Examining cognitive risk and protective factors involved in liability to depression

Shiri D. Davidovich

Division of Psychology and Language Sciences
University College London

Thesis submitted for the degree of Doctor of Philosophy

Prepared under the supervision of:
Dr. Frances Rice, Prof. Chris Brewin & Prof. Eamon McCrory

January 2018

Declaration

I, Shiri D. Davidovich, confirm that the work presented in my thesis is my own.

The studies presented in chapters 2 and 3 employed data collected prior to my PhD study. My role included selecting the research questions, reviewing the literature, conducting statistical analyses and the write up of the studies.

The study presented in chapter 4 is based on data I collected during my PhD. My role included selecting the research question, reviewing the literature, preparing and selecting study measures, attaining ethical approval, recruiting the participants, managing all aspects of data collection, conducting statistical analyses and the write up of the study.

The study presented in chapter 5 is based on data collected during my PhD in collaboration with members of the Educational Psychology Research Group of UCL. My role included selecting the research question, reviewing the literature, attaining ethical approval ,developing and piloting the new measure employed in the study, preparing study measures, managing data entry, conducting statistical analyses and the write up of the study.

Abstract

Major depressive disorder is characterized by emotion dysregulation and an imbalance between negative and positive affect. Cognitive impairments may underlie the difficulties with emotion regulation shown in depression. However, additional studies are needed to explore cognitive risk and protective factors for depression, especially with regard to cognitive processes involved in information processing that are assessed using performance based measures. This thesis explored three areas of cognitive processes that have been highlighted as potential risk and protective factors: executive functions, specificity of autobiographical memory and reward processing. These were examined with a focus on individuals at familial risk for depression.

First, the protective role of executive functioning was explored for adolescent offspring of depressed parents. This examination found that in the presence of a current depressive episode in the parent, adolescents with better executive functions had fewer depressive symptoms. Second, specificity in retrieval of autobiographical memory was examined as a predictor of mental health resilience and social functioning in the same high risk sample. This revealed that impairment in retrieving specific memories was associated with lower mood resilience and poorer social functioning over time. Third, associations between reward learning behavior in a social context and indices of familial risk, anhedonia, depressive symptoms and social functioning were examined in a sample of young adult offspring of parents with and without a history of depression. The findings suggested that familial risk, anhedonia and social functioning were associated with specific patterns of reward learning behavior. Finally, due to a lack of measures evaluating reward processing in adolescents, particularly in a social context, I developed and validated a novel measure to assess sensitivity to social rewards in adolescents. Altogether, the findings presented in the thesis advance understanding of cognitive risk and protective factors in depression, especially in the context of individuals at familial risk for depression.

Acknowledgments

I would like to thank my PhD supervisor, Frances Rice, for her guidance, her patience, her endless devotion and for being one of a kind. I am very lucky that I had you to guide me through this challenging journey and I feel truly grateful for everything you have taught me and for all you have given me over the past three years.

I would also like to thank my secondary supervisors, Chris Brewin and Eamon McCrory for their encouragement, their advice, and their great insight. Many thanks to my collaborators, Sandra Dunsmuir, Antonia Cobbald, Stephan Collishaw, Ajay Thapar, Anita Thapar and Gordon Harold for their help and contribution to my work.

A special thanks for my friends from UCL, Elif, Terry, Lucy, Charlie, Nicola, Antoine and Alexandra for their moral support, for their lovely companionship and for the happy moments we spent together, that were a big part of what made my PhD this interesting and meaningful experience that it was.

Finally, I would like to thank my family, and my amazing partner, Tzachi, for all their support, their love, for being my anchor and for always believing in me.

Publications resulting from this work

Davidovich, S., Collishaw, S., Thapar, A. K., Harold, G., Thapar, A., & Rice, F. (2016). Do better executive functions buffer the effect of current parental depression on adolescent depressive symptoms? *Journal of affective disorders, 199*, 54-64.

Other related publications

Rice, F., Davidovich, S., & Dunsmuir, S. (2017). Emotion regulation in childhood and adolescent depression: maintaining equilibrium between positive and negative affect. In C. A. Essau, S. Leblanc, & T. H. Ollendick (Eds.), *Emotion regulation and psychopathology in children and adolescents*: Oxford University Press.

Table of Contents

List of tables

List of figures

CHAPTER 1: General introduction

This introductory chapter begins by presenting general background information about Major Depressive Disorder (MDD), epidemiology of depression, research designs used to assess risk factors for depression, and offspring of depressed parents as a high risk group, and uses this to justify the focus on early onset depression in the offspring of depressed parents which forms the basis of the majority of the empirical chapters included in this thesis. I also briefly discuss difficulties in social functioning as an outcome associated with depression and a potential risk factor for depression in the general population as well as in offspring of depressed parents. I then describe the role of emotion regulation in depression, outline the main aspects of cognitive models of the etiology of depression and discuss the rationale for focusing on research of cognitive processes involved in depression using performance based assessments. Finally, I will review three cognitive processes that are suggested to be involved in emotion regulation and serve as risk or protective factors for depression: executive functions, specificity of autobiographical memory and reward processing. For each of these areas I describe the cognitive processes involved, how they are assessed, and how they may be associated with risk for depression. I will end the review of each cognitive process by describing the rationale for the specific research questions examined in this thesis.

1.1. MDD

MDD is one of the most prevalent mental health disorders, affecting 13-23% of adults during their lifetime (Hasin, Goodwin, Stinson, & Grant, 2005; Kessler & Wang, 2009). It is one of the leading causes of burden and disability worldwide (Chisholm et al., 2016; Üstün, Ayuso-Mateos, Chatterji, Mathers, & Murray, 2004). Furthermore , it has been estimated by the World Health Organization that by 2020 MDD will be the second leading cause of societal burden as assessed by premature mortality and disability (Murray & Lopez, 1996). MDD is characterised by an array of symptoms that include disturbance in mood such as sadness or irritability, reduced positive affect as expressed by decreased interest or pleasure in activities, disruptions in cognitive processes and vegetative functions such as sleep and appetite, and cause functional impairment in areas such as school or work and social relationships (American Psychiatric Association, 2013; World Health Organization, 2015).

1.2. Epidemiology of MDD

MDD in childhood is uncommon with one year prevalence rate estimates ranging between 0.4-2.8% (Birmaher et al., 1996; Costello, Erkanli, & Angold, 2006; Rice & Rawal, 2010). Rates of depression increase substantially during adolescence to one year prevalence rates of 4-8% (Birmaher et al., 1996; Costello et al., 2006; Rice & Rawal, 2010; Thapar, Collishaw, Pine, & Thapar, 2012) and peak by early adulthood, with 20% of young people experiencing a depressive episode by the end of adolescence (Hankin et al., 1998; Stallard et al., 2012; Thapar et al., 2012).

Depression onset during adolescence and early adulthood is associated with a chronic and severe course of symptoms (V. Dunn & Goodyer, 2006; Klein et al., 1999; Levinson, 2006; Thapar et al., 2012; Weissman et al., 1984). Both depressive symptoms and disorder in adolescence and early adulthood are associated with a range of concurrent and future negative outcomes such as poor academic performance, substance use, health problems, psychosocial impairment, unemployment, and suicidal behavior (Angold, Costello, Farmer, Burns, & Erkanli, 1999; Birmaher et al., 1996; Burcusa & Iacono, 2007; Castaneda et al., 2008; V. Dunn & Goodyer, 2006; Fergusson & Woodward, 2002; Newman et al., 1996; Rice, Lifford, Thomas, & Thapar, 2007; Rohde, Lewinsohn, & Seeley, 1994; Thapar et al., 2012).

Adolescence and early adulthood are important vulnerability periods for the onset of depression given that most affected individuals experience their first episode by early adult life (Hankin et al., 1998). This evidence together with the recurrent and debilitating nature of early onset depression highlights the importance of exploring risk and protective factors during adolescence and young adulthood and focusing prevention efforts on these age groups (Horowitz & Garber, 2006; Rice & Rawal, 2010).

1.3. Research designs used to assess risk factors for depression

Several types of designs are used to identify and assess risk and protective factors for depression. Cross sectional studies comparing individuals at low and high risk of developing depression can help identify risk markers that may be present in high risk individuals prior to the development of disorder(Joormann, Talbot, & Gotlib, 2007). However, there is large heterogeneity in high risk groups and some high risk individuals may not develop psychopathology (Mars et al., 2012). Thus, this design can help identify potential risk factors, but cannot determine a causal association between a risk factor and an outcome, as it cannot be established that the risk factor temporally preceded the

emergence of symptoms/disorder. Additionally, recruiting unaffected high risk individuals may bias the sample as these may represent a highly “resilient” group and thus may not serve as a representative sample of high risk individuals (Luking, Pagliaccio, Luby, & Barch, 2015).

Prospective longitudinal studies in population samples and in high risk groups allow risk factors that precede and increase the rates of subsequent depression to be identified. These studies can be conducted as cohort studies, in which an entire population is selected to participate based on a certain criteria and then risk exposure is assessed in all subjects, or as case control studies in which subjects are included based on initial identification of exposure/non exposure to risk factors. Longitudinal studies have the advantages of establishing temporal precedence of the risk factor and controlling for baseline symptom levels. However, longitudinal studies assessing risk and protective factors for depression are observational, and cannot employ random assignment to exposure groups. Therefore such studies cannot completely rule out alternative explanations. This shortcoming is usually addressed by statistically controlling for potential confounders (Dos Santos Silva, 1999; Rutter, 2007).

Intervention studies can be used to assess the effect of a new treatment on a group of patients, or the effect of a preventive strategy on a community sample/high risk group. Thus, this design can be employed to assess whether a particular intervention can reduce disorder rates among disordered individuals, or whether a certain preventive strategy can “immunise” healthy/high risk subjects and reduce the onset of new disorder. An important advantage of intervention studies is that alternative explanations can be ruled out using random assignment and careful choice of control groups (Dos Santos Silva, 1999). For the sake of brevity, designs used to assess genetic risk factors (Rutter, 2007) are not detailed here as these were not the focus of this thesis.

1.4. Offspring of depressed parents

Understanding risk and protective factors in the aetiology of depression is important for developing effective preventive interventions. As mentioned, studies in groups at high risk of developing depression can help identify risk markers that may be present in vulnerable individuals prior to the development of disorder and can be targeted in prevention programmes.

The benefits of exploring high risk groups are also highlighted by evidence to date which indicates superior outcomes from cognitive therapy-based preventive programmes

targeted at high risk individuals compared to universal prevention programs delivered to all regardless of risk profile (Rice & Rawal, 2010). One common and potent risk factor for depressive symptoms and disorder in children and adolescents is depression in a parent (Beardslee, Gladstone, & O'Connor, 2011; Gladstone, Beardslee, & O'Connor, 2011; Pearson et al., 2013). Offspring of depressed parents are around three to four times more likely to develop depression themselves compared to offspring of healthy parents (Garber, 2006; Rice, Harold, & Thapar, 2002; Rice & Rawal, 2010; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997). Depression in the offspring of depressed parents is also characterised by an earlier onset and a more severe and chronic course compared to depression in the general population (Kovacs & Lopez-Duran, 2010; Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002; Rice & Rawal, 2010). Moreover, depression in the parent is also associated with increased risk for other mental health problems such as behavioral problems and anxiety disorders, as well as impairment in social functioning (Beardslee et al., 2011; S. H. Goodman et al., 2011; Weissman et al., 1997; Weissman et al., 2016). It is not yet clear how risk for depression is transmitted from a parent to offspring, but both environmental and genetic factors are likely to be involved (Beardslee et al., 2011; S. H. Goodman & Gotlib, 1999; Singh et al., 2011; Tully, Iacono, & McGue, 2008). Despite evidence of greater depression risk for the offspring of depressed parents there is also substantial heterogeneity in mental health outcomes in this high-risk group and some of these individuals will not go on to develop mental health problems (Beardslee et al., 2011; Collishaw et al., 2016; Mars et al., 2012; Pargas, Brennan, Hammen, & Le Brocque, 2010). This heterogeneity is intriguing and suggests great clinical potential in exploring which risk and protective factors distinguish "resilient" offspring from those who develop mental health problems.

A few factors have been found to be associated with heterogeneity in mental health outcomes in offspring of depressed parents. First, clinical features of parental depression were shown to be associated with offspring mental health outcomes. Thus, previous studies have indicated that an early age of onset, greater severity and chronicity of parental depression, longer duration of depressive episodes and exposure to a recent episode of parental depression are associated with higher rates of MDD as well as other psychiatric problems (such as externalizing problems) in the offspring (Brennan et al., 2000; Foster et al., 2008; Hammen & Brennan, 2003; Klein, Lewinsohn, Rohde, Seeley, & Olino, 2005; Mars et al., 2012; Weissman et al., 1984).

Second, several characteristics of the offspring have also been found to moderate the effect of parental depression on offspring mental health outcomes. Such characteristics include: higher cognitive ability (Pargas et al., 2010; Radke-Yarrow & Sherman, 1990), social competence and good quality of peer relationships (Collishaw et al., 2016; Conrad & Hammen, 1993; Pargas et al., 2010), positive self-esteem (Lewandowski et al., 2014), and adaptive coping skills (Collishaw et al., 2016; Garber & Little, 1999). Recent evidence from longitudinal studies in high-risk individuals showed that cognitive processes which are suggested to be involved in emotion regulation, such as executive functions, reward processing and retrieval of autobiographical memory, are also associated with heterogeneity in mental health outcomes in offspring of depressed parents(Kilford et al., 2015; Rawal, Collishaw, Thapar, & Rice, 2013; Rawal & Rice, 2012) .

Third, characteristics of the family environment and relationships with family members such as parenting style of both the affected parent and the other parent, support of the other parent, and levels of familial cohesion and conflict, were also found to be associated with offspring mental health outcome (Brennan, Le Brocque, & Hammen, 2003; Collishaw et al., 2016; Conrad & Hammen, 1993; Garber & Little, 1999; Pargas et al., 2010).

1.5. Depression and social functioning

MDD is often associated with a pervasive impairment in social functioning (Hirschfeld et al., 2000). Social functioning is one of the main criteria by which level of functional impairment and severity of depression is determined (American Psychiatric Association, 2013; Angold & Costello, 2000). Social functioning has also been found to be associated with risk for depression. Thus, low social support, poor quality relationships and social isolation serve as a predictors of depressive symptoms and disorder in prospective community studies (Santini, Koyanagi, Tyrovolas, Mason, & Haro, 2015; Teo, Choi, & Valenstein, 2013). Social competence, adaptive social functioning and high social support have been shown to protect against depression in individuals exposed to stress or adversity (Feder, Nestler, & Charney, 2009; Masten & Coatsworth, 1998; Southwick, Vythilingam, & Charney, 2005). Furthermore, offspring of depressed parents are characterised by greater social impairment compared to offspring of non-depressed parents (Weissman et al., 1997; Weissman et al., 2016) which may suggest that social impairment precedes and increases risk for depression in this group. Social impairment may also be especially important to consider in the context of risk for depression in

adolescence. As previously mentioned, adolescence is a vulnerability period for developing depression and is characterised by a substantial increase in depression rates compared to childhood (Thapar et al., 2012). During adolescence the social arena becomes highly salient and adolescents show greater sensitivity to social rewards (Davey, Yücel, & Allen, 2008; Foulkes & Blakemore, 2016; Silk, Steinberg, & Morris, 2003). It has been suggested that experiencing social difficulties and interpersonal stress may be one of the factors involved in the increase in depression rates during that time (Davey et al., 2008; Foulkes & Blakemore, 2016; Silk et al., 2003).

1.6. Emotion regulation in depression

Previous research in emotion regulation has highlighted several challenges within this field of study. First, different definitions for emotion regulation exist in the literature (Campos, Mumme, Kermoian, & Campos, 1994; P. M. Cole, Martin, & Dennis, 2004; Gross, 1998; Thompson, 1994). Second, previous studies have often failed to provide a clear differentiation between the constructs of emotion and emotion regulation (P. M. Cole et al., 2004). Third, the examination of emotion regulation requires the assumptions that: 1) A situation in which emotions may emerge has occurred 2) a regulatory process has taken place. These factors are often difficult to assess. Furthermore, the outcome of the type and magnitude of the emotion that emerged in a certain situation does not provide sufficient evidence of the regulatory process that may have occurred (P. M. Cole et al., 2004).

While acknowledging that different definitions exist in the literature, I will describe here one widely accepted definition that suggests that emotion regulation is a set of processes that are employed to modulate the experience, magnitude, duration and expression of emotions. These processes may be automatic and unconscious or effortful and conscious and may be employed to increase, decrease or maintain both negative as well as positive affect (Gross, 1998). Emotion regulation can occur in several stages of the process of emotion generation. Thus, emotion regulation processes can take place before an emotional response has emerged, for example by modifying a certain situation, or selecting an aspect of a situation on which to focus attention, and they can also take place after an emotional response has emerged by modulating the response itself(Gross, 1998).

MDD has been conceptualised as a disorder of emotion dysregulation (Forbes & Dahl, 2005; Gross & Muñoz, 1995). Depression is characterised by sustained negative affect and difficulty in employing adaptive strategies to recover from negative affect

(American Psychiatric Association, 2013; Forbes & Dahl, 2005; Garber, Braafladt, & Weiss, 1995; Silk et al., 2003; Teasdale, 1988). It is also characterised by reduced capacity to experience positive affect which may be expressed as diminished interest in pleasurable activities, or reduced level of enjoyment in pleasurable activities either in the sense of experiencing positive emotions in a weaker manner or in the sense of experiencing them less frequently compared to previous functioning (American Psychiatric Association, 2013; Forbes & Dahl, 2005).

Thus, depression can be described as a disorder that involves an imbalance between negative and positive affect (American Psychiatric Association, 2013; Forbes & Dahl, 2005; Joormann & Quinn, 2014). Impairments in emotion regulation, such as difficulties in employing adaptive strategies to regulate mood or a tendency to respond to negative mood in a manner that may maintain or exacerbate it, have been shown to be associated with current depression and predict future depressive symptoms and disorder (Berking & Wupperman, 2012; Feng et al., 2009; Gotlib & Joormann, 2010; Haga et al., 2012; Joormann & Siemer, 2014; Kraaij, Pruymboom, & Garnefski, 2002; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). This has emphasised the importance of exploring mechanisms that may underlie the difficulties in emotion regulation displayed in depression that may serve as risk and protective factors for the development of depression.

1.7. Cognitive models of the aetiology of depression

A large body of research suggests that depression is a multifactorial disorder, with different causal risk factors and their interactions contributing to the onset of depression. Thus, environmental, genetic, biological, personality and cognitive factors are all likely to affect the aetiology of depression (Rice & Rawal, 2010; Thapar et al., 2012). It has been hypothesised that distal risk factors, such as inherited and environmental factors, may create a predisposition to depression through their effect on proximal risk factors such as biological and cognitive processes (Feder et al., 2009; Thapar et al., 2012).

The traditional cognitive models of depression focused on cognitive vulnerabilities as risk factors for depression. The original cognitive model of depression by Beck defined cognitive vulnerabilities as maladaptive negative schemas, which are cognitive structures that include dysfunctional attitudes and beliefs about the self, the world and the future regarding interpersonal relationships and achievements. These cognitive structures were suggested to orient the individual's information processing and shape the interpretation of

events (Beck, 1967, 2008; Beck, Brown, Steer, & Weissman, 1991). Different theoreticians have emphasised different cognitive vulnerabilities in their models. For example, whereas Beck focused on dysfunctional attitudes as an important cognitive vulnerability factor(Beck, 1967, 2008; Beck et al., 1991), Abramson focused on a negative attribution style (i.e. a tendency to attribute negative events to internal, stable and global causes) and hopelessness as cognitive vulnerabilities that explain predisposition to depression (Abramson, Metalsky, & Alloy, 1989; Abramson, Seligman, & Teasdale, 1978). Thus, these traditional models have mostly focused on negative cognitions that could be assessed using self-report measures as cognitive vulnerability factors for depression. Furthermore, it has been suggested that the criteria for defining such cognitive vulnerabilities for depression include: 1) demonstrating temporal precedence to depression, 2) having relevant construct validity, 3) showing predictive specificity to depression (vs. other disorders), and 4) being an internal characteristic of the individual (Alloy et al., 1999; Ingram, Miranda, & Segal, 1998; Jacobs, Reinecke, Gollan, & Kane, 2008). Most of the theoreticians exploring these models have also employed a diathesis stress hypothesis in their models, which posits that an interaction between cognitive vulnerability factors and environmental negative events or stressors increases risk to depression (Jacobs et al., 2008). For example, negative life events may activate negative schemas which distort information processing and lead to depressive symptoms such as sad mood.

