

For the combined outcome variable, lifetime suicidal ideation and attempt, a binary logistic regression was utilised. Table 13 (see Appendix) shows the binary logistic regression results of associations between childhood trauma, and its subtypes, with the outcome lifetime suicidal ideation and attempt. Greater levels of childhood trauma were associated with lifetime history of suicide attempt (OR = 1.70, 95% CI [1.53, 2.01]). This relationship is shown in Figure 3 whereby the predicted probability of lifetime suicide attempts varies according to CTQ score. A binary logistic regression showed that all subtypes of childhood trauma were associated with lifetime history of suicide attempt in both the unadjusted and adjusted models (see Appendix, Table 13). In addition, childhood trauma was not associated with an increased likelihood of reporting thoughts to die by suicide as a consequence of the COVID-19 pandemic, but there was a statistically significant increase in the odds of reporting a suicide attempt as a result of the COVID-19 pandemic (see Figure 3, (OR = 1.38, 95% CI [1.15, 1.75])).

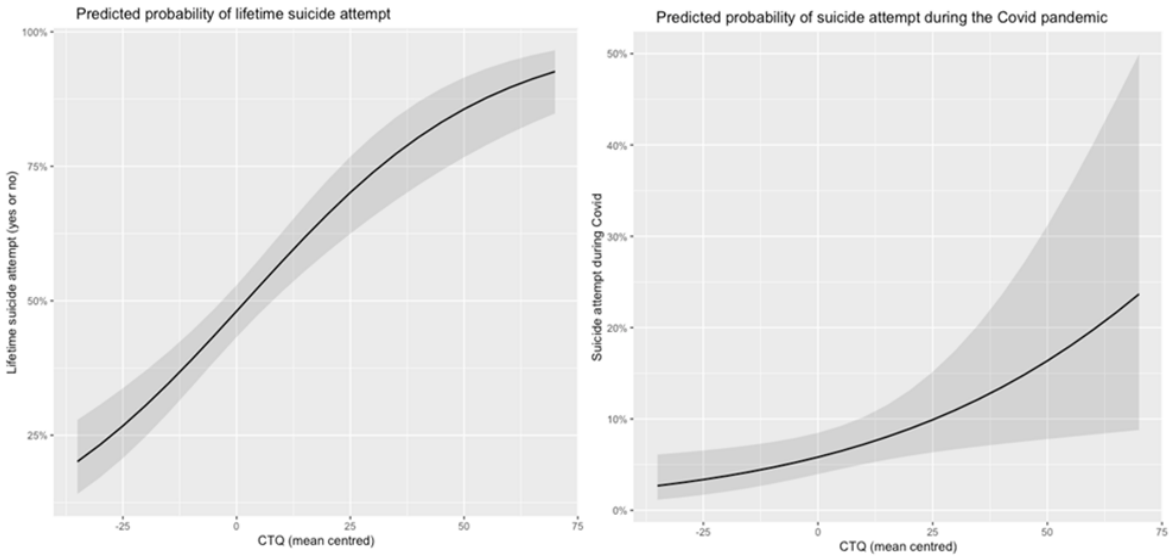


Figure 3. Predicted probability of lifetime suicide attempts (left panel) and COVID suicide attempts (right panel) as a function of CTQ with a 95% CI band

2.4.2.2 Hypothesis 2: The effects of childhood trauma (and sub-types) on suicidal ideation and wellbeing will be mediated by executive functioning and impulsivity

Suicidal ideation

A mediation analysis was run to test the hypothesis using estimates of the indirect effect obtained via percentile bootstrap. The analysis indicated that childhood trauma was significantly associated with executive functioning and executive functioning was significantly associated with suicidal ideation (see Appendix, Table 14, Model 2.1). Moreover, there was a significant indirect effect of childhood trauma on recent suicidal ideation through executive functioning (*b* = 0.02, CI [0.01, 0.04]). See

Figure 4.

For four of the five subscales, there were significant indirect effects on recent suicidal ideation through executive functioning (see Appendix, Table 14, 2.4.1 – 2.4.5); the exception was the sexual abuse subscale.

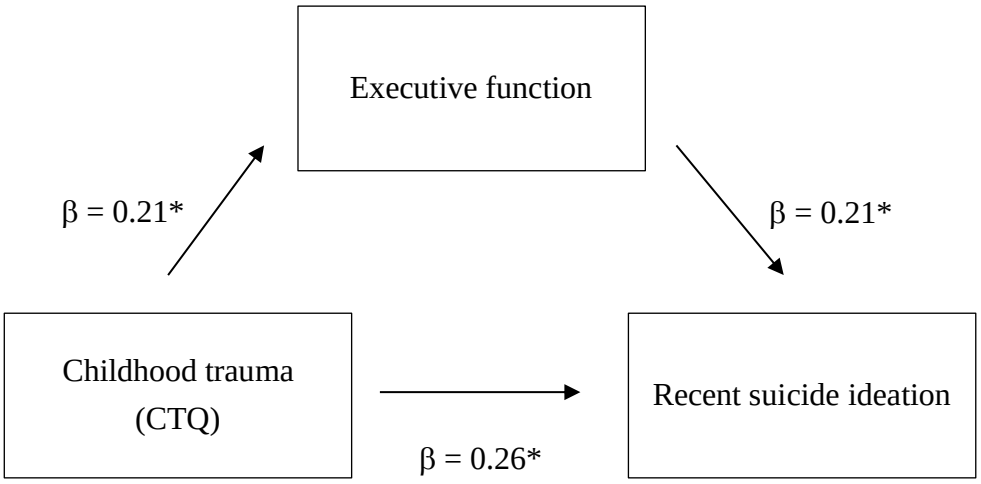


Figure 4. Indirect effects of childhood trauma on recent suicidal ideation through executive functioning

The analysis indicated that childhood trauma was significantly associated with impulsivity and impulsivity was significantly associated with suicidal ideation (see Appendix, Table 14, Model 2.2). There was a significant indirect effect of childhood trauma on recent suicidal ideation through impulsivity (*b* = 0.02, CI [0.01, 0.03]). See Figure 5. For all of the five subscales, there were significant indirect effects on recent suicidal ideation through impulsivity (see Appendix, Table 14, 2.3.1 – 2.3.5).

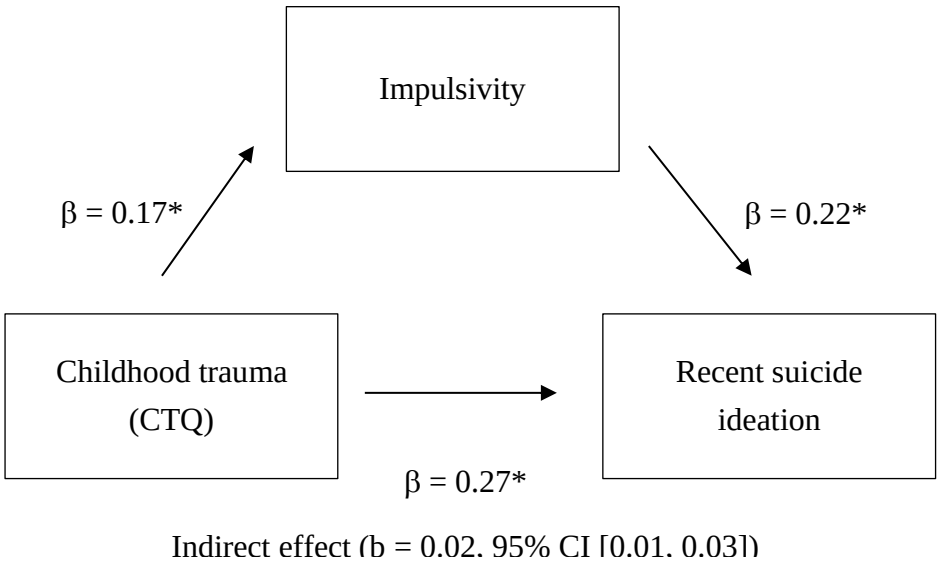
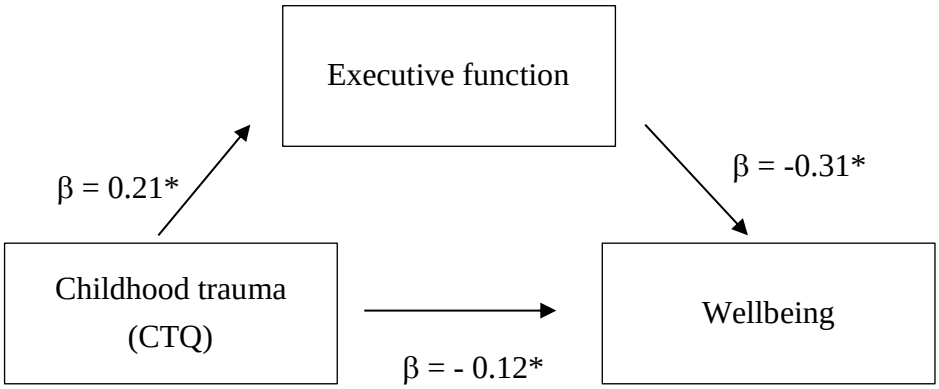


Figure 5. Indirect effects of childhood trauma on recent suicidal ideation through impulsivity

Wellbeing

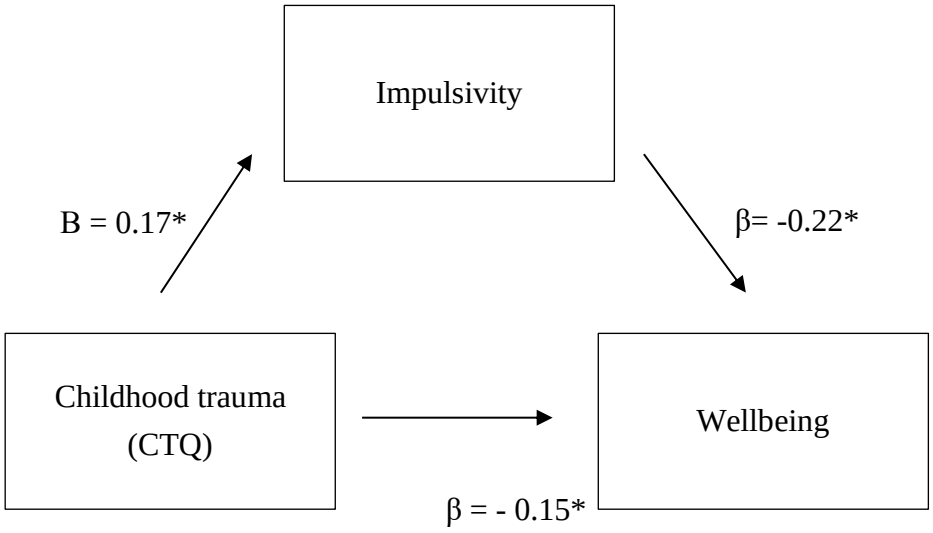
The analysis indicated that childhood trauma was significantly associated with executive functioning and executive functioning was significantly associated with wellbeing. There was a significant indirect effect of childhood trauma on wellbeing through executive functioning (*b* = -0.01, CI [-0.01, -0.00]). See Figure 6. For all subscales, except sexual abuse, there were significant indirect effects on wellbeing through executive functioning (see Appendix, Table 15, 3.4.1 – 3.4.5).



Indirect effect (b = -0.01, 95% CI [-0.01, -0.00])

Figure 6. Indirect effects of childhood trauma on wellbeing through executive functioning

The analysis indicates that childhood trauma was significantly associated with impulsivity and impulsivity was significantly associated with wellbeing. There was a significant indirect effect of childhood trauma on wellbeing through impulsivity (*b* = -0.01, CI [-0.01, -0.00]). See Figure 7. For all of the five subscales, there were significant indirect effects on wellbeing through impulsivity (see Appendix, Table 15, 3.3.1 – 3.3.5).



Indirect effect (b = -0.01, 95% CI [-0.01, -0.00])

Figure 7. Indirect effects of childhood trauma on wellbeing through impulsivity

2.4.2.3 Hypothesis 3: The relationship between childhood trauma (and sub-types) and impulsivity/executive functioning will be moderated by recent stress

Contrary to our predictions, the relationships between childhood trauma, and its subtypes, and executive functioning and impulsivity were not found to be moderated by recent stress (see Appendix, Table 16).

2.4.2.4 Hypothesis 4: The relationship between childhood trauma and recent suicidal ideation and wellbeing will be moderated by recent stress

Similarly, the relationships between childhood trauma and recent suicidal ideation and childhood trauma and wellbeing were not found to be moderated by recent stress (see Appendix, Table 17).

2.5 Discussion

The current study found that experiencing childhood trauma was associated with increased risk of reporting recent suicidal ideation and suicide attempts and these associations held when controlling for gender, age and depressive symptoms. Importantly, the study also found a significant indirect relationship between childhood trauma on recent suicidal ideation and wellbeing through executive functioning; all childhood trauma subtypes apart from sexual abuse also had a significant indirect effect on recent suicidal ideation and wellbeing through executive functioning. A similar indirect relationship was found for childhood trauma, and subtypes, on recent suicidal ideation and wellbeing through impulsivity. Overall, recent stress did not moderate the relationships between childhood trauma and its subtypes and executive functioning, impulsivity, suicidal ideation or wellbeing.

Previous research has established the relationship between childhood trauma and suicide (O'Connor et al., 2018) and argued that poorer executive functioning may be a risk factor that increases the likelihood of suicidal behaviour (McGirr et al., 2010). The current study adds to, and confirms this knowledge, finding an indirect effect of childhood trauma on suicidal ideation through executive functioning. This adds to the existing evidence base that has shown that childhood abuse and neglect are associated with difficulties in executive functioning (Tinajero et al., 2020) and that cumulative exposure to trauma can predict poorer executive functioning; with effects remaining after controlling for

psychopathology symptoms (Letkiewicz, Funkhouser & Shankman, 2021). However, the current study extends our understanding further to reveal a pathway whereby childhood trauma contributes to increased *suicide risk* through poorer executive functioning.

These findings are important as they suggest that experience of childhood trauma may predispose individuals to an increased risk of suicidal ideation in adulthood through disrupted cognitive functioning; both poorer executive functioning, as discussed, and greater impulsivity. Previous research acknowledges that impulsivity is related to both childhood trauma and suicidal behaviour separately (O'Connor et al., 2020c), however the current findings show that the relationship between childhood trauma and suicidal behaviour is also mediated through impulsivity. Previous meta-analytic investigations have found the relationship between impulsivity and suicidal behaviour was significant but small in magnitude, suggesting impulsivity's relationship with suicidal behaviour is likely to be indirect rather than causal (Anestis et al., 2014). Overall, suggesting there are both direct, and indirect pathways, between childhood trauma, and its subtypes, with suicidal ideation and attempt.

In conclusion, the current study provides additional evidence that experiencing childhood trauma is associated with increased risk of reporting recent suicidal ideation and suicide attempts in adulthood, and these associations hold when controlling for gender, age and depressive symptoms. The study also contributes new knowledge to understanding the mechanisms that are associated with increased suicide risk in adulthood in individuals who have experienced childhood trauma. The challenge for researchers is to elucidate how these factors interact across time, and to develop interventions to target these known vulnerability factors affected by childhood trauma to help reduce suicide risk in adulthood.

Chapter 3 - Exploring the relationship between suicide vulnerability, impulsivity and executive functioning during COVID-19: A longitudinal analysis

3.1 Abstract

Public health emergencies increase the presence and severity of multiple suicide risk factors and thus may increase suicide vulnerability. Understanding how suicide risk factors interact throughout the course of a global pandemic can inform how to help the most vulnerable groups in society. The aims of the research were to explore the associations between, and changes in, suicide vulnerability, COVID-related stress, worry, rumination, executive functioning and impulsivity across the first 6 weeks of the UK COVID-19 lockdown (1st April – 17th May, 2020). 418 adults in the UK completed an online survey at three time points during the first lockdown (Time 1 (1st - 5th April), Time 2 (15th – 19th April), Time 3 (13th – 17th May)). Impulsivity and executive functioning remained stable across the first six weeks of UK lockdown. COVID-related stress, worry, and rumination decreased throughout the 6 weeks. Suicide vulnerability was associated with greater impulsivity and poorer executive functioning. Sub-group analysis revealed individuals vulnerable to suicide reported poorer executive function and greater impulsivity than individuals who reported no suicide vulnerability. Individuals vulnerable to suicide appear to have experienced poorer executive functioning and greater impulsivity in the initial phase of the COVID-19 pandemic.

3.2 Introduction

The most recent public health emergency, COVID-19, is estimated to have infected approximately 397 million individuals and resulted in 5.7 million deaths worldwide as of the 9th February 2022 (World Health Organisation, 2020). The first two months of the COVID-19 UK lockdown resulted in a worsened average mental distress score by 8.1% but this was greater for young adults and women who were already at risk for mental health problems, suggesting the pandemic was contributing to growing mental health inequities (Banks & Xu, 2020; Fancourt et al., 2021). There have been the emergence of different mental health trajectories over the course of the pandemic; those who were younger with lower incomes and pre-existing mental health difficulties have experienced higher initial levels of depression and anxiety that continued to increase or stay stable across time despite the cessation of the first UK lockdown (Saunders et al., 2021). The

long-term effects of COVID-19 remain unknown and research has not yet considered the impact of COVID-19 on the mental health trajectories, over time, in individuals who have a history of suicidal behaviour. Despite there being no increase in risk of suicide since the pandemic began (Pirkis et al., 2021); pre-pandemic research indicates there were elevated levels of known risk factors, higher worry and impulsivity, in individuals with a history of suicidal behaviour, compared to individuals with no history (D.B. O'Connor et al., 2021b). However, much less is known about the psychological factors that may be associated with this increased vulnerability to suicide during the current COVID-19 pandemic.

Impulsivity and executive functioning are important constructs implicated in risk of suicidal behaviour as well as other mental health outcomes (Amlung et al., 2019; Han et al., 2016) and are likely to be exacerbated during the stress of a pandemic. Impulsivity is a behavioural construct that reflects impaired self-regulation which can lead to pursuance of actions with little consideration of the consequences. Whereas executive functioning is a broader term referring to a set of cognitive functions that manage thoughts, emotions and actions which in turn help us in goal-directed behaviour. Although the constructs represent a degree of cognitive control and self-regulation, these constructs have been found to be distinct and independent (e.g., Friedman et al., 2020). In a large scale review and meta-analysis of impulsivity and suicidal behaviour, the relationship between impulsivity and suicidal behaviour was found to be significant but small in magnitude (Anestis et al., 2014). In addition, a cross-sectional study in patients with a history of suicide attempt and major depressive disorder by Onat et al. (2018) reported a positive correlation between greater impulsivity scores and poorer executive function. Similarly, poor executive functioning has also been found to be implicated in suicidal behaviour; greater executive functioning has been recently associated with lower levels of suicidal behaviour (Zelazny et al., 2019). The Integrated-Motivational Volitional model (IMV; O'Connor & Kirtley, 2018) acknowledges impulsivity and executive functioning as important volitional moderators translating suicidal ideation to suicidal behaviour. There have also been suggestions that the impulsivity – suicidal behaviour relationship is likely to be indirect, rather than causal, and the presence of stress may exacerbate this relationship (Anestis et al., 2014).

Stress-diathesis models have a long history in the field of suicide research (O'Connor et al., 2020c). An early example was introduced by Schotte and Clum, (1987) in the context of their diathesis-stress-hopelessness model of suicidal behaviour; these authors found

evidence that impaired social problem-solving, a specific cognitive vulnerability factor, acted as a diathesis and it was associated with suicidal behaviour in the presence of stress. More recent research supports this notion by finding that recent life stress can impair executive functioning, specifically working memory (Shields et al., 2016). Thus, it is imperative to understand how these mechanisms interact in relation to suicidal behaviour.

Public health emergencies, such as the outbreak of COVID-19, increase the presence, variety and severity of stressors (Gunnell et al., 2020); a shift to a new normal involving forced working from home or loss of work, isolation or entrapment within difficult family structures, all of which are known risk factors for suicidal behaviour (Moreno et al., 2020). However, there is relatively little known about how changes in stress-related variables might interact with other risk factors such as impulsivity and executive functioning during periods of sustained stress and change. For example, it is likely that increased levels of COVID-related stress, worry and rumination may be associated with more impulsive behaviours or may interfere with aspects of executive functioning. A recent paper found that higher-levels of COVID-related stress, worry and rumination were also associated with poorer mental health (Prudenzi et al., 2023).

To summarise, this longitudinal study aimed to investigate changes in key suicide risk factors: stress, worry, rumination, impulsivity, executive functioning during the early stages of the UK lockdown and to determine whether the effects of COVID-related stress, worry and rumination on impulsivity and executive dysfunction were greater in individuals with a history of suicidal behaviour compared to those with no history of suicidal behaviour. In this study, suicidal behaviour is considered to be individuals who have experienced *either* lifetime suicidal ideation or suicide attempts. The specific aims were:

1. To explore changes in COVID-related stress, rumination and worry, impulsivity and executive functioning during lockdown.
2. To examine whether history of suicidal behaviour was associated with greater impulsivity levels and poorer executive functioning during lockdown.
3. To explore whether COVID-related stress, rumination and worry were associated with greater impulsivity levels and poorer executive functioning, and whether these relationships were moderated by history of suicidal behaviour.

3.3 Methods

3.3.1 Design and Participants

557 participants recruited through social media and Prolific (aged 18 and over and fluent in English) enrolled in the study at time 1 (T1; 1st April 2020), 468 participants (84%) completed at time 2 (T2; 14th April 2020) and 439 participants (78.8%) completed at time 3 (T3; 28th April 2020). Following the final survey (T3), 21 participants were excluded as they did not disclose their history of suicidal behaviour so could not be included in the analysis. The current analysis is based on 418 participants who completed all three time points and disclosed suicide history. Participants were aged between 18 and 75 years of age (M = 35.37, SD = 13.74) and were predominantly Caucasian (See Table 1 for summary of demographics). Participants reported their history of suicidal behaviour as follows; 133 participants reported history of attempting to take their own life or thoughts of suicide throughout their lifetime and 285 participants reported no thoughts or attempts of suicide. The study was approved by the University of Leeds ethics committee (ref: PSYC-23) and the main analyses were preregistered on AsPredicted (reference number: 41985, see Appendix 7.7.1 for further information).

Table 1. Baseline characteristics for participants (n = 418)

Characteristic	Total sample *(n = 418)*	History of suicidal behaviour *(n = 133)*	No history of suicidal behaviour *(n = 285)*
Age (SD)	35.37 (13.74)	33.06 (12.44)	36.45 (14.19)
Sex (% female)	331 (79.2%)	109 (82%)	222 (77.9%)
Ethnic background (%)			
White	386 (92.3%)	125 (94%)	261 (91.6%)
Mixed/Multiple ethnicities	10 (2.4%)	4 (3%)	6 (2.1%)
Asian	17 (4.1%)	3 (2.3%)	14 (4.9%)
Black/African/Caribbean	4 (1%)	1 (0.8%)	3 (1.1%)

Other ethnic group	1 (0.2%)	0 (0%)	1 (0.4%)

3.3.2 Background measures

History of suicidal behaviour

Two questions were taken from the Adult Psychiatric Morbidity Survey (APMS, McManus et al., 2007) to determine both lifetime suicidal ideation: "Have you ever seriously thought of taking your life, but not actually attempted to do so?" and lifetime suicide attempts "Have you ever made an attempt to take your life, by taking an overdose of tablets or in some other way?" Response options were "yes", "no" or "would rather not say". The APMS measure has been used frequently to determine lifetime history of suicidal behaviour (e.g., O'Connor & Kirtley, 2018; Wetherall et al., 2018). Participants who answer yes to either question regarding suicidal ideation or suicide attempts were classified as having a history of suicidal behaviour.

3.3.3 Two weekly measures

COVID-related measures

Single item measures of COVID-related stress, worry and rumination were developed for the purpose of the current study based on previous research which has shown single item measures of stress, rumination and worry to be reliable and valid (Clancy et al., 2020; O'Connor & Ferguson, 2016). These measures were also found to be significant predictors of poorer psychological health in previous research (Prudenzi et al., 2023).

COVID-related stress

Participants were asked "In the past two weeks, to what extent has life become more stressful, difficult or upsetting because of the COVID-19 outbreak?" on a scale of 1 – "not at all stressful, upsetting or bothersome" to 7 – "extremely stressful, upsetting or bothersome".

COVID-related worry

Preceding the question, participants were provided with a definition of COVID-related worry: "Negative, repetitive thoughts about future events which have the potential to be stressful or upsetting. These worrisome thoughts are usually distressing, can be difficult to control and can lead to a spiral of different worries". Participants were then asked "Over the last two weeks, how often did you worry or focus on COVID-19-related things

that may occur or happen in the future?" Participants were required to indicate their answer on a Likert scale from 1 – "never" to 7 – "very often".

COVID-related rumination

A definition of rumination was provided to participants: "Negative, repetitive thoughts about upsetting emotions or events which have happened in the past (including today). These ruminative thoughts are usually distressing, can be difficult to control and can lead to a spiral of different ruminations." Participants were asked "Over the last two weeks how often did you ruminate over COVID-19-related things that have happened to you, or upset you in the past?" Participants rated their response on a Likert scale from 1 – "never" to 7 – "very often".

Executive functioning

Webexec (Buchanan et al., 2010): a 6-item measure of executive functioning whereby participants rate the extent of problems experienced from 1 – "no problems" to 4 – "many problems". The Webexec was modified for this study to reflect on the problems experienced in the past 2 weeks. Example items for this measure asked participants "do you have difficulty carrying out more than one task at a time?" and "do you find it difficult to keep your attention on a particular task?" The greater the summed score indicated poorer executive function. The Webexec had good internal consistency in the study (6 items; T1: α = .87; T2: α = .90; T3: α = .90).

Impulsivity

Momentary impulsivity scale (MIS; Tomko et al., 2014): a 4-item measure asking participants to "Please indicate how much each statement below describes your experience over the past 2 weeks?" This statement was adapted from the original MIS question of "Describe how much each statement described their experience since the last completed prompt". Each statement was rated from 1 – "not at all or very slightly" to 5 – "extremely". Example statements included "I have felt impatient" and "I said things without thinking". The MIS had good internal consistency (4 items; T1: α = .75; T2: α = .76; T3: α = .78).

3.3.4 Procedure

All participants were screened when beginning the questionnaire to ensure participants were aged 18 years or older and they were fluent in English language and provided written online consent. Once participants completed online consent, they completed the online

baseline questionnaire. Participants were then contacted by email two and four weeks later to complete a brief follow-up questionnaire.

3.3.5 Data Analysis

Preliminary analysis to explore data distributions and missingness was conducted. Analyses were conducted using complete case analyses whereby only participants completing all three time points and disclosed their suicide history were eligible for inclusion in the analysis. To examine the pattern of missingness and determine if data were missing completely at random, Little's missing completely at random (MCAR) test was performed; there was missing data in Level 2 that was not MCAR ($X^2(1054) = 1177.87$, $p = .004$). Following the approach adopted by O'Connor et al. (2022), multiple imputations were carried out and 10 imputed datasets were generated; analyses were then conducted on a randomly selected imputed dataset and the results of the multiply imputed dataset and single mean imputation were compared. The results of both analyses were substantively the same, therefore, the findings from the multiple imputed dataset are reported. There was no missing data in Level 1 following removal of non-completers and individuals who did not disclose their suicide history.

The data was analysed using univariate and multivariate analysis of variance (ANOVA and MANOVA) in SPSS v.22 and hierarchical linear modelling in HLM8 (Raudenbush & Congdon, 2021). The analyses were conducted on the entire sample (418 participants). The data was considered to have a two-level hierarchical structure, with Level 1 capturing the within-person relations between the predictors (COVID-related stress, worry and rumination) and the dependent variables (impulsivity and executive function) with level 2 representing the between-person variability (history of suicidal behaviour, age, gender). The level 1 variables were group mean centred and modelled as random as it was assumed that each of the within person variables would be variable. The level 2 dichotomous variables (history of suicidal behaviour, gender) was uncentered and the level 2 continuous variable (age) were grand mean centred. Note that in order to account for multiple testing, the study adopted a more conservative *p* value in all the analyses ($p < .017$; $p < .05/3$) reflecting the number of tests conducted in each set of analyses (i.e., the three main study variables). Lastly, the recommendations set out by (Simmons et al., 2011) regarding transparency were followed so that the unadjusted (no covariates) and adjusted (covariates) analyses are presented. The general form of the HLM model is expressed by the following equation:

Level-1 Model

$$\text{State Impulsivity/Executive function} = \beta_{0j} + \beta_{1j}*(\text{COVID-related stress}) + r_{ij}$$

Level-2 Model

$$\beta_{0j} = \gamma_{00} + \gamma_{01}*(\text{age}) + \gamma_{02}*(\text{gender}) + \gamma_{03}*(\text{history of suicidal behaviour}) + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}*(\text{history of suicidal behaviour}) + u_{1j}$$

3.4 Results

Descriptive statistics for the main study variables are presented in Table 2. For the entire sample the mean impulsivity scores did not alter noticeably across the three time points, yet at each time point individuals with a history of suicidal behaviour demonstrated higher impulsivity scores compared to individuals with no history. A similar pattern emerged for the entire sample when looking at executive function across the three time points, there was no substantial variation in the scores across the lockdown period. Yet, individuals with a history of suicidal behaviour exhibited poorer executive functioning (shown by a higher score) at all-time points compared to individuals with no history. COVID-related stress, worry and rumination each showed decreases across time for the entire sample, and the same pattern emerged for individuals with history of suicidal behaviour reporting greater COVID-related stress, worry and rumination compared to individuals with no history of suicidal behaviour.

Table 2. Descriptive statistics for main study variables at each time point (n = 418)

	Time point								
	1 (1st April 2020)			**2 (14th April 2020)**			**3 (28th April 2020)**		
	History of suicidal behaviour *M (SD)*	*No history of suicidal behaviour* *M (SD)*	*Total* *M (SD)*	*History of suicidal behaviour* *M (SD)*	*No history of suicidal behaviour* *M (SD)*	*Total* *M (SD)*	*History of suicidal behaviour* *M (SD)*	*No history of suicidal behaviour* *M (SD)*	*Total* *M (SD)*
Within-person variables									
COVID-related stress	5.01 (1.41)	4.76 (1.20)	4.84 (1.28)	4.53 (1.41)	4.34 (1.44)	4.40 (1.43)	4.35 (1.57)	4.04 (1.51)	4.14 (1.53)
COVID-related Worry	5.23 (1.58)	5.07 (1.50)	5.12 (1.52)	4.75 (1.61)	4.48 (1.62)	4.56 (1.62)	4.53 (1.66)	4.16 (1.53)	4.28 (1.58)
Rumination	3.95 (1.83)	3.44 (1.65)	3.60 (1.72)	3.83 (1.66)	3.34 (1.66)	3.49 (1.68)	3.76 (1.67)	3.27 (1.67)	3.42 (1.69)

Executive function*	14.92 (4.23)	11.93 (4.07)	12.88 (4.34)	15.50 (4.33)	12.62 (4.47)	13.54 (4.62)	15.26 (4.16)	12.34 (4.44)	13.27 (4.56)
Impulsivity	9.68 (3.30)	8.69 (3.38)	9.00 (3.38)	9.78 (3.41)	8.57 (3.33)	8.95 (3.40)	9.83 (3.69)	8.68 (3.43)	9.05 (3.55)

Note. * = high mean score indicates poorer executive function.

3.4.1 Completer vs non-completer analysis

A series of ANOVAs were performed to determine if there were any differences in COVID-related stress, worry, rumination, executive functioning and impulsivity between individuals who completed (completers (n = 439) all surveys compared to non-completers (n = 118). Specifically, univariate analyses showed that when comparing completers to non-completers, completers were significantly higher in COVID-related stress (F (1, 555) = 13.22; $p < .001$; partial $\eta^2 = .02$), COVID-related worry (F (1, 555) = 10.36; $p = .001$; partial $\eta^2 = .02$) and COVID-related rumination (F (1, 555) = 6.91; $p = .009$; partial $\eta^2 = .01$) compared to non-completers. There were no differences in executive functioning and impulsivity between completers and non-completers that were statistically significant.

In addition to the above ANOVAs, the study explored whether there were any differences in demographic variables amongst completers and non-completers. An independent samples t-test suggested no significant differences in the age of participants depending on whether they completed (M = 35.05, SD = 13.65) or did not complete all the surveys (M = 32.98, SD = 13.29; $t(554) = -1.46$, $p = .144$). Additionally, a chi-square test revealed no significant relationship between completion status and gender ($X^2(2) = 3.92$, $p = .141$).

3.4.2 Changes in COVID-related stress, rumination and worry, impulsivity and executive functioning during UK lockdown

A series of ANOVAs were conducted to test the first hypothesis, exploring the changes in executive function, impulsivity, COVID-related stress, worry and rumination across lockdown. Univariate analyses showed effects of time on both COVID-related stress (F (2, 1251) = 26.30; $p < .001$; partial $\eta^2 = 0.04$) and worry (F (2, 1251) = 30.67; $p < .001$; partial $\eta^2 = 0.05$). Tukey post-hoc comparisons found COVID-related stress was significantly lower at T2 compared to T1 and T3 compared to T1. Although decreases were seen between T2 and T3, when applying Bonferroni corrections to interpret the p-values the decrease was no longer significant. COVID-related worry was significantly lower at T2 compared to T1 and T3 compared to T1, again decreases in COVID-related worry occurred between T2 and T3 but were not significant when adjusting the p-value accounting for multiple testing ($p = .017$). There were no significant changes across lockdown for COVID-rumination, executive function or impulsivity.

3.4.3 Main effects of history of suicidal behaviour on impulsivity and executive function

The HLM analyses found that there was a main effect of history of suicidal behaviour on impulsivity (unadjusted β = 0.29, p <.001; adjusted β = 0.25, p < .001) and executive functioning (unadjusted β = 2.89, p <.001, adjusted β = 2.62, p < .001) such that individuals with a history of suicide reported higher levels of impulsivity and poorer executive function (see Appendix, Tables 3 and 4) compared to individuals with no history of suicide.

3.4.4 Effects of COVID-related stress, rumination and worry on impulsivity and executive function

The HLM analyses also found that the level 1 slope between COVID-related stress and impulsivity was significant in both the unadjusted (β = 0.09, p <.001) and adjusted models (β = 0.09, p <.001) indicating that recent COVID-related stress was associated with greater impulsivity across the initial stages of the UK lockdown. The level 1 slope between COVID-related stress and executive function was significant in both unadjusted (β = 0.34, p <.001) and adjusted (β = 0.34, p <.001) indicating greater COVID-related stress was associated with poorer executive function. After adjusting the p-value criterion using Bonferroni correction, the level 1 slope between COVID-related worry and impulsivity was significant in the unadjusted (β = 0.05, p = .013) and adjusted models (β = 0.05, p = .013). The level 1 slope between COVID-related worry and executive function was significant in both the unadjusted (β = 0.30, p = .002) and adjusted models (β = 0.30, p =.001) indicating that higher levels of COVID-related worry were associated with poorer executive function. The level 1 slope between COVID-related rumination and impulsivity was significant in both the unadjusted (β = 0.06, p = .003) and adjusted models (β = 0.06, p =.004). The level 1 slope between COVID-related rumination and executive function was also significant in both the unadjusted (β = 0.24, p =.008) and adjusted model (β = 0.24, p = .009).

3.4.5 Moderating effects of history of suicidal behaviour on the COVID-related stress, worry and rumination - impulsivity and executive function relationships

History of suicidal behaviour did not moderate the level 1 COVID-related stress – impulsivity slope in either model (unadjusted (β = -0.08 p = .057); adjusted (β = -0.07, p = .059)). History of suicidal behaviour did not moderate the level 1 slope between COVID-related rumination and impulsivity (unadjusted (β = -0.02 p = .622); adjusted (β

= -0.02, p = .625)). The level 1 slope between COVID-related worry and impulsivity was not moderated by history of suicidal behaviour in either model (unadjusted (β = 0.04, p = .340); adjusted (β = 0.04, p = .341)).

Similarly, the level 1 slope between COVID-related stress and executive function was not moderated by history of suicidal behaviour in either the unadjusted (β = -0.03, p = .864) or adjusted model (β = -0.03, p = .884). The level 1 rumination – executive function slope was not moderated by history of suicidal behaviour (unadjusted (β = 0.09, p = .590); adjusted (β = 0.09, p = .586)). Lastly, history of suicidal behaviour did not moderate the level 1 worry – executive function relationship in either model (unadjusted (β = 0.107, p = .547); adjusted (β = 0.107, p = .545)).

3.5 Discussion

This is one of the first longitudinal studies, to our knowledge, that provides evidence for how risk factors for suicide interact throughout the course of a lockdown in a global pandemic. There were four key findings. First, over the course of the 6-week period measured in the first UK lockdown, COVID-related stress and COVID-related worry decreased, whereas rumination, impulsivity and executive functioning remained stable. Second, history of suicidal behaviour was associated with greater impulsivity and poorer executive function during the lockdown period suggesting history of suicidal behaviour may put individuals at a greater risk of negative consequences from the pandemic. Third, in weeks when people experienced greater COVID-related stress, more impulsive behaviours and poorer executive functioning were also reported. However, history of suicidal behaviour did not moderate these associations. Fourth, in weeks when people experienced greater COVID-related worry and rumination, more impulsive behaviours and poorer executive functioning were also reported. Again, history of suicidal behaviour did not moderate these associations.

COVID-related stress and worry decreased over the lockdown period. This is consistent with Fancourt et al. (2020) whereby stress relating to COVID-19 (catching and becoming ill) continued to decrease across the first 5 weeks of lockdown and worries about money, employment and access to food decreased. Whereas COVID-related rumination, impulsivity and executive functioning remained stable across the lockdown period. Combined, the current study, alongside Fancourt et al. (2020), suggests that many individuals adjusted quickly to the lockdown in the UK.

History of suicidal behaviour was found to be associated with poorer executive functioning. This finding aligns with studies whereby self-reported executive functioning was poorer in a sample of individuals reporting suicide attempts, depression and anxiety, compared to a sample with no attempt controls (Loyo et al., 2013). It is important to note the difficultly to directly compare results of the current study to other literature on executive functioning as few studies have measured self-reported executive functioning in relation to suicide, instead opting for performance based measures, especially when there is reported to be minimal overlap with performance and self-reported measures of executive functioning (Toplak et al., 2013). However, one study to our knowledge reported that individuals who had made a recent suicide attempt reported significantly worse self-reported executive functioning compared to individuals who had recently experienced suicidal ideation, suggesting executive functioning (self-reported) may represent an important risk factor for recent suicide attempts (Saffer & Klonsky, 2017). The authors reported no differences in executive dysfunction when comparing lifetime history of suicidal ideation compared to lifetime history of suicide attempts but executive function differences were present when comparing individuals with recent suicidal ideation compared to individuals with recent suicide attempts. Future research should consider the recency of suicidal behaviour in relation to the stress – executive relationship.

The present study found history of suicidal behaviour to be associated with higher impulsivity. This is consistent with previous research whereby aggregated self-reported momentary impulsivity was related to baseline suicide risk in individuals with bipolar disorder (Depp et al., 2016), although this research did not consider the relationship between individual momentary ratings of impulsivity and suicide risk, unlike the present study. Moreover, greater weekly COVID-related stress was associated with more impulsive behaviours. This is in keeping with previous work that found self-reported impulsivity was higher during periods of stress compared to no stress, in a sample of individuals with borderline personality disorder as well as healthy controls (Cackowski et al., 2014). A potential explanation for this is that high levels of impulsive traits may increase vulnerability to problematic coping behaviours during COVID-19, and their influence may be exacerbated during times of stress (Albertella et al., 2021).

Future psychological interventions for individuals with a history of suicide ought to consider targeting changes in executive functioning and impulsivity. Previous research posits that a variety of interventions can improve executive functioning in children aged

4 – 12 years old (Diamond & Lee, 2011). Executive functioning intervention from a younger age may mitigate transition to suicide risk, but whether better executive functioning could become a protective factor against suicidal behaviour is yet to be determined. Future research is needed to explore whether global or individual constructs of executive functioning relate to suicidal behaviour so intervention can target the most relevant constructs and thus be most effective. This is supported by the previous research which states if an intervention is not successful in addressing the executive functioning problems in adolescence, it is possible that over time, some adolescents who experience attentional impairments go on to engage in more lethal suicide attempts (Sommerfeldt et al., 2016).

There are inevitable shortcomings to the current study that ought to be acknowledged. First, the study had no true baseline, the first assessment was taken 1 week into the first UK lockdown, reflecting the window of time between 1-week pre-lockdown and 1-week into the first lockdown, which may have already increased individual stress and impacted wellbeing prior to the first measures being administered. Thus, attributing any changes in measures observed to lockdown is not appropriate. Secondly, the measure of state executive functioning is not a gold standard performance-based measure. There are reported differences between performance-based measures compared to self-report measures of executive function (Keen et al., 2022). Nevertheless, the self-report measure used in the current study has been shown to be reliable and valid (Buchanan et al., 2010).

