Development of Depression in Behaviourally Inhibited Children:

The Role of Risk-Taking and Reward-Seeking in Early Adolescence

Talia Morris

Bachelor of Psychology (Honours)

Centre for Emotional Health, Department of Psychology

Macquarie University

Sydney, Australia

A dissertation submitted in partial fulfilment of the requirements of the degree

of Doctor of Philosophy

23rd of March, 2017

Table of Contents

List of Tables

List of Figures

Thesis Summary

Although depression rates increase rapidly in adolescence, the mechanisms behind this development are not fully understood. This thesis examined the role of several risk factors for adolescent depression with a focus on risk-taking, reward-seeking, early childhood temperament and social interactions. Two samples were used across four empirical papers. First, a sample of behaviourally inhibited (BI) and uninhibited (BUI) preschool children (n = 70). Second, a sample of adolescents initially recruited and classified at BI and BUI at age 4. Paper 1 examined how the temperamental constructs of BI and effortful control related to risk-taking. Effortful control, but not BI, at age 4 was related to reward focused risk-taking on the Balloon Analogue Risk Task (BART). Paper 2 examined the association between BI in childhood, risk-taking and depressive symptoms in adolescence, including how risk-taking as measured by the BART may present a potential risk pathway. An interaction between BI and risk-taking was associated with depressive symptoms in early adolescence. Due to the salience of peers in adolescence and the role that peer interactions might play in risk for adolescent depression, Paper 3 further explored risk-taking by developing a novel measure of social risk-taking and examining the association between scores on this measure and depressive symptoms over a 6-month period. A bidirectional relationship between social risk-taking and depressive symptoms was found. In addition, social acceptance partially mediated the relationship between depressive symptoms and social risk-taking. Finally, Paper 4 focused on reward-seeking using an adapted effort-reward task. No overall difference was found between early adolescents with or without symptoms of depression in their motivation to view a rewarding cartoon. Nevertheless, the task shows promise for future work in this area. Taken together, the results of this thesis present unique information about adolescent depression and the risk factors of temperament, risk-taking, reward-seeking and social risk-taking.

Statement of Candidature

I certify that the work of this thesis entitled "Development of Depression in Behaviourally Inhibited Children: The Role of Risk-Taking and Reward-Seeking in Early Adolescence" has not been previously submitted for a degree to any other university or institution.

I certify that the thesis is an original piece of research that has been written by me, with support from Drs. Jennifer Hudson and Helen Dodd. The individual contributions of co-authors and contributors have been appropriately acknowledged. In addition, I certify that all information sources and literature used when preparing this thesis have been referenced appropriately.

Macquarie University Ethics Committee approval was obtained for all aspects of the research studies presented in this thesis, references 5201000902, 5201100488, HE30MAY2008-R05911 (see Appendix A).

Talia Morris _____

Date _____

List of Thesis Contributors

Mrs. Talia Morris (TM), Dr. Jennifer Hudson (JH), and Dr. Helen Dodd (HD).

Summary of Contributions to Empirical Papers

Contribution	Chapter 2	Chapter 3	Chapter 4	Chapter 5
Study Conception	TM, JH, HD	TM, JH, HD	TM, JH, HD	TM, JH, HD
Data Collection	TM, JH	TM, JH, HD	TM, JH, HD	TM, JH
Data Analysis	TM, JH, HD	TM, JH, HD	TM, JH, HD	TM, JH, HD
Interpretation of Results	TM, JH, HD	TM, JH, HD	TM, JH, HD	TM, JH, HD
Paper Preparation	TM	TM	TM	TM
Paper Revisions	TM, JH, HD	TM, JH, HD	TM, JH, HD	TM, JH, HD

Acknowledgements

First and foremost, I want to acknowledge my wonderful supervisors Drs. Jennie Hudson and Helen Dodd. I am sincerely grateful for the support and guidance you have both provided me with over my candidature. Initially a boss, then my PhD supervisor and finally a mentor, Jennie you have been a fantastic support to me right from the beginning. Helen, you have always made time to provide me with guidance, whether it be with statistics or reading my work. You have both have provided me with so much support, both professionally and personally.

To my amazing level 7 friends and colleagues: particularly Bec, Yuly, Miki, Lauren, Jacqui, Soph, Carly, Sam, and many others. I would never have made it through this degree without your friendship, coffees, lunches, and pep talks. Thank you also to my family on both sides. The endless babysitting and support was very much appreciated.

I want to acknowledge the support and assistance from those I worked with throughout my PhD including the other RAs and all of our hard-working interns. I would also like to offer my appreciation to those who have employed me during my degree as I juggled work, study and a family. You have all been so enthusiastic and supportive of my efforts. Most importantly, thank you to all of the wonderful preschoolers and adolescents and their families who took part in the studies within this thesis.

To my husband Christopher, this journey has seen both challenges and rewards, with you my constant, always by my side. Thank you for your unwavering support and love, without you this thesis would not have happened.

To my beautiful girls, Charlotte and Elliana, thank you for being my little rays of sunshine. This thesis is for you.

Chapter One

General Introduction

Introduction

The overarching aim of this research is to examine risk factors that place an individual at risk of developing depression during adolescence. Specifically, this thesis focuses on risk factors most salient during this developmental period, including puberty, social interactions and changes in reward-seeking and risk-taking behaviour. In addition, the childhood risk factors of temperament and anxiety are included to examine possible pathways to depression in adolescence.

First, this introductory chapter will provide a review of the existing literature examining the role of the different risk factors in the development of depression in early adolescence. In particular, this chapter will identify a number of key questions that will be addressed in the four empirical papers within the thesis. The first study of the thesis examines temperament and risk-taking in pre-school aged children, focusing on whether these risk factors are associated with one another in early childhood. The second study investigates temperament in early childhood and its links to depression in early adolescence, including the potential moderating factors of puberty and risk-taking. In the third study, the particularly salient factors of social interactions and risk-taking are examined together in relation to the development of depressive symptoms in adolescence. The final study of the thesis examines a reward-seeking task in adolescents for the first time, observing its relationship to depressive symptoms. Finally, the concluding chapter will summarise the four studies' findings, examining the implications for our understanding of the development of depression in adolescence and directions for future research.

Definition of Depression

Depression produces the second largest burden of disease in the world today and is the leading cause of disability (World Health Org. Region. Off. South-East Asia 2001). The National Comorbidity Survey, conducted in the United States, found that approximately 1 in 5 women and 1 in 8 men will experience a major depressive episode during their lifetime (Kessler et al., 1994). The Diagnostic and Statistical Manual of Mental Disorders (DSM-

IV-TR, American Psychiatric Association, 2000)[1] defines Major Depressive Disorder (MDD) as depressed mood or loss of interest or pleasure in activities for more than two weeks. For a diagnosis of MDD, a number of other criteria must be met. These include impaired functioning and additional symptoms such as changes in sleep, difficulty concentrating, or feelings of worthlessness.

Adolescence and the Development of Depression

Over the past 25 years, recognition of mood disorders in children and adolescents has increased (Klein, Lewinsohn, Seeley, & Rohde, 2001). Though depression is rare in pre-school aged (Kashani & Carlson, 1987) and preadolescent children (Cohen et. al., 1993), the prevalence of depression increases during adolescence, with the risk of first onset peaking in the early teens and the mid-20s (Kessler, Avenevoli, & Merikangas, 2001). There is a large body of evidence that depression during childhood and adolescence increases the likelihood of depression in adulthood (Rohde, Lewinsohn, Klein, Seeley, & Gau, 2013). Even subclinical depressive symptoms in adolescence strongly predict an episode of MDD in adulthood (Pine, Cohen, Cohen, & Brook, 1999). In fact, most adult cases of depression emerge in adolescence in the form of depression or anxiety (Pine, Cohen, Gurley, Brook, & Ma, 1998).

In addition to increased risk for depression in adulthood, MDD during childhood and adolescence can have debilitating consequences for the individual. For example, adolescent depression has been associated with impaired school performance as well as difficulties with concentration, reading, and writing (Fröjd et al., 2008). Furthermore, adolescent depression is a strong predictor of suicide attempts (Lewinsohn, Rohde, & Seeley, 1994). Adolescent depression can also lead to other mental health disorders with research estimating that 20-30% of adolescents with depression will develop a substance abuse disorder (Birmaher et. al., 1996). In clinical populations, earlier onset of depression is

[1] At the time of the research conducted in this thesis, the DSM-IV was the current version and hence will be used throughout the thesis.

associated with greater social and occupational impairment, as well as poorer quality of life (Zisook et al., 2007). Knowing the detrimental impact of adolescent onset depression, it is essential that developmental pathways to depression be examined so that early risk factors can be identified and targeted within prevention or early intervention programmes (Lau & Eley, 2010).

Although a large body of research has examined risk factors for depression in adults, only more recent work has focused on risk factors for the development of depression in adolescence (e.g Platt, Kadosh, & Lau, 2013). Adolescence is a time of significant change developmentally, with the onset of puberty and the increased focus of independence from parents. During adolescence there is also an increased emphasis on peer relationships and social interactions (Spear, 2000) as well as reward-seeking and risk-taking becoming heightened (Steinberg, 2008). In addition, a number of important neural changes occur during adolescence in areas such as executive functioning and social cognition (Paus, 2005). Silk and colleagues proposed a developmental model of depression in adolescence, highlighting the altered processing of social threats and reward as two key vulnerabilities that are likely to be exacerbated by pubertal processes in adolescence (Silk, Davis, McMakin, Dahl, & Forbes, 2012). In particular, important changes during adolescence including the increased importance of peers and social interactions outside the family, and changes in reward systems intensify the existing issues in processing of social evaluative threat and reward in at risk adolescents leading to adolescent depression (Silk et al., 2012).

It is important to focus on these particular areas to gain insight into why depression increases so significantly in adolescence. This thesis draws on existing evidence regarding risk factors for depression and considers risk within the developmental context of adolescence by focusing on early temperament, as well social issues, puberty and risk-taking/reward-seeking during early adolescence. First, this thesis reviews a number of theoretical models proposed to explain the development of depression, then empirical

research for individual risk factors including temperament, risk-taking, social interactions, social risk-taking, and reward-seeking.

Multivariate Models of Risk For Depression

Previous research has shown that the development of depression is complex, with many contributing factors. A single risk factor is not likely to completely explain the development of depression. Instead, it is the accrual of factors, or interaction among multiple factors that results in depression (Garber, 2006). Yet, there are a multitude of theories concerning vulnerability for depression that focus on a single etiological risk factor. For example, cognitive vulnerability theories such as Beck's cognitive theory (Beck, 1967, 1983) suggest that a negative cognitive style predicts depression (e.g. Carter & Garber, 2011). However, despite the strength of the research support for this model, single-focus vulnerability models have been unable to completely explain when and why a person may become depressed (Hankin, 2012).

More recent work has begun to examine multivariate models of depression. By including a variety of risk factors, such as social, behavioural and genetic factors, and observing how they predict depression over time, these models can provide insight into the complex processes involved. A large-scale study of female twin pairs examined risk factors for depression across the lifespan such as anxiety in early adolescence and social support in late adolescence (Kendler, Gardner, & Prescott, 2002). Using structural equation modelling, the authors found that risk factors for the development of major depression in women fall into three main interlinking pathways: internalising symptoms, externalising symptoms and psychosocial adversity. Risk factors could be identified as being clustered within these three pathways, but they also interacted with each other to predict episodes of depression. The internalising pathway began with a genetic risk for major depression predicting neuroticism, low self-esteem and early onset anxiety in early adolescence. In particular, this pathway was anchored by neuroticism and early onset-anxiety disorders.

These internalising factors then go on to predict an episode of major depression in the last year for the adult participants. The main risk factors anchoring the externalising pathway were conduct disorder in early adolescence and substance misuse in later adolescence. Finally, the psychosocial adversity pathway to depression identified in this study was more wide-ranging. Early childhood factors such as sexual abuse and parental loss, predicted and interacted with later adolescence factors including low social support, as well as difficulties in adulthood including marital problems and stressful life events (Kendler et al., 2002). Although this model is one of the best fitting predictive models of depression in the literature, it still only accounted for approximately 50% of the variance in depression symptoms (Kendler et al., 2002). Thus, more work is needed to continue to explore the development of depression.

Anxiety as a Risk Factor for Depression Anxiety

As mentioned in the predictive model above, anxiety disorders have been implicated in the development of depression. Studies have shown that episodes of depression in adolescence are often preceded by at least one anxiety disorder (Kessler et al., 2001). A longitudinal study measuring depression and anxiety symptoms in children over 3 years demonstrated that high levels of anxiety at stage 1 predicted high levels of depressive symptoms at each subsequent timepoint, even when controlling for previous depressive symptoms levels (Cole, Peeke, Martin, Truglio, & Seroczynski, 1998). However, it has also been argued that depression can precede the development of anxiety almost as often. In a longitudinal study following participants from ages 11 to 32 years, for those who developed co-morbid anxiety and depression in adulthood, depression onset occurred first for one third of participants, anxiety onset first for one third, and depression and anxiety began concurrently in one third (Moffitt et al., 2007). Regardless of direction, high comorbidity has been shown, with anxiety disorders the most common comorbid mental health disorder in adolescents with depression (Rhode et al., 2013). Anxiety and depression co-occur at a rate higher than

chance (Angold, Costello, & Erkanli, 1999). In fact, one of the most consistent genetic findings, when examining the highly comorbid symptoms of anxiety and depression, is that they are influenced mostly by the same genes (Eley & Stevenson, 1999).

It has been suggested that the high level of comorbidity between depression and anxiety may be due to three main factors: an overlap in symptoms, common causation factors such as familial risk or information-processing biases, and the pathway of anxiety leading to an increased risk for the later development of depression (Garber & Weersing, 2010). Further, the Tripartite Model of Anxiety and Depression (Clark & Watson, 1991) posits that there is a tripartite structure with general distress or negative affect common to both depression and anxiety, physiological hyperarousal specific to anxiety and anhedonia specific to depression. Although there is a large body of evidence for the Tripartite model (e.g. Clark, Watson, & Mineka, 1994; Steer, Clark, Beck, & Ranieri, 1995), other findings report deviations. For example, low positive affect and reduced motivation, or anhedonia, has been shown to also appear in social phobia as well as depression (Hughes et al., 2006).

A recent review proposed a model with three pathways towards anxiety-depression comorbidity, in particular that specific anxiety disorders may pose a different risk for depression (Cummings, Caporino, & Kendall, 2014). Specifically, Pathway 1 includes adolescents with either social phobia or separation anxiety and later depression. Pathway 2 includes adolescents with comorbid generalised anxiety disorder and depression, and Pathway 3 describes adolescents with depression experiencing subsequent social phobia. Although evidence exists for all three pathways, the authors emphasise the need for further empirical testing to examine the interplay of depression and anxiety symptoms in adolescents (Cummings et al., 2014), in particular the role risk factors may play in the development of these pathways. Thus, when looking at depression it is important to keep in mind the strong link with anxiety disorders.

Temperament as a Risk Factor for Depression

One important underlying risk factor for both depression and anxiety is childhood temperament. In fact, temperament has been suggested to be one of the main pathways to comorbid anxiety and depression symptoms (Karevold, Ystrom, Coplan, Sanson, & Mathiesen, 2012). Temperament is a biologically based factor which results in individual differences in behaviour and is present early in life (Bates, 1987). Several temperament factors are examined in the literature including Behavioural Inhibition (BI) and Effortful Control (EC).

Behavioural Inhibition (BI). BI was described by Kagan and colleagues as a temperamental trait of withdrawal from the unfamiliar (Garcia Coll, Kagan, & Reznick, 1984). Children classified as high BI are withdrawn in novel situations and hesitant with unfamiliar people (Kagan, Reznick, Snidman, Gibbons, & Johnson, 1988). BI can be identified as early as 4 months of age (Fox, Henderson, Rubin, Calkins, & Schmidt, 2001), with approximately 15% of typically developing children exhibiting this temperament style (see Fox, Henderson, Marshall, Nichols, & Ghera, 2005 for a review). It is relatively stable across childhood (Fox et al., 2005) with the children at the extremes, highly inhibited and highly uninhibited, showing the most stability across time (Kerr, Lambert, Stattin, & Klackenberg-Larsson, 1994). Inhibited toddlers tended to remain quiet and cautious when followed up at 7 years of age, while uninhibited toddlers remained talkative and interacted without difficulty with adults at 7 years of age (Kagan et al., 1988). BI children are more likely to demonstrate socially withdrawn behaviour with their peers at school, remaining solitary and watchful (Rubin, Burgess, & Hastings, 2002). In addition, differences in neural activation have been shown for those with an inhibited temperament. A recent meta-analysis of neural imaging studies found that increased activation in brain areas associated with novelty and threat processing, as well as reward processing, were shown for inhibited participants compared to uninhibited participants (Clauss, Avery, & Blackford, 2015).

Being classified as BI in childhood has been associated with an increased risk for anxiety disorders. It has been demonstrated longitudinally that BI at age 4 predicted anxiety at age 9 over and above any initial anxiety (Hudson & Dodd, 2012). There is also some suggestion of specificity, with high levels of BI in early childhood shown to specifically predict increased risk for lifetime social anxiety in adolescence (Chronis-Tuscano et al., 2009; Clauss & Blackford, 2012). It has been reported that approximately 34-44% of BI pre-schoolers will go on to develop social anxiety by early adolescence (Hirshfeld-Becker, Micco, Simoes, & Henin, 2008).

Studies have also found a link between BI and later depression. A number of cross-sectional studies have shown that retrospectively reported BI is related to depression. For instance, Gladstone and Parker (2006) demonstrated that adults with a lifetime episode of depression reported significantly higher BI in childhood. Research has also shown that BI in adolescence is related to an increased risk for depression. For example, high BI, based upon both child and parent report, is accompanied by high levels of depressive symptoms as well as high levels of anxiety symptoms (Muris, Meesters, & Spinder, 2003). However, only a small number of studies have shown a link between BI observed in childhood and depression in adolescence or adulthood. Children classified as inhibited at age 3 were more likely at age 21 to meet criteria for depression, but not for anxiety disorders (Caspi, Moffitt, Newman, & Silva, 1996). In particular, inhibition in childhood has been shown to be a risk factor for adolescent depression (Jaffee et al., 2002). However, the measurement of BI in childhood for both of these studies involved raters describing the children as inhibited in the testing situation, upset by strangers and having difficulty concentrating on the tasks. This method is in contrast to Kagan and colleagues (Garcia-Coll et al., 1984) who utilized observed laboratory tasks including reactions to an unfamiliar peer, and a novel toy. Further research is needed, utilizing optimal measures of observed BI in childhood and depression in adolescence.

Further research is also needed to understand the mechanisms underlying the link between BI and the development of depression in adolescence. A body of research has examined the hypotheses that BI children are at increased risk of depression because of the social withdrawal and increased peer problems that can occur for these children. For example, social withdrawal in children is linked to later depression and loneliness in early adolescence (Rubin, Chen, McDougall, Bowker, & McKinnon, 1995). A longitudinal study examining inhibition in adolescence and later depressive symptoms (Buck & Dix, 2012) showed that those who became more inhibited during adolescent also presented with an increase in peer issues, such as declining popularity, which also led to increases in depressive symptoms. Taken together, this previous research suggests that BI may increase the risk for social withdrawal and peer problems to lead to depression in adolescence. The role of social interactions in the development of depression will be discussed in detail later in this chapter.

Effortful Control (EC). EC has been described as having a regulatory function, consisting of lower order traits such as executive attention and inhibitory control (Rothbart, Ahadi, Hersey, & Fisher, 2001). EC has been defined as the capacity to repress a dominant reaction in order to carry out a subdominant response (Rothbart, Ellis, Rueda, & Posner, 2003). It allows a child to suppress affect driven tendencies, and change their behaviour in conflict situations (Rothbart et al., 2003). EC is thought to develop later than other temperament traits, with studies showing toddlers are able to complete a simple EC task at 30 months of age (Rothbart et al., 2003). In addition, EC has also been conceptualised more broadly as a facet of executive functioning, or an outcome from the development of executive attention (Jones, Rothbart, & Posner, 2002).

EC has been presented as a risk factor for the development of anxiety and depression. Decreased levels of EC are associated with increased levels of anxiety in non-clinical children (Muris, de Jong, & Engelen, 2004). Further, EC in childhood has been

linked to later depressive symptoms. Lower levels of EC were found to be cross-sectionally associated with higher levels of depressive symptoms (Verstraeten, Vasey, Raes, & Bijttebier, 2009). Examining this association over time, lower levels of laboratory observed EC were associated with a significant increase in depressive symptoms (Kotelnikova, Mackrell, Jordan, & Hayden, 2015). Specifically, increased attention regulation has been shown to be related to decreased depression, and inhibitory control related to lower internalising and externalising problems (Lengua, 2003). Further exploring how EC in childhood may predict depression in adolescence would improve our understanding of this relationship.

Social Interactions and Depression

As summarised above, this thesis examines existing evidence regarding risk factors for depression and considers risk within the developmental context of adolescence initially by focusing on anxiety, and early temperament. Also of importance are factors that become more salient in early adolescence such as social interactions. Interpersonal relations are thought to play an important role in depression, with problematic social interactions identified as a predictor of depression (Joiner, 1997). The interpersonal theory of depression posits that problematic social relationships in people with depression may lead to the escalation of further depressive symptoms (Coyne, 1976).

As mentioned earlier, peers and social interactions begin to take on greater importance during adolescence. Adolescents report that they are most happy when talking with peers, and have been reported to spend close to one-third of normal waking hours talking with peers during an average week but only speaking to adults for 8% of this time (Csikszentmihalyi, Larson, & Prescott, 1977). Adolescents place a greater importance on peer evaluations in shaping their personal feeling of self-worth than children do, with peer rejection viewed as a sign of their 'unworthiness' (O'Brien & Bierman, 1988). Even with unknown peers, adolescents reported a greater decrease in mood, compared to adults,

following ostracism on a ball throwing computer task (Sebastian, Viding, Williams, & Blakemore, 2010). It is thought that the increase in importance of peer-directed social interactions might help an adolescent develop the necessary social skills to transition towards independence from their parents and home environment (Larson & Richards, 1994).

With the increasing importance of social relationships during adolescence, it is not surprising that problems with peers have been found to be associated with depression. Peer rejection in early adolescence, as reported by the child, a parent, and teacher, has been shown to predict depression two years later in a longitudinal study (Nolan, Flynn, & Garber, 2003). Further, a meta-analysis of previous cross-sectional studies demonstrated that there is strong evidence that peer victimization is associated with depression, with increased victimization related to an increased risk for depression (Hawker & Boulton, 2000). Many early adolescents are self-conscious and have increased concerns about how others view them (Elkind, 1978). Thus, experiencing rejection or victimization from peers during this time of already increased demands could be a factor in the increased rates of depressive symptoms observed throughout adolescence (Hankin et al.,1998).

Interestingly, how an adolescent perceives their social acceptance can be just as important as how their peers actually view them. Children with depressive symptoms conceive more negative views of themselves and of their peers, than those without depressive symptoms (Rudolph & Clark, 2001). A predictive relationship has been shown between perceived social competence and later depressive symptoms in adolescence, suggesting that a negative perception of one's social skills may form a risk factor for the development of depression (Lee, Hankin, & Mermelstein, 2010).

A recent review explored the relationship between peer victimisation and its contribution to adolescent depression (Platt et al., 2013), highlighting that the mechanism underlying the increased vulnerability to peer victimisation in adolescents is not clear. The authors of the review speculate that interpretive and attentional cognitive biases, as well as

neural sensitivity, may influence the relationship between peer victimisation and adolescent depression (Platt et al., 2013). As peer victimisation and other social interactions are a prominent source of potential stress in adolescence, further examination of how they influence the risk for adolescent depression in conjunction with existing vulnerabilities is vital to understanding individual differences in the development of adolescent depression.

Risk-taking And Reward-seeking

Reductions in both reward-seeking (engaging in behaviours that result in a positive outcome) and risk-taking behaviour (behaviours that bring a risk of negative consequences as well as reward) have been shown to be present in those with depression (e.g. Henriques & Davidson, 2000; Pizzagali et al., 2009). Experimental paradigms and brain scanning techniques have been utilised to illustrate these differences. Atypical risk-taking behaviour has been identified as an increase in risk aversion on an experimental paradigm for adults with depression when compared to healthy controls (Smoski et al., 2008). Further, with regards to reward-seeking, depressed adults either perform worse after punishment, indicating increased sensitivity, or do not succeed in improving their performance after receiving negative feedback, signifying dulled responses to reinforcement (Eshel & Rosier, 2010). For example, adult patients with depression show altered sensitivity and reactivity to both reward and punishment in experimental gambling tasks (Cella, Dymond, & Cooper, 2010). Changes in reward-seeking have been shown as a blunted response to reward in adults with depression (McFarland & Klein, 2009), with participants not adjusting their pattern of responding on a memory task in response to the reward condition (Henriques & Davidson, 2000). In addition, there is a large body of evidence that neurological reward circuitry has a role in some of the symptoms of mood disorders such as depression (Russo & Nestler, 2013). For example, adult participants with depression showed significantly weaker neural responses to gains on a monetary incentive delay task compared to health control participants (Pizzagali et al., 2009). Additionally, higher levels of reported depressive

symptoms have been shown to be associated with increased behavioural and cognitive avoidance across social and non-social situations (Cribb, Moulds, & Carter, 2006).

Differences in response to reward have also been shown in those at risk for depression. Individuals with a parent who has experienced depression show less neural activation when awaiting a reward and greater response to punishment than those without familial risk (Gotlib et al., 2010). These differences in processing are present despite the high-risk group never having developed depressive symptoms. This effect has been demonstrated with fearful and happy faces as the stimuli. Children with a parent who has experienced depression showed increased neural response to fearful faces, compared with neutral faces, and decreased response to happy faces (Chai et al., 2015). Differences in reward processing have also been demonstrated to predict the development of later depression. In a community sample of adolescent females, a reduced response to reward was shown to predict depression 18 months later, over and above other risk factors such as baseline depressive symptoms (Nelson, Perlman, Klein, Kotov, & Hajcak, 2016). Further, early adolescents who had difficulty differentiating between low and high reward alternatives on an experimental paradigm were found to be more likely to experience depressive symptoms in a follow up one year later (Forbes, Shaw, & Dahl, 2007).

A blunted response to reward in adolescence may reduce the tendency to take part in behaviours that require approach, such as social interactions with peers. Rewarding or pleasurable activities can provide a buffer for stressful life events, and a reduced sensitivity to reward could exacerbate the negative effects of such stress, which may in turn contribute to the development of depression (Pizzagali, 2014). Adolescence in particular is a time of increased stress due to brain development, social changes and physical maturation (Crone & Dahl, 2012). Thus it follows that reductions in response to rewards may have a particularly deleterious effect at this time (Pizzagali, 2014).

Changes in reward-seeking, and in turn risk-taking behaviour, are an important developmental factor to examine in the adolescent period. Numerous studies have shown that adolescents are more likely than adults to undertake risky behaviours such as binge drinking, smoking, and engaging in violent or other criminal behaviour (Steinberg, 2008). It has been posited that this increase in risk-taking behaviour is the product of the interaction of two separate neurobiological systems in a dual systems model (Steinberg, 2008). Adolescent risk-taking is thought to be characterised by an increase in reward-seeking, stimulated by an increase in dopaminergic activity in the socioemotional system around the time of puberty (e.g. Cameron, 2004; Spear, 2000). However, this increase in reward-seeking takes place before the cognitive control system has matured, which provides more advanced self-regulation and impulse control. It is this temporal gap between the surge in the socioemotional system in early adolescence and the full maturation of the cognitive control system, which does not occur until afterward, that creates a period of increased susceptibility to risk-taking during middle adolescence (Steinberg, 2008). Research supports this dual systems model. Age differences in reward-seeking and risk-taking behaviour were measured across the ages of 10 to 30, in 935 individuals (Steinberg, 2010). It was found that reward-seeking followed a curvilinear pattern, increasing from preadolescence to mid adolescence before declining. On the other hand, impulsivity followed a linear pattern, declining steadily from age 10 onwards (Steinberg, 2010). Thus, changes in reward-seeking and cognitive control at this time make risk-taking behaviour a particularly salient element of adolescence, specifically as the pattern of risk aversion in adolescents at risk for depression seems to be in contrast to the general pattern of an increase in risk-taking during adolescence.

Differences between risk-taking and reward-seeking. It is important to note that risk-taking and reward-seeking are considered two different, although related, concepts. As theorised in the dual systems model (Steinberg, 2008), reward-seeking, or reward motivated

behaviour may play an important function in whether an individual undertakes risky behaviour. When deciding whether to undertake a risky behaviour, it is posited that the salience of the potential reward plays an important role. In the context of adolescent depression, it is possible that a reduction in the salience of rewards may play a role in at risk adolescents avoiding risky and sensation-seeking behaviours that are normative for this age period. As these risky or sensation seeking behaviours are normally high in reward value, avoiding these behaviours may lead to lower levels of positive affect relative to healthy peers, as well as increased social withdrawal and isolation (Silk et al., 2012).

The measurement of reward-seeking and risk-taking behaviour are often confounded in experimental tasks. One example, the Iowa Gambling Task (Bechara, Damasio, Damasio, & Anderson, 1994) is one of the most widely used tools in the literature. Although it has been successful in predicting naturalistic risk-taking behaviours, it is difficult to isolate the specific cognitive components involved, such as risk attitudes and sensitivity to reward or loss (Schonberg, Fox, & Poldrack, 2011). Therefore, it is also important to develop new experimental paradigms that are related to natural behaviour and can be broken down into separate variables related to risk-taking, such as sensitivity to reward and attitudes toward risk-taking (Sharp, Viswanathan, Lanyon, & Barton, 2012).

Social risk-taking. The context of the reward salience and thus the subsequent risk-taking measured is important. Previous research has tended not to measure more meaningful rewards such as social rewards, focusing instead on money or pleasant pictures. Without examining such an important reward essential to human functioning, we are potentially underestimating the scope and mechanisms of reward dysfunction in depression (Forbes, 2009).

In fact, the Social Risk Hypothesis of Depressed Mood proposes that depressed states evolved to reduce risk in social situations where the individual feels that their social value to others is significantly low (Allen & Badcock, 2003). This model suggests that

many aspects of depression can be conceptualised as mechanisms that reduce social risk, including an increased sensitivity to social threat, sending signals to others to reduce this social risk, and finally inhibiting their risk-seeking behaviours (such as being confident or acquisitive). Davey and colleagues (2008) present a similar model, focusing on the development of depression in adolescence. Entry into adolescence is a time when rewards are often abstract and future based, such as belonging to a social group, romantic love and social status. Due to the remodelling of the prefrontal cortex and dopaminergic reward system during adolescence, this allows for the pursuit of complex and temporally distant goals. However, these types of goals are also more easily frustrated than more immediate and salient rewards. This model posits that when these distal rewards are thwarted, they suppress the reward system, and when this suppression is far-reaching and continues for a long enough time frame, depression emerges (Davey et al., 2008).

It has been proposed that a reduction in social risk-taking may be one pathway from anxiety to depression (Silk et al., 2012). Based on previous research, it is posited that anxious adolescents have intact reward processing in non-threatening contexts (e.g. Forbes et al., 2006). However, in social contexts that bring a fear of failure or avoidance for those with anxiety, reward processing may be interrupted, and instead the individual focuses on the potential threat. This is in line with previous research that suggests that threat processing can interrupt reward processing, even in non-disordered individuals (e.g. Frewen Dozois, Joanisse, & Neufeld, 2008). Over time, a number of small threats may start to override the reward system, leading to adolescents avoiding social situations that may bring about risk such as going to a party. This is particularly challenging for anxious adolescents, as they are more prone to interpreting even ambiguous social situations as containing threat cues (Creswell, Schniering, & Rapee, 2005). The authors propose that this social withdrawal in turn leads to depression, in line with Davey and colleagues (2008) previous model of depression (Silk et al., 2012). Further examinations of this model using ecologically valid

paradigms of social risk-taking are needed to measure social withdrawal in at risk adolescents.

Puberty

This thesis focuses on the broader phase of adolescence, a gradual progression from childhood to adulthood (Pickles, et al., 1998). Adolescence consists of a number of transitions rather than a specific moment of completion, with puberty being one of these transitions (Spear, 2000). An overall trend has been present in Western populations over the last 150 years with the mean age of puberty in girls decreasing (Pierce & Hardy, 2012). With a number of psychosocial difficulties associated with early pubertal onset (Patton & Viner, 2007), the inclusion of puberty as a risk factor is more important than ever.

Since the categorisation of secondary sexual changes during puberty into stages of development (Marshall & Tanner, 1969, 1970), researchers have examined whether individual differences in this process of maturation may relate to later emotional well-being (Mendle, Harden, Brooks-Gunn, & Graber, 2010). Early pubertal timing in females has been shown to be associated with the onset of depression (Galvao et al., 2014). Across both females and males, early pubertal timing has predicted increases in depressive symptoms in those with more negative cognitive styles (Hamilton, Hamlat, Stange, Abramson, & Alloy, 2014). These findings imply that early pubertal timing may increase the risk for depression in those with pre-existing vulnerabilities. There are a number of different models that propose explanations as to why early puberty influences psychopathology. The most widely accepted explanation is the maturation disparity hypothesis, that the gap between physical and psychosocial maturities places those who physically mature early at risk for psychopathology (Ge & Natsuaki, 2009). Those who mature earlier must struggle with new stressors earlier than peers who attain these same developmental milestones at a later chronological age (Mendle et al., 2010). For example, early maturing girls with more stressful life events were more likely to be subsequently depressed than those with just early

maturation or increased stressful life events (Ge, Conger, & Elder, 2001). Biological theories of early pubertal timing suggest that it is the physical and hormonal changes that occur during early puberty that lead to an increased risk for psychopathology (Mendle, Turkheimer, & Emery, 2007). Early maturing girls have been shown to have differences in their overall hormonal development (e.g. Apter, Reinila, & Vihko, 1989), but even normative changes in hormones may be problematic for early developers as they experience these changes at time when their peers are more stable, leading to feeling isolated or misunderstood (Mendle et al., 2007).

Due to the importance of pubertal timing in the development of depression, as well as the important role it plays during adolescence, it is necessary to assess for pubertal stage when testing in this population. Also, puberty is an important factor to control for due to the vast differences in pubertal stage that can be found in adolescents of the same chronological age.

Present Research

A large body of evidence demonstrates that adolescence is a critical period for the development of depression. The number of factors shown to increase the risk for depression indicates that depression is the likely result of an accumulation of risk factors, and/or interactions among risk factors (Garber, 2006). The studies in this thesis are designed to explore significant risk factors for adolescent depression in a multi-factor context. By focusing on the key aspects of reward-seeking and risk-taking behaviour, particularly in a social context, this thesis aims to examine salient factors in adolescence. This thesis comprises four stand-alone empirical studies that are presented in publication format[2]. The studies are designed to examine reward-seeking and risk-taking in the context of more established risk factors for depression including temperament, and peer victimisation.

[2] As a result of the thesis by publication format, some repetition across papers is to be expected.

Paper one, presented in Chapter 2, examines the association between temperament, and reward-seeking and risk-taking behaviour in preschool children on an experimental task. Although previous research has examined temperament and risk-taking behaviour (e.g. Suhr & Hammers, 2010), this study is the first to observe whether BI and the EC factor of inhibitory control predict risk-taking behaviour in isolation and also whether these factors interact to predict risk-taking. Utilising a preschool aged sample allows the interaction of these risk factors to be examined before the development of depression.

As risk-taking and reward-seeking behaviour are known to increase in the adolescent period, Paper two, presented in Chapter 3, expanded on the association investigated in Paper one. Although the relationship between BI and adolescent depression has previously been examined in the literature, most studies analysed this relationship at a single time point (e.g. Muris et al., 2003) or did not include potential moderating factors (Jaffee et al., 2002). Using a longitudinal design, Paper two uses a new sample to examine the relationship between BI in early childhood and the development of depressive symptoms in early adolescence. Further, this study includes the potentially moderating factors of risk-taking and pubertal timing.

Using the same sample, Paper three, presented in Chapter 4, continues the examination of risk-taking and reward-seeking as an important risk factor for adolescent depression but also incorporates the essential role of peers and social acceptance. Utilising a novel measure of social risk-taking, this study explores the relationship between depressive symptoms and social risk-taking across two time points in early adolescence. Further, the potentially mediating roles of social acceptance and peer victimisation are examined.

Paper four, presented in Chapter 5, sought to examine how reward-seeking, while controlling for other factors such as risk-taking, may predict the development of depression in adolescence. This study also utilised the same sample as Chapters 3 and 4. A reward-

seeking task was adapted for an adolescent population, and its association with depressive symptoms is explored.

Finally, Chapter 6 provides a summary of the four research studies included in this thesis. In particular, the final chapter discusses how an examination of multiple risk factors for adolescent depression could help to provide a clearer picture of the developmental pathways to psychopathology.

References

Allen, N. B., & Badcock, P. B. (2003). The Social Risk Hypothesis of Depressed Mood: Evolutionary, psychosocial, and neurobiological perspectives. *Psychological Bulletin, 129*(6), 887-913. doi:http://dx.doi.org/10.1037/0033-2909.129.6.887

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry*, *40*(1), 57-87.

Apter, D., Reinilä, M., & Vihko, R. (1989). Some endocrine characteristics of early menarche, a risk factor for breast cancer, are preserved into adulthood. *International Journal of Cancer*, *44*(5), 783-787.

Bates, J. E. (1987). Temperament in infancy. In J. D. E. Osofsky (Ed.), *Handbook of Infant Development* (pp. 1101-1149). New York: John Wiley & Sons.

Bechara A., Damasio A.R., Damasio H., & Anderson S.W. (1994) Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition, 50*, 7–15.

Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects*. University of Pennsylvania Press.

Beck, A. T., Epstein, N., & Harrison, R. (1983). Cognitions, attitudes and personality dimensions in depression. *British Journal of Cognitive Psychotherapy*.

Birmaher, B., Ryan, N.D., Williamson, D.E., Brent, D.A., Kaufman, J., Dahl, R.E., Perel, J., & Nelson, B. (1996). Childhood and adolescent depression: A review of the past 10 years. Part I. *Journal of the American Academy of Child & Adolescent Psychiatry, 35,* 1427-1439.