Developmental accounts of depression such as those suggested by Beck (1976, 2008) or Hammen (1992, 2000) posit that early adverse life events or experiences contribute to the formation of cognitive vulnerabilities, and these in turn affect emotion regulation and increase risk for depression when encountering negative or stressful events (Beck, 2008). This hypothesis has been given support from several lines of evidence: various studies have shown that early adversity such as negative life events, childhood maltreatment and negative parenting practices are associated with the development of cognitive vulnerabilities such as dysfunctional attitudes, negative attributional style and increased level of helplessness in youth (Abela & Hankin, 2008). Moreover, diathesis stress models that suggest that individuals with certain cognitive vulnerabilities are more likely to experience depressive symptoms when facing stressful events have received empirical support from studies in both adults and adolescents (Abramson et al., 1999; Ingram et al., 1998; Lakdawalla, Hankin, & Mermelstein, 2007; Scher, Ingram, & Segal, 2005).

As mentioned previously, although traditional depression theories and research referred to aspects of information processing in models of the aetiology of depression, the main focus of the examination of cognitive vulnerability factors was negative cognitions that could be assessed using self-report measures. However, the contribution of shared method variance to observed associations is a significant concern when the same informant reports on a potential risk factor (e.g. attributional style) and outcome (i.e. depressive symptomatology) (Jacobs et al., 2008; Rice et al., 2015; Rutter, Pickles, Murray, & Eaves, 2001). Studies of cognitive vulnerability to depression have therefore begun to include more basic cognitive processes that are related to information processing and are often unconscious (Jacobs et al., 2008; Mathews & MacLeod, 2005; Scher et al., 2005). Examples of such cognitive processes that have been explored are executive functioning, attention, memory, interpretation and reward processing (Beck et al., 1991; Eshel & Roiser, 2010; Gotlib & Joormann, 2010; Jacobs et al., 2008; Roiser, Elliott, & Sahakian, 2012; Scher et al., 2005). These cognitive processes have been examined in experimental paradigms using performance based measures. This is the approach that I take in the majority of empirical chapters included in this thesis and I outline a rationale for this below.

1.8. A rationale for focusing on cognitive processes in exploring risk and protective factors for the development of depression

Several lines of evidence highlight the clinical importance of focusing on cognitive processes in research of risk and protective factors for the development of depression. Firstly, previous research has supported the role of cognitive processes in the aetiology of depression. A range of cognitive vulnerabilities in areas such as cognitive style, affective processing and memory have been found to be present in individuals at high risk for depression (Chan, Goodwin, & Harmer, 2007; Jaenicke et al., 1987; Joormann, Talbot, et al., 2007; Rijsdijk et al., 2009; Roiser et al., 2012; Woody, Burkhouse, & Gibb, 2015). Longitudinal studies have also shown that cognitive factors such as negative attributional style and dysfunctional attitudes, and impairments in memory, executive functions and reward processing, show temporal precedence and predict the subsequent development of depressive symptoms and disorder in adolescents and adults (Castaneda et al., 2008; Forbes, Shaw, & Dahl, 2007; Jacobs et al., 2008; Kilford et al., 2015; Letkiewicz et al., 2014; Rawal et al., 2013; Rawal & Rice, 2012; Scher et al., 2005). Secondly, there are theoretical reasons to suppose that cognitive processes may be important resources that

can protect against the development of depressive symptomatology. For instance, cognitive processes may be important in employing adaptive coping and emotional regulation strategies, and cognition may be an important resource that allows individuals to deal with stressful events more effectively (Feder et al., 2009; Riglin, Collishaw, et al., 2016; Southwick et al., 2005). Thirdly, cognitive factors may be amenable to change and thus can be targeted in interventions (Diamond & Lee, 2011; Garber, 2006; Giesen, Searle, & Sawyer, 2007). In line with this, effective interventions for treating and preventing depression, such as cognitive behavioral therapy, are based on cognitive restructuring techniques aimed at challenging and modifying negative cognitive styles (Forkmann et al., 2014; Friedberg & McClure, 2015; Garber et al., 2009; Rice & Rawal, 2010; Stice, Shaw, Bohon, Marti, & Rohde, 2009). Furthermore, initial evidence suggests that both psychological interventions and antidepressant medications modify cognitive processes such as negative biases and reward processing (Harmer, Goodwin, & Cowen, 2009; Roiser et al., 2012). Such findings are therefore consistent with assertions that cognitive processes may be proximal processes involved the development (and the amelioration) of depressive symptoms (Thapar et al., 2012).

A large body of research has explored cognitive risk factors using self-report measures. Although the assessment of some cognitions requires subjective report, employing only self-report measures may not allow some aspects of information processing to be assessed, and has some notable limitations compared to performance-based measures. Self-report measures can assess cognitive processes that depend on awareness, whereas performance-based measures can also objectively assess processes that the individual may not be aware of or can be assessed only based on overt behavior. Self-report measures of cognitive processes may be prone to biases and may not be independent of reports on depressive symptoms, as usually the same individual reports on both. Thus, shared method variance may contribute to associations where the same informant reports on a cognitive risk factor and a depressive outcome. A important theoretical justification for exploring information processing deficits has been given by recent models of depression that suggest that such lower order cognitive processes may contribute to the development of higher order cognitive factors such as negative schemas (Jacobs et al., 2008; Roiser et al., 2012). Thus, exploring these may serve an important role in understanding the aetiology of depression. Finally, it is worth noting that self-report measures may not always allow cognitive vulnerability factors to be assessed in young people as it is not always clear whether children can consciously report on cognitive

processes to the same extent as adults (Harmer, O'Sullivan, et al., 2009; Jacobs et al., 2008; Rutter et al., 2001). It has been suggested that cognitions associated with depression in adults may only stabilize and serve as a vulnerability factor for depression during adolescence (D. A. Cole et al., 2008; LaGrange et al., 2008; J. E. Turner & Cole, 1994). Furthermore, introspection on cognitive processes requires meta-cognitive abilities which start to develop during childhood and continue to mature until young adulthood (Best & Miller, 2010; King & Kitchener, 2015; Weil et al., 2013). Thus, it is possible that using self-report measures to assess cognitive vulnerabilities for depression may not always be age-appropriate for children and young people.

Given the evidence described above, and as fewer studies have used performance-based measure to assess cognitive risk and protective factors for depression, in this thesis I will focus on cognitive processes assessed using performance-based measures. Specifically, I will focus on three areas that show promise in research of risk and protective factors for depression: executive functioning, reward processing and specificity in retrieval of autobiographical memory. These were selected because they show some of the characteristics that have been suggested for defining cognitive vulnerability factors for depression, which may indicate that they are causally involved in the development of depression (Alloy et al., 1999; Ingram et al., 1998; Jacobs et al., 2008). Namely, there is evidence to suggest that these cognitive factors exist prior to and longitudinally predict the onset of depressive symptomatology. Furthermore, these are all internal characteristics of the individual that have been shown to be associated with emotion regulation, and specifically with difficulties in emotion regulation observed in depression. I next review the evidence to suggest the importance of each of the selected cognitive processes in the development of depression.

1.9. Executive functions

Executive functions are a set of mental processes that facilitate goal directed behaviour, planning, making choices and adapting to novel or complicated situations. Executive functions are therefore inherently important to many areas of adaptive functioning, including academic and professional performance, social relationships and emotion regulation (Diamond, 2013; Suchy, 2009).

There is substantial variation across the literature in which cognitive processes are defined as "executive functions". However, it is widely agreed that three core executive functions are inhibitory control, working memory and mental flexibility (Diamond, 2013).

Inhibitory control involves the ability to exercise control over behaviour, attention, thoughts and emotions in order to make a decision about the appropriate response in a given situation and overcome an automatic or habitual response. Working memory involves the ability to hold relevant information in mind, to update it and manipulate it in order to make a decision or solve a problem. Mental flexibility involves the ability to change or switch between perspectives or responses in order to adapt to a new situation or demand (Diamond, 2013). These three core executive functions are also inter-correlated. For example, working memory depends on inhibitory control, as in order to stay focused on a current task or goal one needs to inhibit irrelevant and distracting material from "occupying" the working memory. Mental flexibility also relies on working memory and inhibitory control. Thus, when switching between different solutions to a problem, one needs to inhibit a previously considered idea, shift to a new idea and hold the relevant one in working memory (Diamond, 2013). It has been suggested that other higher order cognitive processes such as planning, reasoning and problem-solving, which are also often defined as executive functions, may build on the three core executive functions (Davidson, Amso, Anderson, & Diamond, 2006; Diamond, 2013; Miyake et al., 2000; Suchy, 2009). As research on depression is focused on information processing in the context of emotional material, executive functioning tasks in depression studies often involve emotional stimuli such as negative and positive words, or happy and sad faces.

1.9.1. Assessment of executive functions

There are numerous tasks used to assess various aspects of executive functioning. As it is beyond the scope of this thesis to review in detail all the executive functioning tasks, I briefly describe the main features of common paradigms used to assess the three core executive functions in Table 1.1.

Table 1.1. Common paradigms used to assess executive functioning

Aspects of executive functioning	Common paradigms used to assess aspects of executive functioning
Inhibitory control	Paradigms that require the subject to respond in a certain condition while inhibiting a response in a different condition (such as the go/no go task) or to use a decision rule that requires inhibiting a natural or a proponent response (such as the Stroop task) (Cragg & Nation, 2008; MacLeod, 1991).
Working memory	1) Tasks that require subjects to repeat numbers or items that they have just heard and reorder them according to a rule (for example, reordering numbers by size). 2) Complex span tasks such as counting span or reading span where the subject is required to repeat and recall a list of items (such as digits, letter or words) while performing another concurrent task such as counting, doing simple mathematical operations or reading sentences (Hale et al., 2011).
Mental flexibility	1) Set-shifting tasks in which the subject is required to shift quickly between two or more decision rules or stimulus dimensions, such as the Wisconsin Card Sorting Test (Stuss et al., 2000). 2) Tasks such as the verbal fluency task or the alternative uses task[a] in which the subject is required to generate as many items as possible from a certain category (e.g. naming words that start with the letter F) or as many solutions as possible to a problem (e.g. suggesting possible uses for a common household item).

Note: [a] It is suggested that these tasks tap an aspect of mental flexibility that is related to creativity as they require the subjects to generate different strategies and categories from which to extract words or solutions (Baldo, Shimamura, Delis, Kramer, & Kaplan, 2001; Diamond, 2013; Gilhooly, Fioratou, Anthony, & Wynn, 2007).

1.9.2. Executive functioning and risk for depression

Cognitive theories of depression suggest that depression is characterized by difficulties in regulating emotions and in cognitive control of negative thought and affect. Moreover, automatic and uncontrollable negative thoughts, negative biases and a ruminative thinking style, which characterise depressed individuals, are cognitive mechanisms that maintain and intensify negative affect and have been found to predict depressive symptoms and disorder (Abela & Hankin, 2008, 2011; Gotlib & Joormann, 2010; Joormann & Gotlib, 2008; Joormann & Quinn, 2014; Kwon & Oei, 1992; Nolen-Hoeksema, 2000; Olioff, Bryson, & Wadden, 1989; Robinson & Alloy, 2003). A large body of research suggests that executive functioning is essential for emotion regulation and that executive functioning impairment may underlie the difficulties in cognitive control of emotion shown in depression, and particularly the cognitive control of negative emotion.

Executive functioning impairments may be implicated in emotion regulation and risk for depression in several ways. Studies with depressed individuals indicate that they show impairment in inhibitory control when processing emotional information and have difficulties in updating working memory and removing negative material from working memory (Harvey et al., 2004; Joormann & Gotlib, 2008). Difficulties with inhibitory control, updating working memory and ignoring irrelevant information may interfere with disengaging from negative thoughts and emotions, maintaining goal directed behaviour, and recovering from negative mood.

Previous studies have also shown that depression is associated with difficulties in cognitive flexibility when processing emotional material (Deveney & Deldin, 2006; Murphy, Michael, & Sahakian, 2012). Mental flexibility may enable the individual to employ affective regulation strategies such as shifting between different perspectives or interpretations of a stressful situation, using distraction to reduce emotional reactivity, and exercising efficient problem solving by generating possible solutions to a problem (Hendrawan, Yamakawa, Kimura, Murakami, & Ohira, 2012; McRae, Jacobs, Ray, John, & Gross, 2012; Southwick et al., 2005). Applying effective mood regulation strategies may rely on aspects of executive functioning (Gross, 1998; Kross, Ayduk, & Mischel, 2005; McRae et al., 2012; Ochsner & Gross, 2008; Sanchez, Vazquez, Marker, LeMoult, & Joormann, 2013). For example, reappraisal - an important mood regulation strategy - may require inhibitory control in order to disengage from a specific interpretation of a stressful situation, working

memory to hold different options in mind, and mental flexibility in order to generate several interpretations and shift to one that may enable better coping with the situation and alleviate mood (Kross et al., 2005; Ochsner & Gross, 2008). In line with this hypothesis, impairments in inhibitory control, the updating of working memory and set shifting have been found to be associated with higher levels of rumination (i.e. repetitive thinking about the symptoms, causes and consequances of one's negative emotional state; Nolen-Hoeksema, 1991), while better working memory and set shifting ability may be associated with use of reappraisal which is a more adaptive emotional regulation strategy (De Lissnyder, Koster, Derakshan, & De Raedt, 2010; Joormann & Quinn, 2014; McRae et al., 2012).

A large body of research has shown that depressed adults show impairments in different components of executive functioning such as the updating of working memory, inhibitory control and mental flexibility, especially when processing emotional information (Gotlib & Joormann, 2010; Harvey et al., 2004; Joormann & Gotlib, 2008; Murphy et al., 2012; S. Wagner, Doering, Helmreich, Lieb, & Tadić, 2012). Furthermore, a recent meta - analysis (S. Wagner et al., 2012) found that adults with MDD showed worse performance compared to controls on a range of executive functioning tasks assessing inhibitory control and various aspects of mental flexibility such as set shifting and mental generativity. The most pronounced effects were found for tasks assessing inhibitory control and set shifting ability although it is worth noting that the meta-analysis did not examine working memory.

A smaller number of studies have examined executive functioning impairments in depressed children and adolescents. These studies have shown that impairments in executive functioning are also present in depressed youth, although findings are less consistent than those for adults. For example, in a study of children and adolescents, Ledouceur (2005) found that subjects with MDD showed impairment in inhibitory control for negative material compared to healthy controls. Kyte et al. (2005) found that recently depressed adolescents showed difficulties with inhibitory control compared to healthy adolescents, but did not differ on their performance on a set shifting task. Favre (2008) on the other hand found no evidence of group differences in measures of inhibitory control and mental flexibility between currently depressed children and adolescents and controls. It is worth noting that inconsistency of findings in the small number of studies in youth may be related to variation in the methods used to assess depressive symptomatology (i.e. self-report questionnaires vs. diagnostic interviews) and executive functioning. A recent meta-analysis (S. Wagner, Müller, Helmreich, Huss, & Tadić, 2015) reported that children

and adolescents with MDD performed significantly worse compared to healthy controls on measures of inhibitory control, working memory and mental flexibility, with the most marked effects found for measures of inhibitory control. However, evidence of executive functioning impairment in currently depressed individuals does not clarify whether such impairments are a cause or a consequence of depression.

Support for the role of executive functioning in affect regulation has been given by several studies in community samples of children and adults which demonstrated that higher executive functioning abilities are associated with better emotion regulation, as assessed by negative affective responses when facing a negative or stressful event in experimental tasks such as watching aversive video clips (Schmeichel & Tang, 2014). Furthermore, although only a small number of studies examined executive functioning as a risk factor for depression, initial evidence suggests that executive functioning impairment in the context of emotional processing may play a casual role in the onset of depression. Joormann et al. (2007) reported an attentional bias toward negative material (which may reflect difficulties in inhibitory control of attention) in the female offspring of depressed mothers with no personal history of depression. This suggests that such an attentional bias exists in those at elevated risk of depression prior to the development of disorder. Moreover, several longitudinal studies also give support to executive functioning impairments as a risk factor for depression. For example, in a short term longitudinal study, Letkiewicz et al. (2014) reported that working memory deficits were associated over time with increases in depressive symptoms in a sample of undergraduate students. In another short term prospective study in a community sample of children and adolescents, Evans et al (2016) reported that working memory impairment predicted increases in depressive symptoms over time and that there was an indirect effect of mental flexibility on depressive symptoms through coping. Finally, in a study of the adolescent offspring of depressed parents, Kilford et al. (2015) reported that difficulties in inhibitory control in an affective task predicted new onset depression one year later when controlling for baseline depressive symptoms.

This evidence suggests that executive functions are involved in emotion regulation and therefore better executive functions may serve as a protective factor for high-risk individuals, such as the offspring of depressed parents. However, to date, only a small number of studies have examined executive functions as a potential risk factor in this group (Joormann, Talbot, et al., 2007; Kilford et al., 2015) and the protective effect of executive functioning in this group has not been systematically explored in previous

studies. A previous longitudinal study in a community sample of adolescents found no buffering effect for set shifting (as assessed by a performance based task) on the association between events of family adversity and internalising symptoms (Bakker, Ormel, Verhulst, & Oldehinkel, 2011). However, it is important to note that in this study, set shifting, events of family adversity and internalising symptoms were measured at different times, such that set shifting was assessed at a baseline assessment, internalising symptoms were assessed at a follow-up assessment a few years later, and the presence of events of family adversity between baseline and follow up were also assessed at follow-up. It has been suggested that the protective effect of executive functions can be observed when demands for emotion regulation are high, for example when experiencing stress or adversity (Bakker et al., 2011; Muris, van der Pennen, Sigmond, & Mayer, 2008). Therefore it seems reasonable to explore the protective effect of executive functioning in offspring of depressed parents in the context of an ecologically valid source of adversity/ stress that may challenge emotion regulation, such as exposure to a current episode of parental depression. Thus, in chapter 2 I examined the buffering role of executive functioning in the association between the presence of current parental depressive episode and adolescent depressive symptoms.

1.10. Autobiographical memory

Autobiographical memory is a facet of memory that involves one's past personal experiences. Autobiographical memory is of central importance in establishing a coherent sense of self, guides goal directed behaviour, and contributes to problem solving and effective processing of emotional material in light of past experiences (Valentino, 2011; Williams et al., 2007). One aspect of autobiographical memory that has been of importance to research of mental health disorders, and specifically depression, is the specificity of autobiographical memory, meaning, the ability to recall memories of experiences from a specific time and place. Overgeneral memory (OGM) refers to a difficulty retrieving specific memories that is expressed as a tendency to retrieve memories that refer either to categories of repeated events (for example: "I go to the gym every week to exercise") or to things that occurred over extended periods of time (for example: "I did not get good grades in college") (Rawal & Rice, 2012; Williams et al., 2007). OGM has been shown to characterise depressed adults and adolescents (Park, Goodyer, & Teasdale, 2002; Rawal & Rice, 2012; Valentino, 2011; Williams et al., 2007).

Recent accounts of OGM suggest that autobiographical memories are organised hierarchically. When an individual is required to retrieve a specific memory, a generative retrieval is required in which a search is carried out through the hierarchy. It is suggested that executive functioning resources are used to check whether representations that are activated match the required criteria, and to adapt the search and inhibit irrelevant memories. Theories of OGM hypothesise that in individuals with OGM, the search is truncated at a general level, before retrieving a specific memory (Conway & Pleydell-Pearce, 2000; Williams et al., 2007).

The widely accepted model for OGM in depression, the CaR-FA-X model (Williams et al., 2007), suggests that three mechanisms underlie the emergence of OGM in depressed individuals: 1) Capture and Rumination 2) Functional Avoidance 3) impairment in executive functioning. Depressed individuals often have negative schemas (cognitive structures of negative beliefs about the self, the world and the future) and are prone to rumination, a repetitive thinking style, which exacerbates the activation of these schemas (Nolen-Hoeksema et al., 2008; Williams et al., 2007). Thus, according to the CaR-FA-X model, as the search process starts from a general, conceptual level, this may activate negative schemas and rumination, which may result in the "capture" of attention and interference in the retrieval process. Another mechanism that is suggested to underlie OGM is functional avoidance, whereby the search for memories is truncated at a general level to avoid retrieving a specific memory that may include sensory and perceptual details of an event and thus may induce negative mood and interfere with goal directed activity. This functional avoidance is thought to occur in particular in those with a history of traumatic or negative events. For some individuals this may be a flexible strategy, used to reduce negative affect, whereas for some individuals it may turn to an inflexible response style which may be generalised to neutral and positive memories as well. A third mechanism that has been suggested to contribute to the emergence of OGM is reduced executive functioning resources. As mentioned earlier, the retrieval of specific autobiographical memories relies on the use of executive functions. It has been demonstrated in previous research that depressed individuals, as well as those at high risk, show impairments in various aspects of executive functioning (Conway & Pleydell-Pearce, 2000; Joormann, Talbot, et al., 2007; Kilford et al., 2015; S. Wagner et al., 2012; S. Wagner et al., 2015). Impairments in executive functioning may affect one's ability to generate strategies and search descriptions in order to map the memory, inhibit irrelevant information and hold the relevant information in working memory (Conway & Pleydell-

Pearce, 2000; Hertel & Hardin, 1990; Williams et al., 2007; Zacks & Hasher, 1994). Thus, those with executive functioning deficits may experience greater difficulties in retrieving specific memories.