To conclude, this is one of the first longitudinal studies that provides evidence for how risk factors for suicide interact throughout the early stages of a global pandemic lockdown. COVID-related factors decreased in the initial phase of the pandemic. Individuals with a history of suicidal behaviour appear to have experienced poorer executive functioning and greater impulsivity in the initial phase of the COVID-19 pandemic. Psychological intervention for individuals with a history of suicide ought to consider targeting changes in execute functioning and impulsivity.

Chapter 4 - The effects of childhood trauma on stress-related vulnerability factors and indicators of suicide risk: An ecological momentary assessment study

4.1 Abstract

Childhood trauma is experienced by approximately one third of young people in the United Kingdom (UK) and has been shown to confer an increased risk for mental health difficulties in adulthood. Understanding the associations between these factors before negative health outcomes manifest in adulthood is imperative to help inform the development of interventions. The aims of this study were two-fold; first, to investigate the effects of childhood trauma on daily stress-related vulnerability factors over a period of 7 days and to test whether any observed relationships were moderated by protective or risk factors. Second, to explore the indirect effects of childhood trauma on reasons for living, optimism, daily suicidal ideation, defeat and entrapment through the daily stress-related vulnerability factors. 212 participants were recruited to an ecological momentary assessment (EMA) study to complete three diaries per day for a 7-day period. Participants completed daily measures of stress, hassles, executive functioning, impulsivity, sleep quality (stress-related vulnerability factors) as well as measures of reasons for living, optimism, and daily thoughts of suicide, defeat and entrapment. The Childhood Trauma Questionnaire (CTQ) was also completed at baseline. Analyses found that childhood trauma was significantly associated with higher scores on the daily stress-related vulnerability factors and positively related to each of the daily indicators of suicide risk. The study also uncovered key pathways whereby trauma had indirect effects on reasons for living, optimism, daily thoughts of suicide, defeat and entrapment through executive functioning, impulsivity, sleep quality and stress. The measures of executive function and sleep were self-reported and future research ought to replicate the current findings using more objective methods. The findings from this study highlight the complexity of childhood trauma and its damaging impacts on stress-related vulnerability factors and poorer mental health outcomes. Greater understanding of pathways by which trauma may impact later health outcomes is essential for development of interventions.

4.2 Introduction

Childhood trauma is experienced by approximately one third of young people in the UK and two thirds of adults in the United States (US) and is associated with poorer health

outcomes with detrimental impacts on physical and mental health (Lewis et al., 2019; Swedo, 2023). Trauma exposed young people have been found to be twice as likely to develop a mental health condition compared to non-trauma exposed young people and they have also an increased risk of suicide (Lewis et al., 2019; Marshall et al., 2013). Additionally, experiences of trauma are associated with a variety of pre-psychopathological outcomes: dysregulated hypothalamic pituitary adrenal (HPA) axis functioning (O'Connor et al., 2018, 2020a), retrospective assessments of greater perceived stress (Gouin et al., 2012), poor executive functioning (Gould et al., 2012), impulsive behaviours (Lovallo, 2013) and sleep disruption (Tinajero et al., 2020). Each of these pre-psychopathological factors has been shown to confer an increased risk for mental health difficulties in adulthood such as depression, self-harm and suicide; questioning whether a pathway exists whereby childhood trauma confers an increased risk for future mental and physical health outcomes by influencing key pre-psychopathological, or stress-related vulnerability factors, before negative health outcomes manifest.

Effects of childhood trauma on stress-related vulnerability factors

The experience of childhood trauma is associated with a plethora of poor health outcomes, specifically impacting upon daily functioning. There is growing evidence showing that fluctuations in within-person daily stressors are important to understanding stress-outcome processes (Almeida et al., 2005; Gartland et al., 2014; Smyth et al., 2018). Previous research has found childhood abuse to be associated with greater daily hassles (Tinajero et al., 2020) and adverse childhood experiences to significantly predict self-reported stress (Kalmakis et al., 2020). Furthermore, stress, including early life adversity, acute stress, chronic stress and daily hassles can increase the likelihood an individual will experience suicidal ideation and later suicidal behaviour (Howarth et al., 2020; Liu & Miller, 2014).

Childhood trauma has been found to impact on a diverse array of stress-related vulnerability factors such as sleep, impulsivity, executive functioning, defeat and entrapment. Indeed, a recent study found that a greater number of traumatic events during childhood was associated with poorer sleep health as assessed by both actigraphy and sleep diaries in adulthood (Hamilton et al., 2018) and childhood abuse was associated with poorer sleep quality (Tinajero et al., 2020). In addition, impulsivity has also been shown to play a key role in numerous adverse outcomes such as drug addiction (Argyriou

et al., 2018) and suicide (Anestis et al., 2014). Moreover, poorer executive function performance has also been found in adults who have been exposed to childhood trauma and in one study, when considering specific facets of trauma, childhood abuse and neglect were found to be associated with poorer reported and behavioural executive function difficulties (Gould et al., 2012; Tinajero et al., 2020).

As outlined above, childhood trauma is an established risk factor for suicide. A leading model of suicidal behaviour, the Integrated Motivational-Volitional Model (IMV), has identified psychological vulnerability factors such as defeat, entrapment, stress and impulsivity contribute to an increased risk of suicidal ideation and behaviour (O'Connor & Kirtley, 2018). The first part of the model, outlining the pre-motivational and motivational phases, summarises the complex interplay of the aforementioned stress related vulnerability factors, in relation to childhood trauma (as well as other factors), *prior* to the development of suicidal ideation. Specifically, the model postulates that feelings of defeat can trigger feelings of entrapment which in turn predicts suicidal ideation as an escape from unbearable psychological distress. However, there is a lack of evidence linking childhood trauma to daily feelings of defeat and entrapment in adulthood, therefore a key aim of the current study was also to investigate the extent to which childhood trauma is associated with daily feelings of defeat and entrapment.

Furthermore, in the IMV model, there are stage-specific motivational moderators that facilitate or impede the emergence of suicidal ideation. Motivational moderators include resilience, social support, social connectedness and loneliness, as well as previous suicidal history, all influencing the transition from entrapment to suicidal ideation. Recent research has reported lower levels of social support, connectedness and resilience in individuals vulnerable to suicide compared to individuals with no history of suicidal ideation or behaviour, suggesting that these factors are protective (D. B. O'Connor et al., 2021b). Therefore, another aim of the current research was to understand whether the associations between childhood trauma and defeat, entrapment and the other aforementioned daily stress-related vulnerability factors were moderated by known protective-factors (resilience, social support, social connectedness) and/or risk-factors (loneliness and suicide history).

Do stress-related vulnerability factors mediate the childhood trauma – suicide risk relationship?

The final aim of the current study was to investigate the factors that may mediate the effects of childhood trauma on suicide risk. In particular, the study was interested in exploring whether childhood trauma had indirect effects on key indicators of suicide risk (i.e., reasons for living, optimism, defeat, entrapment and suicidal ideation) through influencing the aforementioned stress-related vulnerability factors (i.e., daily stress, executive functioning, impulsivity and sleep). Determining the mechanisms by which childhood trauma is associated with suicide risk factors, *prior* to the development of mental and physical health problems may help further elucidate the pathways that increase future vulnerability and may uncover key targets for intervention.

Finally, it is worth noting that relatively few studies have explored the associations between childhood trauma, stress-related vulnerability factors and suicide risk indicators in naturalistic settings using an EMA methodology. EMA allows the assessment of a phenomenon even if dynamic changes are unobservable when measuring at fixed time points. Moreover, an important avenue for research in this area is understanding the state-dependent dynamics that may help explain changes in daily functioning, such as impulsive behaviours (Tomko et al., 2014) and feelings of stress (Van Nierop et al., 2018). Previous research has revealed that childhood trauma is associated with higher perceived daily hassles (Berhe et al., 2023) and that even in a sample of clinically healthy, asymptomatic individuals with milder forms of childhood trauma, changes in real-life wellbeing is observable as adults (Berhe et al., 2023). In a typical single point measurement study, changes in stress, wellbeing and mental health may be missed, whereas EMA methodology offers an ability to capture changes in risk-associated changes in psychological processes and behaviours within naturalistic settings. A notable recent study by Tinajero et al. (2020) provided useful insights into the links between childhood trauma and pre-psychopathological factors using an EMA approach. This study found childhood abuse to be associated with pre-psychopathological factors such as emotion regulation difficulties, daily hassles and poorer executive function. However, their sample size was relatively small (N = 79), participants were only sampled over 3 days and the study did not explore the possible indirect effects of childhood trauma on known risk factors for suicide through stress-related vulnerability variables. Therefore, in the current study, it sought to recruit a larger sample, to extend the study period to 7 days and to explore whether the effects of childhood trauma on suicide risk factors were mediated through daily stress-related vulnerability variables.

Taken together, the primary aims of the current study were two-fold. First, to investigate the effects of childhood trauma on daily stress-related vulnerability factors (daily stress, hassles, impulsivity, executive functioning and sleep) over a period of 7 days and to test whether any observed relationships were moderated by protective factors (resilience, social support, social connectedness) or risk factors (loneliness and suicide history). The secondary aims were to test the indirect effects of childhood trauma on reasons for living, optimism, daily suicidal ideation, defeat and entrapment through the daily stress-related vulnerability factors.

4.3 Methods

4.3.1 Design and participants

A 7-day EMA study was utilised whereby participants completed three surveys per day. 302 participants were recruited during June 2022 through the Prolific platform to complete a 7-day daily diary survey. Participants were paid £1.50 for the baseline survey (taking 15 – 20 minutes) and 20p for each daily survey (taking 1-2 minutes). This totalled a potential incentive of £5.70. Participants were sent email notifications to complete their daily surveys once in a morning upon awakening (8am), at 12pm and again at 8pm, before the participants went to bed. 211 participants (67.8% female) completed two or more full days of surveys. Participants were required to be aged between 18 and 45 years old and fluent in the English language. Given the study aimed to recruit currently healthy participants, the participants were screened and excluded if they reported having any long-term health condition or chronic illness or a score above 14 for clinical insomnia symptoms (Insomnia Severity Index; Bastien et al., 2001). These exclusion criteria were to prevent potential confounding influences of age related decline or extreme values from sleep or long-term health conditions impacting the measures administered. The main study hypotheses were preregistered at AsPredicted.org (#98604). The current study was approved by the University Of Leeds School of Psychology Ethics Committee (PSYC-522).

The sample size was determined using a summary-statistics-based power analysis to detect a cross-level effect (Murayama et al., 2022) informed by a previous unpublished study dataset (Rogerson, 2019). The power analysis showed that a minimum sample of 220 would be required to achieve 80% power (t = -2.22, df = 130; the cross-level effect of childhood trauma on the level 1 slope between daily stress and executive functioning). Therefore, in order to allow for 10% attrition and drop out between

completing the baseline measures and the diaries, the current study aimed to recruit 242 participants. As noted above, the final sample size included in the main analyses (N=211) is 9 participants less than the minimum sample size indicated by the a priori power analysis.

4.3.2 Background measures

Childhood trauma

Assessed using the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003). This is a 28-item questionnaire that covers five sub-categories of childhood trauma; sexual abuse, physical abuse, emotional abuse, physical neglect and emotional neglect. Each subscale is rated from '1 – Never True' to '5 – Very often true'. Composite scores for overall trauma were calculated by summing all items in the CTQ. The scale had good internal consistency $\alpha = 0.84$; internal consistency was calculated for the scores on each scale for the current sample and good internal consistency in this context is a value of $\alpha \geq .70$ (Nunnally & Bernstein, 1994). Example items for each sub-scale are provided; emotional abuse 'I thought my parents wished I had never been born', emotional neglect 'I felt loved', physical abuse 'I got hit so hard that I had to see a doctor', physical neglect 'I didn't have enough to eat' and sexual abuse 'someone molested me'.

Lifetime suicidal ideation and attempt

Measured using the Adult Psychiatric Morbidity Survey (APMS, McManus et al., 2007) Two questions were taken from this survey to determine both lifetime suicidal ideation: "Have you ever seriously thought of taking your life, but not actually attempted to do so?" and lifetime suicide attempts "Have you ever made an attempt to take your life, by taking an overdose of tablets or in some other way?" Response options were "yes", "no" or "would rather not say". The APMS measure has been frequently used to determine lifetime history of suicidal behaviour (e.g., McDonald et al., 2017; D.B. O'Connor et al., 2021b; Stickley et al., 2016; Wetherall et al., 2018). Participants who answered yes to either question were classified as having a history of suicidal behaviour.

Loneliness

Assessed using the UCLA loneliness scale (Hughes et al., 2004). This 3-item scale (2004) asks participants "how often do you feel that you lack companionship?", "How often do you feel left out?", "How often do you feel isolated from others?". Answers range from '1 – Hardly ever', '2 – Some of the time' and '3 – Often'. The responses are summed to

give a range of scores from 3 – 9. The scale demonstrated good internal consistency, α = 0.88.

Resilience

Measured using the Connor-Davidson Resilience Scale (CD-RISC 10; Campbell-Sills & Stein, 2007). This 10-item measure of resilience's items reflect the degree to which individuals have the ability to cope with adversity rated from '0 – Not true at all' to '4 – True nearly all the time'; items in the scale include, "Not easily discouraged by failure". This version has displayed good psychometric properties and is highly correlated with the original 25-item version (r = 0.92, Campbell-Sills & Stein, 2007; Connor & Davidson, 2003). The scale had good internal consistency, α = 0.92.

Social connectedness

Assessed using the UCLA loneliness Scale-Revised (Russell et al., 1980) which is a 20-item self-report measure. Items are rated on a four-point Likert scale from '1 – Never' to '4 – Always'; including items such as 'I have nobody to talk to'. The Cronbach's alpha value indicated good internal consistency, α = 0.97.

Social support

Measured using the ENRICHD Social Support Inventory (ESSI; Mitchell et al., 2003). A 7-item measure that is comprised of items such as "Is there someone available to you whom you can count on to listen to you when you need to talk?" rated from '1 – None of the time' to '5 – All of the time'. The Cronbach's alpha value indicated good internal consistency, α = 0.90.

4.3.3 Daily measures

Stress was measured using a singular item asking participants "How stressed have you felt, since the last survey?". This item was rated from '0 – not at all stressed' to '4 – extremely stressed'. This item was developed by the research team for the purpose of the current study based on standard single item assessments of stress and has good face validity (O'Connor & Ferguson 2016).

Sleep was measured by asking participants about last night's sleep in the first survey of the day upon waking up at 8am. Sleep quality was assessed with one item whereby participants were asked to rate their sleep quality from '1 – Very bad' to '7 – Very good'; adapted from the Pittsburgh Sleep Quality Index (Buysse et al., 1989). Additionally,

morning tiredness was assessed with one item whereby participants were asked to rate from ‘1 – Not at all’ to ‘5 – Very’ how tired they felt that morning; these items were adapted from (Clancy et al., 2020) and have been shown to have good validity. Higher scores were indicative of poorer sleep quality and greater morning tiredness.

Daily Hassles was measured using the adapted Hassles and Uplifts Scale (DeLongis et al., 1988) as used by Tinajero et al. (2020). Seven categories were chosen which participants rated on a Likert scale from ‘0 – none or not applicable’ to ‘3 – a great deal’ all questions asked ‘Today, how much of a hassle were ___ for you?’ and the categories were friends, work/school, external events, physical health, romantic partner, co-workers. The mean across all categories was taken for each day to indicate the degree of general daily hassles. The scale had an omega value of $\omega = 0.65$.

Defeat was measured using the four-item defeat scale was used from the Short Defeat and Entrapment Scale (SDES; Griffiths et al., 2015), items include “I feel defeated by life” and “I feel powerless” rated on a Likert scale from ‘0 – Never’ to ‘4 – Always’. The total score indicated the sum of the items and greater feelings of defeat. The scale demonstrated good internal consistency, with an omega value of $\omega = 0.96$.

Entrapment was assessed using the Entrapment Short-Form (E-SF; De Beurs et al., 2020), a four-item short form of the 16-item entrapment scale. For the current study, the two items representing internal entrapment were used only. The items “I feel I’m in a deep hole I can’t get out of” and “I feel trapped inside myself” were rated on a Likert scale from ‘0 – Not at all like me’ to ‘4 – extremely like me’. For the current study, the sum of the two items were used to indicate daily feelings of wanting to escape from inner feelings and thoughts (Baumeister, 1990). The scale has an omega value of $\omega = 0.78$.

Executive Functioning was measured using the WEBEXEC (Buchanan et al., 2010). A short self-report measure to assess the degree of executive functioning, designed for internet research. The WEBEXEC scores have been reported to be correlated positively with the DEX and confirmed that it appears suitable for online research (Buchanan et al., 2010). The scale consists of 6-items, such as, “Do you find it difficult to keep your attention on a particular task?” rated from ‘1 – No problems experienced’ to ‘4 – A great many problems experienced’. The total score was calculated for each day with higher scores indicating poorer executive functioning. The scale had an omega value of $\omega = 0.92$.

Impulsivity was measured using the Momentary Impulsivity Scale (Tomko et al., 2014). A 6-item scale where participants were asked to “Describe how much each statement

described their experience since the last completed prompt". In the current study, this item was changed to, "Please indicate how much each statement below describes your experience since the last survey" in order to be suitable for daily use. The items were rated from '1 – very slightly or not at all' to '5 – extremely'. The total score was calculated, higher scores indicated greater impulsive behaviours. The scale had an omega value of, $\omega = 0.75$.

Optimism was assessed using item 4 from the Revised Life Orientation Test (LOT-R; Scheier et al., 1994). Participants were presented with the following statement 'I feel optimistic about my future' to rate from '0 – strongly disagree' to '4 – strongly agree'. This item was adapted from 'I'm always optimistic about my future' to be suitable for daily diary use and is positively correlated with the total LOT-R scale score. The higher the score indicated a greater degree of optimism.

Reasons for living was assessed using item 4 from the Reasons for Living Inventory (RFLA; Linehan et al., 1983)). 'I have a desire to live' rated on a Likert scale from '1 – not at all important' to '6 – extremely important'. This item is positively correlated with the total RFLA scale score. The higher the rated score indicated a greater desire to live.

Thoughts of suicide were assessed in the final diary of each day. The measure consisted of an item from the Beck Suicide Scale (BSI; Beck & Steer, 1991) informed by the daily diary research and shown to be reliable and valid (Coppersmith et al., 2019). Participants were asked to rate the extent to which they: "wish to live", using the following three options '0 – I have a moderate to strong wish to live', '1 – I have a weak wish to live' and '2 – I have no wish to live'.

4.3.4 Statistical Analysis

Analyses were conducted in SPSS v.26 and multilevel models were conducted in HLM8 (Raudenbush & Congdon, 2021). The analysis assessed whether childhood trauma was associated with daily stress-related vulnerability factors and whether these associations were moderated by potential protective and risk factors. In addition, the analysis determined whether childhood trauma was associated with daily reasons for living, suicide thoughts, defeat and entrapment and whether the aforementioned daily stress-related vulnerability factors mediated these relationships. Analyses were performed on individuals who completed at least 2 full days of the study (i.e., at least 6 surveys). This resulted in a total of 211 participants completing 3719 diaries over a 7-day period: 1269 morning surveys; 1236 afternoon surveys; 1214 evening surveys. The dataset was

checked for outliers by scanning the data for any values ± 3 standard deviations away from the mean for all continuous variables. Each measure had the ranges checked to ensure no errors were present in the dataset. Normality was assessed through utilising the Kolmogorov-Smirnov test, all values $p > 0.05$. Histograms and Q-Q plots were also inspected to confirm the data was normally distributed.

Multilevel mediation analysis was conducted using the MLMED macro in SPSS (Rockwood & Hayes, 2017). Using multilevel mediation analysis, the study explored whether there were indirect effects of CTQ total score on daily reasons for living, optimism, suicide thoughts, defeat and entrapment through daily stress-related vulnerability factors. In these analyses, total CTQ score (at level 2) and daily measured reasons for living, optimism, suicide thoughts, defeat and entrapment (at level 1) were the X and Y variables (respectively), the daily stress-related vulnerability factors (at Level 1) were the mediators (M variables). The indirect effects use Monte Carlo simulation to generate the confidence intervals and report the unstandardized estimates, these are summarised in Table 6.

For the daily measured variables included in each of the three surveys; stress, defeat, entrapment, executive functioning, impulsivity, optimism and reasons for living. When any of these variables were entered as an outcome in the moderation, all three time points were utilised, meaning participants could have up to 21 rows of data. In contrast, for daily variables measured once in the morning (sleep quality, morning tiredness) or in the evening survey (daily hassles and suicide thoughts), participants could have up to 7 rows of data. For the mediation models, three datasets were constructed to analyse our research question. First, a dataset for when both outcomes and mediators were measured three times daily; second, when outcomes were measured three times daily but the mediator was measured once daily; third, when both the outcomes and the mediator were measured once daily but at different time points (morning and evening). For the first dataset, all variables utilised each time point measured. For the second and third dataset, any variables measured three times had the mean aggregate taken to allow data analysis to be possible alongside the single measure variables. This meant for the first dataset there were 3719 entries, second dataset had 1269 surveys and lastly, in the mediation analyses investigating both suicide and sleep in the same model, there were a total of 1093 diaries for individuals who had completed both morning and evening survey on the same day.

Hierarchical linear modelling was used to assess the relationship between childhood trauma and daily stress-related vulnerability factors and to test whether any observed relationships were moderated by protective and risk factors. The data were considered to have a two-level hierarchical structure. There were no level 1 predictors. The level 2 variable, suicide history, was uncentered in the model as it was dichotomous in nature. The other level 2 variables (childhood trauma, resilience, social support, social connectedness and loneliness) were grand mean centered as they were continuous variables. The level 1 variables were modelled as random as it was assumed that each of the within-person variables would vary from day to day. The level 2 variables were assumed to be fixed. The main analyses were conducted in three blocks. First, the study tested whether CTQ was associated with each of the stress-related vulnerability factor outcomes over the 7 days by entering CTQ as the level 2 predictor in each model. Second, the study investigated whether childhood trauma interacted with each of the protective or risk factors separately by entering CTQ, an individual risk/protective factor and the CTQ and risk/protective factor interaction term. The analyses were conducted with, and without, covariates (age, sex; cf., Simmons et al., 2011). The unadjusted and adjusted models are presented in the Appendix, Table 20. The main results present the unadjusted models as there was no difference found when adding the covariates. Importantly, due to the number of analyses conducted in this research, a more conservative p-value was adopted based on dividing alpha by the number of outcome variables (0.05 / 5 = p = .01).

4.3.4.1 Treatment of missing data

Missingness was assessed at item level. For the level 1 data, missing value analysis was considered for each survey time point; morning, afternoon and evening. For the morning diaries 0.39% of cases were missing (80 values). When assessing missingness, data was not missing completely at random (X^2 (45) = 101.85, p < .001). For the afternoon diaries, 0.32% of cases were missing (66 values), this was missing completely at random (X^2 (59) = 42.74, p = .945). Lastly, for the evening diaries, 0.57% of cases were missing (140 values). When assessing missingness, data was not missing completely at random (X^2 (98) = 145.62, p = .001). For the level 2 data there was 0.94% missing data (6 values) and this was missing completely at random (X^2 (292) = 320.89, p = .118). Single imputation using an expectation maximisation algorithm was used as less than 5% of the variables were missing therefore less bias is likely to occur. It is also preferred to use this method over simple mean imputation as expectation maximisation imputations are better than

mean imputations because they preserve the relationship with other variables and it provides unbiased parameter estimates (Enders, 2001; Scheffer, 2002).

4.4 Results

Descriptive statistics for the background and daily variables, measured at one or three time points are presented in Table 3. Similar scores were observed in previous research assessing total CTQ (M = 40.52). Scores on the other main study variables were within normal ranges. 72 participants reported suicidal ideation or a suicide attempt history by answering 'yes' to either of the APMS questions. There were 60 individuals reporting only suicide thoughts and 12 individuals reporting both thoughts and attempts. In addition, 105 individuals reported childhood trauma.

Table 3. Mean and standard deviations for level 1 and 2 variables in the total sample (n = 211) and in males (n = 88) and females (n = 143) separately.

	Total		**Male**		**Female**	
Level 1	**M**	**SD**	**M**	**SD**	**M**	**SD**
Stress	1.20	1.00	1.12	0.71	1.24	0.78
Impulsivity	5.76	2.34	5.69	1.90	5.79	1.94
Executive function	8.52	3.32	8.21	2.62	8.67	2.97
Reasons for living	5.33	1.17	5.13	1.31	5.43	1.00
Optimism	3.10	1.06	3.20	0.93	3.06	0.87
Defeat	3.98	4.23	3.33	3.29	4.26	3.71
Entrapment	1.79	2.10	1.67	1.67	1.85	1.84
Sleep quality	4.77	1.78	4.98	1.74	4.68	1.80
Morning tiredness	2.61	1.17	2.50	1.10	2.67	1.20
Hassles	0.39	0.41	0.37	0.39	0.41	0.42
Daily suicide thoughts	0.08	0.30	0.09	0.32	0.08	0.29
Level 2	**M**	**SD**	**M**	**SD**	**M**	**SD**
Age (years)	33.76	6.87	33.52	6.57	33.82	7.03
Childhood trauma	40.52	14.77	39.46	13.82	40.94	15.24

Suicide history (n = history of suicidal behaviour)	n = 72		n = 19		n = 53	
Resilience	35.18	7.69	37.99	7.44	33.88	7.51
Loneliness	5.38	1.93	4.97	1.87	5.58	1.93
Social support	25.84	6.24	26.40	6.71	25.62	6.02
Social connectedness	48.04	3.53	48.18	3.16	47.97	3.71

*Score ranges for level 1 variables: stress (0 – 4), impulsivity (6 – 30), executive function (6 – 24), reasons for living (1 – 6), optimism (0 – 4), defeat (0 – 16), entrapment (0 – 8), sleep quality (1 – 7), morning tiredness (1 – 5), hassles (0 – 3), daily suicide thoughts (0 – 2). Score ranges for level 2 variables: age (19 – 45), childhood trauma (28 – 140), resilience (0 – 40), loneliness (3 – 9), social support (8 – 34), social connectedness (0 – 60).

4.4.1 Effects of childhood trauma on daily stress-related vulnerability factors (hassles, stress, executive functioning, impulsivity and sleep)

The findings for each of the effects of childhood trauma on each of the daily stress-related vulnerability factors are presented in Table 4. The results showed main effects of childhood trauma on daily stress (β = 0.01, $p < .001$), executive functioning (β = .039, $p = .003$) and sleep quality (β = -0.02, $p < .001$) indicating that higher levels of childhood trauma were associated higher daily stress, poorer executive functioning and sleep quality (see Figure 8). However, childhood trauma was not related to daily hassles or morning tiredness.

Table 4. A summary of the main effects of childhood trauma on daily stress-related vulnerability factors.

	β	**Coefficient**	***SE***	***df***	***p* value**
Hassles					
Intercept	β00	0.400	0.021	209	< .001
CTQ	β01	0.004	0.002	209	.029
Stress					
Intercept	β00	1.217	0.039	209	< .001
CTQ	β01	0.010	0.003	209	< .001
Impulsivity					
Intercept	β00	5.780	0.108	209	< .001

CTQ	β01	0.027	0.010	209	.005
Executive function					
Intercept	β00	8.566	0.162	209	<.001
CTQ	β01	0.039	0.013	209	.003
Sleep quality					
Intercept	β00	4.767	0.079	209	< .001
CTQ	β01	-0.018	0.005	209	< .001
Morning tiredness					
Intercept	β00	2.611	0.051	209	< .001
CTQ	β01	0.007	0.004	209	.048

Note: statistical significance = $p < .01$ (see 564.3.4 for further detail on significance)

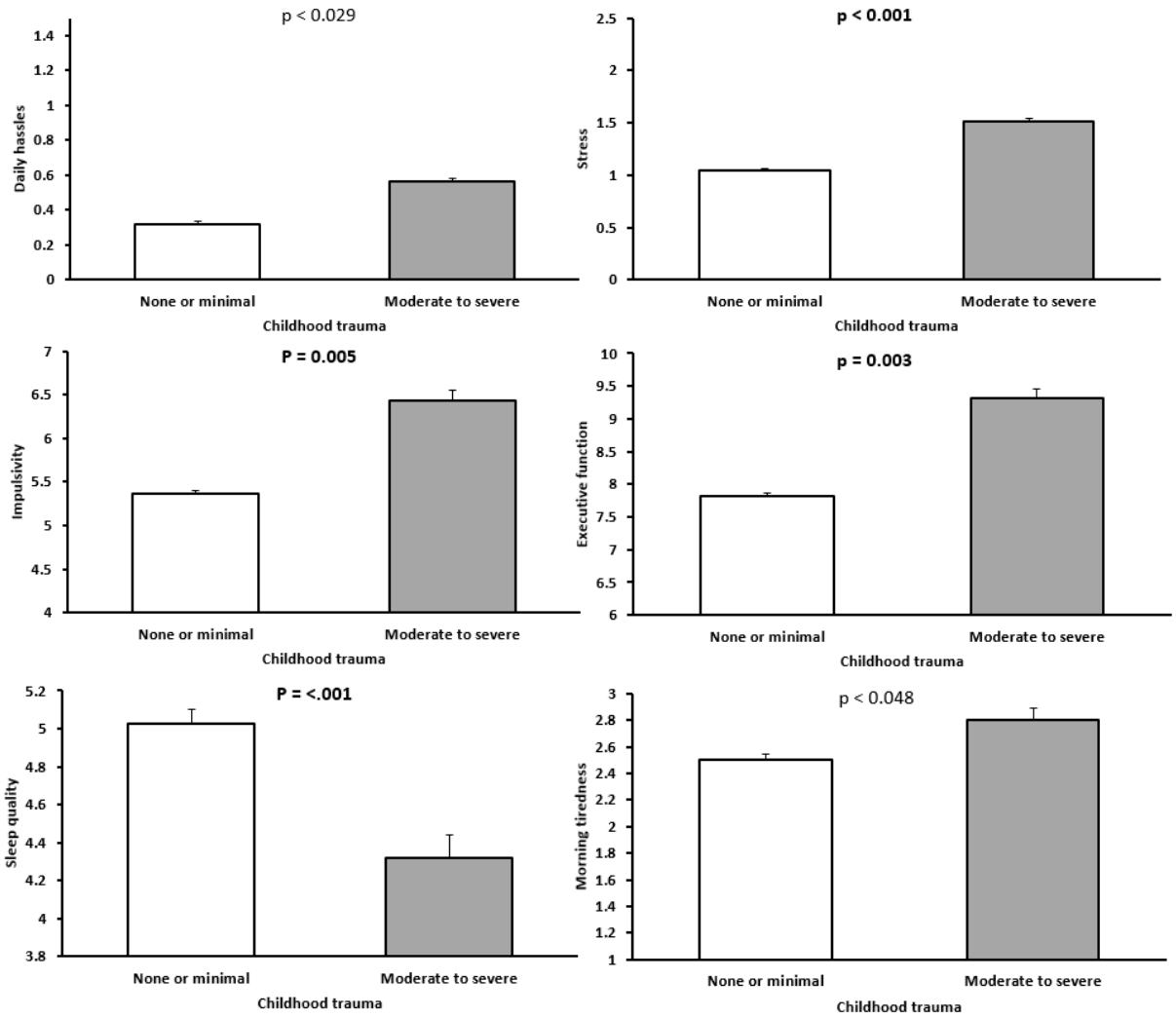


Figure 8. Effects of childhood trauma on daily stress, impulsivity, executive function and sleep outcomes.[1]

4.4.2 Moderating effects of protective factors (resilience, social support, social connectedness) and risk factors (loneliness and suicide history) on the childhood trauma - daily stress vulnerability relationships

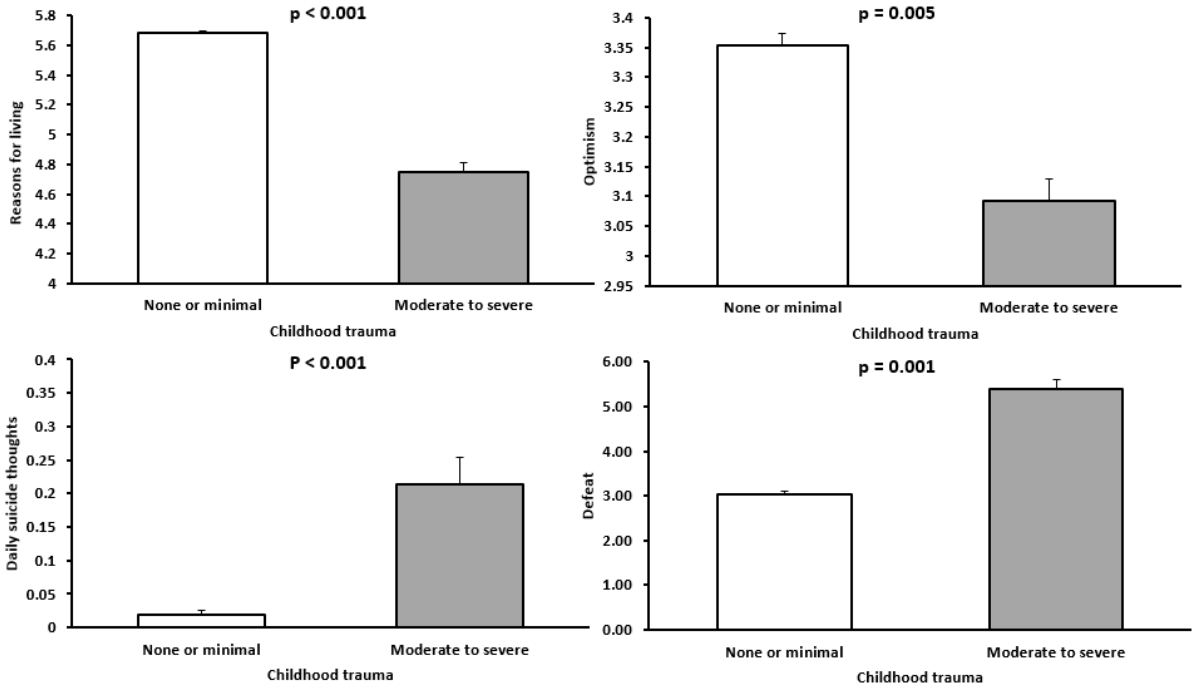
There were no moderating effects of the childhood trauma and daily stress-related variables relationships by any of the protective or risk factors (see Appendix, Table 20). However, there were main effects of loneliness on all daily stress-related vulnerability factors; suggesting that higher levels of loneliness were associated with a higher number of hassles, poorer sleep quality, poorer executive function, greater morning tiredness, greater daily stress, and more impulsive behaviour (see Appendix, Table 20). Similarly, higher levels of resilience were associated with better sleep quality and executive

[1] *Note*: 'none or minimal' < 37; 'moderate to severe' > 55 based on the cut-offs for the subscales reported by Bernstein et al. (2003).

function, lower levels of impulsivity, morning tiredness and daily stress. There were also main effects of suicide history such that individuals who had a suicide history reported greater daily stress, poorer sleep quality, greater morning tiredness and poorer executive function than individuals without a suicide history. Lastly, main effects of social support were found such that lower stress, greater executive functioning and lower impulsivity was reported in individuals who had higher social support compared to those with lower social support. For social connectedness, there were no main effects observed.

4.4.3 Direct effects of childhood trauma on reasons for living, optimism, daily suicide thoughts, defeat and entrapment

Childhood trauma was significantly associated with all daily measured suicide risk factors such that higher levels of childhood trauma were related to lower reasons for living and optimism, and higher daily suicide thoughts, defeat and entrapment (see Figure 9). The results of the aforementioned associations are presented in Table 5 below and are the basis for the subsequent mediation analyses.



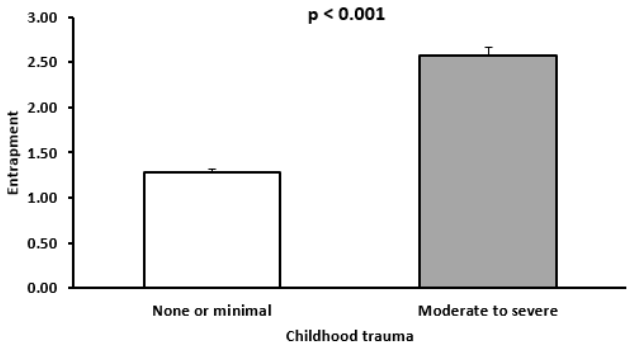


Figure 9. Effects of childhood trauma on daily indicators of suicide risk. [2]

Table 5. A summary of the main effects of childhood trauma on daily suicide risk factors

	β	Coefficient	*SE*	*df*	*p* value
Reasons for living					
Intercept	β00	5.343	0.067	209	< .001
CTQ	β01	-0.022	0.006	209	< .001
Optimism					
Intercept	β00	3.11	0.055	209	< .001
CTQ	β01	-0.011	0.004	209	0.005
Daily suicide thoughts					
Intercept	β00	0.004	0.002	209	< .001
CTQ	β01	0.005	0.002	209	< .001
Defeat					
Intercept	β00	3.986	0.223	209	< .001
CTQ	β01	0.058	0.018	209	0.001
Entrapment					
Intercept	β00	1.792	0.109	209	< .001
CTQ	β01	0.03	0.009	209	< .001

[2] Note: 'none or minimal' < 37; 'moderate to severe' > 55 based on the cut-offs for the subscales reported by Bernstein et al. (2003).

4.4.4 Indirect effects of childhood trauma on reasons for living, optimism, daily suicide thoughts, defeat and entrapment via daily stress-related vulnerability factors

Next, the study tested whether there were indirect effects of childhood trauma on the aforementioned outcome variables through daily stress-related vulnerability factors. In these analyses, childhood trauma (at Level 2) and outcome variables (at Level 1) were the X and Y variables, respectively, and daily stress-related factors (at Level 1) acted as the mediator (M variable). All the analyses are adjusted for age and sex. A summary of the indirect effects, showing unstandardised coefficients, is shown in Table 6 and in Figure 10.