Buck, K. A., & Dix, T. (2012). Can developmental changes in inhibition and peer relationships explain why depressive symptoms increase in early adolescence? *Journal of Youth and Adolescence, 41*(4), 403-413.

Cameron J. (2004). Interrelationships between hormones, behaviour, and affect during adolescence: Understanding hormonal, physical, and brain changes occurring in association with pubertal activation of the reproductive axis. *Annals of the New York Academy of Sciences, 1021*, 110–123.

Carter, J. S., & Garber, J. (2011). Predictors of the first onset of a major depressive episode and changes in depressive symptoms across adolescence: Stress and negative cognitions. *Journal of Abnormal Psychology, 120*(4), 779-796.

Caspi, A., Moffitt, T. E., Newman, D. L., & Silva, P. A. (1996). Behavioral observations at age 3 years predict adult psychiatric disorders: Longitudinal evidence from a birth cohort. *Archives of General Psychiatry, 53*(11), 1033-1039.

Cella, M., Dymond, S., & Cooper, A. (2010). Impaired flexible decision-making in major depressive disorder. *Journal of Affective Disorders, 124*(1-2), 207-210. doi:http://dx.doi.org/10.1016/j.jad.2009.11.013

Chai, X.J., Hirshfeld-Becker, D., Biederman, J., Uchida, M., Doehrmann, O., Leonard, J.A., Salvatore, J., Kenworthy, T., Brown, A., Kagan, E. and de los Angeles, C. (2015). Functional and structural brain correlates of risk for major depression in children with familial depression. *NeuroImage: Clinical*, *8*, 398-407.

Chronis-Tuscano, A., Degnan, K. A., Pine, D. S., Perez-Edgar, K., Henderson, H. A., Diaz, Y., Raggi, V.L., & Fox, N. A. (2009). Stable early maternal report of behavioral inhibition predicts lifetime social anxiety disorder in adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry, 48*(9), 928-935.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*(3), 316-336. doi:http://dx.doi.org/10.1037/0021-843X.100.3.316

Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, *103*(1), 103.

Clauss, J. A., & Blackford, J. U. (2012). Behavioral Inhibition and risk for developing Social Anxiety Disorder: A meta-analytic study. *Journal of the American Academy of Child & Adolescent Psychiatry, 51*(10), 1066-1075.e1061. doi:http://dx.doi.org/10.1016/j.jaac.2012.08.002

Clauss, J. A., Avery, S. N., & Blackford, J. U. (2015). The nature of individual differences in inhibited temperament and risk for psychiatric disease: A review and meta-analysis. *Progress in Neurobiology, 127–128*, 23-45. doi:http://dx.doi.org/10.1016/j.pneurobio.2015.03.001

Cohen, P., Cohen, J., Kasen, S., Velez, C.N., Hartmark, C., Johnson, J., Rojas, M., Brook, J., & Streuning, E.L. (1993). An epidemiological study of disorders in late childhood and adolescence: I. Age- and gender-specific prevalence. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 34,* 851-867.

Cole, D. A., Peeke, L. G., Martin, J. M., Truglio, R., & Seroczynski, A. (1998). A longitudinal look at the relation between depression and anxiety in children and adolescents. *Journal of Consulting and Clinical Psychology, 66*(3), 451-460. doi:http://dx.doi.org/10.1037/0022-006X.66.3.451

Coyne, J. C. (1976). Toward an interactional description of depression. *Psychiatry, 39*(1), 28-40.

Creswell, C., Schniering, C. A., & Rapee, R. M. (2005). Threat interpretation in anxious children and their mothers: comparison with nonclinical children and the effects of treatment. *Behaviour Research and Therapy*, *43*(10), 1375-1381.

Cribb, G., Moulds, M. L., & Carter, S. (2006). Rumination and experiential avoidance in depression. *Behaviour Change*, *23*(03), 165-176.

Crone, E. A., & Dahl, R. E. (2012). Understanding adolescence as a period of social–affective engagement and goal flexibility. *Nature Reviews Neuroscience*, *13*(9), 636-650.

Csikszentmihalyi, M., Larson, R. & Prescott (1977). The ecology of adolescent activity and experience. *Journal of Youth and Adolescence, 6* (3), 281-294. doi:10.1007/BF02138940

Cummings, C. M., Caporino, N. E., & Kendall, P. C. (2014). Comorbidity of Anxiety and Depression in Children and Adolescents: 20 Years After. *Psychological Bulletin, 140*(3), 816.

Davey, C. G., Yucel, M., & Allen, N. B. (2008). The emergence of depression in adolescence: Development of the prefrontal cortex and the representation of reward. *Neuroscience and Biobehavioral Reviews, 32*(1), 1-19. doi:http://dx.doi.org/10.1016/j.neubiorev.2007.04.016

Eley, T. C., & Stevenson, J. (1999). Exploring the covariation between anxiety and depression symptoms: a genetic analysis of the effects of age and sex. *Journal of Child Psychology and Psychiatry*, *40*(8), 1273-1282.

Elkind, D. (1978). Understanding the young adolescent. *Adolescence, 49*, 127–134.

Eshel, N., & Roiser, J. P. (2010). Reward and punishment processing in depression. *Biological Psychiatry, 68*(2), 118-124. doi:http://dx.doi.org/10.1016/j.biopsych.2010.01.027

Forbes, E. E., May, J., Siegle, G. J., Ladouceur, C. D., Ryan, N. D., Carter, C. S., Birhmaher, B., Axelson, D.A., & Dahl, R. E. (2006). Reward-related decision-making in pediatric major depressive disorder: An fMRI study. *Journal of Child Psychology and Psychiatry, 47*(10), 1031-1040. doi:http://dx.doi.org/10.1111/j.1469-7610.2006.01673.x

Forbes, E. E., Shaw, D. S., & Dahl, R. E. (2007). Alterations in reward-related decision making in boys with recent and future depression. *Biological Psychiatry, 61*(5), 633-639. doi:http://dx.doi.org/10.1016/j.biopsych.2006.05.026

Forbes, E. E. (2009). Where's the fun in that? Broadening the focus on reward function in

depression. *Biological Psychiatry, 66*(3), 199-200. doi:http://dx.doi.org/10.1016/j.biopsych.2009.05.001

Fox, N. A., Henderson, H. A., Rubin, K. H., Calkins, S. D., & Schmidt, L. A. (2001). Continuity and discontinuity of behavioral inhibition and exuberance: Psychophysiological and behavioral influences across the first four years of life. *Child Development, 72*(1), 1-21. doi:http://dx.doi.org/10.1111/1467-8624.00262

Fox, N. A., Henderson, H. A., Marshall, P. J., Nichols, K. E., & Ghera, M. M. (2005). Behavioral inhibition: linking biology and behavior within a developmental framework. *Annual Review of Psychology*, *56*, 235-262.

Frewen, P. A., Dozois, D. J., Joanisse, M. F., & Neufeld, R. W. (2008). Selective attention to threat versus reward: Meta-analysis and neural-network modeling of the dot-probe task. *Clinical Psychology Review*, *28*(2), 307-337.

Fröjd, S. A., Nissinen, E. S., Pelkonen, M. U. I., Marttunen, M. J., Koivisto, A. M., & Kaltiala-Heino, R. (2008). Depression and school performance in middle adolescent boys and girls. *Journal of Adolescence, 31*(4), 485-498.

Galvao, T. F., Silva, M. T., Zimmermann, I. R., Souza, K. M., Martins, S. S., & Pereira, M. G. (2014). Pubertal timing in girls and depression: A systematic review. *Journal of Affective Disorders, 155*, 13-19. doi:http://dx.doi.org/10.1016/j.jad.2013.10.034

Garber, J. (2006). Depression in Children and Adolescents. Linking Risk Research and Prevention. *American Journal of Preventive Medicine, 31*(6 SUPPL. 1), 104-125.

Garber, J., & Weersing, V. R. (2010). Comorbidity of anxiety and depression in youth: Implications for treatment and prevention. *Clinical Psychology: Science and Practice, 17*(4), 293-306.

Garcia Coll, C., Kagan, J., & Reznick, J. (1984). Behavioral inhibition in young children. *Child Development, 55*(3), 1005-1019.

Ge, X., Conger, R. D., & Elder, G. H., Jr. (2001). Pubertal transition, stressful life events,

and the emergence of gender differences in adolescent depressive symptoms. *Developmental Psychology, 37*(3), 404-417. doi:http://dx.doi.org/10.1037/0012-1649.37.3.404

Ge, X., & Natsuaki, M. N. (2009). In search of explanations for early pubertal timing effects on developmental psychopathology. *Current Directions in Psychological Science, 18*(6), 327-331. doi:http://dx.doi.org/10.1111/j.1467-8721.2009.01661.x

Gladstone, G. L., & Parker, G. B. (2006). Is behavioral inhibition a risk factor for depression? *Journal of Affective Disorders, 95*(1-3), 85-94. doi:http://dx.doi.org/10.1016/j.jad.2006.04.015

Gotlib, I. H., Hamilton, J., Cooney, R. E., Singh, M. K., Henry, M. L., & Joormann, J. (2010). Neural processing of reward and loss in girls at risk for major depression. *Archives of General Psychiatry, 67*(4), 380-387. doi:http://dx.doi.org/10.1001/archgenpsychiatry.2010.13

Hamilton, J. L., Hamlat, E. J., Stange, J. P., Abramson, L. Y., & Alloy, L. B. (2014). Pubertal timing and vulnerabilities to depression in early adolescence: Differential pathways to depressive symptoms by sex. *Journal of Adolescence, 37*(2), 165-174.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology, 107*(1), 128.

Hankin, B. L. (2012). Future directions in vulnerability to depression among youth: Integrating risk factors and processes across multiple levels of analysis. *Journal of Clinical Child & Adolescent Psychology, 41*(5), 695-718. doi:10.1080/15374416.2012.711708

Hawker, D. S., & Boulton, M. J. (2000). Twenty years' research on peer victimization and psychosocial maladjustment: A meta-analytic review of cross-sectional studies.

Journal of Child Psychology and Psychiatry, 41(4), 441-455.

Henriques, J. B., & Davidson, R. J. (2000). Decreased responsiveness to reward in depression. *Cognition and Emotion, 14*(5), 711-724. doi:http://dx.doi.org/10.1080/026999300501176 84

Hirshfeld-Becker, D. R., Micco, J. A., Simoes, N. A., & Henin, A. (2008, May). High-risk studies and developmental antecedents of anxiety disorders. In *American Journal of Medical Genetics Part C: Seminars in Medical Genetics* (Vol. 148, No. 2, pp. 99-117). Wiley Subscription Services, Inc., A Wiley Company.

Hudson, J. L., & Dodd, H. F. (2012). Informing early intervention: Preschool predictors of anxiety disorders in middle childhood. *PLoS ONE, 7*(8).

Hughes, A. A., Heimberg, R. G., Coles, M. E., Gibb, B. E., Liebowitz, M. R., & Schneier, F. R. (2006). Relations of the factors of the tripartite model of anxiety and depression to types of social anxiety. *Behaviour Research and Therapy, 44*(11), 1629-1641. doi:http://dx.doi.org/10.1016/j.brat.2005.10.015

Jaffee, S. R., Moffitt, T. E., Caspi, A., Fombonne, E., Poulton, R., & Martin, J. (2002). Differences in early childhood risk factors for juvenile-onset and adult-onset depression. *Archives of General Psychiatry, 59*(3), 215-222. doi:http://dx.doi.org/10.1001/archpsyc.59.3.215

Joiner Jr, T. E. (1997). Shyness and low social support as interactive diatheses, with loneliness as mediator: Testing an interpersonal-personality view of vulnerability to depressive symptoms. *Journal of Abnormal Psychology*, *106*(3), 386.

Jones, L. B., Rothbart, M. K., & Posner, M. I. (2003). Development of executive attention in preschool children. *Developmental Science*, *6*(5), 498-504.

Kagan, J., Reznick, J., Snidman, N., Gibbons, J., & Johnson, M.O. (1988). Childhood derivatives of inhibition and lack of inhibition to the unfamiliar. *Child Development, 59*(6), 1580-1589.

Karevold, E., Ystrom, E., Coplan, R. J., Sanson, A. V., & Mathiesen, K. S. (2012). A prospective longitudinal study of shyness from infancy to adolescence: Stability, age-related changes, and prediction of socio-emotional functioning. *Journal of Abnormal Child Psychology*, *40*(7), 1167-1177.

Kashani, J.H., & Carlson, G.A. (1987). Seriously depressed preschoolers. *American Journal of Psychiatry, 144,* 348-350.

Kendler, K. S., Gardner, C. O., & Prescott, C. A. (2002). Toward a comprehensive developmental model for major depression in women. *American Journal of Psychiatry, 159*(7), 1133-1145.

Kerr, M., Lambert, W. W., Stattin, H., & Klackenberg-Larsson, I. (1994). Stability of inhibition in a Swedish longitudinal sample. *Child Development*, *65*(1), 138-146.

Kessler, R.C., Avenevoli, S., & Merikangas, K.R. (2001). Mood disorders in children and adolescents: An epidemiologic perspective. *Biological Psychiatry, 49,* 1002-1014.

Kessler, R.C., McGonagle, K.A., Zhao, S., Nelson, C.B., Hughes, M., Eshleman, S., Wittchen, H-U., & Kendler, K.S. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. *Archives of General Psychiatry, 51,* 8-19.

Klein, D.N., Lewinsohn, P.M., Seeley, J.R., & Rohde, P. (2001). A family study of major depressive disorder in a community sample of adolescents. *Archives of General Psychiatry, 58,* 13-20.

Kotelnikova, Y., Mackrell, S. V., Jordan, P. L., & Hayden, E. P. (2015). Longitudinal associations between reactive and regulatory temperament traits and depressive symptoms in middle childhood. *Journal of Clinical Child & Adolescent Psychology*, *44*(5), 775-786.

Larson, R., & Richards, M. H. (1994). *Divergent realities: The emotional lives of mothers, fathers, and adolescents*. Basic Books.

Lau, J.Y.F., & Eley, T.C. (2010). The genetics of mood disorders. *Annual Review of Clinical Psychology, 6,* 313-337.

Lee, A., Hankin, B. L., & Mermelstein, R. J. (2010). Perceived social competence, negative social interactions, and negative cognitive style predict depressive symptoms during adolescence. *Journal of Clinical Child and Adolescent Psychology, 39*(5), 603-615. doi:http://dx.doi.org/10.1080/15374416.2010.501284

Lejuez, C., Read, J. P., Kahler, C. W., Richards, J. B., Ramsey, S. E., Stuart, G. L., Strong, D.R., & Brown, R. A. (2002). Evaluation of a behavioral measure of risk-taking: The Balloon Analogue Risk Task (BART). *Journal of Experimental Psychology: Applied, 8*(2), 75-84. doi:http://dx.doi.org/10.1037/1076-898X.8.2.75

Lengua, L. J. (2003). Associations among emotionality, self-regulation, adjustment problems, and positive adjustment in middle childhood. *Journal of Applied Developmental Psychology, 24*(5), 595-618. doi:http://dx.doi.org/10.1016/j.appdev.2003.08.002

Lewinsohn, P. M., Rohde, P., & Seeley, J. R. (1994). Psychosocial risk factors for future adolescent suicide attempts. *Journal of Consulting and Clinical Psychology, 62*(2), 297-305.

McFarland, B. R., & Klein, D. N. (2009). Emotional reactivity in depression: Diminished responsiveness to anticipated reward but not to anticipated punishment or to nonreward or avoidance. *Depression and Anxiety, 26*(2), 117-122. doi:http://dx.doi.org/10.1002/da.20513

Marshall, W. A., & Tanner, J. M. (1969). Variations in pattern of pubertal changes in girls. *Archives of Disease in Childhood, 44*(235), 291-303.

Marshall, W., & Tanner, J. (1970). Variations in the pattern of pubertal changes in boys. *Archives of Diseases in Childhood, 45*(239), 13-23. doi:http://dx.doi.org/10.1136/adc.45.239.13

Mendle, J., Turkheimer, E., & Emery, R. E. (2007). Detrimental psychological outcomes associated with early pubertal timing in adolescent girls. *Developmental Review, 27*(2), 151-171. doi:http://dx.doi.org/10.1016/j.dr.2006.11.001

Mendle, J., Harden, K., Brooks-Gunn, J., & Graber, J. A. (2010). Development's tortoise and hare: Pubertal timing, pubertal tempo, and depressive symptoms in boys and girls. *Developmental Psychology, 46*(5), 1341-1353. doi:http://dx.doi.org/10.1037/a0020205

Moffitt, T. E., Harrington, H., Caspi, A., Kim-Cohen, J., Goldberg, D., Gregory, A. M., & Poulton, R. (2007). Depression and generalized anxiety disorder: Cumulative and sequential comorbidity in a birth cohort followed prospectively to age 32 years. *Archives of General Psychiatry, 64*(6), 651-660.

Muris, P., Meesters, C., & Spinder, M. (2003). Relationships between child- and parent-reported behavioural inhibition and symptoms of anxiety and depression in normal adolescents. *Personality and Individual Differences, 34*(5), 759-771. doi:http://dx.doi.org/10.1016/S0191-8869%2802%2900069-7

Muris, P., de Jong, P. J., & Engelen, S. (2004). Relationships between neuroticism, attentional control, and anxiety disorders symptoms in non-clinical children. *Personality and Individual Differences*, *37*(4), 789-797.

Nelson, B. D., Perlman, G., Klein, D. N., Kotov, R., & Hajcak, G. (2016). Blunted neural response to rewards as a prospective predictor of the development of depression in adolescent girls. *American Journal of Psychiatry, 173*(12), 1223-1230.

Nolan, S. A., Flynn, C., & Garber, J. (2003). Prospective relations between rejection and depression in young adolescents. *Journal of Personality and Social Psychology, 85*(4), 745.

O'Brien, S. F., & Bierman, K. L. (1988). Conceptions and perceived influence of peer groups: Interviews with preadolescents and adolescents. *Child Development, 59(*5), 1360–1365.

Patton G. C., & Viner R. (2007). Pubertal transitions in health. *Lancet, 369,* 1130–1139.

Paus, T. (2005). Mapping brain maturation and cognitive development during adolescence. *Trends in Cognitive Sciences*, *9*(2), 60-68.

Pickles A, Pickering K, Simonoff E, Silberg J, Meyer J, Maes H. (1998). Genetic clocks and soft events: A twin model for pubertal development and other recalled sequences of developmental milestones, transitions, or ages at onset. *Behavior Genetics*, *28,* 243–253.

Pierce, M., & Hardy, R. (2012). Commentary: The decreasing age of puberty—as much a psychosocial as biological problem? *International Journal of Epidemiology*, *41*(1), 300–302. http://doi.org/10.1093/ije/dyr227

Pine, D.S., Cohen, E., Cohen, P., & Brook, J. (1999). Adolescent depressive symptoms as predictors of adult depression: Moodiness or mood disorder? *American Journal of Psychiatry, 156,* 133-135.

Pine, D.S., Cohen, P., Gurley, D., Brook, J., & Ma, Y. (1998). The risk for early-adulthood anxiety and depressive disorders in adolescents with anxiety and depressive disorders. *Archives of General Psychiatry, 55,* 56-64.

Pizzagalli, D. A., Holmes, A. J., Dillon, D.G., Goetz, E.L., Birk, J.L., Bogdan, R., Dougherty, D.D., Iosifescu, D.V., Rauch, S.L., & Fava, M. (2009). Reduced caudate and nucleus accumbens response to rewards in unmedicated individuals with major depressive disorder. *American Journal of Psychiatry, 166*(6), 702-710.

Pizzagalli, D. A. (2014). Depression, stress, and anhedonia: toward a synthesis and integrated model. *Annual Review of Clinical Psychology*, *10*, 393-423.

Platt, B., Kadosh, K. C., & Lau, J. Y. (2013). The role of peer rejection in adolescent

depression. *Depression and Anxiety, 30*(9), 809-821. doi:http://dx.doi.org/10.1002/da.22120

Rohde, P., Lewinsohn, P. M., Klein, D. N., Seeley, J. R., & Gau, J. M. (2013). Key characteristics of major depressive disorder occurring in childhood, adolescence, emerging adulthood, and adulthood. *Clinical Psychological Science, 1*(1), 41-53.

Rothbart, M. K., Ahadi, S. A., Hersey, K. L., & Fisher, P. (2001). Investigations of temperament at three to seven years: The Children's Behavior Questionnaire. *Child Development, 72*(5), 1394-1408.

Rothbart, M. K., Ellis, L. K., Rueda, M., & Posner, M. I. (2003). Developing mechanisms of temperamental effortful control. *Journal of Personality, 71*(6), 1113-1143. doi:http://dx.doi.org/10.1111/1467-6494.7106009

Rubin, K. H., Chen, X., McDougall, P., Bowker, A., & McKinnon, J. (1995). The Waterloo Longitudinal Project: Predicting internalizing and externalizing problems in adolescence. *Development and Psychopathology, 7*(04), 751-764.

Rubin, K. H., Burgess, K. B., & Hastings, P. D. (2002). Stability and social-behavioral consequences of toddlers' inhibited temperament and parenting behaviors. *Child Development, 73*(2), 483-495.

Rudolph, K. D., & Clark, A. G. (2001). Conceptions of relationships in children with depressive and aggressive symptoms: Social-cognitive distortion or reality? *Journal of Abnormal Child Psychology, 29*(1), 41-56.

Rudolph, K. D., Flynn, M., & Abaied, J. L. (2008). A developmental perspective on interpersonal theories of youth depression. In J. R. Z. Abela & B. L. Hankin (Eds.), *Handbook of depression in children and adolescents* (pp. 79–102). New York: Guilford Press.

Russo, S. J., & Nestler, E. J. (2013). The brain reward circuitry in mood disorders. *Nature Reviews Neuroscience*, *14*(9), 609-625.

Schonberg, T., Fox, C. R., & Poldrack, R. A. (2011). Mind the gap: bridging economic and naturalistic risk-taking with cognitive neuroscience. *Trends in Cognitive Sciences, 15*(1), 11-19.

Sebastian, C., Viding, E., Williams, K. D., & Blakemore, S. J. (2010). Social brain development and the affective consequences of ostracism in adolescence. *Brain and Cognition, 72*(1), 134-145. doi:10.1016/j.bandc.2009.06.008

Sharp, M. E., Viswanathan, J., Lanyon, L. J., & Barton, J. J. (2012). Sensitivity and bias in decision-making under risk: Evaluating the perception of reward, its probability and value. *PLoS ONE, 7*(4).

Silk, J. S., Davis, S., McMakin, D. L., Dahl, R. E., & Forbes, E. E. (2012). Why do anxious children become depressed teenagers? The role of social evaluative threat and reward processing. *Psychological Medicine*, *42*(10), 2095-2107.

Smoski, M. J., Lynch, T. R., Rosenthal, M., Cheavens, J. S., Chapman, A. L., & Krishnan, R. R. (2008). Decision-making and risk aversion among depressive adults. *Journal of Behavior Therapy and Experimental Psychiatry, 39*(4), 567-576. doi:http://dx.doi.org/10.1016/j.jbtep.2008.01.004

Spear LP (2000). The adolescent brain and age-related behavioral manifestations. *Neuroscience and Biobehavioral Reviews, 24*, 417–463.

Steer, R. A., Clark, D. A., Beck, A. T., & Ranieri, W. F. (1995). Common and specific dimensions of self-reported anxiety and depression: A replication. *Journal of Abnormal Psychology*, *104*(3), 542.

Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review, 28*(1), 78-106.

Steinberg, L. (2010). A dual systems model of adolescent risk-taking. *Developmental Psychobiology, 52*(3), 216-224.

Suhr, J., & Hammers, D. (2010). Who fails the Iowa Gambling Test (IGT)? Personality,

neuropsychological, and near-infrared spectroscopy findings in healthy young controls. *Archives of Clinical Neuropsychology, 25*(4), 293-302. doi:http://dx.doi.org/10.1093/arclin/acq017

Verstraeten, K., Vasey, M. W., Raes, F., & Bijttebier, P. (2009). Temperament and risk for depressive symptoms in adolescence: Mediation by rumination and moderation by effortful control. *Journal of Abnormal Child Psychology*, *37*(3), 349-361.

World Health Org. Region. Off. South-East Asia. (2001). *Conquering Depression.* New Delhi: World Health Org.

Zisook, S., Lesser, I., Stewart, J. W., Wisniewski, S. R., Balasubramani, G. K., Fava, M.,Gilmer, W S., Dresselhaus, T R, Thase, M E, Nierenberg, A A, Trivedi, M H., & Trivedi, M. H. (2007). Effect of age at onset on the course of major depressive disorder. *American Journal of Psychiatry*, *164*(10), 1539-1546.

The following chapter presents the first empirical paper of this thesis entitled "Risk-taking and Inhibitory Control in Behaviourally Inhibited and Disinhibited Preschool Children". This paper examines the association between temperament and risk-taking behaviour in early childhood. The use of a preschool aged sample allows for these two risk factors to be examined before the development of depression in adolescence. In particular, this paper focuses on Behavioural Inhibition and Inhibitory Control, exploring how these risk factors for later psychopathology relate to risk-taking behaviour when examined in preschool aged children. Inhibitory Control has been defined as a lower order trait of the temperament attribute Effortful Control (see Chapter 1). In addition, Inhibitory Control is considered an aspect of executive functioning, as described in this Chapter.

Chapter Two

Risk-Taking and Inhibitory Control in Behaviourally Inhibited and Disinhibited Preschool Children.

Talia M. Morris, Jennifer L. Hudson, & Helen F. Dodd

This paper has been published:

Morris, T. M., Hudson, J. L., & Dodd, H. F. (2014). Risk-taking and inhibitory control in behaviourally inhibited and disinhibited preschool children. *Personality and Individual Differences*, *71*, 113-117.

Abstract

The temperament style behavioural inhibition (BI) has been implicated as a risk factor for the development of internalising disorders such as anxiety. Of interest is what factors influence the developmental trajectories of both inhibited and disinhibited children and the development of psychopathology. One such factor is risk-taking behaviour. Using the computer based Balloon Analogue Risk Task, we assessed risk-taking behaviour in behaviourally inhibited (n = 27) and behaviourally disinhibited (n = 43) children. This is the first study to examine the relationship between BI, executive functioning and risk-taking. The results indicated behavioural inhibition was not related to risk-taking but that inhibitory control predicted reward focused results. These findings illustrate how inhibitory control affects risk-taking and risk avoidance in both inhibited and disinhibited children.

Keywords: Behavioural inhibition; temperament; risk-taking; risk avoidance; inhibitory control; executive functioning

Behavioural inhibition (BI) is a temperament style defined by withdrawal and restraint towards the unfamiliar (Garcia Coll, Kagan, & Reznick, 1984). Around 15% of typically developing children exhibit this temperament style and it is moderately stable across the lifespan (see Fox, Henderson, Marshall, Nichols, & Ghera, 2005 for a review) with children at the extremes showing the most stability across time (Kerr, Lambert, Stattin, & Klackenberg-Larsson, 1994). BI preschoolers take time to warm up to new children or adults and become quiet and socially restrained around unfamiliar people (Coplan, DeBow, Schneider, & Graham, 2009; Kagan, Reznick, Snidman, Gibbons, & Johnson, 1988).

BI has been identified as a risk factor for the development of internalising disorders, such as anxiety, while behavioural disinhibition (BUI) has been identified as a risk factor for externalising disorders, such as attention deficit/hyperactivity disorder (ADHD). For example, a recent longitudinal study by Hudson and Dodd (2012), demonstrated that BI in preschool children significantly predicted anxiety at age 9, over and above initial anxiety. Conversely, early BUI has been associated with increased childhood disruptive behaviour, including ADHD (Hirshfeld-Becker et al., 2007). Early BUI has also been associated with increased aggressive behaviour in preschool children (Kimonis et al., 2006).

While BI has been clearly implicated in the development of psychopathology, not all BI or BUI children go on to develop mental health problems. Thus it is important to identify factors that may protect against or increase risk for psychopathology in BI and BUI children. One such factor implicated in the development and maintenance of anxiety is risk avoidance behaviour. Anxious individuals avoid specific fear-relevant threats. For example, individuals with social phobia avoid interactions with new people (Barlow, 2002). Also, behavioural avoidance in children (as reported by parents) has been demonstrated to predict changes in anxiety over time (Whiteside, Gryczkowski, Ale, Brown-Jacobsen & McCarthy, 2013). In addition to specific avoidance, a more pervasive risk avoidance has also been associated with anxiety symptoms and disorder. For instance, individuals with high levels of

trait anxiety reported less willingness to engage in risk-taking decisions (Maner & Schmidt, 2006). Furthermore, anxious individuals self-report substantially higher risk aversion when compared with other clinical patients and non-clinical controls (Maner et al., 2007). Risk-taking behaviour is also a factor identified as playing a possible role in the development and maintenance of externalising disorders such as ADHD (Humphreys & Lee, 2011). Children with ADHD are more likely to take risks and make poor decisions on a computer gambling task than healthy controls (DeVito et al., 2008).

Risk-taking may play an important role in developmental pathways to psychopathology in BI and BUI children; the more a BI child avoids risk, the less their negative beliefs about potential threats, and their ability to cope with threats, are challenged. Such challenges, or exposures, are necessary learning experiences that enable children to overcome anxiety. By avoiding risk, a BI child's risk for an anxiety disorder may therefore increase. At the other end of the scale, the more a BUI child takes excessive risks, the higher the probability that the behaviour will be inadvertently reinforced (e.g. a reaction from a parent that signifies increased attention to the child), increasing the likelihood that the risky behaviour is repeated. This reinforcement of risk-taking behaviour may potentially increase risk for externalising problems such as aggressive behaviour. Given the possible links between BI and BUI and later risks, we need to further investigate this relationship.

BI is not the only developmental factor associated with risk-taking behaviour. Executive functioning, such as the facet of inhibitory control, has also recently been implicated in the regulation of risk-taking behaviour (for a review see Somerville and Casey 2010). Rothbart and colleagues (2003) define inhibitory control as the capability to repress an overriding response in order to perform a less dominant one. In particular, inhibitory control may be relevant for preventing excessive risk-taking behaviour by helping children to inhibit maladaptive responses in favour of a more balanced choice (Lahat et al., 2012).

Inhibitory control has also been implicated in the development of internalising and externalising problems. Low inhibitory control has been associated with higher levels of internalising and emotional symptoms in non-clinical children aged 8-10 years of age (Vuontela et al., 2013). However, in a clinical sample, depressed children and adolescents show a more conservative response style on neuropsychological tests related to inhibitory control (Cataldo, Nobile, Lorusso, Battaglia, & Molteni, 2005). Evidence in support of the relationship between inhibitory control and externalising symptoms is also mixed. Decreased inhibitory control has been shown to be correlated with increased ADHD symptoms in children (Brocki, Nyberg, Thorell, & Bohlin, 2007). Also, anger-prone infants displayed less inhibitory control than less anger-prone infants (He et al., 2010).

There is some indication that a child's temperament may influence the way inhibitory control is related to later problems. For example, in BUI children, greater inhibitory control has been linked to reduced externalising behaviour problems such as levels of hyperactivity (Thorell, Bohlin, & Rydell, 2004). The role inhibitory control plays in internalising problems for BI children is less clear, with studies demonstrating conflicting findings. For instance, White and colleagues (2011) found that within children who had high levels of inhibitory control, high levels of BI predicted later anxiety. Conversely, BI was not associated with anxiety in children with low levels of inhibitory control (White, et al., 2011). A similar study found that children with both higher levels of BI and high levels of inhibitory control were more likely to experience social anxiety than those with high levels of BI but low levels of inhibitory control (Thorell, et al., 2004). However, a third study reported that increased inhibitory control was linked to fewer internalising and externalising problems in BI children (Lengua, 2003). It is possible that the increased conscious control of impulses in those with higher levels of inhibitory control, may help those children regulate their behaviour and feelings, but for others, such as BI children, it may increase their behavioural tendency to focus on more threatening stimuli (Degnan & Fox, 2007).

Further work is needed to examine the differing impacts inhibitory control has on the developmental trajectories of BI and BUI children, and the implications for later internalising and externalising problems.

'Real life' risk-taking, or risk avoidance behaviour is difficult to replicate in a controlled laboratory setting. Participants may provide socially desirable responses and may potentially lack the insight to provide a true report of their own risk-taking behaviour (Ladouceur et al., 2000). Behavioural measures of risk-taking have been developed, including the Balloon Analogue Risk Task or BART (Lejuez et al., 2002). In this task, participants inflate a balloon that can either grow larger or explode. A larger balloon is naturally associated with an increased probability of explosion. Unlike other behavioural risk-taking tasks in which the risk is arbitrarily controlled, the risk in the BART task is the probability that the balloon will explode. Participants choose whether to continue pumping up the balloon for a larger reward, and therefore have a choice in how much risk they take. The risk in the BART task was designed to model risk in the natural environment, with risk-taking up to a certain point leading to positive consequences (more points) and excessive risk-taking leading to negative consequences (loss of points). The BART correlates with risky behaviour such as substance abuse, both in adults and adolescents (Aklin, Lejuez, Zvolensky, Kahler, & Gwadz, 2005; Lejuez, Aklin, Bornovalova, & Moolchan, 2005). A youth version has been created using a points system, with the points exchangeable for prizes at the end (BART-Y (Lejuez et al., 2007). Although the BART-Y has been shown to be a useful risk-taking measure for adolescents, only one study has examined task performance as well as temperament in preschool aged children thus far (Lahat, et al., 2012).

In the present study, we evaluated the potential relationship between BI, inhibitory control and risk-taking as measured using the BART-Y. Based on previous research, it was hypothesised that: (1) BI children will take less risk on the BART-Y than BUI children; (2)

high levels of inhibitory control will correlate with children taking less risk on the BART-Y; (3) temperament and inhibitory control will interact to predict risk-taking; BUI children with high inhibitory control will have less risk-taking than BUI children with low inhibitory control. As previous findings for how inhibitory control affects risk-taking in BI children have been inconsistent, this aspect of the study was exploratory.

Method

Participants

The sample comprised 60 BI and 86 BUI children, recruited when the children were approximately age 4 (M = 48 months, SD = 4, 45% male) through local preschools and via an advertisement in a free parenting magazine. Advertisements specified that the families would participate in a research project on anxiety in preschool children, and that we were interested in shy and confident children. Due to the exclusion of participants who did not meet the BI/BUI cut off at the second screening (see Measures section), the final sample included 60 participants (35 male) aged between 3 years 5 months and 4 years 6 months (M = 48 months, SD = 3.85 months) when assessed, 27 BUI and 43 BI children. Of this final sample 61% described their ethnicity as Oceanic, with the majority of the remainder being Asian. There were no significant differences between those who were included in the final sample and those who were not on BI classification, maternal age, family income or number of siblings (ps > .05). Significant differences were found for ethnicity, χ^2 (2) = 6.63, p = .04, with greater numbers of children of Asian ethnicity included in the final sample.

Measures

Maternal-report of BI. After completing a screening questionnaire when first calling about the study (Short Temperament Scale for Children, STSC; Sanson, Smart, Prior, Oberklaid, & Pedlow, 1994) children scoring one standard deviation above or below the normative mean on the Approach Scale were classified as BI or BUI, respectively (Cronbach's alpha = .93). To create a more conservative BI classification, mothers

completed the Approach Scale of the STSC again just prior to the laboratory session. Only those whose classification was consistent at both screening questionnaires were included in the study. The STSC has been used previously to classify children as BI (Rapee, Kennedy, Ingram, Edwards, & Sweeney, 2005).

Observed BI. BI was also assessed using laboratory tasks comparable to those used by Kagan and colleagues (Kagan, Reznick, & Gibbons, 1989). Children's responses to a novel toy, new room, same-sex unfamiliar peer and a masked experimenter were observed. Behaviours used to determine inhibition status included: time spent proximal to mother; time spent staring at the peer; time spent talking; number of approaches to the stranger; and number of approaches to the peer. Participants were defined as BI by observation if they scored above a predetermined cut-off on three or more of these five behaviours (Hudson, Dodd & Bovopoulos, 2011; Rapee, et al., 2005).

Risk-taking. Children completed the BART-Y (Lejuez et al., 2007) in the laboratory session. The BART-Y is a computerised task in which a simulated balloon and pump are displayed on the screen along with measures of the participant's progress. Participants were told that each pump of the balloon yields a point, which can be added to an overall prize total displayed on a 'prize meter'. The balloon can explode at any point, and if it does reward points for that round are lost. At each pump, the participant is faced with the choice of saving their points for that round or continuing pumping potentially increasing their round total but also increasing the risk of exploding the balloon, wiping out their reward for that round.

The participants were told they would receive a prize at the end of the BART-Y, and the size of the prize depended upon the total points received. A prize meter was used so that participants could see how many points they had. The task contained 15 balloons in total. Consistent with previous studies (e.g. Lejuez et al., 2007), the average number of pumps across balloons that did not explode was used as a dependent variable, referred to as

'average adjusted pumps'. Also included was the total number of points scored by the participants, as a measure of reward focused behaviour, and total explosions across the task, as another risk-taking variable.

Six participants reached the maximum number of pumps, exploding all 15 balloons across the task. This means they would have an average adjusted pumps score of zero. These cases were removed from all analyses as this was not a true zero, these six participants actually had high levels of risk-taking.

Inhibitory Control. Children completed the Grass-Snow Stroop task as part of the laboratory session (Carlson & Moses, 2001). After confirming that the child could name the colours of grass and snow, the experimenter introduced a white card and a green card. The child was instructed to point to the green card when the experimenter said snow and the white card when the experimenter said grass. The experimenter demonstrated the task before two practise trials were completed. 16 trials were then presented in a pseudorandom sequence. The outcome of interest was the percentage correct across the trials. A small number of children refused to participate in the task and were excluded from analysis (N = 4).

Results

Preliminary statistics

The means and standard deviations of predictor (BI-consistent maternal report and Stroop Performance) and outcome (BART variables) variables are presented in Table 2.1. A series of t tests were used to examine the effect of gender and ethnicity on all outcome and predictor variables, none were significant (all ps > .05). There were no significant differences between temperament groups for child age, maternal age, family income, ethnicity or number of siblings (p >.05).