A recent developmental psychopathology model by Valentino (2011) suggests that environmental factors related to a child's culture, community and family, as well personal characteristics of the child, are likely to contribute to the emergence of OGM. Previous studies exploring environmental effects have mostly examined associations between a history of trauma and OGM. To date, other potential environmental effects that have been suggested as important in theoretical accounts of OGM, such as negative life events, parental depression and aspects of parent-child relationships (Valentino, 2011; Williams et al., 2007; Woody et al., 2015), have hardly been examined in previous studies.

1.10.1. Assessment of specificity of autobiographical memory

OGM is usually assessed using the Autobiographical Memory Test (AMT; Williams & Broadbent, 1986) in which participants are given a cue word and requested to retrieve a memory that occurred at a specific time and place. Participants are given a time limit for each cue word, such as 30 seconds or 1 minute. Answers are typically categorised as specific or overgeneral (either categorical memories that refer to categories of repeated events or extended memories that refer to things that occured over extended periods of time; Williams et al., 2007).

1.10.2. Specificity of autobiographical memory and risk for depression

OGM may increase risk for depression through several pathways. It has been suggested that autobiographical memory may serve as a personal knowledge base that can be used in everyday life to respond adaptively to challenging events and assist in problem solving (Conway & Pleydell-Pearce, 2000). In line with this hypothesis, several studies have shown that impairment in retrieving specific autobiographical memories in adults is associated with lower performance in problem solving tasks (Goddard, Dritschel, & Burton, 1996; Pollock & Williams, 2001; Raes et al., 2005).

Support for the role of specificity of memory in emotion regulation is also provided by studies which examined abstract vs. concrete thinking in depression. It has been suggested that rumination, a core cognitive mechanism in the aetiology of depression, will have a negative effect on mood regulation specifically when it involves an abstract rather than a concrete self-focus. It is hypothesised that an abstract and more conceptual way of

thinking involves less vivid imagery, hinders emotional processing of negative events and is less effective for problem solving, whereas concrete thinking allows generating and applying specific solutions to problems (Davidson et al., 2006; Stöber & Borkovec, 2002; Watkins & Moulds, 2005). The distinction between overgeneral and specific memories is suggested to represent a specific example of the distinction between abstract and concrete thinking. The advantages of concrete thinking compared to abstract thinking have been shown in studies which demonstrated that encouraging individuals to think in a concrete way compared with an abstract way reduced negative mood following a stress induction in healthy adults (Watkins, Moberly, & Moulds, 2008) and improved social problem solving in depressed subjects (Watkins & Moulds, 2005).

Overgeneral memory may also hinder the use of memory as a mood repair strategy. The recollection of positive memories has been demonstrated to be an effective mood regulation strategy used to counterbalance negative mood (Dalgleish & Werner-Seidler, 2014; Erber & Erber, 1994; Rusting & DeHart, 2000). However, previous studies have shown that mood regulation using positive memories is less effective in depressed individuals (Joormann & Siemer, 2004; Joormann, Siemer, & Gotlib, 2007). Although the reasons for these findings are not clear, it seems plausible that difficulties in retrieving details of specific memories may impede the ability to use positive memories to repair mood. This hypothesis was given some support by studies which have shown that following a sad mood induction, individuals with a history of MDD retrieved positive memories which had lower vividness and lower emotional intensity compared to those retrieved by healthy controls (Werner-Seidler & Moulds, 2011, 2012).

A large body of research in adults has indicated that depression is associated with a tendency to retrieve overgeneral memories (Kuyken & Dalgleish, 1995; R. G. Moore, Watts, & Williams, 1988; Williams et al., 2007; Williams & Scott, 1988). Fewer studies have examined over-general autobiographical memory in children and adolescents. However, evidence suggest that difficulty in retrieving specific autobiographical memories also characterises depressed children and adolescents (Kuyken, Howell, & Dalgleish, 2006; Park et al., 2002; Rawal & Rice, 2012; Vrielynck, Deplus, & Philippot, 2007).

Recent studies have also supported the role of overgeneral memory as a risk factor for depression. Firstly, a study by Woody et al. (2015) showed that overgeneral memory differentiates children at high and low familial risk of depression. Specifically, the study showed that never–depressed children of mothers with a history of MDD retrieved more overgeneral autobiographical memories in response to negative cue words compared to

children of healthy mothers. This suggests that OGM exists in high-risk individuals prior to the development of depression and may increase risk for depression. Secondly, two longitudinal studies in adolescents have shown that OGM predicted depressive symptoms (Hipwell, Sapotichne, Klostermann, Battista, & Keenan, 2011; Rawal & Rice, 2012) and new onset depressive disorder over time (Rawal & Rice, 2012).

As mentioned, it has been suggested that autobiographical memory serves as a personal knowledge base which can be used for problem solving, emotional processing, and coping with challenging situations by reflecting on past experiences (Conway & Pleydell-Pearce, 2000; Williams et al., 2007). Therefore, it seems likely that the specificity of autobiographical memory will be also associated with positive outcomes such as adaptive social functioning and mental health resilience. However, previous studies have mostly examined specificity of autobiographical memory in the context of the presence of disorders such as depression and post-traumatic stress disorder (Williams et al., 2007). Thus, the protective effect of specificity of autobiographical memory has not been examined in the context of positive outcomes in high-risk groups and particularly in offspring of depressed parents. Therefore, in chapter 3 I aimed to examine the protective effect of specificity of autobiographical memory by examining longitudinal associations with social functioning and mental health resilience in adolescent offspring of depressed parents. Furthermore, as mentioned, hardly any previous studies have examined environmental factors that may affect the emergence of OGM, beside a history of trauma. Thus, as a secondary aim, I will also examine in chapter 3 whether severity of parental depressive symptoms and negative life events are associated with OGM in adolescent offspring of depressed parents.

1.11. Reward processing

Reward processing is thought to underlie behaviour, cognition and affect related to the pursuit and experience of pleasure. The pursuit of rewarding experiences is suggested to drive engagement in pleasurable activities, to lie at the heart of the innate need to form significant relationships, and is likely to be involved in decision making in aspects of everyday life such as work and academic performance (Forbes, 2009; Forbes & Dahl, 2005, 2012; Rawal et al., 2013). Reward processing is an umbrella term that is suggested to include several dissociable components such as reward learning, motivation for rewards, and the hedonic experience upon receiving a reward (which can also be referred to as reward "liking") (Berridge & Robinson, 2003; Sherdell, Waugh, & Gotlib, 2012).

Reward learning can refer to several types of learning. Some forms of learning are associative and involve acquiring an association between two stimuli or a stimulus and response, such as in Pavlovian conditioning, or between an action and a stimulus, such as in instrumental learning. Other, more developed "cognitive" forms of learning involve awareness and knowledge about causal, temporal, and spatial aspects of relationship between actions and stimuli. Reward learning enables individuals to predict and anticipate rewards, meaning to learn what type of reinforcement is likely to be received as a consequence of an action. Thus, reward learning is involved in goal directed behaviour. Motivation for reward is the desire to receive a certain reward and can also be referred to as the "wanting" of reward. The level of motivation one has to receive a certain reward is likely to affect the level of effort which may be invested in pursing that reward. Reward "liking" is the conscious subjective response of pleasure, which one experiences upon receiving a reward or experiencing a rewarding event (Berridge & Robinson, 2003).

The different aspects of reward processing are inter-correlated and likely to affect one another in a number of ways. For example, a stimulus or an experience that has a higher rewarding value is likely to be pursued with greater motivation compared to one with a lower rewarding value. Reward learning will enable the individual to distinguish between rewards of different magnitudes and thus will contribute to the level of motivation to receive different rewards (Berridge & Robinson, 2003).

1.11.1. Assessment of reward processing

A range of tasks have been used in previous behavioural studies to assess specific elements of reward processing. These are summarised in Table 1.2.

Table 1.2. Common paradigms used to assess reward processing

Aspects of reward processing	Common paradigms used to assess aspects of reward processing
Reward learning	Probabilistic reward tasks that involve signal detection paradigms. In these tasks 1 of 2 stimuli appears quickly on the screen and the subject is required to decide which one appeared (for example a stimulus of a long or short mouth). Correct identification of each stimulus is rewarded at different frequencies such that one stimulus is more frequently reinforced than the other. In the general population this leads to a response bias where subjects tend to more often choose the more frequently rewarded stimulus when asked to choose which of the two possible stimuli appeared on the screen. Thus in this task, response bias serves as a measure of reward learning (Pizzagalli, Jahn, & O'Shea, 2005)
Motivation for receiving rewards	Tasks such as the Effort-Expenditure for Rewards Task (EEfRT; Treadway, Buckholtz, Schwartzman, Lambert, & Zald, 2009). In each trial the subject has to choose between an easy task and a hard task which have different demands for speeded button pressing. The hard task is rewarded with greater monetary reward than the easy task. Different trials are rewarded at different probabilities (low, medium and high). The task assesses the effects of reward magnitude, reward probability and their interaction on subjects' choices between the easy and hard task (i.e. on subjects' willingness to exert effort in order to receive rewards).
Reward "liking" or the hedonic experience when receiving rewards	1) Subjective ratings of the affective value of positive/rewarding stimuli (i.e. how positive a stimuli is or how much it affects the subject's mood; Treadway & Zald, 2011). 2) Self-report questionnaires that assess general hedonic capacity, such as the Snaith-Hamilton Pleasure Scale (SHAPS; Snaith et al., 1995).
Reward seeking behaviour[a]/risk taking	Tasks which examine risk taking as function of sensitivity to probability and magnitude of reward such as the Cambridge Gambling Task (www.camcog.com; Rawal et al., 2013). In this type of task, risk taking is measured as the amount of points or money the subject is willing to bet at trials with different probabilities of gain.

Note: [a] Reward seeking behavior is likely to be related to a few elements of reward processing mentioned previously such as reward learning, motivation to pursue rewards of different magnitude (represented as in some tasks as level of risk taking) or reward "liking"(Berridge & Robinson, 2003; Forbes & Dahl, 2012; Rawal et al., 2013; Treadway & Zald, 2011).

1.11.2. Reward processing and risk for depression

Depression is characterized by dysregulation of both negative and positive affect. Despite the substantial focus on dysregulation of negative affect in depression research, the diminished positive affect shown in depression, and related symptoms such as reduced interest and enjoyment of pleasurable activities (i.e. anhedonia) and social withdrawal, may be of equal importance for understanding risk factors for the onset depression as well as the maintenance of symptoms and functional impairment of depressed individuals (Forbes & Dahl, 2005).

Positive affect is imperative for adaptive functioning and psychological well-being (Forbes & Dahl, 2005; Fredrickson & Joiner, 2002) and is important for social functioning as it plays a pivotal role in initiating and preserving social bonds (Campos et al., 1994; Forbes & Dahl, 2005). For instance, the temperamental trait of low positive affectivity has been shown to be associated with current depression in children and adults (Clark, Watson, & Mineka, 1994; Compas, Connor-Smith, & Jaser, 2004; Klein, Durbin, Shankman, & Santiago, 2002) and to predict depressive symptoms over time in children (Dougherty, Klein, Durbin, Hayden, & Olino, 2010; Lonigan, Phillips, & Hooe, 2003). As positive affect may be considered as an emotional state driven by reward (Forbes & Dahl, 2005; Rolls, 2000; Schultz, 2000), alterations in reward processing are thought to underlie the reduced positive affect, anhedonia and social withdrawal observed in depression (Forbes & Dahl, 2005, 2012; Pizzagalli, 2014).

Anhedonia is suggested to be an important marker for severity and outcome of depression. Longitudinal studies in both adolescents and adults have shown that anhedonia is a predictor of severity and recurrence of depression (McMakin et al., 2012; Spijker, Bijl, De Graaf, & Nolen, 2001). Moreover, anhedonia may be problematic to treat, as previous studies show that Selective Serotonin Reuptake Inhibitors (SSRIs), a common type of antidepressant administered to depressed patients, often has poor treatment response in patients with anhedonia and motivational difficulties (Dunlop & Nemeroff, 2007; McCabe, Mishor, Cowen, & Harmer, 2010; Nutt et al., 2007; Price, Cole, & Goodwin, 2009; Shelton & Tomarken, 2001). This evidence highlights the importance of exploring cognitive mechanisms such as reward processing that may underlie or affect anhedonia.

Behavioral models of depression posit that a reduced frequency of rewarding experiences in one's environment is involved in the onset and maintenance of depressive

symptoms. It is suggested that a reduced frequency of rewarding experiences leads to feelings of sadness as well as to a reduction in behaviors aimed to pursue rewarding experience (i.e. reduced engagement in pleasurable activities and social withdrawal) and this in turn may maintain or exacerbate depressive symptoms (Forbes & Dahl, 2005; Lewinsohn, 1985; Lewinsohn, Hoberman, Teri, & Hautzinger, 1985). It is suggested that the frequency of rewarding experiences in one's environment may depend on a number of factors: 1) The individual's preferences which determine what kind of experiences are perceived as rewarding, 2) the availability of such rewarding experiences in the individual's environment, and 3) the individual's ability to pursue rewarding experiences in their environment. This highlights the importance of exploring how individual differences in reward processing relate to risk for depression.

Impairments in reward processing may increase risk for depression in several ways. Reward learning difficulties may affect the individual's ability to identify opportunities to pursue and receive rewards or to modulate behavior in order to increase the likelihood of receiving rewards. Motivation deficits may lead to reduced engagement in pleasurable activities, and hedonic deficits may lead to a reduced enjoyment from potentially rewarding activities or experiences. These may contribute to reduced positive affect in everyday life (Forbes & Dahl, 2005, 2012; Heerey, 2014; Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2008; Treadway & Zald, 2011). Moreover, individuals with reward processing deficits may be less likely to use rewarding experiences as a mood repair strategy, for example by doing a pleasant activity or recalling positive memories to reduce the impact of negative affect. This, in turn, may increase dysregulation of negative affect (Forbes & Dahl, 2005, 2012; Heerey, 2014; Kovacs & Lopez-Duran, 2010; Pizzagalli et al., 2008; Treadway & Zald, 2011).

Findings from studies in adults suggest that there is substantial heterogeneity with regard to the presence of reward processing deficits in depressed individuals. For example, studies that examine hedonic response to positive stimuli show mixed results, with some studies reporting lower subjective ratings of positive stimuli in depressed subjects compared to controls whereas other studies reported no group differences (Sherdell et al., 2012; Treadway & Zald, 2011). A few studies that have examined reward learning in depression have shown that compared to controls, depressed subjects show a lower tendency to display a reward bias toward a rewarded stimulus, and that this was associated with self-reported anhedonia (Henriques, Glowacki, & Davidson, 1994; Pizzagalli et al., 2008; Pizzagalli et al., 2005). However, a study by Chase et al. (2010) that

examined reward learning in adults with MDD and healthy adults found that the depressed group did not show impairment in reward learning compared to the healthy group but rather that reduced reward learning was associated with level of anhedonia in all subjects. Only a few studies have addressed motivation for reward in depressed subjects. Two studies by Treadway et al. (2012) and Yang et al. (2014) reported that depressed adults were less willing to exert effort in order to receive rewards compared to control subjects, whereas Sherdell et al. (2012) found no evidence of group differences with regard to motivation. However, additional studies are needed to examine motivational deficits in depression.

A smaller number of behavioral studies have examined reward processing deficits in children and adolescents. However, initial findings show that impairments in reward processing are present in depressed adolescents, especially with regard to reward seeking, and to the ability to adapt behavior in order to increase the likelihood of receiving reward. For example, Jazbec et al. (2005) examined improvement in a cognitive control task as a function of reward in adolescents, under the hypothesis that rewards will enhance task performance. Findings of this study indicated that whereas healthy adolescents showed improvement in their performance as a function of reward, depressed adolescents' performance was unaffected by reward. Two other studies reported that depressed children and adolescents show difficulty modifying their behavior according to reward magnitude and display low reward seeking behaviour under conditions of high probability of gain compared to non-depressed children and adolescents (Forbes & Dahl, 2012; Rawal et al., 2013). A study by Kyte et al. (2005) found a different pattern of behavior in a similar task that assessed reward seeking under different probabilities of gain, in that adolescents with recent depression showed a more impulsive pattern of decision making as well as higher risk taking in conditions of low probability of gain. Thus, whilst there are some inconsistencies in findings regarding reward processing in adolescents, existing evidence does suggest that disruptions in reward processing are present in currently depressed adolescents.

Finally, initial evidence from studies comparing those at high and low familial risk, as well as evidence from longitudinal studies, suggests that impairments in reward processing serve as a risk factor for subsequent depression. For instance, Mannie et al. (2015) compared adolescent and young adult offspring of depressed parents and healthy parents and found that high-risk subjects showed lower risk taking compared to low risk subjects in a gambling task . This study thus illustrates that reward processing is altered in

unaffected individuals at high familial risk. In a longitudinal study of adolescents boys, Forbes et al. (2007) reported that a lower tendency to choose an option of a high magnitude reward in a condition of high probability for gain in a gambling task predicted depressive symptoms and disorder one year later. Similarly, in longitudinal study of a high-risk sample of adolescent offspring of depressed parents, Rawal et al. (2013) found that low reward seeking under conditions of high probability of gain in a gambling task predicted depressive symptoms and new onset disorder at a one year follow up when controlling for baseline depressive symptoms. Thus, a number of studies report that reduced reward seeking behavior predicts the subsequent development of depressive symptoms and disorder in adolescents.

There has been a debate regarding the ecological validity of commonly used reward tasks, with questions raised about whether or not current reward paradigms sufficiently reflect reward processing or reward seeking in real life situations (Forbes, 2009; Sharp, Monterosso, & Montague, 2012). In particular, the majority of reward tasks assess reward processing in the context of gambling tasks or probabilistic signal detection tasks (Forbes et al., 2007; Pizzagalli et al., 2005) and use money or points as rewarding stimuli. Therefore they may not tap other types of reward that are meaningful for human functioning and behaviour, such as social rewards (Forbes, 2009). As social functioning has been shown to play an important role in the onset of depression (Forbes, 2009; Santini et al., 2015; Teo et al., 2013), it has been suggested that the lack of studies exploring reward processing in a social context may substantially limit our understanding of the involvement of reward processing in depression (Forbes, 2009). It has been suggested that additional paradigms need to be developed or employed in reward research in order to increase ecological validity of assessment and explore additional types of rewards and especially social reward processing (Forbes, 2009; Mellick, Sharp, & Ernst, 2015; Sharp et al., 2012). Therefore, in this thesis I aimed to address this gap and explore associations between reward processing assessed using social paradigms, depression and social functioning in two age groups which have been shown to be vulnerable periods for the onset of depression: young adults and adolescents (Hankin et al., 1998). Specifically I aimed to:

1) Explore associations between reward learning in a social context and familial risk for depression in young adults. Following previous studies of reward learning in depressed and high-risk individuals, I also aimed to examine whether any associations between reward learning and familial risk were attributable to the severity

of depressive symptoms or anhedonia. Finally, I aimed to examine whether patterns of reward learning that are associated with familial risk, depressive symptoms or anhedonia are associated with everyday social functioning.

2) Develop and validate a measure aimed at assessing sensitivity to social rewards suitable for adolescents, due to the lack of measures assessing reward processing - and specifically social reward processing - suitable for this age group. A further justification for developing the measure for this age group was that as previously mentioned, the increase in rates of depression in adolescence is suggested to be at least partly attributable to the increase in salience of social rewards and social stress in this period (Davey et al., 2008; Foulkes & Blakemore, 2016; Hankin et al., 1998; Silk, Davis, McMakin, Dahl, & Forbes, 2012). I also aimed to explore the association between sensitivity to social rewards and sensitivity to negative social experiences following literature suggesting an association between these two constructs and linking this association to vulnerability to depression during adolescence (Davey et al., 2008; Silk et al., 2003). Finally, I aimed to explore the associations between these two constructs, social functioning and depressive symptoms in adolescents.

1.12. Aims of the current thesis

As described, previous research has shown that impairments in cognitive processes such as executive functioning, retrieval of autobiographical memory and reward processing are present in currently depressed individuals and may also increase risk for depression. However, despite the potential clinical importance of these processes, only a small number of studies examined whether and how such processes may serve as risk and protective factors for depression in young people and high-risk groups. Thus, this thesis will examine these cognitive processes as risk and protective factors for depression in adolescents and young adults, with a particular focus on offspring of depressed parents. As previously detailed, the choices of research design and outcome measures examined in each chapter were based on specific gaps in the literature exploring each cognitive process (i.e. executive functions, retrieval of autobiographical memory and reward processing).

The specific research aims of the subsequent chapters are briefly described below.

In chapter 2, I explored the protective role of two aspects of executive functioning - inhibitory control and mental flexibility - in the association between current parental

depression and adolescent depressive symptoms, in a sample of adolescent offspring of parents with a history of MDD.

In chapter 3, I explored how severity of parental depression and negative life events are associated with specificity of autobiographical memory, and how specificity of autobiographical memory predicts over time indices of mental health resilience and social functioning in a sample of adolescent offspring of parents with a history of MDD.

In chapter 4, I explored associations between reward learning in a social context and familial risk for depression in a sample of young adult offspring of parents with a history of MDD and parents with no history of MDD. Additionally I examined whether any association between reward learning in a social context and familial risk were attributable to depressive symptoms and anhedonia. Finally, I examined how patterns of reward learning related to familial risk, anhedonia or depressive symptoms are associated with social functioning.