Table 6. Summary of all the indirect effects

Indirect effects	b (unstandardised)	SE	Monte Carlo 95% CI		*p*
			Lower	Upper	
Outcome: reasons for living					
1.1 CTQ - hassles - reasons for living	-0.0015	0.0011	-0.0039	0.0002	0.1630
1.2 CTQ - stress - reasons for living	-0.0029	0.0015	-0.0061	-0.0004	0.0469
1.3 CTQ - executive functioning - reasons for living	-0.0050	0.0018	-0.0089	-0.0019	0.0055
1.4 CTQ - impulsivity - reasons for living	-0.0024	0.0014	-0.0054	-0.0001	0.0814
1.5 CTQ - sleep quality - reasons for living	-0.0032	0.0014	-0.0064	-0.0007	0.0288
1.6 CTQ - morning tiredness - reasons for living	-0.0017	0.0011	-0.0042	0.0000	0.1208
Outcome: optimism					
2.1 CTQ - hassles - optimism	-0.0029	0.0013	-0.0056	-0.0007	0.0211
2.2 CTQ - stress - optimism	-0.0069	0.0020	-0.0110	-0.0033	0.0005
2.3 CTQ - executive functioning - optimism	-0.0063	0.0020	-0.0105	-0.0026	0.0015
2.4 CTQ - impulsivity - optimism	-0.0036	0.0014	-0.0066	-0.0013	0.0090
2.5 CTQ - sleep quality - optimism	-0.0044	0.0016	-0.0077	-0.0017	0.0051
2.6 CTQ - morning tiredness - optimism	-0.0031	0.0016	-0.0064	-0.0002	0.0489
Outcome: daily suicide thoughts					
3.1 CTQ - hassles - daily suicide thoughts	0.0011	0.0004	0.0003	0.0021	0.0118
3.2 CTQ - stress - daily suicide thoughts	0.0018	0.0005	0.0008	0.0029	0.0006
3.3 CTQ - executive functioning - daily suicide thoughts	0.0018	0.0006	0.0007	0.0030	0.0017
3.4 CTQ - impulsivity - daily suicide thoughts	0.0013	0.0004	0.0005	0.0022	0.0033
3.5 CTQ - sleep quality - daily suicide thoughts	0.0008	0.0003	0.0002	0.0016	0.0172
3.6 CTQ - morning tiredness - daily suicide thoughts	0.0008	0.0004	0.0001	0.0016	0.0407

Outcome: defeat					
4.1 CTQ - hassles - defeat	0.0194	0.0073	0.0060	0.0352	0.0081
4.2 CTQ - stress - defeat	0.0392	0.0105	0.0193	0.0608	0.0002
4.3 CTQ - executive functioning - defeat	0.0373	0.0110	0.0161	0.0599	0.0007
4.4 CTQ - impulsivity - defeat	0.0261	0.0079	0.0114	0.0430	0.0010
4.5 CTQ - sleep quality - defeat	0.0180	0.0064	0.0068	0.0320	0.0047
4.6 CTQ - morning tiredness - defeat	0.0153	0.0075	0.0011	0.0312	0.0427
Outcome: entrapment					
5.1 CTQ - hassles - entrapment	0.0094	0.0035	0.0029	0.0170	0.0082
5.2 CTQ - stress - entrapment	0.0191	0.0051	0.0094	0.0296	0.0002
5.3 CTQ - executive functioning - entrapment	0.0172	0.0051	0.0074	0.0277	0.0008
5.4 CTQ - impulsivity - entrapment	0.0122	0.0037	0.0053	0.0202	0.0011
5.5 CTQ - sleep quality - entrapment	0.0075	0.0028	0.0026	0.0137	0.0079
5.6 CTQ - morning tiredness - entrapment	0.0067	0.0034	0.0005	0.0138	0.0457

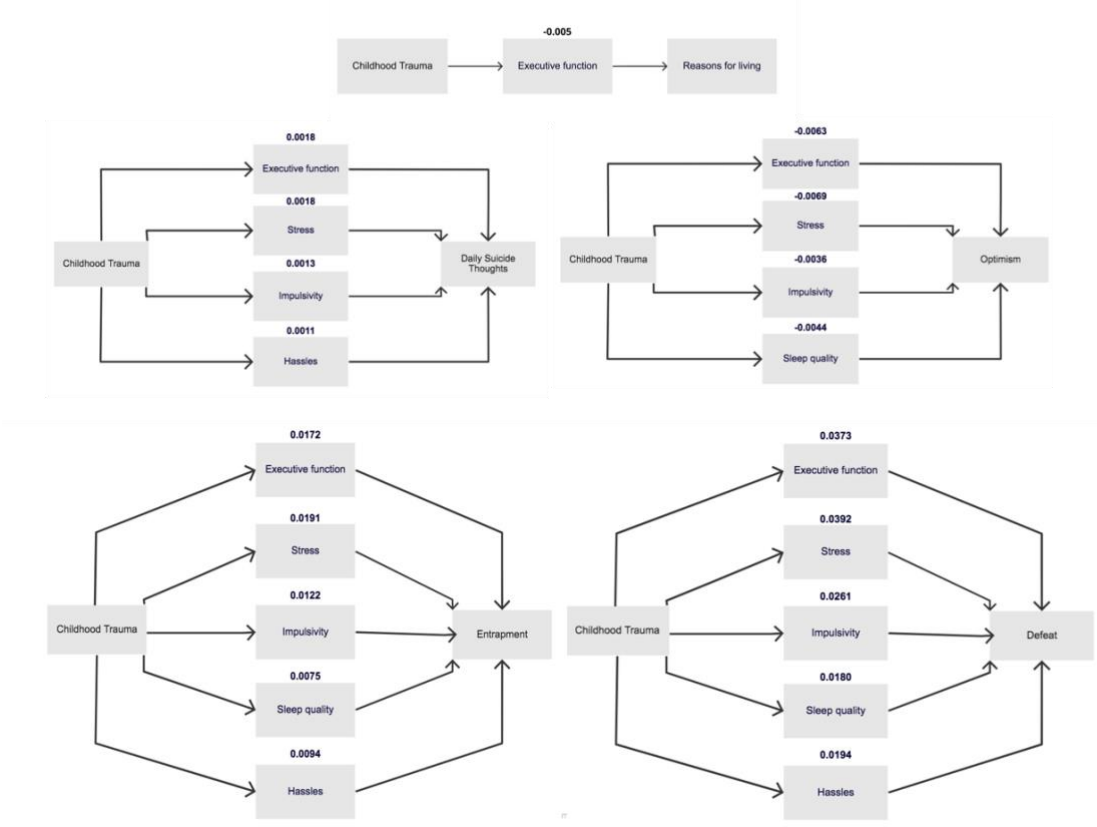


Figure 10. Path diagrams showing the significant indirect effects on outcome variables through daily stress-related vulnerability factors.[3]

[3] All diagrams represent unstandardized B and mediators are grouped for illustrative purposes only, each mediator was in a separate model.

4.4.4.1 Reasons for Living

The study tested whether there were indirect effects of childhood trauma on reasons for living through the daily stress-related vulnerability factors. There were indirect effects of childhood trauma on reasons for living through daily executive functioning (B = -0.005, p = .006). There were no indirect effects through daily hassles, daily impulsivity and daily morning tiredness.

4.4.4.2 Optimism

There were significant indirect effects of childhood trauma on optimism through all of the daily stress-related vulnerability factors: daily stress (B = -0.007, p = .001), daily executive functioning (B = -0.006, p = .002), daily impulsivity (B = -0.004, p = .009) and daily sleep quality (B = -0.004, p = .005).

4.4.4.3 Defeat

There were significant indirect effects of childhood trauma on defeat through all of the daily stress-related vulnerability factors: daily hassles (B = 0.019, p = .008); daily stress (B = 0.039, p < .001); daily executive functioning (B = 0.037, p < .001); daily impulsivity (B = 0.026, p = .001); daily sleep quality (B = 0.018, p = .005).

4.4.4.4 Entrapment

There were significant indirect effects of childhood trauma on entrapment through all of the daily stress-related vulnerability factors: daily hassles (B = 0.009, p = .008); daily stress (B = 0.019, p < .001); daily executive functioning (B = 0.017, p < .001); impulsivity (B = 0.012, p = .001); sleep quality (B = 0.008, p = .008).

4.4.4.5 Suicide thoughts

There were significant indirect effects of childhood trauma on greater daily suicide thoughts through all of the daily stress-related vulnerability factors: daily hassles (B = 0.001, p = .012), daily stress (B = 0.002, p < .001), daily executive functioning (B = 0.002, p = .002) and daily impulsivity (B = 0.001, p = .003).

Taken together, these results show that, in addition to the direct effects on key risk factors for suicide, childhood trauma has indirect effects on suicide vulnerability factors by influencing daily stress-related variables.

4.5 Discussion

First, childhood trauma significantly affected daily functioning by influencing stress-related vulnerability variables (while controlling for age and sex). In particular, childhood trauma was associated with greater daily stress and impulsivity and poor sleep quality and executive functioning. Second, there was robust evidence that childhood trauma affected established indicators of suicide risk (such as defeat and entrapment). Third, childhood trauma indirectly affected suicide risk factors through key daily stress-related variables. Specifically, childhood trauma had indirect effects on daily defeat, entrapment, reasons for living, optimism and suicide thoughts through daily executive functioning. There was also evidence for an indirect effect of childhood trauma on optimism, daily thoughts of suicide, defeat and entrapment through daily impulsivity and stress. Sleep quality was also found to mediate the effects of childhood trauma on optimism, defeat and entrapment. Lastly, an indirect relationship between childhood trauma and daily thoughts of suicide, defeat and entrapment through daily hassles was also found.

The current findings are important as they confirm, and extend previous research suggesting childhood trauma predisposes individuals to poorer health outcomes through impacting a number of stress-related processes in adulthood. A conceptual model posits that exposure to childhood trauma is associated with reduced stress reactivity, altered cognitive abilities and greater impulsive behaviours which in turn contribute to greater risk of experiencing poorer health behaviours (Lovallo, 2013). The study found childhood trauma to negatively impact cognitive ability indexed by daily executive functioning; echoing findings whereby exposure to childhood abuse was associated with poorer executive functioning in daily life in a sample of healthy adults (Tinajero et al., 2020). The current study fails to replicate the finding of trauma impacting daily hassles (Tinajero et al., 2020). This disparity may be, in part, due to the current study assessing general childhood trauma as opposed to childhood abuse, per se. Nevertheless, it was found that greater childhood trauma impacted daily self-reported stress; aligning with the notion that the experience of childhood trauma may predispose individuals to poorer health outcomes in the future by influencing daily levels of stress through prolonged activation of the stress response system. It has been suggested that the cumulative burden of chronic stress and life events, such as childhood trauma, exceed the ability to cope potentially leading to allostatic overload (O'Connor et al., 2020; O'Connor et al., 2021b). Repeated activation of the stress response system can dysregulate immune, cardiovascular and endocrine systems and individuals' daily cortisol awakening response.

The results of this research are also consistent with a growing body of work that has confirmed strong links between childhood trauma and suicide risk in adulthood (Gartland et al., 2022; Lewis et al., 2019; Marshall et al., 2013; O'Connor et al., 2018). However, much of the previous research has been overly reliant on cross-sectional designs, whereas, the current study extends earlier work into naturalistic settings showing that childhood trauma is significantly associated with a range of daily indicators of suicide risk in adulthood; reasons for living, optimism, daily suicide thoughts, defeat and entrapment. Defeat and entrapment have been identified as important variables in understanding suicide risk, for example the IMV model of suicidal behaviour conceptualises suicide as a behaviour that results from a complex interplay of factors; a pathway from ideation to behaviour through defeat and entrapment (O'Connor and Kirtley, 2018). Moreover, optimism is a factor identified in the IMV model, it is predictive of suicide risk, and experiencing childhood adversity is associated with lower self-perceptions of optimism (Mumford et al., 2022). The current study found optimism to be significantly associated with childhood trauma both directly, and indirectly, through all of the daily stress-related vulnerability factors. Therefore, although the negative outcomes associated with childhood trauma are well established, these findings suggest that fostering a greater optimistic outlook may potentially help mitigate, in part, the negative effects on health outcomes, such as suicide risk.

Our findings also suggest that exposure to childhood trauma may create a generalised vulnerability in adulthood across cognitive, stress-related, behavioural and sleep domains. The current study found that childhood trauma had indirect effects on a range of daily suicide vulnerability variables (i.e., reasons for living, optimism, defeat and entrapment) through multiple stress-related variables (daily stress/hassles, executive functioning, impulsivity and sleep quality). This is consistent with a recent study that found indirect effects of childhood trauma on recent suicidal ideation through executive functioning and impulsivity (Rogerson et al., 2022). Importantly, these findings suggest that childhood trauma influences multiple different processes that are likely to influence future psychological health. Therefore, further research should endeavour to investigate the effects of childhood trauma across a broad range of cognitive, psychological, biological and behavioural domains.

Another important finding of the current study is that childhood trauma was shown to influence suicide vulnerability, as indexed by higher levels of daily defeat and entrapment, through poorer levels of sleep quality. This is a notable observation

particularly given the recent work showing links between sleep quality and suicidal ideation. For example, Littlewood et al. (2019), in a sample of participants with current suicidal ideation, found that days preceded with shorter sleep duration and poorer sleep quality were associated with greater levels of suicidal ideation. It is well established that childhood trauma has serious long-term negative consequences on different components of sleep, and poor sleep health in turn has been linked with numerous negative health outcomes (e.g., Buysse, 2014). Moreover, a conceptual model linking childhood trauma and sleep disturbances to poor health outcomes suggests a role for the HPA axis in understanding the relationships between both constructs and later health outcomes (Fuligni et al., 2021). The influence of childhood trauma on HPA axis dysregulation may negatively affect stress reactivity and cortisol awakening responses, but also sleep quality, which in turn may affect poor health outcomes (O'Connor et al., 2018, O'Connor et al., 2024). Collectively, these findings confirm that in both a healthy adult sample, and a sample with individuals at vulnerable to suicide, that childhood trauma is associated with indicators of suicide risk through a poorer sleep quality pathway. Future research ought to continue to consider the role of sleep quality as one of the putative mechanisms through which childhood trauma confers its future physical and mental health risks.

The current findings may have implications for interventions aimed at mitigating the negative impacts of childhood trauma. They suggest that such interventions should incorporate components that target modifiable risk factors such as sleep, stress, impulsivity and executive function. For example, Prudenzi et al. (2022) suggested that acceptance and commitment approaches could benefit stress and worry. There are further avenues for more targeted stress management interventions (Coppersmith et al., 2021; Rogerson et al., 2024) and to improve sleep outcomes (e.g. Murawski et al., 2018; Saruhanjan et al., 2021). There is also promising evidence showing that cognitive enhancement interventions such as goal management training, can be effective in reducing impulsive action and choices (Anderson et al., 2021).

It is recognised that there are a number of shortcomings to the current research. First, the current sample falls a little short of the target sample size determined by the a priori power analysis (i.e., the main analysis was based on 9 participants less than the 220 target sample size). The main reason for this was because we wanted to perform the analysis only on individuals who completed at least 2 full days of the study (i.e., at least 6 surveys). Nevertheless, it is worth noting that the main analysis was still conducted on data from 3719 diaries and the study found strong support for our main hypotheses, suggesting that

the current study was adequately powered to detect the predicted effects. Moreover, in terms of research in this area, this sample is considered relatively large and the within-participants, EMA design, comes with several strengths such as multiple observations, and using each participant as their own control. Nonetheless, exploring the associations between childhood trauma and these important stress-related vulnerability variables in a larger sample and separated into different assessment bouts over time (e.g., Jones et al., 2024) are important next steps for research in this area.

Second, as outlined earlier, the study recruited healthy participants because we wanted to explore mechanisms by which childhood trauma may be associated with suicide risk factors, *prior* to the development of mental and physical health problems. As such, the current sample comprised of healthy young adults aged 18–45 years who were screened for sleep problems and chronic illness and as a result, the sample may not yet evidence deficits in some aspects of executive functioning. That is, it may be that longer-term exposure to stress-related dysfunction such as sleep disturbance leads to broader executive deficits over time (Tinajero et al., 2020). Moreover, it is recognised that adopting these inclusion criteria limits the generalisability and representativeness of the sample. The generalisability of our findings to older populations where executive function, impulsivity and cognitive measures may differ is unknown and thereby further investigation is required in individuals capturing all age groups including older populations.

Third, it is acknowledged that there was limited variability in suicidal ideation scores across the 7 days. This may be, in part, due to the healthy sample of adult participants included in the current research compared to other studies that recruited participants who reported suicidal ideation following psychiatric hospitalisation or that targeted recruitment at participants with a history of suicide (e.g., Kleiman et al., 2017; O'Connor et al., 2024, O'Connor et al., 2020). Therefore, future research ought to attempt to replicate these findings in a more representative sample that includes individuals with and without a mental and physical health conditions.

Finally, there are a number of potential shortcomings relating to the measures included in the current study. Of note, that our measures of executive function and sleep are self-reported and future research ought to attempt to replicate our results using more objective methods such as polysomnography and laboratory-based neuropsychology tests. It is recognised that suicidal ideation and history was assessed using only two questions from

the Adult Psychiatry Morbidity Survey. Although these questions are used widely in the literature (e.g., McDonald et al., 2017; O'Connor et al., 2021a; Stickley et al., 2016; Wetherall et al., 2018), it is acknowledged that such brief assessments may have limitations and do not provide more detailed and nuanced data on suicidal ideation, plans, intent, gestures or type of suicide attempts. Future research should endeavour, where possible, to include more comprehensive assessment tools such as Self Injurious Thoughts and Behaviours Interview (Nock et al., 2007).

In conclusion, the current study found that childhood trauma was significantly associated with higher scores on daily stress-related vulnerability variables and daily indicators of suicide risk. Moreover, it identified key pathways whereby childhood trauma had indirect effects on suicide vulnerability through executive functioning, impulsivity, sleep quality and stress in adulthood. Taken together, these findings highlight the complexity of childhood trauma and its potentially damaging effects on stress-related vulnerability factors and poorer mental health outcomes. A greater understanding of the pathways by which childhood trauma influences later health outcomes is essential for development of appropriate, and timely, intervention.

Chapter 5 - Effectiveness of stress management interventions to change cortisol levels: a systematic review and meta-analysis

Abstract

Stress has a damaging impact on our mental and physical health, and as a result, there is an on-going demand for effective stress management interventions. However, there are no reviews or meta-analyses synthesising the evidence base of randomised controlled trials testing the effectiveness of psychological interventions on changing cortisol levels (the stress hormone) in non-patient groups. Therefore, the primary aim of this systematic review and meta-analysis was to address this gap. Six databases (Medline, PsychInfo, Embase, CINAHL, Cochrane and Web of Science) were searched (1171 studies identified) with 58 studies (combined N = 3508) included in the meta-analysis. The interventions were coded into one of four categories; mind body therapies, mindfulness, relaxation or talking therapies. A random effects meta-analyses on cortisol as measured in blood, saliva or hair found that stress management interventions outperformed pooled control conditions with a medium positive effect size (g = 0.282). The studies that utilised cortisol awakening measures (g = 0.644) revealed larger effects of stress management interventions than those that measured diurnal cortisol (g = 0.255). Mindfulness (g = 0. 345) and relaxation (g = 0. 347) interventions were most effective at changing cortisol levels, while mind body therapies (g = 0. 129) and talking therapies (g = 0.107) were shown to have smaller and non-significant effect sizes. Additionally, studies that utilised an active control group (g = 0. 477) over passive control group (g = 0.129) were found to have stronger effects. Length of the intervention, study quality, risk of bias, age and gender did not influence the effectiveness of interventions and there was no evidence of publication bias. Overall, the current findings confirm that stress management interventions can positively influence cortisol levels. Future research should investigate the longer term implications for health and health outcomes.

5.1 Introduction

Stress is a profound public health concern and an important mechanism through which the social and physical environment can impact later health outcomes (D.B. O'Connor et al., 2021a). It is well established that experiencing stressful life events and reporting greater perceived stress over sustained periods of time are associated with poorer mental and physical health (Epel et al., 2018; O'Connor et al., 2021). Additionally, experiencing

traumatic life events across one's life have also been consistently found to be associated with poorer health outcomes (Howarth et al., 2020; Liu & Miller, 2014).

A key mechanism regulating how the environment impacts the stress process is the stress hormone – cortisol. Cortisol is a product of the hypothalamic-pituitary adrenal (HPA) axis system which plays an essential role in regulating the body's biological systems - from metabolic to immune systems (Lupien et al., 2009; Sapolsky et al., 2000). The dysregulation of the HPA axis is well documented to have links with negative health outcomes: the chronic over-activation of the HPA axis through experiencing acute stress or stressful life events can lead to allostatic load (McEwen, 1998). Most recently, allostatic *overload* was conceptualised referring to the detrimental impacts of stress on the body's biological systems when stress mediators, such as cortisol, are released to respond to stress in one's environment but their excessive and prolonged use, as well as dysregulation, leads to tissue damage (McEwen & Rasgon, 2018). Collectively, stress, and by part, cortisol, impacts psychological and physical body functioning; subsequently implicated in mental and physical health outcomes, suggesting cortisol regulation plays a key mediating role in the relationship between stress exposure and later negative health outcomes (Adam et al., 2017; Chrousos & Gold, 1992; D. B. O'Connor, Thayer, et al., 2021a).

5.1.1 The stress response and health outcomes

Low and high cortisol responses to stress may be associated with poor health outcomes; research has emerged to suggest that smaller increases, or a blunted cortisol response, to stress may be indicative of current ill-health or future health risks (Lovallo, 2016). Lower cortisol stress reactivity has been shown to be associated with the risk of obesity and with symptoms of depression and anxiety (de Rooij, 2013). In other research it was found that individuals who had previously made a suicide attempt exhibited low levels of cortisol in response to an acute stressor compared to control participants (O'Connor et al., 2017). Moreover, the results of a meta-analysis found evidence of an association between early-life adversity and a blunted cortisol response to social stress (Bunea et al., 2017). Conversely, literature exists whereby heightened cortisol responses are associated with poorer health outcomes. Specifically, in trauma participants, it has been shown that there is an increase in cortisol to a stressor (Heim et al., 2000). Additionally, in another study, an elevated cortisol response to a stressor increased the odds of experiencing hypertension and progression to coronary artery calcification 3 years later (Hamer & Steptoe, 2012).

Collectively, evidence points towards both heightened and blunted cortisol responses being associated with poorer health outcomes in the future.

5.1.2 Cortisol across the day

The cortisol awakening response (CAR) is also implicated in later health status; linked to an array of health outcomes as confirmed in a meta-analysis whereby enhanced CAR is linked to job stress and general life stress. Conversely, reduced CAR has also been found to be associated with fatigue, exhaustion and burnout (Chida & Steptoe, 2009). The natural cortisol fluctuations throughout the day also play an important role in relation to later health. A flatter diurnal slope represented by low morning and high evening levels has also been suggested to be indicative of HPA dysregulation. Flatter diurnal cortisol slopes across the waking day may be one mechanism by which stress influences negative health outcomes (Adam & Kumari, 2009). A number of studies have found that there is an association between a flatter cortisol slope and negative health outcomes such as depression, cardiovascular disease, obesity and suicide attempt (Matthews et al., 2006; O'Connor et al., 2020; Ruttle et al., 2013). This is synthesised in a meta-analysis that found consistent evidence that flatter cortisol slopes were associated with numerous poor health outcomes, from cancer, to depression and even obesity (Adam et al., 2017).

5.1.3 Stress management interventions

Therefore, taken together, it is clear that stress can be damaging for our mental and physical health, and as a result, there is an on-going demand for effective stress management interventions. An abundance of stress management interventions exist, however, which type of intervention is most effective? Is there evidence that they can influence cortisol? How do they perform in randomised controlled trials? For example, some of the most increasingly popular intervention approaches are mindfulness based (Khoury et al., 2013). A previous systematic review reported varied success for mindfulness-based interventions on changing cortisol outcomes, finding mindfulness-based interventions had limited effectiveness but that they were more effective when standardised measures of cortisol were assessed such as the CAR and diurnal slope, instead of unstandardised measures such as averages of raw cortisol concentrations (Sanada et al., 2016). A recent meta-analysis found that meditation interventions were effective at lowering cortisol levels but only in highly stress samples that assessed cortisol in blood (Koncz et al., 2021). There is also evidence that psychological interventions can influence cortisol levels in patients with cancer, psychiatric conditions and other health

issues (e.g., Antoni et al., 2023, Saban et al., 2022). However, there are no reviews or meta-analyses synthesising the evidence base of randomised controlled trials testing the effectiveness of psychological interventions on changing cortisol levels in non-patient groups.

Therefore, the primary aim of the current systematic review and meta-analysis was to examine the effectiveness of psychological interventions to reduce cortisol levels in healthy adults that used randomised controlled trial designs. The secondary aim was to investigate the heterogeneity of any observed effects in terms of the type of cortisol measurement (in blood, hair or saliva), control group (active, inactive, waitlist or active/passive) and intervention together with exploring the moderating effects of sample size, study quality and risk of bias.

5.2 Methods

5.2.1 Protocol and registration

The inclusion and exclusion criteria, methods for analysis and protocol for the current systematic review and meta-analysis were preregistered on PROSPERO with the following registration number: CRD42019120066. Meta-analyses data are available on the Open Science Framework (https://rb.gy/tkrfp).

5.2.2 Eligibility Criteria

To be included in the review, studies had to have utilised a randomised controlled trial design to investigate the effectiveness of a psychological intervention(s) on cortisol outcomes *and* to have measured cortisol at baseline and post-intervention in order to determine the change in cortisol from pre- to post-intervention. The full study inclusion and exclusion criteria (PICOS) are outlined in Table 7.

Table 7. Outline of the study selection criteria

	Inclusion criteria	**Exclusion criteria**
Population	Healthy adult subjects (aged > 18 years). Subjects can be stressed or not stressed prior to the study.	Patients with cancer, diseases, obese, pregnancy, psychiatric or other health issues.

Interventions	Any psychological stress-management interventions: including, mindfulness, CBT.	Other pharmacological interventions
Control group	Waitlist control or other intervention	No control group
Outcome	Cortisol level measures in blood, saliva and hair. Cortisol can be measured with and without out an acute stress test.	Heart rate, blood pressure, only stress test assessments.
Studies	RCTs. Published in English language, journal articles, humans, published any year	Non-RCTs, open trials with a pre-post analysis. Published in other languages, reviews, posters, presentations, case reports, dissertations, letters.

Note: RCT = randomised controlled trial, CBT = Cognitive Behaviour Therapy

5.2.3 Search

The search was completed across six electronic databases: Medline, PsychInfo, Embase, CINAHL, Cochrane and Web of Science. The key terms such as “cortisol”, “stress management intervention” were used. Table 8 provides an example of the search strategy used in Embase. The search was regularly updated to ensure all relevant articles were included. The date of the last search was 06/04/23. Additionally, Google Scholar was used to thoroughly search through all studies citing the included studies. Figure 11 shows the selection of studies throughout the meta-analysis.

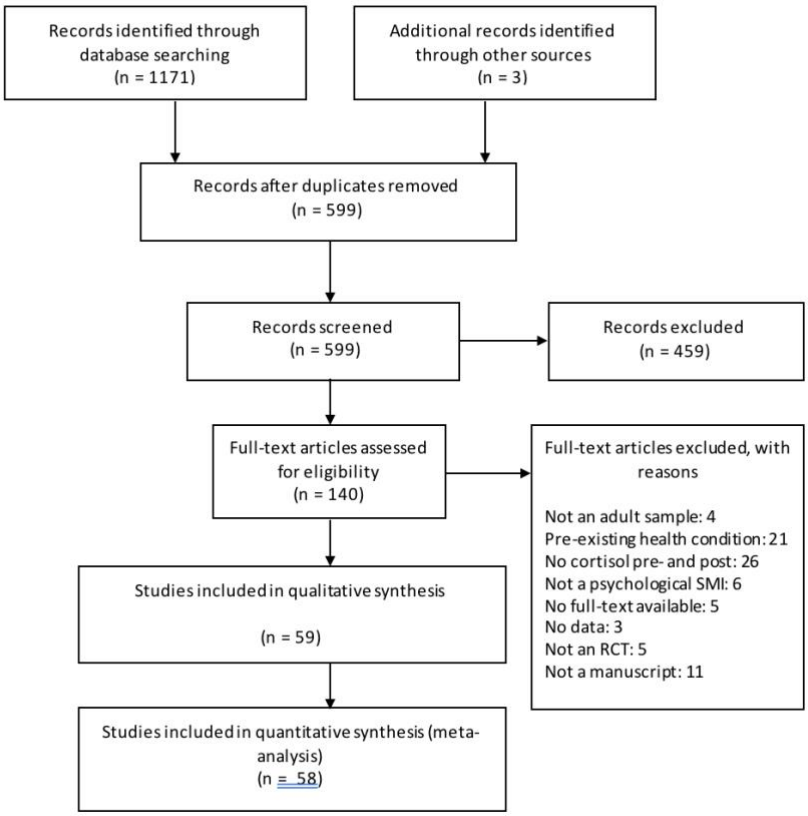


Figure 11. PRISMA study flow diagram of studies retained in the review. Reasons for exclusion included.

Table 8. Search strategy for Embase

1.	"adult" or "adulthood" or "man" or "men" or "women" or "woman" or "young adult" or "worker" or "employee"
2.	"mindfulness" or "mindfulness-based stress reduction" or "MBSR" or "meditation" or "stress management" or "cognitive behavioural stress management" or "CBSM" or "stress management training" or "stress management intervention" or "internet-based CBSM" or "IB-CBSM" or "internet-based stress management intervention" or "internet-based stress management" or "IBSM" or "iSMI" or "stress inoculation training" or "time management training" or "progressive muscle relaxation" or "biofeedback" or "guided imagery"
3.	"cortisol" or "cortisol response" or "cortisol awakening response" or "awakening cortisol response" or "saliva" or

	"salivary" or "hair cortisol" or "hypothalamic-pituitary-adrenal axis" or "HPA axis" or "salivary free cortisol response" or "diurnal cortisol" or "diurnal"
4.	"random allocation" or "randomised" or "randomized" or "RCT" or "random* trial" or "random* control trial" or "pilot study"
5.	1 AND 2 AND 3 AND 4
6.	Limit 5 limits for abstracts, human, English language, clinical trial (RCT), human age groups (adult 18-64 and 65+), source types (journal) , publication types (article)

5.2.4 Study Selection

A total of 1171 studies were identified during the searches and 3 additional papers through Google Scholar. Title and abstract screening were completed for eligibility by OR and a 20% overlap completed by SW. Duplicates were detected and removed through Endnote library. Full text screening was done by OR and 20% overlap completed by SW. Any disagreements were resolved by consensus and if an agreement could not be reached, a third researcher was required (DO'C). The inter-rater reliability on study selection was calculated to indicate a high level of agreement (K = 0.76, $p < .001$).

5.2.5 Data collection process and coding procedure

A data extraction table was used for extracting key information from the studies, this was based upon the Cochrane collaborative data collection template form (Cochrane Training, 2014). Additional components were added to the table, taken from O'Connor et al., (2016), to ensure data was extracted specific to cortisol measurement. In any instance of study information for data extraction not being clear, study authors were contacted to ask for more detail.

In instances when the mean age was not available in a study paper, the mean age was calculated from the age range information (e.g., Christopher et al., 2018; MacLean et al., 1997; Tsiouli et al., 2014). For some studies, overall mean age was calculated through taking the average of the intervention and control groups (Bottaccioli et al., 2020; Danucalov et al., 2013; Feicht et al., 2013; MacDonald & Minahan, 2018).

For some included studies, the standard error (SE) was presented. The standard deviation was calculated from SE and sample size using the following formula (SE x $\sqrt{N}$; Cochrane,

2014). This formula was utilised for the following papers: Domes et al. (2019), Fan et al. (2014), MacLean et al. (1997), Nyklíček et al. (2013) and Rosenkranz et al. (2013). Although for Rosenkranz et al., (2013) the average SE was first calculated across the 5 measures. In one included study the 95% confidence intervals were presented (e.g., Laudenslager, 2015). Therefore the SD was calculated using the following formula: SD = $\sqrt{N}$ x (upper limit – lower limit) / 3.92.

The current meta-analysis prioritised diurnal measures of cortisol over single measures. If the diurnal mean was possible to be calculated from the data included in a study, this was done using the following formula: sum of the mean at each time point/number of time points. for the following studies: Fotiou et al. (2016); Oken et al. (2010); Rosenkranz et al. (2013). To calculate the standard deviation when the diurnal mean was produced, this was done using the following formula: SQRT((sum of the SD at each time point^2 + b + c)/k). As one study, Rosenkranz et al., (2013), provided SEM so this was converted to SD first then the above formula was used to produce the SD in relation to the diurnal mean calculation.

For studies whereby the sample was not clear if N represented participants who completed both baseline and post-intervention, the author was contacted in the first instance. If we could not obtain additional information, the smaller of the two sample sizes were chosen to avoid overestimation of the effect size. For instance, Fendel et al., (2021) we took the T2 sample size as the intervention/control group size. For Jensen et al., (2012), for the mindfulness group, n = 14 was taken. Finally, Jensen et al., (2015) was contacted and responded regarding cortisol sample (n = 47).

In the current meta-analysis there were three crossover trials. In these instances, we inflated the sample size – for instance, in Benvenutti et al. (2017) they had a sample size of 24 who all completed the intervention and control conditions - therefore we inflated the total sample size to 48.

In cases of studies that had multiple active or passive control groups, we included both groups and divided the intervention sample size by the number of control groups to prevent inflation of the effect size and allow comparison against a variety of controls. The meta-analytic software used to conduct the analysis, Comprehensive Meta-Analysis (CMA), takes the average of the effect sizes in one study as these are not independent from each other before calculating a grand average.

When studies had more than one intervention group, the main psychological intervention was used in the meta-analysis and we treated the remaining intervention as a control condition because we were exploring determinants of effectiveness (*as per* Michie et al. (2009). This was the case for both studies by Bowden et al. (2012) and Brinkmann et al. (2020) who had two intervention groups; Bowden et al. (2012) compared brain wave vibration and mindfulness compared to yoga. The meta-analysis compared mindfulness to two comparison groups – brain wave vibration and yoga. Whereas Brinkmann et al. (2020) investigated the effects of biofeedback and mindfulness compared to waitlist controls. The current meta-analysis considered mindfulness as the intervention only.

5.2.6 Risk of bias and study quality

The Cochrane Collaborations tool for assessing risk of bias in RCTs was used (RoB2; Sterne et al., 2019). The first reviewer covered all studies, whilst the second reviewer (AP) reviewed 50% of the studies. Kappa coefficients were calculated for the all items in the RoB2 and indicated a moderate level of agreement ($K = 0.60$ $p < .001$). Following the assessment, the discrepancies lay in cortisol assessment criteria and these were resolved through discussion.

Since there is no validated rating scale available assessing the consideration of confounding influences during measurement of cortisol concentrations, we utilised a cortisol quality index from the existing literature (Laufer et al., 2018). This scale consists of several items which influence the measurement, and accuracy, of cortisol measurement dependent on whether it is measured in saliva or blood. We applied the scale to also consider hair cortisol in this instance. Items can be allocated to one of four categories: report of sampling design; reported strategies enhancing accuracy of sampling; consideration of confounders on the particular sampling day ("state covariates"; Stalder et al., 2016), consideration of confounders with regard to sociodemographic and health variables ("trait covariates"; Stalder et al., 2016). Items include whether cortisol was measured over consecutive days, if authors considered time of awakening and even the use of oral contraception in female samples. For each item, it is rated as either '0 – not considered', '1 – considered' or N/A as not all items are applicable to the study, depending on how cortisol was measured. The term 'considered' was indicated if the study addressed the potential confounder in one of the following: sampling instructions, a covariate in the analyses, reported in the descriptive statistics or included in the exclusion criteria of the study sample. The sum scores for each of the four categories were

calculated and divided by the maximum score the study could achieve in that category, based on the modality of cortisol. This created a percentage used to rate consideration as good consideration (100% - 66.1%), moderate consideration (66% - 33.1%) or low consideration (33% - 0%).

5.2.7 Data extraction plan

The following data was extracted from each study: number of participants analysed with cortisol, the number of participants in the intervention and control group(s), the mean age of the entire sample and separate intervention/control groups (if available). The percentage of females in the study, the included control conditions (active, inactive, waitlist), pooled control conditions (active/passive), type of intervention, broad intervention category, length of intervention in absolute minutes (if available), an interpretation of length of intervention (as short (0 – 250 minutes), medium (251 – 800), long (>801 minutes), type of cortisol sampling (blood/saliva/hair), categorisation of cortisol measurement (awakening/diurnal), number of days cortisol was measured on, number of times per day cortisol measured, timing of cortisol measurement (AM/PM/AM – PM), study quality (as described above) and whether the sample was stressed or non-stressed.

5.2.8 Meta-analytic procedure

All analyses were conducted using the Comprehensive Meta-Analysis 4.0 (CMA) software (Borenstein, 2022). The aim of the meta-analysis was to determine the effectiveness of stress management interventions on the change in cortisol levels from pre-intervention to post-intervention; meaning the dependent variable was the standardised mean difference change in cortisol from pre- to post-intervention between the intervention and comparator group. By utilising the standardised mean difference it permitted us to summarise evidence when studies used a variety of sampling strategies; from single measure, cortisol awakening response to diurnal cortisol. Following the procedure of Koncz et al. (2021) we devised a hierarchy of cortisol reporting, should different indices be available in a study; selecting the AUCg measure first, followed by the mean of multiple measures then choosing a single measurement. Additionally, if a study reported more than one control condition we included both contrasts (for instance, Errazuriz et al. (2022) utilised an active and waitlist control group). CMA software takes an average of multiple effects sizes in one study, as these are not independent of one another, before calculating a grand average. The current meta-analysis utilised the

random effects model and Hedges *g* as a measure of effect size; the magnitude of the effect is interpreted using the following parameters where a low effect size is approximately 0.20, medium is 0.50 and large is 0.80 (Cohen, 2013).

When considering the direction of effect, a positive effect size indicates favouring the intervention condition, shown by a larger decrease, or a smaller increase, in change in cortisol levels from pre- to post-test. As the included studies employed varied in the samples, interventions, control conditions and cortisol sampling approaches, average effect sizes and corresponding 95% confidence intervals were calculated based on the random-effects model, which accounts for between-study variances (Borenstein et al., 2009).

Funnel plots were inspected to determine the degree of publication bias whereby we can visually plot how the inherent difficulties of publishing non-significant results can lead to an overrepresentation of significant findings in the literature. We utilised Egger's regression coefficient to identify publication bias (Egger et al., 1997) and Duval and Tweedie's trim and fill analysis to understand the number of missing studies to the left and the right of the mean (Duval & Tweedie, 2000).

Lastly, sensitivity analyses were also performed by removing each study from the analyses one at a time. Further subgroup analyses investigated the effectiveness of types of intervention relative to control conditions (active, inactive and waitlist controls, as well as broader active/passive control groups), types of cortisol sampling (blood, saliva, hair), intervention group (mindfulness, relaxation, mind body therapy and talking therapy; see below), length of intervention (short, medium and long), study quality (low, average and high), stress risk (low risk, high risk), risk of bias (low, some concerns, high) and cortisol measurement (awakening, diurnal). Meta-regressions were also conducted to identify moderating variables (time elapsed between the end of the intervention and post-intervention cortisol measure and sample demographics)).

5.3 Results

5.3.1 Study characteristics

Of the 59 studies, 56 were RCTs and 3 were crossover trials (Benvenutti et al., 2017; Bittman et al., 2001; Lai & Li, 2011). 57 studies provided a baseline and post-intervention measure, 2 studies provided the pre-post intervention change in cortisol. In total, there were 3508 participants who were included in the meta-analysis, with the individual study

sample size ranging from 12 – 154. There were 1648 participants allocated to the intervention condition and 1860 allocated to the control condition. Collectively, there was a mean age of 35.84 years and the proportion of included females was 64.84%. The average intervention length was 19 hours in length across the studies but this ranged from 20 minutes to 4560 minutes (see Appendix, Table 21, for study characteristics). A total of 15 studies included samples with individuals considered to be at a stress risk. The remaining 44 studies were considered to have samples with no stress risk. For the type of cortisol measured, 13 were in blood, 43 were in saliva and 3 were in hair. We also characterised the cortisol measurements in relation to the time the cortisol measurement was taken; in the morning only (AM), in the afternoon/evening only (PM) or taken both in the morning and the afternoon (AM and PM). Moreover, we characterised the cortisol measurements as awakening or diurnal cortisol.

We conceptualised the control comparison groups as active, inactive or passive. We also followed previous meta-analyses (e.g. Koncz et al., 2021) to look at whether collapsing the inactive and waitlist groups into a larger, passive control group made a difference to understanding subgroup differences in explaining the heterogeneity of our results.

When considering the risk of bias, a large proportion of the included studies were categorised as ‘some concerns’, with six studies being ‘high risk’. As seen in Figure 14 below, the greatest risk of bias stemmed from the category ‘missing outcome data’; often due to participants dropping out of the study. There was also a greater risk derived from lack of detail in relation to the method of cortisol sampling and failure to conduct sensitivity analyses in the included studies to understand if the findings were biased by missing data. Additionally, there was a lack of clarity regarding the category ‘selection of the reported result’ where despite a standardised cortisol collection procedure being implemented, the study did not make clear whether the study personnel were aware of group allocation.

As the outcome of interest was cortisol, as measured in either saliva, blood or hair, it was essential to recognise the variability of the quality of cortisol measurement across studies and its potential impact on determining the effectiveness of interventions in the changes in cortisol. The current meta-analysis utilised the cortisol quality tool as devised by (Laufer et al., 2018), we adapted this measure to additionally be used for hair cortisol; previously this tool was used in saliva and blood only. The cortisol quality measure uncovered patterns in the cortisol sampling that may confound effectiveness of the

interventions utilised. Notably the lack of reporting of state confounders that could influence cortisol measurement, such as time of day the measurement was taken, consideration of medication or menstrual phase in female samples were the most frequent indicators of poorer cortisol sampling. See Appendix, Table 21, for a summary of the study characteristics.

5.3.2 Categorising the interventions

There was a great variety of interventions included in the meta-analysis. For the purpose of analyses, and to improve understanding of differential effectiveness of different broad types of interventions, we summarised the underlying concepts of the interventions and this allowed us to categorise each intervention into one of four broad categories to allow meaningful comparison of key intervention components (see Figure 12*).*

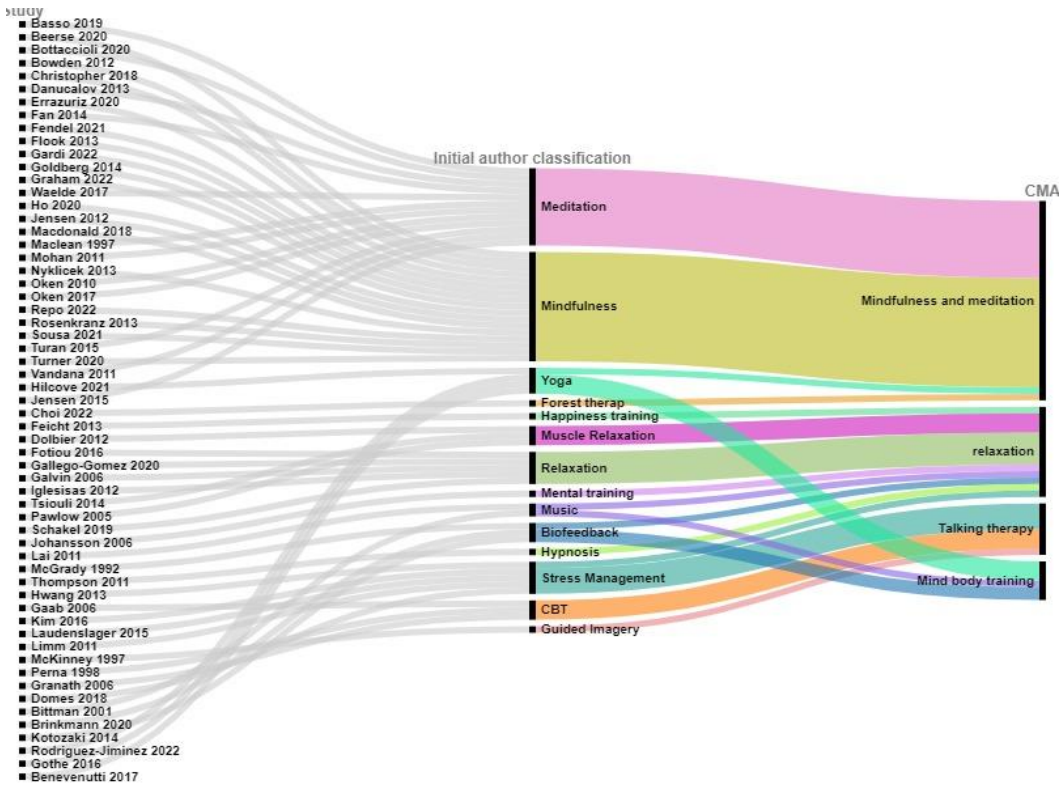


Figure 12. An alluvial diagram mapping the categorisation of study interventions.