Table 2.1

Means and Standard Deviations of Predictor and Outcome Variables

Variable	*M*	*SD*
Percentage correct grass-snow Stroop	59.0	40.4
BI (STSC score)	2.8	1.7
BART performance		
Average adjusted pumps	4.0	1.8
Total number of explosions	4.6	3.1
Total number of points	37.4	14.5

Note: BI = Behavioural Inhibition, STSC = Short Temperament Scale for Children

BI and BART performance

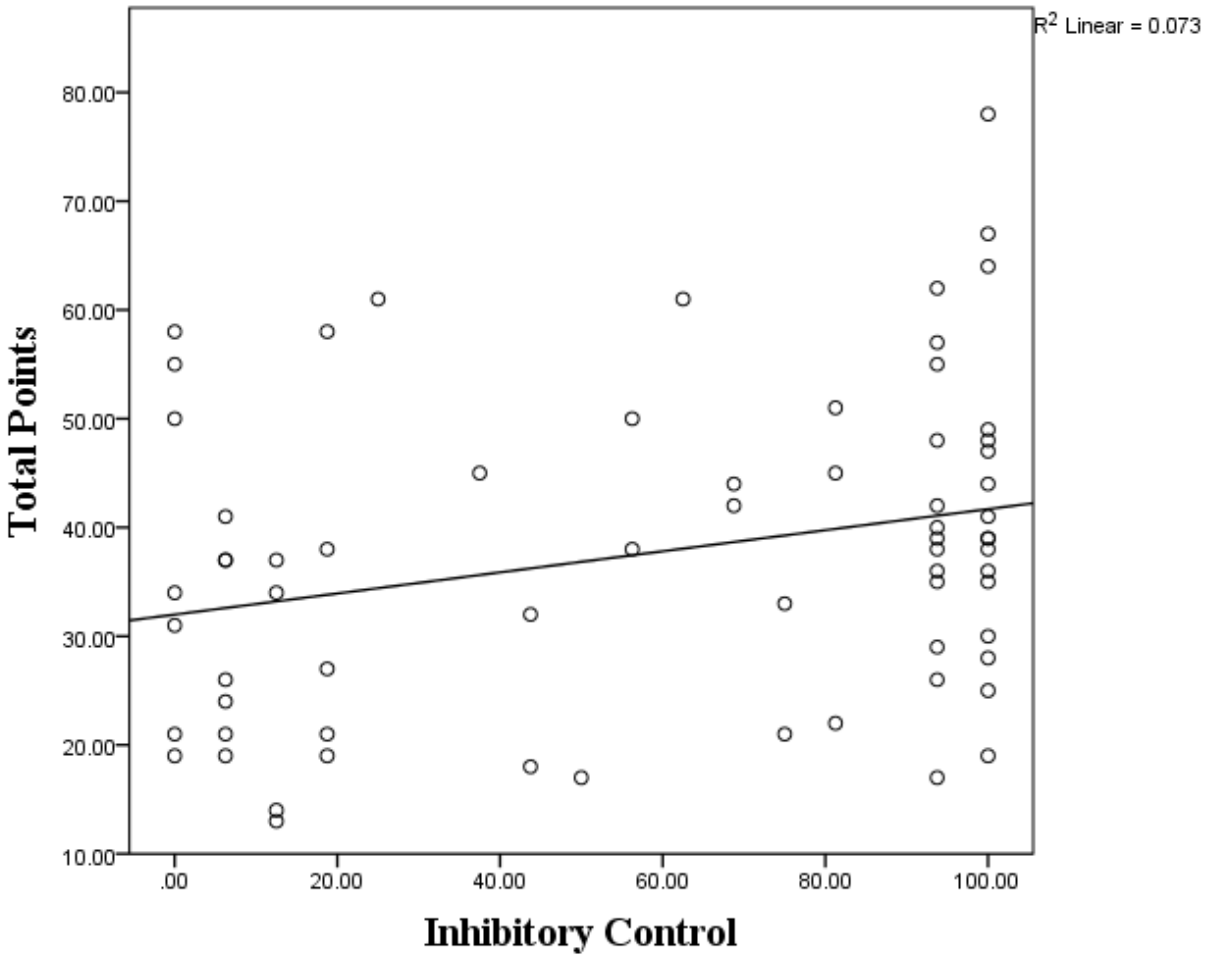
To examine possible group differences for BI and BUI children in their performance on the BART (average adjusted pumps, total points and total number of explosions) a number of *t* tests were performed. No significant effects were found for inhibition level (maternal report) and BART performance (all *p*s > .05).

Inhibitory control and BART performance

There was a significant positive correlation between inhibitory control and total points achieved ($r = .27$, $p = .03$). The more inhibitory control demonstrated, the more reward focused behaviour the participant displayed, by achieving a higher number of total points. No significant relationship was found for average adjusted pumps and inhibitory control, nor explosions and inhibitory control.

Figure 2.1:

Correlation between the executive functioning facet of inhibitory control and BART performance (total points).



BI, Inhibitory control and BART performance

To examine whether inhibitory control interacted with BI group (as determined by maternal report) to predict performance on the BART, three separate multiple regression analyses were conducted, with each BART outcome variable as a dependent variable. BI group, inhibitory control and their interaction were entered into a single model. To aid in interpretation, mean centred predictors were used, and interaction terms were created as the product between the mean-centred continuous measure of inhibitory control and BI group.

Average adjusted pumps The full model was not significant, $F(3, 64) = 0.02$, $p = .99$, and neither BI nor inhibitory control were significantly related to the average adjusted pumps score on the BART (*p*s > .05).

Table 2.2

Multiple Regression for Average Adjusted Pumps

Variable	*B*	*SE B*	*β*
Constant	4.10	0.24	
BI	0.02	0.24	.01
Inhibitory control	0.00	0.01	.03
BI * Inhibitory control	0.00	0.01	.01

Note: $R^2 = .001$, power = 0.053 BI = Behavioural Inhibition

Total point score The full model was not significant, $F(3,64) = 1.70$, $p = .18$. Although BI and the interaction term did not significantly relate to point score, inhibitory control significantly predicted point score on the BART, $\beta = 0.28$, $t(64) = 2.11$, $p = .04$.

Table 2.3

Multiple Regression for Total Point Score

Variable	*B*	*SE B*	*β*
Constant	37.44	1.73	
BI	-0.22	1.82	-.02
Inhibitory control	0.10	0.05	.28*
BI * Inhibitory control	0.00	0.04	.01

Note: R^2 = .074 * p < .05, power = 0.398 BI = Behavioural Inhibition

Total exploded balloons The full model was not significant, $F(3,64) = 0.96$, $p = .42$, and neither BI nor inhibitory control were significantly related to the average adjusted pumps score on the BART (ps > .05).

Table 2.4

Multiple Regression for Total Exploded Balloons

Variable	*B*	*SE B*	*β*
Constant	4.86	0.42	
BI	0.12	0.42	.04
Inhibitory control	-0.02	0.01	-.20
BI * Inhibitory control	0.00	0.01	.04

Note: R^2 = .043, power = 0.234 BI = Behavioural Inhibition

When these analyses were conducted using only a reduced sample of only those participants with consistent BI classifications across parent report and observation (BI = 16, BUI = 36), the pattern of significance was identical, except that Inhibitory Control for total point score was no longer significant ($p = .13$).

Discussion

BI and BUI have both been associated with later psychopathology, one factor that might influence this pathway is risk-taking behaviour. The executive function of inhibitory control has also been linked with risk-taking. The current study was the first to examine whether BI and inhibitory control predict risk-taking behaviour in isolation and whether these factors interact to predict risk-taking.

The findings demonstrated that the higher the level of inhibitory control, the more points a child achieved on the BART. Although average adjusted pumps and number of explosions were not significantly related to inhibitory control, the results for total points scored suggests that children who are high in inhibitory control are taking some risk but they are able to balance this risk with the chance of a reward, the prize at the end. This result is in line with previous research on the role of executive function in risk-taking behaviour. For example, our results are consistent with Suhr and Hammers (2010), who demonstrated that executive functioning is related to performance on the Iowa Gambling Test.

Inconsistent with hypothesis 1, no significant difference between the BI and BUI children was found on the BART. Further, there was no significant interaction between BI group and inhibitory control in predicting BART performance. Although there was no interaction, it remains possible that the mechanism underpinning the relationship between risk-taking and inhibitory control is different for BI and BUI children. Potentially, inhibitory control assists the BI children in suppressing their dominant urge to avoid risk-taking on the BART, allowing them to pump more, saving more points. For the BUI children it is possible that inhibitory control allows them to suppress the urge to continue pumping balloons and instead save their points before the balloon explodes. This suggests that for both temperament extremes, inhibitory control may be acting as a protective factor against risk-taking and risk avoidance behaviour. Further research is needed to examine this possibility. Longitudinal research following participants into childhood and adolescence could examine

the relationship between early BI, inhibitory control and later risk-taking and risk avoidance behaviour. Another possibility is the use of treatment studies that may modify inhibitory control.

The findings support previous research demonstrating that inhibitory control is a possible protective factor for BUI children (Thorell, et al., 2004). However, the results are somewhat contrary to research with BI children. For example, White and colleagues demonstrated that increased inhibitory control led to increased risk for anxiety symptoms in BI children (White, et al., 2011). Taken together with the present findings, this suggests that the additive effect of high inhibitory control and BI as a risk factor for internalising disorders may not manifest through a pathway of increased risk aversion.

The lack of between group difference on risk-taking was surprising, given research showing BI is associated with reticence. It is possible that these children are not generally risk-averse but instead are averse to risk that is associated specifically with their fears. BI children are particularly reticent in social situations (Coplan, et al., 2009). It remains possible that between group differences might be found on a task measuring social risk-taking. A further consideration is whether the 'risk' posed on the BART task was significant enough to trigger risk avoidance behaviour in BI children. Several limitations of the present study should be noted. First, the participants were approximately 4 years of age and it is unclear whether performance on the BART-Y at this age predicts later real-world risk behaviours (Lahat, et al., 2012). Second, only one other study has examined the performance of preschool-aged children on the BART-Y and the role of temperament. Lahat et al. (2012), found that executive functioning had a differing impact on risk-taking for children of different temperaments. However, the Lahat et al. (2012) study focused on the temperament trait of exuberance, which has been demonstrated to be distinct from BI (Putnam & Stifter, 2005).

In conclusion, the results suggest that inhibitory control, but not BI impacts preschool children's risk-taking and reward-focused performance. Further studies could examine whether a risk task better tailored to the fears of BI children, such as social situations, will demonstrate a difference in risk-taking and avoidance between these temperament extremes.

References

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

Barlow, D. H. (2002). *Anxiety and its disorders: The nature and treatment of anxiety and panic (2nd ed.).* Guildford Press.

Brocki, K. C., Nyberg, L., Thorell, L. B., & Bohlin, G. (2007). Early concurrent and longitudinal symptoms of ADHD and ODD: Relations to different types of inhibitory control and working memory. *Journal of Child Psychology and Psychiatry, 48*, 1033-1041.

Carlson, S. M., & Moses, L. J. (2001). Individual differences in inhibitory control and children's theory of mind. *Child Development, 72*, 1032-1053.

Cataldo, M. G., Nobile, M., Lorusso, M. L., Battaglia, M., & Molteni, M. (2005). Impulsivity in depressed children and adolescents: A comparison between behavioral and neuropsychological data. *Psychiatry Research, 136*, 123-133.

Coplan, R. J., DeBow, A., Schneider, B. H., & Graham, A. A. (2009). The social behaviours of inhibited children in and out of preschool. *British Journal of Developmental Psychology, 27*, 891-905.

Degnan, K. A., & Fox, N. A. (2007). Behavioral inhibition and anxiety disorders: Multiple levels of a resilience process. *Development and Psychopathology, 19*, 729-746.

DeVito, E. E., Blackwell, A. D., Kent, L., Ersche, K. D., Clark, L., Salmond, C. H, Dezsery, A. M., & Sahakian, B. J. (2008). The effects of methylphenidate on decision making in attention-deficit/hyperactivity disorder. *Biological Psychiatry, 64*, 636-639.

Fox, N. A., Henderson, H. A., Marshall, P. J., Nichols, K. E., & Ghera, M. M. (2005). Behavioral inhibition: Linking biology and behavior within a developmental framework. *Annual Review of Psychology, 56*, 235-262.

Garcia Coll, C., Kagan, J., & Reznick, J. (1984). Behavioral inhibition in young children. *Child Development, 55*, 1005-1019.

He, J., Degnan, K. A., McDermott, J. M., Henderson, H. A., Hane, A. A., Xu, Q., & Fox, N. A. (2010). Anger and approach motivation in infancy: Relations to early childhood inhibitory control and behavior problems. *Infancy, 15*, 246-269.

Hirshfeld-Becker, D. R., Biederman, J., Henin, A., Faraone, S. V., Micco, J. A., van Grondelle, A., et al. (2007). Clinical outcomes of laboratory-observed preschool behavioral disinhibition at five-year follow-up. *Biological Psychiatry, 62*, 565-572.

Hudson, J. L., Dodd, H. F., & Bovopoulos, N. (2011). Temperament, family environment and anxiety in preschool children. *Journal of Abnormal Child Psychology, 39*, 939-951.

Hudson, J. L., & Dodd, H. F. (2012). Informing early intervention: Preschool predictors of anxiety disorders in middle childhood. *PLoS ONE, 7*.

Humphreys, K. L., & Lee, S. S. (2011). Risk-taking and sensitivity to punishment in children with ADHD, ODD, ADHD+ODD, and controls. *Journal of Psychopathology and Behavioral Assessment, 33*, 299-307.

Kagan, J., Reznick, J., & Gibbons, J. (1989). Inhibited and uninhibited types of children. *Child Development, 60*, 838-845.

Kagan, J., Reznick, J., Snidman, N., Gibbons, J., & Johnson, M. O. (1988). Childhood derivatives of inhibition and lack of inhibition to the unfamiliar. *Child Development, 59*, 1580-1589.

Kerr, M., Lambert, W. W., Stattin, H., & Klackenberg-Larsson, I. (1994). Stability of inhibition in a Swedish longitudinal sample. *Child Development, 65*, 138-146.

Kimonis, E. R., Frick, P. J., Boris, N. W., Smyke, A. T., Cornell, A. H., Farrell, J. M., & Zeanah, C. H. (2006). Callous-unemotional features, behavioral inhibition, and

parenting: Independent predictors of aggression in a high-risk preschool sample. *Journal of Child and Family Studies, 15*, 745-756

Ladouceur, R., Bouchard, C., Rheaume, N., Jacques, C., Ferland, F., Leblond, J., & Walker, M. (2000). Is the SOGS an accurate measure of pathological gambling among children, adolescents and adults? *Journal of Gambling Studies, 16*, 1-24.

Lahat, A., Degnan, K. A., White, L. K., McDermott, J. M., Henderson, H. A., Lejuez, C., & Fox, N. A. (2012). Temperamental exuberance and executive function predict propensity for risk-taking in childhood. *Development and Psychopathology, 24*, 847-856.

Lejuez, C., Aklin, W., Daughters, S., Zvolensky, M., Kahler, C., & Gwadz, M. (2007). Reliability and validity of the youth version of the Balloon Analogue Risk Task (BART-Y) in the asssessment of risk-taking behavior among inner-city adolescents. *Journal of Clinical Child and Adolescent Psychology, 36*, 106-111.

Lejuez, C., Aklin, W. M., Bornovalova, M. A., & Moolchan, E. T. (2005). Differences in risk-taking propensity across inner-city adolescent ever- and never-smokers. *Nicotine & Tobacco Research, 7*, 71-79.

Lejuez, C., Read, J. P., Kahler, C. W., Richards, J. B., Ramsey, S. E., Stuart, G. L., et al. (2002). Evaluation of a behavioral measure of risk-taking: The Balloon Analogue Risk Task (BART). *Journal of Experimental Psychology: Applied, 8*, 75-84.

Lengua, L. J. (2003). Associations among emotionality, self-regulation, adjustment problems, and positive adjustment in middle childhood. *Journal of Applied Developmental Psychology, 24*, 595-618.

Maner, J. K., Richey, J., Cromer, K., Mallott, M., Lejuez, C. W., Joiner, T. E., & Schmidt, N. B. (2007). Dispositional anxiety and risk-avoidant decision-making. *Personality and Individual Differences, 42*, 665-675.

Maner, J. K., & Schmidt, N. B. (2006). The role of risk avoidance in anxiety. *Behavior Therapy, 37*, 181-189.

Putnam, S. P., & Stifter, C. A. (2005). Behavioral approach-inhibition in toddlers: Prediction from infancy, positive and negative affective components, and relations with behavior problems. *Child Development, 76*, 212-226.

Rapee, R., M., Kennedy, S., Ingram, M., Edwards, S., & Sweeney, L. (2005). Prevention and early intervention of anxiety disorders in inhibited preschool children. *Journal of Consulting and Clinical Psychology, 73*, 488-497.

Rothbart, M. K., Ellis, L. K., Rueda, M., & Posner, M. I. (2003). Developing mechanisms of temperamental effortful control. *Journal of Personality, 71*, 1113-1143.

Sanson, A. V., Smart, D. F., Prior, M., Oberklaid, F., & Pedlow, R. (1994). The structure of temperament from age 3 to 7 years: Age, sex, and sociodemographic influences. *Merrill-Palmer Quarterly (1982-)*, 233-252.

Somerville, L. H., & Casey, B. (2010). Developmental neurobiology of cognitive control and motivational systems. *Current Opinion in Neurobiology, 20*, 236-241.

Suhr, J., & Hammers, D. (2010). Who fails the Iowa Gambling Test (IGT)? Personality, neuropsychological, and near-infrared spectroscopy findings in healthy young controls. *Archives of Clinical Neuropsychology, 25*, 293-302.

Thorell, L. B., Bohlin, G., & Rydell, A.-M. (2004). Two types of inhibitory control: Predictive relations to social functioning. *International Journal of Behavioral Development, 28*, 193-203.

Vuontela, V., Carlson, S., Troberg, A.-M., Fontell, T., Simola, P., Saarinen, S., & Aronen, E. T. (2013). Working memory, attention, inhibition, and their relation to adaptive functioning and behavioral/emotional symptoms in school-aged children. *Child Psychiatry and Human Development, 44*, 105-122.

White, L. K., McDermott, J. M., Degnan, K. A., Henderson, H. A., & Fox, N. A. (2011). Behavioral inhibition and anxiety: The moderating roles of inhibitory control and attention shifting. *Journal of Abnormal Child Psychology, 39*, 735-747.

Whiteside, S. P. H., Gryczkowski, M., Ale, C. M., Brown-Jacobsen, A. M., & McCarthy, D.M. (2013). Development of child- and parent-report measures of behavioral avoidance related to childhood anxiety disorders. *Behavior Therapy, 44*, 325-337.

The next chapter presents the paper "Behavioural Inhibition in Childhood and the Development of Adolescent Depression: The Role of Puberty and Risk-Taking". Although a relationship between Behavioural Inhibition (BI) and risk-taking behaviour in early childhood was not found in Chapter 2, this may have been due to the age of the participants. Therefore, the next chapter builds on Chapter 2 by examining how risk-taking behaviour in early adolescence may affect the developmental pathway from BI in early childhood to depression in adolescence. In addition, the study explores how pubertal status may influence this developmental pathway. Chapter 3 introduces the use of a new group of participants, a longitudinal sample of 202 BI and BUI early adolescents initially recruited at 3.5 to 4.5 years of age (Hudson, Dodd, & Bovopoulos, 2011) [3].

[3] Hudson, J. L., Dodd, H. F., & Bovopoulos, N. (2011). Temperament, family environment and anxiety in preschool children. *Journal of Abnormal Child Psychology: An official publication of the International Society for Research in Child and Adolescent Psychopathology, 39*(7), 939-951.

Chapter Three

Behavioural Inhibition in Childhood and the Development of Adolescent Depression: The Role of Puberty and Risk-Taking

Talia M. Morris, Jennifer L. Hudson, & Helen F. Dodd

Abstract

Several risk factors have been identified in the development of depression in adolescence, with current research focusing on how these factors interact to increase risk. The present study examined the relationship between Behavioural Inhibition (BI) in childhood and depression in a sample of 11-12 year-old early adolescents (n = 137), considering the potential moderating role of risk-taking, using the Balloon Analogue Risk Task – Youth version (BART-Y), and pubertal timing, as measured by the Sexual Maturation Scale. Although not consistent across all measures, there was evidence that the relationship between BI in childhood and depressive symptoms in early adolescence was moderated by risk-taking behaviour and pubertal status. The results provide some initial indication that the association between temperament and symptoms of depression may differ according to the risk-taking behaviour demonstrated by the adolescent.

Approximately seventeen percent of people experience a depressive disorder in their lifetime (Kessler et al., 1994). In particular, adolescence is a time of increased vulnerability for the initial development of depression. For example, one study demonstrated a dramatic increase in prevalence rates for major depressive disorder (MDD) from 2% in early adolescence to 15% in middle adolescence (Hankin et al., 1998).

There is substantial evidence that depression in adolescence increases the risk of depression in adulthood. For example, those who experienced an episode of depression during adolescence had an average annual rate of subsequent episodes in young adulthood of 9.0% compared to 3.7% for those who had not experienced depression in adolescence (Lewinsohn, Rohde, Klein, & Seeley, 1999). In fact, even subclinical depressive symptoms in adolescence strongly predict an episode of MDD in adulthood (Pine, Cohen, Cohen, & Brook, 1999) and most adult cases of depression emerge in adolescence in the form of depression or anxiety (Pine, Cohen, Gurley, Brook, & Ma, 1998). Child and adolescent depression has been associated with impairment in school performance, increased risk of suicide behaviours, tobacco use, and abuse of alcohol and other substances (see Birmaher et. al., 1996 for a review). Adolescent depression has a significant and long-term impact, thus it is important to examine the processes that lead to its development. One of the long-term goals of research into MDD is to identify early risk factors that can be targeted for preventative treatment (Lau & Eley, 2010).

It is unlikely that single risk factors are able to explain the development of depression, instead it is the accrual or interaction of multiple factors that lead to an increased vulnerability (Garber, 2006). For example, one of the best fitting predictive models of depression in the current literature found three main interlinking pathways to depression in women (Kendler, Gardner, & Prescott, 2002). However, even this combination of early childhood, adolescence and adulthood factors still only accounted for approximately 50% of the variance in depression (Kendler et al., 2002). Therefore,

continued exploration into the different risk factors for depression across the lifespan is vital.

One factor that has been implicated in the development of depression in adolescents is the temperament trait of behavioural inhibition (BI). BI has been defined as a reserved and shy reaction to unfamiliar adults and peers (Garcia Coll, Kagan, & Reznick, 1984). BI children are less likely to interact with peers at preschool (Rubin, Burgess, & Hastings, 2002), and tend to remain watchful of others (Gersten, 1989). A link between early BI and anxiety disorders is well established and there is some indication BI may also be linked to depression. For example, Caspi and colleagues (1996) found that BI preschool children were more likely to be diagnosed not only with anxiety disorders but also with depression in early adulthood compared to uninhibited children. In another study, BI in childhood was linked to childhood-onset and recurrent depression (Jaffee et al., 2002). Concurrent BI and depression has also been demonstrated, with high levels of child and parent reported BI linked to high levels of depression in young adolescents (Muris, Meesters, & Spinder, 2003). Based on this emerging body of research, Hirshfeld-Becker and colleagues (2008) suggested that further follow up of children initially assessed for BI in childhood could advance our understanding of the development of later disorders such as depression.

Although BI has been linked to later depression, not all BI children develop depression, nor does disinhibition protect against the development of depression. For example, in Caspi and colleagues' study (1996), although 53% of BI children were found to have a diagnosis of either depression or anxiety at age 21, 47% did not. Thus, it is important to examine what other factors may increase the risk for depression in BI children and what factors may be protective.

Due to the significant increase in depressive symptoms during adolescence, a body of research has focused on examining why rates of depression increase at this point in the lifespan. A number of important changes occur during this period, including pubertal and

behavioural changes. Firstly, adolescence is a time of increased risk-taking and reward-seeking behaviour. Adolescents are more likely to engage in binge drinking, smoke cigarettes, or engage in violent and criminal behaviour than younger or older individuals (Steinberg, 2008). The dual systems model of risk-taking in adolescence posits that this vulnerability to risk-taking is a result of increased reward-seeking behaviour and low impulse control. This is reflected in proposed neurological changes that occur around the time of puberty. Initially, it is suggested that a rapid and dramatic increase in dopaminergic activity in the socioemotional system of the brain directs an increase in reward-seeking behaviour (Steinberg, 2008). Importantly however, this increase takes place before the cognitive control system, responsible for advanced self-regulation and impulse control, has completely developed. Therefore, it is proposed that a period of increased vulnerability occurs in middle adolescence where adolescents are stimulated to seek rewards but do not yet possess the self-regulation to effectively assess the risks involved (Steinberg, 2008). A neurological longitudinal study demonstrated this mismatch in the timing of structural maturation of the two systems across childhood, adolescence, and young adulthood (Mills, Goddings, Clasen, Giedd, & Blakemore, 2014). This mismatch has also been demonstrated in behavioural studies with an increase in risk-taking behaviour over the adolescent period in an inverted U function, with a peak in middle adolescence, followed by a decline in late adolescence (e.g. Steinberg, 2008). Of interest to the current study is how changes in risk-taking behaviour may lead to an increased risk for the development of depression.

Those at higher risk for depression, for example children who have a parent who has experienced depression, show a decreased responsiveness to reward and increased response to negative feedback from risk-taking, for example the loss of a reward (e.g. Forbes, Shaw & Dahl, 2007). For instance, Gotlib and colleagues found that individuals with familial risk for depression showed less neural activation when anticipating rewards, and greater activation in response to punishment than those without familial risk (Gotlib et al., 2010).

These differences in neural response occurred despite the high-risk group never having developed depressive symptoms. In longitudinal studies, differences in reward processing have also been shown to predict the onset of depression in adolescents. In a community sample, a blunted neural response to reward was shown to predict depressive scores 18 months later in adolescent females (Nelson, Perlman, Klein, Kotov, & Hajcak, 2016). Also, Forbes et al., (2007) examined whether differences in reward processing predicted subsequent depressive symptoms in early adolescence. It was demonstrated that early adolescents who had difficulty distinguishing between low and high reward options on the experimental paradigm were more likely to experience depressive symptoms in a follow up one year later (Forbes et al., 2007). Therefore, changes in risk-taking and reward-seeking behaviour have also been demonstrated in populations at an increased risk for depressive symptoms.

A body of research has examined neural responses to reward in BI adolescents. Compared to behaviourally uninhibited (BUI) adolescents, BI adolescents show increased striatal response when anticipating rewards (Guyer et al., 2006). Striatal areas, such as the nucleus accumbens, are implicated in the process of coding the properties of reward and risk cues (e.g. Knutson, Adams, Fong, & Hommer, 2001). Other studies have demonstrated that BI adolescents show the opposite pattern of activation in the striatum to BUI adolescents, with BI adolescents having a greater activation in response to the omission of a reward, and diminished activation to the collection of a reward (Helfinstein et al., 2011). Furthermore, a meta-analysis of neural imagining studies demonstrated that BI is associated with increased activity in regions associated with threat and reward processing (Clauss, Avery, & Blackford, 2015). It is possible that this different pattern of risk sensitivity and decreased response to reward may, in part, explain why BI children are at increased risk for later depression. This possibility has not yet been explored using a reward-seeking/risk-taking behavioural paradigm.

The dramatic increase in depression rates in adolescence also coincides with the onset of puberty. In particular, the timing of puberty is thought to affect the incidence of depression in adolescents. Early pubertal timing has been shown to predict increases in depressive symptoms over early adolescence for both boys and girls (Mendle, Harden, Brooks-Gunn, & Graber, 2010). This is further supported by a recent meta-analysis which suggested that early puberty (particularly in girls) may increase the risk of depression (Galvao et al., 2014). Recent work has extended this research by examining the interaction between pubertal timing and pre-existing vulnerabilities to depression. For example, Hamilton and colleagues (2014) looked at the influence of pubertal timing on depressive symptoms in adolescents with differing cognitive styles and levels of emotional clarity. Emotional clarity is defined as the ability to identify and understand one's own emotions. Early pubertal timing was linked to heightened depressive symptoms for adolescent boys and girls with a negative cognitive style and for adolescent girls with poor emotional clarity. These findings suggest that early pubertal timing may increase the risk for depression in adolescents with pre-existing vulnerability to depression. To our knowledge, no study has examined whether pubertal timing may increase the risk for depression in adolescents classified as BI in childhood. Following previous research on other pre-existing vulnerabilities, it is expected that BI adolescents who also experience early pubertal timing will be at increased risk for depressive symptoms, compared to BI adolescents with later pubertal timing and BUI adolescents.

Although the relationship between BI and adolescent depression has been examined, most studies have looked at this relationship at a single time point (e.g. Muris et al., 2003). Jaffee et al. (2002) examined the BI-depression relationship longitudinally but they did not attend to moderating factors that may have led to the rise in depressive symptoms during early adolescence for those who were inhibited in childhood.

The present study examined the relationship between BI in childhood and depression in 11-12 year-old early adolescents, considering the potential moderating role of risk-taking and pubertal timing. Overall it is expected that early adolescents identified as BI in childhood will have more depressive symptoms compared to those identified initially as BUI. Further, it was hypothesised that lower risk-taking would be associated with increased symptoms of depression for both BI and BUI early adolescents. It was further anticipated that pubertal status and risk-taking would interact with early childhood BI to predict depressive symptoms in early adolescence such that they have a more deleterious effect on BI individuals compared to BUI individuals.

Method

Participants

Participants were 102 BI and 100 BUI children who were initially recruited for an ongoing longitudinal study via local preschools and advertisements (see Hudson, Dodd & Bovopoulos, 2011 for full details). BI was initially assessed at baseline using the mothers' report on the Approach Scale of the Short Temperament Scale for Children (Sanson, Prior, Garino, Oberklaid, & Sewell, 1987; STSC), which is described below. Children scoring 1 standard deviation above or below the normative mean on the STSC were invited to take part in the full study. These participants were classified as BI or BUI respectively. Baseline assessments were performed when the children were aged approximately 4 years (mean age: 48.2 months, SD = 4.26; 50% male). Based on the baseline observation of BI, 92 participants from the original sample were classified as inhibited and 110 participants as uninhibited. For 74% of participants, the classifications agreed with the original parent-report groups (71 BI and 79 BUI).

All participants who could be contacted were invited to take part in a follow-up eight years after the baseline assessment. Of the original sample, 56 BI and 81 BUI early adolescents, 137 in total, took part in this follow up. As several participants were no longer

able to attend a research session on campus due to a change in circumstances, 112 of these early adolescents took part in the full laboratory session (68 BUI and 44 BI). The remaining participants completed only a diagnostic interview over the phone and questionnaires online. Participants were aged approximately 12 years (mean age: 140 months, sd = 3.01), with 55 female and 57 male participants. Most participants (64 %) identified as being Oceanic, 21% as European, and 9% as Asian with the remainder being American, African or Middle Eastern. For income, 56% of the sample were from middle to high income families.

There were no significant differences between those who participated at the 8-year follow up and those that did not on child gender, maternal anxiety, maternal education, ethnicity or family income as measured at baseline ($p > .05$). Participants who did not take part were, however, more likely to have been classified as BI, χ^2 (1, $N = 202$) = 15.76, $p < .001$, and more likely to have met criteria for an anxiety disorder at baseline, χ^2 (1, $N = 202$) = 6.77, $p = .009$.

Measures

Baseline Measures. Below is a brief summary of the relevant baseline measures. Full details of these measures and the procedure at baseline can be found in Hudson et al., (2011).

Maternal-report of BI. As described above, BI was assessed at baseline using the approach scale of the STSC. Seven items make up the Approach Scale, for example 'My child is shy when first meeting new children'. High scores on the approach scale indicate lack of approach. The STSC has adequate validity, good internal consistency and reliability (Sanson et al., 1987). The internal consistency for the approach scale in the present sample at baseline was $\alpha = .92$. The STSC has been used previously to classify children as BI (Rapee, Kennedy, Ingram, Edwards, & Sweeney, 2005).

Observed BI. BI was also assessed at baseline using laboratory tasks similar to those used by Kagan and colleagues (1984). Children were observed responding to a novel room,

novel toy, same-sex unfamiliar peer, and a masked experimenter. To determine inhibition status, the following behaviours were examined: time spent proximal to mother; time spent staring at the peer; time spent talking; number of approaches to the stranger; and number of approaches to the peer. To be defined as BI by observation, participants needed to score above a predetermined cutoff on three or more of these five behaviours (Rapee et al., 2005).

Follow-Up Measures. The following measures were conducted at 8-year follow-up.

Pubertal status. As part of the research session, participants completed Tanner's Sexual Maturation Scale (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987). This task uses drawings to depict five stages of pubertal development, either female breast and pubic hair or male genitalia and pubic hair. Participants were asked to select the drawing that corresponds to their stage of pubertal development. The SMS is often used when a physical examination by a qualified physician is impractical or would be too confronting for a participant or their parents. Previous studies have reported a high level of agreement between self-report using the SMS and physician examination within one pubertal stage (e.g. 85% agreement, Schmitz et al., 2004).

Risk-taking. Participants completed the Balloon Analogue Risk Task, youth version (BART-Y; Lejuez et al., 2007). The BART-Y has been shown to significantly predict risk-taking behaviour and sensation seeking in adolescents (Lejuez et al., 2007). The BART-Y is a computer task in which participants pump up a simulated balloon displayed on the screen. Participants are told that each pump of the balloon yields a point, which can be added to an overall prize total, displayed on a 'prize meter'. The balloon can explode at any pump point and if it explodes, reward points for that round are lost. For each pump of the balloon, participants are faced with the choice of saving their points for the round so far or continuing to pump up the balloon, which may yield more points but also comes with the risk of the balloon exploding and them losing their points for that round.

Before the task began, participants were told they will receive a prize and that the size of the prize was dependent on the total points received. During the task, the participants could see their points total tallied on a prize meter. As they received points, the prize meter was coloured in the appropriate amount, making it easy for the participants to see how many points they had accumulated thus far. The task included 30 trials/balloons with 30 being the maximum numbers of pumps before the balloon exploded and two the minimum. The maximum number of pumps was randomised across the task, with all participants receiving the same randomised order of balloons. Consistent with previous studies (e.g. Lejuez et al., 2007), the average number of pumps across balloons that did not explode was used as a dependent variable. This adjusted value is used as it includes only balloons for which the participant's behaviour was not constrained by the explosion point of the balloon. To complement this and ensure that excessive risk-taking resulting in a high proportion of exploded balloons was captured, total explosions across the task was also included as a second risk-taking measure (Morrongiello, Kane, McArthur, & Bell, 2012). Finally, the total number of points scored by the participants was included as a measure of reward focused behaviour. Participants more focused on the reward at the end of the task would be expected to aim for a higher number of points, balancing the risk and reward.

Depressive symptoms. Participants completed two measures of symptoms of depression.

Short Mood and Feelings Questionnaire. Prior to attending the 8-year follow up laboratory session, participants and their mothers completed the Short Mood and Feelings Questionnaire (SMFQ: Angold et al., 1995) as part of an online questionnaire battery. The SMFQ consists of 13 items that assess depressive symptoms occurring over the previous two weeks on a 3-point scale. The SMFQ has good internal consistency ($\alpha = 0.85$, Angold et al., 1995), and the test-retest reliability is 0.66 over a two-week period (Costello & Angold, 1988). The SMFQ correlates moderately with other child-reported measures of

depressive symptoms (0.67 with the Children's Depressive Inventory; Angold et al., 1995). The internal consistency in our sample was high, $\alpha = .87$ for the child report.

Kiddie schedule for affective disorders and schizophrenia. The Depression Rating Scales of the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS-DRS/MRS; Kaufman et al., 1997) was used to assess symptoms of depression. Early adolescents and their mothers were interviewed independently during the laboratory session. The KSADS is a semi-structured interview designed to assess for mood disorders in children and adolescents. It is one of the most frequently used diagnostic interviews with youths and has demonstrated strong evidence of reliability and validity (Kaufman et al. 1997; Klein, Dougherty & Olino 2005). The interviewer gave the participant a score of 0-2 for each screening question, as per the KSADS instructions. In this study, inter rater agreement on each screening question across the KSADS-DRS, was ICC (2, 1) = .81. The KSADS can be used to diagnose depression in participants, however as no participants met the full criteria for major depressive disorder these results are not reported. This low diagnosis rate is not unusual given the participants' age. Instead, depressive symptom presence was examined, using the child and mother reports. Participants who had at least one depressive symptom as reported on the KSADS, by the child or mother, were coded as 'symptom present', while those not meeting criteria were coded as 'symptom absent'. This resulted in two KSADS groups.

Procedure

Macquarie University Human Ethics Committee approved the procedures for the study including the consent procedures. Children who met the criteria following the initial STSC screening were invited to attend a 2-hour research session on campus at Macquarie University both at baseline and at follow up eight years later. For both assessments, written consent was provided by the mothers for both their and their child's participation. Following a brief description of the research, children also provided verbal assent/consent. Written

questionnaires were completed prior to attending the research session by the mother only for the baseline session and by both the mother and child for the follow up session, including the SMFQ. At both sessions the family were reimbursed $50 and children were given a small age appropriate gift. At the follow up session both mother and child completed the KSADS interview separately with a trained administrator. Prior to the KSADS, early adolescents completed the SMS and BART-Y task and received another small gift as their prize. Additional observed tasks and questionnaires were completed at baseline and at the follow up which are not presented here.

Results

Analysis Plan

There was a small amount of missing data (<5%) due to issues with the computer based tasks, and participants not completing their questionnaires in full. Due to missing data, multiple imputation was applied to create 20 datasets with complete data for the follow up variables: KSADS, SMFQ child and mother report, puberty and the three BART-Y variables (Sinharay, Stern, & Russell, 2001). All the baseline variables described in the methods, including demographics, were used as independent variables in the multiple imputation. The results reported are for the pooled outcomes across the 20 imputed datasets unless noted. Also, as scores on the child and mother report SMFQ were negatively skewed, they were successfully transformed using a log +1 transformation.