In chapter 5, I developed and examined the validity of a new measure assessing sensitivity to socially rewarding situations and negative social situations in adolescents. Furthermore, I examined associations between sensitivity to socially rewarding situations, sensitivity to negative social situations, social functioning and depressive symptoms in a community sample of adolescents.

CHAPTER 2: Do better executive functions buffer the effect of current parental depression on adolescent depressive symptoms?

Offspring of parents with a history of major depressive disorder (MDD) and especially those exposed to a current episode of parental depression have been found to be at increased risk for developing depression themselves. Exposure to a current parental depressive episode also reduces the efficacy of interventions in high risk or depressed adolescents. This highlights the need to identify protective factors for adolescents exposed to a current parental depressive episode. Executive functions serve as an important cognitive resource, involved in the ability to regulate mood and thoughts and cope with stressful events. This study examined the buffering role of two components of executive functioning, inhibitory control and mental flexibility, in the association between a current parental episode of MDD and adolescent depressive symptoms. A high-risk sample of 288 adolescent offspring of parents with recurrent major depressive disorder completed an Affective Go/No Go and a Verbal Fluency task. Parents and adolescents underwent psychiatric interviews. Findings of this study indicated that in the presence of a current parental depressive episode in the parent, adolescents with better inhibitory control and mental flexibility had fewer depressive symptoms after controlling for age, gender and IQ.

These findings suggest that executive functions may protect against adolescent depression in the presence of a parental depressive episode. It may be beneficial to target executive functions in preventive programs for individuals at high-risk for depression.

2.1. Introduction

Parental depression has been identified as a major risk factor for depression in childhood and adolescence with children of depressed parents three to four times more likely to develop depression than offspring of non-depressed parents (Garber, 2006; Rice & Rawal, 2010; Weissman et al., 1997). Despite the strong association found between parental depression and offspring depression, there is heterogeneity in outcomes for children of depressed parents which is partly attributable to clinical features of parental depression. Evidence suggests that exposure to a current parental depressive episode is an important feature of parental depression that increases risk for offspring. For instance, children of parents with a history of recurrent depression whose parents have had a recent episode of major depressive disorder (MDD) show elevated rates of psychiatric disorder and depressive symptoms (Mars et al., 2012). Exposure to a recent episode of depressive disorder in a parent has also been shown to moderate the efficacy of treatment and prevention programs (Beardslee et al., 2013; D. A. Brent et al., 1998; Garber et al., 2009). Thus, exposure to a current parental episode may serve as a particularly salient risk factor among those at familial risk of depression. This evidence, taken together with the long-term adverse consequences of depression in childhood and adolescence (V. Dunn & Goodyer, 2006; Fergusson, Boden, & Horwood, 2007; Rice et al., 2007; Rutter, Kim-Cohen, & Maughan, 2006) emphasizes the need to identify protective factors for high-risk offspring and especially for those currently exposed to a depressive episode in the parent.

Several lines of evidence suggest that executive functions may confer protection against depression for the adolescent offspring of depressed parents. First, cognitive models of depression suggest that the mood regulation difficulties that characterise currently depressed individuals (De Lissnyder et al., 2010; Harmer, Goodwin, et al., 2009; Roiser et al., 2012) may arise from difficulties with executive functions such as inhibition and mental flexibility. Inhibitory control involves controlling attention, thoughts and behaviours in order to override an automatic or dominant response and mental flexibility allows switching between different mental sets or perspectives in order to adjust to changing or novel circumstances (Davidson et al., 2006; Diamond, 2013; Miyake et al., 2000). It is plausible that the ability to inhibit thoughts and flexibly switch between thoughts and perspectives can protect against being "captured" by negative thoughts or low mood. Thus, both inhibitory control and mental flexibility have been associated with

more effective emotional regulation strategies including lower levels of rumination - a cognitive style which perpetuates negative affect (Gotlib & Joormann, 2010; Joormann & Quinn, 2014) and higher levels of reappraisal – an effective mood repair strategy (McRae et al., 2012; Ochsner & Gross, 2008). Studies conducted with depressed adults have indicated particular impairments in inhibitory control and mental flexibility when processing emotional information (Deveney & Deldin, 2006; Gotlib & Joormann, 2010; Murphy et al., 2012). Although fewer studies have examined if these impairments occur in depressed children and adolescents, several lines of evidence suggest these deficits are also present in depressed children and adolescents (Kyte et al., 2005; Ladouceur et al., 2005; S. Wagner et al., 2015). Moreover, preliminary evidence suggests that difficulties in executive functioning, such as impairments in inhibition on emotional tasks, may precede and increase risk for depression (Joormann, Talbot, et al., 2007; Kilford et al., 2015).

A second line of evidence that suggests executive functioning may protect against depression is that efficacious interventions for treating and preventing adolescent depression such as Cognitive Behavioural Therapy (CBT) (Stice et al., 2009; Weisz, McCarty, & Valeri, 2006) involve training in evaluating and challenging thoughts and introducing alternatives (Forkmann et al., 2014; Friedberg & McClure, 2002). These mood regulation skills involve elements of executive functioning, for instance in inhibiting negative thoughts (i.e. inhibitory control) and thinking about an issue from a different perspective to introduce alternative thoughts (i.e. mental flexibility). In a similar way, when healthy individuals are instructed to look at an emotionally salient event from different perspectives this has significant impact on emotional regulation and reactivity as measured by self-report and physiological measures (Kross et al., 2005; Schartau, Dalgleish, & Dunn, 2009; Southwick et al., 2005).

A third source of evidence comes from findings which suggest that executive functions may serve as an important cognitive resource that protects individuals at familial risk of psychiatric disorders (M. H. Johnson, 2012). Johnson (2012) suggests that better executive functions may help those at genetic risk for developmental disorders by allowing compensatory brain systems to be recruited during cognitive operations. Thus, it is plausible that executive functions may also serve as a protective factor in those at increased familial risk of depression.

Although previous research has shown that currently depressed individuals are characterized by executive functioning impairments and that these impairments may increase risk for depression, past studies have not directly examined whether better

executive functions serve as a protective factor for those at increased familial risk of depression. In order to address this gap in the literature, I aimed to examine the protective effect of two components of executive functioning (inhibitory control and mental flexibility) in a high risk sample of adolescent offspring of parents with a history of recurrent MDD. The primary research question of this study was whether inhibitory control and mental flexibility protected against the predicted risk effect of a current parental depressive episode on adolescent depressive symptoms. On the basis of evidence involving inhibitory control and mental flexibility with dysregulation of thoughts and emotions I also examined whether the proposed moderating effect of executive functions was present for both cognitive-emotional depressive symptoms (e.g. low mood/irritability and concentration difficulties) and for vegetative-somatic depressive symptoms (e.g. change in appetite or weight and psychomotor retardation/agitation). I hypothesized that for adolescents exposed to a current parental episode, the buffering effect of executive functions would be more consistently present for cognitive-emotional depressive symptoms than for vegetative-somatic depressive symptoms given that the former indicate dysregulation of thoughts and emotion and therefore might be more attenuated in those with better executive functions.

2.2. Methods

2.2.1. Participants and procedure

The present study utilized an existing data set collected by others prior to my PhD. The sample came from a longitudinal study of parents with recurrent major depression and their adolescent offspring (aged 9-17), the Early Prediction of Adolescent Depression (EPAD) study. Data collection of the EPAD sample was completed during the years 2007-2011.Previous work employing data from the EPAD study has yielded papers exploring a wide range of topics including mental health, cognitive processing, family processes, resilience, screening and measurement development (Collishaw et al., 2016; Hammerton, Zammit, Potter, Thapar, & Collishaw, 2014; Kilford et al., 2015; Mars et al., 2012; Rawal et al., 2013; Rawal & Rice, 2012; Sellers et al., 2014).Parents were recruited from general practices in south Wales (78%), from advertisements in primary care health centres (3%) and from previous studies of recurrent unipolar depression (19%) - a genetic study of siblings with depression (Depression Network study; DeNT; Korszun et al., 2004) and a case control study (Depression Case-Control study; DeCC; Korszun et al., 2004).Following initial screening over the phone, parents underwent a psychiatric interview to confirm a

history of recurrent unipolar depression. Families were excluded if the index parent met criteria for a bipolar disorder, mania/hypomania or psychotic disorder at the time of the interview; the child wasn't living at home or had an IQ lower than 50. If more than one child in the household was eligible to participate in the study, the youngest child was selected in order to prevent selection bias.

At baseline the sample included 337 families. The parent sample included 315 mothers and 22 fathers (age range 26-55; mean age 41.7). The adolescent sample included 197 females and 140 males (age range: 9-17; mean age 12.4). 70% of the families in the baseline sample were a 2 parent household (approximately comparable rates to 77% found in a recent epidemiological study in the UK; Office for National Statistics, 2009). No substantive differences were found between parents recruited from the previous studies of recurrent unipolar depression and parents recruited from general practices/advertisements in primary care health centres on indices of past severity of depression such as age of onset of depression [t(324)=-1.752, p=.08; Parents recruited from previous studies: M=24.5, SD=7.40, Parents recruited from general practices/advertisements: M=26.57, SD=8.34] and the average DSM-IV Global Assessment of Functioning (GAF) score (American Psychiatric Association, 1994) of the worst two past depressive episodes as assessed in baseline [t(79.59)=-1.63,p=11; Parents recruited from previous studies: M=43.01, SD=19.26, Parents recruited from general practices/advertisements: M=47.27, SD=15.36].

The EPAD study was undertaken with approval of the Multi- Centre Research Ethics Committee for Wales. Assessments were administered in families' homes. The data utilized for the current investigation was collected at the second assessment of the study because adolescents completed a battery of cognitive tests including measures of executive functioning at this assessment.

At the second assessment of the study 288 parents and 275 adolescents completed research psychiatric interviews, the Child and Adolescent Psychiatric Assessment (CAPA), for assessing psychopathology in the offspring. Among 288 adolescents for whom a psychopathology assessment was completed (by either the parent and offspring or by the parent only), data on performance on the Verbal Fluency (VF) task was available for 264 (91.6%) adolescents and data on performance on the Affective Go/No-Go Task (AGN) was available for 187 (64.9%). Participation rates and reasons for non-completion are outlined in Appendix 2.1. There were no systematic differences between adolescents who completed the Verbal Fluency task and the AGN and those who

did not in terms of gender [VF: $\chi^2(1)=1.52, p=.22$; AGN: $\chi^2(1)=.10, p=.75$], age [VF: $t(286)=1.40$, $p=.16$; AGN: $t(286)=.89$, $p=.37$] and depressive symptoms [VF: $t(282)=.96$, $p=.34$; AGN: $t(282)=-.07$, $p=.95$]. However, participants who completed the VF and AGN had higher IQ scores than those who did not [VF: $t(328)=-4.54$, $p<.001$; AGN: $t(328)=-2.83$, $p<.01$]. IQ was therefore included as a covariate in all analyses that follow. As detailed in Table 2.1., the mean number of CAPA defined adolescent depressive symptoms was 1.93. Additionally, 6% of adolescents in this sample met DSM-IV criteria for MDD. These rates are substantially higher than those found in community studies where similarly stringent criteria are used (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Green, McGinnity, Meltzer, Ford, & Goodman, 2005; Silberg et al., 1999).

2.2.2. Measures

2.2.2.1. Parent psychiatric assessment

Depressive symptoms and disorder in the parent were assessed using the Schedules for Clinical Assessment in Neuropsychiatry(SCAN; Wing et al., 1990) .The primary exposure variable was the presence/absence of a DSM-IV episode of MDD in the parent. However, I also assessed past parental severity (the presence of a previous severe episode), parental age of onset and the adolescent's exposure to previous parent depressive episodes (the number of previous parental depressive episodes the adolescent was exposed to). These variables were included as covariates in analyses that follow. The SCAN is a psychiatric interview which provides a detailed assessment of adult psychopathology. A life history calendar approach was used to compile a timeline of the affected parent's previous episodes and to define age of onset (Caspi et al., 1996; Freedman, Thornton, Camburn, Alwin, & Young-DeMarco, 1988). Based on the SCAN and the timeline data, the presence of a parental current episode of MDD, the presence of a previous severe episode, parental age of onset and the adolescent's exposure to previous parent depressive episodes were defined according to the following criteria: the presence of a current depressive episode in the parent was defined as whether an episode of DSM-IV defined MDD had occurred in the previous month (yes; no); parent age of onset was dichotomized to ≤20 versus 21 years or older in accordance with previous research (Weissman et al., 1984); a previous parental episode was considered severe if it involved severe functional impairment (a score of GAF≤30 on the Global Assessment of Functioning scale in DSM-IV; American Psychiatric Association, 1994) or hospitalization

(Hammen & Brennan, 2003; Mars et al., 2012); using a life history calendar approach, a count of the number of parental episodes the adolescent was exposed to from birth until the year prior to the current assessment was calculated.

2.2.2.2. Adolescent Psychiatric assessment

Depressive symptoms in the adolescent were assessed using the Child and Adolescent Psychiatric Assessment (CAPA; Angold & Costello, 2000). The CAPA is a semi-structured interview which assesses psychopathology over the previous 3 months. Interviews were conducted separately both with the parent and the adolescent. Adolescent total number of depressive symptoms was the primary outcome variable (range 0-9). Cognitive-emotional and vegetative-somatic symptoms were also considered separately as secondary outcome variables using definitions based on previous research (Cavanaugh, 1984; Cook, Spring, McChargue, & Doran, 2010; Osman, Kopper, Barrios, Gutierrez, & Bagge, 2004). Number of cognitive-emotional symptoms included the symptoms of irritable or low mood, loss of interest, feelings of worthlessness or guilt, inefficient thinking/indecisiveness, suicidal thoughts/plans/behaviour (range 0-5). Number of vegetative-somatic symptoms included the symptoms of change in appetite, sleep disturbance, loss of energy, psychomotor agitation or retardation (range 0-4). The three outcome measures of symptom counts consisted of a combined report of the parent and the adolescent such that a symptom was considered present if it was reported by either the adolescent or the parent, as supported by common clinical practice (Angold, Prendergast, et al., 1995; Costello et al., 2003). Sensitivity analyses additionally examined adolescent-rated depressive symptoms as the outcome variable. Attention Deficit Hyperactivity Disorder (ADHD) was also assessed using the CAPA and analyses were re-run excluding individuals meeting DSM-IV criteria for any of the three subtypes of ADHD: the combined type, the predominantly inattentive type and the predominantly hyperactive-impulsive type.

2.2.2.3. IQ

The 10 subscales from the Wechsler Intelligence Scale for Children- Fourth edition (WISC-IV; Wechsler, 2003) were used to measure full-scale IQ.

2.2.2.4. Executive Functioning

The Verbal Fluency task (VF;Benton, 1968) was administered to assess mental flexibility (Diamond, 2013; Suchy, 2009). Participants are required to generate as many words as possible beginning with the letters F, A, S with 1 minute given for each letter. Participants were instructed that proper nouns (e.g. France), repetitions and more than one word of the same origin (e.g. act, acting) were not acceptable and they were given examples of each. The task involves the ability to devise different strategies for coming up with as many words as possible and to generate categories from which to extract words (Fossati, Bastard Guillaume, Ergis, & Allilaire, 2003). Outcome measures were: 1) Total number of correct responses; 2) number of disallowed responses i.e. words that violated the rules of the task; 3) number of repetitions (Regard, Strauss, & Knapp, 1982; M. A. Turner, 1999).

The Affective go/no go task (AGN; www.camcog.com; Murphy et al., 1999) was used to measure inhibitory control and set-shifting. A series of words is rapidly presented in the centre of the screen. Words can be positive or negative. Participants are given a target valence and asked to press the press pad when they see a word that matches this valence, while withholding response to words of the other valence (distractors). There are 2 practice blocks and 8 test blocks of 18 words each. The target word changes during the task, so that half of the blocks are shift blocks. The task measures inhibitory control and set-shifting ability in the context of processing emotional information. A number of outcomes measures were initially examined: 1) Commission errors (the number of responses to distractor stimuli i.e. false alarms) for all trials and for shift blocks only. 2) Omission errors (the number of missed responses to targets) for all trials and for shift blocks only. 3) Shifting costs (calculated as the difference between the mean reaction time in non-shift blocks and the mean reaction time in shift blocks). Larger differences represent difficulties in set shifting ability (Karbach & Kray, 2009).

2.2.3. Statistical analysis

A Principal Component Analysis (PCA) on all outcome measures from the Verbal Fluency and Affective Go No Go Tasks was conducted to identify measures assessing inhibitory control and mental flexibility which are unique components of executive functioning (Diamond, 2013; Suchy, 2009). This derived one measure of inhibitory control errors (number of commission errors on the AGN) and two measures of mental flexibility (number of correct responses on the Verbal Fluency task and AGN shifting costs).

In addressing the main hypotheses, I first examined the main effect of current parental depression (the presence of a current parental depressive episode) on adolescent executive functioning variables using linear regression while controlling for the additional parental depression variables (the presence of a previous severe episode, age of onset and the adolescent's exposure to previous parent depressive episodes). Next, I tested the main effect of current parental depression on adolescent depressive symptoms and assessed whether any observed association remained when controlling for the covariates of the presence of a previous severe episode in the parent, parent age of onset and the adolescent's exposure to previous parent depressive episodes. I entered current parental depression and the three covariates mentioned above in separate steps of the regression in order to examine whether these covariates had additional contribution to the regression models above current parental depression.

I next examined executive functioning variables as moderators of the association between current parental depression and offspring depressive symptoms using linear regression. Analyses were conducted separately for each of the three measures of executive functioning identified by the PCA. Independent and control variables were standardized (Cohen, Cohen, West, & Aiken, 2003). Covariates identified as significantly associated with executive functioning measures or with offspring depressive symptoms were retained in the test of moderation. In the first step of each regression, adolescent age and gender were entered as covariates. Next, full scale IQ was entered as a covariate. In the third step, the main effects of current parental depression (currently affected yes (1); no (0)) and the measure of executive functioning were included. In the fourth step the interaction term between current parental depression and executive functioning was entered. Significant interactions were illustrated by plotting counts of depressive symptoms for adolescents with high (top tertile) and low (bottom tertile) performance on executive functioning measures by current parental depression. In order to rule out the possibility that the observed pattern of results was attributable to biased rating of adolescent depressive symptoms by currently depressed parents, analyses were repeated with adolescent-rated depressive symptoms as the outcome variable. To rule out the possibility that ADHD served as a confounder as it has been associated with both executive functioning impairment and higher depressive symptoms (Humphreys et al., 2013; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005), I repeated the analysis excluding adolescents meeting DSM-IV diagnostic criteria for ADHD (n=16). Additionally, I repeated the analyses while controlling for the three additional parental depression variables examined earlier

(the presence of a previous severe episode, age of onset and the adolescent's exposure to previous parent depressive episodes). Finally, I examined whether the buffering effect of executive functioning was present for both cognitive-emotional symptoms and for vegetative-somatic symptoms by conducting linear regression separately for each symptom group.

2.3. Results

2.3.1. Deriving inhibitory control and mental flexibility components of offspring executive functioning

A PCA with direct Oblimin rotation was performed on adolescent executive functioning measures (Table 2.2.). This derived four components, three that assessed aspects of executive functioning and one that assessed attention. High component loadings of individual measures as well as distributive properties and theoretical understanding of the construction of executive functioning (Suchy, 2009) were the criteria used to select measures tapping unique aspects of executive functioning for analysis. This resulted in one measure of inhibitory control errors i.e. failure to inhibit an inappropriate behavioural response to a distractor (number of commission errors on the AGN) and two measures tapping separate aspects of mental flexibility i.e. mental generativity and shifting costs. Mental generativity was assessed by number of correct responses on the Verbal Fluency task which is suggested to represent the ability to generate and create ideas and responses to problems (Hendrawan et al., 2012; Suchy, 2009). Although both VF repetitions and VF correct responses had loadings higher than .4 on the mental generativity component, I chose to focus on VF correct responses over VF repetitions since repetitions rarely occurred (Table 2.2.) and the measure of VF correct responses has received more support in previous studies as a measure of mental generativity (Hendrawan et al., 2012; Suchy, 2009). AGN shifting costs was assessed as the difference in reaction times between shift blocks and non-shift blocks with larger differences representing greater difficulty in shifting (Karbach & Kray, 2009). Results of the PCA suggested that mental generativity and shifting costs constitute two separate aspects of mental flexibility (Table 2.2.).