We conceptualised four categories of intervention: 1) *mindfulness and meditation*, incorporating any mindfulness meditation, mindfulness based therapy, including mindfulness based stress reduction and mindfulness based cognitive therapy where the

central core of the intervention is to gain a greater awareness of one's physical, mental and emotional condition; 2) *talking therapies* included psychological interventions involving talking one-to-one, in a group, online, over the phone or with friends, family or co-workers, an example of talking therapy being cognitive behavioural therapy; 3) *relaxation*, included any intervention specifying muscle relaxation, biofeedback assisted relaxation and breathing exercises; 4) *mind body training*, incorporated yoga and biofeedback where there was an awareness of bodily movement to influence mental state.

5.3.3 Grand meta-analysis

This analysis is based on 58 studies that investigated the effect of stress management interventions on cortisol (as measured in blood, hair or saliva). The meta-analysis excluded one study, Danucalov et al. (2013), due to being identified as an outlier with inflated effect sizes. The grand meta-analysis found that stress management interventions led to a small-to-medium, and heterogeneous, positive effect on cortisol levels (g = 0.282, 95% CI = 0.166, 0.398, Z = 4.749, $p < 0.001$; I^2 = 60.3%, $Q_{(57)}$ = 143.603, p < 0.001) reflecting a favourable outcome for the psychological intervention compared to the control condition. See Appendix, Figure 15 for the high resolution plot of effect sizes.

5.3.4 Publication bias and sensitivity analysis

Egger's regression coefficient did not indicate presence of publication bias when all studies were considered together (see Figure 13; intercept = 1.284, df = 56, p = .082). Duval and Tweedie's trim and fill analyses indicated there were no missing studies either side of the mean. Sensitivity analyses were performed to determine the impact of removing each study from the analyses, one at a time. These analyses did not detect any studies that had a significant independent impact on the overall effect size at post-intervention (effect sizes (hedges g) ranged from 0.250 - 0.298).

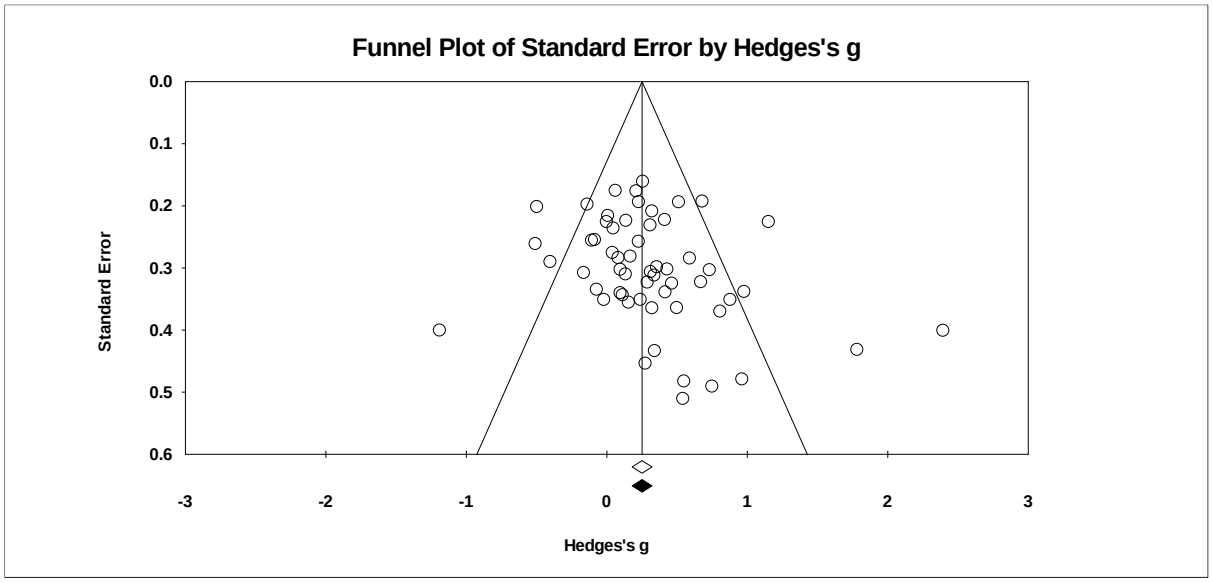


Figure 13. Funnel plot based on Hedge's g, 95% CI's for cortisol.

5.3.5 Subgroup analyses

5.3.5.1 Cortisol measurement type

To compare the effectiveness of the interventions in studies utilising different cortisol outcomes, as measured in blood, hair or saliva, a subgroup analysis was conducted. As outlined earlier, there were only 3 studies utilising hair cortisol, therefore, this category was omitted from the analysis as there were too few studies to have adequate power to conduct the analysis. There was a main effect of the interventions, when compared to controls, in blood (g = 0.331, SE = 0.136, p = .015) and saliva (g = 0.284, SE = 0.074, p < .001). However, there was no evidence that the effect sizes varied as a function of cortisol outcome measure (Q = 0.093, p = .761).

5.3.5.2 Types of intervention

We explored whether the type of intervention impacted the effectiveness of stress management interventions (see Appendix, Table 22). The interventions were grouped into one of four categories; mind body therapies, mindfulness, relaxation or talking therapies. The subgroup analysis revealed the largest, significant effect sizes for mindfulness (g = 0.345, SE = 0.085, p < .001) and relaxation (g = 0.347, SE = 0.125, p = .005). We observed much smaller, non-significant, effect sizes for mind body therapies (g = 0.129, SE = 0.187, p = .492) and talking therapies (g = 0.107, SE = 0.162, p = .510). Overall, there was no evidence that the effect sizes varied as a function of the type of intervention received (Q = 2.643, p = .450).

5.3.5.3 Comparison group

In this subgroup analysis we only included studies with *one* control group; for instance, a study that had two control groups would be excluded (e.g. Errazuriz et al., 2022). In studies where the intervention group was compared against an active control group, we observed a large, significant, effect size (g = 0.477, SE = 0.109, p < .001). In studies where the intervention was compared against a passive control group there was a much smaller, non-significant, effect observed (g = 0.129, SE = 0.076, p = .093). Additionally, the effect sizes varied as a function of the type of comparison group the intervention was compared against and was significantly different across conditions. The analyses indicated that when the stress management interventions were compared against an active control group the effect sizes were much larger and significantly different than when compared to a passive control group (Q = 6.967, p = .009). The same pattern emerged when the comparison groups were classified into active, inactive and waitlist categories (for main effects of each control group, see Appendix, Table 22).

5.3.5.4 Awakening or diurnal

Next, analyses were conducted to explore whether the effectiveness of interventions on cortisol varied based on the *type* of cortisol measure – awakening or diurnal cortisol. The analyses found a large, significant effect when studies utilised awakening measures of cortisol (g = 0.644, SE = 0.153, p < .001), and smaller, but also significant, effects when using diurnal measures of cortisol (g = 0.225, SE = 0.063, p < .001). Moreover, the magnitude of effect was significantly different in studies that assessed the awakening response compared to diurnal levels, indicating that the interventions were more effective at changing cortisol in the morning awakening measures compared to diurnal cortisol measures (Q = 6.37, p = .012).

5.3.5.5 Length of intervention

One study was excluded from this subgroup analysis as it did not provide detail on the length of the intervention (Johansson & Unestål, 2006). When considering the length of intervention, categorised as short, medium or long in length, there was a significant effect for long interventions (more than 801 minutes; g = 0.348, SE = 0.093, p < .001) as well as for short interventions (less than 250 minutes; g = 0.306, SE = 0.084, p < .001). However, no significant effect was found for medium length interventions (251 – 800 minutes; g = 0.150, SE = 0.147, p = .308). Overall, there was no significant difference on

the effectiveness of the intervention based on the length of the intervention (Q = 1.299, *p* = .522).

5.3.5.6 Study quality

We conducted subgroup analysis to determine the effect of study quality on the effectiveness of interventions on change in cortisol. For studies with moderate study quality we observed significant effects (*g* = 0.346, *SE* = 0.080, *p* < .001). However, high study quality was not significant (*g* = 0.212, *SE* = 0.130, *p* = .103) and low study quality had the smallest effect size but also non-significant (*g* = 0.195, *SE* = 0.144, *p* = 0.178). Overall, we found no difference in effect sizes based on study quality (Q = 1.272, *p* = 0.529).

5.3.5.7 Risk of bias

We explored the impact of risk of bias on the observed effect sizes. For studies with 'low risk' of bias, we observed significant effect sizes (*g* = 0.295, *SE* = 0.100, *p* = .003) and studies categorised as 'some risk of bias' observed a similar effect size (*g* = 0.303, *SE* = 0.087, *p* < .001). However, for studies with high risk of bias there were smaller, non-significant effects (*g* = 0.207, *SE* = 0.186, *p* = .267). Overall there were no significant differences in effect sizes according to the risk of bias categorisation (Q = 0.224, *p* = 0.894). A summary of the evaluation of the risk of bias across studies can be seen in Figure 14.

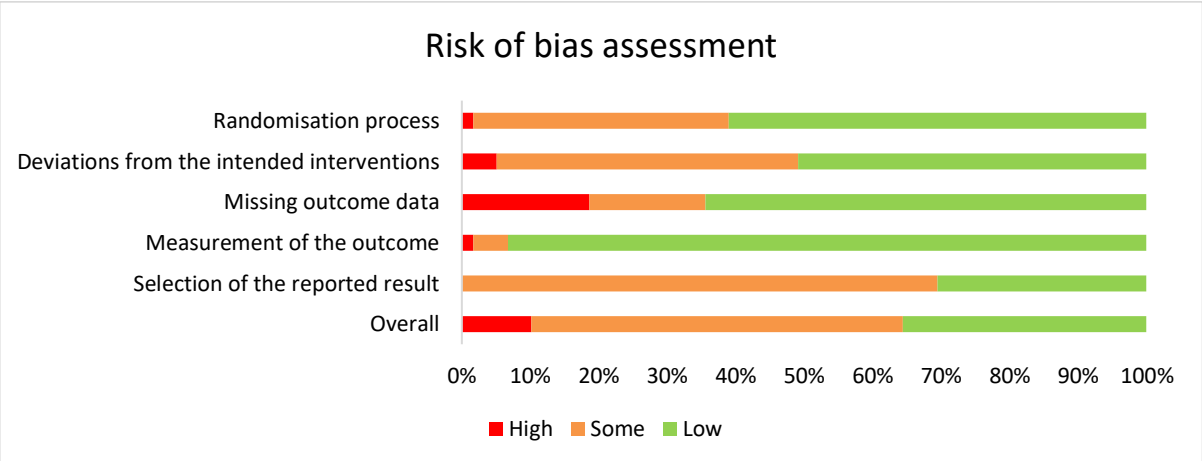


Figure 14. A summary of risk of bias across studies.

5.3.5.8 Stress risk

When considering the stress risk of the participants in the included studies, we explored whether having a 'stress risk' sample influenced the effectiveness of the interventions on

change in cortisol. We found that the interventions were effective in non-stressed samples, shown by a medium sized significant effect (g = 0.351, SE = 0.075, p < .001). However, in samples experiencing stress, the interventions were much less effective and this was shown by a smaller, non-significant effect size (g = 0.135, SE = 0.098, p = .169). Overall, there was no significant differences of the stress risk of the sample on the effectiveness of the intervention on cortisol (Q = 3.078, p = .079).

5.3.6 Meta-regressions

5.3.6.1 Time elapsed between end of intervention and cortisol measurement

This analysis was conducted on the 45 studies which provided detail on the time elapsed between the end of the intervention and post-intervention cortisol measure. There were no significant relationships between the time elapsed after the intervention and post-intervention measure (B = -0.0002, SE = 0.001, *95% CI* [-0.002, 0.001], p = .734).

5.3.6.2 Demographics

When considering whether the total number of participants included in the study influenced the observed effect sizes, there was no significant effect of total sample size on the observed effect (B = -0.002, SE = 0.002, p = .273). Second, when considering the demographics of the samples, the meta-regressions were conducted on the 28 studies which reported the demographics for the participants providing cortisol samples, as opposed to the total study sample. There was no significant effect of age (B = 0.012, SE = 0.074, 95% CI -0.0025, 0.0264]. p = .1048) or gender (B = .0002, SE = 0.0029, p = .955) on the effect sizes of the observed studies.

5.4 Discussion

The current systematic review and meta-analysis explored the effectiveness of stress management interventions in changing cortisol levels and considered moderators influencing the effectiveness of the interventions. There was clear evidence that stress management interventions had a positive effect in improving cortisol levels from pre- to post-intervention. The review was comprehensive; considering healthy individuals with no reported pre-existing health conditions, yet inclusive of samples that may experience periods of short- or long-term stress where it is imperative to have effective stress management interventions. Previous reviews of the effectiveness of stress management interventions on cortisol levels have focussed on a singular form of intervention, such as

meditation (Koncz et al., 2021). However, a plethora of stress management interventions exist and the effects of these interventions could vary. In the current review and meta-analysis we considered the array of interventions available to reflect the heterogeneity of stress management interventions, aiming to provide a more comprehensive overview of the effects of stress management interventions on cortisol levels.

The current meta-analysis acknowledges potential moderating variables influencing the effectiveness of stress management interventions, such as: cortisol sampling strategies (diurnal, awakening), cortisol outcomes (blood, hair, saliva), control conditions (active, passive), quality of cortisol measurement, risk of bias within studies and sample demographics. Specifically we found that mindfulness and relaxation interventions appeared most effective at changing cortisol levels. We also found interventions that compared against an active control group, rather than a passive control group, were also more effective at reducing cortisol levels. This is consistent with previous literature whereby mindfulness-based interventions were slightly superior to other active controls in adults when analysing a variety of health outcomes, including stress (Goldberg et al., 2022). Additionally, studies that measured awakening cortisol revealed greater effectiveness of interventions in changing cortisol levels than those measuring diurnal cortisol. However, the type of intervention, length of the intervention, study quality, and risk of bias did not appear to influence the effectiveness of interventions. The findings emphasise the need to recognise the diversity of interventions, and cortisol measurement, especially when interpreting the disparate findings observed in previous literature regarding the success of stress management interventions.

To our knowledge, this is the first meta-analysis to compare the effectiveness of different types of interventions for cortisol changes from pre- to post-intervention in a single statistical model. It is apparent from our analysis that there is no clear indication of one intervention being *more* effective than another intervention when directly compared, per se. However, we can conclude that meditation and mindfulness and relaxation were the only statistically significant effective interventions and yielded the largest effect sizes. It is worth noting that meditation, mindfulness and relaxation studies also represent the largest study groups and generally were longer interventions, therefore, we cannot rule out the possibility that as the number of studies increase, that these conclusions may need to change. Nevertheless, the question remains as to what is the underlying driver of these differential findings? Is it the intervention content, length, delivery, or sample size that is driving the observed effects. The current findings provide further evidence for the

effectiveness of mindfulness and meditation-based interventions. For example, a recent meta-analysis found that mindfulness-based interventions had beneficial effects on cortisol in healthy adults but also recognised the heterogeneity in delivery of studies and what is the true driver of the effect (Sanada et al., 2016). These congruent findings open opportunities to understand the extent to which third wave interventions that include mindfulness, such as Acceptance Commitment Therapy, could influence cortisol levels (Prudenzi et al., 2021). Further research is needed to understand the nuanced effects of different interventions.

Contrary to expectations, the results of this meta-analysis suggest that stress management interventions were more effective when compared to active controls, as opposed to passive controls. There are several possible explanations for this pattern of results. One possibility is that the studies with active control conditions were of higher quality and this was reflected in enhanced intervention delivery and fidelity leading to improved outcomes. Of course, the converse may also be true, the studies with passive control conditions may have had inferior intervention delivery and fidelity. This finding is somewhat surprising when considering previous meta-analyses found studies with inactive controls had larger effect sizes than active (Witarto et al., 2022) and larger magnitude of effects specific to mindfulness interventions were when compared to passive controls, with smaller, yet still significant effects when compared to some active controls (Goldberg et al., 2022). Nonetheless, it is noteworthy that only with an appropriate active control group can we attribute differential improvements to the potency of the stress management intervention and it is a more rigorous test of intervention efficacy as to whether these interventions should be considered for stress reduction. For example, if an active control group receives an evidence-based intervention, then we can be more confident that the change in cortisol levels seen in the stress management intervention group is due to the specific components of that intervention, rather than simply the fact that participants were receiving any intervention at all. Additionally, unless the design of the study is a double-blind design, the true effectiveness of an intervention cannot be concluded (Boot et al., 2013). Future research should use appropriate active control groups and double-blind designs to more accurately assess the effectiveness of stress management interventions.

The meta-analysis also found interventions to be *more* effective in “no stress” risk samples than in “stress risk” samples; contrary to previous research only yielding significant effects for stress risk samples, with no significant effects in non-stressed

samples, when considering blood cortisol (Koncz et al., 2021). However, the lack of statistical power in previous research meant that direct comparisons between stressed and non-stressed groups could not be conducted. The current study was able to conduct analyses to directly compare stress and no stress risk groups, finding no statistically significant differences in intervention effectiveness depending on whether participants were at stress risk or not. It is also important to recognise that the "stress risk" grouping in the Koncz et al. (2021) review differed slightly from the stress risk samples in the current meta-analysis. For example, the stress risk groups in previous research included low-income family members, dementia caregivers, cancer survivors or cancer patients, while the stressed samples in the current meta-analysis were comprised of caregivers, healthcare workers, and people who reported prolonged stress. The key difference being the current meta-analysis did not include anyone with a diagnosed somatic or mental illness. These differential findings are difficult to reconcile and highlight the need for more careful consideration of how samples are classified as stress risk versus no stress as this may not be a useful arbitrary distinction. It is likely there is a large amount of variability within and across groupings and samples. Future research ought to consider this issue further.

The current meta-analysis also found stronger evidence for intervention effectiveness when studies utilised the cortisol awakening response compared to a diurnal cortisol measure. The smaller effects for diurnal cortisol measures highlight potential divergence in the sensitivity of different diurnal cortisol indices to training effects. The diurnal cortisol measures were still significantly influenced by interventions, although the effects being smaller could be due to one of many factors such as the varied and inconsistent quantification of diurnal cortisol utilised, differences in the number and timing of daily samples across the day as well as variation in daily lifestyle factors. Whereas cortisol levels measured after awakening may be less confounded by the diverse influences of the day (e.g., food intake, exercise), and thus are less 'noisy' measures (Engert et al., 2023). It could be said that if studies were better controlled and quality checked, different effects may emerge. Although, when conducting further analysis we did not find any significant difference in intervention effectiveness based on study quality, nor a relationship between study quality and type of cortisol measurement.

Two quality assessment tools were used, the RoB2 and a cortisol quality assessment tool (Laufer et al., 2018; Sterne et al., 2019). Determining the quality of the cortisol measurements in the included studies was imperative to consider because the methods of

cortisol collection are likely to impact study findings of intervention effectiveness (Adam et al., 2017). It is apparent from the study quality assessments that studies lack true consideration of state covariates such as time of day, psychotropic medication, oral contraceptives and somatic disease and there is room for improvement in this area especially considering these factors greatly influence cortisol measurement (Stalder et al., 2016). We found studies with the poorest study quality, and greatest risk of bias, to have the smallest effect sizes and these main effects were not significant suggesting that poorer controlled studies fail to determine the true effectiveness of stress management interventions. However, there were no significant differences between categories of study quality or risk of bias groups; this could be attributed partially to the heterogeneity of the sampling procedures across the included studies. Nevertheless, the current findings highlight the importance for researchers in this area to ensure that their intervention studies are designed to be of the highest quality in order to robustly and accurately test the effectiveness of their interventions.

There are inevitable shortcomings to any research including the current meta-analysis. First, due to the heterogeneity of the included participant samples, psychological interventions and cortisol measurement procedures; there was a great variety in frequency, timings, procedures and measures of cortisol which may have caused further confounding of the true effectiveness of the included interventions. Second, we recognise the small number of hair cortisol studies available in the current meta-analysis which prevented us from comparing effectiveness of the interventions against studies that utilised blood and saliva samples. The studies utilising hair cortisol are more recent publications, possibly represent better controlled studies and it is hoped that further research continues to utilise this measurement parameter in the future. Third, when categorising the stress management interventions it is inevitably vulnerable to a degree of subjectivity therefore it may be that others may consider the interventions to reflect different intervention mechanisms. However, we ensured a second screener independently categorised a proportion of the interventions and reached consensus with the first reviewer prior to categorisation. Lastly, the scope of the review focussed exclusively on healthy participants which limits our conclusions to a degree. Future research is needed to confirm these findings and to identify the most effective interventions for reducing cortisol levels stratified by different populations.

Overall, the current systematic review and meta-analysis found a positive effect of stress management interventions on cortisol, with robust conclusions for blood and saliva

cortisol. Interventions were more effective when compared to active control groups than passive control groups and more effective at changing the cortisol awakening response measures compared to diurnal cortisol measures. There was no significant difference in the effectiveness of interventions based on the type of cortisol measurement (blood, saliva, or hair) nor for the length of the intervention. Mindfulness and relaxation interventions were found to be most effective yielding the largest effect sizes, while mind body therapies and talking therapies were shown to have smaller and non-significant effect sizes. The current findings confirm that stress management interventions can positively influence cortisol levels. Future research should investigate the longer term implications for health and health outcomes.

Chapter 6 - General discussion

6.1 Thesis aims and summary

In this discussion chapter the findings from the empirical research studies (study 1, 2 and 3) and the meta-analysis (study 4) will be discussed in context of the overall thesis aims. The strengths and limitations of each study will also be considered, as well as outlining the potential practical and clinical implications of the research presented in the thesis. The avenues for future research will be outlined and overall conclusions will be presented.

6.1.1 Overall discussion

It is well-established that early life adversity, including childhood trauma, can have long-lasting, negative, impacts on mental and physical health outcomes in adulthood (Danese & McEwen, 2012; Fergusson et al., 2013; Hughes et al., 2017; Noteboom et al., 2021). There is contributing evidence that cognitive functioning (e.g. executive function and impulsivity) is a central mechanism through which childhood trauma has a negative impact on mental and physical health in adulthood (see Oshri et al., 2018; Trossman et al., 2021). The central neurobiological systems that are influenced by childhood trauma (e.g., neural, endocrine, and immune systems) are also those that are adversely affected by stress and suicide (Wiebenga et al., 2022). However, much less is known about the precise mechanisms that may link childhood trauma to stress-related processes and suicidal behaviour. This thesis attempted to shed light on this knowledge gap. It provides evidence that executive functioning and impulsivity are central and important mechanisms through which early life adversity has a negative impact on mental health, through stress-related processes, to later impact suicidal behaviour.

There have been various mechanisms put forward connecting childhood trauma to later negative health outcomes. Previous research has shown that childhood abuse history significantly increases inflammatory responses to daily stressors in adulthood (Gouin et al., 2012). The model by Lovallo (2013) theorises that childhood trauma may have damaging effects on health and well-being by causing modifications in frontolimbic brain functioning which may have the capacity to lead directly to reduced stress reactivity and altered cognition, such as impulsive behaviours and a focus on short term goals. More recent work has found that childhood trauma is also associated with HPA axis dysregulation in adulthood (O'Connor et al., 2018, 2020a). Furthermore, a meta-analysis of longitudinal studies revealed that experiences such as sexual and emotional abuse, as

well as physical neglect, were associated with suicide attempts later in life (Zatti et al., 2017). Additional research indicates that approximately 80% of individuals who have attempted suicide reported experiencing at least one moderate to severe form of childhood trauma (O'Connor et al., 2018). Therefore, the thesis examined the effects of childhood trauma on mental health outcomes in a sample that included participants who had a history of suicide thoughts and behaviours (study 1 and 2) and then to attempt to replicate the findings in a more generalisable sample who were considered healthy with no history of suicidal behaviour (study 3).

The overarching question of this thesis was to understand why are individuals who experience childhood trauma at increased risk of later experiencing suicide thoughts and behaviours? The thesis sought to first identify mechanisms that linked childhood trauma to stress and suicide, then to understand how these mechanisms interact. Across the series of research studies conducted, there was evidence to support that childhood trauma impacted both stress and later suicidal behaviour through maladaptive cognitive functioning. In two of the research studies, there was consistent evidence that childhood trauma was associated with increased risk of reporting recent suicidal ideation and suicide attempts (study 1 and 3). The present thesis demonstrated that childhood trauma was associated with poorer executive function and greater impulsivity using a cross-sectional methodology (study 1) and that childhood trauma had an indirect relationship on daily defeat, daily entrapment, daily optimism and daily suicide thoughts through daily executive functioning and impulsivity, using EMA methodology (study 3). This is consistent with Tinajero et al. (2020) who also found that childhood abuse was significantly associated with poorer daily reported executive function and Blackwell (2022) who found that emotional abuse was significantly associated with greater daily impulsive behaviours. Taken together, this is particularly concerning as disrupted cognitive functioning in turn has been linked with numerous negative health outcomes (e.g. Halse et al., 2022). It is also important to consider how stress may influence the aforementioned relationships. During periods of increased stress, such as the COVID-19 pandemic, stress appears to be strongly linked to poorer executive functioning (study 2). Together, the three empirical studies show that both stress and childhood trauma negatively impact executive function. Furthermore, executive function appears to be a mechanism through which trauma increases the risk of experiencing known suicide risk factors and suicidal thoughts. This suggests that stress is not only detrimental to executive function, as previous research suggests (Shields et al., 2015), but that individuals who

experience trauma may also be at greater risk of poorer executive function. Future research should continue to explore the complex associations between childhood trauma, executive function, and suicide in the context of stress, in order to better understand the nuances with the individual facets of executive functioning. Overall, these findings provide further evidence that childhood trauma may have long-term effects on health by influencing daily stress-related processes.

The thesis also sheds light on how prolonged stress can impact later suicidal behaviour. To our knowledge, the thesis reports one of the first longitudinal studies examining how risk factors for suicide interact during a national lockdown (study 2). The findings highlight the associations between suicide vulnerability, impulsivity, and poorer executive functioning during the COVID-19 pandemic, a period of intense stress, as well as emphasising the potential exacerbating effects of daily COVID-related stress and worry on impulsive behaviours and executive function. There was consistent evidence, aligning with study 1, that history of suicide was associated with greater impulsivity and poorer executive function, during the initial lockdown period, suggesting a history of suicide may put these individuals at a greater risk of negative consequences from the pandemic. This suggests potential poor cognitive functioning of information processing and response, especially in stressful time periods. With the lockdown announcement in the UK on the 24th March 2020, there were growing concerns over the impact of how the protection of physical health comes with the potential detrimental effects to mental health, especially in vulnerable groups including key workers (Holmes et al., 2020). This study therefore contributes to the body of literature that began to understand the short- and medium-term psychological impacts of COVID-19. As well as being one of the first longitudinal studies, to our knowledge, that provided evidence for how risk factors for suicide interact throughout the course of a lockdown in a global pandemic.

Surprisingly, there was an absence of evidence of moderation effects by key variables throughout the research studies in the thesis. History of suicidal behaviour did not moderate the associations between COVID-related stress, worry, and rumination, with impulsive behaviours and executive functioning (study 2). It could be the case that recent suicidal behaviour may have moderated the relationship as opposed to lifetime history. Moreover, there were no moderating effects of the relationship between childhood trauma and daily stress-related variables (e.g. daily measures of stress, hassles, executive

functioning, impulsivity, sleep quality) by any of the protective or risk factors (resilience, social support, social connectedness, loneliness, and suicide history) (study 3). Similarly, recent stress did not moderate the relationships between childhood trauma and executive functioning, impulsivity, recent suicidal ideation, or wellbeing (study 1). In this instance, it could be that a mediated pathway exists whereby childhood trauma impacts stress-related processes which then influences known suicide risk factors. If a mediated pathway were to exist, it could likely be a bi-directional relationship between stress and suicide risk factors, e.g. a reinforcing cycle wherein heightened stress levels exacerbates suicide risk factors, while those same risk factors may amplify an individual's susceptibility to stress, creating a complex interplay that perpetuates the likelihood of suicidal behaviour. Further research needs to investigate this possibility and further elucidate the possible complex and nuanced pathways through which childhood trauma influences mental and physical health. Another reason for the lack of moderation effects may be the complexity of the relationships being studied. It is possible that the variables examined in this research interact in intricate ways that were not captured by the analyses conducted. Despite this, the thesis contributes valuable insights into understanding the mechanisms associated with increased suicide risk in individuals who have experienced increased life stress, including childhood trauma. Moving forward, researchers face the challenge of elucidating how these factors interact over time and developing interventions to target vulnerability factors affected by childhood trauma to mitigate suicide risk in adulthood.

From the literature, and the research in the current thesis, it appears imperative to gather a more nuanced understanding of the day to day processes by which executive function and impulsivity relate to other negative health behaviours. The reason being is that it has been suggested that both executive functioning and impulsivity are transdiagnostic risk factors, that when impaired, can affect a broad range of mental health problems (Berg et al., 2015). Previous research findings converge to suggest that various mental health disorders, despite their phenotypic diversity, may stem from a shared factor or susceptibility to dysfunction, which correlates with broad neurocognitive impairments, including both executive function and impulsivity (Berg et al., 2015; McTeague et al., 2016; Snyder et al., 2015). Executive function impairments are associated with most forms of pre-psychopathology, including high levels of rumination, worry, and emotion dysregulation (Aldao et al., 2010; Snyder et al., 2015), all of which are risk factors for various forms of psychopathology such as depression and anxiety (Lynch et al., 2021; McLaughlin & Nolen-Hoeksema, 2011). It is well documented that executive functioning

is transdiagnostically associated with both the mental health disorders generally associated with childhood trauma, as well as having strong associations to childhood trauma itself (McTeague et al., 2016; Op Den Kelder et al., 2018). Further research into transdiagnostic factors would allow the exploration of commonalities across psychopathologies to better understand the contributing mechanisms to suicidal behaviour, as well as allow for better targeted interventions that target general processes instead of domain-specific symptomology.

The current research in the thesis found childhood trauma to be associated with a number of daily stress-related processes in adulthood. In Study 3, childhood trauma was significantly associated with higher scores on the daily stress-related vulnerability factors. Also, trauma had indirect impact on feelings of defeat, entrapment, reasons for living, optimism and even feelings of suicide through stress. The findings are concerning given the well-established effects of stress on HPA axis regulation and cortisol dynamics, on the nervous system and other health outcomes including chronic illnesses and suicide risk (D.B. O'Connor et al., 2021a).

One of the key elements of this thesis was the systematic review and meta-analysis (study 4) that examined the effectiveness of psychological interventions to improve cortisol levels in healthy adults. The findings of the study provide clear evidence that stress management interventions had a positive effect in improving cortisol levels from pre- to post-intervention. Specifically, interventions that were compared against an active control group, rather than a passive control group, were more effective at reducing cortisol levels. This is consistent with previous literature whereby mindfulness-based interventions were slightly superior to other active controls in adults when analysing a variety of health outcomes, including stress (Goldberg et al., 2022). Additionally, intervention studies that measured awakening cortisol were found to be more effective in changing cortisol levels than those measuring diurnal cortisol. However, the type of intervention, length of the intervention, study quality, and risk of bias did not appear to influence the effectiveness of interventions. These findings highlight the importance of assessing the different components of the diurnal cortisol profile before drawing firm conclusions about the effectiveness of various stress management interventions. Moreover, future research ought to explore whether stress management interventions can mitigate the effects of childhood trauma on daily stress-related processes and prevent the transition to longer-term negative effects on health.

Overall, the thesis considers the findings in light of the IMV model of suicidal behaviour and broadly supports this theoretical model (O'Connor & Kirtley, 2018). The IMV acknowledges there is a scenario whereby stress regulation, alongside cognitive functioning, could form a pathway by which childhood trauma increases the risk of suicidal behaviour through these mechanisms. The current findings are consistent with this proposed pathway and support the theoretical importance in understanding the impact of childhood trauma in development of later mental health difficulties, particularly concerning its relationship with the IMV model of suicidal behaviour (O'Connor & Kirtley, 2018). We can confirm that childhood trauma significantly influences various facets of psychological functioning, including cognitive processes, stress regulation, and emotional regulation, all of which are central components of the IMV model. Throughout the thesis there is evidence for this pathway through cognitive functioning to later suicidal behaviour. By elucidating the pathways through which childhood trauma influences psychological functioning, particularly within the framework of the IMV model, we are able to enhance understanding of the mechanisms underlying suicidal behaviour. This understanding has potential implications for suicide prevention and intervention efforts, highlighting the importance of addressing trauma-related vulnerabilities and promoting stress management techniques in individuals at risk for suicide. The results highlight the possibility for psychological interventions to include a component which focuses on factors such as stress, executive function, and impulsive behaviours to mitigate the negative effects from stressful life events. These approaches are in keeping with the majority of models of suicidal ideation and behaviour, which place the experience of stressful life events as the initiating stressor or precipitating event, as well as focus on the interpretation of these events by individuals.

6.2 Strength, limitations and avenues for future research

6.2.1 Strengths

Understandably, there are likely important variations in stress, executive function and impulsivity that contribute to the development of suicidal ideation that would be missed in a traditional cross-sectional study. A way to capture short-term variations in stress-related vulnerability factors is to measure these at a momentary level using techniques such as EMA methods, allowing data to be collected virtually in real-time. The meta-analysis by Franklin et al. (2017) highlighted that suicide research often misses important variations in suicidal behaviour as the studies often have long follow ups, and that longer

follow up does not necessarily improve predictive ability. Therefore, a strength of the current thesis was the adoption of a multi-methodological approach, avoiding reliance merely on cross-sectional methods. Utilising a variety of methodological techniques such as longitudinal designs, EMAs, and meta-analytic techniques allowed for comprehensive exploration and validation of findings across different research methodologies, ensuring confidence in the inferences drawn. Leveraging diverse methodologies across research studies offers a means to mitigate inherent limitations associated with individual approaches. If one experimental design is limited in a particular way, complementing with another approach that is stronger in that aspect (but perhaps limited in another) can provide a more complete picture. This also implicitly acknowledges that scientific rigor does not proceed only from the single study; convergent evidence may proceed from a multitude of methods. For instance, the longitudinal methodology permitted the understanding of changes in, and the impact of stress on, known suicide risk factors (study 2) whereas the EMA design captured within-participant variations and explored the relationship of *daily* stress-related vulnerability factors and suicide risk (study 3). Additionally, the meta-analytic approach in the current thesis offers several benefits, including increased statistical power, the ability to detect small effects, and the ability to assess the consistency and generalizability of results across multiple studies (study 4). As well as allowing for the exploration of potential moderators and provides more precise estimates of effect sizes, thus strengthening the overall conclusions drawn from the research.

Lastly, it is noteworthy that the studies presented in this thesis were all preregistered, with the resulting data made readily available through open repositories. Through preregistration, researchers outline the specifics of their confirmatory hypotheses, thus distinguishing them from exploratory hypothesis-generating endeavours. While sharing results from exploratory work remains crucial for scientific advancement, presenting such findings as if they were confirmatory misrepresents the scientific process (Bosnjak et al., 2022; Munafò et al., 2017). To highlight, study 1 was a registered report which, to our knowledge, represents the first registered report in this research area. By conducting research using registered reports it not only improves rigor, but also reduces publication bias and increases the transparency of science by allowing peer review of research studies before results are known (O'Connor, 2021). This publication format mitigates the use of questionable research practices and helps to reduce the 'file drawer' effect. Indeed, an evaluation of 113 registered reports found that only 39.5% of primary hypotheses were

supported, whereas, between 80% and 95% of primary hypotheses were supported in a random sample of studies using the traditional publication format (Allen & Mehler, 2019). Moreover, similar findings were reported in a comparison of 71 published registered reports with a random sample of 152 hypothesis testing studies from the traditional literature (Scheel et al., 2020). This study found 44% positive results in the registered reports compared to 96% in the traditional literature. Therefore, the use of these approaches throughout the thesis aimed to augment the quality and reliability of the findings in the realm of suicide research and to enhance the transparency and accessibility of the work to the wider scientific community. Consequently, this thesis not only furthers our understanding of suicidal behaviour but also stresses the importance of rigorous research practices in contributing to collective scientific knowledge.

6.2.2 Limitations

All the studies included in the current thesis employed quantitative methodology to help understand the pathways between childhood trauma and suicide. The current thesis has clearly mapped how these mechanisms interact and proposed pathways. However, now that the mechanisms are identified it would be beneficial to utilise qualitative or a mixed-methodological approach that would permit a deep dive investigation of the nuances of individual experiences of suicide risk factors over short term and long term time periods. Quantitative methods may have difficulty to capture the complexity and interplay of these factors adequately. This limitation could result in oversimplified models of suicide risk and prevention strategies that fail to address the diverse needs of at-risk populations. As such, conducting qualitative explorations would be beneficial to validate the current thesis findings and ensure theoretical models are reflective of these developments.

Another limitation relates to study 2. This study had no true baseline, the first measurement was taken 1 week into the first UK lockdown, reflecting the window of time between 1-week pre-lockdown and 1-week into the first lockdown, which may have already increased individual stress and impacted wellbeing prior to the first measures being administered. Given the unprecedented speed at which the disease spread, it was not possible for the research to include appropriate baseline measurements prior to lockdown. Without an appropriate baseline, there is not sufficient information to attribute any findings to lockdown specifically because participants might have felt the same during a ‘typical’ six week period (although, this is unlikely). However, it should be noted

that the measures all had normative data therefore allowing comparisons to be made with scores from the pre-COVID period.

It is important to acknowledge that childhood trauma assessment relies on retrospective self-report measures and due to the time in which these events occurred many years ago, it may be that this is susceptible to social desirability biases and memory distortions which may impact on the reliability of the CTQ. Nevertheless, it is noteworthy that scores on the CTQ align with expectations, consistently showing higher scores in suicide risk groups across various studies, consistent with prior literature (O'Connor et al., 2021b). If anything, retrospective self-report tools may underestimate the true prevalence of childhood trauma (Hardt & Rutter, 2004). It is believed that false negatives are more prevalent than false positives in reporting past traumas, potentially leading to an underestimation of childhood trauma in the current research (Hardt & Rutter, 2004). Overall, self-report measures rely on the perception of oneself in relation to other people and these self-perceptions may sometimes be erroneous. Although, it is unavoidable in many instances to not assess childhood trauma using self-report measures. Not all occurrences of childhood trauma are reported at the time of occurrence to healthcare or social services. Therefore without utilising self-report measures we would simply not determine the true impact of childhood trauma on later health difficulties in the population.

It is also recognised that there is no gold standard measure of state executive functioning. There are questions surrounding the extent to which self-reported executive function measures reflect objectively measured executive problems. In some literature, there are documented instances where individuals have poorer executive functioning on performance based measures, yet their self-reported measures of executive function do not align and perhaps they measure different constructs entirely (Barkley & Fischer, 2011; Buchanan, 2016; Keen et al., 2022; Toplak et al., 2013). This questions what factors may influence response on these self-report measures. Some researchers have suggested that, in non-clinical samples especially, these self-report measures may not be suitable as proxies for actual executive functioning performance based tests (Buchanan, 2016). It is possible that personality may be a factor influencing self-reports of executive function as it has been found that self-reported executive function measures are negatively associated with neuroticism and conscientiousness (Buchanan, 2016). However, there are strong arguments for the use of self-report measures of trait and state executive function (Johnco et al., 2014), as well as several self-report measures that exist, including the DEX (Wilson

et al., 1996) and the WEBEXEC (Buchanan et al., 2010). In the current research we found that state and trait measures of executive function were correlated (Study 2, Study 3) and we argue that having a brief measure of executive function is invaluable for researchers looking to assess executive function in survey research. Most importantly, the self-report measure used in the current research has been shown to be reliable and valid (Buchanan et al., 2010).