Three outcome variables were used to capture depression: KSADS group; child report SMFQ; and mother report SMFQ. Initial analyses examined the direct association between BI, risk-taking and puberty on depression. Next, a series of regression analyses were conducted to examine the primary research aim of whether risk-taking and pubertal status moderate the BI-depression relationship. For each regression BI was entered as step 1, with the other IVs (pubertal status (SMS) & BART-Y: explosions, total points or average adjusted points) and interaction terms (BI*SMS, BI*BART-Y: explosions, total points or

average adjusted points) entered as step 2. Interaction terms were calculated for the linear regressions by multiplying mean-centred variables. Each regression analysis was run three times to account for the three BART-Y outcome IVs. The R square results are based on the original data as no pooled estimation of R square is produced.

For all analyses, maternal report was used for the BI variable. Based on the child's behaviour during the baseline laboratory assessment of BI, 39 of the original sample were classified as inhibited and 66 as uninhibited. For 77% of participants this classification agreed with the maternal report of BI. All analyses were conducted again using the subsample of participants whose maternal-report classification was consistent with their laboratory-based classification. If differences were found in the pattern of effects, these are reported.

Descriptive analyses

A series of *t* tests were used to examine the effect of gender, ethnicity, child age, maternal age, family income, or number of siblings on the three BART-Y outcome variables and Tanner's SMS. None were significant (all *p*'s > .05). There were no significant differences between temperament groups or depressive symptoms for child age, gender, maternal age, family income, or number of siblings ($p > .05$). Significant differences were found for ethnicity and temperament, χ^2 (2) = 11.48, $p = .01$, with greater numbers of children of Asian ethnicity in the BI group. As ethnicity was not related to any of the outcome variables ($p > .05$) it was not controlled for in future analyses.

Pooled means and standard deviations for all variables are reported in table 3.1. Pooled means were calculated in SPSS (Version 22, IBM) and pooled standard deviations were calculated using the following formula: where SD is standard deviation, p is pooled, n is number of participants and 1, 2 and k are the pooled dataset number.

$$SD_p = \sqrt{\frac{(n_1-1)SD_1^2+(n_2-1)SD_2^2+\ldots+(n_k-1)SD_k^2}{n_1+n_2+\ldots+(n_k-k)}}$$

Table 3.1

Means and standard deviations for predictor and outcome variables at 8-year follow up for parent-reported behaviourally inhibited (BI) and uninhibited (BUI) groups.

	BI N = 102		BUI N = 100	
	Mean	SD	Mean	SD
SMS	2.36	0.94	2.33	0.84
BART-Y: Total points	102.60	23.93	102.72	21.01
BART-Y: Explosions	11.79	3.40	11.94	3.49
BART-Y: Average adjusted pumps	5.83	1.66	5.93	1.65
KSADS	0.28	0.45	0.23	0.42
SMFQ: Child report	0.41	0.31	0.41	0.31
SMFQ: Mother report	0.36	0.31	0.35	0.34

Note: Pooled MI dataset BI = Behavioural inhibition, SMS = Sexual maturation scale, BART-Y = Balloon analogue risk task, KSADS = Kiddie schedule for affective disorders and schizophrenia, SMFQ = Short mood and feelings questionnaire.

BI and Depression

T-tests were used to observe whether BI measured in early childhood predicted differences in SMFQ depression symptoms in early adolescence. Neither maternal report SMFQ $t(455) = -0.24$, $p = .81$, nor child report SMFQ $t(257) = -0.04$, $p = .97$ were significantly different between BI and BUI participants. A chi-square analysis was also not significant for the KSADS groups, χ^2 (1) = 0.98, $p = .32$.

Risk-taking and Depression

Point biserial correlations were conducted to examine whether the three BART-Y risk-taking variables were related to the KSADS groups. None were significant (Total points: $r_{pb}(200) = .03$, p = .784, Explosions: $r_{pb}(200) = .01$, p = .894, Average adjusted pumps: $r_{pb}(200) = .02$, p = .771). Pearson correlations between the three BART-Y risk variables and child report SMFQ were also not significant (Total points: $r(200) = .30$, p = .759. Explosions: $r(200) = -.01$, p = .937. Average adjusted pumps: $r(200) = .02$, p = .848). Finally, Pearson correlations between the three BART-Y risk variables and mother report SMFQ were also not significant (Total points: $r(200) = -.002$, p = .986. Explosions: $r(200) = .01$, p = .950. Average adjusted pumps: $r(200) = .01$, p = .899).

Pubertal Status and Depression

Pearson correlations explored whether there was a relationship between pubertal status and depressive symptoms. None of the correlations were significant: maternal report SMFQ ($r(200) = .03$, p = .75), child report SMFQ ($r(200) = -.05$, p = .63), nor KSADS ($r(200) = .07$, p = .40).

BI, Risk-taking, Pubertal Status and Depressive Symptoms

KSADS. Logistic regression was used to examine the impact of several factors on the likelihood that participants would report a depressive symptom on the KSADS. The full model containing all predictor variables (BI, BART-Y: total points, explosions or average adjusted pumps) and interactions (BI*pubertal status and BI*BART-Y) was significant for total points, χ^2 (5) = 10.02, $p = .040$, and average adjusted pumps, χ^2 (5) = 10.49, $p = .047$. It

was non-significant for explosions, χ^2 (5) = 10.09, p = .073. The chi-square results are based on the original data set as no pooled data output is produced for this test.

Total points. Overall the model as a whole explained between 11.2% (Cox and Snell R square) and 17.4% (Nagelkerke R square) of the variance in depressive symptom presence, see Table 3.2. None of the independent variables nor interactions made a unique statistically significant contribution to the model.

Explosions. Overall the model as a whole explained between 10.2% (Cox and Snell R square) and 15.8% (Nagelkerke R square) of the variance in depressive symptom presence. BI, explosions, SMS, and BI*pubertal status were not significant predictors within the model. The interaction between BI and explosions made a unique statistically significant contribution to the model as seen in Table 3.3 This suggests that the relationship between explosions and depressive symptom presence differs for BI and BUI participants. Within-group point bi-serial correlations revealed that for BI children there was a positive non-significant correlation between the number of explosions and KSADS group r(100) = .20, p = .08, such that there was a trend for increased risk-taking being associated with increased chance of depressive symptom presence. For BUI children the correlation was in the negative direction r(98) = -.19, p = .07, a trend of decreased risk-taking being associated with an increased chance of depressive symptom presence.

Table 3.2

Logistic regression predicting likelihood of reporting symptom presence on the KSADS from BI, SMS and total points.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	-1.21	0.27	0.30*	.00
BI	0.24	0.38	1.28	.52
Step 2				
Constant	-2.24	1.82	0.11	.22
BI	0.57	2.37	1.77	.81
SMS (pubertal status)	0.53	0.40	1.69	.19
Total Points (BART-Y)	-0.00	0.02	1.00	.87
BI * SMS	-0.56	0.53	0.57	.30
BI * Total Points	0.01	0.02	1.01	.62

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .11$, *p < 0.05, BI = Behavioural Inhibition, BART-Y = Balloon Analogue Risk Task - Youth

Average adjusted pumps. Overall the model as a whole explained between 11.7% (Cox and Snell R square) and 18.1% (Nagelkerke R square) of the variance in depressive symptom presence. BI, average adjusted pumps, SMS, and BI*pubertal status were not significant predictors within the model. The interaction between BI and average adjusted pumps made a unique statistically significant contribution to the model as seen in Table 3.4. Within group point bi-serial correlations demonstrated the same pattern of results as those for Explosions. For BI children there was a positive correlation between the number of average adjusted pumps and KSADS group $r(100) = .19$, $p = .10$, such that there was a trend for increased risk being associated with a greater chance of reporting a depressive symptom. For BUI children the correlation was in the negative direction $r(98) = -.16$, $p = .17$, a trend of decreased risk-taking being associated with an increased chance of depressive symptom presence.

When these analyses were conducted using the reduced sample of only participants with consistent BI classifications across parent-report and observation, the pattern of results remained the same for all three logistic regression analyses (see Supplementary Tables 1-9; Appendix B).

Table 3.3

Logistic regression predicting likelihood of reporting symptom presence on the KSADS from BI, SMS and explosions.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	-1.21	0.27	0.30*	.00
BI	0.24	0.38	1.28	.52
Step 2				
Constant	-0.89	1.58	0.41	.57
BI	-1.69	2.07	0.19	.42
SMS (pubertal status)	0.44	0.41	1.56	.29
Explosions (BART-Y)	-0.12	0.08	0.89	.13
BI * SMS	-0.50	0.56	0.60	.37
BI * Explosions	0.27	0.12	1.30*	.02

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .10$, *p < 0.05, BI = Behavioural Inhibition, BART-Y = Balloon Analogue Risk Task - Youth

Table 3.4

Logistic regression predicting likelihood of reporting symptom presence on the KSADS from BI, SMS and average adjusted points.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	-1.21	0.27	0.30*	.00
BI	0.24	0.38	1.28	.52
Step 2				
Constant	-1.18	1.50	0.31	.44
BI	-1.36	2.00	0.26	.50
SMS (pubertal status)	0.46	0.41	1.59	.26
Average Adjusted Pumps (BART-Y)	-0.20	0.21	0.82	.24
BI * SMS	-0.49	0.55	0.62	.38
BI * Average Adjusted Pumps	0.47	0.24	1.60*	.05

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .12$,*$p < 0.05$, BI = Behavioural Inhibition, BART-Y = Balloon Analogue Risk Task - Youth

SMFQ. Linear regressions were conducted to examine the relationship between BI, risk-taking, pubertal status and depressive symptoms on the SMFQ for child and mother report separately. All reported R^2 and F values are using the original data set as no pooled output was produced.

Total points. In combination all five predictor variables explained 10.5% of the variance in SMFQ child report symptom level, $R^2 = .105$, adjusted $R^2 = .048$, $F(5, 79) = 1.86$, $p = .112$. BI, SMS, total points and BI*total points did not significantly contribute to the model. The interaction term BI*SMS significantly predicted symptom level on the SMFQ child report in the model as seen in Table 3.5.

Within-group correlations were conducted to further explore the relationship between BI and pubertal status as measured using the SMS. For BI children, a non-significant relationship was demonstrated between pubertal status and depressive symptom report on child-reported SMFQ $r(98) = .08$, $p = .49$. For BUI children a negative non-significant relationship was also reported, but in the opposite direction to BI children $r(100) = -.16$, $p = .24$. Even though the correlations were non-significant, the significant interaction term demonstrated that the relationship between pubertal status and depressive symptoms differs for BI and BUI children.

Explosions. In combination all five predictor variables explained 10.7% of the variance in SMFQ symptom level, $R^2 = .107$, adjusted $R^2 = .050$, $F(5, 79) = 1.88$, $p = .107$. BI, SMS, explosions and BI*explosions did not significantly contribute to the model. The interaction term BI*SMS significantly predicted symptom level on the SMFQ child report in the model as seen in Table 3.6. Within group correlations between SMS and SMFQ symptom level are stated above.

Average adjusted points. In combination all five predictor variables explained 10.5% of the variance in SMFQ symptom level, $R^2 = .105$, adjusted $R^2 = .048$, $F(5, 79) = 1.85$, $p = .113$. BI, SMS, average adjusted points and BI*average adjusted points did not

significantly contribute to the model. The interaction term BI*SMS significantly predicted symptom level on the SMFQ child report in the model as seen in Table 3.7. Within group correlations between SMS and SMFQ symptom level are stated above.

When these analyses were conducted using the reduced sample of only participants with consistent BI classifications across parent-report and observation, the interaction term BI*SMS is no longer significant for all three regression analyses, however the pattern of results remained the same ($p > .05$).

Mother report SMFQ. Tables for the three regressions using the mother report SMFQ results are included in Appendix B (Supplementary Tables 10-12). No predictors were found to significantly predict symptom level on the SMFQ mother report across all three regression analyses.

Table 3.5

Linear regression predicting symptom level on the SMFQ child report with BI, SMS and total points.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.40	0.04	9.77*	.00
BI	-0.04	0.06	-0.64	.52
Step 2				
Constant	0.56	0.25	2.24*	.03
BI	-0.04	0.06	-0.70	.48
SMS (pubertal status)	-0.04	0.04	-0.90	.37
Total Points (BART-Y)	-0.00	0.00	-0.33	.74
BI * SMS	-0.08	0.04	-2.12*	.03
BI * Total Points	0.00	0.00	-0.11	.92

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .11$, *p < 0.05, BI = Behavioural Inhibition, BART-Y = Balloon Analogue Risk Task - Youth

Table 3.6

Linear regression predicting symptom level on the SMFQ child report with BI, SMS and explosions.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.40	0.04	9.77*	.00
BI	-0.04	0.06	-0.64	.52
Step 2				
Constant	0.53	0.15	3.47*	.00
BI	-0.05	0.06	-0.72	.47
SMS (pubertal status)	-0.03	0.04	-0.78	.44
Explosions (BART-Y)	-0.00	0.01	-0.46	.65
BI * SMS	-0.08	-0.04	-2.13*	.03
BI * Explosions	-0.00	0.01	-0.44	.66

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .11$, *p < 0.05, BI = Behavioural Inhibition, BART-Y = Balloon Analogue Risk Task - Youth

Table 3.7

Linear regression predicting symptom level on the SMFQ child report with BI, SMS and average adjusted pumps

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.40	0.04	9.77*	.00
BI	-0.04	0.06	-0.64	.52
Step 2				
Constant	0.55	0.17	3.32*	.00
BI	-0.05	0.06	-0.72	.47
SMS (pubertal status)	-0.04	0.04	-0.86	.39
Average Adjusted Pumps (BART-Y)	-0.01	0.02	-0.56	.58
BI * SMS	-0.08	0.04	-2.14*	.03
BI * Average Adjusted Pumps	-0.00	0.02	-0.21	.84

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .11$,*$p < 0.05$, BI = Behavioural Inhibition, BART-Y = Balloon Analogue Risk Task - Youth

Discussion

Although an increase in diagnosed depression from childhood to adolescence is widely reported, the mechanisms by which this increase occurs are not fully understood. The primary aims of the current study were to examine whether BI classified in childhood was associated with symptoms of depression in early adolescence and whether this effect was moderated by risk-taking and pubertal status. In contrast to our hypothesis, BI participants did not show more symptoms of depression overall on the KSADS clinician report, parent report or child report measure of depressive symptoms. However, there was some evidence that the relationship between BI and depression was moderated by risk-taking and by pubertal status. These interactions were not consistent across all measures of risk-taking and depression. For example, the interaction between BI and risk-taking was significant when risk-taking was measured using the BART-Y variables of explosions and average adjusted pumps, but not for total points, and not when depression was measured using child or maternal report (SMFQ). Similarly for pubertal status, there was a significant interaction between BI and pubertal status when depression was measured using child report but not the KSADS or maternal report.

Although not consistent across measurements of depression and risk-taking, the pattern of results suggest that the relationship between BI and clinician rated depression differed depending on the adolescent's level of risk-taking on the BART-Y. The pattern of results for the BUI group was consistent with previous research (e.g. Nelson et al., 2016; Smoski et al., 2008); there was a non-significant negative relationship between risk-taking and symptoms of depression such that reduced risk-taking/reward-seeking was associated with an increased risk of symptoms of depression. In contrast, for the BI group, higher risk-taking was associated with an increased risk of symptoms of depression. As these bivariate correlations did not reach significance, possibly due to sample size, interpretation of the interaction needs to be tentative. Nonetheless, these results conflict with previous research,

which shows reduced risk-taking is associated with increased depression (Gotlib et al., 2010). If replicated, the results suggest that the association between temperament and symptoms of depression may differ according to the risk-taking behaviour demonstrated by the adolescent.

One possible explanation of the differential impact of risk-taking on the relationship between BI and depression is that risk-taking behaviour has the potential for rewards as well as negative consequences. For an inhibited child, negative consequences may be more impactful than for an uninhibited child. For example, in a social situation increased risk-taking behaviour may lead to peer victimisation or rejection, a known risk factor for depression (Nolan, Flynn, & Garber, 2003).

Although a significant moderating effect was shown for two of the BART variables (explosions and average adjusted pumps), this effect was not demonstrated for total points. Explosions and average adjusted pumps have been used previously as outcome measures of risk-taking on the BART-Y (Lejuez et al., 2007; Morrongiello et al., 2012). On the other hand, total points has been conceptualised as a measure of reward focused risk-taking (Morris, Hudson, & Dodd, 2014; Chapter 2). The inconsistent findings across the BART-Y variables suggests that the moderating effect on the relationship between BI and depressive symptoms may be related to risk-taking alone, rather than reward-focused risk-taking.

The finding that pubertal status may moderate the relationship between BI and depressive symptoms is an interesting addition to prior research. Pubertal status has been previously linked with depressive symptoms; an early or late pubertal status in comparison to their peers' pubertal status is associated with elevated levels of depressed mood in both boys and girls (Natsuaki, Biehl, & Ge, 2009). Although there was no main effect of pubertal status on depressive symptoms in the current study, there was an interaction effect on child-reported depressive symptoms only. Pubertal status moderated the relationship between BI in early childhood and depressive symptoms in early adolescence. However, due to the

small size of the correlations, conclusive statements cannot be made about the direction of these relationships, just that the relationship between pubertal status and symptoms of depression differed for adolescents identified as either BI or BUI in early childhood. The possibility that pubertal status may play a role in the pathway from BI to later depression is an important avenue for future research.

The prospect of pubertal status and risk-taking behaviour impacting the risk for depression in BI children provides a potential insight into the mechanisms of this pathway. It is feasible that the transition from late childhood to early adolescence may be more stressful for BI children than BUI children (Lahat, Hong, & Fox, 2011). Biological factors such as puberty begin to play a role, while peer groups and social interactions take on a more important function in the children's lives (Rubin, Coplan, & Bowker, 2009). Furthermore, previous research has found that children who become more inhibited during early adolescence report increasing depressive symptoms (Buck & Dix, 2012). The authors suggested that increasing inhibition as an adaptation to the stresses of adolescence is a risk factor for increases in depressive symptoms (Buck & Dix, 2012). It is possible that risk factors for the development of depressive symptoms such as pubertal status and risk-taking behaviour impact early adolescents who are BI or BUI differently. This is in line with multivariate theories of depression (e.g. Kendler et al., 2002).

The current findings provide a number of important directions for future research. Due to the salience of social risk-taking for adolescents, further exploring the area of reduction in social risk-taking and its links to later depression is an important area for future research. Although the BART has been shown to correlate with risk-taking behaviour such as smoking in adolescents (Lejuez, Aklin, Bornovalova, & Moolchan, 2005), to our knowledge, it has not been examined in depression before. In relation to the current findings of no significant association between risk-taking and depressive symptoms, it is possible that the BART-Y does not capture the reduction in risk-taking behaviour due to the type of

reward used. Thus, it is important to consider how applicable the risk and reward presented in the BART-Y is to real-life situations. In particular, real-life situations that are salient to those at risk for depression, such as social risk-taking.

Furthermore, the BART-Y is a measure that captures both reward-seeking and risk-taking behaviour. Participants seek the reward of more points (and a larger prize), while also balancing the risk of the balloon bursting. This is particularly prudent when examining relationships between the BART-Y results and depressive symptoms as risk-taking and reward may play different roles. For example, those with familial risk for depression demonstrated less neural activation when anticipating rewards, and greater activation in response to punishment than those without familial risk (Gotlib et al., 2010). Results from the BART-Y cannot be separated into reward-seeking and risk-taking behaviour individually. This may, in part, explain the absence of a main effect between risk-taking on the BART and depressive symptoms in our sample. Future research would benefit from the use of tasks specifically measuring these two aspects of behaviour.

Also of note is the age of the participants when they took part in the current study. As our participants were aged between 11 and 12 years of age when they reported on their depressive symptoms, it is possible that the increase in depressive symptom presence observed in early adolescence had not yet fully occurred. This seems likely given that only a small number of participants experienced significant depressed mood. In previous research, the large increase in depression onset occurs in middle adolescence. For example, a longitudinal study following participants from age 11 to age 21 demonstrated a significant rise in depression occurrence from age 15 to age 18 (Hankin et al., 1998). Nonetheless, the examination of risk factors for depression in early adolescence provides important insights into the development of this disorder as it begins. Furthermore, examining depressive symptoms is important as subclinical depressive symptoms have still been associated with impaired functioning (Gotlib, Lewinsohn, & Seeley, 1995) and the onset of later depressive

disorders (van Lang, Ferdinand, & Verhulst, 2007). Future research would benefit from continuing to follow the early adolescents into the peak range of 15 to 18 years of age to capture the full range of adolescent depressive symptoms.

One of the strengths of the present study is that multiple factors measured in early adolescence were examined alongside BI (reported and observed) when the participants were 3-4 years of age. Research examining the association between BI and depression has often examined BI retrospectively or at the same time as depressive symptoms. For example, child and parent reported BI in adolescence was associated with higher reported anxiety and depression measured at the same time point (Muris et al., 2003). Only a small number of other studies have used longitudinal designs measuring BI in childhood and depression in adolescence. For example, a long-term prospective study found that inhibition at age 3 was linked to juvenile depression, particularly juvenile depression that recurred in adulthood (Jaffee et al., 2002). Ideally, a study should measure BI in early childhood, following participants into adolescence. Nonetheless, in contrast to previous studies (e.g. Jaffee et al., 2002), a main effect of BI on the development of symptoms of depression was not found in the current study. There are several possible explanations for the current study not demonstrating this hypothesised relationship. Previous studies that have shown a direct relationship between BI and depression have been cross sectional (Neal, Edelmann, & Glachan, 2002) measured self-reported BI retrospectively (Muris et al., 2001) or measured depression in late adolescence or adulthood (Caspi et al., 1996). Thus, the differences in our study design may explain the differences in our findings.

Another strength of the current research is the use of both child and mother report for a structured interview and questionnaire to assess depressive symptoms. It is considered optimal to obtain information from multiple informants when assessing depression including the child and parents (Klein et al., 2005). Parent, clinician, and child ratings have

been shown to all explain significant unique differences in predicting ensuing outcomes (Verhulst, Dekker, & Ende, 1997).

There are also a few limitations to consider. Firstly, there are limitations in the generalisability of the sample used in the current study. The clear majority of participants identified their ethnicity as western, with over half of the sample from middle to high income families. Furthermore, the use of imputation to account for the missing data should be taken into consideration when interpreting the findings.

Overall, the results of the current study indicate that the association between risk-taking and depressive symptoms and between pubertal status and depressive symptoms may vary between early adolescents classified as BI and those classified as BUI in childhood. Although there were no significant main effects of temperament, puberty or risk-taking on depressive symptoms, there were significant interactions such that BI and risk-taking on the BART-Y, and BI and pubertal status interacted to predict depressive symptoms. The possibility that the pathway from BI in childhood to an increased risk for depression in adolescence may be moderated by risk-taking behaviour or pubertal status provides an important direction for future research.

References

Allen, N. B., & Badcock, P. B. (2003). The Social Risk Hypothesis of Depressed Mood: Evolutionary, psychosocial, and neurobiological perspectives. *Psychological Bulletin, 129*(6), 887-913. doi:http://dx.doi.org/10.1037/0033-2909.129.6.887

Angold, A., Costello, E. J., Messer, S. C., Pickles, A., Winder, F., & Silver, D. (1995). Development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *International Journal of Methods in Psychiatric Research*(5), 237-249.

Birmaher, B., Ryan, N. D., Williamson, D. E., Brent, D. A., Kaufman, J., Dahl, R. E., Perel, J., & Nelson, B. (1996). Childhood and adolescent depression: A review of the past 10 years. Part I. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*(11), 1427-1439.

Brooks-Gunn, J., Warren, M. P., Rosso, J., & Gargiulo, J. (1987). Validity of self-report measures of girls' pubertal status. *Child Development, 58*(3), 829-841.

Buck, K. A., & Dix, T. (2012). Can developmental changes in inhibition and peer relationships explain why depressive symptoms increase in early adolescence? *Journal of Youth and Adolescence, 41*(4), 403-413.

Caspi, A., Moffitt, T. E., Newman, D. L., & Silva, P. A. (1996). Behavioral observations at age 3 years predict adult psychiatric disorders: Longitudinal evidence from a birth cohort. *Archives of General Psychiatry, 53*(11), 1033-1039.

Clauss, J. A., Avery, S. N., & Blackford, J. U. (2015). The nature of individual differences in inhibited temperament and risk for psychiatric disease: A review and meta-analysis. *Progress in Neurobiology, 127–128*, 23-45. doi:http://dx.doi.org/10.1016/j.pneurobio.2015.03.001

Costello, E. J., & Angold, A. (1988). Scales to assess child and adolescent depression: checklists, screens, and nets. *Journal of the American Academy of Child & Adolescent Psychiatry, 27*(6), 726-737.

Forbes, E. E., Shaw, D. S., & Dahl, R. E. (2007). Alterations in reward-related decision making in boys with recent and future depression. *Biological Psychiatry, 61*(5), 633-639. doi:http://dx.doi.org/10.1016/j.biopsych.2006.05.026

Galvao, T. F., Silva, M. T., Zimmermann, I. R., Souza, K. M., Martins, S. S., & Pereira, M. G. (2014). Pubertal timing in girls and depression: a systematic review. *Journal of Affective Disorders*, *155*, 13-19.

Garber, J. (2006). Depression in children and adolescents. Linking risk research and prevention. *American Journal of Preventive Medicine, 31*(6 SUPPL. 1), 104-125.

Garcia Coll, C., Kagan, J., & Reznick, J. (1984). Behavioral inhibition in young children. *Child Development, 55*(3), 1005-1019.

Gersten, M. (1989). Behavioral inhibition in the classroom. *Reznick, J Steven [Ed]*, 71-91.

Gladstone, G. L., & Parker, G. B. (2006). Is behavioral inhibition a risk factor for depression? *Journal of Affective Disorders, 95*(1-3), 85-94. doi:http://dx.doi.org/10.1016/j.jad.2006.04.015

Gotlib, I. H., Hamilton, J., Cooney, R. E., Singh, M. K., Henry, M. L., & Joormann, J. (2010). Neural processing of reward and loss in girls at risk for major depression. *Archives of General Psychiatry, 67*(4), 380-387. doi:http://dx.doi.org/10.1001/archgenpsychiatry.2010.13

Gotlib, I. H., Lewinsohn, P. M., & Seeley, J. R. (1995). Symptoms versus a diagnosis of depression: differences in psychosocial functioning. *Journal of Consulting and Clinical Psychology, 63*(1), 90.

Guyer, A. E., Nelson, E. E., Perez-Edgar, K., Hardin, M. G., Roberson-Nay, R., Monk, C. S., Bjork, J. M., Henderson, H. A., Pine, D. S., & Fox, N. A. (2006). Striatal

functional alteration in adolescents characterized by early childhood behavioral inhibition. *The Journal of Neuroscience, 26*(24), 6399-6405.

Hamilton, J. L., Hamlat, E. J., Stange, J. P., Abramson, L. Y., & Alloy, L. B. (2014). Pubertal timing and vulnerabilities to depression in early adolescence: Differential pathways to depressive symptoms by sex. *Journal of Adolescence, 37*(2), 165-174.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology, 107*(1), 128.

Helfinstein, S. M., Benson, B., Perez-Edgar, K., Bar-Haim, Y., Detloff, A., Pine, D. S., Fox., N. A., & Ernst, M. (2011). Striatal responses to negative monetary outcomes differ between temperamentally inhibited and non-inhibited adolescents. *Neuropsychologia, 49*(3), 479-485.

Hirshfeld-Becker, D. R., Micco, J., Henin, A., Bloomfield, A., Biederman, J., & Rosenbaum, J. (2008). Behavioral inhibition. *Depression and Anxiety, 25*(4), 357-367. doi:http://dx.doi.org/10.1002/da.20490

Hudson, J. L., Dodd, H. F., & Bovopoulos, N. (2011). Temperament, family environment and anxiety in preschool children. *Journal of Abnormal Child Psychology: An official publication of the International Society for Research in Child and Adolescent Psychopathology, 39*(7), 939-951.

Jaffee, S. R., Moffitt, T. E., Caspi, A., Fombonne, E., Poulton, R., & Martin, J. (2002). Differences in early childhood risk factors for juvenile-onset and adult-onset depression. *Archives of General Psychiatry, 59*(3), 215-222. doi:http://dx.doi.org/10.1001/archpsyc.59.3.215

Kaufman, J., Birmaher, B., Brent, D., Rao, U. M. A., Flynn, C., Moreci, P., Williamson, D., & Ryan, N. (1997). Schedule for Affective Disorders and Schizophrenia for School-

Age Children-Present and Lifetime Version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(7), 980-988. doi:http://dx.doi.org/10.1097/00004583-199707000-00021

Kendler, K. S., Gardner, C. O., & Prescott, C. A. (2002). Toward a comprehensive developmental model for major depression in women. *American Journal of Psychiatry, 159*(7), 1133-1145.

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., Wittchen, H. U., & Kendler, K. S. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: Results from the National Comorbidity Survey. *Archives of General Psychiatry, 51*(1), 8-19.

Klein, D. N., Dougherty, L. R., & Olino, T. M. (2005). Toward guidelines for evidence-based assessment of depression in children and adolescents. *Journal of Clinical Child and Adolescent Psychology, 34*(3), 412-432.

Knutson, B., Adams, C. M., Fong, G. W., & Hommer, D. (2001). Anticipation of increasing monetary reward selectively recruits nucleus accumbens. *Journal of Neuroscience, 21*(16), RC159.

Lahat, A., Hong, M., & Fox, N. A. (2011). Behavioural inhibition: Is it a risk factor for anxiety? *International Review of Psychiatry, 23*(3), 248-257.

Lau, J. Y., & Eley, T. C. (2010). The genetics of mood disorders. *Annual Review of Clinical Psychology, 6*, 313-337. doi:http://dx.doi.org/10.1146/annurev.clinpsy.121208.131308

Lejuez, C., Aklin, W., Daughters, S., Zvolensky, M., Kahler, C., & Gwadz, M. (2007). Reliability and validity of the Youth Version of the Balloon Analogue Risk Task (BART-Y) in the assessment of risk-taking behavior among inner-city adolescents. *Journal of Clinical Child and Adolescent Psychology, 36*(1), 106-111. doi:http://dx.doi.org/10.1207/s15374424jccp3601_11

Lejuez, C., Aklin, W. M., Bornovalova, M. A., & Moolchan, E. T. (2005). Differences in risk-taking propensity across inner-city adolescent ever- and never-smokers. *Nicotine & Tobacco Research, 7*(1), 71-79.

Lewinsohn, P. M., Rohde, P., Klein, D. N., & Seeley, J. R. (1999). Natural course of adolescent major depressive disorder: I. Continuity into young adulthood. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(1), 56-63.

Mendle, J., Harden, K., Brooks-Gunn, J., & Graber, J. A. (2010). Development's tortoise and hare: Pubertal timing, pubertal tempo, and depressive symptoms in boys and girls. *Developmental Psychology, 46*(5), 1341-1353. doi:http://dx.doi.org/10.1037/a0020205

Mills, K. L., Goddings, A.-L., Clasen, L. S., Giedd, J. N., & Blakemore, S.-J. (2014). The developmental mismatch in structural brain maturation during adolescence. *Developmental Neuroscience, 36*(3-4), 147-160.

Morris, T. M., Hudson, J. L., & Dodd, H. F. (2014). Risk-taking and inhibitory control in behaviourally inhibited and disinhibited preschool children. *Personality and Individual Differences*, *71*, 113-117.

Morrongiello, B. A., Kane, A., McArthur, B. A., & Bell, M. (2012). Physical risk-taking in elementary-school children: Measurement and emotion regulation issues. *Personality and Individual Differences, 52*(4), 492-496. doi:http://dx.doi.org/10.1016/j.paid.2011.11.003

Muris, P., Meesters, C., & Spinder, M. (2003). Relationships between child- and parent-reported behavioural inhibition and symptoms of anxiety and depression in normal adolescents. *Personality and Individual Differences, 34*(5), 759-771. doi:http://dx.doi.org/10.1016/S0191-8869%2802%2900069-7

Natsuaki, M. N., Biehl, M. C., & Ge, X. (2009). Trajectories of depressed mood from early adolescence to young adulthood: The effects of pubertal timing and adolescent

dating. *Journal of Research on Adolescence, 19*(1), 47-74. doi:http://dx.doi.org/10.1111/j.1532-7795.2009.00581.x

Neal, J. A., Edelmann, R. J., & Glachan, M. (2002). Behavioural inhibition and symptoms of anxiety and depression: Is there a specific relationship with social phobia? *British Journal of Clinical Psychology, 41*(4), 361-374. doi:http://dx.doi.org/10.1348/014466502760387489

Nelson, B. D., Perlman, G., Klein, D. N., Kotov, R., & Hajcak, G. (2016). Blunted neural response to rewards as a prospective predictor of the development of depression in adolescent girls. *American Journal of Psychiatry, 173*(12), 1223-1230.

Nolan, S. A., Flynn, C., & Garber, J. (2003). Prospective relations between rejection and depression in young adolescents. *Journal of Personality and Social Psychology, 85*(4), 745.

Pine, D. S., Cohen, E., Cohen, P., & Brook, J. (1999). Adolescent depressive symptoms as predictors of adult depression: Moodiness or mood disorder? *American Journal of Psychiatry, 156*(1), 133-135.

Pine, D. S., Cohen, P., Gurley, D., Brook, J., & Ma, Y. (1998). The risk for early-adulthood anxiety and depressive disorders in adolescents with anxiety and depressive disorders. *Archives of General Psychiatry, 55*(1), 56-64.

Rapee, R., M., Kennedy, S., Ingram, M., Edwards, S., & Sweeney, L. (2005). Prevention and Early Intervention of Anxiety Disorders in Inhibited Preschool Children. *Journal of Consulting and Clinical Psychology, 73*(3), 488-497. doi:http://dx.doi.org/10.1037/0022-006X.73.3.488

Rubin, K. H., Burgess, K. B., & Hastings, P. D. (2002). Stability and social-behavioral consequences of toddlers' inhibited temperament and parenting behaviors. *Child Development, 73*(2), 483-495.

Rubin, K. H., Coplan, R. J., & Bowker, J. C. (2009). Social Withdrawal in Childhood. *Annual Review of Psychology, 60*, 141-171.

Rubin, K. H., Coplan, R. J., Fox, N. A., & Calkins, S. D. (1995). Emotionality, emotion regulation, and preschoolers' social adaptation. *Development and Psychopathology, 7*(1), 49-62. doi:http://dx.doi.org/10.1017/S0954579400006337

Sanson, A., Prior, M., Garino, E., Oberklaid, F., & Sewell, J. (1987). The structure of infant temperament: Factor analysis of the Revised Infant Temperament Questionnaire. *Infant Behavior and Development, 10*(1), 97-104.

Schmitz, K. E., Hovell, M. F., Nichols, J. F., Irvin, V. L., Keating, K., Simon, G. M., Gehrman, C., & Jones, K. L. (2004). A validation study of early adolescents' pubertal self-assessments. *Journal of Early Adolescence, 24*(4), 357-384. doi:10.1177/0272431604268531

Sinharay, S., Stern, H. S., & Russell, D. (2001). The use of multiple imputation for the analysis of missing data. *Psychological Methods, 6*(4), 317.

Smoski, M. J., Lynch, T. R., Rosenthal, M., Cheavens, J. S., Chapman, A. L., & Krishnan, R. R. (2008). Decision-making and risk aversion among depressive adults. *Journal of Behavior Therapy and Experimental Psychiatry, 39*(4), 567-576. doi:http://dx.doi.org/10.1016/j.jbtep.2008.01.004

Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review, 28*(1), 78-106.

van Lang, N. D., Ferdinand, R. F., & Verhulst, F. C. (2007). Predictors of future depression in early and late adolescence. *Journal of Affective Disorders, 97*(1), 137-144.

Verhulst, F. C., Dekker, M., & Ende, J. v. d. (1997). Parent, teacher and self-reports as predictors of signs of disturbance in adolescents: Whose information carries the most weight? *Acta Psychiatrica Scandinavica, 96*(1), 75-81.

Next is the second empirical paper with an early adolescent population. Chapter 3 demonstrated that risk-taking may play a role in the relationship between BI in childhood and depressive symptoms in early adolescence. Using the same sample as chapter 3, this study further explores the importance of risk-taking in the development of adolescent depression by focusing on social risk-taking. Adolescence is a time of increased importance for peer relationships, with peer victimisation and acceptance being previously shown to impact psychopathology. Thus, a novel measure was utilised to examine the relationship between depressive symptoms and social risk-taking over a 6-month period. In addition, measures of social acceptance and peer victimisation are examined as potential mediators of this relationship.

Note that the primary aims of this paper do not involve the examination of BI. Whilst participants had initially been recruited as BI/BUI in early childhood, in order to maximise both statistical power and clarity, BI was not examined in the following paper. The relationship between BI and social risk-taking was examined to ensure this decision was appropriate. There was no significant relationship between BI and social risk-taking, the results are presented in Appendix C.

This chapter focuses on measures captured at two time points, 6 months apart. At the second time point solely online questionnaires were used, this meant that only the two Short Mood and Feelings Questionnaires (parent and child report) were available for both time points. Whilst the previous chapter utilised clinician, parent and child reported depression, in this chapter only the Short Mood and Feelings Questionnaire (parent and child report) was used as a measure of depressive symptoms.

Chapter Four

The Relationship Between Social Risk-Taking and Depression in Adolescence: The Mediating Effect of Perceived Social Acceptance.

Talia M. Morris, Jennifer L. Hudson, & Helen F. Dodd

Abstract

Adolescence is a time of change, including growth in the importance of social interactions, rises in risk-taking behaviour and a significant increase in the risk for developing depression. Utilising a novel measure of social risk-taking, the current study examined the relationship between social risk-taking and depressive symptoms and the possibility that this relationship is mediated by experiences with peers. Using a longitudinal design (n = 76), it was hypothesised that decreased social risk-taking at time 1 during early adolescence (11-12 years of age) may be associated with increased depressive symptoms at time 2, six months later. Further, it was hypothesised that depressive symptoms at time 1 may be associated with later reductions in social risk-taking behaviour. A bidirectional relationship was shown. Increased depressive symptoms at time 1 predicted reduced social risk-taking at time 2, providing some support to the social risk hypothesis of depression. Increased social risk-taking at time 1 predicted increased depressive symptoms at time 2. Finally, social acceptance partially mediated the relationship between depressive symptoms and social risk-taking: more depressive symptoms at time 1 was associated with less social acceptance at time 1 and less social risk-taking at time 2. This finding provides evidence of a possible mechanism by which depressive symptoms may impact social experiences for early adolescents.