2.3.2. Preliminary analyses

Correlations between all study variables and descriptive statistics are presented in Table 2.1. Measures of executive functioning were not significantly associated with

adolescent depressive symptoms nor with indicators of current parental depression, the presence of a previous severe episode in the parent, parental age of onset or the adolescent's exposure to previous parent depressive episodes (Table 2.1). Linear regression analyses were conducted to examine the main effect of parental current depression on adolescent executive functioning and adolescent depressive symptoms while controlling for age, gender and the additional parental depression variables. As presented on Table 2.3, after controlling for age, gender and additional parental depression variables, current parental depression was not significantly associated with adolescent executive functioning (Parental current depression: AGN inhibitory control errors β=-.03, p=.66; VF mental generativity β=-.10, p=.12; AGN shifting costs β=.04, p=.65). However, as predicted, current parental depression was significantly associated with adolescent depressive symptoms after controlling for age and gender [β=.20, p<.01]. This effect was present when controlling for the covariates of the presence of a previous severe episode in the parent, age of onset in the parent and the adolescent's exposure to previous parent depressive episodes. The covariates of parent previous severe episode, parent age of onset and the adolescent's exposure to previous parent depressive episodes were not found to be significantly associated with offspring executive functions [parent previous severe episode: AGN inhibitory control errors β=.01, p=.86, VF mental generativity β=.06, p=.40, AGN shifting costs β=.04, p=.66; parent age of onset: AGN inhibitory control errors β=.02, p=.74, VF mental generativity β=.01, p=.90, AGN shifting costs β=.03, p=.72; adolescent's exposure to previous parent depressive episodes: AGN inhibitory control errors β=-.10, p=.17, VF mental generativity β=.11, p=.08, AGN shifting costs β=.04, p=.61] or with offspring depressive symptoms [parent previous severe episode β=.06, p=.36; parent age of onset β=.05, p=.42; adolescent's exposure to previous parent depressive episodes β=.004, p=.95].

Table 2.1. Descriptive statistics and correlations between study variables

	Mean (SD) or N%	Range (Min, Max)	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Age of adolescent	13.77 (2.04)	8 (10,18)													
2. Gender of adolescent (%male, % female)	40.3, 59.7	-	.08												
3. IQ of adolescent	96.02 (12.67)	81 (50,131)	-.15*	.11											
4. Current parental depression (%yes, %no)	18.2, 81.8	-	-.05	.02	-.06										
5. Parental age of onset (%≤20,%>20)	27, 73	-	-.002	-.08	-.04	-.15*									
6. Parent previous severe episode (% yes, % no)	27.8, 72.2	-	.06	-.06	.02	.26**	-.22**								
7. Adolescent exposure to previous parent episodes	3.8 (12.8)	170 (0,170)	.04	-.03	.02	.01	-.06	-.02							
8. Mental generativity(VF)	27.14 (8.58)	46 (8,54)	.22**	.06	.40**	-.08	.02	.02	.09						
9. Inhibitory control errors (AGN)	19.52 (13.26)	66 (1,67)	-.37**	-.18*	-.12	.02	.10	.02	-.13	-.18*					
10. Shifting costs (AGN; msec)	-11.65 (44.04)	320.72 (-200.52,120.2)	.06	.08	-.11	.02	.04	.06	.04	-.03	-.05				
11. Adolescent total depressive SY	1.93 (1.94)	9 (0,9)	.19**	.12*	-.16**	.14*	-.01	.10	.01	-.03	.06	.06			
12. Adolescent Cognitive-emotional SY	.65 (1.08)	5 (0,5)	.10	.15*	-.19**	.18**	-.02	.16**	-.04	-.06	.04	.05	.86**		
13. Adolescent Vegetative-somatic SY	1.27 (1.13)	4 (0,4)	.22**	.09	-.09	.08	-.04	.03	.05	.01	.07	.05	.88**	.53**	

Note: SY= Symptoms; Min= minimal value; Max=maximal value; msec= milliseconds; *p<.05; **p<.01

Correlations were calculated for 288 adolescents for whom psychopathology data was available; N ranges from 183 to 288 due to missing values.

Table 2.2. Principal component analysis on measures of executive functioning from the AGN and VF tasks

Measure	**Descriptive data (N=186)**		**Component Loadings**			
	Mean (SD)	Range	Attention	Inhibitory control	Mental Generativity	Shifting costs
AGN: omissions (shift blocks)	8.40 (6.78)	33	**.99**	-.07	.06	-.05
AGN: Total omissions	16.56 (13.02)	66	**.99**	-.05	.05	-.05
AGN: commissions (shift blocks)	10.30 (6.75)	36	-.04	**.98**	.04	-.05
AGN: Total commissions	19.62 (13.23)	66	-.06	**.98**	.04	-.04
VF: repetitions	0.42 (1.12)	11	.14	.11	**.95**	.10
VF: correct responses	27.06 (8.37)	42	-.39	-.28	**.47**	-.20
AGN: shifting costs (msecs)	-11.65 (44.04)	320.72	-.09	.03	.06	**.75**
VF: disallowed responses	.99 (1.78)	12	-.03	.10	.01	**-.69**
Component			**Correlations among components**			
Attention						
Inhibitory control errors			.05			
Mental generativity			-.14	-.09		
Shifting costs			.06	-.05	-.07	

Note: The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis, KMO= .50; Bartlett's test of sphericity χ^2 (28) =1070.45, p<.001 indicated that correlations between measures were sufficiently large for a principal component analysis; Loadings of .4 or more were considered informative.

Descriptive data is presented for subjects with data on all measures of AGN and VF tasks. Minor variations in the descriptive data presented in Tables 2.1. and 2.2. are due to the use of pairwise deletion in Table 2.1. and listwise deletion in Table 2.2.

Table 2.3. Main effects of parent current depression, parent past severity, parent age of onset and the adolescent's exposure to previous parent depressive episodes on offspring executive functioning and depressive symptoms

	Inhibitory control errors (N=172)					Mental generativity (N=239)					Shifting costs (N=171)					Adolescent depressive symptoms				
	∆ R²	**p**	**B(S.E.)**	**β**	**p**	**∆ R²**	**p**	**B(S.E.)**	**β**	**p**	**∆ R²**	**p**	**B(S.E.)**	**B**	**P**	**∆ R²**	**p**	**B(S.E.)**	**β**	**p**
Step 1	.18	<.001				.04	<.01				.01	.39				.04	<.01			
Gender of adolescent			-4.69(1.89)	-.17	<.05			.74(1.12)	.04	.51			5.46(6.84)	.06	.43			.37(.24)	.09	.13
Age of adolescent			-5.16(.96)	-.37	<.001			1.80(.56)	.20	<.01			3.86(3.49)	.08	.27			.33(.12)	.17	<.01
Step 2	.001	.70				.01	.13				.002	.61				.04	<.01			
Gender of adolescent			-4.67(1.90)	-.17	<.05			.75(1.12)	.04	.50			5.27(6.86)	.06	.44			.35(.24)	.09	.14
Age of adolescent			-5.18(.97)	-.38	<.001			1.77(.56)	.20	<.01			3.93(3.50)	.09	.26			.35(.12)	.18	<.01
Current PD			-.95(2.44)	-.03	.70			-2.18(1.45)	-.10	.13			4.57(8.91)	.04	.61			1.03(.31)	.20	<.01
Step 3	.01	.53				.02	.30				.003	.93				.005	.73			
Gender of adolescent			-4.51(1.91)	-.17	<.05			.79(1.13)	.04	.48			5.48(6.95)	.06	.43			.38(.24)	.10	.12
Age of adolescent			-5.14(.98)	-.37	<.001			1.74(.56)	.20	<.01			3.64(3.55)	.08	.31			.34(.12)	.18	<.01
Current PD			-1.12(2.57)	-.03	.66			-2.34(1.50)	-.10	.12			4.28(9.41)	.04	.65			.99(.32)	.20	<.01
Parent previous severe episode			.41(2.25)	.01	.86			1.10(1.31)	.06	.40			3.56(8.18)	.04	.66			.25(.27)	.06	.36
Parent age of onset			.75(2.28)	.02	.74			.16(1.32)	.01	.90			2.96(8.29)	.03	.72			.22(.27)	.05	.42
Adolescent's exposure to previous parent episodes			-4.99(3.66)	-.10	.17			4.26(2.41)	.11	.08			6.68(13.24)	.04	.61			.01(.13)	.004	.95

Note: PD=Parental depression

2.3.3. Buffering effect of executive functions on the association between current parental depression and adolescent total depressive symptoms

I next tested whether executive functioning moderated the association between current parental depression and adolescent depressive symptoms. I tested this separately for each executive functioning measure while controlling for covariates associated with offspring executive functioning or offspring depressive symptoms (age, gender and IQ, Table 2.4.; Step 4). Results were consistent across the three measures of executive functioning which assess aspects of inhibitory control and mental flexibility.

2.3.3.1. Main effects

In all three analyses lower adolescent IQ was associated with a greater number of adolescent depressive symptoms [AGN Inhibitory control errors: β=-.15, p<.05; VF mental generativity: β=-.13, p=.06; AGN shifting costs: β=-.16 p<.05]. Consistent with previous research and as expected, parental current depression was also associated with adolescent depressive symptoms. Thus, the number of depressive symptoms was significantly higher in adolescents whose parents met DSM-IV criteria for a current depressive episode in all three analyses [AGN Inhibitory control errors: β=.33, p<.001; VF mental generativity: β=.20, p<.01; AGN Shifting costs: β=.30 p<.001]. There were no significant main effects of executive functioning on adolescent depressive symptoms although a trend was observed for inhibitory control [AGN Inhibitory control errors: β=.15, p=.052; VF mental generativity: β=.02, p=.75; AGN Shifting costs: β=-.09 p=.23].

2.3.3.2. Interactive effects

Results of the interaction terms between current parental depression and executive functioning measures showed that both AGN inhibitory control errors and AGN shifting costs significantly moderated the association between current parental depression and offspring depressive symptoms in the expected direction [AGN Inhibitory control errors: β=.16, p<.05; AGN Shifting costs: β=.18, p<.05], where more errors of inhibitory control and greater shifting costs were associated with increased adolescent depressive symptoms. The interaction between current parental depression and mental generativity was associated at trend level with adolescent symptoms in the expected direction [VF mental generativity: β=-.13, p=.06] where lower mental generativity was associated with increased adolescent depressive symptoms. Results are presented graphically in order to illustrate the difference in depressive symptoms for adolescents with higher and lower

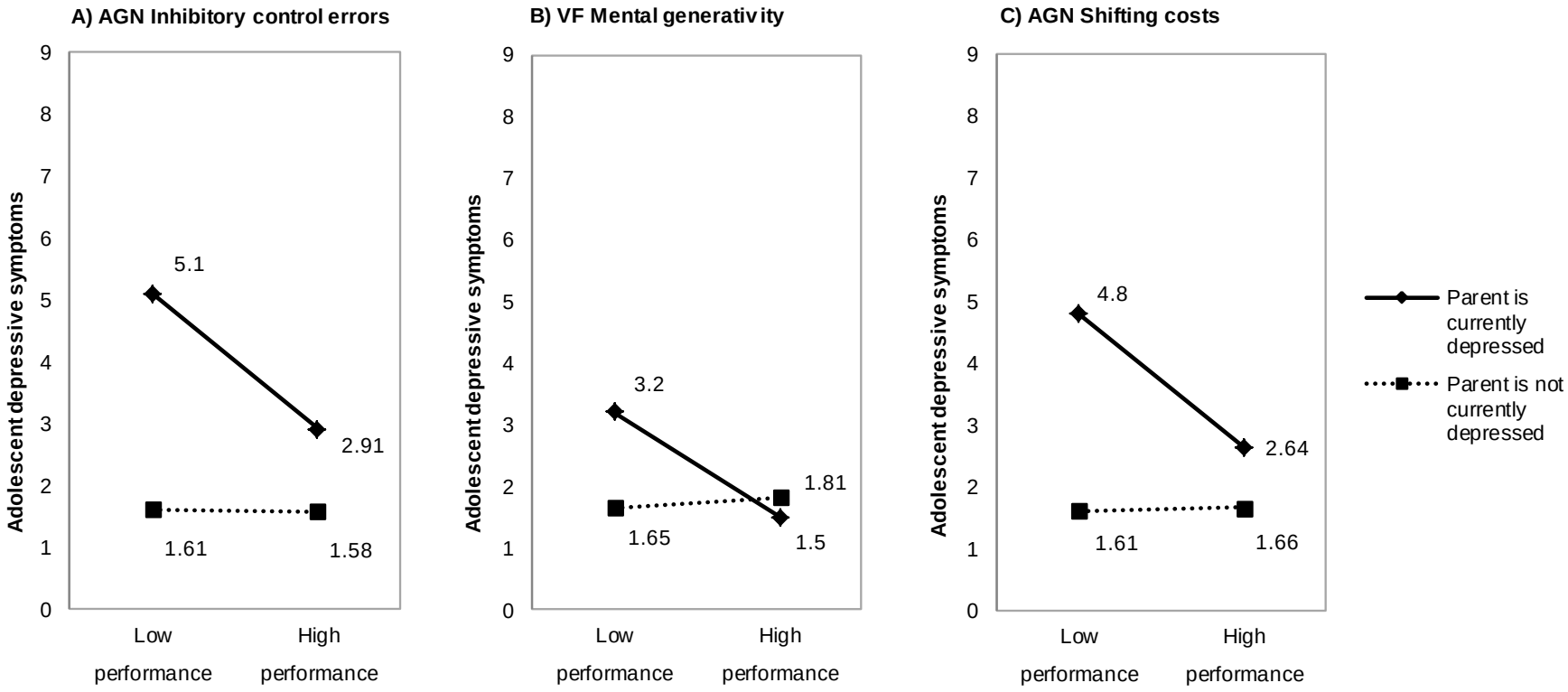
performance on executive functioning measures, separately for those exposed and not exposed to a current parental depressive episode (Figure 2.1.). High and low performance in executive functioning measures were defined as scoring in the top or bottom tertile of the distribution, respectively. On average, higher executive functioning was associated with a decrease of two depressive symptoms in the presence of a current parental episode. The same pattern of interactive effects between current parental depression and executive functioning measures emerged when analyses were conducted with adolescent-rated depressive symptoms as the outcome [AGN Inhibitory control errors: β=.15, $p<.05$; VF mental generativity: β=-.12, $p=.08$; AGN Shifting costs: β=.19, $p<.05$]. Interactions also replicated when excluding adolescents with a diagnosis of ADHD and were significant for all three measures of executive functioning [Combined parent-adolescent depressive symptoms: AGN Inhibitory control errors β=.19, $p<.05$; VF mental generativity β=-.18, $p<.01$; AGN Shifting costs β=.21, $p<.01$; Adolescent-rated depressive symptoms: AGN Inhibitory control errors β=.20, $p<.05$; VF mental generativity β=-.16, $p<.05$; AGN Shifting costs β=.21, $p<.05$]. Furthermore, when I repeated the analyses while controlling for the three additional parental depression variables (parent previous severe episode, parent age of onset and the adolescent's exposure to previous parent depressive episodes), none of the additional parent depression covariates were associated with the outcome measure, and the general pattern of interactive effects replicates with only minor differences (Appendix 2.2.).

Table 2.4. Current parental depression, offspring executive functioning and their interaction predicting offspring depressive symptoms

	AGN Inhibitory control errors (N=182)					VF Mental generativity (N=254)					AGN Shifting costs (N=181)				
	∆ R²	**p**	**B(S.E.)**	**β**	**p**	**∆ R²**	**p**	**B(S.E.)**	**β**	**p**	**∆ R²**	**p**	**B(S.E.)**	**β**	**p**
Step 1	.05	<.05				.06	<.001				.05	<.05			
Gender of adolescent			.43(.29)	.11	.13			.48(.24)	.12	<.05			.42(.29)	.11	.15
Age of adolescent			.37(.15)	.18	<.05			.40(.12)	.20	<.01			.36(.15)	.18	<.05
Step 2	.04	<.01				.03	<.01				.04	<.01			
Gender of adolescent			.55(.29)	.14	.06			.56(.24)	.14	<.05			.54(.29)	.14	.06
Age of adolescent			.32(.14)	.16	<.05			.36(.12)	.18	<.01			.31(.14)	.16	<.05
IQ of adolescent			-.44(.16)	-.20	<.01			-.34(.13)	-.17	<.01			-.43(.16)	-.20	<.01
Step 3	.13	<.001				.04	<.01				.10	<.001			
Gender of adolescent			.65(.27)	.16	<.05			.53(.23)	.14	<.05			.45(.28)	.12	.10
Age of adolescent			.53(.14)	.27	<.001			.39(.12)	.20	<.01			.36(.14)	.18	<.05
IQ of adolescent			-.33(.15)	-.15	<.05			-.29(.14)	-.14	<.05			-.38(.15)	-.18	<.05
Current PD			1.59(.34)	.32	<.001			1.04(.29)	.21	<.01			1.59(.35)	.32	<.001
EF measure (adolescent)			.44(.15)	.22	<.01			-.06(.13)	-.03	.65			-.02(.14)	-.01	.86
Step 4	.02	<.05				.01	.06				.02	<.05			
Gender of adolescent			.58(.27)	.15	<.05			.50(.23)	.13	<.05			.36(.27)	.09	.19
Age of adolescent			.51(.14)	.26	<.01			.38(.12)	.20	<.01			.38(.14)	.19	<.01
IQ of adolescent			-.32(.15)	-.15	<.05			-.26(.14)	-.13	.06			-.34(.15)	-.16	<.05
Current PD			1.63 (.33)	.33	<.001			.99(.29)	.20	<.01			1.54(.34)	.30	<.001
EF measure (adolescent)			.31 (.16)	.15	.05			.04(.14)	.02	.75			-.18(.15)	-.09	.23
EF measure (adolescent) x current PD			.92(.40)	.16	<.05			-.54(.28)	-.13	.06			.78(.33)	.18	<.05

Note: PD= Parental depression, EF=Executive functioning; Coding of the variables: AGN Inhibitory control errors: higher scores represent worse performance; VF Mental generativity: higher scores represent better performance; AGN Shifting costs: higher scores represent worse performance.

Figure 2.1. The interaction between current parental depression and measures of executive functioning as a predictor of adolescent depressive symptoms



2.3.4. Adolescent cognitive-emotional depressive symptoms and vegetative-somatic depressive symptoms

I next examined whether the buffering effect of executive functioning measures was present for both cognitive-emotional symptoms and vegetative-somatic symptoms. The interaction between all three executive function measures and current parental depression was consistently associated with adolescent cognitive-emotional depressive symptoms in the expected direction [AGN Inhibitory control errors: β=.14, p<.05; VF mental generativity: β=-.17, p<.05; AGN Shifting costs: β=.21, p<.01; Tables 2.5.1.-2.5.3.]. Only the interaction between current parental depression and AGN inhibitory control errors was significantly associated with vegetative-somatic symptoms (β=.15, p<.05; Table 2.5.1.). The interactions for the other two measures of executive functioning were non–significant [VF mental generativity: β=-.07, p=.32; AGN shifting costs β=.12, p=.14; Tables 2.5.2.-2.5.3.].

Table 2.5.1. Current parental depression, offspring inhibitory control errors and their interaction predicting offspring cognitive-emotional and vegetative-somatic depressive symptoms

	Cognitive-emotional symptoms (N=183)					Vegetative-somatic symptoms (N=184)				
	ΔR^2	**p**	**B(S.E.)**	**β**	**p**	**ΔR^2**	**p**	**B(S.E.)**	**β**	**p**
Step 1	.02	.17				.07	<.01			
Gender of adolescent			.25(.16)	.12	.12			.20(.17)	.09	.23
Age of adolescent			.08(.08)	.07	.33			.28(.08)	.24	<.01
Step 2	.05	<.01				.02	<.05			
Gender of adolescent			.31(.16)	.15	<.05			.25(.17)	.11	.14
Age of adolescent			.05(.08)	.05	.52			.26(.08)	.22	<.01
IQ of adolescent			-.26(.09)	-.22	<.01			-.18(.09)	-.14	<.05
Step 3	.12	<.001				.09	<.001			
Gender of adolescent			.32(.15)	.15	<.05			.33(.16)	.14	<.05
Age of adolescent			.14(.08)	.13	.09			.38(.09)	.33	<.001
IQ of adolescent			-.21(.08)	-.18	<.05			-.12(.09)	-.10	.18
Current PD			.92(.18)	.34	<.001			.66(.20)	.22	<.01
AGN inhibitory control errors			.15(.08)	.14	.07			.28(.09)	.24	<.01
Step 4	.02	<.05				.02	<.05			
Gender of adolescent			.29(.15)	.14	.05			.29(.16)	.12	.08
Age of adolescent			.12(.08)	.12	.12			.37(.09)	.32	<.001
IQ of adolescent			-.20(.08)	-.17	<.05			-.12(.09)	-.09	.19
Current PD			.94(.18)	.35	<.001			.69(.20)	.23	<.01
AGN inhibitory control errors			.08(.09)	.08	.33			.21(.09)	.18	<.05
AGN inhibitory control errors x Current PD			.44(.22)	.14	<.05			.49(.24)	.15	<.05

Note: PD=Parental depression.