EMA techniques offer a unique opportunity to explore dynamic psychological processes while mitigating recall bias and enhancing ecological validity by capturing participants' behaviours in their daily lives (Myin-Germeys et al., 2018; Trull & Ebner-Priemer, 2014). Although these methods present numerous benefits, they also entail complex challenges that demand meticulous planning and decision-making from researchers. A concern arises regarding the measurement of psychological constructs through EMA, as there exists inherent flexibility in daily measurements, which can lead to unforeseen issues. While comprehensive trait measures are suitable for in-depth evaluations, relying on single items for immediate reporting of current feelings poses challenges. This practice introduces potential psychometric consequences, such as item omissions or modifications, as well as inconsistencies in item usage across studies. The limited availability of psychometrically validated items is recognised as a limitation in current EMA research in this domain (Murray et al., 2023). This could contribute to a wider issue in the EMA literature whereby there is no tendency for researchers to agree on measures over time which opens up to the jingle fallacy whereby some measures actually quantify different things but share similar labels, as well as the jangle fallacy where other measures quantify the same thing as existing measures but under a different label (Gonzalez et al., 2021; Kelley, 1927). In the current thesis, both study 2 and study 3 had a series of single item measures developed to minimise participant burden. Despite the psychometric consequences being unknown in some instances and that single item measures have received criticism with concerns around measurement error in the past, recent theorising has contested this notion, presenting compelling arguments supporting the utilisation of single-item measures given their face validity, criterion validity, predictive validity and concurrent validity (see Allen et al., 2022). Moreover, some measures such as the worry and rumination measures (see Clancy et al., 2020; O'Connor et al., 2023) and COVID-19 measures (Wilding et al., 2022) have been used in wider literature finding that the former have good face validity, criterion validity, predictive validity and concurrent validity. Especially for the EMA measure of impulsivity, the MIS (Tomko et al., 2014), is

validated that impulsive states can be reliably measured with strong convergent, divergent and person-level criterion validity (Halvorson et al., 2021). Lastly, the recent development of the EMA item repository aims to address this issue by providing templates and tutorials for study pre-registration, as well as facilitating the citation and funding of efforts to psychometrically validate items within the repository (Kirtley et al., 2020). This ongoing discussion underscores the importance of grounding the findings of the present thesis within this evolving landscape.

A final limitation pertains to thinking about the sample sizes recruited throughout the thesis. For study 3, the sample size might be perceived as relatively small when compared to large-scale epidemiological studies of suicide. However, within the realm of experimental research in this domain, this sample size is relatively large. Furthermore, it incorporates the advantages associated with employing a within-participants, daily diary design, such as allowing multiple observations and utilising each participant as their own control. The within-person perspective of the study is an asset, as most theories in mental health describe within-person processes, but are rarely tested in studies with sufficient time frames to observe such developments.

6.3 Avenues for future research

Research should continue to identify and explore mechanisms aimed at improving the understanding of the relationship between childhood trauma and suicidal ideation and behaviours. In particular, research ought to explore further the potential buffers to the long-term damage imposed by trauma during childhood, which could be targeted clinically. By monitoring risk and protective factors over prolonged periods, the temporal and causal relationships between study variables may be established (Tucker et al., 2015). Ideally, future research could adopt a life span approach, where children are monitored through to adulthood to allow for conclusions regarding causality to be confirmed. Life-course studies are essential to further our understanding of the short and long-term effects of childhood experiences. This would help understand the developmental pathways that play a role in explaining the relationship between childhood trauma and suicide vulnerability in adulthood, utilising child and adolescent samples. Moreover, suicide research needs to examine complex relationships between suicide predictors. For example there are suggestions of taking this one step further; with a growing interest in machine learning to detect a wide range of associations, amongst a great number of factors, algorithms to predict suicidality may be the next step for suicide research (Walsh et al., 2017).

Future psychological interventions for individuals vulnerable to suicide ought to consider targeting changes in executive functioning and impulsivity. Previous research suggests that a variety of interventions can improve executive functioning in children aged 4 – 12 years old (Diamond & Lee, 2011). Executive functioning interventions from a younger age may help mitigate transition to suicide risk, but whether better executive functioning could become a protective factor against suicidal behaviour is yet to be determined. Future research is needed to explore whether global or individual constructs of executive functioning relate to suicidal behaviour so interventions can target the most relevant constructs and thus be most effective alongside exploring the relationships with performance based measures of executive function. This is supported by the previous research which states if an intervention is not successful in addressing the executive functioning problems in adolescence, it is possible that over time, some adolescents who experience attentional impairments go on to engage in more lethal suicide attempts (Sommerfeldt et al., 2016).

The current thesis is based upon the IMV (O'Connor & Kirtley, 2018). Yet, the psychological models of the pathways to suicidal ideation and behaviours would benefit from considering the body of literature that looks at fluctuations in, and temporal relationships between, daily stress-related variables and suicide risk over time spans of hours, in addition to the follow-up time points of months often used in prospectively designed studies. Alternative theoretical accounts may be needed to understand the mechanisms that lead to short-term proximal changes in suicidal ideation over the course of hours, such as it has been suggested that the association between entrapment and suicidal ideation is moderated by variables such as interpersonal factors and resilience (O'Connor & Kirtley, 2018). Future research should consider whether there are any moderating effects to short-term changes in suicidal ideation.

It is essential that the field of suicide research recognises and utilises the importance of EMA methodology; recent research utilising EMA found that suicidal ideation varies substantially in short time frames of hours and days (Hallensleben et al., 2018; Kleiman et al., 2017). With up to 36% of the variance in passive and active suicidal ideation being due to within-person variability (Hallensleben et al., 2019). The integration of real-time, repeated assessments in research offers potential clinical and practical utility; identified variables could be potential intervention targets. Based on the current study, it could be that interventions targeting the daily stress related vulnerability factors may be key targets, with the knowledge that daily stress related vulnerability factors have an indirect relationship with suicidal ideation could have potential to indirectly target suicidal ideation. If poorer executive functioning, or increased impulsivity, entrapment or defeat could be considered indicators for the emergence of suicidal ideation, interventions could be developed to contact individuals when entrapment levels feel overwhelming or uncontrollable for them, in order to prevent development to suicidal ideation (Kelly et al.,

2012; van Ballegooijen et al., 2022). Different theoretical frameworks might be necessary to understand the processes driving short-term fluctuations in suicidal ideation within hourly intervals (van Ballegooijen et al., 2022).

It would be beneficial if future research considered incorporating important bio-markers of suicide risk, such as cortisol levels, in order to help understand the interplay between the central components of the IMV model and related biological mechanisms. Adopting a psychobiological approach to understanding the complex interplay of factors from childhood trauma to suicide in relation to the IMV would permit this exploration. A growing body of research shows a link between dysregulation of the HPA axis and suicidal behaviour (Melhem et al., 2016; O'Connor et al., 2020a). Recent findings by O'Connor et al. (2020a) suggest that individuals with increased vulnerability to suicide exhibit notably lower total cortisol awakening response (CAR). Within this framework, researchers face the significant challenge of comprehending the factors that could both foster and mitigate HPA axis dysfunction. For instance, it has been found that childhood trauma has an indirect effect on suicidal behaviour through lower daily CAR levels (O'Connor et al., 2020a) and that higher levels of trait impulsivity were associated with lower total CAR (D.B. O'Connor et al., 2021b). Knowing that dysregulation of the HPA axis, as measured via cortisol, is associated with suicidal behaviour and that childhood trauma is an important distal factor also associated with HPA axis dysregulation, future research is needed to elucidate how CAR changes over time, as well as how these changes may interact with childhood trauma and suicide risk. It is therefore essential for future research to utilise cortisol measurements to better understand the trajectory and transition from childhood trauma to suicidal behaviour.

The next step for research is to integrate cortisol measures as part of EMA methodology to understand the intricacies between daily life stressors and cortisol. This could ultimately support the identification of stressors causing physiological stress reactions in daily life and contribute to the prediction of disease risk and the development of tailored interventions based on the profiling of risk (Weber et al., 2022). It is integral that methodological considerations are the forefront of EMA study design to truly understand the role of cortisol in the associations under investigation. Following recommendations that both stressors and physiological reactions are to be considered concurrently under dense temporal resolution with at least three measurement points per day instead of multiple cortisol measures and only one self-reported stress indicator, as previous research has had a tendency to do (Weber et al., 2022). To capture a comprehensive cortisol profile, it is essential to measure cortisol levels at various time points throughout the day (morning, afternoon, evening) over consecutive days (Gartland, O'Connor, Lawton, & Bristow, 2014). Future research should also measure confounders (gender, age, medication, waking time, etc.). The proposed approach would ensure an accurate depiction of cortisol dynamics, with appropriate measurement that considers factors

known to impact cortisol levels, such as sampling accuracy and relevant confounders, to truly understand daily life stressors, cortisol and associations with negative health behaviours.

It was beyond the scope of this thesis to consider genetic influences on the relationship between childhood trauma and suicidal behaviour. Traditional research methods often lack the capacity to adequately consider genetic influences or other unmeasured confounders. Although, a promising direction for future research is to utilise quasi-experimental methods to better understand and disentangle the causal effects of how child maltreatment impacts mental health from confounding factors (Baldwin et al., 2023). The current thesis finds evidence that individuals who have experienced childhood trauma can be predisposed to developing later mental health difficulties due to the experiences of abuse and neglect. Building upon these findings, a further point to consider is whether the elevated risk of later mental health difficulties is driven, in part, by other genetic and environmental factors for health problems, which may confound the aforementioned associations. It may be that these individuals could already have an increased risk of mental health difficulties due to genetic and environmental risk factors, which confound previously observed associations between childhood trauma and mental health difficulties (Baldwin et al., 2023). For instance, there is evidence indicating that individuals reporting a history of childhood trauma often have family history of mental health difficulties including depression (Streit et al., 2023) and may possess elevated polygenic scores for depression, ADHD and schizophrenia (Ratanatharathorn et al., 2021; Sallis et al., 2021). These co-occurring risk factors have not been fully accounted for in the majority of previous research on child maltreatment and mental health, which have relied on traditional epidemiological methods such as multiple regression that has limited ability to account for unmeasured confounders. The current thesis overcomes some of these difficulties using EMA methodology to consider these relationships in real time environments, with modelling techniques to account for confounders. However, there is still a need to consider the genotypes and family history of suicide in understanding why individuals may be predisposed to later mental health difficulties, including suicide. As previous research suggests that the effects of childhood trauma directly impacts cortisol awakening response which is implicated in psychopathologies; specifically, the rs1006737 polymorphism of the CACNA1C has been strongly linked in psychiatric disorders (Liu et al., 2011) and recently a genotype-by-trauma interaction was found whereby early life stress lead to higher CAR overall and specifically this was lower in the rs1006737 risk allele A carriers (Klaus et al., 2018). This warrants further exploration to consider both genetic and environmental influences on the relationship between childhood trauma and suicidal behaviour.

6.4 Practical and clinical implications

The thesis found childhood trauma to be associated with suicide risk in adulthood and that poorer executive functioning and higher levels of impulsivity, as well as other stress related variables, contribute to this increased risk (study 1 and study 3). The identification of these associations highlight the critical importance of addressing childhood trauma and associated factors (e.g., executive function, impulsivity, stress) as an integral part of suicide prevention. First, interventions aimed at targeting trauma in early childhood could play a crucial role in reducing the risk of suicide and promoting long-term mental well-being. The importance of trauma informed care, along with interventions designed to help adults who have experienced childhood trauma, have been well documented in previous literature (Goddard, 2021; Melton et al., 2020). The thesis findings have clinical significance, wherein clinicians could use these findings to inform their work with individuals with histories of childhood trauma. Clinicians could screen for such vulnerability factors and develop targeted interventions to improve functioning. The thesis findings present an opportunity to not only consider trauma based interventions but also to utilise stress management interventions to support the management of daily stress related factors, especially in those with a history of childhood trauma.

Moreover, the findings reported in this thesis suggest that there may be utility in exploring childhood stressors and cognitive ability as part of suicide risk assessments, in addition to individual traumatic events. One approach might involve incorporating the daily measures of impulsivity, executive function, within the therapeutic setting and using the evidence around these measures to guide a clinicians approach. This would allow assessment of current feelings to be validated across visits as well as to be compared against a stable trait measure. This would capture further contextual information as necessary to allow the clinician to better assess the individuals potential risk of suicidal ideation and behaviour. Enhancing our comprehension of factors contributing to suicide risk is paramount for developing precise theories, risk evaluations, and treatment strategies for at-risk individuals. That said, suicide risk assessment remains a controversial subject (Bolton et al., 2015) and assessments need to be a collaboration between clinicians and patients – rather than solely relying on scores on scale measures (Branley-Bell et al., 2019). For example, a recent clinical study suggested scale measures were unable to predict repeat suicidal behaviour (Steeg et al., 2018), therefore it is essential that utilisation of scales alone is not used to determine treatment or predict future risk.

Identifying those at most risk for suicide should continue to be a priority across health services and although numerous suicide risk assessment tools are utilised within the National Health Service (NHS), a recent nationwide investigation found a lack of uniformity in the application of risk evaluation instruments in mental health services (Graney et al., 2020). The current approach, waiting for individuals to reach a crisis before intervention, is flawed. The focus needs to be on prevention in reaching crisis. Screening

tools are only effective for determining the severity of a crisis, but they do not determine nor explain the likely complexity of factors intervening that *cause* a crisis. Therefore screening for high levels of stress related vulnerability factors could be an opening for the transition from checklist-based approaches towards a more person-centred methodology (Graney et al., 2020). Traditional risk assessments often focus on immediate susceptibility to suicide (Beck & Steer, 1988; Posner et al., 2011), concentrating on questions concerning current risk factors such as suicidal plans and access to lethal means. Transitioning towards a preventive assessment paradigm, where foundational suicide risk factors such as trauma and cognitive functioning are monitored, may enable the earlier detection of risk and reduce the likelihood of reaching crisis.

Lastly, clinicians, from a pragmatic perspective, are likely to be more concerned with assessing acute suicide risk over hours, days or weeks rather than across time periods spanning months and years. Similarly, for people with suicidal ideation, it may feel more urgent and relevant to explore the most recent antecedents to those experiences. Identification of factors that predict and reflect suicidal experiences over a time span of hours or days rather than weeks, months or years would greatly help clinicians to effectively evaluate and organise care to address an individual's immediate needs with respect to suicidal ideation and behaviours and to address acute risk. Such an approach has the potential to offer increased understanding of psychological factors that 'trigger' or lead to suicidal experiences, and the interactions between those psychological factors, which may amplify suicidal ideation and acts on an hour-by-hour, day-to-day basis.

6.5 Conclusion

In conclusion, this thesis has presented an overview and understanding of the multifaceted interplay between childhood trauma, stress and suicidal behaviour through cognitive functioning and other stress-related vulnerability factors. Through four studies employing diverse methodologies, it has helped outline the pathways linking childhood trauma to suicidal behaviour, expanding upon existing theoretical models and incorporating novel approaches. The findings highlight the significance of psychological pathways, specifically impaired executive function and impulsivity, in increasing suicide risk. Moving forward, there is a need for research to simultaneously examine the impact of both distal and proximal determinants of suicidal behaviour, integrating the role of cortisol alongside identified risk factors and pathways. By elucidating these complex relationships, the thesis emphasises the importance of early intervention strategies aimed at reducing the detrimental impacts of childhood trauma, ameliorating cognitive deficits and mitigating stress-related vulnerabilities to prevent an increased risk of suicide in adulthood. Furthermore, the thesis advocates for the integration of real-time assessments

and longitudinal approaches in suicide research to capture the dynamic nature of suicide risk factors and inform timely interventions. Taken together, the findings pave the way for future research to explore novel strategies to address the underlying psychological mechanisms linking childhood trauma, stress and to suicidal behaviour, ultimately contributing to suicide prevention efforts.

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Appendix

7.6 Study 1

7.6.1 Confirmatory analysis plan

Preliminary correlation analyses will be used to investigate the relationships between childhood trauma, impulsivity, executive functioning, stress, depression, wellbeing and suicide ideation and lifetime history of suicide. Logistic and hierarchical linear regression will be used to investigate whether childhood trauma (and its sub-types) are associated with lifetime suicide ideation and attempt or recent suicide ideation (H1). Next the PROCESS macro tool for SPSS will be utilised to test the models of mediation (H2: model 4 (Hayes, 2013)) and moderation (H3 & H4: model 1 (Hayes (2013)) using regressions and the percentile bootstrap technique to estimate the confidence intervals (Yzerbyt et al., 2018). All analyses will be run with and without covariates (age, gender and depression) as recommended by Simmons, Nelson and Simonsohn (2011) and all continuous predictor variables will be mean centered to allow better interpretation. Missing data will be handled using multiple imputation (or an equivalent such as full information maximum likelihood).

7.6.2 Odds ratio adjustment

The odds ratios presented in the manuscript appear to be small but need to be taken in context with the range of the CTQ scores (25 – 125). For such a wide range of scores a 1-unit change does not accurately reflect the increased risk of childhood trauma for most participants on the outcome variables. We therefore decided a meaningful unit change would be to compute the average difference in CTQ score between the categories of risk (None/minimal, low/moderate, moderate/severe, severe). An alternative scaling would be the present the *OR* for a 1 *SD* change in CTQ. As the *SD* of the CTQ is 19.4 this would lead us to present an *OR* for an even larger change in CTQ.

To compute the average difference the following procedure was adopted: taking the midpoint for each of the five subscales, for each risk category. For instance, for the None (or minimal risk) category, the following midpoints for each subscale were calculated; emotional abuse = 6.5, physical abuse = 6, sexual abuse = 5, emotional neglect = 7 and physical neglect = 6. Next, for each of the four risk categories, the average midpoint was calculated. The difference between each risk category average midpoint was computed.

Finally, the average difference was calculated which resulted in 14.3; the value adopted to be the meaningful unit of change to contextualise the odds ratios.

7.6.3 Transformations

Lifetime suicidal behaviour: for the APMS score, the following coding procedure would be introduced , 1 – ideation but no attempt and 2 – attempts.

Suicide ideation: the SSI (Beck et al., 1979) would be scored whereby each of the 21 items have a score from 0 – 2, suicide ideation is taken from the first 19 items with a resulting total score ranging from 0-38. The final two items measure number of suicide attempts and intent to die during the last attempt. Cronbach's $\alpha = 0.88$.

COVID-19 suicidal behaviour: two items which reflect the extent of suicidal behaviour as a result of COVID-19.

CTQ: two approaches will be used, following Bernstein (2003) whereby a summed score from 5 - 25 is created for each of the five subscales, allowing analysis of both individual scales and global childhood trauma score. As a result, individual scale scores for the CTQ ranged from 5 – 25 and global scores for the CTQ ranged from 25 - 125.

Impulsivity: summed score, greater score, greater impulsivity. The BIS-11 can be decomposed to 2nd Order factor components – attentional (comprised of items 5, 9, 11, 20, 28, 6 ,24 & 26), motor (items 2, 3, 4, 16, 17, 19, 21, 22, 23, 25 & 30) and non-planning (items 1, 7, 8, 10, 12, 13, 14, 15, 18, 27 & 29).

Stress: greater summed score, greater perceived stress. The scale is comprised of items 2, 4, 5 and 10 from the 10-item PSS scale.

For the executive dysfunctioning, depression and wellbeing measures, the greater the summed score, the greater executive dysfunctioning, depressive symptoms and overall wellbeing.

Table 9. Proposed univariate analyses

Hypothesis	Proposed Statistical Analysis	Interpretation given different outcomes	Sampling plan
H1: Childhood trauma (and sub-types) will be associated with both	H1: hierarchical linear regression (DV: recent	No evidence of a difference between childhood trauma and	See power analysis

recent and lifetime suicide ideation and attempt.	suicide ideation and attempt) and ordinal logistic regression analysis (DV: lifetime suicide ideation and attempt).	suicide ideation and attempt.	summary (below)
H2: The effects of childhood trauma (and sub-types) on suicide ideation and wellbeing will be mediated by executive functioning and impulsivity	H2: hierarchical linear regression analysis with a mediation component (model 4; Hayes, (2013))	No evidence of a difference of childhood trauma on suicide ideation and wellbeing.	
H3: The relationship between childhood trauma (and sub-types) and impulsivity/executive functioning will be moderated by recent stress	H3: hierarchical linear regression analysis with a moderation component (model 1; Hayes, (2013))	No evidence of a difference between childhood trauma and impulsivity/executive function. No evidence of this relationship being moderated by recent stress.	

H4: The relationship between childhood trauma and recent suicide ideation and wellbeing will be moderated by recent stress	H4: hierarchical linear regression with a moderation component (model 1; Hayes, (2013))	No evidence of a difference between childhood trauma and recent suicide ideation/wellbeing. No evidence of this relationship being moderated by recent stress.	

7.6.4 Magnitude of the indirect effects

There was a significantly indirect effect of childhood trauma on recent suicide ideation through executive functioning, *b* = 0.02, CI [0.01, 0.04]. This represents a relatively small effect k^2 = 0.04, 95% CI [0.02, 0.08]. The mediation represented a relatively small effect for all subscales; Emotional Abuse (k^2 = 0.08, CI (0.04, 0.14); Physical abuse (k^2 = 0.02, CI (0.00, 0.05); Sexual abuse (k^2 = 0.023, CI (0.001, 0.050); Emotional neglect (k^2 = 0.04, CI (0.01, 0.07); Physical neglect (k^2 = 0.04, CI (0.02, 0.07). There was no significant indirect effects for sexual abuse on recent suicide ideation through impulsivity.

There was a significantly indirect effect of childhood trauma on recent suicide ideation through impulsivity, *b* = 0.02, CI [0.01, 0.03]. This represents a relatively small effect k^2 = 0.04, 95% CI [0.01, 0.07]. The mediation represented a relatively small effect for four subscales; Emotional Abuse (k^2 = 0.04, CI (0.02, 0.07)); Physical abuse (k^2 = 0.02, CI (0.00, 0.05); Sexual abuse (k^2 = 0.02, CI (0.00, 0.05), Emotional neglect (k^2 = 0.03, CI (0.01, 0.06); physical neglect (k^2 = 0.03, CI (0.00, 0.05).

There was a significantly indirect effect of childhood trauma on wellbeing through executive functioning, *b* = -0.01, CI [-0.01, -0.00]. This represents a relatively small effect k^2 = -0.06, CI [-0.10, -0.03]. The mediation represented a relatively small effect for four subscales; Emotional Abuse (k2 = -0.08, CI (-0.12, -0.04); Physical abuse (k^2 = -0.03, CI (-0.06, -0.00); Emotional neglect (k^2 = -0.05, CI (-0.08, -0.02); physical neglect (k^2 = -0.06, CI (-0.09, -0.02). However, there was no significant indirect effects of sexual abuse on recent suicide ideation through executive functioning.

There was a significantly indirect effect of childhood trauma on wellbeing through impulsivity. *b* = -0.01, CI [-0.01, -0.00]. This represents a relatively small effect k^2 = -0.04, CI [-0.07, -0.01]. The mediation represented a relatively small effect for four subscales; Emotional Abuse (k^2 = -0.04, CI (-0.07, -0.01)); Physical abuse (k^2 = -0.02, CI

(-0.05, -0.00); Sexual abuse (k^2 = -0.02, CI (-0.05, -0.00), Emotional neglect (k^2 = -0.03, CI (-0.06, -0.01); physical neglect (k^2 = -0.07=2, CI (-0.05, -0.00).

Table 10. Demographics of the sample

		Total sample	**Suicide ideation group**	**Suicide attempt group**
			Mean (SD)	
N		457	238	219
Age		32.43 (11.22)	31.78 (11.40)	33.13 (11.00)
Sex (n) (%)				
	Female	345 (75.5%)	173 (72.7%)	172 (78.5%)
	Male	104 (22.8%)	60 (25.2%)	44 (20.1%)
	Not disclosed	8 (1.8%)	5 (2.1%)	3 (1.4%)
Ethnicity				
	White	431 (89.9%)	213 (89.5%)	198 (90.4%)
	Mixed	16 (3.5 %)	8 (3.4%)	8 (3.7%)
	Asian	18 (4%)	11 (4.6%)	7 (3.2%)
	Black	8 (1.7 %)	4 (1.7%)	4 (1.8%)
	Arabic	1 (0.2%)	0	1 (0.5%)
	Other	3 (0.7%)	2 (0.8%)	1 (0.5%)
Depression		36.16 (13.10)	33.51 (12.97)	39.05 (12.65)
Recent Suicide Ideation		11.49 (9.28)	8.83 (8.37)	14.39 (9.37)
Executive Function		38.35 (13.04)	37.13 (12.82)	39.67 (13.17)

Total CTQ		56.13 (19.37)	50.12 (15.90)	62.66 (20.68)
CTQ subscales				
	Emotional abuse	14.72 (5.97)	13.17 (5.56)	16.39 (5.95)
	Physical abuse	7.90 (4.42)	6.77 (3.14)	9.13 (5.21)
	Sexual abuse	9.08 (6.29)	7.53 (5.11)	10.76 (7.00)
	Emotional neglect	15.22 (5.38)	14.04 (5.01)	16.50 (5.50)
	Physical neglect	9.22 (3.98)	8.61 (3.54)	9.88 (4.32)
Wellbeing		17.09 (2.76)	17.48 (2.79)	16.65 (2.67)
Impulsivity		70.31 (11.89)	68.11 (11.48)	72.69 (11.89)
Perceived stress		10.73 (2.63)	10.46 (2.73)	11.03 (2.49)

Table 11. Correlations with confidence intervals

Variable	1	2	3	4	5	6	7	8	9	10	11
1. **Depressive symptoms**											
2. Recent suicide ideation	.52**										
	[.45, .58]										
3 Impulsivity	.35**	.26**									
	[.27, .43]	[.17, .34]									
4. Childhood trauma	.29**	.30**	.17**								
	[.20, .37]	[.22, .39]	[.08, .26]								
5. Emotional abuse	.25**	.26**	.17**	.83**							
	[.16, .33]	[.17, .34]	[.08, .26]	[.80, .86]							
6. Emotional	.23**	.29**	.15**	.80**	.67**						

neglect											
	[.14, .31]	[.20, .37]	[.06, .24]	[.77, .83]	[.62, .72]						
7. Physical abuse	.23**	.24**	.10*	.74**	.54**	.45**					
	[.14, .31]	[.16, .33]	[.01, .19]	[.69, .78]	[.47, .60]	[.37, .52]					
8. Physical neglect	.17**	.17**	.11*	.74**	.50**	.67**	.51**				
	[.08, .26]	[.08, .26]	[.01, .20]	[.69, .78]	[.43, .56]	[.61, .71]	[.44, .57]				
9. Sexual abuse	.19**	.17**	.09*	.62**	.33**	.24**	.35**	.23**			
	[.10, .28]	[.08, .26]	[.00, .18]	[.56, .68]	[.25, .41]	[.15, .33]	[.27, .43]	[.15, .32]			
10. Executive functioning	.44**	.27**	.67**	.21**	.25**	.16**	.10*	.17**	.08		
	[.37, .51]	[.18, .35]	[.61, .71]	[.12, .29]	[.16, .33]	[.07, .25]	[.01, .19]	[.08, .26]	[-.01, .17]		
11. Perceived stress	.64**	.39**	.24**	.09	.09	.08	.09	.04	.04	.35**	
	[.57, .70]	[.32, .47]	[.15, .33]	[-.01, .18]	[-.00, .18]	[-.02, .17]	[.00, .17]	[-.06, .12]	[-.05, .13]	[.26, .43]	
12. Wellbeing	-.69**	-.46**	-.24**	-.18**	-.13**	-.21**	-.16**	-.10*	-.09*	-.33**	.09*
	[-.74, -	[-.53, -	[-.33, -	[-.27, -	[-.22, -	[-.29, -	[-.25, -	[-.19, -	[-.18, -	[-.41, -	[.00,

	.64]	.39]	.15]	.09]	.04]	.12]	.07]	.00]	.00]	.25]	.18]

Table 12. Hierarchical linear regression for recent suicide ideation

Adjusted Model							
		***b* (95% CI)**	**SE *b***	**Beta**	***t***	**R^2**	**ΔR^2**
Step							
1	Age	-0.01 [-0.08, 0.05]	0.03	-0.02 [-0.10, 0.06]	-0.39		
	Gender	-0.67 [-2.32, 0.99]	0.84	-0.03 [-0.11, 0.05]	-0.79		
	Depression	0.37** [0.31, 0.42]	0.03	0.52 [0.44, 0.60]	12.88	R^2 = .270** [0.20, 0.33]	
Predictor: CTQ							
2	Age	-0.04 [-0.10, 0.03]	0.03	-0.05 [-0.13, 0.03]	-1.15		
	Gender	-1.28 [-2.92, 0.36]	0.84	-0.06 [-0.14, 0.02]	-1.53		
	Depression	0.33** [0.28, 0.39]	0.03	0.47 [0.39, 0.55]	11.42		
	CTQ	0.09** [0.05, 0.13]	0.02	0.19 [0.10, 0.27]	4.40	R^2 = .300** [0.23, 0.36]	ΔR^2 = 0.030** [0.00, 0.06]
Predictor: Emotional abuse							
2	Age	-0.02 [-0.09, 0.04]	0.03	-0.03 [-0.11, 0.05]	-0.71		
	Gender	-1.06 [-2.70, 0.59]	0.84	-0.05 [-0.13, 0.03]	-1.26		
	Depression	0.34**[0.29, 0.40]	0.03	0.49 [0.41, 0.57]	11.83		

	Emotional abuse	0.23** [0.10, 0.35]	0.06	0.15 [0.06, 0.23]	3.51	R^2 = .289** [0.22,0.35]	ΔR^2 = .019** [-0.00, 0.04]
Predictor: Physical abuse							
2	Age	-0.05 [-0.11, 0.02]	0.03	-0.05 [-0.14, 0.03]	-1.30		
	Gender	-0.83 [-2.47, 0.80]	0.84	-0.04 [-0.12, 0.04]	-1.00		
	Depression	0.35** [0.29, 0.40]	0.03	0.49 [0.41, 0.57]	12.04		
	Physical abuse	0.31** [0.14, 0.49]	0.09	0.15 [0.07, 0.23]	3.51	R^2 = .289** [0.22,0.35]	ΔR^2 = .019** [-.00, .04]
Predictor: Sexual abuse							
2	Age	-0.02 [-0.08, 0.05]	0.03	-0.02 [-0.10, 0.06]	-0.48		
	Gender	-1.00 [-2.68, 0.68]	0.86	-0.05 [-0.13, 0.03]	-1.17		
	Depression	0.36** [0.30, 0.41]	0.03	0.51 [0.42, 0.59]	12.33		
	Sexual abuse	0.12* [0.00, 0.24]	0.06	0.08 [0.00, 0.16]	1.97	R^2 = .276** [0.20, 0.33]	ΔR^2 = .006* [-0.01, 0.02]
Predictor: Emotional neglect							
2	Age	-0.04 [-0.11, 0.03]	0.03	-0.05 [-0.13, 0.03]	-1.20		
	Gender	-1.15 [-2.77, 0.48]	0.83	-0.06 [-0.13, 0.02]	-1.38		
	Depression	0.34** [0.28, 0.40]	0.03	0.48 [0.40, 0.56]	11.86		

	Emotional neglect	0.33** [0.19, 0.47]	0.07	0.19 [0.11, 0.27]	4.71	R^2 = .304** [0.23, 0.36]	ΔR^2 = .034** [0.01, 0.06]
Predictor: Physical neglect							
2	Age	-0.02 [-0.09, 0.05]	0.03	-0.02 [-0.10, 0.06]	-0.58		
	Gender	-0.76 [-2.41, 0.89]	0.84	-0.04 [-0.12, 0.04]	-0.91		
	Depression	0.36** [0.30, 0.42]	0.03	0.51 [0.43, 0.59]	12.41		
	Physical neglect	0.20* [0.02, 0.39]	0.10	0.09 [0.01, 0.17]	2.15	R^2 = .277** [0.21, 0.34]	ΔR^2 = .007** [-0.01, 0.02]

Table 13. Binary logistic regression showing the coefficients of the model predicting lifetime history of suicide ideation or attempts [95% bootstrap confidence intervals based on 1000 samples]

		Unadjusted				***Adjusted***			
		b	**95% CI for odds ratio**			***b***	**95% CI for odds ratio**		
Step		95% CI	**Lower**	**Odds**	**Upper**	**95% CI**	**Lower**	**Odds**	**Upper**
Step 1	Age					0.01 [-0.01, 0.03]	0.99	1.01	1.03
	Sex					0.36 [-0.12, 0.84]	0.90	1.43	2.26
	Depressive symptoms					0.03 [0.02, 0.05]	1.02	1.03	1.05
Step 2	Age					0.00 [-0.02, 0.02]	0.98	1.00	1.02
	Sex					0.17 [-0.30, 0.65]	0.74	1.19	1.92
	Depressive symptoms					0.02 [0.01, 0.04]	1.01	1.02	1.04
	Childhood trauma	0.04 [0.03, 0.05]	1.03	1.04	1.05	0.03 [0.02, 0.05]	1.02	1.04	1.05
Step 2	Age					0.01 [-0.01, 0.02]	0.99	1.01	1.02
	Sex					0.24 [-0.23, 0.71]	0.80	1.27	2.04
	Depressive symptoms					0.03 [0.01, 0.04]	1.01	1.03	1.04

	Emotional abuse	0.10 [0.06, 0.13]	1.06	1.10	1.14	0.08 [0.05, 0.12]	1.05	1.09	1.13
Step 2	Age					-0.00 [-0.02, 0.02]	0.98	1.00	1.02
	Sex					0.34 [-0.16, 0.86]	0.88	1.41	2.26
	Depressive symptoms					0.03 [0.01, 0.04]	1.01	1.03	1.04
	Physical abuse	0.14 [0.09, 0.20]	1.09	1.15	1.21	0.13 [0.08, 0.20]	1.08	1.14	1.20
Step 2	Age					0.01 [-0.01, 0.03]	0.99	1.01	1.03
	Sex					0.16 [-0.32, 0.63]	0.73	1.17	1.88
	Depressive symptoms					0.03 [0.01, 0.04]	1.01	1.03	1.05
	Sexual abuse	0.09 [0.06, 0.13]	1.06	1.09	1.13	0.08 [0.04, 0.12]	1.05	1.08	1.12
Step 2	Age					0.00 [-0.02, 0.02]	0.99	1.00	1.02
	Sex					0.28 [-0.19, 0.76]	0.83	1.32	2.10
	Depressive symptoms					0.03 [0.01, 0.04]	1.01	1.03	1.05
	Emotional neglect	0.09 [0.06, 0.13]	1.05	1.09	1.13	0.08 [0.04, 0.12]	1.04	1.08	1.12
Step 2	Age					0.01 [-0.01, 0.03]	0.99	1.01	1.03
	Sex					0.34 [-0.13, 0.83]	0.89	1.41	2.23
	Depressive symptoms					0.03 [0.02, 0.05]	1.02	1.03	1.05

Physical neglect	0.08 [0.04, 0.13]	1.03	1.09	1.14	0.07 [0.02, 0.12]	1.02	1.07	1.12

Table 14. Mediation analysis with recent suicide ideation as the outcome variable

Outcome: recent suicide ideation				
Models of individual predictions	***b* (unstandardised)**	***t***	***p***	**95% CI**
Associations of models tested				
2.1				
childhood trauma → executive function	0.14	4.49	< .001	0.08, 0.20
executive function → recent suicide ideation	0.15	4.82	< .001	0.09, 0.22
2.2				
childhood trauma → impulsivity	0.10	3.65	< .001	0.05, 0.16
impulsivity → recent suicide ideation	0.17	4.88	< .001	0.10, 0.24
2.3				
2.3.1 emotional abuse → impulsivity	0.34	3.67	<.001	0.16, 0.52
impulsivity → recent suicide ideation	0.18	5.00	< .001	0.11, 0.24
2.3.2 physical abuse → impulsivity	0.27	2.19	.029	0.03, 0.52
impulsivity → recent suicide ideation	0.19	5.38	< .001	0.12, 0.25
2.3.3 sexual abuse → impulsivity	0.17	1.98	.048	0.00, 0.35

impulsivity → recent suicide ideation	0.19	5.51	< .001	0.12, 0.26
2.3.4 emotional neglect → recent suicide ideation	0.33	3.22	.001	0.13, 0.53
impulsivity → recent suicide ideation	0.17	5.04	< .001	0.11, 0.24
2.3.5 physical neglect → recent suicide ideation	0.32	2.28	.023	0.04, 0.59
impulsivity → recent suicide ideation	0.19	5.46	< .001	0.12, 0.26
2.4				
2.4.1 emotional abuse → executive function	0.54	5.44	< .001	0.35, 0.74
executive function → recent suicide ideation	0.16	4.78	< .001	0.09, 0.22
2.4.2 physical abuse → executive function	0.29	2.12	.034	0.02, 0.56
executive function → recent suicide ideation	0.18	5.57	< .001	0.11, 0.24
2.4.3 sexual abuse → executive function	0.17	1.75	.081	-0.02, 0.36
executive function → recent suicide ideation	0.18	5.72	< .001	0.12, 025
2.4.4 emotional neglect → executive function	0.40	3.55	< .001	0.18, 0.62
executive function → recent suicide ideation	0.17	5.12	< .001	0.10, 0.22
2.4.5 physical neglect → executive function	0.56	3.69	< .001	0.26, 0.86
executive function → recent suicide ideation	0.18	5.41	< .001	0.11, 0.24

indirect effects	***b***	**SE**	**Boostrapped 95% CI (N)**	
2.1				
childhood trauma → executive function → recent suicide ideation	0.02	0.01	0.01	0.04
2.2				
childhood trauma → impulsivity → recent suicide ideation	0.02	0.01	0.01	0.03
2.3				
2.3.1 emotional abuse → impulsivity → recent suicide ideation	0.06	0.02	0.02	0.10
2.3.2 physical abuse → impulsivity → recent suicide ideation	0.05	0.03	0.00	0.11
2.3.3 sexual abuse → impulsivity → recent suicide ideation	0.03	0.02	0.00	0.07
2.3.4 emotional neglect → impulsivity → recent suicide ideation	0.06	0.02	0.02	0.11
2.3.5 physical neglect → impulsivity → recent suicide ideation	0.06	0.03	0.01	0.13
2.4				
2.4.1 emotional abuse → executive function → recent suicide ideation	0.08	0.03	0.04	0.14
2.4.2 physical abuse → executive function → recent suicide ideation	0.05	0.03	0.01	0.11
2.4.3 sexual abuse → executive function → recent suicide ideation	0.03	0.02	-0.00	0.05
2.4.4 emotional neglect → executive function → recent suicide ideation	0.06	0.02	0.02	0.12

2.4.5 physical neglect → executive function → recent suicide ideation	0.10	0.03	0.02	0.07
Direct effects after inclusion of mediator	***b***	***t***	***p***	**95% CI**
2.1				
childhood trauma → recent suicide ideation	0.12	5.83	< .001	0.08, 0.17
2.2				
childhood trauma → recent suicide ideation	0.13	6.06	< .001	0.09, 0.17
2.3				
2.3.1 emotional abuse → recent suicide ideation	0.34	4.85	< .001	0.20, 0.48
2.3.2 physical abuse → recent suicide ideation	0.46	4.94	< .001	0.28, 0.64
2.3.3 Sexual abuse → recent suicide ideation	0.21	3.21	.001	0.08, 0.34
2.3.4 Emotional neglect → recent suicide ideation	0.44	5.80	< .001	0.29, 0.59
2.3.5 Physical neglect → recent suicide ideation	0.34	3.22	.001	0.13, 0.54
2.4				
2.4.1 Emotional abuse → recent suicide ideation	0.31	4.41	< .001	0.17, 0.45
2.4.2 Physical abuse → recent suicide ideation	0.46	4.95	< .001	0.28, 0.64
2.4.3 Sexual abuse → recent suicide ideation	0.22	3.26	.001	0.09, 0.35

2.4.4 Emotional neglect → recent suicide ideation	0.43	5.70	< .001	0.28, 0.58
2.4.5 Physical neglect → recent suicide ideation	0.30	2.84	.005	0.09, 0.51
Total effect of X on Y	***b***	***t***	***p***	**95% CI**
2.1				
Childhood trauma → executive function → recent suicide ideation	0.15	6.81	< .001	0.10, 0.19
2.2				
Childhood trauma → Impulsivity → recent suicide ideation	0.15	6.81	< .001	0.10, 0.19
2.3				
2.3.1 Emotional abuse → impulsivity → recent suicide ideation	0.40	5.64	< .001	0.26, 0.54
2.3.2 Physical abuse → impulsivity → recent suicide ideation	0.51	5.35	< .001	0.32 0.70
2.3.3 Sexual abuse → impulsivity → recent suicide ideation	0.25	3.62	< .001	0.11, 0.38
2.3.4 Emotional neglect → impulsivity → recent suicide ideation	0.50	6.45	<.001	0.35, 0.65
Physical neglect → impulsivity → recent suicide ideation	0.40	3.70	<.001	0.19, 0.61
2.4				
2.4.1 Emotional abuse → executive function → recent suicide ideation	0.40	5.64	< .001	0.26, 0.54
2.4.2 Physical abuse → executive function → recent suicide ideation	0.51	5.35	< .001	0.32, 0.70

2.4.3 Sexual abuse → executive function → recent suicide ideation	0.25	3.62	< .001	0.11, 0.38
2.4.4 Emotional neglect → executive function → recent suicide ideation	0.50	6.45	< .001	0.35, 0.65
2.4.5 Physical neglect → executive function → recent suicide ideation	0.40	3.70	< .001	0.19, 0.61