Depression is a common mental health problem in adolescence, with estimated prevalence rates of between 4 and 5% in mid-adolescence (Costello, Erkanli, & Angold, 2006). The prevalence of depression in childhood is low, less than 1% in most studies (Kessler, Avenevoli, & Ries Merikangas, 2001), however increases significantly throughout adolescence (Green, McGinnity, Meltzer, Ford, & Goodman, 2005). For example, one-year point prevalence rates of depression rise radically from around 2% in early adolescence (age 13-15 years) to approximately 15% in middle adolescence (15-18 years) (Hankin et al., 1998).

This increase in depressive disorders during adolescence could be attributable to many different factors, as this developmental period is exemplified by several social and biological changes. Pubertal changes are occurring, including physiological changes (e.g. changes in hormonal concentrations). Adolescence is also a time of change in the social environment, with amplified self-consciousness, an enhanced understanding of others and an increased importance of peer relationships, all features of adolescence (Steinberg & Morris, 2001). Finally, the brain circuits and neural pathways involved in response to reward and risk are also changing. An increase in risk-taking behaviour is common during adolescence (Steinberg, 2008) and may be attributable to an increase in sensation seeking associated with changes in dopaminergic activity around the time of puberty (Steinberg, 2008). The social environment, risk-taking behaviour and pubertal changes may independently or in interaction with each other, be associated with the increase of depression during adolescence. Each of these factors will now be discussed in turn.

Adolescence is a time of enhanced cognitive maturation including increased social understanding and self-awareness. Psychological changes occur that influence an adolescent's sense of identity, self-consciousness, and their relationships with others (Steinberg & Morris, 2001). A central part of adolescent self-identity is acceptance by peers, which also has a strong influence on psychological adjustment (Harter, 1998). Low self-

perceived social acceptance has been associated with increases in adolescent depressive symptoms over a six-month period (Uhrlass, Crossett, & Gibb, 2008). Peer victimisation has been found to be associated with depression in adolescents across a number of studies. In a meta-analytic review of cross-sectional studies, peer victimisation was found to be strongly related to depression (Hawker & Boulton, 2000). In particular, it has been demonstrated that adolescents who experience relational victimisation from peers report higher levels of depressive symptoms compared with other teens (La Greca & Harrison, 2005). In contrast to overt aggression, relational aggression uses a teen's relationships, or their friendships, as a way of causing social impairment, for example exclusion from social situations, and the spreading of rumours (e.g. Crick, 1995, 1997; Galen & Underwood, 1997). Specifically, increases in peer rejection have also been shown to significantly predict increases in depressive symptoms over a one year period in 10 to 14 year olds (Panak & Garber, 1992). Furthermore, adolescents who experienced moderate to high levels of loneliness across adolescence displayed more depressive symptoms than other teens and experienced a larger gain in depressive symptoms over time (Ladd & Ettekal, 2013).

The relationship between social victimisation and depression is complex, with the direction of the causal relationship not entirely clear. Several researchers have suggested that peer victimisation can lead to depressive symptoms in adolescence. In support of this, a number of longitudinal studies have demonstrated that peer rejection temporally predicts adolescent depression (e.g. Panak & Garber, 1992; Nolan, Flynn & Garber, 2003). In contrast, others propose that depression increases the likelihood of experiencing peer victimisation and rejection later in adolescence. For example, Rubin and colleagues (2003) argue that socially withdrawn children attribute their negative relationships with peers to internal causes and as a result may encounter feelings of loneliness and depression. Also, depressed adolescents' behaviours have been shown to lead to a decrease in positive behaviour from a partner compared to healthy controls (Heller & Tanaka-Matsumi, 1999).

Interpersonal theories of depression propose that depressive symptoms and social victimisation are actually part of an escalating cycle where problematic social relationships of depressed persons are hypothesised to have an additive effect on the development of their depressive symptoms (Coyne, 1976). Perhaps depressed adolescents are drawn more to peers who confirm their negative self-beliefs (Swann, Stein-Seroussi, & Giesler, 1992) or instead, they experience more victimisation because of increased reassurance seeking (Coyne, 1976). Depressive symptoms may be associated with increased social risk aversion, due to the reduction in motivation to seek social rewards. Increased depressive symptoms may be associated with negative peer interactions such as increased peer victimisation or reduced social acceptance which then leads to reduced risk-taking in social situations. To examine the temporal nature of depressive symptoms and social experiences in adolescents, further research using longitudinal studies is vital.

Another important change occurring during adolescence is a shift in risk-taking behaviour. There is an increase in risk-taking behaviour over the adolescence period in an inverted U function, with risk-taking peaking in middle adolescence and then declining through late adolescence (e.g. Steinberg, 2008). The dual systems model of risk-taking in adolescence suggests that vulnerability to risk-taking is a result of high reward-seeking and low impulse control. In particular, neurobiological changes occur that impact these two important elements of risk-taking behaviour. It is proposed that a rapid and dramatic increase in dopaminergic activity in the socioemotional system of the brain around the time of puberty leads to an increase in reward-seeking behaviour (Steinberg, 2008). However, this increase in reward-seeking behaviour occurs before the cognitive control system, involved in advanced self-regulation and impulse control, has fully matured. Thus, it is hypothesised that a period of heightened vulnerability to risk-taking is created, peaking in middle adolescence, where teenagers are more motivated to seek rewards, however they do not possess the self-regulation to adequately assess the risks (Steinberg, 2008). Thus

heightened motivation to seek rewards leads to an increase in risk-taking behaviours. This increased vulnerability to risk-taking is reflected in real world behaviours. For example, adolescents are more likely to binge drink, smoke cigarettes or engage in violent and other criminal behaviour than younger or older individuals (Steinberg, 2008).

Both decreased risk-taking behaviour and a reduction in reward-seeking behaviour have been linked to risk for depression in adolescence. Two interrelated neural circuits that continue to mature during adolescence are known to be active in the response to danger and to learning about rewards, and have been closely associated with an increased risk for depression in both adults and adolescents (Feder, Nestler, & Charney, 2009; Forbes & Dahl, 2005). One of these circuits is the connection between the amygdala and hippocampus and ventral expanses of the prefrontal cortex (PFC). This pathway is linked to the hypothalamic-pituitary-adrenal (HPA) axis activity. Individuals with depression as well as those at high risk of depression have been shown to have increased activity in this circuit compared to healthy control participants (Brody et al., 1999). The second neural circuit associated with depression includes the striatum and its connection to the PFC and ventral dopamine-based systems. Reduced activity in this circuit during tasks involving rewards has been demonstrated in depressed individuals and individuals with depressed parents (Forbes et al., 2009). Behavioural studies have also demonstrated that a reduction in reward-seeking behaviour during this period, relative to other adolescents, can be a risk factor for the development of depression. For example, a longitudinal study demonstrated that early adolescents who had difficulty distinguishing between low and high reward options on an experimental paradigm were more likely to experience depressive symptoms in a follow up one year later (Forbes, Shaw, & Dahl, 2007). Also, adults with depression demonstrate more risk aversion on an experimental risk-taking paradigm than healthy controls (Smoski et al., 2008). It is possible that a reduction in reward-seeking behaviour in those at risk for depression may also be present in real world settings such as social interactions, leading to a

reduction in risk-taking in these settings (Brinkmann, Franzen, Rossier, & Gendolla, 2014). For example, in a non-clinical adult sample, behavioural and cognitive avoidance in social situations was shown to be associated with increased depressive symptoms (Moulds, Kandris, Starr, & Wong, 2007).

The idea that depression may be linked to a diminished response to social rewards has been examined briefly in the literature. One model of depression, the social risk hypothesis, proposes that depressed mood evolved to allow for risk aversion in social situations that might lead to an individual being excluded (Allen & Badcock, 2003). Specifically, depression is seen as a mechanism to deal with social challenges by heightening sensitivity for indicators of risk in social situations.

Other researchers suggest that a reduction in social reward-seeking occurs before depression (Davey, Yucel, & Allen, 2008). As a result of not being able to experience more distant and abstract rewards, such as rewards in social and romantic relationships, a suppression of the reward system can occur (Davey et al., 2008). If this suppression transpires for an extended period of time and is combined with other vulnerability factors, it is theorised that depression can ensue. Consequently, although previous research and theories have discussed a link between reduced social reward-seeking behaviour in those experiencing depression (Rubin, Coplan, Fox, & Calkins, 1995), to date the direction of this link is not clear. In the present research, this question is explored by examining whether this reduction in social reward-seeking manifests as a decrease in risk-taking in social situations for those experiencing, or at risk of, depression. Thus, this research measures social risk-taking.

Methods for capturing social risk-taking in a laboratory setting are scarce. Although computer tasks such as ball throwing (Williams, Cheung, & Choi, 2000) have measured social rejection, none have examined real world social risk-taking. The current study uses a new questionnaire measure of social risk-taking. Utilising a set of vignettes about social

situations applicable to adolescents, participants are required to respond with an open answer as to how they would act in response to that situation. Hypothetical vignettes were used as this methodology allowed us to measure the response of the adolescents to potentially anxiety provoking situations that would be difficult to observe (Pellegrini & Bartini, 2000). Previous research has used hypothetical vignettes to assess for children's responses to important interpersonal situations (e.g. Dirks, Suor, Rusch, & Frazier, 2014). Furthermore, past studies have shown that how adolescents report they would behave in social situations is correlated with how others perceive their actual behaviour (e.g. Hughes, Webster-Stratton, & Cavell, 2004). Therefore, a new measure utilising vignettes is an appropriate method to measure social risk-taking in adolescents.

The final risk factor to be examined in the current study is pubertal status. The onset of puberty has been suggested to play an important part in the increase of depression during adolescence. Puberty involves both morphological (e.g. secondary sex changes) and physiological development (e.g. hormones). A number of researchers maintain that the changes in adrenal and gonadal hormones during pubertal development increase risk for psychopathology, including depressive symptoms (e.g. Angold, Costello, Erkanli, & Worthman, 1999). Furthermore, the increase in depressive symptoms during adolescence has been linked more closely to changes in female hormones than to chronological age (Angold et al., 1999). In fact, a previous study has demonstrated that pubertal status can moderate the relationship between Behavioural Inhibition (BI), a risk factor for depression, and depressive symptoms in early adolescence (Morris, Hudson, & Dodd, 2017; Chapter 3). In addition, early onset of puberty is associated with a greater level of depressive symptoms in adolescent females and males (Mendle, Harden, Brooks-Gunn, & Graber, 2010). This may be explained by the greater social and emotional challenge puberty presents for a younger child less able to manage such changes than an older peer (Ge, Conger, & Elder, 2001).

The current study aims to examine social risk-taking in an early adolescent sample across two time points. The previous research summarised above suggests that decreased risk-taking is linked with increased depression. Thus, it is hypothesised that decreased social risk-taking at time 1 during early adolescence (11-12 years of age) will be associated with increased depressive symptoms at time 2, six months later. Further, it is possible that depressive symptoms at time 1 may be associated with later reductions in social risk-taking behaviour.

In addition, the current study aims to examine the possibility that the relationship between social risk-taking and depressive symptoms is mediated by experiences with peers. Based on previous research we would expect that the bidirectional relationship between social risk-taking and depression occurs via perceived social acceptance or reported peer victimisation. Furthermore, due to the importance of pubertal changes to adolescence (Morris et al., 2017; Chapter 3), pubertal status will be included as a control for analyses.

Method

Participants

Participants were 76 children and their mothers taking part in a larger longitudinal study (see Hudson, Dodd, & Bovopoulos, 2011). Participants were originally recruited as highly inhibited or uninhibited at age 3-4 years via local preschools and an advertisement in a free parenting magazine. When parents initially contacted researchers, they completed a screening questionnaire for BI in their child. Using the mothers' report on the Approach Scale of the Short Temperament Scale for Children (Sanson, Prior, Garino, Oberklaid, & Sewell, 1987; STSC), children scoring 1 standard deviation above or below the normative mean on the STSC were invited to take part in the full study. For the current study, at time 1 participants were aged between 11 and 13 years (M = 11 years and 8 months, SD = 3.6 months), with 40 males and 36 females. For ethnicity, 50% of children identified as

Oceanic, 13% as European and 9% Asian, with the remainder identified as African, American or Middle Eastern.

Measures

Depressive symptoms. Participants and their mothers completed the Short Mood and Feelings Questionnaire (SMFQ: Angold et al., 1995) as part of an online questionnaire battery both at time 1 and six months later at time 2. The SMFQ consists of 13 items that assess depressive symptoms occurring over the previous two weeks on a 3-point scale. The SMFQ has good internal consistency ($\alpha = 0.85$; Angold et al., 1995), and the test-retest reliability is 0.66 over a two week period (Costello & Angold, 1988). The SMFQ correlates moderately with other child-reported measures of depressive symptoms (0.67 with the Children's Depressive Inventory; Angold et al., 1995). The internal consistency in our sample was high, $\alpha = .87$ for the child report, $\alpha = .87$ for the maternal report at time 1.

Social risk-taking. A new measure was created for the current study to capture social risk-taking. Thirteen vignettes were generated describing everyday situations involving social risk. For example, "After class one day, one of your friends asks you to play basketball. You notice that you don't know any of the other kids playing but that they look nice. What do you do?" Initially the Social Risk-Taking Questionnaire (SRTQ) was administered to 20 pilot participants (aged 10 – 13 years) who gave feedback on the wording of the vignettes and how they would respond to them. All pilot participants felt that one vignette was potentially problematic as it asked participants whether they would attend a party they had not been invited to. The feedback from pilot participants was that it is rude to attend, regardless of whether you were willing to go to a party with unfamiliar people or not. Thus, this item was removed from the final questionnaire. A full list of the 12 vignettes is included in Appendix D. Participants' responses to each vignette are open-ended, with responses coded for the presence (1) or absence (0) of risk-taking. Risk-taking was defined as approaching the other child in the story. For example, a response to the vignette above

coded as risk-taking would be "I would join in". A total score is calculated for each participant by summing the score received for each vignette, with a higher score indicating more social risk-taking. All responses were double coded by two trained coders. The scoring agreement for the total score on the SRTQ showed a high degree of reliability for time 1 (intraclass correlation ICC = .84) and for time 2 (ICC = .93).

Peer victimisation. Participants completed the Revised Peer Experiences Questionnaire (RPEQ; Prinstein, Boergers & Vernberg, 2001) at the time 1 laboratory session and as part of the time 2 online questionnaire battery. The RPEQ contains nine items. For each item, participants were asked how often a behaviour had been directed towards the informant (e.g., "A peer chased me like he or she was really trying to hurt me") on a 5-point scale (1 = *never*, 2 = *once or twice,* 3 = *a few times*, 4 = *about once a week,* 5 = *a few times a week*). Overt and relational subscales are computed by summing the items included in each subscale. Good internal consistency estimates have been reported (Prinstein et al., 2001; overt α = .79, relational α = .76). For this study, good internal consistency was found for both the victimisation subscales (overt α = .82, relational α = .78)

Social acceptance. Harter's (1988) Self-Perception Profile for Adolescents (SPPA) assesses adolescents' self-perceptions in six domains (e.g. romantic appeal, friendship competence, etc.). For this study the social acceptance subscale containing six items, was included in the online questionnaire battery completed at baseline and the 6-month follow up by participants. Each item is coded with a score of 1 through 4, with a mean calculated across all six items. Higher scores represent higher perceived social acceptance. Good internal consistency for the SPPA has been reported (Harter, 1998; Cronbach's α = .74-.93). In our sample, the subscale demonstrated good internal consistency (α=.79).

Pubertal status. Participant's assessed their current pubertal status using Tanner's Sexual Maturation Scale (SMS: Marshall & Tanner, 1969, 1970). This task uses drawings to

depict five stages of pubertal development, either female breast and pubic hair or male genitalia and pubic hair. The SMS is often used when a physical examination by a qualified physician is impractical or would be too confronting for a participant or their parents. Previous studies have reported a high level of agreement between self-report using the SMS and physician examination within one pubertal stage (e.g. 85% agreement Schmitz et al., 2004). Participants were asked to select the drawing that corresponds to their stage of pubertal development.

Procedure

The procedures for the current study, including consent procedures, were approved by the Macquarie University Human Ethics Committee. Children were invited to participate in a 2-hour research session on campus at Macquarie University for the time 1 assessment as well as an online questionnaire battery at time 1 and at time 2. At both time points, written consent was provided by the mothers for both their and their child's participation and, following a brief description of the research, children provided verbal assent/consent. Written questionnaires were completed prior to attending the research session at time 1 for both mother and child. At the research session the family were reimbursed $50 and the children were given a small age appropriate gift. At time 2, the mother and child completed the online questionnaire battery after being approached by the researchers over the phone. Additional observed tasks and questionnaires were completed at both time points which are not presented as part of this study.

Analysis Plan

All analyses for the current study were completed using SPSS (v24, IBM). Due to incomplete surveys and technical issues, there was a small amount of missing data at time 2 on the SRTQ, PVQ and SPPC. In total 9% of items were missing from the dataset. A non-significant Little's MCAR test, $\chi 2(323) = 319.48$, $p = .55$, revealed that the data were missing completely at random (Little, 1988). When data are missing completely at random

and only a small portion of data are missing, a single imputation using the expectation maximization algorithm provides unbiased parameter estimates and improves statistical power of analyses (Enders, 2001; Scheffer, 2002). Missing data were imputed using Missing Values Analysis within SPSS 24.0.

To explore the relationship between social risk-taking and depressive symptoms at both time points, a series of Pearson correlations were conducted, also including the other time 1 predictor variables. To further examine the directionality of the relationship between social risk-taking and depression, three regression analyses were completed, one for each of the time 2 outcome variables (child report depressive symptoms, maternal report depressive symptoms, and social risk-taking). Each regression included the outcome variable (social risk-taking, or depressive symptoms) and the additional predictor variables (social risk-taking, social acceptance, overt victimisation, and relational victimisation) that were significantly correlated with the outcome variable in the Pearson correlations, as well as the time 1 measure of the outcome variable as a control. Pubertal status was also included as a control measure.

Finally, the possibility of mediation was examined between depressive symptoms/social risk-taking at time 1 and social risk-taking/depressive symptoms at time 2. It was predicted that social acceptance and/or peer victimisation would act as the mediating variable. Potential mediation was examined using the PROCESS tool for SPSS (Hayes, 2012). As recommended for small samples, nonparametric bootstrapping analyses were used to test the mediational models (Preacher & Hayes, 2004; Preacher, Rucker, & Hayes, 2007). In these analyses, mediation was considered significant if the 95% bias corrected and accelerated confidence intervals for the indirect effect do not include zero (Preacher & Hayes, 2004; Preacher, al., 2007).

Results

Descriptive and Preliminary Analyses

The mean and SD of the SMFQ child and mother report, SRTQ, victimisation, social acceptance and pubertal status at both time points is provided in Table 4.1.

Table 4.1
Mean and SD of depressive symptoms, social risk-taking, victimisation, social acceptance and pubertal status by time point.

		Time 1	Time 2
Depressive Symptoms	Child Report	2.48 (3.49)	2.69 (2.89)
	Mother Report	1.78 (2.50)	2.50 (3.62)
Social Risk-taking		10.35 (1.52)	10.04 (2.02)
Peer Victimisation	Overt Victimisation	1.39 (0.53)	0.24 (0.43)
	Relational Victimisation	1.44 (0.49)	0.33 (0.40)
Social Acceptance		3.20 (0.55)	3.33 (0.61)
Pubertal Status		2.40 (0.78)	2.90 (0.87)

Correlations were conducted to examine whether maternal age, family income, and ethnicity were associated with the predictor and outcome variables. Most were non-significant (all *p*'s > .05) but overt victimisation at time 1 was significantly correlated in a negative direction with family income (r^2 = -.28, *p* = .02). Those with a lower family income rated more overt victimisation on the RPEQ. Maternal age was also significantly negatively correlated with total victimisation at time 1 (r^2 = -.23, *p* = .047). As neither family income, nor maternal age were significantly related to our four outcome variables, they were not included in later analyses. No significant differences on any predictor and outcome variables by gender were found (all *p*'s > .05).

Relationships Between Predictor and Outcome Variables

The relationships between the time 1 predictor variables (child report depressive symptoms, maternal report depressive symptoms, social risk-taking, social acceptance, pubertal status, overt victimisation and relational victimisation) and the time 2 outcome variables (child report depressive symptoms, maternal report depressive symptoms, and social risk-taking) were examined using Pearson correlations coefficients. Several significant correlations were found for each outcome variable and all Pearson correlation coefficients are presented in Table 4.2. Time 1 social risk-taking, relational victimisation, and child and maternal report SMFQ were significantly positively correlated with time 2 child report depressive symptoms (*p*'s < .05). Time 1 maternal report SMFQ and social risk-taking were also positively correlated with time 2 maternal report SMFQ (*p* < .05).

A number of time 1 factors were significantly correlated with social risk-taking at time 2. Social risk-taking and social acceptance at time 1 were correlated in a positive direction with social risk-taking at time 2, while relational victimisation, and child and maternal report SMFQ were in a negative direction.

Table 4.2

Pearson correlations examining the relationships between the baseline predictor variables and 6-month follow up outcomes.

	Time 2 outcome variables		
	Child report depressive symptoms	Maternal report depressive symptoms	Social risk-taking
Time 1 variables			
Child report depressive symptoms	.34**	.13	-.67***
Maternal report depressive symptoms	.54***	.60***	-.49***
Social risk-taking	.34**	.30**	.33**
Social acceptance	-.22	.02	.48***
Overt victimisation	.07	.10	-.14
Relational victimisation	.29**	.14	-.29*
Pubertal status	.16	.03	.19

Note: * $p < .05$ ** $p < .01$ *** $p < .001$

Predicting Depressive Symptoms

Child report SMFQ. A regression analysis was conducted to assess the ability of the predictor variables, particularly social risk-taking, at time 1 to predict levels of depressive symptoms at time 2 after controlling for depressive symptoms at time 1. Only the predictor variables found to significantly correlate with follow-up depressive symptoms were included in the analysis (maternal report depressive symptoms, social risk-taking and relational peer victimisation) along with time 1 child report depressive symptoms, and pubertal status as a control measure.

The results from the regression analysis are summarised in Table 4.3. SMFQ maternal report *(*$\beta = 0.40$, $t(73) = 3.74$, $p < .0001$), pubertal status *(*$\beta = 0.26$, $t(73) = 2.74$, $p = .01$), and Social Risk-taking *(*$\beta = 0.28$, $t(73) = 3.01$, $p = .004$) significantly contributed to the model $F(5,70) = 10.84$, $p < .0001$. Higher maternal reported depressive symptoms at time 1 were associated with higher child report depressive symptoms at follow up, and increased social risk-taking at time 1 was also associated with higher child report depressive symptoms. Furthermore, increased pubertal status at time 1 was significantly associated with increased child report depressive symptoms at time 2. Relational peer victimisation was not a significant predictor of child report depressive symptoms at time 2, when controlling for time 1 depressive symptoms (all p's $> .05$). As neither of the potential mediating variables, peer victimisation or social acceptance, were found to significantly predict time 2 child report depressive symptoms, mediation was not explored for this outcome variable.

Table 4.3

Regression analysis predicting child report depressive symptoms at 6-month follow up from baseline predictors.

Variable	*B*	*SE B*	*β*
Constant	-6.81	2.15	
Baseline child report depressive symptoms	0.16	0.09	0.20
Baseline pubertal status	0.95	0.35	0.26**
Baseline maternal report depressive symptoms	0.46	0.12	0.40***
Baseline social risk-taking	0.52	0.17	0.28**
Relational victimisation	0.40	0.59	0.07

Note: $R^2 = .44$ ** $p < .01$ *** $p < .001$

Mother report SMFQ. A second regression analysis was conducted to assess the ability of social risk-taking and the other predictor variables at time 1 to predict levels of maternal report depressive symptoms at time 2 after controlling for depressive symptoms at time 1. Social risk-taking was the only predictor variable found to significantly correlate with follow-up maternal report depressive symptoms thus it was included in the analysis along with time 1 maternal report depressive symptoms and pubertal status as control measures.

The results from the regression analysis are summarised in Table 4.4. The control measure, depressive symptoms at time 1 SMFQ maternal report ($\beta = 0.57$, $t(73) = 6.15$, $p < .0001$) significantly contributed to the model $F(3,72) = 16.20$, $p < .0001$. Social risk-taking ($\beta = 0.21$, $t(73) = 2.26$, $p = .03$) was also a significant predictor of maternal report depressive symptoms at time 2, when controlling for time 1 depressive symptoms (all p's $> .05$). An increase in social risk-taking was associated with increased maternal report depressive symptoms at follow up. Pubertal status did not significantly predict maternal report depressive symptoms at time 2 ($p > .05$).

As neither of the potential mediating variables, peer victimisation or social acceptance, were found to significantly predict time 2 child report depressive symptoms, mediation was not explored for this outcome variable.

Social risk-taking. A final regression analysis was conducted to assess the ability of depressive symptoms and other predictor variables at time 1 to predict levels of social risk-taking at time 2 after controlling for social risk-taking at time 1. Only the predictor variables found to significantly correlate with follow-up social risk-taking were included in the analysis (child report depressive symptoms, maternal report depressive symptoms, social acceptance, and relational peer victimisation) along with time 1 social risk-taking and pubertal status as control measures.

Table 4.4

Regression analysis predicting maternal report depressive symptoms at 6-month follow up from baseline predictors.

Variable	*B*	*SE B*	*β*
Constant	-4.96	2.54	
Baseline maternal report depressive symptoms	0.82	0.13	0.57***
Baseline pubertal status	0.36	0.43	0.08
Baseline social risk-taking	0.50	0.13	0.57***

Note: $R^2 = .40$ *** $p < .001$

The results from the regression analysis are summarised in Table 4.5. Social risk-taking at time 1 (β = 0.41, $t(73) = 6.20$, $p < .0001$), child report depressive symptoms at time 1 (β = -0.39, $t(73) = -4.75$, $p < .0001$), maternal report depressive symptoms at time 1 (β = -0.32, $t(73) = -4.18$, $p < .0001$), and social acceptance at time 1 (β = 026, $t(73) = 3.66$, $p < .0001$) all significantly contributed to the model $F(6,69) = 28.19$, $p < .0001$. Neither relational peer victimisation, nor pubertal status were significant (*p's* > .05). Time 1 social acceptance demonstrated a positive relationship with social risk-taking, such that increased social acceptance at time 1 is correlated with higher social risk-taking at time 2. Conversely, a negative relationship was demonstrated with child and maternal report depressive symptoms, increased depressive symptoms at time 1 are associated with decreased social risk-taking at follow up.

Table 4.5

Regression analysis predicting social risk-taking at 6-month follow up from baseline predictors.

Variable	*B*	*SE B*	*β*
Constant	2.32	1.41	
Baseline social risk-taking	0.54	0.09	0.41***
Baseline pubertal status	0.18	0.17	0.07
Baseline child report depressive symptoms	-0.23	0.05	-0.39***
Baseline social acceptance	0.96	0.26	0.26***
Baseline maternal report depressive symptoms	-0.26	0.06	-0.32***
Baseline relational peer victimisation	-0.26	0.29	-0.06

Note: $R^2 = .71$ *** $p < .001$

Mediation Analysis

Using the PROCESS macro developed by Hayes (2012), we examined whether the relationship between depressive symptoms at time 1 and social risk-taking at time 2 was mediated by social acceptance. We conducted this first for child report of depression symptoms and second for parent report. Note that these were the only mediation analyses that were appropriate based on the findings reported above.

Social acceptance as mediator. Mediation analyses based on 10000 bootstrapped samples using bias-corrected and accelerated 95% confidence intervals (Preacher & Hayes, 2004) showed that controlling for the effect of time 1 social risk-taking (*b*= .46, *se* = .10, *p* <.001), child report depressive symptoms had a significant Total Effect (TE) on social risk-taking at follow up (*TE* = -.39, *se* = .04, *p* < .00001), a significant Direct Effect (*DE* = -.33, *se* = .04, *p* < .00001) and a significant Indirect Effect (*IE* = -.06, *se* = .03, *Lower level CI* = -.14, *Upper level CI* = -.01). Thus, social acceptance partially mediated the relationship between child report depressive symptoms at time 1 and social risk-taking at time 2. Whilst the beta for depressive symptoms decreases once the mediator (social acceptance) is added to the model, it remains significant. Figure 4.1 presents the partial mediation model and betas.

The mediation model was also completed with maternal report depressive symptoms at time 1 as the predictor variable. Controlling for the effect of time 1 social risk-taking (*b*= .55, *se* = .10, *p* <.0001), maternal report depressive symptoms had a significant Total Effect (TE) on social risk-taking at follow up (*TE* = -.45, *se* = .09, *p* < .00001), a significant Direct Effect (*DE* = -.41, *se* = .07, *p* < .00001) and a non-significant Indirect Effect (*IE* = -.04, *se* = .05, *Lower level CI* = -.15, *Upper level CI* = .03). Thus, social acceptance did not mediate the relationship between maternal report depressive symptoms at time 1 and social risk-taking at time 2 as the Indirect Effect is not significant.

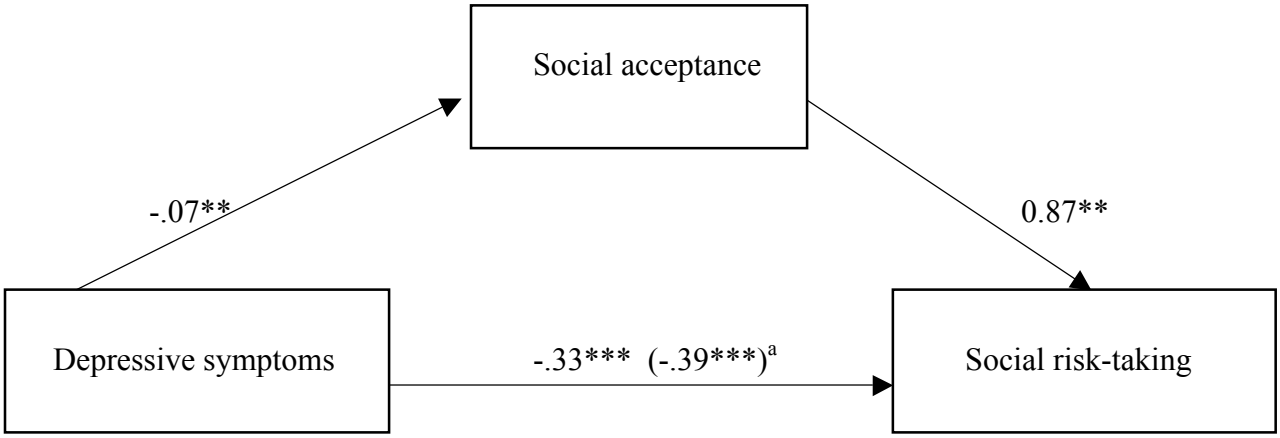


Figure 4.1 Social acceptance at baseline mediates the relationship between baseline child report depressive symptoms and 6-month follow up social risk-taking.

Note: Betas used are from the bootstrapping analysis. [a] Two betas are presented for depressive symptoms, with the mediator in the model (and without the mediator).

* $p < .05$ ** $p < .01$ *** $p < .001$

Discussion

This study examined the relationship between social risk-taking and depressive symptoms in early adolescents. Previous research has shown an association between risk-taking behaviour on experimental paradigms and depression, as well as social interactions and depression. However, the directionality of these relationships is unclear. Furthermore, bringing together the risk factors of social interactions and risk-taking and examining how they collectively influence the development of depressive symptoms, provides further insight into the mechanisms behind adolescent depression. The current study utilised a longitudinal design, measuring social risk-taking, depressive symptoms and social interactions at two time points, six months apart.

While controlling for the level of depressive symptoms at time 1, social risk-taking at time 1 predicted increased depressive symptoms at time 2. In contrast, when controlling for social risk-taking at time 1, depressive symptoms at time 1 predicted reduced social risk-taking at time 2. Although these findings appear to be contradictory, together they present a potential insight into the differing mechanisms behind the development of depression in adolescence.

Firstly, the finding that increased depressive symptoms predicted reduced social risk-taking provides support to the social risk hypothesis of depression. This model suggests that a depressed mood developed so that individuals could avoid social situations which may lead to them being excluded (Allen & Badcock, 2003). Specifically, that increased depression is a way for individuals to deal with social challenges by heightening sensitivity for suggestions of risk in social situations, thus leading to risk avoidance.

In contrast, the second main finding, that increased social risk-taking predicted increased depressive symptoms, appears to be in contrast to previous theories. For example, it has been suggested that a reduction in social risk-taking during the adolescent period can lead to a suppression of the reward system (Davey et al., 2008). If this suppression then

continues for an extended period, it is possible that depression can follow. Why instead might increased, rather than decreased, social risk-taking predict depressive symptoms as seen in the current study? It is possible that taking social risks may not always lead to a positive experience. For example, approaching a new classmate, or attending a party where the adolescent does not know anyone may lead to social victimisation. Experiencing social victimisation may also lead to a suppression of the reward system, as theorised by Davey et al., (2008), which may contribute to the development of increased depressive symptoms. As this is the first study to examine the direction of the relationship between social risk-taking and depression, the current findings are exploratory. Further research is needed, examining the temporal relationship between social risk-taking and depression, as well as following the development of depressive symptoms into depressive disorders.

Expanding on the reported finding of increased depressive symptoms predicting decreased social risk-taking, other possible mediating factors were examined. In particular, social acceptance at time 1 was also found to be significantly related to social risk-taking at time 2, with increased social acceptance predicting increased social risk-taking. A mediation analysis found that social acceptance partially mediated the relationship between depressive symptoms and social risk-taking. Increased depressive symptoms at time 1 were associated with decreased social acceptance at time 1 and decreased social risk-taking at time 2. Previous studies have also shown that depression is associated with decreased social acceptance or a negative response from peers. For example, in response to depressed adolescents' behaviours, a decrease in positive behaviour from a partner has been shown when compared to healthy controls (Heller & Tanaka-Matsumi, 1999). It has also been theorised that individuals with depressive symptoms may also experience less acceptance from peers because of increased reassurance seeking (Coyne, 1976). Thus, the partial mediation demonstrated in the current finding is in line with previous research but it is the

first to show a relationship between depressive symptoms, social acceptance and social risk-taking.

It was hypothesised that peer victimisation would also be a potential mediator of the relationship between social risk-taking and depressive symptoms. Although relational victimisation was significantly negatively correlated with social risk-taking at time 2, when social risk-taking at time 1 was controlled for, this relationship was no longer significant. Neither overt nor relational victimisation were significantly associated with later depressive symptoms. This is an interesting finding when compared to previous research. Others have found that experiencing victimisation can both predict depression (e.g. La Greca & Harrison, 2005) and occur at higher rates in those already experiencing depression (e.g. Hawker & Boulton, 2000). However, most of the previous research has examined the concepts of loneliness and peer rejection, rather than outright peer victimisation (e.g. Panak & Garber, 1992). Perhaps it is an individual's perception of how their peers view them, and whether they feel socially accepted that is most closely linked to the development and maintenance of depression. As the adolescent was the only informant for the current study, it was not possible to examine this. Future research could also gather information from others such as peers or a teacher about whether a participant is accepted or victimised by their peers. Nonetheless, previous research has shown that when comparing children's perceived versus actual peer acceptance, their perception, rather than actual dislike by peers, was most directly linked to their functioning (Zimmer–Gembeck, Hunter, & Pronk, 2007). The use of a longitudinal design in the current study has allowed for initial exploration of the complicated relationship between depressive symptoms, social experiences and social risk-taking in early adolescents.

The development of a new questionnaire to measure the concept of social risk-taking is an exciting advance in the current study. Previous research has examined risk-taking and reward-seeking and the relationship of these behaviours with depression using computer

based tasks, such as the BART (e.g. Morris, et al., 2017; Chapter 3). However, it is also important to consider real-world risk-taking, and how this is related to the development or maintenance of depression in adolescents. The use of vignettes asking participants how they would respond in real-world social situations was able to capture the overall level of social risk participants were willing to take, as well as demonstrating a reciprocal relationship with depressive symptoms. Future research comparing participant responses on the SRTQ with their performance on previously studied risk-taking measures such as the BART (Lejuez et al., 2002) would provide information on the potential overlap between social risk-taking and risk-taking on a gambling style task.

However, using a new measure also raises a number of potential limitations. For example, the reliability and construct validity have not yet been fully examined in the SRTQ. Importantly, the significant associations with depressive symptoms and social acceptance provide some initial support for the construct validity of the measure. Other limitations include the small amount of missing data across the participants, and as this study examined associations only, we are unable to make strong claims about causation. Also, the generalisability of the results to the general adolescent population is not completely clear. The participants mostly identified as being from a Western based culture, and were mainly from middle to high income families. Further, initially at age 3.5-4.5 years, the participants in this study were recruited based on the temperament trait of behavioural inhibition (BI). Only those classified as highly inhibited or highly uninhibited were included in the baseline assessments.

Also, although the SRTQ provides a real-world example of risk-taking and reward-seeking behaviour in social situations, it is not possible to separate motivation to seek the social reward and the risk-taking behaviour reported by the participant. This is particularly important when examining risk-taking and reward-seeking in the development of depression as reward-seeking in particular appears to be specifically associated with depression

(Sherdell, Waugh, & Gotlib, 2012). Therefore, being able to measure this behaviour alone may be important when examining the mechanisms behind the development of depression. Future research may wish to expand on the current measure by including a rating of how highly a participant rates the potential social reward in each situation, or by examining neural responses during the completion of the questionnaire.

Finally, depressive symptoms were explored in the current study, rather than focusing on clinical diagnoses. As the participants ranged from 11 to 13 years of age, they had not yet reached the peak adolescent period for the development of depressive disorders (Hankin et al., 1998). Examining depressive symptoms allows for an exploration of those at risk for depression prior to its development. Expanding on the current findings by following this group of early adolescents into the high risk age category of 15 to 18 years of age would be beneficial as mid-adolescence is also the peak time for risk-taking behaviour (Steinberg, 2008). This would allow for an exploration of whether the same association between social risk-taking and depression continues as the risk for both increases.