Table 2.5.2. Current parental depression, offspring mental generativity and their interaction predicting offspring cognitive-emotional and vegetative-somatic depressive symptoms

	Cognitive-emotional symptoms (N=256)					Vegetative-somatic symptoms (N=258)				
	ΔR^2	**p**	**B(S.E.)**	**β**	**p**	**ΔR^2**	**p**	**B(S.E.)**	**β**	**p**
Step 1	.02	.07				.08	<.001			
Gender of adolescent			.22(.13)	.10	.09			.22(.14)	.10	.11
Age of adolescent			.10(.07)	.09	.14			.29(.07)	.25	<.001
Step 2	.03	<.01				.01	.11			
Gender of adolescent			.27(.13)	.13	<.05			.25(.14)	.11	.07
Age of adolescent			.08(.07)	.07	.25			.28(.07)	.24	<.001
IQ of adolescent			-.21(.07)	-.18	<.01			-.12(.07)	-.10	.11
Step 3	.06	<.001				.02	.10			
Gender of adolescent			.25(.13)	.12	.05			.24(.14)	.10	.09
Age of adolescent			.09(.07)	.09	.17			.29(.07)	.25	<.001
IQ of adolescent			-.19(.08)	-.16	<.05			-.10(.08)	-.08	.25
Current PD			.65(.16)	.24	<.001			.37(.18)	.13	<.05
VF mental generativity			-.02(.07)	-.02	.81			-.03(.08)	-.03	.68
Step 4	.02	<.05				.004	.32			
Gender of adolescent			.24(.13)	.11	.07			.23(.14)	.10	.10
Age of adolescent			.08(.07)	.08	.20			.29(.07)	.25	<.001
IQ of adolescent			-.16(.08)	-.14	<.05			-.09(.08)	-.07	.30
Current PD			.61(.16)	.22	<.001			.36(.18)	.12	<.05
VF mental generativity			.06(.08)	.06	.45			.00(.08)	.00	.99
VF mental generativity x Current PD			-.40(.16)	-.17	<.05			-.17(.17)	-.07	.32

Note: PD=Parental depression.

Table 2.5.3. Current parental depression, offspring shifting costs and their interaction predicting offspring cognitive-emotional and vegetative-somatic depressive symptoms

	Cognitive-emotional symptoms (N=182)					Vegetative-somatic symptoms (N=183)				
	ΔR^2	p	B(S.E.)	β	p	ΔR^2	p	B(S.E.)	β	p
Step 1	.02	.19				.06	<.01			
Gender of adolescent			.24(.16)	.11	.13			.19(.17)	.08	.26
Age of adolescent			.08(.08)	.07	.35			.28(.08)	.24	<.01
Step 2	.04	<.01				.02	.05			
Gender of adolescent			.31(.16)	.14	.05			.24(.17)	.10	.16
Age of adolescent			.05(.08)	.04	.54			.26(.08)	.22	<.01
IQ of adolescent			-.25(.09)	-.22	<.01			-.18(.09)	-.14	.05
Step 3	.11	<.001				.05	<.01			
Gender of adolescent			.26(.15)	.12	.08			.21(.16)	.09	.21
Age of adolescent			.08(.08)	.07	.30			.28(.08)	.24	<.01
IQ of adolescent			-.22(.08)	-.19	<.01			-.16(0.9)	-.12	.08
Current PD			.92(.19)	.34	<.001			.66(.21)	.22	<.01
AGN shifting costs			-.01(.07)	-.01	.87			-.01(.08)	-.01	.87
Step 4	.03	<.01				.01	.14			
Gender of adolescent			.20(.15)	.10	.17			.17(.17)	.07	.30
Age of adolescent			.09(.07)	.08	.23			.28(.08)	.24	<.01
IQ of adolescent			-.20(.08)	-.17	<.05			-.14(.09)	-.11	.12
Current PD			.89(.19)	.33	<.001			.64(.21)	.22	<.01
AGN shifting costs			-.11(.08)	-.10	.19			-.07(.09)	-.06	.43
AGN shifting costs x Current PD			.48(.18)	.21	<.01			.30(.20)	.12	.14

Note: PD=Parental depression

2.4. Discussion

This is the first study to examine whether higher executive functions confer protection against adolescent depressive symptoms in the presence of a current episode of parental depression. Consistent with a number of previous studies and a previous analysis of this cohort (Mars et al., 2012), the findings of this study confirmed that within a high-risk cohort of adolescents, a current episode of MDD in the parent was associated with higher levels of depressive symptoms in the offspring. The main findings of this study indicated that when a parent was in a current episode of depression, adolescents with better executive functioning had significantly fewer total depressive symptoms compared to adolescents with poorer performance on these measures. Previous research has emphasized the importance of executive functioning in regulating thoughts and emotions and in coping with stress (Sanchez et al., 2013). Mental flexibility has been suggested to be associated with mechanisms underlying effective coping with adversity such as the generation of solutions to problems and the positive reappraisal of negative events (Diamond, 2013; McRae et al., 2012; Stahl & Pry, 2005). Although further research is required to examine the mechanisms behind the buffering effects of executive functioning observed in this study, the findings of this study suggest that inhibitory control and mental flexibility may serve as important cognitive resources that facilitate the ability of young people to cope with having a currently depressed parent.

The mean difference in DSM-IV total depressive symptoms between adolescents with higher (upper tertile) versus lower (lower tertile) executive functioning was fairly substantial, around two depressive symptoms that met strictly defined thresholds according to a semi-structured clinical interview. As the DSM-IV diagnostic criteria for a major depressive episode requires the presence of 5 symptoms (out of 9 possible symptoms) this difference might have clinical and functional implications and highlights the importance of executive functioning as a potential protective factor. Thus, executive functioning appears to be a potentially clinically important protective factor for the adolescent offspring of depressed parents. Whilst interventions targeting executive functioning have been assessed for educational and neurodevelopmental difficulties in children and adults as well as for currently depressed adults (Melby-Lervåg & Hulme, 2013; Rapport, Orban, Kofler, & Friedman, 2013; Siegle, Ghinassi, & Thase, 2007; Titz & Karbach, 2014), to my knowledge such programmes have not been tested in relation to

individuals at high risk for mood disorders. It has been suggested that common intervention strategies such as CBT, encourage patients to employ "executive control" over negative automatic thoughts and affective responses (Siegle et al., 2007). It is possible that those with difficulties in executive functioning find these requirements of CBT demanding and considering enhancing executive functioning as an adjunct to more traditional preventive approaches may be warranted.

It is worth noting that inhibitory control and shifting costs were assessed by a task that involves the processing of affective information whereas mental generativity was assessed by a task that did not involve an explicit emotional component. Previous research has emphasized cognitive control impairments in the context of emotionally salient information as a factor associated with risk for depression and persistence of symptoms (Gotlib & Joormann, 2010; Gotlib, Joormann, & Foland-Ross, 2014; Kilford et al., 2015) which suggests that the buffering effect observed in the present study might be expected to be larger for 'emotional' executive functioning. Overall, the present study found relatively little evidence of this with similar effect sizes observed for both types of executive functioning task. Although the buffering effect of VF mental generativity only approached significance I did not interpret it differently from the significant buffering effects of AGN inhibitory control errors and AGN shifting costs as each of the three interactive effects had similar effect sizes and I did not directly test whether effects sizes were significantly different. I also found little evidence of valence specific effects when the interaction between current parental depression and AGN inhibitory control errors was examined separately for inhibitory control errors for blocks with a negative target and blocks with a positive target [negative target blocks (β=.17, $p<.05$); positive target blocks (β=.15, $p<.05$)].

There was some indication that the protective effects of executive functioning were somewhat more consistently observed for cognitive-emotional symptoms than for vegetative-somatic symptoms. However, it is important to note that I did not formally test differences. Executive functioning might be predicted to particularly confer protection against cognitive-emotional depressive symptoms given the important role that executive functions have in emotional regulation. The finding that only the interaction with inhibitory control and current parent depression was significant for both cognitive-emotional and vegetative-somatic symptoms is intriguing but requires replication.

I did not find evidence for significant main effects of executive functioning measures on adolescent depressive symptoms and instead found a main effect of IQ on

adolescent depressive symptoms. The finding of a main effect for IQ on adolescent depressive symptoms is consistent with evidence that IQ protects against familial risk for depression and other psychiatric disorders (Kendler, Ohlsson, Sundquist, & Sundquist, 2015; Zammit et al., 2004). One possible reason for no main effect of executive functioning on adolescent depressive symptoms may be differences between depressive symptoms and disorder. Previous studies in children and adolescents that demonstrated an association between depression and impairments in executive functioning examined those with a current episode of MDD (Maalouf et al., 2011; S. Wagner et al., 2015). Thus, executive dysfunction may appear in a full blown episode, rather than with depressive symptoms. Furthermore, previous studies in adults have showed that executive functioning impairments may be related to current and past severity and/or chronicity in those with MDD, which may suggest a "scarring effect" that can lead to the persistence of executive functioning difficulties in those with severe/chronic depression (Basso & Bornstein, 1999; Karabekiroğlu, Topçuoğlu, Gimzal Gönentür, & Karabekiroğlu, 2010; McDermott & Ebmeier, 2009). In the EPAD sample, only 38 individuals (11%) met DSM-IV criteria for current MDD as assessed by the CAPA interview at any of the three assessments of the study. Although these rates are higher than those found in community studies where similarly stringent criteria where applied (1-2%; Copeland, Shanahan, Worthman, Angold, & Costello, 2012; Ford, Goodman, & Meltzer, 2003), reflecting the increased familial risk of MDD in this sample, they nevertheless indicate that the majority of the sample had not experienced an MDD episode. Finally, executive functioning impairments in those with MDD may be related to comorbidity with other disorders such as ADHD, anxiety disorders, or disruptive behaviour disorders (Snyder, 2013). Indeed, MDD in young people is very often comorbid with other psychiatric disorders, with previous studies reporting comorbid disorders in 40-70% of children and adolescents with MDD (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015; Birmaher et al., 1996; Ford et al., 2003). However, the association between executive functioning impairments and comorbidity with other disorders in those with MDD has not been thoroughly examined in previous studies, especially in young people. As exploring this was beyond the scope of this study, it may be beneficial to examine this in future studies.

It is also worth noting that overall findings in this study are consistent with findings in a previous examination of the associations between measures of parental depression severity and course and adolescent depressive symptoms in this cohort (Mars et al., 2012). Minor differences in results are related to the use of different analyses and different

outcome measures. I did not find evidence for main effects of current parental depression as well as other indicators of parental depression (e.g. age of onset, past severity and the adolescent's exposure to previous parent depressive episodes) on adolescent executive functioning. A few studies have examined whether there is an association between parental depression and offspring executive functions and results have been mixed. Hughes et al.(2013), found that mothers' depressive symptoms when the child age was 2 as well as the change in maternal depressive symptoms between the child ages of 2-6, predicted childrens' executive functioning at age 6 in a community sample of low income families. Studies examining parental diagnosis of MDD have found no evidence of an association between parental depression and offspring executive functioning (Klimes-Dougan, Ronsaville, Wiggs, & Martinez, 2006; Micco et al., 2009; C. Wagner, Abramson, & Alloy, 2015). This cross-sectional examination assessed adolescents whose parents had a history of recurrent depression, so it is not possible to rule out that other indicators of parental depression had earlier effects on offspring executive functions or that changes in parental depression had effects on the development of offspring executive functioning over time.

I conducted analyses for three executive functioning measures and as suggested by Rothman (1990, 2014) did not correct for multiple comparisons. I examined comorbidity with ADHD but was not able to examine the role of comorbid anxiety because of the high correlation with depression in this sample (Gorman, 1996). It is therefore unclear whether anxiety may affect the observed buffering effect of executive functioning on depressive symptoms. Further research is required in order to investigate this.

It is important to note the wide age range of this sample (10-18 years). As executive functions continue to develop during adolescence (Best & Miller, 2010) it is possible that the buffering effect of executive functions may differ for younger and older adolescents. It is also possible that younger and older adolescents may experience the executive functioning tasks differently in terms of understanding of the instructions and level of difficulty and thus complete it differently (Kilford et al., 2015). In this study, I controlled for age but did not examine two way interactions of age and executive functioning or 3 way interactions of exposure to current parental depression, age and executive functioning. Thus, future studies should examine whether the protective effect of executive functioning may vary across adolescence.

Additional limitations include the cross-sectional and observational nature of the data. Thus, it is important to note that both unmeasured environmental and genetic factors, that is, residual confounding, could account for the associations between parent depression and offspring depression and between offspring executive functioning and offspring depression. As a cross sectional examination of the association between a current episode in the parent and offspring depressive symptoms was conducted, it is not possible to infer any causal relationship between indicators of parental depression and offspring depressive symptoms. Whilst it is possible that reduced executive functioning may stem from aspects of depression not examined in this study such as rumination (Davis & Nolen-Hoeksema, 2000; Watkins & Brown, 2002) or from motivational problems (Ellis, 1991), if poor executive functioning was a result of depressed mood or another feature of depression such as rumination, significant correlations between depressive symptoms and performance in executive functioning measures would be expected. In this sample I did not find such correlations (Table 2.1.) but rather an interaction effect of parental depression and executive functioning measures on depressive symptoms. Findings of a protective effect of executive functioning are cross-sectional meaning that the protective effect on depressive symptomatology may not persist over time. The focus on a high-risk sample of adolescents may also limit the extent to which findings generalise to other populations.

Further research is required to elucidate the pathways through which risk for depression is transmitted from parents to offspring and examine how executive functions may buffer this risk. Thus, longitudinal studies that assess executive functioning early in life and prior to the development of offspring depression are warranted in order to rule out the possibility of earlier effects of parental or offspring depression on the development of offspring executive functions. It would also be informative to examine executive functioning as a mediator of depressive symptom change in preventive trials of high risk groups and to consider the possibility of incorporating executive functioning training in prevention programs aimed at increasing resilience in these groups.

This is the first study to demonstrate that executive functions may protect against depressive symptoms when adolescents are exposed to a current episode of parental depression. The assessment of executive function skills may help in the early detection of vulnerable individuals that are likely to be less ‘resilient’ when exposed to parental depression. These findings have therapeutic implications as preventive interventions could

target executive functions as a way to promote resilience and to enhance the efficacy of existing interventions in high risk groups.

CHAPTER 3: Examining overgeneral autobiographical memory as a predictor of mental health resilience and social functioning in high risk adolescents

Previous research has demonstrated that difficulties in retrieving specific autobiographical memories (referred to as overgeneral memory; OGM), characterise individuals with depression and a history of trauma, and may also serve as a cognitive risk factor for developing depression. However, OGM has not been examined as a predictor of positive outcomes such as mental health resilience and social functioning. Moreover, despite its clinical importance, little is known about potential developmental antecedents of OGM. This study examined whether OGM serves as a predictor of "better than expected" mental health resilience and social functioning in longitudinal study of adolescent offspring of parents with a history of MDD. Furthermore, this study also explored whether environmental factors such as recent negative life events and current parental depression severity are associated with OGM in adolescents. "Better than expected" mental health resilience was assessed as lower than predicted mood and behavioral symptoms given indices of familial risk for depression. Social functioning was assessed as quality of peer relationships as reported separately by adolescents and their parents. Results indicated that the retrieval of overgeneral memories, primarily in response to negative cue words, prospectively predicted adolescent rated quality of peer relationships and resilience to mood symptoms but not resilience to behavioral symptoms. There was no evidence of an association between current parental depression severity and the retrieval of overgeneral memories. There was however evidence of an association between recent negative life events and a greater retrieval of overgeneral memories primarily in response to negative cue words. Finally, mediation analysis suggested that that the retrieval of overgeneral memories in response to negative cue words mediated the association between recent negative life events and adolescent rated quality of peer relationships. These findings suggest that difficulties in retrieving specific autobiographical memories may diminish resilience to mood symptoms and impair social functioning. Also, findings suggest that recent negative life events may be associated with the development of OGM.

3.1. Introduction

Recent research has highlighted the potential role of autobiographical memory in the development of emotional disorders. A large body of research has shown that difficulties in retrieving specific autobiographical memories characterise individuals with depression or a history of trauma (Kuyken et al., 2006; Park et al., 2002; Vrielynck et al., 2007; Williams et al., 2007). Such difficulties are referred to as overgeneral memory (OGM). OGM describes a difficulty in retrieving autobiographical memories that occurred at a specific time and place when requested to do so, and a tendency instead to describe a category of events, or a memory that occurred over an extended period of time (Valentino, 2011; Williams et al., 2007). As described in chapter 1, a widely accepted theoretical model of the emergence of OGM, the CaR-FA-X model (Williams et al., 2007), suggests that three possible mechanisms may account for the emergence of OGM: 1) A response of "functional avoidance" to exposure to traumatic/negative events, which is expressed as a tendency to truncate the retrieval process before retrieving a specific memory in order to avoid recalling details of memories of that may cause negative mood. It has been hypothesised that this response may develop initially as a coping mechanism for negative material, but then generalise with time to other material (Dalgleish, Spinks, Yiend, & Kuyken, 2001; Raes, Hermans, de Decker, Eelen, & Williams, 2003; Williams et al., 2007). 2) A tendency for rumination among depressed individuals that may lead to attention being "captured" by irrelevant negative material during the retrieval process. 3) Impairments in executive functions that affect the ability to retrieve specific memories.

OGM has been examined mostly in the context of patients with depression and post-traumatic stress disorder (PTSD) (Valentino, 2011; Williams et al., 2007) but has not been systematically explored in the context of positive outcomes such as mental health resilience and adaptive functioning. Thus, the primary aim of this chapter will be to investigate OGM as a predictor of mental health resilience and adaptive functioning in adolescents. As previous studies examined OGM mostly as a risk factor for depression in young people (Hipwell et al., 2011; Rawal & Rice, 2012; Woody et al., 2015), a secondary aim will be to investigate the specificity of the association of OGM with resilience to mood symptoms, as opposed to resilience to behavioral symptoms. Furthermore, a third research aim will be to explore potential developmental antecedents of OGM, as this topic has received surprisingly little empirical examination (Valentino, 2011).

3.1.1. Resilience and Mental Health

Resilience has been generally defined as an individual's ability to cope adaptively with stressful events or adversity (Feder et al., 2009). Mental health resilience has been defined in various ways, but is commonly conceptualised as a "better than expected" mental health outcome given exposure to risk or adversity compared to other individuals who have been exposed to similar levels of risk (Collishaw et al., 2016; Rutter, 2013). Resilience may be brought about by reinforcing protective factors, and preventive interventions are at least partly driven by the notion that mental health resilience can be enhanced in high risk populations (Feder et al., 2009; Haglund, Nestadt, Cooper, Southwick, & Charney, 2007; Rice & Rawal, 2010). Several known psychosocial mechanisms have been suggested in previous research as factors that promote mental health resilience: active coping strategies (such as problem solving, reappraisal and planning), optimism, positive affectivity, spirituality, social competence and the ability to utilise social support (Feder et al., 2009; Southwick et al., 2005).

It has been suggested that the ability to retrieve autobiographical memories is essential for a coherent sense of self, and important for facilitating effective problem-solving and processing of emotional information by reflection on past experiences (Williams et al., 2007). Thus, autobiographical memory seems to be an important cognitive mechanism to consider in the context of mental health resilience.

3.1.2. Autobiographical Memory, Resilience and Mental Health

Previous studies on autobiographical memory specificity and mental health have mostly focused on exploring impairments in retrieving specific autobiographical memories in currently depressed adults, suicidal patients, and patients suffering from PTSD (S. A. Moore & Zoellner, 2007; Valentino, 2011). A few prospective studies have also indicated that OGM may act as a risk marker for depression in adolescents (Hipwell et al., 2011; Rawal & Rice, 2012). However, previous studies have not examined OGM as a predictor of positive outcomes such as mental health resilience and adaptive functioning. It has been suggested that OGM may increase risk specifically for affective disorders (Rawal & Rice, 2012; Williams et al., 2007). Furthermore, as only one study to date has examined OGM as a predictor of several disorders (Rawal & Rice, 2012), it is not clear whether OGM may predict resilience specifically to mood symptoms or also to symptoms of other disorders common in childhood and adolescence, such as behavioral disorders.

3.1.3. Autobiographical Memory and Social Functioning

To date OGM has not been examined as a predictor of aspects of adaptive functioning, such as social functioning. Social functioning is an essential part of development and is strongly associated with happiness, health and emotional well-being (Baumeister & Leary, 1995). It has been argued that the ability to form relationships and receive social support has protective effects against mental illness, and has been linked with better mental health resilience under adversity or stressful circumstances (Baumeister & Leary, 1995; Feder et al., 2009; Rutter, 2013; Southwick et al., 2005). It has been shown that greater specificity of autobiographical memory is associated with better social problem solving in experimental tasks in depressed or suicidal adults (J. Evans, Williams, O'loughlin, & Howells, 1992; Goddard et al., 1996; Pollock & Williams, 2001; Raes et al., 2005). As such, it is reasonable to hypothesise that autobiographical memory specificity may influence the nature of an individual's social functioning in everyday life. The extant experimental studies, in addition to lacking ecological validity, were cross sectional and did not examine whether OGM can predict any aspect of everyday social functioning over time.