Table 15. Mediation analysis with wellbeing as the outcome variable

Outcome: wellbeing				
Models of individual predictions	***b***	***t***	***p***	**95% CI**
Associations of models tested				
3.1				
Childhood trauma → executive function	0.14	4.49	< .001	0.08, 0.20
Executive function → Wellbeing	-0.07	-6.84	< .001	-0.08, -0.05
3.2 Childhood trauma → impulsivity	0.10	3.65	< .001	0.05, 0.16
Impulsivity → wellbeing	-0.05	-4.75	< .001	-0.07, -0.03
3.3				
3.3.1 Emotional abuse → impulsivity	0.34	3.67	< .001	0.16, 0.52
Impulsivity → wellbeing	-0.05	-4.92	< .001	-0.07, -0.03
3.3.2 Physical abuse → impulsivity	0.27	2.19	.029	0.03, 0.52
Impulsivity → wellbeing	-0.05	-5.02	< .001	-0.07, -0.03
3.3.3 Sexual abuse → impulsivity	0.17	1.98	.048	0.00, 0.35
Impulsivity → wellbeing	-0.05	-5.15	< .001	-0.08, -0.03

3.3.4 emotional neglect → wellbeing	0.33	3.22	.001	0.13, 0.53
Impulsivity → wellbeing	-0.05	-4.76	<.001	-0.07, -0.03
3.3.5 physical neglect → wellbeing	0.32	2.28	.023	0.04, 0.59
Impulsivity → wellbeing	-0.05	-5.12	<.001	-0.08, -0.03
3.4				
3.4.1 Emotional abuse → executive function	0.54	5.44	<.001	0.35, 0.74
Executive function → wellbeing	-0.07	-7.01	<.001	-0.09, -0.05
3.4.2 Physical abuse → executive function	0.29	2.12	.034	0.02, 0.56
Executive function → wellbeing	-0.07	-7.23	< .001	-0.08, -0.05
3.4.3 Sexual abuse → executive function	0.17	1.75	.081	-0.02, 0.36
Executive function → wellbeing	-0.07	-7.36	< .001	-0.09, -0.05
3.4.4 emotional neglect → wellbeing	0.40	3.55	< .001	0.18, 0.62
Executive function → wellbeing	-0.06	-6.91	<.001	-0.08, -0.04
3.4.5 physical neglect → wellbeing	0.56	3.69	< .001	0.26, 0.86
Executive function → wellbeing	-0.07	-7.24	< .001	-0.09, -0.05

Indirect effects	***b***	**SE**	**Bootstrapped 95% CI (N)**

3.1				
Childhood trauma → executive function → wellbeing	-0.01	0.02	-0.10	-0.03
3.2				
Childhood trauma → impulsivity → wellbeing	-0.01	0.00	-0.01	-0.00
3.3				
3.3.1 Emotional abuse → impulsivity → wellbeing	-0.02	0.01	-0.03	-0.01
3.3.2 Physical abuse → impulsivity → wellbeing	-0.01	0.01	-0.03	-0.00
3.3.3 Sexual abuse → impulsivity → wellbeing	-0.01	0.01	-0.02	-0.00
3.3.4 Emotional neglect → impulsivity → wellbeing	-0.02	0.01	-0.03	-0.00
3.3.5 Physical neglect → impulsivity → wellbeing	-0.02	0.01	-0.04	-0.00
3.4				
3.4.1 Emotional abuse → executive function → wellbeing	-0.04	0.01	-0.06	-0.02
3.4.2 Physical abuse → executive function → wellbeing	-0.02	0.01	-0.04	-0.00
3.4.3 Sexual abuse → executive function → wellbeing	-0.01	0.01	-0.03	0.00
3.4.4 Emotional neglect → executive function → wellbeing	-0.03	0.01	-0.04	-0.01
3.4.5 Physical neglect → executive function → wellbeing	-0.04	0.01	-0.06	-0.02

Direct effects after inclusion of mediator	***b***	***t***	***p***	**95% CI**
3.1				
Childhood trauma → wellbeing	-0.02	-2.68	.008	-0.03, -0.00
3.2				
Childhood trauma → wellbeing	-0.02	03.21	.001	-0.03, -0.01
3.3				
3.3.1 Emotional abuse → wellbeing	-0.04	-1.92	.056	-0.08, 0.00
3.3.2 Physical abuse → wellbeing	-0.09	-3.08	.002	-0.14, -0.03
3.3.3 Sexual abuse → wellbeing	-0.03	-1.56	.119	-0.07, 0.01
3.3.4 Emotional neglect → wellbeing	-0.09	-3.85	< .001	-0.14, -0.04
3.3.5 Physical neglect → wellbeing	-0.05	-1.55	.121	-0.11, 0.01
3.4				
3.4.1 Emotional abuse → wellbeing	-0.02	-1.04	.298	-0.06, 0.02
3.4.2 Physical abuse → wellbeing	-0.08	-2.98	.003	-0.03, -0.13
3.4.3 Sexual abuse → wellbeing	-0.03	-1.50	.135	-0.07, 0.01
3.4.4 Emotional neglect → wellbeing	-0.08	-3.53	< .001	-0.12, -0.04

3.4.5 Physical neglect → wellbeing	-0.03	-0.90	.368	-0.09, 0.03
Total effect of X on Y	***b***	***t***	***p***	**95% CI**
3.1				
Childhood trauma → executive function → wellbeing	**-0.03**	**-3.98**	**< .001**	**-0.04, -0.01**
3.2				
Childhood trauma → impulsivity → Wellbeing	-0.03	-3.98	< .001	-0.04, -0.01
3.3				
3.3.1 Emotional abuse → impulsivity → wellbeing	-0.06	-2.72	.007	-0.10, -0.02
3.3.2 Physical abuse → impulsivity → wellbeing	-0.10	-3.52	< .001	-0.16, -0.04
3.3.3 Sexual abuse → impulsivity → wellbeing	-0.04	-1.99	.047	-0.08, -0.00
3.3.4 Emotional neglect → impulsivity → wellbeing	-0.11	-4.50	< .001	-0.15, -0.06
3.3.5 Physical neglect → impulsivity → wellbeing	-0.07	-2.05	.041	-0.13, -0.00
3.4				
3.4.1 Emotional abuse → executive function → wellbeing	-0.06	-2.72	.007	-0.10, -0.02
3.4.2 Physical abuse → executive function → wellbeing	-0.10	-3.52	< .001	-0.16, -0.04
3.4.3 Sexual abuse → executive function → wellbeing	-0.04	-1.99	.047	-0.08, -0.00

3.4.4 Emotional neglect → executive function → wellbeing	-0.11	-4.50	< .001	-0.15, -0.06
3.4.5 Physical neglect → executive function → wellbeing	-0.07	-2.05	.041	-0.13, -0.00

Table 16. Moderation analysis for the outcomes impulsivity and executive functioning

	b	*t*	*p*	95% CI	
Outcome: impulsivity					
4.1 Childhood trauma → impulsivity	0.09	3.25	.001	0.04	0.15
4.2 Stress * Childhood trauma → impulsivity	0.00	0.01	.989	-0.02	0.02
4.3 Emotional abuse → impulsivity	0.30	3.32	.001	0.12	0.48
4.4 Stress * emotional abuse → impulsivity	0.03	1.00	.319	-0.03	0.10
4.5 Physical abuse → impulsivity	0.22	1.78	.077	-0.02	0.47
4.6 stress* Physical abuse → impulsivity	0.00	0.04	.970	-0.09	0.10
4.7 Sexual abuse → impulsivity	0.15	1.78	.076	-0.02	0.32
4.8 stress* Sexual abuse → impulsivity	0.03	0.86	.391	-0.04	0.09
4.9 Emotional neglect → impulsivity	0.29	2.93	.004	0.10	0.49
5.1 stress*emotional neglect → impulsivity	-0.02	-0.59	.554	-0.09	0.05
5.2 physical neglect → impulsivity	0.30	2.23	.026	0.04	0.57
5.3 stress * physical neglect → impulsivity	-0.05	-0.83	.405	-0.15	0.06
Outcome: executive function					

6.3 Childhood trauma → executive function	0.09	3.25	.001	0.04	0.15
6.4 Stress * Childhood trauma → executive function	0.00	0.01	.989	-0.02	0.02
6.5 Emotional abuse → executive function	0.48	5.07	< .001	0.29	0.66
6.6 Stress * emotional abuse → executive function	0.01	0.18	.859	-0.06	0.07
6.7 Physical abuse → executive function	0.20	1.49	.137	-0.06	0.46
6.8 stress* Physical abuse → executive function	0.02	0.40	.687	-0.08	0.12
6.9 Sexual abuse → executive function	0.14	1.54	.125	-0.04	0.32
7.1 stress* Sexual abuse → executive function	0.00	0.03	.977	-0.07	0.07
7.2 Emotional neglect → executive function	0.33	3.11	.002	0.12	0.54
7.3 stress*emotional neglect → executive function	0.00	0.07	.945	-0.07	0.08
7.4 physical neglect → executive function	0.52	3.65	<.001	0.24	0.80
7.5 stress * physical neglect → executive function	-0.01	-0.20	.839	-0.12	0.10

Table 17. Moderation analysis for the outcomes recent suicide ideation and wellbeing

	b	***t***	***p***	**95% CI**	
Outcome: recent suicide ideation					
4.1 Childhood trauma → recent suicide ideation	0.13	6.51	< .001	0.09	0.17
4.2 Stress * Childhood trauma → recent suicide ideation	-0.00	-0.27	.789	-0.02	0.01
4.3 Emotional abuse → recent suicide ideation	0.35	5.29	< .001	0.22	0.47
4.4 Stress * emotional abuse → recent suicide ideation	-0.00	-0.16	.869	-0.05	0.04
4.5 Physical abuse → recent suicide ideation	0.45	4.95	< .001	0.27	0.62
4.6 stress* Physical abuse → recent suicide ideation	-0.00	-0.07	.941	-0.07	0.07
4.7 Sexual abuse → recent suicide ideation	0.22	3.51	<.001	0.10	0.34
4.8 stress* Sexual abuse → recent suicide ideation	0.02	0.82	.413	-0.03	0.07
4.9 Emotional neglect → recent suicide ideation	0.45	6.25	< .001	0.31	0.59
5.1 stress*emotional neglect → recent suicide ideation	-0.01	-0.46	.647	-0.06	0.04
5.2 physical neglect → recent suicide ideation	0.36	3.62	< .001	0.16	0.56

5.3 stress * physical neglect → recent suicide ideation	0.02	0.53	.600	-0.06	0.10
Outcome: wellbeing					
6.3 Childhood trauma → wellbeing	-0.02	-3.60	< .001	-0.03	-0.01
6.4 Stress * Childhood trauma → wellbeing	0.00	0.04	.965	-0.00	0.00
6.5 Emotional abuse → wellbeing	-0.03	-1.95	0.052	-0.06	0.00
6.6 Stress * emotional abuse → wellbeing	-0.01	-1.32	.189	-0.02	0.00
6.7 Physical abuse → wellbeing	-0.07	-3.27	.001	-0.11	-0.03
6.8 stress* Physical abuse → wellbeing	0.01	1.20	.231	-0.01	0.03
6.9 Sexual abuse → wellbeing	-0.03	-1.91	.057	-0.06	0.00
7.1 stress* Sexual abuse → wellbeing	-0.00	-0.12	.905	-0.01	0.01
7.2 Emotional neglect → wellbeing	-0.08	-4.43	< .001	-0.11	-0.04
7.3 stress*emotional neglect → wellbeing	-0.00	-0.74	.458	-0.02	0.01
7.4 physical neglect → wellbeing	-0.05	-2.22	0.027	-0.10	-0.01
7.5 stress * physical neglect → wellbeing	0.01	1.25	.211	-0.01	0.03

7.6.5 Power analysis and sample size estimation

The general approach taken to power analysis is to start with reasonable values of the parameters (e.g., effect size, correlations between predictors, base rates of outcomes) and estimate power as a function of *n*. As the parameters aren't known with any degree of certainty we also vary the values slightly around those reasonable starting points to gauge sensitivity to the key parameters and present these graphically. For complex analyses the values for power are simulated and all analyses were undertaken in R 4.03 (R Core Team, 2020). All analyses assume *alpha* = .05 unless otherwise stated.

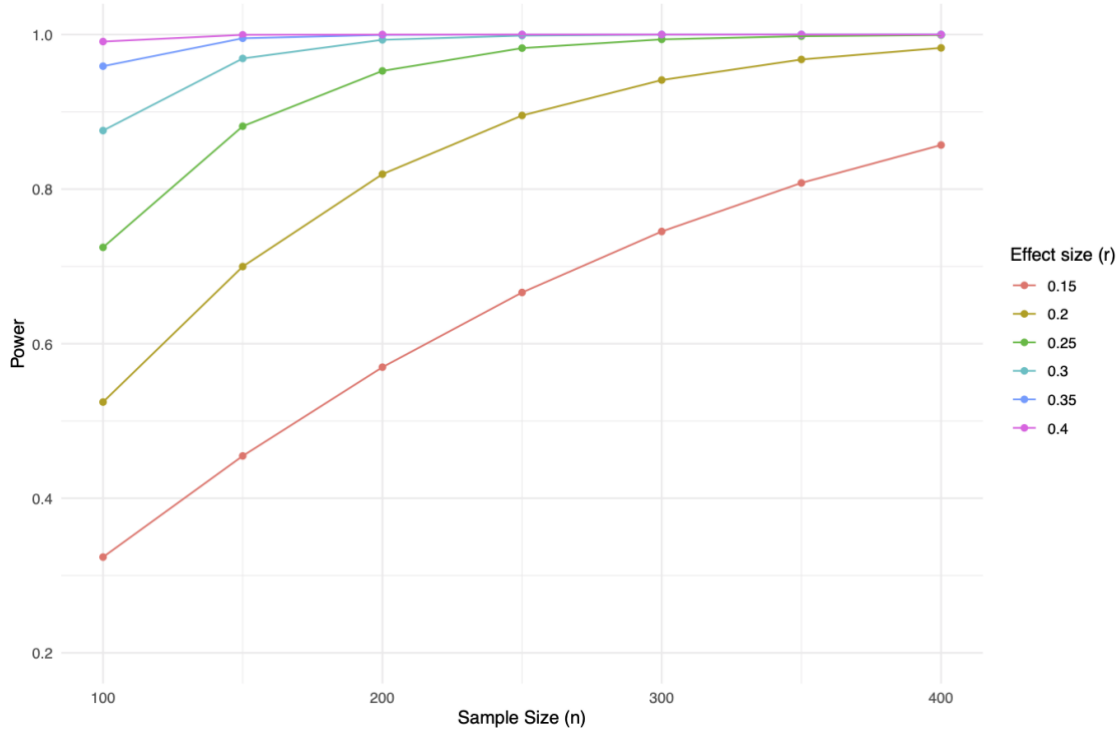
In summary the aim is not to arrive at a single number for each test but arrive at an overall sample size that will have good power (e.g., approximately 80% or more) for a wide range of effect sizes.

7.6.5.1 Hypothesis 1a

Predictor: Childhood trauma (CTQ)

Outcome: Recent suicide ideation (outcome 1)

Recent research suggests correlations ranging from .2 to .4 for different subscales and the overall CTQ measure (Bahk et al., 2017).



Interpretation: With sample sizes of 350 and above power is relatively high (80% or more) for correlations > .15 and for correlations as low as .20 $n = 200$ would be sufficient.

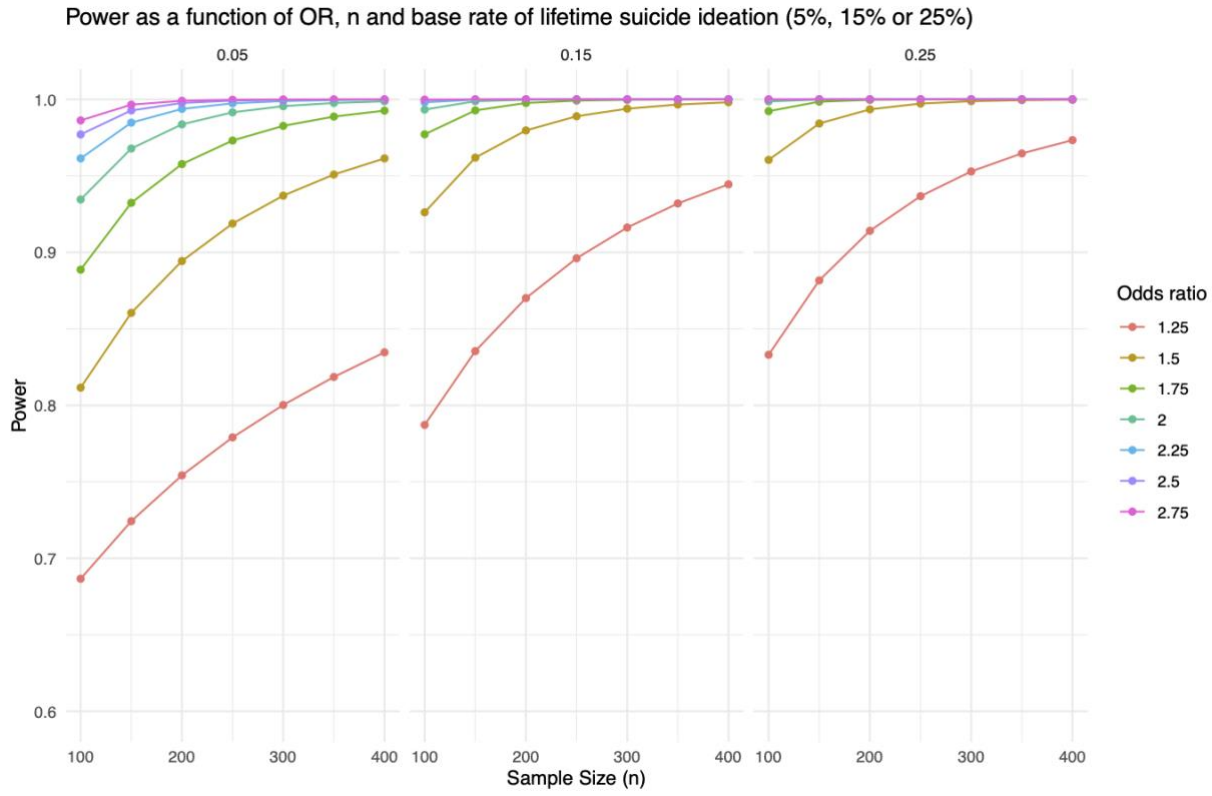
7.6.5.2 Hypothesis 1b and c

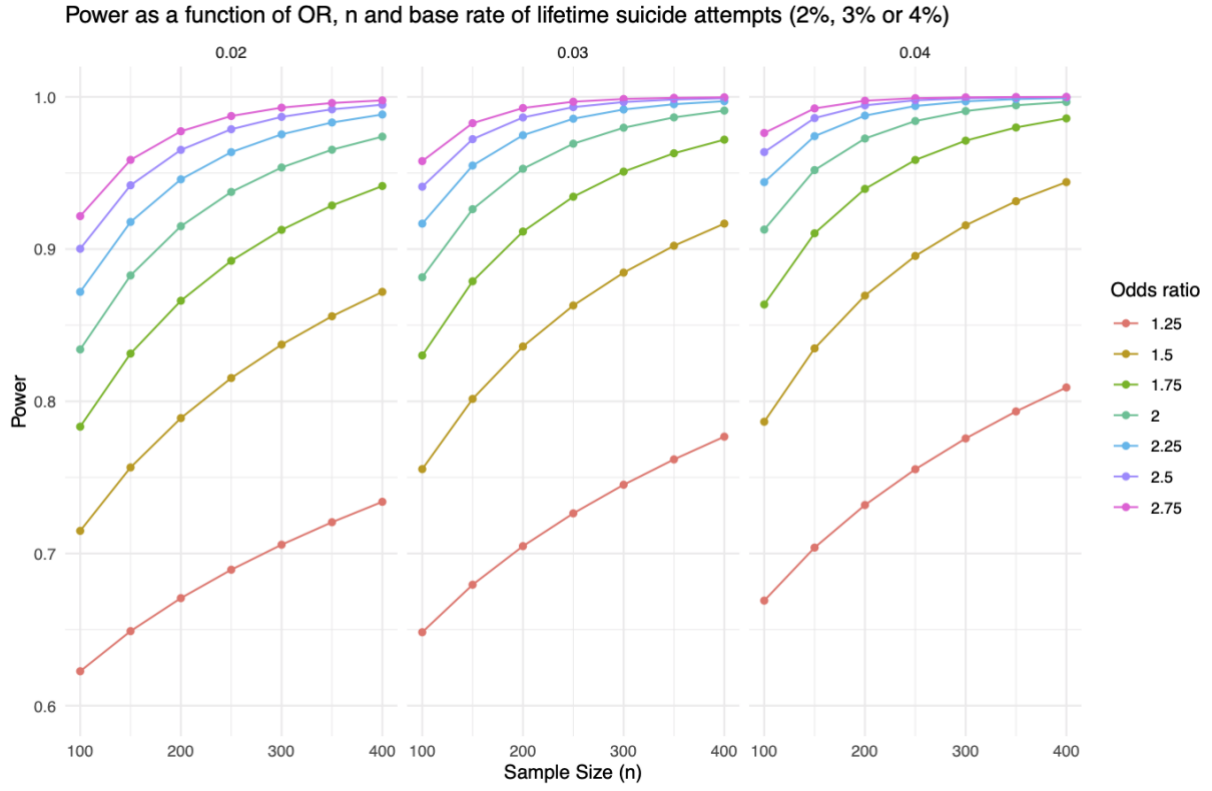
Predictor: Childhood trauma (CTQ)

Outcome: Lifetime suicide ideation or attempt (outcomes 2 and 3)

Previous research suggests an odds ratio (*OR*) of 2.66 (1.63) for ideation and 2.09 (1.45) for attempt (Angelakis et al., 2019). Here we used the power estimation approach for logistic regression of Vittinghoff et al. (2009). Importantly the key parameters are the *OR* (for a standardized predictor, i.e., the OR for a 1 *SD* increase in the predictor) and the base rate of the outcome coded 1 (here lifetime suicide ideation). As the original *OR* seems to be from a dummy coded dichotomous predictor it needs to be rescaled to have an *SD* of 1. This produces more conservative estimates of the *OR*s as 1.63 and 1.45 (as halving the effect on the log odds scale is equivalent to taking the square root of the *OR*).

Approaches that ignore the base rate could be wildly wrong (as when the outcome is rare or common this dramatically reduces power relative to outcomes with prevalence around .50). Base rate of lifetime suicide ideation was estimated as around .135 (Kessler et al., 1999). Other estimates are lower so a wider range is used here. For attempts prevalence is estimated around 3% (Nock, Borges, Bromet, Alonso, et al., 2008).





Interpretation: For lifetime suicide ideation reasonable power is maintained even with the lower prevalence estimate of 5% provided the *OR* is at least 1.5 for a wide range of *n*.

With *n* = 300 power is over 80% to detect an *OR* of 1.25. For suicide attempts prevalence is much lower and with prevalence at the lower end of what the literature suggests (2%) power is only satisfactory if the *OR* is 1.5 or greater and *n* at least 250.

7.6.5.3 Hypothesis 2ai

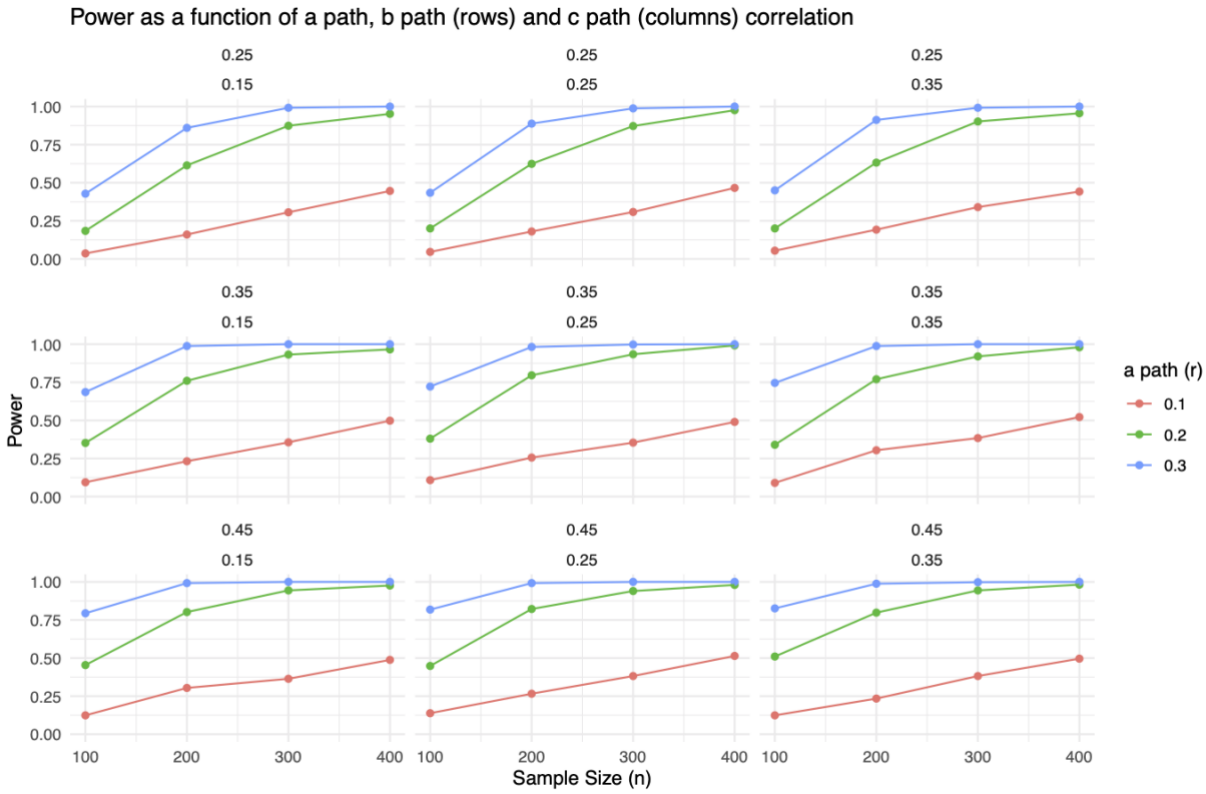
Predictor: Childhood trauma (CTQ)

Mediator: Executive function

Outcome: Recent suicide ideation

Power analysis for simple mediation (the *a* times *b* path in the model) depends on the correlations between the three variables. Power could be further impacted by additional covariates/predictors either reducing the error in the model or introducing collinearity (respectively increasing or decreasing power), but simulating simple mediation for a range of plausible correlation values should give a good idea of the sensitivity to assumed parameter values at different sample sizes. From previous research correlation between predictor and mediator (*a* path) is around .21 (Op den Kelder et al., 2018), between mediator and outcome (*b* path) .35 (Saffer & Klonsky, 2017) and (*c* path) predictor and outcome .26 (Angelakis et al., 2019).

Note that for all mediation tests power is likely to depend on the weakest of the *a* and *b* paths. This is because the mediation effect is *a* times *b* then is either *a* or *b* is close to zero then *ab* will necessarily be close to zero. This is a feature not a bug – power ought to be low when the mediator effect is near zero.



Interpretation: With executive function as a mediator power depends critically on the *a* path but is over 80% when *n* = 300 or more as long as the a path is around *r* = 0.2 or greater.

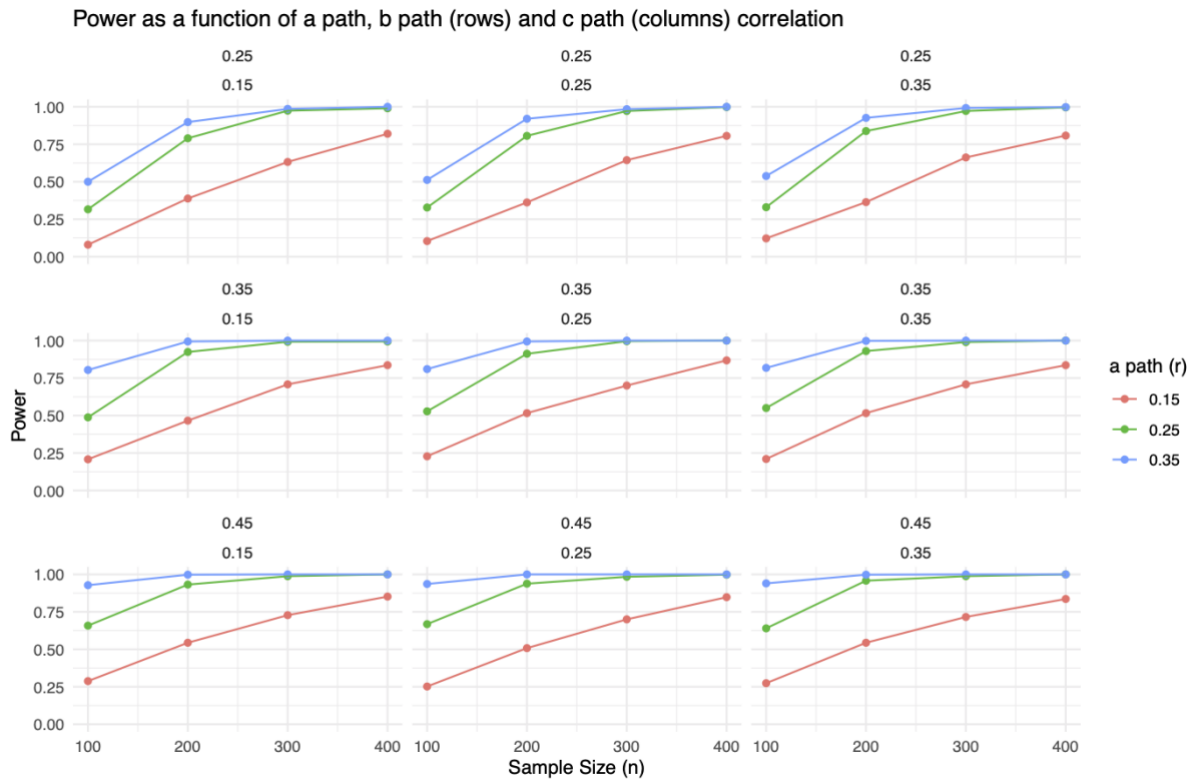
7.6.5.4 Hypothesis 2aii

Predictor: Childhood trauma (CTQ)

Mediator: Impulsivity

Outcome: Recent suicide ideation

From previous research correlation between predictor and mediator is around .253 (Dal Santo et al., 2020) between mediator and outcome .33 (Kleiman et al., 2012) and predictor and outcome .26 (as above).



Interpretation: Power tends to be poor when *a* or *b* is small (as one would expect) but is reasonable at *n* = 400 even when both paths are as weak as *r* = .15. For larger effects *n* = .200 may well be sufficient.

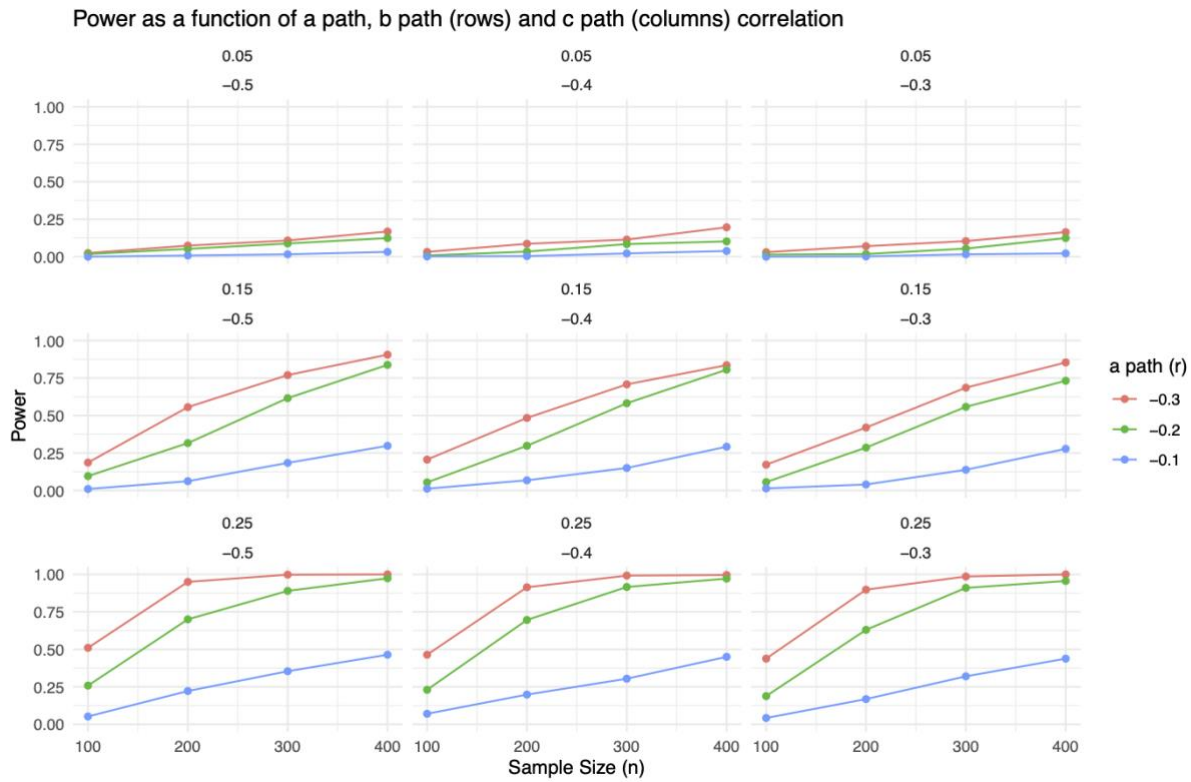
7.6.5.5 Hypothesis 2bi

Predictor: Childhood trauma (CTQ)

Mediator: Executive function

Outcome: Wellbeing

From previous research correlation between predictor and mediator is around -.21 (Op den Kelder et al., 2018), between mediator and outcome .10 (Gray-Burrows et al., 2019) and predictor and outcome -.39 (McElroy & Hevey, 2014).



Interpretation: With wellbeing as the outcome and executive function as mediator power depends largely on the *b* path which previous research suggests is a relatively small effect. As long as the *b* path effect is around .15 power is acceptable (over 75%) with $n = 400$ or more. It would be important to maximize reliability of the executive function and wellbeing measures.

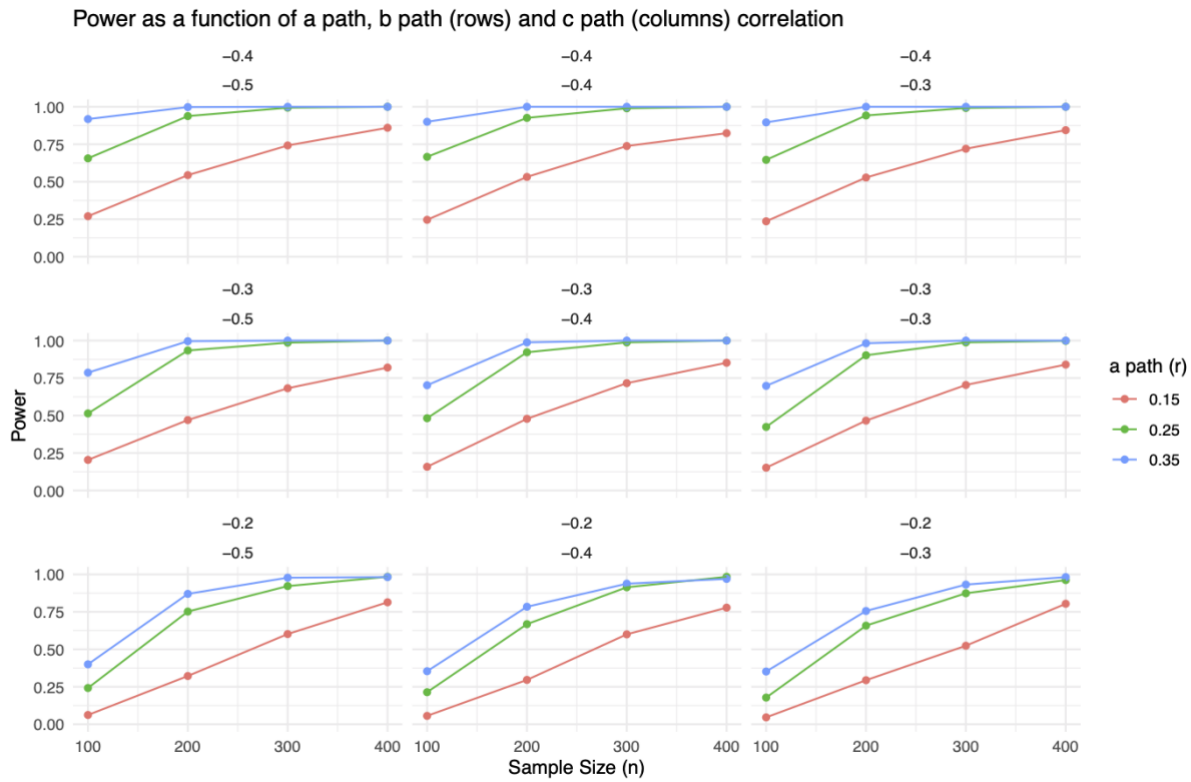
7.6.5.6 Hypothesis 2bii

Predictor: Childhood trauma (CTQ)

Mediator: Impulsivity

Outcome: Wellbeing

From previous research correlation between predictor and mediator is .253 (as above), between mediator and outcome -.302 (Goodwin et al., 2017) and predictor and outcome -.39 (as above).



Interpretation: Overall, power is reasonable for $n > 250$ except when the *a* path correlation is .15 or lower, but even then is acceptable for $n = 400$.

7.6.5.7 Hypothesis 3a

Predictor: Childhood trauma (CTQ)

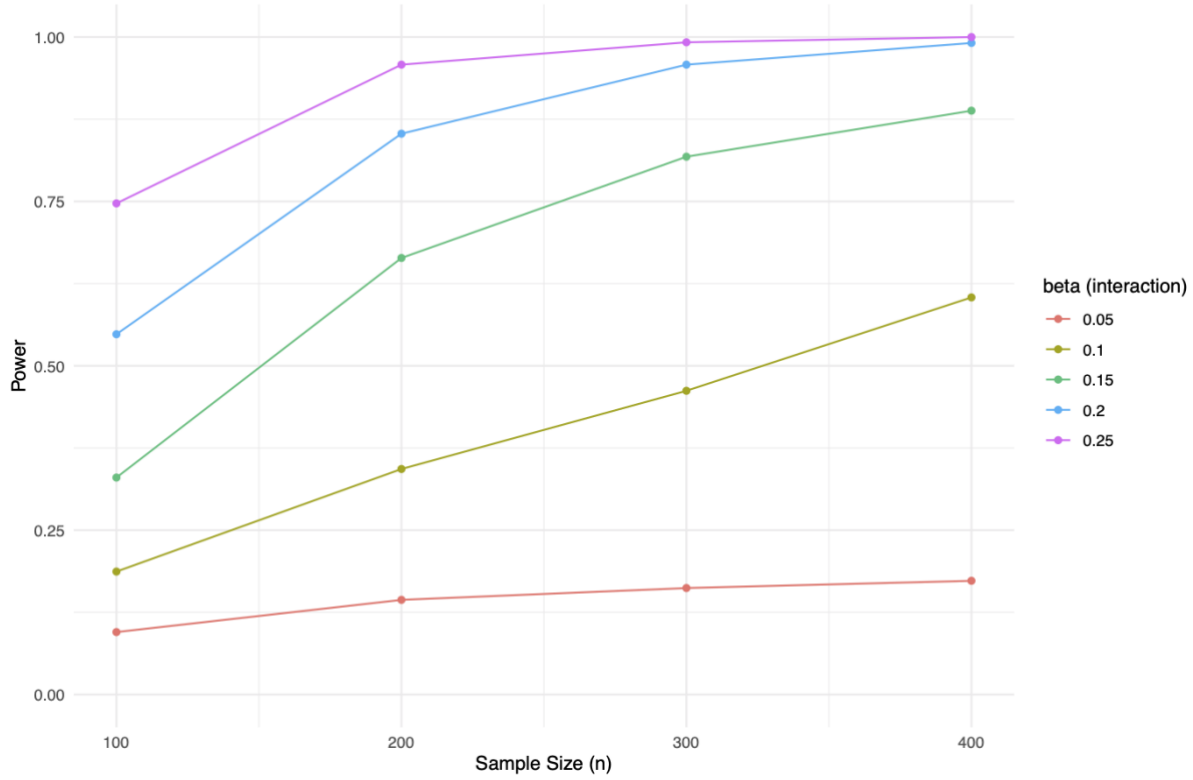
Moderator: Recent stress

Outcome: Impulsivity

Moderator effects (interactions between two continuous predictors) are notoriously low in power. While the correlations between three variables impact the power (as do collinearity with other predictors), what matters most in simulations likely to be the change in standardized coefficient of the predictor when there is a one *SD* increase in the moderator (*beta*). This tends to be small in practice because of range restriction in the product term (predictor times mediator) that is, in effect, the predictor of interest. This tends to lead to small *beta* for the interaction unless extreme values of both predictor and moderator are common (which they tend not to be).