The current study provides an examination of social risk-taking and depressive symptoms in early adolescents. A novel measure was used to examine social risk-taking and reward-seeking in the context of adolescent depression. Social risk-taking and depressive symptoms had a bidirectional relationship over a six-month period with increased social risk-taking predicting increased depressive symptoms, and increased depression predicting decreased social risk-taking. Although this first finding is not directly in line with previous research, it suggests that social risk-taking may have a negative effect on adolescents' mood. When possible mediators were explored, it was found that perceived social acceptance partially mediated the relationship between depressive symptoms and social risk-taking, such that increased depression was associated with decreased social acceptance and decreased later social risk-taking. This suggests a possible mechanism by which depressive symptoms might impact social experiences for early adolescents.

References

Allen, N. B., & Badcock, P. B. (2003). The Social Risk Hypothesis of Depressed Mood: Evolutionary, psychosocial, and neurobiological perspectives. *Psychological Bulletin, 129*(6), 887-913. doi:http://dx.doi.org/10.1037/0033-2909.129.6.887

Angold, A., Costello, E., Erkanli, A., & Worthman, C. (1999). Pubertal changes in hormone levels and depression in girls. *Psychological Medicine, 29*(05), 1043-1053.

Angold, A., Costello, E. J., Messer, S. C., Pickles, A., Winder, F., & Silver, D. (1995). Development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *International Journal of Methods in Psychiatric Research*(5), 237-249.

Brinkmann, K., Franzen, J., Rossier, C., & Gendolla, G. H. (2014). I don't care about others' approval: Dysphoric individuals show reduced effort mobilization for obtaining a social reward. *Motivation and Emotion, 38*(6), 790-801.

Brody, A. L., Saxena, S., Silverman, D. H., Fairbanks, L. A., Phelps, M. E., Huang, S.-C., Wu, H. M., Maidment, K., Baxter, L. R., & Alborzian, S. (1999). Brain metabolic changes in major depressive disorder from pre-to post-treatment with paroxetine. *Psychiatry Research: Neuroimaging, 91*(3), 127-139.

Costello, E., Erkanli, A., & Angold, A. (2006). Is there an epidemic of child or adolescent depression? *Journal of Child Psychology and Psychiatry, 47*(12), 1263-1271.

Costello, E. J., & Angold, A. (1988). Scales to assess child and adolescent depression: checklists, screens, and nets. *Journal of the American Academy of Child & Adolescent Psychiatry, 27*(6), 726-737.

Coyne, J. C. (1976). Toward an interactional description of depression. *Psychiatry, 39*(1), 28-40.

Crick, N. R. (1995). Relational aggression: The role of intent attributions, feelings of distress, and provocation type. *Development and Psychopathology, 7*(02), 313-322.

Crick, N. R. (1997). Engagement in gender normative versus nonnormative forms of aggression: Links to social–psychological adjustment. *Developmental Psychology, 33*(4), 610.

Davey, C. G., Yucel, M., & Allen, N. B. (2008). The emergence of depression in adolescence: Development of the prefrontal cortex and the representation of reward. *Neuroscience and Biobehavioral Reviews, 32*(1), 1-19. doi:http://dx.doi.org/10.1016/j.neubiorev.2007.04.016

Dirks, M. A., Suor, J. H., Rusch, D., & Frazier, S. L. (2014). Children's responses to hypothetical provocation by peers: Coordination of assertive and aggressive strategies. *Journal of Abnormal Child Psychology, 42*(7), 1077-1087.

Enders, C. K. (2001). A primer on maximum likelihood algorithms available for use with missing data. *Structural Equation Modeling, 8,* 128-141. doi: 10.1207/S15328007SEM0801_7

Feder, A., Nestler, E. J., & Charney, D. S. (2009). Psychobiology and molecular genetics of resilience. *Nature Reviews Neuroscience, 10*(6), 446-457.

Forbes, E. E., & Dahl, R. E. (2005). Neural systems of positive affect: Relevance to understanding child and adolescent depression? *Development and Psychopathology, 17*(3), 827-850. doi:http://dx.doi.org/10.1017/S095457940505039X

Forbes, E. E., Hariri, A. R., Martin, S. L., Silk, J. S., Moyles, D. L., Fisher, P. M., Brown, S. M., Ryan, N. D., Birhamer, B., Axelson, D. A., & Dahl, R. E. (2009). Altered striatal activation predicting real-world positive affect in adolescent major depressive disorder. *American Journal of Psychiatry*.

Forbes, E. E., Shaw, D. S., & Dahl, R. E. (2007). Alterations in Reward-Related Decision Making in Boys with Recent and Future Depression. *Biological Psychiatry, 61*(5), 633-639. doi:http://dx.doi.org/10.1016/j.biopsych.2006.05.026

Galen, B. R., & Underwood, M. K. (1997). A developmental investigation of social aggression among children. *Developmental Psychology, 33*(4), 589.

Ge, X., Conger, R. D., & Elder, G. H., Jr. (2001). Pubertal transition, stressful life events, and the emergence of gender differences in adolescent depressive symptoms. *Developmental Psychology, 37*(3), 404-417. doi:http://dx.doi.org/10.1037/0012-1649.37.3.404

Green, H., McGinnity, Á., Meltzer, H., Ford, T., & Goodman, R. (2005). *Mental health of children and young people in Great Britain, 2004*: Palgrave Macmillan Basingstoke.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology, 107*(1), 128.

Harter, S. (1988). *Self-perception profile for adolescents*. University of Denver.

Harter, S. (1998). The development of self-representations. In W. Damon & N. Eisenberg (Eds.), *Handbook of child psychology, 5th ed.* (Vol. 3. Social, emotional, and personality development, pp. 553-617). Hoboken, NJ, US: John Wiley & Sons Inc.

Hawker, D. S., & Boulton, M. J. (2000). Twenty years' research on peer victimization and psychosocial maladjustment: A meta-analytic review of cross-sectional studies. *Journal of Child Psychology and Psychiatry, 41*(4), 441-455.

Hayes, A. F. (2012). PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling: University of Kansas, KS.

Heller, M. C., & Tanaka-Matsumi, J. (1999). A sequential analysis of depressive behaviors within adolescent peer interactions. *Journal of Psychopathology and Behavioral Assessment, 21*(3), 249-273.

Hudson, J. L., Dodd, H. F., & Bovopoulos, N. (2011). Temperament, family environment and anxiety in preschool children. *Journal of Abnormal Child Psychology: An*

official publication of the International Society for Research in Child and Adolescent Psychopathology, 39(7), 939-951.

Hughes, J. N., Webster-Stratton, B. T., & Cavell, T. A. (2004). Development and validation of a gender-balanced measure of aggression-relevant social cognition. *Journal of Clinical Child and Adolescent Psychology, 33*(2), 292-302.

Kessler, R. C., Avenevoli, S., & Ries Merikangas, K. (2001). Mood disorders in children and adolescents: An NIMH perspective. *Biological Psychiatry, 49*(12), 962-969.

La Greca, A. M., & Harrison, H. M. (2005). Adolescent peer relations, friendships, and romantic relationships: Do they predict social anxiety and depression? *Journal of Clinical Child and Adolescent Psychology, 34*(1), 49-61.

Ladd, G. W., & Ettekal, I. (2013). Peer-related loneliness across early to late adolescence: Normative trends, intra-individual trajectories, and links with depressive symptoms. *Journal of Adolescence, 36*(6), 1269-1282.

Lejuez, C., Read, J. P., Kahler, C. W., Richards, J. B., Ramsey, S. E., Stuart, G. L., Strong, D. R., & Brown, R. A. (2002). Evaluation of a behavioral measure of risk-taking: The Balloon Analogue Risk Task (BART). *Journal of Experimental Psychology: Applied, 8*(2), 75-84. doi:http://dx.doi.org/10.1037/1076-898X.8.2.75

Little, R. J. (1988). A test of missing completely at random for multivariate data with missing values. *Journal of the American Statistical Association*, *83*(404), 1198-1202.

Marshall, W., & Tanner, J. (1970). Variations in the pattern of pubertal changes in boys. *Archives of Diseases in Childhood, 45*(239), 13-23. doi:http://dx.doi.org/10.1136/adc.45.239.13

Marshall, W. A., & Tanner, J. M. (1969). Variations in pattern of pubertal changes in girls. *Archives of Disease in Childhood, 44*(235), 291-303.

Mendle, J., Harden, K., Brooks-Gunn, J., & Graber, J. A. (2010). Development's tortoise and hare: Pubertal timing, pubertal tempo, and depressive symptoms in boys and

girls. *Developmental Psychology, 46*(5), 1341-1353. doi:http://dx.doi.org/10.1037/a0020205

Morris, T. M., Hudson, J. L., & Dodd, H. F. (2017). *Behavioural inhibition in childhood and the development of adolescent depression: The role of puberty and risk-taking.* Manuscript in preparation.

Moulds, M. L., Kandris, E., Starr, S., & Wong, A. C. (2007). The relationship between rumination, avoidance and depression in a non-clinical sample. *Behaviour Research and Therapy*, *45*(2), 251-261.

Nolan, S. A., Flynn, C., & Garber, J. (2003). Prospective relations between rejection and depression in young adolescents. *Journal of Personality and Social Psychology, 85*(4), 745.

Panak, W. F., & Garber, J. (1992). Role of aggression, rejection, and attributions in the prediction of depression in children. *Development and Psychopathology, 4*(01), 145-165.

Pellegrini, A. D., & Bartini, M. (2000). An empirical comparison of methods of sampling aggression and victimization in school settings. *Journal of Educational Psychology, 92*(2), 360-366. doi:10.1037/0022-0663.92.2.360

Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments, & Computers, 36*(4), 717-731.

Preacher, K. J., Rucker, D. D., & Hayes, A. F. (2007). Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. *Multivariate Behavioral Research, 42*(1), 185-227.

Prinstein, M. J., Boergers, J., & Vernberg, E. M. (2001). Overt and relational aggression in adolescents: Social-psychological adjustment of aggressors and victims. *Journal of*

Clinical Child Psychology, 30(4), 479-491. doi:http://dx.doi.org/10.1207/S15374424JCCP3004_05

Rao, U., Daley, S. E., & Hammen, C. (2000). Relationship between depression and substance use disorders in adolescent women during the transition to adulthood. *Journal of the American Academy of Child & Adolescent Psychiatry, 39*(2), 215-222.

Rubin, K. H., Burgess, K. B., Kennedy, A. E., & Stewart, S. L. (2003). Social withdrawal in childhood. In E. J. M. R. A. Barkley (Ed.), *Child Psychopathology, 2nd ed* (pp. 372-406). New York, NY, US: Guilford Press.

Rubin, K. H., Coplan, R. J., Fox, N. A., & Calkins, S. D. (1995). Emotionality, emotion regulation, and preschoolers' social adaptation. *Development and Psychopathology, 7*(1), 49-62. doi:http://dx.doi.org/10.1017/S0954579400006337

Sanson, A., Prior, M., Garino, E., Oberklaid, F., & Sewell, J. (1987). The structure of infant temperament: Factor analysis of the Revised Infant Temperament Questionnaire. *Infant Behavior and Development, 10*(1), 97-104.

Scheffer, J. (2002). Dealing with missing data. Research Letters in the Information and Mathematical Sciences, 3, 153-160. Retrieved from http://equinetrust.org.nz/massey/fms/Colleges/College%20of%20Sciences/IIMS/RLIMS/Volume03/Dealing_with_Missing_Data.pdf

Schmitz, K. E., Hovell, M. F., Nichols, J. F., Irvin, V. L., Keating, K., Simon, G. M., Gehrman, C., & Jones, K. L. (2004). A validation study of early adolescents' pubertal self-assessments. *Journal of Early Adolescence, 24*(4), 357-384. doi:10.1177/0272431604268531

Smoski, M. J., Lynch, T. R., Rosenthal, M., Cheavens, J. S., Chapman, A. L., & Krishnan, R. R. (2008). Decision-making and risk aversion among depressive adults. *Journal*

of Behavior Therapy and Experimental Psychiatry, 39(4), 567-576. doi:http://dx.doi.org/10.1016/j.jbtep.2008.01.004

Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review, 28*(1), 78-106.

Steinberg, L., & Morris, A. S. (2001). Adolescent development. *Journal of Cognitive Education and Psychology, 2*(1), 55-87.

Swann, W. B., Stein-Seroussi, A., & Giesler, R. B. (1992). Why people self-verify. *Journal of Personality and Social Psychology, 62*(3), 392.

Uhrlass, D. J., Crossett, S. E., & Gibb, B. E. (2008). Self-perceived competence, relational victimization, and children's depressive symptoms: Evidence for a sex-specific vulnerability-stress model. *International Journal of Cognitive Therapy, 1*(4), 284-297. doi:http://dx.doi.org/10.1521/ijct.2008.1.4.284

Williams, K. D., Cheung, C. K., & Choi, W. (2000). Cyberostracism: effects of being ignored over the Internet. *Journal of Personality and Social Psychology, 79*(5), 748.

Wilson, K., Asbridge, M., Kisely, S., & Langille, D. (2010). Associations of risk of depression with sexual risk-taking among adolescents in Nova Scotia high schools. *Canadian Journal of Psychiatry-Revue Canadienne De Psychiatrie, 55*(9), 577-585.

Zimmer–Gembeck, M. J., Hunter, T. A., & Pronk, R. (2007). A model of behaviors, peer relations and depression: Perceived social acceptance as a mediator and the divergence of perceptions. *Journal of Social and Clinical Psychology, 26*(3), 273-302.

Next is the final empirical paper, “Reward-Seeking Behaviour on an Effort-Reward Task and Symptoms of Depression in Early Adolescents”. Chapters 2, 3 and 4 examined risk-taking behaviour utilising measures that were unable to separate out risk-taking and reward-seeking behaviour. Reward-seeking behaviour has been hypothesised to be a unique risk factor for the development of depression. Chapter 5 builds on the previous findings by examining reward-seeking behaviour, without the confound of risk-taking, and how it relates to depressive symptoms in early adolescents. In particular, whether differences in motivation to seek rewarding stimuli are associated with depressive symptoms. As the primary focus of the paper was the examination of the adapted effort-reward task, to maximise statistical power and clarity, behavioural inhibition (BI) and pubertal status were not included as variables. The data for this paper were collected at a single time point, therefore the clinician measure of depressive symptoms was available and is included alongside the Short Mood and Feeling Questionnaire, parent and child report.

Chapter Five

Reward-Seeking Behaviour on an Effort-Reward Task and Symptoms of Depression in Early Adolescents

Talia M. Morris, Jennifer L. Hudson, & Helen F. Dodd

Abstract

Reductions in reward-seeking behaviour have been identified in those at risk for depression and for those experiencing depression. Previous research has utilised reward-seeking tasks that confound reward-seeking with risk-taking as there is the potential for negative feedback when rewards are sought. The current pilot study examines the performance of early adolescents with and without depressive symptoms (N = 41) on an adapted effort-reward task, a computerised measure of reward-seeking behaviour. The task successfully captured reward-seeking as evidenced by increased motivation to seek rewarding cartoon stimuli. However, there was no significant difference between participants with or without depressive symptoms. This pilot study provides initial information about early adolescents' reward-seeking behaviour and suggests that the task adapted for the purposes of this paper may be a useful tool for examining reward-seeking in adolescents.

Previous research has examined the risk factor of diminished reward-seeking in depression. For example, adults with depression have been shown to be less willing to expend effort to gain a reward than healthy controls (Treadway, Bossaller, Shelton, & Zald, 2012). Similarly, adolescents with a familial risk for developing depression show a blunted neural reward response in a computerised task compared to those with no familial risk (Weinberg, Liu, Hajcak, & Shankman, 2015). However, many of the preceding studies have used tasks that examine reward-seeking but also include negative feedback in response to risk-taking. For example, the Balloon Analogue Risk Task (BART: Lejuez et al., 2002) involves pumping up a virtual balloon, with each pump gaining a point. However, the balloon can explode at any time; if this happens, participants lose their points. Thus, the BART involves risk as well as the potential for reward. The BART has been criticised for not differentiating between the cognitive components that affect performance, for example sensitivity to reward and risk attitudes (Lejuez et al., 2002). Another example, the Social Risk-taking Questionnaire (SRTQ; Morris, Hudson, & Dodd, 2017; Chapter 4), provides a real world example of risk-taking and reward-seeking behaviour with participants responding to a social situation involving risk (e.g. social rejection) that could lead to a rewarding social interaction. Again however, it is not possible to separate motivation to seek the social reward and the risk-taking behaviour reported by the participant. As reward-seeking in particular appears to be specifically associated with depression (Sherdell, Waugh, & Gotlib, 2012), being able to measure this behaviour alone may be important when examining the mechanisms behind the development of depression.

It has been theorised that anxiety and depression have distinct associations with risk-taking and reward-seeking behaviour. Greater risk avoidant behaviour has been demonstrated in anxious adult participants compared to those with a mood disorder (Maner et al., 2007). Furthermore, when controlling for negative affect, a common feature of both anxiety and depression (Clark & Watson, 1991), the correlation between anxiety symptoms

and risk-taking remained unchanged (Maner et al., 2007). This suggests that risk avoidance may be specific to the development of anxiety rather than depression. In contrast, when considering reward-seeking behaviour, anxiety is associated with increased reward-related functioning in the striatum in adolescents (Guyer et al., 2012) whereas reduced striatal functioning in response to reward has been shown in depression for adults (e.g. Epstein et al., 2006). These findings therefore suggest that distinct responses to rewarding stimuli can be seen in anxiety and depression.

Reward-seeking behaviour has previously been examined in the effort-reward task, which unlike the BART and SRTQ, does not confound reward-seeking and risk-taking. The effort-reward task was originally created by Waugh and Gotlib (2008) as a means of separating the 'liking' of a reward from the effort participants are willing to exert to obtain the reward. This computer task involves manipulating the effort required for participants to view a reward (humorous vs. non-humorous cartoon) via a square-clicking task. As the necessary effort to view a humorous cartoon increases, participants are less likely to select the reward of viewing the humorous cartoon. Individual differences in 'liking' of the cartoon predicted cartoon choice when low levels of effort were required but not at high levels of effort, indicating that participants were willing to exert some effort to view cartoons that they liked, but not a lot of effort. It would be expected that all rewards have a level of effort that participants are willing to exert to receive the reward, but that this level of effort varies by individual and reward.

A modified version of the effort-reward task has been used with depressed and non-depressed adult participants (Sherdell et al., 2012). Although the control and depressed groups did not differ overall in how much they liked the reward (humorous cartoons), the depressed group exerted less effort to seek the rewarding cartoon. Also, only in the control group did levels of reward liking predict the amount of effort participants were willing to exert to view the reward. This association was not present for depressed participants

suggesting that depressed participants did not base their cartoon choice on how much they liked the rewarding cartoon. This indicates that depression may be associated with atypical reward-seeking processes.

It is not currently clear whether this unusual reward-seeking is present in those at risk for depression. Work with other reward tasks has shown that those with depressive symptoms, rather than a clinical diagnosis, have reduced reward-seeking behaviour (e.g. Weinberg et al., 2015) providing some indication that reward-seeking may be atypical even in individuals at risk for depression. However, the tasks used in this previous work tend to confound reward-seeking with risk-taking as there is a potential for negative feedback. To our knowledge, no previous paper has examined reward-seeking in relation to depression risk in adolescents using a task that does not also include an element of risk-taking.

The effort-reward task has not previously been used with an adolescent sample. The first aim of the present study was therefore to adapt the effort-reward task and provide pilot data as to whether it is appropriate for assessing depression-related reward-seeking in adolescents. To be considered appropriate, a number of fundamental aspects of the task must be demonstrated. First, participants would need to like the humorous cartoons more than the non-humorous. By liking the humorous cartoons more, this would indicate that these stimuli are considered more rewarding than the non-humorous cartoons. In addition, there would need to be no difference in the level of liking of the humorous and non-humorous cartoons between depressive and non-depressive groups, in particular the humorous cartoons, so that the 'reward' was equal across groups. Third, the amount of effort participants are willing to exert to view the humorous cartoons should be greater when the alternative non-humorous cartoon also requires some effort (15 clicks) as compared to when the non-humorous cartoon requires no effort (0 clicks). Finally, to demonstrate that the square-clicking task was 'liked/disliked' equally for the different participant groups (for example depressive and non-depressive), there should be no

significant differences between groups on their 'liking' ratings of the effort task. If such a difference in liking the task were shown between groups, this would mean that any differences in levels of effort on the task could be due to differences in liking of the effort task rather than differences in motivation to seek the rewarding stimuli. If all four of these criteria are met then we can conclude that the task has been appropriately adapted for use examining the association between symptoms of depression and reward-seeking in adolescents.

Assuming that the above criteria were met, we hypothesised that participants with depressive symptoms would show reduced motivation to seek the rewarding cartoon when compared with non-depressive participants by having a lower number of clicks they are willing to exert overall for the humorous (rewarding) cartoons. Furthermore, in line with Sherdell et al. (2012), it was expected that for non-depressive participants, greater 'liking' of the humorous cartoons would be associated with increased motivation to exert effort to view the cartoons. In contrast, 'liking' and motivation were hypothesised to be unrelated in depressive participants.

To examine the utility of the task, we used a sample of early adolescents (aged 11-12 years) identified in childhood as being at risk of anxiety and depression based on temperament (that is, behavioural inhibition, BI). We do not explore any associations with BI so as to focus on the specific aims stated above. However, given that BI is associated with anxiety and depression, this is a relevant sample in which to pilot the new measure.

Method

Participants

Participants were originally recruited as part of a larger longitudinal study at the age of 3-4 years via local preschools and an advertisement in a free parenting magazine (see Hudson, Dodd, & Bovopoulos, 2011). The original sample included 202 children who were initially screened for the temperament trait BI. BI was initially assessed at baseline using the

mothers' report on the Approach Scale of the Short Temperament Scale for Children (Sanson, Prior, Garino, Oberklaid, & Sewell, 1987; STSC). Children scoring 1 standard deviation above or below the normative mean on the STSC were invited to take part in the full study. These participants were classified as BI or behaviourally uninhibited (BUI) respectively. Every participant who could be contacted was invited to take part in a follow up research session eight years later, with 137 participants agreeing to participate. All participants (N = 41) who took part in a follow up research session within the final six months recruitment took part in the present task.

The sample for the current study comprised of 41 early adolescents, 22 male, 19 female, aged approximately 12 years (M = 138.66 months, SD = 2.57). For 63% of the sample they described their ethnicity as Oceanic, with the majority of the remainder being European (20%). Compared to the larger overall follow-up sample of 137 participants, there were no significant differences between those who participated in the current study and those who did not on gender, ethnicity, family income or the variables of interest. There was a difference in age in months with those who participated in the subset sample on average two months younger than those who did not, t (115,1) = 3.54, p = .001.

Measures

Depressive symptoms. Three measures were used to assess depressive symptoms; a self-report measure, parent report measure and a clinician rated measure.

Short Mood and Feelings Questionnaire. To measure symptoms of depression as reported by the child and their mother the self-report (SMFQ) and parent-report (SMFQ-P) versions of the Short Mood and Feelings Questionnaire were used (Angold, Costello, Messer, & Pickles, 1995). The parent and child versions of the scale are matched closely and contain 11 items. Participants use a 3-point Likert scale (0-2) to report whether a specific phrase is indicative of their/their child's feelings and behaviour. For example, 'I

(s/he) felt miserable or unhappy'. In the full follow up sample internal consistency was good for child self-report; Cronbach's alpha = .86, and for parent report; Cronbach's alpha = .88.

Kiddie Schedule for Affective Disorders and Schizophrenia. To further assess depressive symptoms (clinician rated), the Depression Rating Scales of the Kiddie Schedule for Affective Disorders and Schizophrenia were used (KSADS-DRS; Kaufman et al., 1997). Participants and their mothers were interviewed using the KSADS, a semi-structured interview. It is one of the most often used diagnostic interviews with youths and has previously demonstrated good reliability and validity (Kaufman et al., 1997, Klein, Dougherty, & Olino, 2005). Although the KSADS was used to diagnose depression in the participants, none met full criteria for major depressive disorder. Given the age of the participants this low diagnosis rate is not unexpected. Instead, using the child and mother reports, the presence of depressive symptoms was examined. Participants who had at least one depressive symptom as reported on the KSADS were coded as 'symptom present' while those not meeting criteria were coded as 'symptom absent'. In the full follow up sample an inter-rater reliability analysis using the Kappa statistic was performed to determine consistency among the two raters. Agreement on KSADS-DRS symptom presence/absence ratings amongst raters was found to be Kappa = 0.71 ($p < .0001$).

Reward-seeking. To examine reward-seeking, we modified the task used by Sherdell and colleagues (2012) which is based on the effort-reward task (Waugh & Gotlib, 2008). The version used in the current study is that same as that used by Sherdell and colleagues but with the cartoon stimuli updated to be age-appropriate. In the effort-reward task, participants first rate how much they enjoy stimuli (cartoons) from 'reward' (i.e., humorous) and 'non-reward' (i.e. non-humorous) categories. They then make choices about how much effort they are willing to exert before viewing a cartoon from a selected group.

Reward stimuli. Single-panel cartoons are used as the stimuli in this task. Consistent with other rewarding stimuli, humorous and non-humorous cartoons have been shown to

activate mesolimbic reward areas of the brain (Mobbs, Greicius, Abdel-Azim, Menon, & Reiss, 2003). A total of 104 cartoons were used by Sherdell et al. (2012), with half of the cartoons humorous and the other half containing non-humorous captions which made the cartoon 'unfunny'. Sherdell et al. (2012)'s task was originally administered to adults, therefore a number of the humorous cartoons were not appropriate for early adolescents. Seventeen of the original 52 humorous cartoons from Sherdell and colleagues' (2012) task were deemed to be suitable for children and were retained. To replace the inappropriate cartoons, a range of age appropriate cartoons was assessed. Forty-seven age appropriate humorous cartoons were tested with 27 pilot participants who rated the cartoons from 1 to 10 on how humorous they found them. The 35 cartoons rated as most humorous across all pilot participants were included in the current version of the effort-reward task along with the original 17 humorous cartoons from Sherdell et al. (2012). All of the original non-humorous cartoons were age-appropriate and were included in the current study.

Humorous and non-humorous cartoons were presented to participants as belonging to two different 'decks' of cartoons. These decks were given the nonsense names of "LUM" and "GUP" to avoid influencing ratings or choice behaviour. The allocation of these two names to the humorous and non-humorous decks was counterbalanced across participants.

Reward 'liking'. Participants were then presented with the same 20 cartoons one at a time (along with their deck label) and asked to rate how much they liked the cartoon. A bipolar visual analogue scale was used along the bottom half of the screen from 0 pixels ('extremely disliked') to 1000 pixels ('extremely liked'). A white bar in the middle designated "neither liked nor disliked'.

Motivation. To calculate motivation, the amount of effort participants were willing to put in to view a cartoon from the preferred deck was measured. Thirty-six choice trials were included. Each choice trial began with a screen displaying two rectangles representing the 'LUM' and 'GUP' decks. A number was written on each rectangle to indicate the 'click-

cost' for viewing a cartoon from that deck. The 'click-cost' referred to how many times the participant would have to click on a 2 × 2 inch black square as it appeared in random locations on the screen before they were able to view the cartoon from the chosen deck. This square-clicking task was used to induce effort and is similar to those used in the past (Klein, Bhatt, & Zentall, 2005). Participants were asked to select which deck they wanted to view a cartoon from. Then, once the required number of clicks were completed, the participants viewed the cartoon from their chosen deck. Whilst viewing the cartoon, they were asked to rate how much they liked the cartoon using the aforementioned visual analogue scale.

The 'click-cost' for the humorous deck was always greater than for the non-humorous deck. While the non-humorous deck click-cost was always either 0 or 15 clicks, the humorous deck click-cost was decided by a random adjusting-amount algorithm adapted from Richards, Zhang, Mitchell, and de Wit (1999). Depending on the participant's previous choices, the algorithm was programmed to narrow the range of values from which the subsequent click-costs were chosen. As the trials progressed, the number of clicks associated with viewing a cartoon from the humorous deck was adjusted relative to the non-humorous deck until the range of the upper and lower bounds of the click-cost reached five clicks. The lower limit of this range represented the indifference point for the participants, the click-cost at which they were equally likely to choose either deck.

For each participant, two indifference points were determined (when the click cost for the non-humorous deck was 0 clicks or 15 clicks). This provided a measure of how willing a participant is to work for a reward when there is no "opt out" option, they must exert some effort regardless of choice. As per Sherdell et al. (2012), the two indifference points were used as a measure of a participant's motivation. Once the indifference points were established, randomly presented click counts were used until the full 36 trials were completed.

For five participants (out of 41), the algorithm was unable to calculate the participant's indifference point by the end of the 36 trials. In these cases, an approximate indifference point was calculated by adjusting the required range between the upper and lower bounds of the click-cost to be 10 clicks, rather than five, in line with previous research (Sherdell et al., 2012).

Procedure

All procedures for this study, including the consent procedures, were approved by the Macquarie University Human Ethics Committee. After completing the initial STSC phone screening, children who met criteria were invited to attend a 2-hour laboratory session at Macquarie University both at baseline and eight years later at follow up. At both laboratory sessions written consent was provided by the mothers for both themselves and their child's participation. Families were reimbursed $50 at both sessions and children received a small age appropriate gift.

At the follow up session, both mother and child completed the KSADS interview separately with a trained administrator. Prior to completing their interviews, the participants completed the effort-reward task. Additional observed tasks and questionnaires were completed at baseline and the follow up which are not presented here.

Analyses Plan

All analyses were conducted for the current study using SPSS (Version 22, IBM). For all analyses the Shapiro-Wilk, F_{max} and Levene's test statistics were used to test the assumptions of normality and homogeneity of variance. The assumptions for the mixed model analysis of variance (ANOVA) were not violated.

First we examined whether the cartoon task appeared to be appropriate for examining reward-seeking and depression symptoms in adolescents by examining whether the following four previously stated criteria were met: 1) participants should like the humorous cartoons more than the non-humorous; 2) there should be no difference between

KSADS groups on participants' liking of the cartoons, this is particularly important for the humorous cartoon; 3) participants should increase their effort to view the humorous cartoon as the click-cost for the non-humorous cartoon increased from 0 to 15 clicks; 4) there should be no difference in liking of the square clicking task between KSADS groups. The results for these criteria are reported first. Next we report whether motivation to view the rewarding humorous cartoon was associated with depressive symptoms. Finally we report whether relative liking of the humorous cartoons over the non-humorous cartoons influences the indifference point.

Results

Descriptive analyses

T-tests were used to examine the effect of gender and ethnicity on the SMFQ child and mother report, no significant associations were found (all *p*s > .05). Further, there were no significant differences between temperament groups, depressive symptoms or anxiety diagnosis with any of the demographic variables (ethnicity, gender, child age, maternal age, and family income; $p > .05$). Pearson's correlations were used to examine child age, maternal age, and family income on the SMFQ child and mother report. Only maternal age was found to be significantly negatively correlated with maternal report SMFQ, $r = -.18$, $p = .046$. Maternal age was controlled for in the relevant analyses. Means and standard deviations for all variables are reported in table 5.1.

Due to non-normally distributed data, the SMFQ child report was transformed using a log + 1 transformation before analyses were completed.

Table 5.1
Descriptive statistics for complete sample.

	Complete sample (n = 41)
Child age, Mean (SD)	11.55 (0.21)
Maternal age, Mean (SD)	44.97 (4.27)
Males, *n* (%)	22 (46.3)
Family income	
Low income, *n* (%)	6 (14.7)
Middle income, *n* (%)	11 (26.8)
High income, *n* (%)	22 (53.7)
Ethnicity	
Oceanic, *n* (%)	26 (63.4)
European, *n* (%)	8 (19.5)
Asian, *n* (%)	2 (4.9)

Overall Cartoon task statistics

Criteria 1 and 2.

A mixed between-within subjects ANOVA was conducted to compare participants' 'liking' ratings of the non-humorous and humorous cartoons and to assess whether KSADS groups (presence/absence) differed in 'liking' across the two different cartoon types (humorous and non-humorous).

There was a significant main effect for cartoon type on liking, Wilks' Lambda = .31, F (1,39) = 83.77, $p < .0001$, partial $\eta^2 = .68$, with both groups 'liking' the humorous cartoon more than the non-humorous cartoons (see Table 5.2), satisfying Criteria 1. The main effect comparing the two KSADS groups (KSADS depressive symptom presence and absence) was not significant, F (1,39) = 1.06, $p = .31$, partial $\eta^2 = .03$, but there was a significant interaction between depressive symptom presence and cartoon type, Wilks' Lambda = .90, F (1,39) = 4.54, $p = .04$, partial $\eta^2 = .10$. Examination of the parameter estimates demonstrates that there was a trend for participants with KSADS depressive symptoms to like the non-humorous cartoon less than those without KSADS depressive symptoms, β = 102.14, $t = 2.00$, $p = .05$. However, there was no difference for the level of liking of the humorous cartoons between groups ($p > .05$). See figure 5.1. Criteria 1 and 2 are therefore adequately met.

Table 5.2

Level of cartoon 'liking' in the different groups across the two cartoon decks

KSADS Group	Non-humorous	Humorous
Depressive*	337.71 (170.11)	702.48 (141.73)
Non-depressive *	439.85 (146.75)	666.84 (108.43)

*Mean (SD) *Depressive symptom presence or absence on the KSADS*

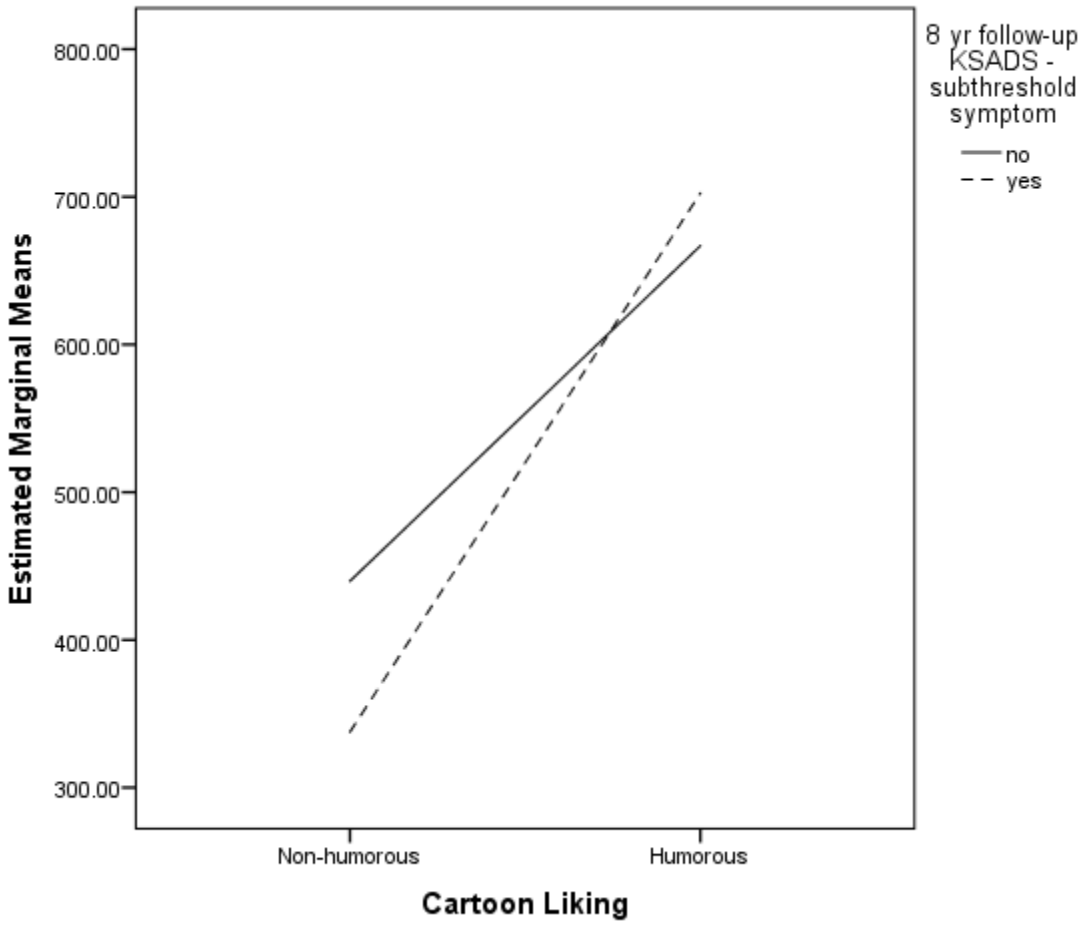


Figure 5.1. Differences in liking of the humorous and non-humorous cartoons across KSADS depressive symptoms groups

Further mixed model ANOVA's were conducted examining depressive symptoms as a continuous variable on the SMFQ. When child report on the SMFQ was utilised, a significant main effect of cartoon type was also demonstrated, however no main effect for depression nor interaction effect was found (Supplementary Tables 1-2; Appendix E). In contrast, when mixed ANOVAs, using between and within subjects variables, were conducted with the maternal report depressive symptoms on the SMFQ, the pattern of results showed no significant effects on depression or reward-seeking. Neither of the main effects were significant, nor was the interaction term (all p values > 0.05; see Supplementary Tables 1-2; Appendix E).

Criteria 3.

A paired samples t-test was conducted to examine whether participants were willing to increase their level of effort to view the humorous cartoon when the click-cost for the non-humorous cartoon was increased from 0 to 15 points. A significant difference was shown, with the indifference point increasing when the click-cost was set to 15 points (M = 28.08, SD = 16.37) compared to 0 points (M = 12.69, SD = 15.25), $t(38) = -5.39$, $p < .0001$. Criteria 3 was therefore met.

Criteria 4.

A t-test showed that there were no differences in participants' ratings of how much they liked the square-clicking task between children with and without depressive symptoms ($t(39) = -0.18$, $p = .86$). Criteria 4 was therefore met.

In addition, Pearsons correlations were conducted to examine whether there were differences in liking of the square-clicking task across child and maternal report depressive symptoms on the SMFQ. These were also non-significant (see Supplementary Tables 3; Appendix E).

Motivation/effort

A mixed ANOVA was conducted to assess the impact of KSADS group (depression symptom presence/absence), on the participants' level of effort across the two different non-humorous click-cost (0pt and 15pt). There was a significant main effect of non-humorous cartoon click-cost, F (1,37) = 30.39, $p < .0001$, partial $\eta^2 = .45$, as described above with reference to criteria 3 (see Table 5.3.). In contrast to hypotheses, the main effect of KSADS group was not significant, F (1,37) = .02, $p = .89$, partial $\eta^2 = .001$, suggesting no difference in the overall motivation to view the cartoons between the two KSADS groups. There was no significant interaction between KSADS group and non-humorous cartoon click-cost, Wilks' Lambda = .96, F (1,37) = 1.65, $p = .21$, partial $\eta^2 = .04$.