3.1.4. Antecedents of Autobiographical Memory Functioning

Despite the clinical importance of OGM and its emerging role in mental health functioning, surprisingly little is known about environmental factors that may contribute to its emergence. It has been suggested that early trauma may contribute to the development of OGM (S. A. Moore & Zoellner, 2007; Williams, 1996). Thus, Williams hypothesised that when retrieving memories, individuals who experienced traumatic events may employ “functional avoidance" in order to prevent the retrieval of specific negative memories that may elicit negative emotions, and this will lead to other memories becoming overgeneral (Williams, 1996; Williams et al., 2007). Various studies, most of them cross-sectional, have compared autobiographical memory in those with and without PTSD or those with or without exposure to potentially traumatic events such as abuse, burn injuries and breast cancer (S. A. Moore & Zoellner, 2007). However, few studies have examined other environmental factors that might increase risk for OGM. For example, it is not known whether common negative life events (which may not be considered at “traumatic” events), such as parental divorce or the loss of someone close, may also influence OGM. It is plausible to hypothesise that those who experience negative life events may also develop OGM to avoid the retrieval of painful memories.

Another common environmental factor that has been suggested to affect autobiographical memory is parental depression (Valentino, 2011; Woody et al., 2015). Developmental research suggests that parental depression may affect several aspects of parent-child relationships that are important for the development of autobiographical memory. For example, it has been suggested that parent-child conversations about past events, especially following exposure to stressful events, may facilitate the development of autobiographical memory and reduce the likelihood of the child developing an avoidant coping style which may be associated with the emergence of OGM as suggested by Williams' functional avoidance theory (Bird & Reese, 2006; Valentino, 2011; Williams et al., 2007). Parental depression is likely to affect the parent's resources to engage in parent-child conversations and to give support following negative or stressful events (Lovejoy, Graczyk, O'Hare, & Neuman, 2000). However, only one study to date has examined parental depression as a risk factor for OGM in children (Woody et al., 2015). Indeed, Woody et al. (2015) found that children of depressed mothers retrieved fewer specific memories in response to negative cue words compared to children of non-depressed mothers.

One high risk group that may be especially relevant for examining both the antecedents and the outcomes of OGM are offspring of depressed parents. Previous research has suggested that children of depressed parents might be at increased risk for developing OGM as they are exposed to environmental factors that may contribute to the development of OGM such as parental depression and stressful life events. For example, it has been found that children of depressed parents are exposed to more environmental stressors such as marital discord and financial problems compared to children of healthy parents (S. H. Goodman & Gotlib, 1999; Hammen, 2002). Moreover, children of depressed parents are at increased risk for both internalising and externalising problems (Beardslee et al., 2011; S. H. Goodman et al., 2011) as well as social impairment (Weissman et al., 1997). Due to the detrimental outcomes of familial risk for depression, there has been call for studies to examine cognitive mechanisms related to risk and resilience in this high-risk group (Beardslee et al., 2011; Rice & Rawal, 2010). Evidence of the potential clinical importance of OGM suggests that examining how it may be related to mental health resilience in the context of familial risk for depression may help inform future prevention efforts.

3.1.5. Study Aims

The primary aim of this study is to examine OGM as a predictor of indices of mental health resilience and social functioning in a high risk sample of adolescent offspring of parents with a history of MDD. Specifically, I examined whether OGM is associated over time with resilience to both mood and behavioural symptoms (given exposure to familial risk) and with quality of adolescent peer relationships. The specificity of the influence of OGM on resilience to mood symptoms compared with resilience to behavioral symptoms was examined as a secondary aim. Following theoretical accounts of OGM and previous findings (Rawal & Rice, 2012; Williams et al., 2007) it was hypothesised that OGM would be associated with social functioning and mental health resilience over time, and specifically with resilience to mood symptoms. As a third aim, I examined whether proximal environmental risk factors such as recent negative life events and current parental depression severity were associated with adolescent OGM. It was hypothesised that recent negative life events and current parental depression severity would be associated with OGM (Williams et al., 2007; Woody et al., 2015). Finally, if any hypothesised associations are found between environmental risk factors and OGM, and between OGM and indices of mental health resilience and social functioning, the potential role of OGM as a mediator of the association between environmental risk factors and indices of mental health resilience and social functioning will also be examined. As previous studies showed mixed findings with regard to cue word valence effects of OGM in current depression and risk for depression (Hipwell et al., 2011; Rawal & Rice, 2012; Valentino, 2011; Williams et al., 2007; Woody et al., 2015; Young, Bellgowan, Bodurka, & Drevets, 2013), I examined OGM separately for positive and negative cue words when exploring the aforementioned research questions.

3.2. Methods

3.2.1. Participants and procedure

Participants of this study were part of a longitudinal study of parents with a history of MDD and their adolescent offspring (EPAD: the Early Prediction of Adolescent Depression). Details on the recruitment of subjects and inclusion and exclusion criteria are included in chapter 2. This study uses data collected at the second assessment of the EPAD study, when adolescents completed the Autobiographical Memory Test, and from 1-year follow-up. Thus, the second assessment will be referred to in this study as baseline.

As detailed in Appendix 3.1., 337 families were eligible to participate in the baseline sample of the study. Among these, 2 families were excluded because the index parent was diagnosed with bipolar disorder, and 4 additional families were excluded for the purpose of this study because the adolescents were not exposed to parental depression during their lifetime (Collishaw et al., 2016). Full psychopathology data was available for 285 adolescents at baseline, of whom 254 also had data from the autobiographical memory task. Among these, 240 adolescents had psychopathology data at follow up, 232 adolescents completed questionnaire ratings of peer relationship quality and 238 parents completed ratings of adolescent peer relationship quality.

3.2.2. Measures

3.2.2.1. Autobiographical memory

OGM was assessed using the Autobiographical Memory Test(AMT; Williams & Broadbent, 1986) . In this task, participants were required to retrieve specific autobiographical memories in response to emotional cue words. For each cue word, participants were asked to describe a specific event which the word reminded them of. The event had to last less than a day and occur at a particular time and place. Prior to beginning the task, three practice cue-words were given with feedback if non-specific responses were provided by the participant. Participants were then read 12 emotional cue words, 6 negative and 6 positive (alternating between positive and negative) and were required to retrieve a specific autobiographical memory in 30 seconds. To select the cue words used in this study, first a list of cue words used in previous studies with adolescents was compiled(Kuyken et al., 2006; Park et al., 2002; Swales, Williams, & Wood, 2001). The frequency of each cue word was then examined and rated for emotionality and imageability by a panel of researchers. Finally, 2 sets of 6 negative cue words (Word set 1: mistake, rejected, weakness, needy, angry, tired; word set 2: failure, disliked, ugly, useless, worse, lonely) and 2 sets of 6 positive cue words (word set 1: loyal, joy, smile, achieve, loved, ambitious; word set 2: friendly, happy, respect, caring, sunny, perfect) that were matched on frequency, emotionality and imageability were selected(Williams et al., 2007).

If participants did not recall a specific memory, they were prompted with questions aimed to get more details on the memory and encourage them to recall a particular time. Answers were taped and transcribed. Participants' responses were coded as specific

(memories that regarded a specific time and place), overgeneral (either categorical responses that referred to repeated events, or extended responses that referred to events that happened over long periods of time), semantic associates (responses that relate to the cue word but do not include a memory) and omissions (if no response was given). Two independent raters coded responses from 45 participants at the first administration of AMT. Inter-rater agreement was high (average agreement for all 12 cue words: k=.93). The number of total overgeneral memories, overgeneral memories for positive cue words and overgeneral memories for negative cue words served as the outcome measures (referred to later on as OGM total, OGM negative, OGM positive).

3.2.2.2. Quality of peer relationships

Adolescent rating

A questionnaire of 10 statements was used to assess quality of peer relationships. The questionnaire included statements about friendships (e.g. “other children want to be my friend”) and peer rejection (e.g. “other children often tease me”). Adolescents were asked to rate each statement on a 5 point scale (1-strongly agree, 5-strongly disagree). Answers were coded so that higher scores reflected poorer quality of peer relationships. This questionnaire was devised for the purpose of this study (Appendix 3.2.). Cronbach's alpha at baseline was 0.87. The total score on this questionnaire was significantly correlated with the score of the peer problems subscale in the Strengths and Difficulties Questionnaire (SDQ) as rated by the adolescent ($r=.59$, $p<.001$), which provides good evidence of convergent validity.

Parent rating

A shorter 3-item questionnaire was used to assess parental rating of the quality of the adolescents’ peer relationships. The questionnaire included questions about friendships and peer rejection. This questionnaire was devised for the purpose of this study (Appendix 3.3.). Answers were coded such that higher scores reflected poorer quality of adolescent peer relationships. Cronbach's alpha at baseline was 0.68. The total score on this questionnaire was significantly correlated with the adolescent rating of quality of peer relationships ($r=.44$, $p<.01$) and parent rated SDQ peer problems subscale ($r=.66$, $p<.001$) which provides evidence of convergent validity.

3.2.2.3. Familial risk indices

The following indices of familial risk for depression were assessed: parental age of onset, parental depression severity and family history of depression in first and second degree relatives (additional to the parent). Parents were interviewed at each assessment with the Schedules for Clinical Assessment in Neuropsychiatry (SCAN; Wing et al., 1990) in order to assess the presence of depressive episodes in the month preceding the assessment and between the assessments. The SCAN is a well validated psychiatric interview that provides an assessment of mental health disorders in adults. A life history calendar approach was used to assess past episodes, functional impairment of the parent's worst two episodes and age of onset (Caspi et al., 1996; Freedman et al., 1988). Additionally, the parent was also interviewed regarding family history of depression in the adolescent's other parent, siblings and grandparents. Parental age of onset was defined as either ≤20 or 21 years or older following previous research (Weissman et al., 1984). Parental depression severity was dichotomized (coded 0,1) and was considered as present if any past episode prior to the baseline assessment of the study involved hospitalization or severe functional impairment (as defined by a score of GAF≤30 on the Global Assessment of Functioning scale in DSM-IV; American Psychiatric Association, 1994). Family history of depression was defined as a count of relatives with history of MDD among the adolescent's parents, siblings and grandparents.

3.2.2.4. Adolescent mood/behavioural symptoms

Mood and behavioural symptoms in the adolescent were assessed using the Child and Adolescent Psychiatric Assessment (CAPA; Angold & Costello, 2000). The CAPA is a well validated semi-structured interview used to assess psychiatric symptoms and disorders over the last 3 months in children and adolescents aged 9-18. Adolescents and their parents were interviewed separately regarding the adolescent's psychopathology. The sections assessing depressive disorders and behaviour disorders (oppositional defiant disorder and conduct disorder) were used to determine current number of mood/ behavioural symptoms at each assessment. A combined report of the parent and the adolescent was used to assess symptoms count, such that a symptom was considered present if reported by either the adolescent or the parent, as conducted in common clinical practice (Angold, Prendergast, et al., 1995; Costello et al., 2003). The outcome measures of total number of mood symptoms and total number of behavioural symptoms were used

to devise indices of mental health resilience (i.e. resilience to mood symptoms and resilience to behavioural symptoms). The outcome measure of total number of mood symptoms was also used as a control variable in sensitivity analyses as described later.

3.2.2.5. Mental health resilience

Mental health resilience was defined as "better than expected" mood and behavioural symptoms scores given several indices of familial risk for depression (Amstadter, Myers, & Kendler, 2014; Rutter, 2013). These scores were adapted from Collishaw et al. (2016). Resilience to mood and behavioural symptoms scores were calculated by regressing mood and behavioural symptoms counts at follow up on familial risk indices (parent depression age of onset, parent depression severity, and family history of depression). Prior to computing the resilience scores, mood symptoms counts were log-transformed to approximate normality of residuals. Residual scores derived from the regression analyses served as resilience scores. Negative residual scores represent "better than expected" resilience to mood/behavioural symptoms.

3.2.2.6. Environmental risk factors

Recent negative life events

Negative life events were assessed by the life events checklist (J. H. Johnson & McCutcheon, 1980).The adolescent and parent reported separately whether a list of negative life events had occurred in the adolescent's life over the recent year prior to the baseline assessment. The list included 20 items of events such as divorce, death in the family, losing a close friend through an argument etc. An event was considered as present if reported by either the adolescent or the parent (Gest, Reed, & Masten, 1999). The number of events was summed to a total score.

Parent current depression severity

Parent current depression severity at baseline was assessed using the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979; Sellers et al., 2013). The BDI is a widely used self-report questionnaire which assesses current severity of depressive symptoms. The questionnaire includes 21 items scored on a scale of 0-3. Higher scores reflect greater severity of symptoms.

3.2.3. Statistical analysis

In order to explore whether OGM was associated with mental health resilience and social functioning over time, I first examined (using Pearson's correlation coefficients) the associations between OGM variables at baseline and indices of resilience and social functioning at follow up (i.e. scores of "better than expected" resilience to mood/behavioural symptoms, and adolescent and parent rated quality of peer relationships scores). Significant correlations were followed up with hierarchical multiple linear regression analyses in which I examined the effect of baseline OGM variables on indices of resilience and functioning at follow-up, while controlling for baseline scores of mental health resilience/social functioning and the covariates of age, gender, and IQ. Separate analyses were conducted for OGM total score, OGM negative (negative cue words) and OGM positive (positive cue words). Variables were entered in the following steps: 1) OGM variable (total/ negative/positive) 2) baseline score of the mental health resilience/social functioning index examined as the outcome measure 3) covariates of age, IQ and gender.

To examine whether environmental risk factors were associated with OGM, I first examined Pearson's correlation coefficients between environmental risk factors (i.e. parental depression severity at baseline, recent negative life events at baseline) and OGM variables. Significant correlations were followed up by hierarchical multiple regression analyses in which I examined the effects of environmental risk factors on OGM variables while controlling for age, IQ and gender. Variables were entered in the following steps: 1) OGM variable (total/ negative/positive) 2) covariates of age, IQ and gender.

In light of the extant literature establishing an association between OGM and mood (van Vreeswijk & de Wilde, 2004; Williams et al., 2007) a sensitivity analysis examined whether any effect of OGM on social functioning was driven by an effect of mood on social functioning. This was explored by controlling for baseline mood symptoms in the regression analyses examining the associations between baseline OGM variables and follow-up adolescent/parent rated quality of peer relationships. It is worth noting that examining mood as a covariate was not necessary in the regression analyses exploring associations between baseline OGM variables and resilience to mood symptoms at follow-up, as these analyses controlled for baseline resilience to mood symptoms (which was calculated based on scores of baseline mood symptoms).

Furthermore, in light of the extant literature establishing an association between negative life events and mood (Bolger, DeLongis, Kessler, & Schilling, 1989; Hammen,

2005), a sensitivity analysis examined whether any effects of negative life events on OGM were driven by an effect of mood on OGM. This was explored by controlling for baseline mood symptoms in the regression analyses examining associations between recent negative life events and baseline OGM measures.

Finally, in order to investigate whether OGM mediated the association between environmental risk factors and indices of mental health resilience and social functioning, first associations between environmental risk factors and adolescent indices of mental health resilience and social functioning at follow-up were examined using Pearson's correlation coefficients. Indirect effects of environmental risk factors on adolescent mental health resilience and social functioning were then examined, via OGM variables according to criteria outlined by Hayes (Hayes, 2009, 2012). Indirect effects were tested using 95% bias-corrected bootstrapped confidence intervals. Bias-corrected bootstrapping was found to be a robust method of testing indirect effects (Hayes, 2009; MacKinnon, Lockwood, & Williams, 2004)

3.3. Results

3.3.1. Preliminary analysis

As presented in Table 3.1., the variables of OGM total, OGM negative and OGM positive at baseline were significantly associated with adolescent-rated quality of peer relationships (OGM total r=.23, p<.01; OGM negative r=.24, p<.01; OGM positive r=.14, p<.05) and parent-rated quality of peer relation at follow up (OGM total r=.21, p<.01; OGM negative r=.16, p<.05; OGM positive r=.18, p<.01) in the expected direction. Among OGM variables, only OGM negative at baseline was significantly associated with resilience to mood symptoms at follow up in the expected direction (OGM total r=.11, p=.11; OGM negative r=.18, p<.01; OGM positive r=-.02, p=.77). Furthermore, none of the OGM variables at baseline were associated with resilience to behavioural symptoms at follow up (OGM total r=.005, p=.94; OGM negative r=.10, p=.15; OGM positive r=-.09, p=.16). The covariate of age was associated at a trend level with baseline parent report of adolescent quality of peer relationships and was significantly associated with follow-up parent report of adolescent quality of peer relationships (baseline: r=.11, p<.1; follow-up: r=-.13, p<.05). Age was also significantly associated with resilience to mood symptoms at baseline and follow up (baseline: r=.24, p<.01; follow-up: r=.17, p<.01). The covariate of gender was associated with resilience to mood symptoms at follow-up (r=.15, p<.05). The covariate of IQ was associated at a trend level with resilience to mood symptoms at baseline [r=-.12,

p<.1]. Therefore, follow-up regression analyses aimed to examine baseline OGM variables as predictors of social functioning and mental health resilience at follow up, while controlling for baseline scores of mental health resilience/social functioning and covariates of age, IQ and gender, were conducted only for the following outcomes: adolescent rated quality of peer relationships, parent rated quality of peer relationships, and resilience to mood symptoms.

Among environmental risk factors, recent negative life events were significantly associated with baseline OGM total and OGM negative in the expected direction, but not with OGM positive (OGM total r=.13, p<.05; OGM negative r=.14, p<.05; OGM positive r=.07, p=.29). The covariates of age, IQ and gender were associated with recent negative life events (age: r=.13, p<.05; IQ: r=-.12, p<.05; gender: r=.17, p<.01). Recent negative life events were significantly associated with adolescent-rated peer relationships (r=.29, p<.01), parent-rated peer relationships (r=.18, p<.01), resilience to mood symptoms at follow up (r=.30, p<.01) and resilience to behavioural symptoms at follow up (r=.29, p<.01). Baseline parent depression severity was not associated with any of the OGM variables at baseline (OGM total r=-.04, p=.55; OGM negative r=-.02, p=.82; OGM positive r=-.05, p=.45). Thus, a follow up regression analysis aimed at examining environmental risk factors as predictors of OGM variables at baseline while controlling the covariates of age, IQ and gender was conducted only for recent negative life events.

Table 3.1. Correlations between study variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1.B overgeneral total																	
2.B overgeneral negative	.82**																
3.B overgeneral positive	.82**	.34**															
4.B Recent negative life events	.13*	.14*	.07														
5.B parent symptom severity	-.04	-.02	-.05	.22**													
6.B adolescent rated quality of peer relationships	.12a	.12a	.07	.27**	.12*												
7.B parent rated quality of peer relationships	.14*	.11	.12a	.24**	.17**	.44**											
8.FU adolescent rated quality of peer relationships	.23**	.24**	.14*	.29**	.11a	.69**	.45**										
9.FU parent rated quality of peer relationships	.21**	.16*	.18**	.18**	.12a	.42**	.72**	.47**									
10.B resilience to mood symptoms	.03	.11a	-.07	.38**	.18**	.32**	.18**	.27**	.13a								
11.FU resilience to mood symptoms	.11	.18**	-.02	.30**	.11	.17*	.13a	.28**	.14*	.55**							
12.B resilience to behavioral symptoms	.01	.09	-.09	.30**	.09	.20**	.06	.20**	-.04	.39**	.31**						
13.FU resilience to behavioral symptoms	.005	.10	-.09	.29**	.05	.26**	.03	.26**	.002	.32**	.36**	.72**					
14.B mood symptoms	.07	.10	.01	.41**	.24**	.30**	.24**	.26**	.16**	.92**	.54**	.36**	.30**				
15.Age	-.10	-.06	-.09	.13*	.13*	-.09	-.11a	-.01	-.13*	.24**	.17**	.13*	-.01	.19**			
16.IQ	.06	.07	.03	-.12*	-.12**	.01	.003	-.08	-.02	-.12a	-.07	-.14*	-.19**	-.16**	-.16**		
17.Gender	.004	-.002	.004	.17**	.17**	-.04	.03	.07	.03	.12a	.15*	-.05	.00	.12*	.09	.10a	

Note: B= Baseline; FU=Follow up; ap<.1, *p<.05, **p<.01

3.3.2. OGM as a predictor of adolescent social functioning and mental health resilience

3.3.2.1. Adolescent-rated quality of peer relationships

As presented in Table 3.2. (step 3), regression analyses showed that after controlling for baseline adolescent-rated quality of peer relationships, age, gender and IQ, baseline OGM total and OGM negative were significantly associated with adolescent-rated quality of peer relationships at follow up in the expected direction (OGM total: β=.14, p<.01; OGM negative: β=.14, p<.01). In other words, greater levels of OGM were associated with higher scores on the quality of peer relationships measure (with higher scores reflecting poorer peer functioning). OGM positive at baseline was also associated with adolescent-rated peer relation at a trend level (β=.09, p=.09). This suggests that a higher retrieval of overgeneral memories, primarily in response to negative cue words, was associated over time with poorer quality of peer relationships as reported by the adolescent. Gender was also significantly associated with adolescent rated quality of peer relationships in all three analyses (OGM total r=.12, p<.05; OGM negative r=.12, p<.05; OGM positive r=.12, p<.05), indicating that females were likely to report lower quality of peer relationships compared to males.