We simulated *beta* from 0.05 to 0.25 for a range of plausible correlations between the variables. (The simulated variables are standardized and therefore centred, but this doesn't impact the estimate of the interaction effect; however centering is advised in the actual analysis to aid interpretation – particularly if there are other covariates). The first

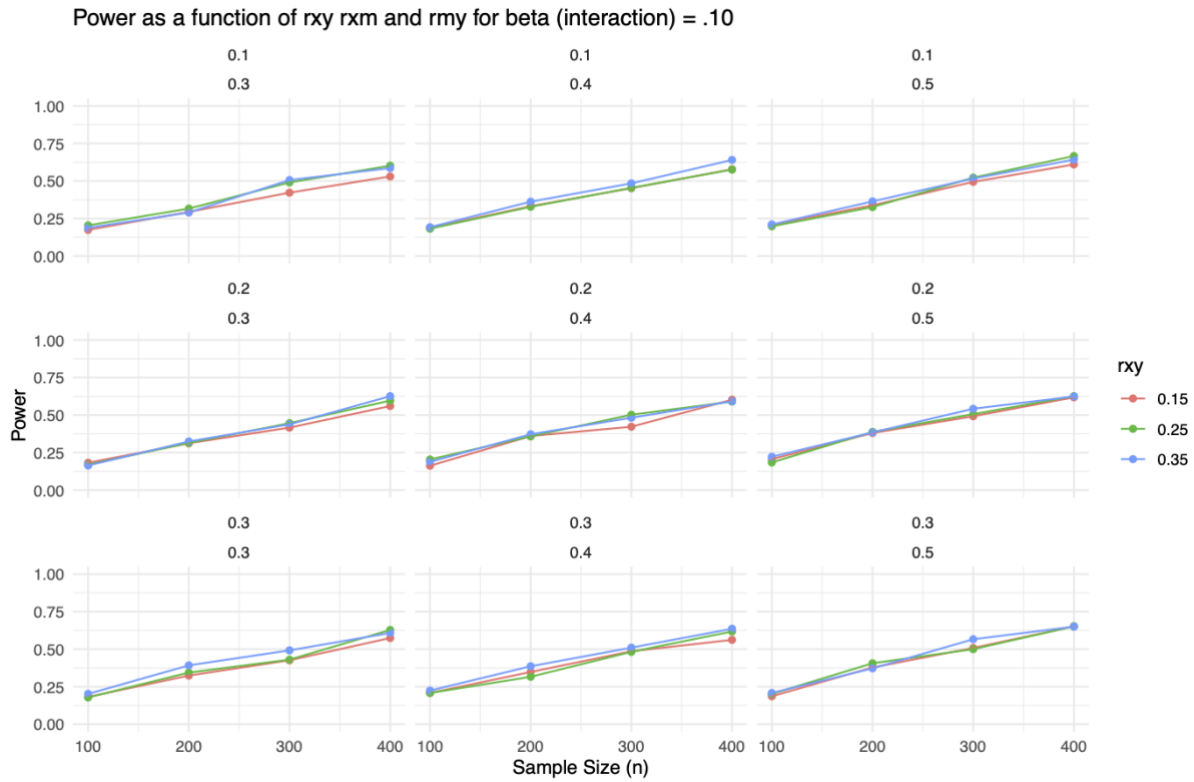
plot shows the power with the predictor-moderation correlation fixed at .412 (McElroy & Hevey, 2014), the predictor-outcome at .253 (as above) and moderator-outcome *r* = .192 (Ansell et al., 2012).



Interpretation: Power to detect the moderator effect is reasonable for *n* = 250 or greater if the *beta* for the interaction is around 0.15 or greater. For small standardized effects (which might include meaningful effects given the presence of range restriction for the moderator) of 0.05 to 0.10 power tends to be poor. However, at least one of the predictors is skewed and that may limit the impact of range restriction (power to detect moderators can be increased by skew and kurtosis as there are more extreme observations). A cautious approach would be to work with samples of 400 plus, but moderator effects are notoriously hard to detect (McClelland & Judd, 1993).

Sensitivity check:

For each moderator analysis we also varied the correlations around the original values (in this case with *beta* = 0.10). This doesn't have a huge impact on the power estimates. The sensitivity plot for Hypothesis 3a is shown below (but not for subsequent analyses as it isn't that informative).



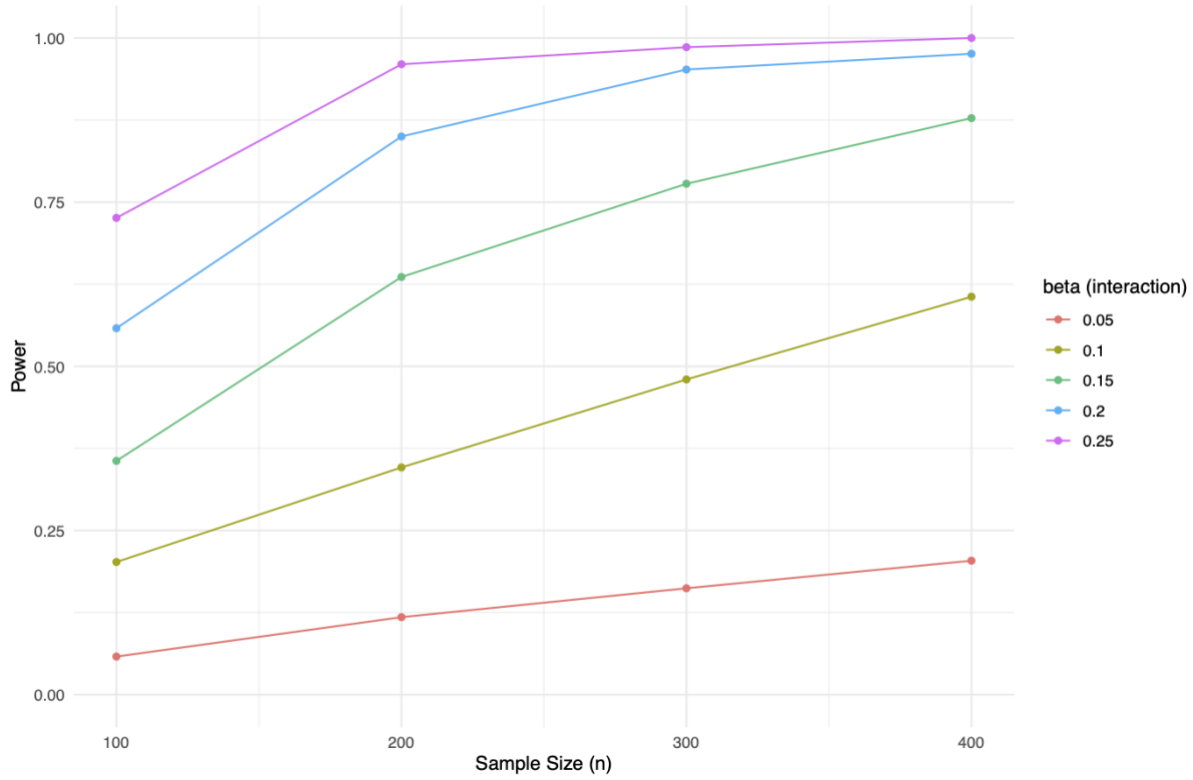
7.6.5.8 Hypothesis 3b

Predictor: Childhood trauma (CTQ)

Moderator: Recent stress

Outcome: Executive function

The plot below shows the power with the predictor-moderation correlation fixed at .412 (as above), the predictor-outcome at -.21 (Op den Kelder et al., 2018) and moderator-outcome *r* of -.10 to -.30 (Shields et al., 2016) representing different measures of executive function.



Interpretation: Power to detect the moderator effect is reasonable for *n* = 300 or greater if the *beta* for the interaction is 0.15 or greater. Power is approaching 60% for *n* = 400 when beta is 0.1. Again it would desirable to have samples of 400 or greater.

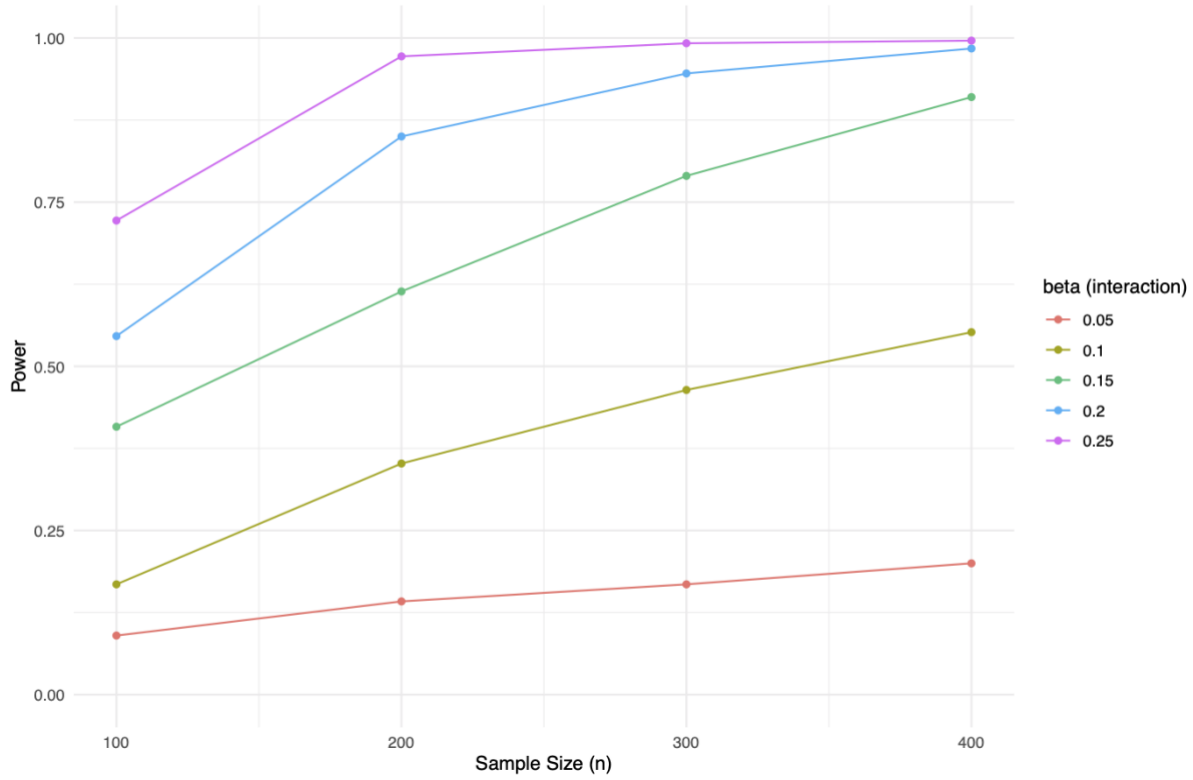
7.6.5.9 Hypothesis 4a

Predictor: Childhood trauma (CTQ)

Moderator: Recent stress

Outcome: Recent suicide ideation

As for hypothesis 3a and 3b we simulated *beta* from 0.05 to 0.25 for a range of plausible correlations between the variables. The plot shows power with the predictor-moderation correlation fixed at .412, the predictor-outcome at .092 (Angelakis et al., 2019) and moderator-outcome *r* = .24 (Polanco-Roman et al., 2016).



Interpretation: Here the pattern is very similar pattern to the hypothesis 3b. Power to detect the moderator effect is reasonable for *n* = 300 or greater if the *beta* for the interaction is 0.15 or greater. As before, it would be desirable to have samples of 400 or greater.

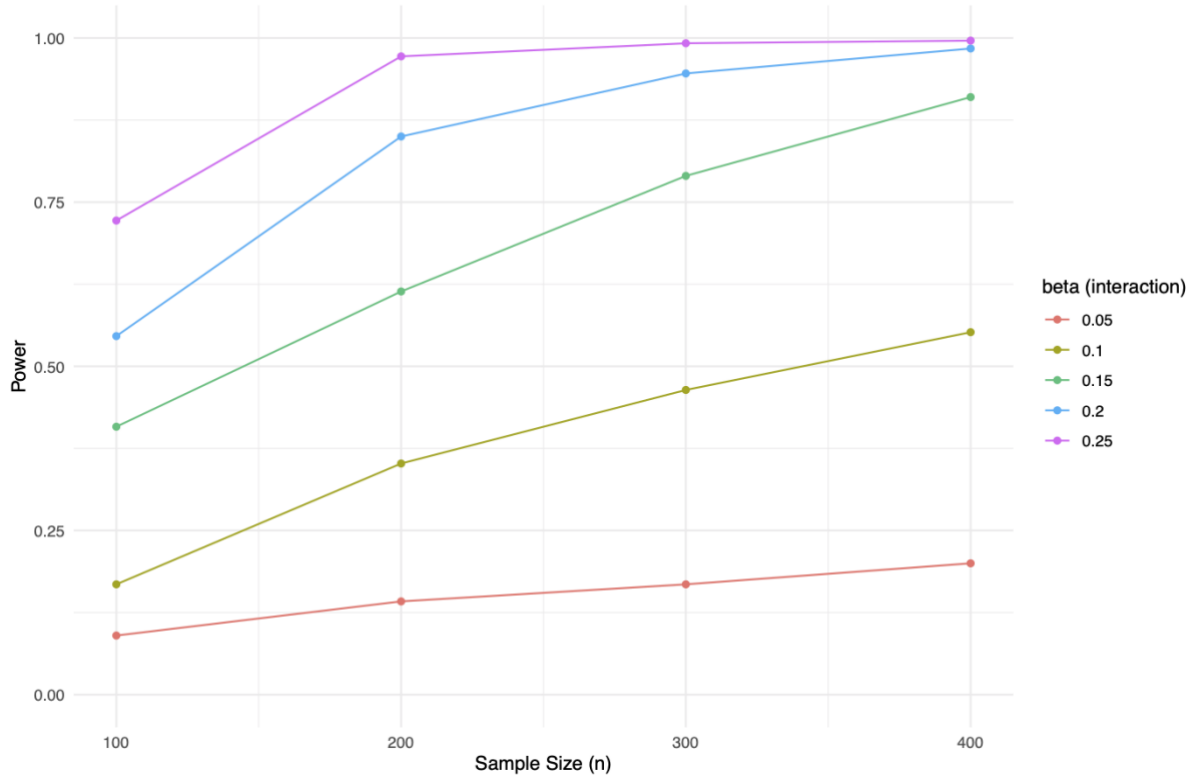
7.6.5.10 Hypothesis 4b

Predictor: Childhood trauma (CTQ)

Moderator: Recent stress

Outcome: Wellbeing

For hypothesis 4b we simulated *beta* from 0.05 to 0.25 for a range of plausible correlations between the variables. The plot shows power with the predictor-moderation correlation fixed at .412, the predictor-outcome at -.39, and moderator-outcome *r* = -.41 (McElroy & Hevey, 2014).



Interpretation: Here the pattern is very similar pattern to the hypothesis 4a. As before, it would be desirable to have samples of 400 or greater.

7.6.6 Missing data & data exclusion

Missing data will include items missed by participants and those who selected the option 'would rather not say' to the APMS suicidal behaviour questions. If a participant had completed at least 75% of a psychological measure, their data will be retained for analyses. Otherwise their scores for that measure will be treated as missing. In cases where the data are missing multiple imputation (MI) or full information maximum likelihood (FIML) methods which assume data are Missing At Random (MAR) (Little, 1988) will be used. MI can be more flexible as it allows the inclusion of auxillary variables that predict missingness but aren't in the model used for analysis, but FIML is implemented in some SEM software (e.g., MPLUS, lavaan) which may be used for some analyses. For null effects additional Bayesian analyses may be conducted with R to obtain Bayes factors to assess the degree of support for the null hypothesis and for computationally demanding analyses (e.g., multiple imputation if the proportion of missing data is high).

7.7 Study 2

7.7.1 Notes on AsPredicted Preregistration

The preregistration represents a larger COVID study in which several manuscripts have been developed. For this study, we refer to the overall aim of the pre-registration; to explore relations between risk factors for suicide during the COVID-19 pandemic and suicidal behaviour. Next, the current study explores preregistered aim 1 which informs hypothesis 1. The preregistered aims 2 and 4 inform hypothesis 2. Lastly, the preregistered aims 2, 4 and 6 inform hypothesis 3.

7.7.2 Unadjusted and adjusted models

Table 18. Effects of COVID-related stress, worry, rumination and time on state impulsivity

	Original, single imputation complete case										Multiple imputation complete case									
	Unadjusted					Adjusted					Unadjusted					Adjusted				
Outcome: impulsivity	β	CoF	SE	df	p-value	β	CoF	SE	df	p-value	β	CoF	SE	df	p-value	β	CoF	SE	df	p-value
Predictor: COVID-related stress																				
Intercept	β_{00}	8.644	0.174	416	<.001	β_{00}	8.210	0.650	414	<.001	β_{00}	2.159	0.043	416	< .001	β_{00}	2.054	0.163	414	< .001
Age	β_{01}	-	-	-	-	β_{01}	-0.043	0.010	414	<.001	β_{01}	-	-	-	-	β_{01}	-0.011	0.003	414	< .001
Gender	β_{02}	-	-	-	-	β_{02}	0.270	0.348	414	.438	β_{02}	-	-	-	-	β_{02}	0.065	0.087	414	.454
History of suicide behaviour	β_{03}	1.122	0.309	416	<.001	β_{03}	0.965	0.305	414	.002	β_{03}	0.293	0.077	416	< .001	β_{03}	0.254	0.077	414	< .001
Level 1 Slope																				
Stress - impulsivity	β_{10}	0.373	0.087	416	<.001	β_{10}	0.368	0.087	416	<.001	β_{10}	0.093	0.021	416	< .001	β_{10}	0.091	0.021	416	< .001
History of suicide behaviour	β_{11}	-0.303	0.158	416	.055	β_{11}	-0.300	0.157	416	.057	β_{11}	-0.076	0.040	416	.057	β_{11}	- 0.074	0.039	416	.059
Predictor: COVID-related rumination																				
Intercept	β_{00}	8.644	0.174	416	<.001	β_{00}	8.181	0.651	414	<.001	β_{00}	2.159	0.043	416	< .001	β_{00}	2.047	0.163	414	< .001
Age	β_{01}	-	-	-	-	β_{01}	-0.043	0.010	414	<.001	β_{01}	-	-	-	-	β_{01}	- 0.011	0.002	414	< .001

Gender	β_{02}	-	-	-	-	β_{02}	0.286	0.349	414	.412	β_{02}	-	-	-	-	β_{02}	0.070	0.087	414	.426
History of suicide behaviour	β_{03}	1.122	0.310	416	<.001	β_{03}	0.964	0.306	414	.002	β_{03}	0.293	0.078	416	< .001	β_{03}	0.254	0.077	414	< .001
Level 1 Slope																				
Rumination – impulsivity	β_{10}	0.226	0.077	416	.004	β_{10}	0.227	0.077	416	.004	β_{10}	0.057	0.019	416	.003	β_{10}	0.057	0.019	416	.003
History of suicide behaviour	β_{11}	-0.062	0.142	416	.660	β_{11}	-0.061	0.142	416	.660	β_{11}	-0.018	0.036	416	.622	β_{11}	-0.018	0.036	416	.625
Predictor: COVID-related worry																				
Intercept	β_{00}	8.644	.174	416	<.001	β_{00}	8.181	.649	414	<.001	β_{00}	2.159	0.043	416	<.001	β_{00}	2.047	0.163	414	< .001
Age	β_{01}	-	-	-	-	β_{01}	-0.044	.010	414	<.001	β_{01}	-	-	-	-	β_{01}	-0.011	0.003	414	< .001
Gender	β_{02}	-	-	-	-	β_{02}	0.287	.348	414	.410	β_{02}	-	-	-	-	β_{02}	0.069	0.087	414	.428
History of suicide behaviour	β_{03}	1.122	.310	416	<.001	β_{03}	0.963	.306	414	.002	β_{03}	0.293	0.078	416	< .001	β_{03}	0.254	0.077	414	< .001
Level 1 Slope																				
Worry- impulsivity	β_{10}	0.183	0.076	416	.017	β_{10}	0.182	0.076	416	.017	β_{10}	0.048	0.019	416	.013	β_{10}	0.048	0.019	416	.013
History of suicide behaviour	β_{11}	0.163	0.145	416	.261	β_{11}	0.162	0.145	416	.264	β_{11}	0.035	0.036	416	.340	β_{11}	0.035	0.036	416	.341

Note: participants who reported *either* lifetime suicidal ideation or suicide attempts were considered to have a history of suicidal behaviour.

Table 19. Effects of COVID-related stress, worry, rumination and time on state executive functioning

	Original, single imputation complete case										Multiple imputation complete case									
	Unadjusted					Adjusted					Unadjusted					Adjusted				
Outcome: executive function	β	CoF	SE	df	p-value	β	CoF	SE	df	p-value	β	CoF	SE	df	p-value	β	CoF	SE	df	
Predictor: COVID-related stress																				
Intercept	β_{00}	12.294	0.227	416	<.001	β_{00}	11.465	0.721	414	<0.001	β_{00}	12.328	0.227	416	< .001	β_{00}	11.520	0.733	414	< .001
Age	β_{01}	-	-	-	-	β_{01}	-0.075	0.012	414	<.001	β_{01}	-	-	-	-	β_{01}	-0.075	0.012	414	< .001
Gender	β_{02}	-	-	-	-	β_{02}	0.511	0.398	414	.200	β_{02}	-	-	-	-	β_{02}	0.498	0.403	414	.218
History of suicidal behaviour	β_{03}	2.935	0.395	416	<.001	β_{03}	2.661	0.387	414	<0.001	β_{03}	2.893	0.397	416	< .001	β_{03}	2.618	0.388	414	< .001
Level 1 Slope																				
Stress - EF	β_{10}	0.346	.095	416	<.001	β_{10}	0.342	0.095	416	<0.001	β_{10}	0.339	0.094	416	< .001	β_{10}	0.335	0.094	416	< .001
History of suicidal behaviour	β_{11}	-0.051	.189	416	.785	β_{11}	-0.047	0.189	416	0.826	β_{11}	-0.033	0.190	416	.864	β_{11}	-0.028	0.190	416	.884
Predictor: COVID-related rumination																				
Intercept	β_{00}	12.294	0.227	416	<.001	β_{00}	11.448	0.719	414	<.001	β_{00}	12.327	0.228	416	< .001	β_{00}	11.504	0.731	414	< .001
Age	β_{01}	-	-	-	-	β_{01}	-0.075	0.012	414	<.001	β_{01}	-	-	-	-	β_{01}	-0.075	0.012	414	< .001
Gender	β_{02}	-	-	-	-	β_{02}	0.051	0.397	414	.190	β_{02}	-	-	-	-	β_{02}	0.507	0.402	414	.208
History of suicidal behaviour	β_{03}	2.935	0.395	416	<.001	β_{03}	2.661	0.387	414	<.001	β_{03}	2.893	0.398	416	< .001	β_{03}	2.619	0.388	414	< .001
Level 1 Slope																				

Rumination - EF	β_{10}	0.233	0.091	416	.011	β_{10}	0.234	0.091	416	.011	β_{10}	0.238	0.090	416	.008	β_{10}	0.237	0.090	416	.009
History of suicidal behaviour	β_{11}	0.105	0.167	416	.530	β_{11}	0.107	0.166	416	.529	β_{11}	0.091	0.169	416	.590	β_{11}	0.092	0.169	416	.586
Predictor: COVID-related worry																				
Intercept	β_{00}	12.294	0.227	416	<.001	β_{00}	11.478	0.722	414	<.001	β_{00}	12.328	0.225	416	< .001	β_{00}	11.537	0.734	414	< .001
Age	β_{01}	-	-	-	-	β_{01}	-0.075	0.012	414	<.001	β_{01}	-	-	-	-	β_{01}	-0.075	0.012	414	< .001
Gender	β_{02}	-	-	-	-	β_{02}	0.504	0.398	414	.207	β_{02}	-	-	-	-	β_{02}	0.489	0.404	414	.226
History of suicidal behaviour	β_{03}	2.935	0.395	416	<.001	β_{03}	2.662	0.387	414	<.001	β_{03}	2.893	0.404	416	< .001	β_{03}	2.620	0.388	414	< .001
Level 1 Slope																				
Worry – EF	β_{10}	0.290	0.092	416	.002	β_{10}	0.290	0.092	416	.002	β_{10}	0.296	0.093	416	.002	β_{10}	0.296	0.092	416	.001
History of suicidal behaviour	β_{11}	0.126	0.176	416	.475	β_{11}	0.126	0.177	416	.476	β_{11}	0.107	0.177	416	.547	β_{11}	0.107	0.177	416	.545

Note: participants who reported *either* lifetime suicidal ideation or suicide attempts were considered to have a history of suicidal behaviour.

7.8 Study 3

Table 20. Adjusted and unadjusted HLM models for Study 3, Research Question 1.

		Unadjusted							Adjusted						
		B	**CoF**	**b**	**SE**	**SD**	**df**	**p-value**	**B**	**CoF**	**b**	**SE**	**SD**	**df**	**p-value**
Outcome:	**Hassles**														
	Intercept	β00	0.4	0.4	0.021	0.305	207	< .001	β00	0.407	0.407	0.073	1.060	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.005	0.000	0.003	0.044	205	0.081
	Sex	β02	-	-	-	-	-	-	β02	-0.004	-0.003	0.046	0.668	205	0.928
	Resilience	β03	-0.007	-0.001	0.003	0.044	207	0.029	β03	-0.049	-0.016	0.024	0.349	205	0.037
	CTQ	β04	0.004	0.0004	0.002	0.029	207	0.078	β04	0.004	0.0001	0.002	0.029	205	0.066
	CTQ*Resilience	β05	0.027	0.037	0.029	0.421	207	0.36	β05	0.027	0.011	0.029	0.421	205	0.343
	Intercept	β00	0.399	0.399	0.021	0.305	207	< .001	β00	0.38	0.38	0.077	1.118	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.005	0.000	0.003	0.044	205	0.104
	Sex	β02	-	-	-	-	-	-	β02	0.012	0.007	0.044	0.639	205	0.791
	Support	β03	-0.046	-0.053	0.024	0.349	207	0.051	β03	-0.042	-0.014	0.024	0.349	205	0.07
	CTQ	β04	0.003	0.000	0.002	0.029	207	0.108	β04	0.003	0.0001	0.002	0.029	205	0.091
	CTQ*Support	β05	-0.002	-0.003	0.032	0.465	207	0.948	β05	-0.0003	0.000	0.031	0.450	205	0.993

Intercept	β00	0.4	0.4	0.021	0.305	207	< .001	β00	0.371	0.371	0.077	1.118	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.005	0.000	0.003	0.044	205	0.128
Sex	β02	-	-	-	-	-	-	β02	0.017	0.010	0.044	0.639	205	0.695
Connectedness	β03	0.028	0.028	0.021	0.305	207	0.194	β03	0.023	0.006	0.021	0.305	205	0.288
CTQ	β04	0.004	0.0004	0.002	0.029	207	0.039	β04	0.004	0.0001	0.002	0.029	205	0.032
CTQ*Connectedness	β05	0.016	0.019	0.025	0.363	207	0.529	β05	0.012	0.004	0.025	0.363	205	0.641
Intercept	β00	0.399	0.399	0.021	0.305	207	< .001	β00	0.404	0.404	0.075	1.089	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.005	0.000	0.003	0.044	205	0.094
Sex	β02	-	-	-	-	-	-	β02	-0.003	-0.002	0.043	0.625	205	0.944
Loneliness	β03	0.074	0.074	0.021	0.305	207	< .001	β03	0.073	0.021	0.022	0.320	205	0.001
CTQ	β04	0.002	0.0002	0.002	0.029	207	0.203	β04	0.002	0.0001	0.002	0.029	205	0.174
CTQ*Loneliness	β05	0.007	0.009	0.027	0.392	207	0.794	β05	0.004	0.001	0.026	0.378	205	0.882
Intercept	β00	0.373	0.373	0.025	0.363	207	< .001	β00	0.356	0.356	0.076	1.104	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.005	0.000	0.003	0.044	205	0.127
Sex	β02	-	-	-	-	-	-	β02	0.011	0.006	0.044	0.639	205	0.811
Suicide	β03	0.08	0.141	0.044	0.639	207	0.07	β03	0.077	0.045	0.044	0.639	205	0.08
CTQ	β04	0.002	0.0002	0.002	0.029	207	0.297	β04	0.002	0.0001	0.002	0.029	205	0.209
CTQ*Suicide	β05	0.064	0.156	0.061	0.886	207	0.297	β05	0.052	0.041	0.06	0.872	205	0.386

Outcome:	**Sleep quality**													
	Intercept	β00	4.767	4.767	0.077	1.118	207	< .001	β00	5.043	5.043	0.283	4.111	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.003	0.000	0.011	0.160	205	0.815
	Sex	β02	-	-	-	-	-	-	β02	-0.163	-0.093	0.162	2.353	205	0.316
	Resilience	β03	0.237	0.255	0.083	1.206	207	0.005	β03	0.22	0.065	0.083	1.206	205	0.009
	CTQ	β04	-0.227	-0.221	0.075	1.089	207	0.003	β04	-0.227	-0.061	0.076	1.104	205	0.003
	CTQ*Resilience	β05	-0.067	-0.068	0.078	1.133	207	0.39	β05	-0.072	-0.020	0.078	1.133	205	0.362
	Intercept	β00	4.767	4.767	0.079	1.148	207	< .001	β00	5.187	5.187	0.291	4.227	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.002	0.000	0.012	0.174	205	0.863
	Sex	β02	-	-	-	-	-	-	β02	-0.249	-0.140	0.164	2.382	205	0.131
	Support	β03	0.149	0.179	0.095	1.380	207	0.118	β03	0.143	0.047	0.096	1.394	205	0.136
	CTQ	β04	-0.226	-0.237	0.083	1.206	207	0.007	β04	-0.222	-0.063	0.083	1.206	205	0.008
	CTQ*Support	β05	-0.114	-0.111	0.077	1.118	207	0.147	β05	-0.114	-0.031	0.079	1.148	205	0.151
	Intercept	β00	4.767	4.767	0.079	1.148	207	< .001	β00	5.211	5.211	0.294	4.271	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.001	0.000	0.012	0.174	205	0.907
	Sex	β02	-	-	-	-	-	-	β02	-0.263	-0.148	0.166	2.411	205	0.115
	Connectedness	β03	-0.009	-0.010	0.087	1.264	207	0.922	β03	-0.014	-0.004	0.087	1.264	205	0.87
	CTQ	β04	-0.271	-0.261	0.076	1.104	207	< .001	β04	-0.264	-0.067	0.075	1.089	205	< .001

	CTQ*Connectedness	β05	-0.001	-0.001	0.071	1.031	207	0.99	β05	-0.0002	0.000	0.071	1.031	205	0.997
	Intercept	β00	4.767	4.767	0.077	1.118	207	< .001	β00	5.071	5.071	0.283	4.111	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.002	0.000	0.011	0.160	205	0.853
	Sex	β02	-	-	-	-	-	-	β02	-0.18	-0.103	0.162	2.353	205	0.266
	Loneliness	β03	-0.315	-0.327	0.08	1.162	207	< .001	β03	-0.303	-0.089	0.083	1.206	205	< .001
	CTQ	β04	-0.17	-0.181	0.082	1.191	207	0.038	β04	-0.169	-0.049	0.082	1.191	205	0.041
	CTQ*Loneliness	β05	0.055	0.055	0.077	1.118	207	0.474	β05	0.052	0.015	0.079	1.148	205	0.501
	Intercept	β00	4.991	4.991	0.098	1.424	207	< .001	β00	5.371	5.371	0.288	4.183	205	< .001
	Age	β01	-	-	-	-	-	-	β01	0.0002	0.000	0.012	0.174	205	0.983
	Sex	β02	-	-	-	-	-	-	β02	-0.228	-0.126	0.159	2.310	205	0.152
	Suicide	β03	-0.657	-1.133	0.169	2.455	207	< .001	β03	-0.641	-0.374	0.168	2.440	205	< .001
	CTQ	β04	-0.315	-0.350	0.109	1.583	207	0.004	β04	-0.313	-0.121	0.111	1.612	205	0.005
	CTQ*Suicide	β05	0.289	0.454	0.154	2.237	207	0.062	β05	0.293	0.160	0.157	2.281	205	0.064
Outcome:	**Morning tiredness**														
	Intercept	β00	2.61	2.61	0.048	0.697	207	< .001	β00	2.607	2.607	0.181	2.629	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.009	0.000	0.007	0.102	205	0.188
	Sex	β02	-	-	-	-	-	-	β02	0.003	0.002	0.104	1.511	205	0.98
	Resilience	β03	-0.272	-0.300	0.053	0.770	207	< .001	β03	-0.268	-0.081	0.055	0.799	205	< .001

CTQ	β04	0.054	0.061	0.054	0.784	207	0.327	β04	0.056	0.017	0.054	0.784	205	0.301
CTQ*Resilience	β05	0.011	0.016	0.069	1.002	207	0.878	β05	0.012	0.005	0.068	0.988	205	0.862
Intercept	β00	2.611	2.611	0.051	0.741	207	< .001	β00	2.398	2.398	0.19	2.760	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.011	0.000	0.007	0.102	205	0.15
Sex	β02	-	-	-	-	-	-	β02	0.126	0.072	0.108	1.569	205	0.244
Support	β03	-0.088	-0.090	0.052	0.755	207	0.093	β03	-0.078	-0.023	0.055	0.799	205	0.16
CTQ	β04	0.801	0.942	0.06	0.872	207	0.178	β04	0.084	0.024	0.055	0.799	205	0.13
CTQ*Support	β05	0.046	0.051	0.057	0.828	207	0.413	β05	0.05	0.014	0.055	0.799	205	0.361
Intercept	β00	2.611	2.611	0.051	0.741	207	<.001	β00	2.384	2.384	0.186	2.702	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.009	0.000	0.007	0.102	205	0.224
Sex	β02	-	-	-	-	-	-	β02	0.135	0.078	0.107	1.554	205	0.208
Connectedness	β03	0.056	0.060	0.055	0.799	207	0.31	β03	0.048	0.014	0.054	0.784	205	0.379
CTQ	β04	0.099	0.101	0.052	0.755	207	0.059	β04	0.099	0.028	0.052	0.755	205	0.06
CTQ*Connectedness	β05	0.055	0.057	0.053	0.770	207	0.304	β05	0.046	0.013	0.052	0.755	205	0.375
Intercept	β00	2.612	2.612	0.049	0.712	207	< .001	β00	2.491	2.491	0.179	2.600	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.01	0.000	0.007	0.102	205	0.159
Sex	β02	-	-	-	-	-	-	β02	0.072	0.041	0.102	1.482	205	0.482
Loneliness	β03	0.24	0.269	0.055	0.799	207	< .001	β03	0.233	0.073	0.056	0.813	205	< .001

	CTQ	β04	0.025	0.030	0.058	0.842	207	0.666	β04	0.028	0.009	0.058	0.842	205	0.627
	CTQ*Loneliness	β05	0.013	0.016	0.06	0.872	207	0.825	β05	0.008	0.003	0.06	0.872	205	0.895
	Intercept	β00	2.471	2.471	0.061	0.886	207	< .001	β00	2.287	2.287	0.176	2.557	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.012	0.000	0.007	0.102	205	0.095
	Sex	β02	-	-	-	-	-	-	β02	0.113	0.065	0.101	1.467	205	0.265
	Suicide	β03	0.409	0.758	0.113	1.641	207	< .001	β03	0.396	0.254	0.113	1.641	205	< .001
	CTQ	β04	0.123	0.149	0.074	1.075	207	0.098	β04	0.137	0.058	0.075	1.089	205	0.07
	CTQ*Suicide	β05	-0.151	-0.287	0.116	1.685	207	0.193	β05	-0.181	-0.115	0.112	1.627	205	0.108
Outcome:	**Stress**														
	Intercept	β00	1.217	1.217	0.035	0.508	207	< .001	β00	1.25	1.25	0.13	1.888	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.002	0.000	0.005	0.073	205	0.698
	Sex	β02	-	-	-	-	-	-	β02	-0.019	-0.011	0.073	1.060	205	0.791
	Resilience	β03	-0.236	-0.243	0.036	0.523	207	< .001	β03	-0.237	-0.067	0.037	0.537	205	< .001
	CTQ	β04	0.109	0.112	0.036	0.523	207	0.003	β04	0.109	0.030	0.036	0.523	205	0.003
	CTQ*Resilience	β05	0.029	0.035	0.042	0.610	207	0.485	β05	0.029	0.009	0.041	0.596	205	0.492
	Intercept	β00	1.217	1.217	0.038	0.552	207	< .001	β00	1.076	1.076	0.137	1.990	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.003	0.000	0.005	0.073	205	0.579
	Sex	β02	-	-	-	-	-	-	β02	0.084	0.048	0.079	1.148	205	0.288

Support	β03	-0.126	-0.129	0.039	0.567	207	0.001	β03	-0.122	-0.037	0.041	0.596	205	0.003
CTQ	β04	0.113	0.119	0.04	0.581	207	0.005	β04	0.113	0.033	0.04	0.581	205	0.005
CTQ*Support	β05	0.064	0.071	0.042	0.610	207	0.124	β05	0.065	0.020	0.042	0.610	205	0.116
Intercept	β00	1.218	1.218	0.039	0.567	207	< .001	β00	1.055	1.055	0.135	1.961	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.002	0.000	0.006	0.087	205	0.722
Sex	β02	-	-	-	-	-	-	β02	0.096	0.055	0.078	1.133	205	0.22
Connectedness	β03	0.041	0.043	0.041	0.596	207	0.315	β03	0.04	0.012	0.042	0.610	205	0.342
CTQ	β04	0.145	0.134	0.036	0.523	207	< .001	β04	0.144	0.038	0.036	0.523	205	< .001
CTQ*Connectedness	β05	0.042	0.042	0.039	0.567	207	0.283	β05	0.04	0.0116	0.039	0.567	205	0.306
Intercept	β00	1.217	1.217	0.036	0.523	207	< .001	β00	1.159	1.159	0.131	1.903	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.003	0.000	0.005	0.073	205	0.547
Sex	β02	-	-	-	-	-	-	β02	0.034	0.020	0.076	1.104	205	0.652
Loneliness	β03	0.232	0.245	0.038	0.552	207	< .001	β03	0.229	0.070	0.04	0.581	205	< .001
CTQ	β04	0.076	0.076	0.036	0.523	207	0.04	β04	0.077	0.0217	0.037	0.537	205	0.037
CTQ*Loneliness	β05	-0.023	-0.022	0.035	0.508	207	0.51	β05	-0.025	-0.007	0.035	0.508	205	0.484
Intercept	β00	1.124	1.124	0.046	0.668	207	< .001	β00	0.994	0.994	0.13	1.888	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.003	0.000	0.005	0.073	205	0.586
Sex	β02	-	-	-	-	-	-	β02	0.079	0.047	0.078	1.133	205	0.314

	Suicide	β03	0.273	0.499	0.084	1.220	207	0.001	β03	0.267	0.177	0.086	1.249	205	0.002
	CTQ	β04	0.111	0.111	0.046	0.668	207	0.016	β04	0.113	0.040	0.046	0.668	205	0.015
	CTQ*Suicide	β05	0.028	0.048	0.079	1.148	207	0.721	β05	0.021	0.013	0.079	1.148	205	0.795
Outcome:	**EF**														
	Intercept	β00	8.565	8.565	0.15	2.179	207	< .001	β00	8.792	8.792	0.535	7.771	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.054	-0.002	0.022	0.320	205	0.013
	Sex	β02	-	-	-	-	-	-	β02	-0.134	-0.075	0.3	4.358	205	0.656
	Resilience	β03	-0.939	-0.958	0.153	2.222	207	< .001	β03	-0.935	-0.274	0.157	2.281	205	< .001
	CTQ	β04	0.387	0.397	0.154	2.237	207	0.013	β04	0.402	0.138	0.183	2.658	205	0.029
	CTQ*Resilience	β05	0.094	0.096	0.153	2.222	207	0.54	β05	0.093	0.029	0.167	2.426	205	0.579
	Intercept	β00	8.565	8.565	0.155	2.252	207	< .001	β00	8.156	8.156	0.551	8.004	205	<.001
	Age	β01	-	-	-	-	-	-	β01	-0.053	-0.002	0.022	0.320	205	0.014
	Sex	β02	-	-	-	-	-	-	β02	0.243	0.140	0.318	4.619	205	0.445
	Support	β03	-0.737	-0.732	0.154	2.237	207	< .001	β03	-0.699	-0.200	0.158	2.295	205	< .001
	CTQ	β04	0.312	0.354	0.176	2.557	207	0.078	β04	0.33	0.104	0.173	2.513	205	0.058
	CTQ*Support	β05	-0.044	-0.048	0.168	2.440	207	0.796	β05	-0.027	-0.008	0.164	2.382	205	0.871
	Intercept	β00	8.566	8.566	0.159	2.310	207	< .001	β00	8.007	8.007	0.568	8.251	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.049	-0.002	0.022	0.320	205	0.028

	Sex	β02	-	-	-	-	-	-	β02	0.332	0.190	0.325	4.721	205	0.308
	Connectedness	β03	0.378	0.397	0.167	2.426	207	0.025	β03	0.33	0.097	0.167	2.426	205	0.049
	CTQ	β04	0.499	0.552	0.176	2.557	207	0.005	β04	0.511	0.155	0.172	2.498	205	0.003
	CTQ*Connectedness	β05	0.219	0.191	0.139	2.019	207	0.116	β05	0.177	0.043	0.138	2.005	205	0.2
	Intercept	β00	8.564	8.564	0.145	2.106	207	< .001	β00	8.507	8.507	0.513	7.452	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.054	-0.002	0.02	0.291	205	0.007
	Sex	β02	-	-	-	-	-	-	β02	0.035	0.020	0.295	4.285	205	0.907
	Loneliness	β03	1.114	1.137	0.148	2.150	207	< .001	β03	1.099	0.326	0.152	2.208	205	< .001
	CTQ	β04	0.152	0.168	0.16	2.324	207	0.341	β04	0.172	0.053	0.158	2.295	205	0.278
	CTQ*Loneliness	β05	0.16	0.164	0.149	2.164	207	0.281	β05	0.13	0.037	0.146	2.121	205	0.371
	Intercept	β00	8.131	8.131	0.173	2.513	207	< .001	β00	7.744	7.744	0.562	8.164	205	< .001
	Age	β01	-	-	-	-	-	-	β01	-0.055	-0.002	0.022	0.320	205	0.014
	Sex	β02	-	-	-	-	-	-	β02	0.237	0.135	0.321	4.663	205	0.461
	Suicide	β03	1.272	2.618	0.356	5.171	207	< .001	β03	1.238	0.784	0.356	5.171	205	< .001
	CTQ	β04	0.237	0.211	0.154	2.237	207	0.125	β04	0.296	0.081	0.153	2.222	205	0.054
	CTQ*Suicide	β05	0.451	1.066	0.409	5.941	207	0.271	β05	0.326	0.231	0.398	5.781	205	0.414
Outcome:	**impulsivity**														
	Intercept	β00	5.780	5.780	0.104	1.511	207	< .001	β00	5.979	5.979	0.363	5.273	205	<.001

Age	β01	-	-	-	-	-	-	β01	-0.05	-0.002	0.015	0.218	205	0.001
Sex	β02	-	-	-	-	-	-	β02	-0.117	-0.066	0.205	2.978	205	0.57
Resilience	β03	-0.397	-0.428	0.112	1.627	207	< .001	β03	-0.392	-0.119	0.11	1.598	205	< .001
CTQ	β04	0.335	0.506	0.157	2.281	207	0.034	β04	0.348	0.147	0.153	2.222	205	0.024
CTQ*Resilience	β05	0.178	0.241	0.141	2.048	207	0.207	β05	0.177	0.067	0.137	1.990	205	0.195
Intercept	β00	5.779	5.779	0.104	1.511	207	<.001	β00	5.768	5.768	0.389	5.651	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.048	-0.002	0.016	0.232	205	0.002
Sex	β02	-	-	-	-	-	-	β02	0.007	0.004	0.219	3.181	205	0.973
Support	β03	-0.452	-0.474	0.109	1.583	207	< .001	β03	-0.424	-0.116	0.106	1.540	205	< .001
CTQ	β04	0.246	0.326	0.138	2.005	207	0.077	β04	0.266	0.095	0.139	2.019	205	0.057
CTQ*Support	β05	-0.023	-0.035	0.157	2.281	207	0.882	β05	-0.009	-0.004	0.157	2.281	205	0.957
Intercept	β00	5.780	5.780	0.108	1.569	207	< .001	β00	5.689	5.689	0.407	5.912	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.05	-0.002	0.016	0.232	205	0.002
Sex	β02	-	-	-	-	-	-	β02	0.055	0.031	0.228	3.312	205	0.81
Connectedness	β03	0.112	0.118	0.114	1.656	207	0.326	β03	0.059	0.016	0.112	1.627	205	0.596
CTQ	β04	0.379	0.477	0.136	1.976	207	0.006	β04	0.398	0.133	0.136	1.976	205	0.004
CTQ*Connectedness	β05	0.121	0.128	0.114	1.656	207	0.291	β05	0.08	0.023	0.117	1.700	205	0.494
Intercept	β00	5.779	5.779	0.104	1.511	207	< .001	β00	5.878	5.878	0.403	5.854	205	< .001

Age	β01	-	-	-	-	-	-	β01	-0.05	-0.002	0.016	0.232	205	0.002
Sex	β02	-	-	-	-	-	-	β02	-0.058	-0.032	0.225	3.268	205	0.799
Loneliness	β03	0.437	0.429	0.102	1.482	207	< .001	β03	0.429	0.109	0.102	1.482	205	< .001
CTQ	β04	0.237	0.289	0.127	1.845	207	0.064	β04	0.255	0.081	0.128	1.859	205	0.047
CTQ*Loneliness	β05	0.086	0.104	0.126	1.830	207	0.492	β05	0.058	0.018	0.126	1.830	205	0.647
Intercept	β00	5.646	5.646	0.126	1.830	207	< .001	β00	5.602	5.602	0.414	6.014	205	< .001
Age	β01	-	-	-	-	-	-	β01	-0.051	-0.002	0.016	0.232	205	0.001
Sex	β02	-	-	-	-	-	-	β02	0.03	0.017	0.229	3.326	205	0.895
Suicide	β03	0.393	0.699	0.224	3.254	207	0.081	β03	0.373	0.196	0.218	3.167	205	0.089
CTQ	β04	0.287	0.269	0.118	1.714	207	0.015	β04	0.346	0.099	0.118	1.714	205	0.004
CTQ*Suicide	β05	0.175	0.450	0.324	4.706	207	0.591	β05	0.058	0.045	0.32	4.648	205	0.855

7.9 Study 4

Figure 15. High resolution plot of effect sizes (Hedge's g) and 95% CI of stress management interventions on cortisol outcomes.