Table 5.3.
Level of effort participants were willing to take to view cartoons in the different groups across the two levels of click-cost for non-humorous cartoons

KSADS Group	0pt Click-cost	15pt Click-cost
Depressive*	10.42 (14.99)	31.25 (19.32)
Non-depressive*	13.70 (15.54)	26.67 (15.06)

*Mean (SD) *Depressive symptom presence or absence on the KSADS*

Further mixed model ANOVAs were conducted to assess the impact of depressive symptoms as a continuous variable using the SMFQ, on the participants' level of effort across the two different non-humorous click-cost (0pt and 15pt). The child report SMFQ demonstrated the same pattern of results as the KSADS analyses (see Appendix E). In contrast, the pattern of results showed non-significance across all effects when maternal report of depressive symptoms on the SMFQ (and maternal age as a control factor) were examined as predictors of the participants' level of effort across the two different non-

humorous click-cost (0pt and 15pt). Neither of the main effects were significant, nor was the interaction term (p's > .05; see Supplementary Tables 4-5; Appendix E).

Relation Between Reward Liking and Motivation

To examine the relationship between participants' reward liking and their motivation to view the reward, multiple regression analyses were conducted. A reward liking difference score was created to capture how much participants liked the humorous cartoons over and above their liking of the non-humorous cartoons, with the participants' liking of the non-humorous cartoons deducted from their liking of the humorous cartoons. Reward liking, KSADS group and their interaction were included as independent variables whereas indifference point (when the non-humorous deck was set to 15 clicks) was the dependent variable. Reward liking was centred to reduce collinearity.

There was no significant main effect of KSADS group (β = .03, t = .06, p = .96) on motivation. There was also no main effect of reward liking (β = .02, t = .14, p = .89), and no significant interaction (β = .24, t = .45, p = .66); R^2 = .08, $F(3,35) = 1.00$, p = .41.

Furthermore, when the continuous independent depressive symptom variables were included instead of group (SMFQ child and SMFQ mother) the pattern of the results was the same: neither of the main effects were significant, nor was the interaction term (all p values > .05, see Supplementary Tables 6-7; Appendix E).

Discussion

Atypical reward-seeking behaviour has been shown to be present in adults experiencing depression, or at increased risk for depression (e.g. Treadway et al., 2012). However, the majority of previous studies have used tasks that confound reward-seeking with risk-taking, as the reward-seeking activity can lead to negative feedback. To clarify the relative importance of reward-seeking and risk-taking in depression it is important to be able to explore reward-seeking alone. The effort-reward task (Waugh & Gotlib, 2008) has been developed for this purpose but has only been used thus far with adults.

The current study is the first to observe the performance of early adolescents on the adapted effort-reward task (Sherdell et al., 2012). The first aim was to demonstrate the validity of the effort-reward task for examining reward-seeking in relation to symptoms of depression in early adolescents by evaluating four criteria. This aim was achieved. First, participants liked the humorous cartoons more than the non-humorous, suggesting that the age appropriate cartoons used in the current study were rewarding for participants. Second, the results suggest there was no difference in the level of overall liking of the humorous cartoons between depressive and non-depressive groups both when using the KSADS and SMFQ measures. Although there was some indication that participants showing symptoms of depression on the KSADS liked the non-humorous cartoons less than those not showing any symptoms of depression on the KSADS. Third, the early adolescents were willing to put in more effort to view humorous cartoons when the non-humorous cartoon click cost was increased from 0 to 15 clicks. Finally, the square-clicking task was "disliked" equally by both depressive and non-depressive groups. Thus, using the four criteria, the current study provides some support of previous findings that this task is able to elicit reward-seeking behaviour (Sherdell et al., 2012). Furthermore, the current findings suggest that the adapted effort-reward task, which had previously been used with adults, may also appropriate for use with early adolescents. Future research could validate the task further across a range of age groups but it offers promise as a measure of reward-seeking behaviour.

When considering the suitability of the adapted effort-reward task, it is necessary to take note of the unexpected finding of an interaction between reward liking and depressive symptoms. As stated above, depressive participants (as measured by the KSADS) reported lower levels of liking of the non-humorous cartoons than non-depressive participants. Importantly for criteria 2, there was no significant difference in their liking of the humorous cartoons. Nonetheless, the lower ratings of liking of the non-humorous cartoon in the depressive group needs further consideration. No difference was found between the

depressive groups on liking of the humorous cartoons, nor for the square-clicking task. Thus, this finding appears to be uniquely related to liking of the non-humorous cartoons, rather than a difference in liking of the effort-reward task as a whole. Upon further examination, it is possible that the non-humorous cartoon can be conceptualised as a negative stimuli. The participant has exerted a level of effort on the square-clicking task and then received the non-reward of a non-humorous cartoon. Previous work has shown an increased neural response to negative stimuli in depressed adult participants compared to non-depressed controls (e.g. Mueller, Pechtel, Cohen, Douglas, & Pizzagalli, 2015; Tucker, Luu, Frishkoff, Quiring, & Poulsen, 2003). Interestingly, this relationship was shown to be inversely related to symptom severity with decreased depression severity associated with increased neural sensitivity for negative feedback (Mueller et al., 2015). Thus, the difference in liking of the non-humorous cartoon across depressive groups in the current study may be partially due to the subclinical level of depression reported by the participants being associated with an increased sensitivity for negative feedback. However, it is important to keep in mind that this relationship was only found at trend level, and only for the KSADS groups, not the SMFQ child or maternal report. Thus, at this stage it does not present a major concern regarding the utility of the adapted effort-reward task with early adolescents.

The second aim of the current study was to utilise the effort-reward task to examine differences in reward-seeking behaviour across groups of participants, in particular expanding on previous work with depressed and non-depressed adults (Sherdell, et al., 2012). The current study extended these findings by exploring differences in responses of early adolescents with and without symptoms of depression. When examining differences between participants, it was hypothesised that the depressive group would show reduced motivation to seek the reward of the humorous cartoons in comparison to the non-depressive group. However, the results showed that there was no significant difference on motivation to

view the rewarding cartoon. One possible reason the findings were not exactly as expected, could be due to fact that the participants did not yet meet full criteria for a depressive disorder. It is possible that the differences in motivation to seek the rewarding cartoon are not yet present at a sub clinical level of depression. Furthermore, as the participants were in early adolescence, the changes in reward-seeking behaviour linked to depression may not have yet manifested as previous research has demonstrated that reward-seeking increases into mid-adolescence (Steinberg, 2010). Further study could examine this probability, using the effort-reward task with adolescents with clinical depression and adults with sub clinical depressive symptoms.

Finally, following from previous work (Sherdell et al., 2012), it was expected that there would be an association between reward liking and motivation to seek the reward for non-depressed participants only. This association was not present for either group, suggesting that the number of clicks a participant was willing to make to view the humorous cartoon was not associated with how much they rated their liking of the cartoon. From this finding it can be suggested that when making their decision about how much effort they were willing to exert for the reward, the participants in this study did not consider how much they liked the rewarding stimulus, or instead that their liking of the cartoons fluctuated over the course of the task. It is possible that this finding is related to the age group of the participants, and how they make their decisions regarding rewards. Further examination of how different age groups make decisions on the effort-reward task will help to clarify this finding.

There are several strengths of the current study and a few limitations to consider. This was the first study to use the effort-reward task in an adolescent population and to examine the findings in relation to depressive symptoms. However, the current study provided a cross sectional consideration which prevents any discussion on the temporal nature of the relationship between depressive symptoms and reward-seeking. Future

research could utilise a longitudinal design to examine whether performance on the effort-reward task is stable across time, and further, whether it is predictive of later depression. As this was only a small pilot study, the sample size may have impacted on the significance of the findings. In addition, the sample were selected as being BI and BUI at age 4 so may not be entirely representative of the general population. Further investigation with a larger sample size selected from the general population or using a case control design could expand on these initial findings.

In conclusion, this is the first study to examine the adapted effort-reward task (Sherdell et al., 2012) using age appropriate stimuli in an early adolescent sample at risk for depression. Although there was no difference in motivation to seek reward based on symptoms of depression, a significant interaction demonstrated that those with depressive symptoms 'liked' the non-humorous cartoons less than those without depressive symptoms. The non-significant difference in motivation to view the rewarding cartoon based on symptoms of depression may be due to the age of the participants, or presence of depressive symptoms rather than a clinical disorder. Nevertheless, these initial findings suggest that the task is suitable for use with this age group and would be useful for future research examining reward-seeking in adolescence.

References

Angold, A., Costello, E. J., Messer, S. C., & Pickles, A. (1995). Development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *International Journal of Methods in Psychiatric Research*.

Australian Bureau of Statistics. (2008). *National Survey of Mental Health and Wellbeing: Summary of Results, 2007.* Cat. no. (4326.0). Canberra: ABS.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*(3), 316-336. doi:http://dx.doi.org/10.1037/0021-843X.100.3.316

Epstein, J., Pan, H., Kocsis, J. H., Yang, Y., Butler, T., Chusid, J., Hochberg, H., Murrough, J., Strohmayer, E., Stern, E. & Silbersweig, D.A. (2006). Lack of ventral striatal response to positive stimuli in depressed versus normal subjects. *American Journal of Psychiatry*.

Guyer, A. E., Choate, V. R., Detloff, A., Benson, B., Nelson, E. E., Perez-Edgar, K., Fox, N.A., Pine, D.S. & Ernst, M. (2012). Striatal functional alteration during incentive anticipation in pediatric anxiety disorders. *The American Journal of Psychiatry, 169*(2), 205-212.

Hudson, J. L., Dodd, H. F., & Bovopoulos, N. (2011). Temperament, family environment and anxiety in preschool children. *Journal of Abnormal Child Psychology: An official publication of the International Society for Research in Child and Adolescent Psychopathology, 39*(7), 939-951.

Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., Williamson, D. & Ryan, N. (1997). Schedule for affective disorders and schizophrenia for school-age children-present and lifetime version (K-SADS-PL): initial reliability and validity data. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(7), 980-988.

Klein, D. N., Dougherty, L. R., & Olino, T. M. (2005). Toward guidelines for evidence-based assessment of depression in children and adolescents. *Journal of Clinical Child and Adolescent Psychology, 34*(3), 412-432.

Klein, E. D., Bhatt, R. S., & Zentall, T. R. (2005). Contrast and the justification of effort. *Psychonomic Bulletin & Review, 12*(2), 335-339.

Lejuez, C., Read, J. P., Kahler, C. W., Richards, J. B., Ramsey, S. E., Stuart, G. L., Strong, D.R. & Brown, R. A. (2002). Evaluation of a behavioral measure of risk taking: The Balloon Analogue Risk Task (BART). *Journal of Experimental Psychology: Applied, 8*(2), 75-84. doi:http://dx.doi.org/10.1037/1076-898X.8.2.75

Maner, J. K., Richey, J., Cromer, K., Mallott, M., Lejuez, C. W., Joiner, T. E., & Schmidt, N. B. (2007). Dispositional anxiety and risk-avoidant decision-making. *Personality and Individual Differences, 42*(4), 665-675.

Mobbs, D., Greicius, M. D., Abdel-Azim, E., Menon, V., & Reiss, A. L. (2003). Humor modulates the mesolimbic reward centers. *Neuron, 40*(5), 1041-1048.

Morris, T. M., Hudson, J. L., & Dodd, H. F. (2017). *The relationship between social risk-taking and depression in adolescence: The mediating effect of perceived social acceptance.* Manuscript in preparation.

Mueller, E. M., Pechtel, P., Cohen, A. L., Douglas, S. R., & Pizzagalli, D. A. (2015). Potentiated processing of negative feedback in depression is attenuated by anhedonia. *Depression and Anxiety*, *32*(4), 296-305.

Richards, J. B., Zhang, L., Mitchell, S. H., & Wit, H. (1999). Delay or probability discounting in a model of impulsive behavior: Effect of alcohol. *Journal of the Experimental Analysis of Behavior, 71*(2), 121-143.

Sanson, A., Prior, M., Garino, E., Oberklaid, F., & Sewell, J. (1987). The structure of infant temperament: Factor analysis of the Revised Infant Temperament Questionnaire. *Infant Behavior and Development, 10*(1), 97-104.

Sherdell, L., Waugh, C. E., & Gotlib, I. H. (2012). Anticipatory pleasure predicts motivation for reward in major depression. *Journal of Abnormal Psychology*, *121*(1), 51.

Steinberg, L. (2010). A dual systems model of adolescent risk-taking. *Developmental Psychobiology, 52*(3), 216-224.

Treadway, M. T., Bossaller, N. A., Shelton, R. C., & Zald, D. H. (2012). Effort-based decision-making in major depressive disorder: a translational model of motivational anhedonia. *Journal of Abnormal Psychology, 121*(3), 553.

Tucker, D. M., Luu, P., Frishkoff, G., Quiring, J., & Poulsen, C. (2003). Frontolimbic response to negative feedback in clinical depression. *Journal of Abnormal Psychology*, *112*(4), 667.

Waugh, C. E., & Gotlib, I. H. (2008). Motivation for reward as a function of required effort: Dissociating the 'liking' from the 'wanting' system in humans. *Motivation and Emotion, 32*(4), 323-330.

Weinberg, A., Liu, H., Hajcak, G., & Shankman, S. A. (2015). Blunted neural response to rewards as a vulnerability factor for depression: Results from a family study. *Journal of Abnormal Psychology, 124*(4), 878.

Chapter Six

General Discussion

Thesis Summary

Rates of depression increase rapidly in adolescence, with the highest risk of first onset in the early teens and mid-20s (Kessler, Avenevoli, & Merikangas, 2001). Depression in adolescence is a strong predictor of suicide attempts (Lewinsohn, Rohde, & Seeley, 1994), and has been linked to later mental health disorders such as substance abuse (Birmaher et. al., 1996). Furthermore, adolescent depression has detrimental consequences including impaired school performance (Fröjd et al., 2008) and poorer quality of life (Zisook et al., 2007). Due to the negative impact of adolescent onset depression, it is necessary for developmental pathways to depression to be examined. This will allow for the identification of early risk factors, which may be targeted via prevention or early intervention programs (Lau & Eley, 2010).

This thesis focused on the complex nature of the development of depression in adolescence by examining both early childhood predictors of depression and risk factors that are particularly relevant to the developmental stage of adolescence. Adolescence is a time of substantial change, there is an intensified focus on peer relationships (Spear 2000), increases in risk-taking and reward-seeking (Steinberg, 2008), and the onset of puberty. Thus, it is important to focus on these specific areas of change to provide a deeper understanding of the development of depression in adolescence. Paper 1 examined the relationship between temperament and risk-taking in early childhood. This provided insight into the relationship between risk factors for adolescent depression early in life, important for early intervention. Following from this, Paper 2 examined the relationship between early temperament and depression in early adolescence, and considered the potential moderating role of risk-taking. The next study, Paper 3, utilised a novel measure of social risk-taking to examine the important area of peer interactions and social risk-taking in adolescence and how this relates to the development of depression. Finally, Paper 4 explored the specific role of reward-seeking in relation to depression in early adolescence using a novel task. The vast

majority of previous studies examining reward-seeking in depression have utilised tasks that also involve an element of risk, or negative feedback (e.g. Weinberg, Liu, Hajcak, & Shankman, 2015). This study was the first to examine reward-seeking without this confounding factor and its relation to depression in early adolescence.

The four studies in this thesis utilised a multi-factor context to examine important risk factors for the development of adolescent depression. This thesis aimed to examine salient factors in adolescence - specifically risk-taking and reward-seeking behaviours - in a social context in conjunction with other established risk factors for depression including temperament and social victimisation.

Review of Thesis Papers and Outcomes

Paper 1: Risk-taking and inhibitory control in behaviourally inhibited and disinhibited preschool children.

The temperament style behavioural inhibition (BI) has previously been linked to the development of depression (e.g. Jaffee et al., 2002). The mechanism by which BI may confer risk for depression is not clear. Paper 1 was the first study to examine whether BI and executive functioning, (i.e. the temperament trait effortful, or inhibitory control) predicted risk-taking behaviour in isolation or whether they interacted to predict risk-taking. It was hypothesised that BI children would take less risks on the Balloon Analogue Risk Task youth version (BART-Y; Lejuez et al., 2007) compared to uninhibited children and that their performance on an effortful control task would further interact with BI to predict risk-taking behaviour. Children high in inhibitory control were significantly more likely to score a higher number of points on the BART-Y suggesting that although children high in inhibitory control take risks, they are better able to balance the risk of the balloon popping with the prize at the end (i.e. the reward), compared to children with low inhibitory control. This finding was in line with previous research showing that executive functioning is associated with performance on the Iowa Gambling Task (Suhr & Hammers, 2010).

However in contrast to our hypotheses, no significant difference was found in risk-taking for BI and BUI children, nor was there a significant interaction between BI and inhibitory control. This suggests that BI children may not be generally risk-averse, but instead may be averse to risk specifically associated with novelty or their fears. For example, BI children are known to be particularly reticent in social situations (Coplan, DeBow, Schneider, & Graham, 2009), and BI has been shown to be a risk factor for social anxiety disorder in particular (Clauss & Blackford, 2012). Perhaps then, a task examining social risk-taking would have elicited differences between the BI and BUI children. However, if this was indeed the case, we would have expected BI to be significantly associated with social risk-taking in adolescents, which was in contrast to our findings.

Paper 2: Behavioural inhibition in childhood and the development of adolescent depression: The role of puberty and risk-taking.

This study built on Paper 1 and aimed to examine whether BI classified in childhood was associated with depressive symptoms in early adolescence. Additionally, as previous research has shown that risk-taking and reward-seeking behaviour increase into adolescence (e.g. Steinberg, 2008), Paper 2 utilised an early adolescent sample to examine the impact of risk-taking behaviour, as well as pubertal status, on the relationship between temperament and depressive symptoms. The approach of Paper 2 was novel as the association between BI and risk for depression has previously been examined mostly at single time points (e.g. Muris, Meester, & Spinder, 2003) or without consideration of potential moderating factors (e.g. Jaffee et al., 2002). In contrast to hypotheses, no overall relationship was demonstrated between BI in early childhood and depressive symptoms in early adolescence. However, risk-taking on the BART-Y was shown to significantly moderate this relationship, providing an initial indication that the relationship between BI in childhood and depression in adolescence differs according to the risk-taking behaviour shown by the adolescent. This finding was demonstrated for two of the three BART variables, suggesting that the

moderating effect on the relationship between BI and depressive symptoms may be related to risk-taking alone, rather than reward-focused risk-taking. Specifically, higher risk-taking was associated with an increased risk for depressive symptoms for the BI group, whereas for the BUI group a non-significant negative correlation was found between risk-taking and depressive symptoms, such that reduced risk-taking was associated with an increased risk for depressive symptoms. As these correlations did not reach significance the interpretation of these relationships needs to be tentative. Further, this effect was not consistent across all measures of risk-taking or all measures of depression. Nonetheless, this finding provides a possible insight into the pathway by which temperament may influence the development of depression.

In addition to risk-taking, Paper 2 also examined pubertal status as a potential moderating factor on the relationship between BI in early childhood and depression in early adolescence. Previous research has shown that early or late puberty, relative to peers, is associated with increased levels of depressed mood (Natsuaki, Biehl, & Ge, 2009). Although no main effect of pubertal status was found on depressive symptoms, pubertal status did show a moderation effect, influencing the relationship between BI and depressive symptoms on one measure of depression. This finding provides an interesting possibility that pubertal status may play a role in the pathway from BI to later depression. It is possible that the period from late childhood to early adolescence may be more stressful for BI children than BUI children (Lahat, Hong, & Fox, 2011). Therefore, the risk factors for the development of depression during the period of adolescence, such as pubertal status and risk-taking behaviour, may impact BI and BUI early adolescents differently, thus providing a potential pathway to depression.

Paper 3: The relationship between social risk-taking and depression in adolescence: The mediating effect of perceived social acceptance.

In adolescence, relationships with peers and social interactions increase in salience (Steinberg & Morris, 2001). In addition, low perceived social acceptance (Uhrlass, Crossett, & Gibb, 2008) and peer victimsation (Hawker & Boulton, 2000) in adolescence have been shown to be associated with an increased risk for depression. Consistent with the social risk hypothesis (Allen & Badcock, 2003), depression is hypothesised to be a mechanism to deal with social challenges by increasing a person's sensitivity for indicators of risk in social situations, allowing them to avoid taking social risks that might lead to their exclusion. However, other researchers suggest that the reduction in social risk-taking precedes the development of adolescent depression (Davey, Yucel, & Allen, 2008).

Thus, building on the work of the previous chapter, Paper 3 utilised a novel measure of social risk-taking and examined its relationship to a measure of depressive symptoms in adolescents over a six-month period. Further, measures of social interactions including perceived social acceptance and peer victimisation were included. It was expected that decreased social risk-taking at 12 years of age would be associated with increased depressive symptoms 6 months later. Furthermore, it was hypothesised that increased depressive symptoms at 12 years may be associated with reductions in social risk-taking 6 months later.

A bidirectional relationship between social risk-taking and depressive symptoms was demonstrated in this paper. Increased depressive symptoms at 12 years predicted reduced social risk-taking 6 months later. This finding provides support to the social risk hypothesis of depression (Allen & Badcock, 2003), that increased depression is a method for individuals to deal with social challenges by increasing their sensitivity to the possibility of risk in social situations, therefore leading to risk avoidance. In addition, this relationship was partially mediated by social acceptance, with increased depressive symptoms at 12 years associated with decreased social acceptance also at 12 years of age and decreased social risk-taking 6 months later. This is consistent with previous research demonstrating

that depression is associated with decreased social acceptance from peers (e.g. Heller & Tanaka-Matsumi, 1999). Importantly, this paper is the first to show a relationship between depresive symptoms, social acceptance and social risk-taking.

Unexpectedly, Paper 3 also demonstrates that increased social risk-taking at 12 years of age predicts increased depressive symptoms 6 months later. Although this finding initially appears to contrast with previous theories, it raises the possibility that taking social risks may not always lead to a positive interaction; instead it may lead to social victimisation. Being subjected to social victimisation may also lead to a suppression of the reward system, as theorised by Davey et al., (2008), which may play a role in the development of increased depressive symptoms. However, in the current study, this relationship was not found to be mediated by social acceptance, suggesting future research is needed to assess this possible pathway.

Paper 4: Reward-seeking behaviour on an effort-reward task and symptoms of depression in early adolescents.

Reduced reward-seeking, or anhedonia, has been demonstrated as a risk factor for the development of depression in prior studies (e.g. Weinberg et al., 2015). However, the majority of previous work examining reward-seeking in depression utilise tasks that confound reward-seeking with some element of risk-taking or negative feedback in response to risk. Reward-seeking is potentially a unique risk factor for depression, rather than shared with anxiety like risk avoidance (e.g. Maner et al., 2007; Guyer et al., 2012; Epstein et al., 2006), thus measuring this behaviour alone may be important when examining the mechanisms behind the development of depression. Reward-seeking behaviour has previously been examined in the effort-reward task (Waugh & Gotlib, 2008), which unlike the BART and SRTQ, does not confound reward-seeking and risk-taking. Paper 4, the final study in this thesis, adapted the modified effort-reward task (Sherdell, Waugh, & Gotlib, 2012) for use with adolescents. This task requires participants to choose between viewing a

humorous or non-humorous cartoon based on how much effort they are willing to exert on a square-clicking task. It was hypothesised that the adapted task would effectively measure reward-seeking in adolescents, and that there would be differences in motivation to view the rewarding cartoon based on depressive symptoms. The adapted effort-reward task was found to effectively measure reward-seeking in an adolescent sample, with participants liking the humorous cartoons more than the non-humorous cartoons, and participants willing to put in greater effort to view the humorous cartoon as the effort required was increased. However, in contrast to Sherdell and colleagues (2012) study with adults, there was no significant difference in motivation to view the humorous cartoons between depressive groups. This unexpected finding may be due to the age of the participants, or the severity of their depression (as they did not meet full criteria for a depressive disorder). It is possible that the variations in motivation to seek the reward of a humorous cartoon are not yet present at sub clinical levels of depression.

Theoretical implications for the development of depression

The findings of this thesis provide important additions to the existing adolescent depression literature in several areas. Previous work has suggested that the development of depression is unlikely to result from a single risk factor, rather the accrual of factors, or interaction amongst factors is what leads to depression (Garber, 2006). Although there is a large body of evidence examining the developmental pathways of depression in adults, it is only more recently that work has looked at the development of adolescent depression.

This thesis examined the risk factors of BI temperament and risk-taking behaviour, both previously found to be risk factors for the development of anxiety and depression (e.g. Jaffee et al., 2002; Hudson & Dodd, 2012; Smoski et al., 2008; Maner et al., 2007). No relationship was found between BI and risk-taking behaviour in preschool aged children. This may have been due to the risk-taking measure used not being salient enough to promote risk aversion in BI children. In contrast, the relationship between BI identified in

childhood and depressive symptoms in adolescence was impacted by risk-taking behaviour in adolescence. This finding suggests that although there may not be a direct relationship between BI and risk-taking behaviour in early childhood, these two factors may work in conjunction to increase the risk for adolescent depression. As both BI and risk-taking are risk factors for anxiety and depression in adolescence, this may also provide an insight into the interplay of anxiety and depression symptoms in adolescence. Indeed, it has been suggested that the high level of comorbidity between anxiety and depression (Angold, Costello, & Ekanli, 1999) could be due to common causation factors (Garber & Weersing, 2010).

Other theories of depression have focused on the role of social interactions in the development of adolescent depression. The interpersonal theory of depression suggests that the problematic social relationships of those with depression may lead to an increase in further depressive symptoms (Coyne, 1976). During adolescence, peers and social interactions become particularly salient. For example, adolescents place more importance on peer evaluation than children do (O'Brien & Bierman, 1988), thus it follows that peer interactions may be an important risk factor for adolescent depression. A review of experimental paradigms has shown that peer rejection plays a role in adolescent depression (Platt, Kadosh, & Lau, 2013). However, it is not clear what mechanisms increase the impact of peer victimisation for at risk adolescents (Platt et al., 2013). Although previous research and theories have considered this link between reduced social reward-seeking behaviour and depression (Rubin et al., 1995), the direction of this link is not evident. Therefore, the finding in Paper 3 that the relationship between social risk-taking and depressive symptoms may be bidirectional provides an important insight into the potential mechanisms of depression development.

Furthermore, the finding that increased social risk-taking at 12 years is associated with increased depressive symptoms 6 months later provides a potential insight into the different

ways social risk-taking and social interactions can increase the risk for depression. If an adolescent is undertaking social risks, such as trying to interact with new people, and is experiencing unsatisfactory results such as victimisation, it fits that this may lead to an increase in their risk for depression. This line of thought supports Davey and colleagues (2008)'s model that the thwarting of social goals in adolescence can eventually lead to the development of depression.

Reward-seeking has been found to be particularly important in the development of depression (e.g. Weinberg et al., 2015). However, this thesis found no relationship between depressive symptoms and reward-seeking behaviour. It is possible that the relationship between depression and reward-seeking behaviour occurs only at a clinical level, rather than with subclinical symptoms. Nonetheless, this finding is also important in the context of the findings related to risk-taking in this thesis. The BART and the SRTQ can be conceptualised as including both risk-taking and reward-seeking behaviour, which can not be easily separated. For instance, scenarios in the SRTQ require the early adolescent to undertake social risk (e.g. approach an unknown peer) for the chance of a reward. Thus, the finding that scores on the SRTQ were associated with depressive symptoms, but reward-seeking on the effort-reward task was not, suggests that it is the risk-taking aspect of social risk-taking, rather than the potential for reward, that is important for depression in early adolescence. Future research could further examine this possibility by examining the SRTQ and effort-reward task in one population of adolescents and how they uniquely predict depression.

Clinical implications

The papers included in this thesis provide evidence for risk factors in the development of depression in adolescence indicating further support to the importance of early intervention. Participants included in each study had not yet developed clinical depression, nevertheless risk factors including the interaction of temperament and risk taking, as well as the importance of social interactions, were associated with subclinical levels of depression.

As previously discussed, adolescent depression is associated with an increased risk of suicide, and impaired school performance (e.g. Fröjd et al., 2008; Lewinsohn et al., 1994). Although treatment programs are reasonably effective (e.g. Kennard et al., 2006), approximately 75% of depressed adolescents will not receive treatment (Hirschfeld et al., 1997). Thus, previous research has examined the efficacy of early intervention programs with adolescents at risk for the development of depression (e.g. Clarke et al., 2001). Although intervention programs have been shown to be effective in adolescents, not all participants have ongoing positive outcomes. For example, almost one-third of adolescents who achieved the "best" treatment outcomes went on to experience a depressive episode during the 33-month follow up (Beardslee et al., 2013).

It has been suggested that one method to strengthen intervention effects is to utilise other aspects of the adolescent's world as a focus during intervention (Reivich, Gillham, Chaplin, & Seligman, 2013). As demonstrated in Paper 3, depressive symptoms are associated with decreased perceived social acceptance and a reduction in later social risk-taking. Across the findings of this thesis, Paper 3 provides one of the clearest areas for potential clinical intervention. By identifying adolescents with depressive symptoms before the development of depressive disorders, it may be possible to provide an intervention that is aimed at reducing the impact on the adolescent's social interactions, thus preventing a reduction in social risk-taking. Providing at risk adolescents with the skills and resilience needed to prevent social withdrawal may help reduce the later development of clinical depression, thus breaking the potential negative cycle indicated by the bidirectional relationship demonstrated in Paper 3. In addition, results from Paper 2 showing that risk-taking behaviour impacts the relationship between BI and depressive symptoms suggests that those identified as BI in early childhood may also benefit from intervention in early adolescence. No direct relationship was shown between BI and depressive symptoms in Paper 2, however the inclusion of risk-taking behaviour led to an interaction effect. Thus,

providing an intervention targeting risk aversion may also be important for early adolescents previously identified as BI in early childhood. However, it is also important to note that increased social risk-taking at time 1 was associated with increased depressive symptoms at time 2 in Paper 3. Thus, the paradox is that reducing social withdrawal may prevent the development of adolescent depression, but increased social risk-taking may also lead to increased risk for depression. Further examination of the mechanisms behind the association between increased social risk-taking and depression will be important to understand whether these behaviours pose a risk for all individuals or whether there are other moderating factors to be considered. By understanding which individuals are at greater risk of negative outcomes following increases in social risk-taking, interventions can be tailored to increase positive outcomes for those individuals at risk. By providing adolescents with the skills and resilience needed for positive social interactions, it may be possible to reduce their risk for depression.

Thesis strengths

This thesis has a number of important strengths in both the use of longitudinal samples and the inclusion of new and adapted measures. Firstly, Paper 3 presented a novel social risk-taking measure: Social Risk-Taking Questionnaire (SRTQ). Previous work examining risk-taking in adolescents have utilised tasks that measure risk-taking using money or points (e.g. Gotlib et al., 2010). We know that social interactions are highly salient for adolescents (Spear 2000), thus risk-taking in this context could be particularly influential in this developmental period. In response to this gap in the literature, Paper 3 demonstrated the first use of the SRTQ. Notably, this study showed an association between SRTQ and depressive symptoms, social acceptance, and peer victimisation. These initial findings are an important starting point for examining real-life risk-taking in adolescents and how it relates to the development of depression.

Another strength was the use of the adapted effort-reward task (Sherdell et al., 2012). Initially based on the effort-reward task (Waugh & Gotlib, 2008), the adapted version of the effort reward task has previously been used to measure reward-seeking in adults and how it relates to depression. Paper 4 utilised age appropriate cartoons to modify this measure to be suitable for adolescents for the first time. Several earlier studies have used tasks that observe reward-seeking but also include negative feedback in response to risk-taking. For example, the Balloon Analogue Risk Task (BART: Lejuez et al., 2002) entails pumping up a virtual balloon, with each pump gaining a point. However, the balloon can explode at any time; if this happens, participants lose their points. Consequently, the BART includes risk as well as the possibility for reward. The BART has been critiqued for not making a distinction between the cognitive components that influence a participant's performance, for example sensitivity to reward and risk attitudes (Lejuez et al., 2002). Using the adapted effort-reward task in Paper 4 provides a unique look at reward-seeking in adolescents and its relationship with depressive symptoms without the possible influence of risk. Although the results were not as predicted, the new knowledge that the effort-reward task can be effectively used in adolescents to measure reward-seeking opens exciting avenues for future research.

Another strength of the current research is the use of both child and mother report, and the inclusion of a structured interview and questionnaires to assess depressive symptoms. It is considered optimal to obtain information from multiple informants when assessing depression, including the child and parents (Klein, Dougherty, & Olino, 2005). This is because parent, clinician and child ratings have been shown to all explain significant unique differences in predicting ensuing outcomes (Verhulst, Dekker, & Ende, 1997). However, agreement between the informants has been shown to often be only fair to moderate (Achenbach, McConaughy, & Howell, 1987). The KSADS interview is considered a gold standard measure due to the use of a semi-structured interview by a trained administrator

with both mother and early adolescent. The SMFQ scale is also a well-used measure in the depression literature that has been shown to discriminate depressed from non-depressed subjects in a general population (Angold, Costello, Messer, & Pickles, 1995). The current thesis found both consistent and contrasting results for the two different measures. However, the use of two differing measures of depressive symptoms may have allowed for diverging aspects of depression to be captured. The KSADS is focused more on depression diagnosis, whereas the SMFQ is a continuous measure of symptomology.

Thesis limitations and future directions

This thesis has some notable limitations. Consistent with most longitudinal studies, there was missing data present in Papers 2 and 3. Both studies utilised methods to deal with missing data: Paper 2 utilised multiple imputation (Sinharay, Stern, & Russell, 2001) while Paper 3 utilised single shot imputation (Enders, 2001; Scheffer, 2002). While imputation methods allow for analysis of a more complete data set, as a small number of data points are created statistically rather than from participants it is important for the findings to be replicated in future research.

A further methodological limitation that impacts the generalisability of the findings across this thesis is the sample selection basis. Both the pre-school aged sample in Paper 1 and the early adolescent sample in Papers 2, 3, and 4 were initially selected based on temperament using an extreme groups design. Specifically, participants were included if they met the criteria for BI or were behaviourally uninhibited. This extreme groups design allows for the examination of the important risk factor of BI and how it relates to the development of depression in adolescents. However, this also limits whether the current findings can be applied to the general population. In particular, the findings may relate to unique factors to these two extreme groups and thus future research is needed to replicate the findings with a more representative population.

Another important consideration is the use of depressive symptoms rather than a diagnosis of depression throughout the thesis. Although this thesis assessed for depression diagnoses, none of the participants in studies 2, 3 and 4 met the full criteria for clinical depression. This may have been due to the age range of 11-14 years of age. Nonetheless, examining risk factors for the development of depressive symptoms does provide an insight into depression before it develops into a disorder. The findings of this thesis do not provide evidence of BI or risk-taking in isolation being predictive of depression in the early adolescent age group. Moderating effects were shown however, with risk-taking and pubertal demonstrated to moderate the relationship between BI in childhood and depressive symptoms in early adolescence. These results provide important information about the timeline, or developmental boundaries of risk factors for adolescent depression. That is, in the current sample of early adolescents a pattern of moderating and mediating effects was shown to be associated with increased depressive symptoms. Continuing this research with older adolescents will allow future research to capture how the association between these risk factors and depression varies according to developmental stage. For example, is early BI particularly associated with depression that emerges in late adolescence subsequent to an anxiety diagnosis? Is social risk-taking particularly relevant for depression that has an earlier onset? It seems likely that different risk factors could be differentially linked to depression at specific developmental stages and this body of work contributes to our understanding of these developmental pathways.

The use of a subclinical population does present limitations, particularly for the use of the KSADS interview. The KSADS is a diagnostic semi-structured interview, used to determine if the interviewee meets the criteria for a clinical disorder. In this thesis, to capture the subclinical levels of depression while still utilising the KSADS, symptom presence/absence was the outcome measure instead of depressed/non-depressed. The assessment of inter-rater reliability to ensure consistency across the allocation of

participants to either the symptom presence or absence group was a strength of the current research. However, as this method had not been previously used in the literature it is important to replicate the findings in future research to demonstrate the validity of this method. In addition, one method of assessing construct validity throughout the thesis was the use of the SMFQ child and maternal report. Nonetheless, the SMFQ provides a measure of depressive symptoms rather than the presence of clinically significant symptoms.

Further, not all variables were able to utilised across all the studies included in this thesis. Although the samples used were originally selected based on whether they met criteria for being behaviourally inhibited or uninhibited, BI was not included as a measure in Papers 3 or 4 as it was not a focus of the papers' aims. In Paper 3 the focus was on the bi-directional relationship between depressive symptoms and social risk-taking over a 6-month period. As participants completed online questionnaires only at the time 2 follow up, this prevented the examination of BI. Furthermore, the KSADS was not included in Paper 3 as the use of an interview at time point 2 was not possible. Thus, each of the papers in this thesis do not utilise all available measures, due to the differing focus points for each paper.

Finally, the number of participants presents a limitation across the thesis. Although significant findings were found, in some analyses non-significance may have been due to a lack of power. In particular, interaction effects were unable to be fully explored in Paper 2 due to the non-significance of post-hoc correlations.

Direct implications for future research

The inclusion of the novel measure SRTQ is an important contribution to the field of adolescent depression. However, as with all new measures, the psychometric properties of the SRTQ need to be further examined. Paper 3 initially examined how findings on the SRTQ were associated with depressive symptoms, social acceptance, and peer victimisation. In this study, participants' responses on the SRTQ at time1 and time 2 were significantly correlated presenting evidence of test-retest reliability. Future research can provide

additional testing of the reliability and validity of the SRTQ. For example, the use of the measure in a large, representative sample of adolescents could provide important information on normative responding. Furthermore, comparison of responses on the SRTQ to other gold standard measures will help provide construct validity for the SRTQ as a measure of social risk-taking behaviour in adolescence.

An important strength of this thesis is the use of a longitudinal design to examine directionality in the relationship between adolescent depression and risk factors such as social risk-taking. An important direction for future research would be to extend this time frame. The population in Paper 3 were approximately 12-13 years at the time of the second follow up. Previous research has shown that the rate of depression begins to rise most dramatically in mid adolescence, from approximately 15 years of age (Hankin et al., 1998). Examining depression at the symptom level is important given subclinical depressive symptoms have still been shown to be associated with impaired functioning (Gotlib, Lewinsohn, & Seeley, 1995). However, following up into adolescence and adulthood would provide a more complete picture of adolescent depression development and how different risk factors interact together.

Conclusion

This thesis has brought together existing evidence regarding risk factors for depression and has particularly focused on risk within the context of early adolescence. It has done this by focusing on early temperament, including BI and inhibitory control, as well as factors crucial in early adolescence including social interactions, puberty, and risk-taking and reward-seeking. The studies in this thesis are amongst the first to examine these risk factors for adolescent depression in conjunction with one another. First, while BI and risk-taking on the BART-Y were not directly associated with depressive symptoms in early adolescents, these risk factors interacted to predict depressive symptoms. That is, risk-taking had a significant effect on the relationship between BI and depressive symptoms in

adolescence. Second, social risk-taking as assessed by the novel measure SRTQ, was shown to have a bidirectional relationship with depressive symptoms in early adolescents, providing support for previous theories of the importance of social interactions in the development of adolescent depression. Finally, the role of reward-seeking behaviour was examined without the confounding factor of risk-taking. The establishment of the adapted effort-reward task as a suitable measure for adolescents provides a potential avenue for future research. This thesis contributes to our understanding of the development of depression in young people and provides a number of important areas for future research.

References

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, *101*(2), 213.

Allen, N. B., & Badcock, P. B. (2003). The Social Risk Hypothesis of Depressed Mood: Evolutionary, psychosocial, and neurobiological perspectives. *Psychological Bulletin, 129*(6), 887-913. doi:http://dx.doi.org/10.1037/0033-2909.129.6.887

Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry*, *40*(1), 57-87.

Angold, A., Costello, E. J., Messer, S. C., & Pickles, A. (1995). Development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *International Journal of Methods in Psychiatric Research*.

Beardslee, W.R., Brent, D.A., Weersing, V.R., Clarke, G.N., Porta, G., Hollon, S.D., Gladstone, T.R., Gallop, R., Lynch, F.L., Iyengar, S. & DeBar, L., (2013). Prevention of depression in at-risk adolescents: longer-term effects. *JAMA Psychiatry*, *70*(11), 1161-1170.

Birmaher, B., Ryan, N.D., Williamson, D.E., Brent, D.A., Kaufman, J., Dahl, R.E., Perel, J., & Nelson, B. (1996). Childhood and adolescent depression: A review of the past 10 years. Part I. *Journal of the American Academy of Child & Adolescent Psychiatry, 35,* 1427-1439.

Clarke, G. N., Hornbrook, M., Lynch, F., Polen, M., Gale, J., Beardslee, W., O'Connor, E., & Seeley, J. (2001). A randomized trial of a group cognitive intervention for preventing depression in adolescent offspring of depressed parents. *Archives of General Psychiatry, 58*(12), 1127-1134.

Clauss, J. A., & Blackford, J. U. (2012). Behavioral Inhibition and risk for developing Social Anxiety Disorder: A meta-analytic study. *Journal of the American Academy*

of Child & Adolescent Psychiatry, 51(10), 1066-1075.e1061. doi:http://dx.doi.org/10.1016/j.jaac.2012.08.002

Coplan, R. J., DeBow, A., Schneider, B. H., & Graham, A. A. (2009). The social behaviours of inhibited children in and out of preschool. *British Journal of Developmental Psychology, 27*, 891-905.

Coyne, J. C. (1976). Toward an interactional description of depression. *Psychiatry, 39*(1), 28-40.

Davey, C. G., Yucel, M., & Allen, N. B. (2008). The emergence of depression in adolescence: Development of the prefrontal cortex and the representation of reward. *Neuroscience and Biobehavioral Reviews, 32*(1), 1-19. doi:http://dx.doi.org/10.1016/j.neubiorev.2007.04.016

Enders, C. K. (2001). A primer on maximum likelihood algorithms available for use with missing data. *Structural Equation Modelling, 8,* 128-141. doi: 10.1207/S15328007SEM0801_7

Epstein, J., Pan, H., Kocsis, J. H., Yang, Y., Butler, T., Chusid, J., Hochberg, H., Murrough, J., Strohmayer, E., Stern, E., & Silbersweig, D. A. (2006). Lack of ventral striatal response to positive stimuli in depressed versus normal subjects. *American Journal of Psychiatry*.

Fröjd, S. A., Nissinen, E. S., Pelkonen, M. U. I., Marttunen, M. J., Koivisto, A. M., & Kaltiala-Heino, R. (2008). Depression and school performance in middle adolescent boys and girls. *Journal of Adolescence, 31*(4), 485-498.

Garber, J. (2006). Depression in Children and Adolescents. Linking Risk Research and Prevention. *American Journal of Preventive Medicine, 31*(6 SUPPL. 1), 104-125.

Garber, J., & Weersing, V. R. (2010). Comorbidity of anxiety and depression in youth: Implications for treatment and prevention. *Clinical Psychology: Science and Practice, 17*(4), 293-306.

Gladstone, G. L., & Parker, G. B. (2006). Is behavioral inhibition a risk factor for depression? *Journal of Affective Disorders, 95*(1-3), 85-94. doi:http://dx.doi.org/10.1016/j.jad.2006.04.015

Gotlib, I. H., Hamilton, J., Cooney, R. E., Singh, M. K., Henry, M. L., & Joormann, J. (2010). Neural processing of reward and loss in girls at risk for major depression. *Archives of General Psychiatry, 67*(4), 380-387. doi:http://dx.doi.org/10.1001/archgenpsychiatry.2010.13

Gotlib, I. H., Lewinsohn, P. M., & Seeley, J. R. (1995). Symptoms versus a diagnosis of depression: Differences in psychosocial functioning. *Journal of Consulting and Clinical Psychology, 63*(1), 90

Guyer, A. E., Choate, V. R., Detloff, A., Benson, B., Nelson, E. E., Perez-Edgar, K., Fox, N. A., Pine, D. S., & Ernst, M. (2012). Striatal functional alteration during incentive anticipation in pediatric anxiety disorders. *The American Journal of Psychiatry, 169*(2), 205-212.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology, 107*(1), 128.

Hawker, D. S., & Boulton, M. J. (2000). Twenty years' research on peer victimization and psychosocial maladjustment: A meta-analytic review of cross-sectional studies. *Journal of Child Psychology and Psychiatry, 41*(4), 441-455.

Heller, M. C., & Tanaka-Matsumi, J. (1999). A sequential analysis of depressive behaviors within adolescent peer interactions. *Journal of Psychopathology and Behavioral Assessment, 21*(3), 249-273.

Hirschfeld, R.M., Keller, M.B., Panico, S., Arons, B.S., Barlow, D., Davidoff, F., Endicott, J., Froom, J., Goldstein, M., Gorman, J.M. & Guthrie, D., (1997). The National

Depressive and Manic-Depressive Association consensus statement on the undertreatment of depression. *Jama*, *277*(4),.333-340.

Hudson, J. L., & Dodd, H. F. (2012). Informing early intervention: Preschool predictors of anxiety disorders in middle childhood. *PLoS ONE, 7*(8).

Jaffee, S. R., Moffitt, T. E., Caspi, A., Fombonne, E., Poulton, R., & Martin, J. (2002). Differences in early childhood risk factors for juvenile-onset and adult-onset depression. *Archives of General Psychiatry, 59*(3), 215-222. doi:http://dx.doi.org/10.1001/archpsyc.59.3.215

Kennard, B., Silva, S., Vitiello, B., Curry, J., Kratochvil, C., Simons, A., Hughes, J., Feeny, N., Weller, E., Sweeney, M. & Reinecke, M., (2006). Remission and residual symptoms after short-term treatment in the Treatment of Adolescents with Depression Study (TADS). *Journal of the American Academy of Child & Adolescent Psychiatry*, *45*(12), 1404-1411.

Kessler, R. C., Avenevoli, S., & Ries Merikangas, K. (2001). Mood disorders in children and adolescents: An NIMH perspective. *Biological Psychiatry, 49*(12), 962-969.

Klein, D. N., Dougherty, L. R., & Olino, T. M. (2005). Toward guidelines for evidence-based assessment of depression in children and adolescents. *Journal of Clinical Child and Adolescent Psychology, 34*(3), 412-432.

Lahat, A., Hong, M., & Fox, N. A. (2011). Behavioural inhibition: Is it a risk factor for anxiety? *International Review of Psychiatry, 23*(3), 248-257.

Lau, J.Y.F., & Eley, T.C. (2010). The genetics of mood disorders. *Annual Review of Clinical Psychology, 6,* 313-337.

Lejuez, C., Aklin, W., Daughters, S., Zvolensky, M., Kahler, C., & Gwadz, M. (2007). Reliability and validity of the Youth Version of the Balloon Analogue Risk Task (BART-Y) in the assessment of risk-taking behavior among inner-city adolescents. *Journal of Clinical Child and Adolescent Psychology, 36*(1), 106-111.

doi:http://dx.doi.org/10.1207/s15374424jccp3601_11

Lejuez, C., Read, J. P., Kahler, C. W., Richards, J. B., Ramsey, S. E., Stuart, G. L., Strong, D.R. & Brown, R. A. (2002). Evaluation of a behavioral measure of risk taking: The Balloon Analogue Risk Task (BART). *Journal of Experimental Psychology: Applied, 8*(2), 75-84. doi:http://dx.doi.org/10.1037/1076-898X.8.2.75

Lewinsohn, P. M., Rohde, P., & Seeley, J. R. (1994). Psychosocial risk factors for future adolescent suicide attempts. *Journal of Consulting and Clinical Psychology, 62*(2), 297-305.

Maner, J. K., Richey, J., Cromer, K., Mallott, M., Lejuez, C. W., Joiner, T. E., & Schmidt, N. B. (2007). Dispositional anxiety and risk-avoidant decision-making. *Personality and Individual Differences, 42*(4), 665-675.

Muris, P., Meesters, C., & Spinder, M. (2003). Relationships between child- and parent-reported behavioural inhibition and symptoms of anxiety and depression in normal adolescents. *Personality and Individual Differences, 34*(5), 759-771. doi:http://dx.doi.org/10.1016/S0191-8869%2802%2900069-7

Natsuaki, M. N., Biehl, M. C., & Ge, X. (2009). Trajectories of depressed mood from early adolescence to young adulthood: The effects of pubertal timing and adolescent dating. *Journal of Research on Adolescence, 19*(1), 47-74. doi:http://dx.doi.org/10.1111/j.1532-7795.2009.00581.x

O'Brien, S. F., & Bierman, K. L. (1988). Conceptions and perceived influence of peer groups: Interviews with preadolescents and adolescents. *Child Development*, 1360-1365.

Platt, B., Kadosh, K. C., & Lau, J. Y. (2013). The role of peer rejection in adolescent depression. *Depression and Anxiety, 30*(9), 809-821. doi:http://dx.doi.org/10.1002/da.22120

Reivich, K., Gillham, J. E., Chaplin, T. M., & Seligman, M. E. (2013). From helplessness to optimism: The role of resilience in treating and preventing depression in youth *Handbook of resilience in children* (pp. 201-214): Springer.

Rubin, K. H., Coplan, R. J., Fox, N. A., & Calkins, S. D. (1995). Emotionality, emotion regulation, and preschoolers' social adaptation. *Development and Psychopathology, 7*(1), 49-62. doi:http://dx.doi.org/10.1017/S0954579400006337

Scheffer, J. (2002). Dealing with missing data. Research Letters in the Information and Mathematical Sciences, 3, 153-160. Retrieved from http://equinetrust.org.nz/massey/fms/Colleges/College%20of%20Sciences/IIMS/RLIMS/Volume03/Dealing_with_Missing_Data.pdf

Sherdell, L., Waugh, C. E., & Gotlib, I. H. (2012). Anticipatory pleasure predicts motivation for reward in major depression. *Journal of Abnormal Psychology*, *121*(1), 51.

Sinharay, S., Stern, H. S., & Russell, D. (2001). The use of multiple imputation for the analysis of missing data. *Psychological Methods, 6*(4), 317.

Smoski, M. J., Lynch, T. R., Rosenthal, M., Cheavens, J. S., Chapman, A. L., & Krishnan, R. R. (2008). Decision-making and risk aversion among depressive adults. *Journal of Behavior Therapy and Experimental Psychiatry, 39*(4), 567-576. doi:http://dx.doi.org/10.1016/j.jbtep.2008.01.004

Spear, L. P. (2000). The adolescent brain and age-related behavioral manifestations. *Neuroscience & Biobehavioral Reviews, 24*(4), 417-463. doi:http://dx.doi.org/10.1016/S0149-7634(00)00014-2

Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review, 28*(1), 78-106.

Steinberg, L., & Morris, A. S. (2001). Adolescent development. *Journal of Cognitive Education and Psychology, 2*(1), 55-87.

Suhr, J., & Hammers, D. (2010). Who fails the Iowa Gambling Test (IGT)? Personality,

neuropsychological, and near-infrared spectroscopy findings in healthy young controls. *Archives of Clinical Neuropsychology, 25*(4), 293-302. doi:http://dx.doi.org/10.1093/arclin/acq017

Uhrlass, D. J., Crossett, S. E., & Gibb, B. E. (2008). Self-perceived competence, relational victimization, and children's depressive symptoms: Evidence for a sex-specific vulnerability-stress model. *International Journal of Cognitive Therapy, 1*(4), 284-297. doi:http://dx.doi.org/10.1521/ijct.2008.1.4.284

Verhulst, F. C., Dekker, M. C., & Ende, J. V. D. (1997). Parent, teacher and self-reports as predictors of signs of disturbance in adolescents: whose information carries the most weight?. *Acta Psychiatrica Scandinavica*, *96*(1), 75-81.

Waugh, C. E., & Gotlib, I. H. (2008). Motivation for reward as a function of required effort: Dissociating the 'liking' from the 'wanting' system in humans. *Motivation and Emotion, 32*(4), 323-330.

Weinberg, A., Liu, H., Hajcak, G., & Shankman, S. A. (2015). Blunted neural response to rewards as a vulnerability factor for depression: Results from a family study. *Journal of Abnormal Psychology, 124*(4), 878.

Zisook, S., Lesser, I., Stewart, J. W., Wisniewski, S. R., Balasubramani, G. K., Fava, M.,Gilmer, W S., Dresselhaus, T R, Thase, M E, Nierenberg, A A, Trivedi, M H., & Trivedi, M. H. (2007). Effect of age at onset on the course of major depressive disorder. *American Journal of Psychiatry*, *164*(10), 1539-1546.

Appendix A

Macquarie University Ethics Approvals

MACQUARIE University

Talia Morris <talia.morris@mq.edu.au>

Fwd: Ethics amendments for HE30MAY2008-R05911

Helen Dodd <helen.dodd@mq.edu.au> Fri, Apr 15, 2011 at 3:57 PM
To: Margie Brammall <margiebram@optusnet.com.au>, Talia Morris <talia.morris@mq.edu.au>

All done! Finally :)

H

---------- Forwarded message ----------
From: Ethics Secretariat <ethics.secretariat@mq.edu.au>
Date: 15 April 2011 05:49
Subject: Re: Ethics amendments for HE30MAY2008-R05911
To: Helen Dodd <helen.dodd@mq.edu.au>

Dear Helen

Thank you for your email. The following amendments have been approved (with the exception of the cheek swab collection amendment):

1. Amendment 1. The addition of the following measures:
The Youth Version of the Balloon Analogue Risk Task (BART-Y), Self-Perception Profile for Children (SPPC), the Pubertal Development Scale (PDS), the Social Risk Taking Questionnaire and a questionnaire about challenging behaviours to be completed by the participant's mother and father.

2. Amendment 2.

(a) The addition of the following to the approved experimental tasks:
Revised Peer Experience Questionnaire and Interpretation bias in social networking site (SNS) task.

(b) Prior to the session participants will be asked to complete the following questionnaires online:
Revised Cyber Victimisation Scale and the Electronic Media Usage Questionnaire

3. Amendment 1 & 2. The addition of the following students to the study:
Ms Margaret Brammall, Ms Veronica Engel, Ms Amy He, Ms Emily Ramsay and Ms Kerrie Lam.

4. The Tanner diagrams will be removed from the online questionnaires and children will be asked to complete them in the session, still using an online format. Parents will be shown a copy of the diagrams and will be asked to tick an additional box of the information and consent form if they are happy for their child to complete these questions. If the parent chooses not to tick the box, the child will not be shown the Tanner diagrams.

5. The information and consent form has been amended to reflect the above changes.

Please do not hesitate to contact the Ethics Secretariat if we can be

of any assistance.

Kind regards
Fran

On Fri, Apr 15, 2011 at 12:54 AM, Helen Dodd <helen.dodd@mq.edu.au> wrote:
>
> Dear Fran,
>
> Thank you for forwarding on the committee's thoughts on our amendments.
>
> Following the advice of the committee, we have decided to remove the
> Tanner diagrams from the online questionnaires and ask children to
> complete them in the session (still using an online format). At the
> beginning of the session when we go through consent and
> confidentiality with participants, we will show the parent a copy of
> the diagrams and ask them to check an additional box on the consent
> form to show that they consent to their child answering the questions
> that refer to the diagrams. If they choose not to select this box, the
> child will not complete these questions.
>
> Please find attached the amended consent form with an additional tick
> box at the end of page 2.
>
> I will look at the points raised in relation to the genetic study
> early next week because I'll need to speak to Jennie about some of
> them before I can provide a response.
>
> Once again for your help navigating this complex amendment. Don't
> hesitate to contact me if there are any further questions or concerns.
>
> Many thanks,
> Helen
>
> On 14 April 2011 04:40, Ethics Secretariat <ethics.secretariat@mq.edu.au> wrote:
> > Dear Helen
> >
> > I was about to prepare the approval for amendment 2 (cyber ivictimisation0,
> > and realised that I can't do this until we have received and approved your
> > response to the Tanner Charts. This is because the information and consent
> > form for amendment 2 incorporates all the students and information for both
> > amendments. One amendment can't be approved without the other (but the cheek
> > swab collection aspect probably could be separated.
> >
> > To resolve this, it might be easier for you to provide a separate response
> > to the Committee's Tanner Charts query.
> >
> > Hope this makes sense.
> >
> > Please do not hesitate to ring me if you have any questions.
> >
> > Kind regards
> > Fran
> >
> > On Thu, Apr 14, 2011 at 1:25 PM, Ethics Secretariat
> > <ethics.secretariat@mq.edu.au> wrote:
> >>
> >> Dear Helen
> >>
> >> Thank you for your email. I left you a message on your work phone, but
> >> thought I would email too anyway.

MACQUARIE University

Talia Morris <talia.morris@mq.edu.au>

Ethics application Reference- 5201000902- Final Approval

1 message

Ethics Secretariat <ethics.secretariat@mq.edu.au> Wed, Aug 11, 2010 at 9:41 AM
To: A/Prof Jennie Hudson <jennie.hudson@mq.edu.au>
Cc: Dr Helen Dodd <helen.dodd@mq.edu.au>, Mrs Talia Morris <talia.higman@mq.edu.au>

Dear A/Prof Hudson

Re: "A cognitive behavioural intervention program for behaviourally inhibited children and their parents"

Thank you for your recent correspondence. Your response has addressed the issues raised by the Human Research Ethics Committee and you may now commence your research.

The following personnel are authorised to conduct this research:

A/Prof Jennifer Leanne Hudson- Chief Investigator
Dr Helen Dodd, Dr Thalia Eley & Mrs Talia Morris- Co-Investigator

Please note the following standard requirements of approval:

1. The approval of this project is conditional upon your continuing compliance with the National Statement on Ethical Conduct in Human Research (2007).

2. Approval will be for a period of five (5) years subject to the provision of annual reports. Your first progress report is due on 11th August 2011.

If you complete the work earlier than you had planned you must submit a Final Report as soon as the work is completed. If the project has been discontinued or not commenced for any reason, you are also required to submit a Final Report for the project.

Progress reports and Final Reports are available at the following website:

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/forms

3. If the project has run for more than five (5) years you cannot renew approval for the project. You will need to complete and submit a Final Report and submit a new application for the project. (The five year limit on renewal of approvals allows the Committee to fully re-review research in an environment where legislation, guidelines and requirements are continually changing, for example, new child protection and privacy laws).

4. All amendments to the project must be reviewed and approved by the Committee before implementation. Please complete and submit a Request for Amendment Form available at the following website:

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/forms

5. Please notify the Committee immediately in the event of any adverse

effects on participants or of any unforeseen events that affect the continued ethical acceptability of the project.

6. At all times you are responsible for the ethical conduct of your research in accordance with the guidelines established by the University. This information is available at the following websites:

http://www.mq.edu.au/policy/

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/policy

If you will be applying for or have applied for internal or external funding for the above project it is your responsibility to provide the Macquarie University's Research Grants Management Assistant with a copy of this email as soon as possible. Internal and External funding agencies will not be informed that you have final approval for your project and funds will not be released until the Research Grants Management Assistant has received a copy of this email.

If you need to provide a hard copy letter of Final Approval to an external organisation as evidence that you have Final Approval, please do not hesitate to contact the Ethics Secretariat at the address below.

Please retain a copy of this email as this is your official notification of final ethics approval.

Yours sincerely
Dr Karolyn White
Director of Research Ethics
Chair, Human Research Ethics Committee

MACQUARIE University

Talia Morris <talia.morris@mq.edu.au>

Final Approval- Ethics application reference-5201100488

Ethics Secretariat <ethics.secretariat@mq.edu.au> Tue, Jul 5, 2011 at 3:38 PM
To: A/Prof Jennie Hudson <jennie.hudson@mq.edu.au>
Cc: talia.morris@mq.edu.au

Dear A/Prof Hudson

Re: "Temperament, environment and cognitive processes in confident children" (Ethics Ref: 5201100488)

Thank you for your recent correspondence. Your response has addressed the issues raised by the Human Research Ethics Committee and you may now commence your research.

The following personnel are authorised to conduct this research:

A/Prof Jennie Hudson- Chief Investigator/Supervisor
Dr Helen Dodd, Mrs Talia Maree Morris & Dr Thalia Eley-Co-Investigators

NB. STUDENTS: IT IS YOUR RESPONSIBILITY TO KEEP A COPY OF THIS APPROVAL EMAIL TO SUBMIT WITH YOUR THESIS.

Please note the following standard requirements of approval:

1. The approval of this project is conditional upon your continuing compliance with the National Statement on Ethical Conduct in Human Research (2007).

2. Approval will be for a period of five (5) years subject to the provision of annual reports. Your first progress report is due on 05 July 2012.

If you complete the work earlier than you had planned you must submit a Final Report as soon as the work is completed. If the project has been discontinued or not commenced for any reason, you are also required to submit a Final Report for the project.

Progress reports and Final Reports are available at the following website:

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/forms

3. If the project has run for more than five (5) years you cannot renew approval for the project. You will need to complete and submit a Final Report and submit a new application for the project. (The five year limit on renewal of approvals allows the Committee to fully re-review research in an environment where legislation, guidelines and requirements are continually changing, for example, new child protection and privacy laws).

4. All amendments to the project must be reviewed and approved by the Committee before implementation. Please complete and submit a Request for Amendment Form available at the following website:

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/forms

5. Please notify the Committee immediately in the event of any adverse effects on participants or of any unforeseen events that affect the continued ethical acceptability of the project.

6. At all times you are responsible for the ethical conduct of your research in accordance with the guidelines established by the University. This information is available at the following websites:

http://www.mq.edu.au/policy/

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/policy

If you will be applying for or have applied for internal or external funding for the above project it is your responsibility to provide the Macquarie University's Research Grants Management Assistant with a copy of this email as soon as possible. Internal and External funding agencies will not be informed that you have final approval for your project and funds will not be released until the Research Grants Management Assistant has received a copy of this email.

If you need to provide a hard copy letter of Final Approval to an external organisation as evidence that you have Final Approval, please do not hesitate to contact the Ethics Secretariat at the address below.

Please retain a copy of this email as this is your official notification of final ethics approval.

Yours sincerely
Dr Karolyn White
Director of Research Ethics
Chair, Human Research Ethics Committee

Appendix B

Supplementary Tables from Chapter 3

KSADS

Supplementary Table 1

Logistic regression predicting likelihood of reporting symptom presence on the KSADS with BI, SMS and total points.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	-1.39	0.32	0.25*	.00
Combined BI	0.49	0.45	1.63	.27
Step 2				
Constant	-1.88	2.32	0.15	.42
Combined BI	0.76	2.83	2.14	.79
SMS (pubertal status)	0.58	0.46	1.79	.21
Total Points (BART)	-0.01	0.02	0.99	.67
Combined BI * SMS	-0.69	0.59	0.50	.25
Combined BI * Total Points	0.01	0.02	1.01	.57

Note: Step 1: $R^2 = .04$, Step 2: $R^2 = .17$, *p < 0.05, BI = Behavioural Inhibition

Supplementary Table 2

Logistic regression predicting likelihood of reporting symptom presence on the KSADS with BI, SMS and explosions.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	-1.39	0.32	0.25*	.00
Combined BI	0.49	0.45	1.63	.27
Step 2				
Constant	-0.92	1.90	0.40	.63
BI	-1.89	2.49	0.15	.45
SMS (pubertal status)	0.48	0.48	1.62	.32
Explosions (BART)	-0.15	0.10	0.87	.16
Combined BI * SMS	-0.63	0.63	0.53	.32
Combined BI * Explosions	0.33	0.14	1.39*	.02

Note: Step 1: $R^2 = .04$, Step 2: $R^2 = .20$, *p < 0.05, BI = Behavioural Inhibition

Supplementary Table 3

Logistic regression predicting likelihood of reporting symptom presence on the KSADS with BI, SMS and average adjusted pumps.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	-1.39	0.32	0.25*	.00
Combined BI	0.49	0.45	1.63	.27
Step 2				
Constant	-1.16	1.85	0.31	.53
Combined BI	-1.50	2.32	0.22	.52
SMS (pubertal status)	0.51	0.48	1.70	.29
Average Adjusted Pumps (BART)	-0.26	0.21	0.77	.22
Combined BI * SMS	-0.62	0.61	0.53	.31
Combined BI * Average AdjPumps	0.60	0.29	1.81*	.04

Note: Step 1: $R^2 = .04$, Step 2: $R^2 = .21$,*$p < 0.05$, BI = Behavioural Inhibition

Supplementary Table 4

Linear regression predicting symptom level on the SMFQ child report with BI, SMS and total points.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.40	0.04	10.57*	.00
Combined BI	0.01	0.06	0.14	.89
Step 2				
Constant	0.43	0.19	2.31*	.02
Combined BI	0.01	0.06	0.15	.88
SMS (pubertal status)	-0.02	0.04	-0.42	.67
Total Points (BART)	-0.00	0.00	0.03	.98
Combined BI * SMS	-0.03	0.03	-1.02	.31
Combined BI * Total Points	0.00	0.00	-.27	.79

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .14$, *p < 0.05, BI = Behavioural Inhibition

Supplementary Table 5

Linear regression predicting symptom level on the SMFQ child report with BI, SMS and explosions.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.40	0.04	10.57*	.00
Combined BI	0.01	0.06	0.14	.89
Step 2				
Constant	0.53	0.14	3.84*	.00
BI	0.01	0.06	0.21	.84
SMS (pubertal status)	-0.02	0.04	-0.46	.65
Explosions (BART)	-0.01	0.01	-0.97	.33
Combined BI * SMS	-0.03	0.03	-0.92	.36
Combined BI * Explosions	-0.00	0.01	-0.20	.84

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .11$, *p < 0.05, BI = Behavioural Inhibition

Supplementary Table 6

Linear regression predicting symptom level on the SMFQ child report with BI, SMS and average adjusted pumps.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.40	0.04	10.57*	.00
Combined BI	0.01	0.06	0.14	.89
Step 2				
Constant	0.52	0.15	3.53*	.00
Combined BI	0.01	0.06	0.19	.85
SMS (pubertal status)	-0.02	0.04	-0.47	.64
Average Adjusted Pumps (BART)	-0.01	0.02	-0.80	.43
Combined BI * SMS	-0.03	0.03	-0.97	.33
Combined BI *Average Adj Pumps	-0.00	0.02	-0.23	.82

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .12$,*$p < 0.05$, BI = Behavioural Inhibition

Supplementary Table 7

Linear regression predicting symptom level on the SMFQ mother report with BI, SMS and total points.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.35	0.04	9.04*	.00
Combined BI	0.02	0.06	0.34	.73
Step 2				
Constant	0.37	0.19	1.98*	.05
Combined BI	0.02	0.06	0.33	.74
SMS (pubertal status)	0.00	0.03	0.05	.96
Total Points (BART)	0.00	0.00	-0.15	.89
Combined BI * SMS	-0.01	0.03	-0.16	.87
Combined BI * Total Points	0.00	0.00	0.11	.91

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .02$, *p < 0.05, BI = Behavioural Inhibition

Supplementary Table 8

Linear regression predicting symptom level on the SMFQ mother report with BI, SMS and explosions.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.35	0.04	9.04*	.00
Combined BI	0.02	0.06	0.34	.73
Step 2				
Constant	0.18	0.14	1.31	.19
Combined BI	0.02	0.06	0.27	.79
SMS (pubertal status)	0.01	0.04	0.28	.78
Explosions (BART)	0.01	0.01	1.47	.14
Combined BI * SMS	-0.01	0.04	-0.39	.69
Combined BI * Explosions	-0.01	0.01	-1.08	.28

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .18$, *p < 0.05, BI = Behavioural Inhibition

Supplementary Table 9

Linear regression predicting symptom level on the SMFQ mother report with BI, SMS and average adjusted pumps.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.35	0.04	9.04*	.00
Combined BI	0.02	0.06	0.34	.73
Step 2				
Constant	0.24	0.15	1.54	.13
Combined BI	0.02	0.06	0.39	.77
SMS (pubertal status)	0.01	0.04	0.23	.82
Average Adjusted Pumps (BART)	0.02	0.02	0.87	.39
Combined BI * SMS	-0.01	0.04	-0.32	.75
Combined BI * Average AdjPumps	-0.01	0.02	-0.77	.44

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .13$,*$p < 0.05$, BI = Behavioural Inhibition

Linear regressions using maternal report BI and mother SMFQ report as the output variable

Supplementary Table 10

Linear regression predicting symptom level on the SMFQ mother report with BI, SMS and total points.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.32	0.04	7.71*	.00
BI	-0.01	0.06	-0.13	.90
Step 2				
Constant	0.32	0.26	1.25	.22
BI	-0.07	0.07	-0.11	.91
SMS (pubertal status)	0.01	0.04	0.28	.78
Total Points (BART)	0.00	0.00	-0.13	.89
BI * SMS	-0.03	0.04	-0.63	.53
BI * Total Points	0.00	0.00	0.20	.84

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .01$, *p < 0.05, BI = Behavioural Inhibition

Supplementary Table 11

Linear regression predicting symptom level on the SMFQ mother report with BI, SMS and explosions.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.32	0.04	7.71*	.00
BI	-0.01	0.06	-0.13	.90
Step 2				
Constant	0.21	0.16	1.32	.19
BI	-0.07	0.07	-0.11	.92
SMS (pubertal status)	0.02	0.04	0.44	.66
Explosions (BART)	0.01	0.01	0.61	.54
BI * SMS	-0.03	0.04	-0.75	.45
BI * Explosions	-0.01	0.01	-0.76	.45

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .09$, *p < 0.05, BI = Behavioural Inhibition

Supplementary Table 12

Linear regression predicting symptom level on the SMFQ mother report with BI, SMS and average adjusted pumps.

Variable	*B*	*SE B*	*β*	*p*
Step 1				
Constant	0.32	0.04	7.71*	.00
BI	-0.01	0.06	-0.13	.90
Step 2				
Constant	0.23	0.18	1.30	.19
BI	-0.01	0.07	-0.11	.91
SMS (pubertal status)	0.02	0.04	0.39	.70
Average Adjusted Pumps (BART)	0.01	0.02	0.39	.70
BI * SMS	-0.03	0.04	-0.72	.47
BI * Average Adjusted Pumps	-0.01	0.02	-0.49	.62

Note: Step 1: $R^2 = .01$, Step 2: $R^2 = .07$,*$p < 0.05$, BI = Behavioural Inhibition

Appendix C

Additional Analysis of BI and Social Risk-Taking

Supplementary Table 1

Means and Standard Deviations for Social Risk-Taking by BI groups

	BI N = 35		BUI N = 41	
	Mean	SD	Mean	SD
SRTQ Time 1	10.18	1.49	10.49	1.56
SRTQ Time 2	10.04	2.14	10.04	1.92

SRTQ: Social Risk-Taking Questionnaire, BI = Behavioural inhibition

Supplementary Table 2

Differences in Social Risk-Taking across BI groups

	df	*t*	*p*
SRTQ Time 1	74	0.89	.38
SRTQ Time 2	74	-0.01	.99

SRTQ: Social Risk-Taking Questionnaire, BI = Behavioural inhibition

Appendix D

Social Risk-Taking Questionnaire from Chapter 4

Social Risk-Taking Questionnaire (SRTQ)

There are 12 different situations described below, read each one and imagine that it is happening to you. In the box, describe briefly what you would do in each situation.

1. You have just started high school and don't know any of the people in your classes. In first period the teacher places everybody in pairs to complete an assignment. Your partner is nice and you work well together. Later that day, in fourth period, you notice that there is a spare seat next to your partner from earlier. What do you do?

2. There is a new student in your English class who seems nice. When you log onto Facebook that night, you notice that a few of your friends have added the new student as a friend. What do you do?

3. You have joined a new sporting team this year. When you arrive at the first training session, you notice that one of the team members used to be in your team when you were younger. You are not sure if they would remember you. What do you do?

4. You are at the shops, waiting for your mum to finish the grocery shopping. You notice that a kid from school is also waiting for their mum. Although you aren't friends with them you have met them before because they are friends with some of your friends. What do you do?

5. Tonight one of your friends is having a group of kids over for movies and pizza. None of your good friends are going but you have met some of the other kids going. What do you do?

6. There's a kid in your class who you don't know that well, but would like to be friends with. One afternoon after school, when you log onto Facebook, you see they are online. What do you do?

7. After class one day, one of your friends asks you to play basketball. You notice that you don't know any of the other kids playing, but that they look nice. What do you do?

8. Your parents have organised a BBQ with another family. You have met the kids from the other family a few times and they seemed nice. What do you do?

9. At year 7 camp, you are paired up with a kid you haven't met before. You have fun completing the different activities together, and when it's time to go home you swap mobile numbers. What do you do?

10. It's the school holidays and your family is going to the beach for the day. Your mum tells you that you can invite a friend to come along but your best friend is away on holidays with their family. What do you do?

11. You are making the invitations to your birthday party. There is a kid at school who you don't know that well but would like to be friends with. You aren't sure whether you should invite them or not. What do you do?

12. Every Christmas holidays your family goes to the same holiday park. Last year you hung out with the kid who stayed in the cabin next door. When you arrive this year you see that the same family are staying next door. What do you do?

Appendix E

Supplementary Tables from Chapter 5

Supplementary Table 1

Differences in liking of humorous and non-humorous cartoons across child report depressive symptoms

	df	*F*	η^2	*p*
Child SMFQ	1	0.80	.21	.62
Error	35			
Cartoon type	1	24.58	.48	<.001***
Cartoon type * Child SMFQ	1	0.54	.83	.83
Error	35			

SMFQ: Short Mood and Feelings Questionnaire * $p < .0001$

Supplementary Table 2

Differences in liking of humorous and non-humorous cartoons across maternal report depressive symptoms, controlling for maternal age

	df	*F*	η^2	*p*
Maternal SMFQ	1	0.87	.02	.36
Maternal age	1	0.12	.003	.73
Error	35			
Cartoon type	1	1.80	.05	.19
Cartoon type * Maternal SMFQ	1	0.35	.01	.56
Cartoon type * Maternal age	1	0.39	.01	.54
Error	35			

SMFQ: Short Mood and Feelings Questionnaire

Supplementary Table 3

Pearson correlations between SMFQ (child and maternal report) and task liking (square-clicking)

	Square-task liking	
	r	*p*
Child SMFQ	-.02	.89
Maternal SMFQ	.07	.69

SMFQ: Short Mood and Feelings Questionnaire

Supplementary Table 4

Differences in motivation to view humorous cartoons across child report depressive symptoms

	df	*F*	η^2	*p*
Child SMFQ	1	0.30	.10	.97
Error	33			
Motivation	1	4.33	.15	.048*
Motivation * Child SMFQ	1	1.52	.35	.19
Error	33			

SMFQ: Short Mood and Feelings Questionnaire * $p < .05$

Supplementary Table 5

Differences in motivation to view humorous cartoons across maternal report depressive symptoms, controlling for maternal age

	df	*F*	η^2	*p*
Maternal SMFQ	1	0.49	.02	.49
Maternal age	1	1.68	.05	.20
Error	33			
Motivation	1	0.13	.004	.72
Motivation * Maternal SMFQ	1	0.19	.01	.66
Motivation * Maternal age	1	.91	.03	.35
Error	33			

SMFQ: Short Mood and Feelings Questionnaire

Supplementary Table 6

Does liking of the humorous cartoon influence viewing motivation across depressive symptoms – SMFQ child report

	df	*β*	*t*	*p*
Error	31		10.53	.000
SMFQ child	1	-0.17	-1.02	.32
Liking	1	0.32	1.54	.14
Liking * SMFQ	1	0.01	0.02	.98

R^2 = .14, $F(3, 31) = 1.62$, p = .21.

SMFQ: Short Mood and Feelings Questionnaire

Supplementary Table 7

Does liking of the humorous cartoon influence viewing motivation across depressive symptoms – SMFQ maternal report

	df	*β*	*t*	*p*
Error	32		10.09	.000
SMFQ mother	1	-0.03	-0.15	.88
Liking	1	0.30	1.62	.12
Liking * SMFQ	1	0.03	0.14	.89

R^2 = .10, $F(3,32) = 1.16$, p = .34.

SMFQ: Short Mood and Feelings Questionnaire