Study name	Outcome	Time point	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Basso 2019	Saliva	Post	0.671	0.323	0.104	0.038	1.304	2.078	0.038
Beerse 2020	Saliva	Post	-0.000	0.226	0.051	-0.443	0.443	-0.000	1.000
Benevenutti 2017	Saliva	Post	0.083	0.284	0.081	-0.474	0.640	0.293	0.770
Bittman 2001	Blood	Post	-0.084	0.255	0.065	-0.584	0.415	-0.331	0.741
Bottaccioli 2020	Saliva	Post	0.339	0.312	0.098	-0.273	0.951	1.087	0.277
Bowden 2012	Saliva	Post	0.114	0.344	0.118	-0.560	0.788	0.331	0.741
Brinkmann 2020	Saliva	Post	0.357	0.299	0.089	-0.229	0.943	1.195	0.232
Choi 2022	Saliva	Post	0.098	0.303	0.092	-0.496	0.692	0.324	0.746
Christopher 2018	Saliva	Post	0.591	0.285	0.081	0.033	1.150	2.077	0.038
Dolbier 2012	Saliva	Post	0.063	0.176	0.031	-0.282	0.408	0.358	0.720
Domes 2018	Saliva	Post	0.733	0.304	0.092	0.138	1.328	2.413	0.016
Errazuriz 2020	Saliva	Post	-0.105	0.256	0.066	-0.607	0.397	-0.412	0.681
Fan 2014	Saliva	Post	0.880	0.351	0.124	0.191	1.569	2.503	0.012
Feicht 2013	Saliva	Post	0.291	0.324	0.105	-0.343	0.925	0.899	0.369
Fendel 2021	Hair	Post	0.229	0.194	0.038	-0.152	0.609	1.178	0.239
Flook 2013	Saliva	Post	0.964	0.479	0.230	0.024	1.904	2.011	0.044
Fotiou 2016	Saliva	Post	-0.507	0.261	0.068	-1.019	0.006	-1.939	0.053
Gaab 2006	Saliva	Post	-1.188	0.401	0.161	-1.974	-0.403	-2.965	0.003
Gallego-Gomez 2020	Blood	Post	0.682	0.193	0.037	0.303	1.060	3.529	0.000
Galvin 2006	Saliva	Post	0.544	0.511	0.261	-0.457	1.545	1.065	0.287
Gardi 2022	Saliva	Post	1.153	0.226	0.051	0.710	1.596	5.102	0.000
Goldberg 2014	Hair	Post	0.276	0.454	0.206	-0.614	1.166	0.608	0.543
Gothe 2016	Saliva	Post	-0.496	0.202	0.041	-0.892	-0.100	-2.458	0.014
Graham 2022	Saliva	Post	0.979	0.339	0.115	0.315	1.643	2.892	0.004
Granath 2006	Saliva	Post	0.241	0.351	0.123	-0.447	0.930	0.687	0.492
Hilcove 2021	Saliva	Post	0.228	0.258	0.066	-0.277	0.733	0.884	0.377
Ho 2020	Saliva	Post	0.042	0.276	0.076	-0.499	0.583	0.152	0.879
Hwang 2013	Saliva	Post	-0.071	0.335	0.112	-0.727	0.586	-0.211	0.833
Iglesisas 2012	Saliva	Post	2.395	0.401	0.161	1.609	3.182	5.972	0.000
Jensen 2012	Saliva	Post	0.464	0.325	0.106	-0.173	1.102	1.428	0.153
Jensen 2015	Saliva	Post	-0.162	0.308	0.095	-0.766	0.441	-0.527	0.598
Johansson 2006	Blood	Post	0.120	0.534	0.285	-0.925	1.166	0.226	0.822
Kim 2016	Blood	Post	0.009	0.216	0.047	-0.414	0.433	0.044	0.965
Kotozaki 2014	Saliva	Post	0.808	0.370	0.137	0.082	1.534	2.182	0.029
Lai 2011	Blood	Post	0.514	0.194	0.038	0.133	0.894	2.645	0.008
Laudenslager 2015	Saliva	Post	-0.137	0.198	0.039	-0.525	0.251	-0.693	0.488
Limm 2011	Saliva	Post	0.258	0.161	0.026	-0.058	0.574	1.601	0.109
Macdonald 2018	Saliva	Post	0.551	0.483	0.233	-0.395	1.497	1.142	0.253
Maclean 1997	Blood	Post	1.783	0.432	0.187	0.937	2.630	4.129	0.000
McGrady 1992	Blood	Post	-0.019	0.351	0.124	-0.707	0.670	-0.053	0.958
McKinney 1997	Blood	Post	0.342	0.434	0.188	-0.508	1.193	0.789	0.430
Mohan 2011	Blood	Post	0.751	0.491	0.241	-0.211	1.713	1.530	0.126
Nyklicek 2013	Saliva	Post	0.311	0.232	0.054	-0.143	0.765	1.343	0.179
Oken 2010	Saliva	Post	0.324	0.365	0.133	-0.391	1.039	0.888	0.374
Oken 2017	Saliva	Post	0.211	0.177	0.031	-0.135	0.557	1.194	0.232
Pawlow 2005	Saliva	Post	0.314	0.307	0.094	-0.287	0.915	1.023	0.306
Pema 1998	Blood	Post	0.418	0.339	0.115	-0.247	1.083	1.232	0.218
Repo 2022	Hair	Post	0.139	0.224	0.050	-0.301	0.578	0.618	0.537
Rodriguez-Jiminez 2022	Saliva	Post	0.500	0.365	0.133	-0.215	1.214	1.371	0.170
Rosenkranz 2013	Saliva	Post	0.169	0.282	0.079	-0.383	0.721	0.600	0.548
Schakel 2019	Saliva	Post	0.323	0.209	0.044	-0.086	0.733	1.548	0.122
Sousa 2021	Blood	Post	0.135	0.310	0.096	-0.473	0.744	0.436	0.663
Thompson 2011	Saliva	Post	0.098	0.341	0.116	-0.569	0.766	0.288	0.773
Tsiouli 2014	Saliva	Post	0.431	0.302	0.091	-0.161	1.024	1.427	0.154
Turan 2015	Saliva	Post	0.048	0.236	0.056	-0.415	0.511	0.203	0.839
Turner 2020	Blood	Post	-0.401	0.290	0.084	-0.971	0.168	-1.382	0.167
Vandana 2011	Blood	Post	0.414	0.223	0.050	-0.023	0.851	1.859	0.063
Waelde 2017	Saliva	Post	0.157	0.356	0.127	-0.541	0.854	0.440	0.660
Pooled			0.282	0.059	0.004	0.166	0.398	4.749	0.000
Prediction Interval			0.282			-0.406	0.970		

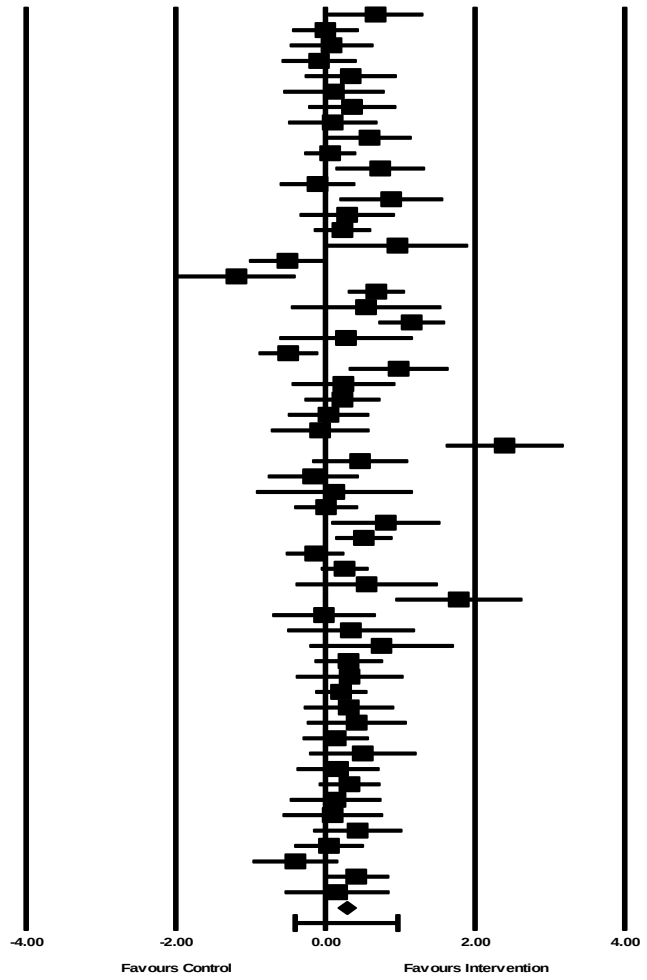


Table 21. Summary of the included study characteristics (n = 58)

Study name	Participants (age in years and gender distribution)	Risk for elevated cortisol levels	Intervention (type of intervention)	Intervention (sample size)	Control (type of intervention)	Control (sample size)	Total intervention time	Cortisol measurements
Basso 2019	age: not reported; 64% female	No risk	Meditation	19	Active (podcast listening)	20	12. 1 h (One initial 17-minute introductory meditation, followed by 8 weeks of daily meditation lasting 13 minutes)	One saliva sample taken at pre- and post-intervention between 1 - 5pm; PM
Beerse 2020	age: *M* = 19.7 , *SD = 1.557, range = 18 - 24 ;* gender: 85.7% female	No risk	Mindfulness based art therapy	41	Active (neutral clay task)	36	2.5 h (5-week intervention covering 10 modules each lasting 15 minutes, the first and last being in person and facilitated)	One saliva sample taken pre- and post-intervention at a time slot between 7 and 10am; AM
Benevenutti 2017	age: *M* = 22.9, *SD = 3.5;* gender: 45.8% female	No risk	Hatha yoga	12	Inactive (watching TV)	12	0.5 h (one 30-minute session of yoga)	between 1 and 4pm; PM
Bittman 2001	age: not reported*;* gender: 48.3% female	No risk	Music therapy	30	Inactive (read magazines)	30	1 h (one session of group drumming and guided imagery)	One blood sample drawn; PM

Bottaccioli 2020	age: *M* = 24.6, *SD = 3.39;* gender: 50% female	No risk	Meditation	20	Active (academic lessons)	20	30 h (4 consecutive days consisting of 15h of learning and 15h of meditation)	One baseline saliva sample taken 30 minutes after waking pre- and post-intervention; AM
Bowden 2012	age: *M* = 34, *range = 18 - 50;* gender: 63.64% female	No risk	Mindfulness	12	Yoga	12	24.2 h (two 75-minute sessions for 5 consecutive weeks. At home practice for 10 minutes each day)	Two saliva samples taken after 10 and 40 minutes into the assessment pre- and post between 11am and 3pm; AM - PM
Bowden 2012	age: *M* = 34, *range = 18 - 50;* gender: 63.64% female	No risk	Mindfulness	12	Brain Wave Vibration	9	24.2 h (two 75-minute sessions for 5 consecutive weeks. At home practice for 10 minutes each day)	Two saliva samples taken after 10 and 40 minutes into the assessment pre- and post between 11am and 3pm; AM - PM
Brinkmann 2020	age: *M* = 43.27, *SD = 10.45;* gender: 71.2% female	No risk	Mindfulness	18	Active (heart rate variability-biofeedback)	19	36.6 h (6-week intervention consisting of four consecutive half days and 30 minute daily self-guided training for 6 weeks. Two booster	One saliva sample after waking; AM

							sessions in weeks 1 and 3)	
Brinkmann 2020	age: M = 43.27, SD = 10.45; gender: 71.2% female	No risk	Mindfulness	18	Waitlist	15	36.6 h (6-week intervention consisting of four consecutive half days and 30 minute daily self-guided training for 6 weeks. Two booster sessions in weeks 1 and 3)	One saliva sample after waking; AM
Christopher 2018	age: *M* = 44, *SD* = *6.03, range = ;* gender: 11.5% female	Stress risk (law enforcement officers)	Mindfulness	24	Inactive (no intervention)	25	22 h (Eight-week intervention with weekly 2-hour sessions and one extended 6-hour class in week 7)	Three saliva samples taken at awakening, +30 and +45 minutes after awakening on three consecutive days pre- and post-intervention to calculate AUCi; AM
Danucalov 2013	age: *M* = 54.45, *SD* = *8.15;* gender: 89.13% female	Stress risk (caregivers)	Yoga and compassion meditation	25	Inactive (no intervention)	21	30 h (2 months interventions with three sessions per	Two saliva samples taken after waking and +30 minutes

							week s lasting 1 hour and 15 minutes)	after on two consecutive days pre- and post-intervention; AM
Dolbier 2012	age: *M* = 19, *SD =1.6;* gender: 58.6% female	No risk	Abbreviated progressive muscle relaxation	66	Inactive (resting)	62	0.3 h (single session of APMR lasting 20 minutes)	One saliva sample taken in the morning; AM
Domes 2019	age: *M* = 46.8, *SD = 9.2;* gender: 0% female	No risk	Internet based stress management	16	Active (progressive muscle relaxation)	16	4.5 h (6-week intervention with weekly modules lasting 45 minutes)	Cortisol sampled pre-intervention and post-intervention prior to the TSST; PM
Domes 2019	age: *M* = 46.8, *SD = 9.2;* gender: 0% female	No risk	Internet based stress management	16	Waitlist	17	4.5 h (6-week intervention with weekly modules lasting 45 minutes)	Cortisol sampled pre-intervention and post-intervention prior to the TSST; PM
Errazuriz 2020	age: *M* = 40.2, *SD = 11.7;* gender: 98.1% female	Stress risk (healthcare workers)	Mindfulness based stress reduction	21	Active (stress management course)	23	16 h (8-week intervention with a weekly 2-hour session)	Cortisol sampled upon awakening and before bedtime to calculate the AUC; AM- PM

Errazuriz 2020	age: *M* = 40.2, *SD* = *11.7;* gender: 98.1% female	Stress risk (healthcare workers)	Mindfulness based stress reduction	21	Waitlist	25	16 h (8-week intervention with a weekly 2-hour session)	Cortisol sampled upon awakening and before bedtime to calculate the AUC; AM- PM
Fan 2014	age: *M* = 20.87, *SD* = *0.26 ;* gender: 52.94% female	No risk	Integrative meditation	17	Active (relaxation)	17	8.3 h (five 20 - 30-minute sessions for four weeks)	Saliva sample taken before a stress test pre- and post-intervention; PM
Feicht 2013*	age: *M* = 37.19, *SD* = *9.07;* gender: 69.3% female	No risk	Happiness training	18	Waitlist	19	1.5 h (one session at home lasting 10 - 15 minutes for seven weeks)	Two saliva samples taken 30 minutes after awakening and at 8pm to calculate circadian rhythm amplitude; AM - PM
Fendel 2021	age: *M* = 31.02, *SD* = *3.43;* gender: 65.31% female	No risk	Mindfulness based stress reduction	57	Active (mindfulness description booklet)	49	30.75 h (8 guided sessions (once per week), one 6-hour retreat and three, monthly boosters during the maintenance phase)	One hair sample taken at each measurement point (pre-intervention, post-intervention and follow-up)

Flook et al., 2013	age: *M* = 43.06, SD = *9.87;* gender: 88.89% female	No risk	Modified mindfulness-based stress reduction adapted for teachers	10	Waitlist	8	26 h (8 sessions through 8 weeks and a day long immersion for 6 hours)	One saliva sample 30 minutes after waking on three consecutive days; AM
Fotiou et al., 2015	age: median = 34.5 , *IQR = 32.5, 40.5;* gender: 52.5% female	Stress risk (parents with baby's in a tertiary maternity hospital)	Relaxation training course (involving techniques such as progressive muscle relaxation, deep breathing and guided imagery)	31	Inactive (information sessions for parents)	28	7.5 h (5 interactive training courses lasting approximately 90 minutes each)	We calculated the diurnal secretion of cortisol from taking the average from - one saliva sample upon awakening, +30 minutes after waking and before bed on one day; AM - PM
Gaab et al., 2006	age: *M (intervention)* = 22.5 , *SD(intervention)* = *1.7, M (control)* = 24.3, *SD (control)* = 4.5, *range* = not reported*;* gender: 39.29% female	No risk	Cognitive Behavioural Stress Management	13	Waitlist	15	10.5 h (4 weekly CBSM sessions and a booster session)	Four saliva samples taken at 8am, 11am, 3pm and 8pm, AUCg was calculated from these measures; AM - PM

Gallego Gome 2020	age: *M* = 24.3, *SD = 6.2;* gender: 75% female	No risk	Music therapy and progressive muscle relaxation	56	Inactive (no intervention)	56	1 h	One blood sample drawn; AM
Galvin 2006	age: *M* = 71.3, *SD = not reported, range = 66 - 80;* gender: 53.33% female	No risk	Relaxation response training	7	Inactive (no intervention)	7	13.2 h (90 minute led relaxation response training followed by daily 20-minute training following a relaxation response audiotape)	One saliva sample taken, before 9:30am, pre- and post-intervention; AM
Gardi 2022	age: M = 46.58, SD = 10.76; gender: 55.56% female	No risk	Mindfulness meditation retreat	46	Active (vacation retreat)	44	30 h (3-day retreat with 10h of meditation per day)	One saliva sample 30 minutes after waking pre- and post-intervention between 5 and 6am; AM
Goldberg 2014	age: *M* = 42.2, *SD* = 11.4*;* gender: 55.6% female	No risk	Mindfulness training for smokers	10	Active (freedom from smoking)	8	42.38 h (7 sessions lasting 2.5 hour each. Additionally a 6.5-hour day retreat and 15 - 30 minutes of daily meditation)	One hair sample taken at post quit study visit
Gothe 2016	age: *M* = 62, *SD* = 5.6*;* gender: 77.97% female	No risk	Yoga	53	Inactive (stretching)	47	24 h (3 sessions per week for 8 weeks)	One saliva sample taken prior to

								cognitive assessments; PM
Graham 2022	age: M = 33; gender: 93.4% female	No risk	Mindfulness based stress management	20	Active (relaxing music)	21	3.5 h (6-week online course with at least 5 minutes of daily meditation practice. The sessions ranged from 5 - 20 minutes in length)	Six saliva samples at home over one day at pre- and post-intervention to calculate the AUCg change between visits; AM - PM
Granath 2006	age: not reported*;* gender: 72.97% female	No risk	Cognitive Behavioural Therapy	16	Yoga	15	10 sessions over 4 months (one session per week for 4 week, 3 sessions every other week then 3 sessions once every 3 weeks)	One saliva sample; no timing reported
Hilcove 2021	age: *M* = 42.41, *SD* = 12.12, *range* = 24 - 69*;* gender: 94.87% female	No risk	Mindfulness based yoga	29	Inactive (no intervention)	30	20 h (Weekly group sessions and home yoga practice for 6 weeks)	Three saliva samples during the day (awakening, 45 minutes after awakening and 12-14 hours after awakening) to calculate AUC; AM - PM

Ho 2020	age: *M* = 38.75, *SD = 5.41;* gender: 96.08% female	No risk	Mindfulness based intervention	6	Waitlist	25	9 h (6-week intervention, each session lasting 1.5 hours)	Four saliva samples taken after waking up (around 7:30am), before lunch (around 12pm), late afternoon (around 5:30pm) and before sleep around 9:30pm); AM - PM
Hwang 2013	age: *M* = 41.02, *SD = 11.66*, *range = 21 - 59;* gender: 78% female	Stress risk (regularly reported stress)	Brief Qigong-based stress reduction (BQSRP)	17	Waitlist	17	18 h (one 2-hour session in week 1, then one 1-hour session per week for 3 weeks. Participants also practiced meditation twice daily for 15 minutes)	One saliva sample 1 hour after awakening; AM
Iglesias 2012	age: *M* = 23*;* gender: 75% female	No risk	Relaxation	12	Active (cognitive behavioural techniques)	13	17.5 h (10 weeks of 90 - 120 minute sessions)	One saliva sample immediately after awakening; AM
Iglesias 2012	age: *M* = 23*;* gender: 75% female	No risk	Relaxation	12	Active (relaxation with cognitive behavioural techniques)	14	17.5 h (10 weeks of 90 - 120 minute sessions)	One saliva sample immediately after awakening; AM

Iglesias 2012	age: *M* = 23*;* gender: 75% female	No risk	Relaxation	12	Waitlist	13	17.5 h (10 weeks of 90 - 120 minute sessions)	One saliva sample immediately after awakening; AM
Jensen 2012	age: *range = 20 - 36 ;* gender: 66% female	No risk	Mindfulness based stress reduction	14	Active (non-mindfulness stress reduction)	11	83 h (8 week intervention with each session lasting 2.5 hours. Also has formal home assignments (45 minutes) and informal (15 minutes)	Five saliva samples taken upon awakening and then 4 further samples every 15 minutes for the subsequent hour, to calculate the AUCg; AM
Jensen 2012	age: *range = 20 - 36 ;* gender: 66% female	No risk	Mindfulness based stress reduction	14	Inactive (no intervention)	14	83 h (8 week intervention with each session lasting 2.5 hours. Also has formal home assignments (45 minutes) and informal (15 minutes)	Five saliva samples taken upon awakening and then 4 further samples every 15 minutes for the subsequent hour, to calculate the AUCg; AM
Jensen 2015	age: *M* = 42, *SD = 9, range = 18 - 59;* gender: 65% female	Stress risk (complained to their GP about	Relaxation	32	Inactive (treatment as usual)	15	25.5 h (9 week intervention with weekly 2.5 hour group sessions and two 1.5	Five saliva samples taken upon awakening and then 4 further samples every 15 minutes for

		prolonged stress)					hour individual sessions)	the subsequent hour, to calculate the AUCg; AM
Johansson 2006	age: *M* = 35 , *range = 25 - 47;* gender: 75% female	No risk	Mental training	6	Inactive (no intervention)	6	Not reported (group meetings every other week for 6 months)	One blood sample drawn; AM
Kim 2016	age: *M* = 23.26, *SD = 2.3;* gender: 0% female	Stress risk (distressed college students)	Stress management	43	Waitlist	41	16 h (eight 2 hour sessions over four weeks, two sessions per week)	One blood sample drawn; AM
Kotozaki 2014	age: *M* = 42.3, *SD = 7.9, range = 23 - 53;* gender: 0% female	No risk	Biofeedback	15	Inactive (no intervention)	15	2.3 h (4 week intervention with daily 5 minute practicing)	One saliva sample taken at 4pm pre- and post-intervention; PM
Lai 2011	age: *M* = 23.4, *SD = 2.46;* gender: 100% female	No risk	Music therapy	54	Inactive (resting)	54	0.5 h (one 30 minute session)	One blood sample drawn pre- and post-intervention; PM
Laudenslager 2015	age: *M* = 53.5, *range = 21 - 80;* gender: 75.7% female	Stress risk (caregivers)	Psychoeducation, Paced Respiration and Relaxation	48	Inactive (treatment as usual)	53	19 h (eight 1-1 sessions lasting 60 - 75 minutes. At home 15 minute practicing every day for 4 -5 times per week)	Three saliva samples on three consecutive days pre- and post-intervention (taken at awakening, before lunch and +10 hours); AM - PM

Limm 2011	age: *M* = 40.9, *SD* = *7.72;* gender: 99% female	No risk	Stress management	75		79	18h (eight sessions lasting 90 minutes over 2 consecutive days followed by two booster sessions within 3 - 6 months lasting 180 minutes per session)	Four saliva samples taken at 8am, 11am, 3pm and 8pm, AUCg was calculated from these measures; AM - PM
MacDonald 2018	age: *M* = 25.9, *SD* = *5.45;* gender: 31.25% female	No risk	Mindfulness training for smokers	8	Waitlist	8	26.5 h (5 sessions per week lasting 15 minutes for the first 2 weeks. 5 sessions per week lasting 50 minutes for the final 6 weeks)	One saliva sample taken in the morning; AM
MacLean 1997	age: *M* = 25, range = 18 - 32 ; gender: 0% female	No risk	Meditation	16	Active (stress education)	13	65.3 h (daily practice for 15 - 20 minutes for 4 months)	Six blood samples taken across a single stress session to calculate AUC; AM
McGrady 1992	age: *M* = 25*;* gender: 43.75% female	No risk	Biofeedback	14	Inactive (no intervention)	17	4 h (four 30 minute group relaxation and four 30 minute individual biofeedback sessions over 4 weeks)	One blood sample drawn; PM

McKinney 1997	age: *M* = 37.4, *SD = 6.1, range* = 23 - 45*;* gender: 85.71% female	No risk	Guided imagery and music	11	Waitlist	9	10.5 h (six individual sessions lasting 1.5 - 2 hours every 2 weeks)	One blood sample drawn; AM
Mohan 2011	age: *M* =27.3 , *SD = 1.8, range = 23 - 30;* gender: 0% female	No risk	Meditation	8	Inactive (no intervention)	8	0.3 h (one 20 minute session of meditation)	One blood sample drawn; PM
Nyklicek 2013	age: *M* = 46.1, *SD = 10.6;* gender: 70.59% female	Stress risk (stress related complaints)	Mindfulness based stress reduction	37	Waitlist	36	26 h (eight weekly 2.5 hour group sessions plus a weekly at home session lasting 45 minutes)	Depends which measurements we take; PM
Oken 2010	age: *M = 64.8, SD = 9.98, range = 45 - 85;* gender: 80.65% female	Stress risk (caregivers)	Mindfulness meditation	10	Active (education class)	11	9 h (six weekly 90 minute sessions per week across 7 weeks)	Three saliva samples taken within 5 minutes and 30 minutes of awakening, then before bed (10-11pm). We calculated the diurnal mean; AM - PM

Oken 2010	age: *M = 64.8, SD = 9.98, range = 45 - 85;* gender: 80.65% female	Stress risk (caregivers)	Mindfulness meditation	10	Inactive (respite only)	10	9 h (six weekly 90 minute sessions per week across 7 weeks)	Three saliva samples taken within 5 minutes and 30 minutes of awakening, then before bed (10-11pm). We calculated the diurnal mean; AM - PM
Oken 2017	age: M = 59.8, SD = 6.85, *range = 50 - 85;* gender: 79.85% female	Stress risk (mildly stressed)	Meditation	60	Waitlist	68	33.75 h (six weekly 60 - 90 minute sessions, suggested daily home practice for 30 - 45 minutes)	Two saliva samples taken upon awakening and before bed to calculate the diurnal slope; AM - PM
Pawlow 2005	age: *M* = 23.96, *SD = 7.54, range = 19 - 57;* gender: 52.73% female	No risk	Progressive muscle relaxation	41	Inactive (no intervention)	14	0.3 h (one 20 - 25 minute session of muscle relaxation)	One saliva sample taken pre- and post-intervention; AM
Perna 1998	age: *M* = 19.2, *SD = 1.3;* gender: 58.82% female	No risk	Cognitive Behavioural Stress Management	18	Active (stress education)	16	5.25 h (two 45 minute sessions each week for the first 3 weeks with one weekly meeting in week 4)	One blood sample drawn; AM

Puhlmann 2022	age: *M* = 40.74, *SD* = 9.24, range = 20 - 55; gender: 59.3% female	No risk	Contemplative Mental Training	29	Active (affect only training cohort)	36	238.5 h (39 week intervention consisting of three 3 month modules. Each module began with a 3 day retreat (est. 7 hour per day). Participants then had weekly 2-hour groups sessions and engaged in 30 minutes daily practice on 5 days per week)	One hair sample taken at each measurement point (pre-intervention and post-intervention)
Puhlmann 2022	age: *M* = 40.74, *SD* = 9.24, range = 20 - 55; gender: 59.3% female	No risk	Contemplative Mental Training	29	Inactive (no intervention)	42	238.5 h (39 week intervention consisting of three 3 month modules. Each module began with a 3 day retreat (est. 7 hour per day). Participants then had weekly 2-hour groups sessions and engaged in 30 minutes daily practice on 5 days per week)	One hair sample taken at each measurement point (pre-intervention and post-intervention)

Repo 2022	age: not reported ; gender: 72.5% female	No risk	Mindfulness	35	Active (online mindfulness and ACT)	15	29.7 h (Eight week intervention with eight sessions lasting 75-90 minutes. At home daily mindfulness for 10-30 minutes)	One hair sample taken at each measurement point (pre-intervention, post-intervention and follow-up)
Repo 2022	age: not reported ; gender: 72.5% female	No risk	Mindfulness	35	Inactive (support as usual)	30	29.7 h (Eight week intervention with eight sessions lasting 75-90 minutes. At home daily mindfulness for 10-30 minutes)	One hair sample taken at each measurement point (pre-intervention, post-intervention and follow-up)
Rodriguez-Jiminez 2022	age: M = 43.3, SD = 7.5; gender: 64.5% female	No risk	Yoga	11	Active (body movement awareness)	10	12 h (one 90 minute session per week for 8 weeks)	Saliva sample taken before a stress test pre- and post-intervention; no data
Rodriguez-Jiminez 2022	age: M = 43.3, SD = 7.5; gender: 64.5% female	no risk	Yoga	11	Inactive (no intervention)	10	12 h (one 90 minute session per week for 8 weeks)	Saliva sample taken before a stress test pre- and post-intervention; no data
Rosenkranz 2013	age: *M* = 45.89, *SD = 10.92, range = 19 -59;* gender: 79.59% female	No risk	Mindfulness based stress reduction	24	Active (health enhancement program)	25	76 h (8 weekly 2.5 h sessions and one full-day session lasting 7 hours.	Five saliva samples taken at awakening, 30 minutes post-awakening, before

							Daily at-home practice for 45 - 60 minutes)	lunch, 3pm, and before bed; AM - PM
Schakel 2019	age: *M* = 22.1, *SD = 2.3, range = 18 - 29;* gender: 81.67% female	No risk	Relaxation with verbal suggestions	30	Active (relaxation)	29	0.3 h (one 20 - 25 minute session of relaxation)	Cortisol sampled pre- and post-intervention and TSST; PM
Schakel 2019	No risk	No risk	Relaxation with verbal suggestions	30	Active (verbal suggestions)	29	0.3 h (one 20 - 25 minute session of relaxation)	Cortisol sampled pre- and post-intervention and TSST; PM
Schakel 2019	age: *M* = 22.1, *SD = 2.3, range = 18 - 29;* gender: 85% female	No risk	Relaxation with verbal suggestions	30	Inactive (no intervention)	28	0.3 h (one 20 - 25 minute session of relaxation)	Cortisol sampled pre- and post-intervention and TSST; PM
Sousa 2021	age: *M* = 24.15, *SD = 3.56, range = 18 - 30;* gender: 50% female	No risk	Brief mindfulness training	20	Active (listening to health education audio and colouring)	20	1.5 h (three 30 minute sessions over three days)	One blood sample drawn; AM
Thompson 2011	age: *M* = 22.1, *SD = 3.4;* gender: % female	No risk	Hypnosis (animated imagery)	12	Active (verbal imagery)	12	3.3 h (10 self-hypnosis sessions each taking around 20 minutes)	Two saliva samples taken pre- and post-intervention; AM - PM

Thompson 2011	age: *M* = 22.1, *SD = 3.4;* gender: % female	No risk	Hypnosis (animated imagery)	12	Active (relaxation)	11	3.3 h (10 self-hypnosis sessions each taking around 20 minutes)	Two saliva samples taken pre- and post-intervention; AM - PM
Tsiouli 2014	age: *M* = 43.28, *range = 31 - 65;* gender: 79.55% female	Stress risk (parents of children with type 1 diabetes)	Progressive muscle relaxation	19	Active (education)	25	69 h (One session lasting 37 minutes then twice daily for 8 weeks)	Five saliva samples taken at 8am, 12pm, 3pm, 6pm and 9pm; AM - PM
Turan 2015	age: *M* = 40.61, *SD = 10.28, range = 25.05 - 60.92;* gender: 100% female	No risk	Meditation	35	Inactive (no intervention)	35	42 h (4 all day sessions and 4 evening sessions over 8 weeks)	Two saliva samples taken pre-stress task at pre- and post-intervention; no timing reported
Turner 2020	age: *not reported ;* gender: 70.37% female	Stress risk (exam stress)	Mindfulness	22	Inactive (treatment as usual)	25	11 h (8 weekly sessions lasting 75 - 90 minutes)	One blood sample drawn; no timing reported
Vandana 2011	age: *range = 18 - 21;* gender: 81.45% female	No risk	Integrated Amrita Meditation	29	Active (progressive muscle relaxation)	23	59.7 h (regular practice over an 8 month period, asked to do a minimum of four sessions per week. Each session lasting 28 minutes)	One blood sample drawn; AM

Vandana 2011	age: range = 18 - 21; gender: 81.45% female	No risk	Integrated Amrita Meditation	29	Inactive (no intervention)	23	59.7 h (regular practice over an 8 month period, asked to do a minimum of four sessions per week. Each session lasting 28 minutes)	One blood sample drawn; AM
Waelde 2017	age: *M* = 59.6, *SD = 11.9, range = 37 - 83;* gender: 100% female	Stress risk (caregivers)	Meditation	15	Active (psychoeducation and telephone support)	15	18 h (nine 1.5 hour sessions over 8 weeks. 3 hour retreat held in week 7 and a booster session in week 12)	Three saliva samples taken at awakening, 5pm and 9pm on two consecutive days pre- and post-intervention; AM - PM

*** the saliva sampling may not represent all samples taken in the study; the samples are chosen here that fulfil the criteria of the meta-analysis**

Table 22. Summary of the sub-group analyses representing the mixed effects analysis model for each moderator variable

				95% CI		
Moderator	**g**	**k**	**SE**	**LL**	**UL**	***p***
Cortisol outcome						
Blood	0.331	13	0.136	0.065	0.598	0.015
Saliva	0.284	42	0.074	0.139	0.429	0.000
Intervention group						
Mindfulness	0.345	30	0.085	0.178	0.511	0.000
Relaxation	0.347	14	0.125	0.102	0.591	0.005
Mind body	0.129	6	0.187	-0.238	0.496	0.492
Talking therapy	0.107	8	0.162	-0.211	0.424	0.510
Collapsed control group						
Active	0.477	16	0.109	0.264	0.690	0.000
Passive	0.129	30	0.076	-0.021	0.278	0.093
Control group						
Active	0.477	16	0.110	0.261	0.693	0.000
Inactive	0.131	20	0.095	-0.055	0.318	0.168

Waitlist	0.124	10	0.134	-0.138	0.386	0.354
Awakening/diurnal						
Awakening	0.644	9	0.153	0.343	0.945	0.000
Diurnal	0.225	49	0.063	0.103	0.348	0.000
Length of intervention						
Short	0.306	15	0.084	0.142	0.471	0.000
Medium	0.150	14	0.147	-0.139	0.439	0.308
Long	0.348	28	0.093	0.166	0.530	0.000
Study quality						
Low	0195	11	0.144	-0.088	0.477	0.178
Moderate	0.346	34	0.080	0.189	0.503	0.000
High	0.212	13	0.130	-0.043	0.468	0.103
Risk of Bias						
High	0.207	6	0.186	-0.158	0.572	0.267
Some Concerns	0.303	31	0.087	0.132	0.474	0.001
Low	0.295	21	0.100	0.100	0.490	0.003
Stress risk of sample						

Low risk	0.351	43	0.075	0.204	0.498	0.000
High risk	0.135	15	0.098	-0.057	0.327	0.169