3.3.2.2. Parent rated quality of peer relationships

As presented in Table 3.3. (step 3), after controlling for baseline parent-rated quality of peer relationships, age, gender and IQ, baseline OGM total and OGM negative were not significantly associated with parent-rated quality of peer relationships at follow up (OGM total: β=.08, p=.10; OGM negative: β=.04, p=.41). OGM positive at baseline was associated with parent-rated quality of peer relationships at trend level (β=.09, p=.07). Age was associated with parent-rated quality of peer relationships at trend level in all three analyses (OGM total: β=-.08, p=.08; OGM negative: β=-.09, p=.06, OGM positive: β=-.08, p=.08).

3.3.2.3. Adolescent resilience to mood symptoms

As presented in Table 3.4. (step 3), after controlling for baseline resilience to mood symptoms, age, gender and IQ, only baseline OGM negative was significantly associated with resilience to mood symptoms at follow up in the expected direction (β=.13, p<.05). Baseline OGM total was associated with resilience to mood symptoms at follow up at trend level (β=.10,p=.09) and OGM positive was not significantly associated with resilience to mood symptoms (β=.02,p=.67). This suggests that greater retrieval of overgeneral

memories in response to negative cue words was associated with lower levels of resilience to mood symptoms. Gender was positively associated with follow up resilience to mood symptoms in all three analyses (OGM total: $\beta=.12$, $p<.05$; OGM negative: $\beta=.12$, $p<.05$, OGM positive: $\beta=.12$, $p<.05$), indicating that females had poorer resilience to mood symptoms compared to males.

Table 3.2. OGM variables predicting adolescent-rated quality of peer relations

OGM measure	OGM total			OGM negative			OGM positive		
	B (S.E.)	**β**	**p**	**B (S.E.)**	**β**	**p**	**B (S.E.)**	**β**	**p**
Step 1									
OGM measure	1.37 (.43)	.22	<.01	1.38 (.44)	.21	<.01	.93 (.43)	.15	<.05
Step 2									
OGM measure	.85 (.32)	.14	<.01	.88 (.33)	.14	<.01	.55 (.32)	.09	.09
Baseline adolescent-rated quality of peer relationships	4.46 (.34)	.66	<.001	4.47 (.34)	.66	<.001	4.52 (.35)	.67	<.001
Step 3									
OGM measure	.87 (.32)	.14	<.01	.90 (.33)	.14	<.01	.54 (.32)	.09	.09
Baseline adolescent-rated quality of peer relationships	4.50 (.34)	.66	<.001	4.51 (.34)	.67	<.001	4.56 (.34)	.67	<.001
Age	.18 (.32)	.03	.58	.16 (.32)	.03	.61	.12 (.33)	.02	.71
IQ	-.49 (.36)	-.07	.17	-.50 (.36)	-.07	.16	-.46 (.36)	-.06	.21
Gender	1.52 (.65)	.12	<.05	1.54 (.65)	.12	<.05	1.56 (.66)	.12	<.05

Table 3.3. OGM variables predicting parent-rated quality of peer relationships

OGM measure	OGM total			OGM negative			OGM positive		
	B (S.E.)	**β**	**p**	**B (S.E.)**	**β**	**p**	**B (S.E.)**	**β**	**p**
Step 1									
OGM measure	.40 (.15)	.17	<.05	.29 (.158)	.12	.06	.37 (.15)	.16	<.05
Step 2									
OGM measure	.20 (.11)	.09	.07	.11 (.11)	.05	.32	.22 (.11)	.10	<.05
Baseline parent-rated quality of peer relationships	1.72 (.11)	.71	<.001	1.73 (.11)	.72	<.001	1.72 (.11)	.72	<.001
Step 3									
OGM measure	.18 (.11)	.08	.10	.09 (.11)	.04	.41	.20 (.11)	.09	.07
Baseline parent-rated quality of peer relationships	1.70 (.11)	.71	<.001	1.72 (.11)	.71	<.001	1.71 (.11)	.71	<.001
Age	-.192 (.11)	-.08	.08	-.20 (.11)	-.09	.06	-.19 (.11)	-.08	.08
IQ	-.05 (.12)	-.02	.659	-.05 (.12)	-.02	.69	-.04 (.12)	-.02	.71
Gender	.31 (.22)	.07	.15	.31 (.22)	.07	.16	.32 (.22)	.07	.15

Table 3.4. OGM variables predicting resilience to mood symptoms

OGM measure	OGM total			OGM negative			OGM positive		
	B (S.E.)	β	p	B (S.E.)	β	p	B (S.E.)	β	p
Step 1									
OGM measure	.13 (.07)	.12	.07	.18 (.06)	.19	<.01	.00 (.07)	.00	.99
Step 2									
OGM measure	.10 (.06)	.09	.09	.12 (.05)	.12	<.05	.03 (.06)	.03	.65
Baseline resilience to mood symptoms	.53 (.06)	.54	<.001	.52 (.06)	.52	<.001	.53 (.06)	.54	<.001
Step 3									
OGM measure	.10 (.06)	.10	.09	.12 (.05)	.13	<.05	.03 (.06)	.02	.67
Baseline resilience to mood symptoms	.50 (.06)	.50	<.001	.48 (.06)	.492	<.001	.50 (.06)	.51	<.001
Age	.03 (.06)	.03	.61	.03 (.06)	.03	.58	.02 (.06)	.02	.69
IQ	-.04 (.06)	-.04	.52	-.04 (.06)	-.04	.46	-.04 (.06)	-.03	.56
Gender	.24 (.11)	.12	<.05	.24 (.11)	.12	<.05	.24 (.11)	.12	<.05

3.3.3. Environmental risk factors as predictors of adolescent OGM

3.3.3.1. Recent negative life events as a predictor of adolescent OGM

As presented in Table 3.5. (step 2), after controlling for age, gender and IQ, recent negative life events were significantly associated with baseline OGM total and OGM negative in the expected direction (OGM total β=.14 ,p<.05 ; OGM negative β=.17, p<.05), but not with OGM positive (β=.06, p=.35). This suggests that a higher rate of recent negative life events was associated with a higher retrieval of overgeneral memories at baseline, primarily in response to negative cue words.

3.3.4. Sensitivity analyses

The first sensitivity analysis tested whether the observed effect of OGM on social functioning was due to an effect of mood symptoms on social functioning. As presented in Appendix 3.4. (Step 4), after controlling for OGM variables, baseline adolescent-rated quality of peer relationships and the covariates of age, IQ and gender, baseline mood symptoms weren't significantly associated with follow up adolescent-rated quality of peer relationships in any of the analyses (OGM total: β=.004, p=.94; OGM negative: β=-.003, p=.96; OGM positive: β=.01, p=.82). Similarly, as presented in Appendix 3.5.(Step 4), after accounting for OGM variables, baseline parent-rated quality of peer relationships and the aforementioned covariates, baseline mood symptoms were also not significantly associated with follow up parent-rated quality of peer relationships in any of the analyses (OGM total: β=.02, p=.66; OGM negative: β=.02,p=.64; OGM positive: β=.03,p=.56).

The second sensitivity analysis assessed whether the observed effect of negative life events on OGM was due to an effect of mood symptoms on OGM, given the known association between mood symptoms and negative life events (Bolger et al., 1989; Hammen, 2005). As presented in Appendix 3.6. (step 4), after accounting for OGM variables and the covariates, baseline mood symptoms were not significantly associated with baseline OGM measures in any of the analyses (OGM total: β=.10, p=.18; OGM negative: β=.10, p=.16; OGM positive: β=.06, p=.45). This illustrates that the observed association between negative life events and OGM was unlikely to be driven by an association with mood symptoms. The covariate of baseline mood symptoms was therefore not explored further in the mediation analyses.

3.3.5. Mediation analyses

Following results of the previous analyses, mediation analyses were conducted for recent negative life events as an environmental risk factor, and for adolescent-rated quality of peer relationships and adolescent resilience to mood symptoms as outcome measures. OGM negative was examined as a mediator because among OGM variables, OGM negative was the only variable that was consistently associated with recent negative life events, as well as with resilience to mood symptoms and adolescent rated quality of peer relationships. Furthermore, the covariate of gender was also included in the mediation analyses because it emerged as being significantly associated with both resilience to mood symptoms and adolescent rated quality of peer relationships in previous analyses.

3.3.5.1. OGM negative as a mediator of the association between recent negative life events and adolescent rated quality of peer relationships

As noted, recent negative life events at baseline were found to be significantly associated with adolescent-rated quality of peer relationships at follow up ($r=.29$, $p<.01$; Table 3.1.). OGM negative at baseline (i.e. the hypothesised mediator) was significantly associated with both recent negative life events at baseline ($\beta=.17$, $p<.05$; Table 3.5.) and adolescent rated quality of peer relationships at follow up ($\beta=.14$, $p<.01$; Table 3.2.).

An analysis of the indirect effect (Figure 3.1.A), while adjusting for gender, revealed a significant indirect effect between recent negative life events and adolescent-rated quality of peer relationships via OGM negative (standardised indirect effect= .03; standardised 95% bias-corrected bootstrapped C.I.=.001, .09). Thus, OGM negative partially mediated the association between negative life events and adolescent-rated quality of peer relationships.

3.3.5.2. OGM negative as a mediator of the association between recent negative life events and resilience to mood symptoms

As noted, recent negative life events at baseline were found to be significantly associated with adolescent resilience to mood symptoms at follow up ($r=.30$, $p<.01$; Table 3.1.)**.** OGM negative at baseline (i.e. the hypothesised mediator) was significantly associated with both recent negative life events at baseline ($\beta=.17$, $p<.05$; Table 3.5.) and resilience to mood symptoms at follow up ($\beta=.12$, $p<.05$; Table 3.4.).

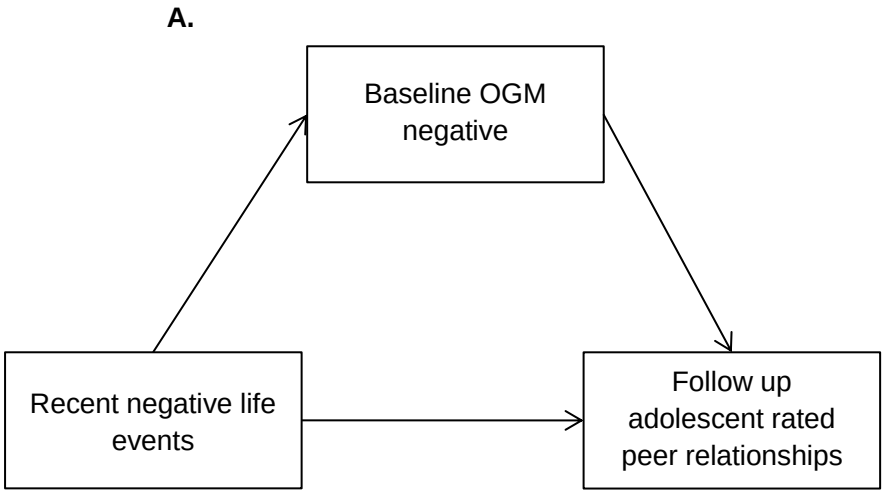
An analysis of the indirect effect (Figure 3.1.B), while adjusting for gender, found no evidence for a significant indirect effect between recent negative life events at baseline

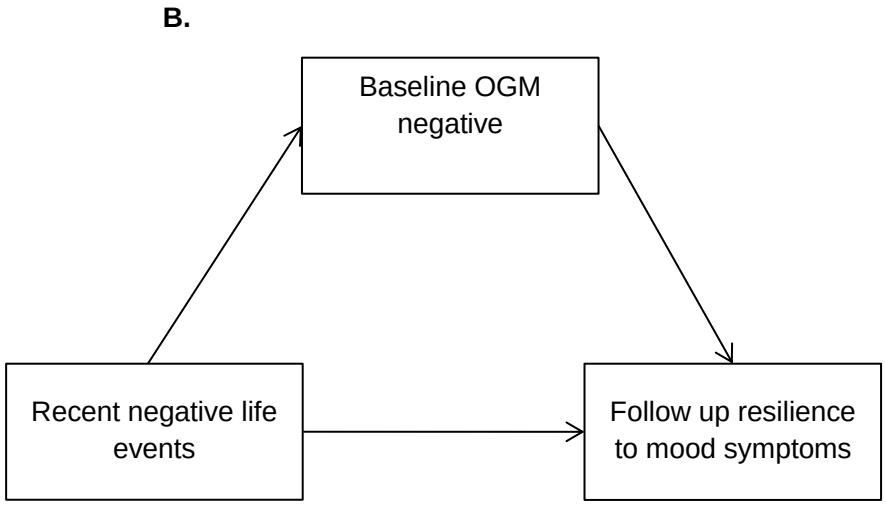
and adolescent resilience to mood symptoms at follow up (standardized indirect effect= .01; standardized 95% bias-corrected bootstrapped C.I.=-.001,.05). Thus, there was no evidence for mediation.

Table 3.5. Recent negative life events predicting baseline OGM variables

Outcome variable/ predictors	OGM total			OGM negative			OGM positive		
	B (S.E.)	β	p	B (S.E.)	β	p	B (S.E.)	β	p
Step 1									
Recent negative life events	.24 (.13)	.12	.06	.18 (.08)	.15	<.05	.06 (.08)	.05	.44
Step 2									
Recent negative life events	.27 (.13)	.14	<.05	.19 (.08)	.17	<.05	.07 (.08)	.06	.35
Age	-.24 (.12)	-.13	.052	-.11 (.07)	-.10	.13	-.13 (.08)	-.11	.09
IQ	.13 (.13)	.06	.32	.10 (.08)	.08	.22	.04 (.08)	.03	.67
Gender	-.04 (.25)	-.01	.86	-.04 (.15)	-.02	.80	-.01 (.15)	-.003	.96

Figure 3.1. Mediation models





3.4. Discussion

The present study sought to examine the following questions: 1) Is OGM associated over time with indices of mental health resilience and social functioning? 2) Is OGM specifically related to resilience to mood symptoms, or is it associated with resilience to behavioural symptoms as well? 3) Are environmental risk factors such as parental current depression severity and recent negative life events associated with OGM? 4) Does OGM serve as a mediator of the associations between environmental risk factors and adolescent mental health resilience/social functioning? These questions were examined in a longitudinal design in a high-risk sample of adolescent offspring of parents with a history of MDD.

The findings of this study suggest that retrieval of overgeneral memories, especially in response to negative cue words, was associated with adolescent rated quality of peer relationships and resilience to mood symptoms over time. No evidence was found for an association between OGM and resilience to behavioural symptoms over time.
It has been suggested that the ability to retrieve specific details of autobiographical memory is important for affect regulation, adaptive functioning, for remaining oriented to personal goals and for generating solutions to problems (Williams et al., 2007). Thus, difficulties in retrieving specific autobiographical memories may increase vulnerability for mental health disorders and impair adaptive functioning. The findings of this study are consistent with this notion and demonstrate that those who retrieved fewer overgeneral memories in response to negative cues showed better subsequent resilience to mood symptoms and had better quality of peer relationships compared to those who retrieved a higher number of overgeneral memories in response to negative cue words. The findings also suggest that OGM may be a cognitive mechanism that is specific to resilience to mood symptoms and may not affect resilience to behavioural symptoms. This is consistent with the theoretical account of OGM by Williams (2007) which suggests that OGM may be specifically associated with psychopathology of depression and PTSD. It is also consistent with the finding of Rawal & Rice (2012), which indicated the OGM in response to negative cues predicted new onset depressive disorder but not new onset externalising disorders (oppositional-defiant disorder, conduct disorder or attention deficit/hyperactivity disorder) in high-risk adolescents.

Nevertheless, the finding that OGM did not predict resilience to behavioural symptoms may be surprising, in light of the association found between OGM and executive functioning (Dalgleish et al., 2007; Valentino, Bridgett, Hayden, & Nuttall, 2012; Williams et al., 2007) and the association found in previous studies between executive functioning, specifically behavioural inhibition, and behavioural problems (Burke, Loeber, & Birmaher, 2002; Moffitt, 1993). However, OGM may involve a specific pattern of executive functioning difficulties in the context of memory retrieval that may be specifically associated with mood symptoms. Thus, executive functioning difficulties such as impaired inhibition of irrelevant material, reduced working memory capacities and diminished initiative in performance of cognitive processes have been found to be associated with mood problems and are also suggested to be important for specificity of retrieval autobiographical memory (Gotlib & Joormann, 2010; Harvey et al., 2004; Hertel & Hardin, 1990; Williams et al., 2007).It is plausible to assume that different patterns of executive dysfunction may contribute to different clinical outcomes. Furthermore, although executive functioning difficulties are suggested to be an important factor involved in OGM, OGM is also thought to involve other processes such as avoidance and rumination, specifically rumination focused on one's symptoms and negative feelings, and these may be specifically associated with mood problems (Dodge, 1993; Williams et al., 2007). Moreover, behavioural disorders are suggested to have a multi-factorial aetiology and thus impairment in executive functioning may not be present in all adolescents with behavioural symptoms (Burke et al., 2002).The lack of association between OGM and resilience to behavioural symptoms reported here may also be surprising given the association found in this study between OGM and resilience to mood symptoms and the high comorbidity between mood problems and behavioural problems in young people (Angold & Costello, 1993; Lewinsohn, Rohde, & Seeley, 1998). However, it is possible that among depressed young people, those with "pure" depression and those with comorbid behavioural problems may represent two distinct sub-groups, with different aetiology. For example, it has been found in previous studies that a younger age of onset and lower IQ is associated with the co-occurrence of depressive symptoms and behavioural symptoms (D. Brent & Weersing, 2009; Harrington, 2000; Riglin, Thapar, et al., 2016). Thus, it is possible that an association between OGM and resilience to both mood symptoms and behavioural symptoms might be observed in children or young adolescents because of the greater likelihood of a profile of depressive symptoms in which behavioural symptoms are also present. The examination of an association between OGM and behavioural symptoms

should be considered in future studies as it may be beneficial to explore such an association in other samples of young people.

As noted, retrieval of autobiographical memory is thought to rely on executive resources (Dalgleish et al., 2007; Williams et al., 2007). Thus, the finding of this study with regard to the association between OGM and resilience to mood symptoms is consistent with the findings of chapter 2, which showed that adolescents with better executive functions had fewer depressive symptoms when exposed to a current parental depressive episode. Thus, as specificity of autobiographical memory involves cognitive control in the context of memory retrieval, this finding gives additional support to the hypothesised role of cognitive control in emotion regulation.

These findings may also shed light on the pathways through which OGM may increase vulnerability for depression. Social functioning is an important part of well-being and adaptive functioning, and it has been shown that experiencing difficulties in the social arena increases risk for depression over time (Cacioppo, Hughes, Waite, Hawkley, & Thisted, 2006; Santini et al., 2015; Teo et al., 2013). Thus, it is plausible to hypothesise that if OGM predicts poor social functioning, this may be one of the pathways through which it may influence mental health resilience and confer risk for depression. However, it is important to mention that this hypothesis was not directly tested in this study; thus additional longitudinal studies are needed to determine whether social functioning may mediate the association between OGM and resilience to mood symptoms.

This study found more consistent effects for overgeneral memory for negative cues on quality of peer relationships and resilience to mood symptoms than for overgeneral memory to positive cues. As mentioned, previous studies that have examined OGM in currently depressed adults or explored OGM as a risk marker for depression in children and adolescents have yielded mixed results with regard to cue word valence effects (Hipwell et al., 2011; Rawal & Rice, 2012; Valentino, 2011; Williams et al., 2007; Woody et al., 2015). However, the findings of this study are consistent with those of Woody et al. (2015) and Rawal & Rice (2012), both of which have examined OGM as a risk marker for depression in young people. It has been hypothesised that overgeneral autobiographical memory develops initially as an avoidant coping mechanism for negative material which, with time, can generalize to other more positive material (Dalgleish et al., 2001; Raes et al., 2003; Williams et al., 2007). This may serve as a possible explanation for the valence effects shown in this sample. However, it is important to note that the valence of the cue words does not necessarily match the valence of the memory retrieved (Young, Erickson,

& Drevets, 2012). Thus, future studies should examine how the valence of the memories retrieved, and not only the valence of the cue word, is associated with mental health resilience and social functioning.

It is worth noting that no significant associations between OGM and parent-rated quality of peer relationships were found in this study. This may be related to the use of a modified, shorter questionnaire for the parent report (3 items), meaning that the parents' measure might not have indexed social functioning as effectively as the adolescent measure (10 items). Moreover, in this study, adolescent and parent reports for quality of peer relationships were only moderately correlated (baseline: $r=.44$, $p<.01$, follow-up: $r=.47$, $p<.01$; Table 3.1.). Discrepancies between child and parent report are often observed, with previous studies reporting low to moderate agreement between parents and children/adolescents on questionnaires assessing quality of life, emotional problems, and behaviour (Achenbach, McConaughy, & Howell, 1987; Klassen, Miller, & Fine, 2006; Moretti, Fine, Haley, & Marriage, 1985; Van der Meer, Dixon, & Rose, 2008).

Findings of this study also suggested that recent negative life events were associated with a greater retrieval of overgeneral memories for negative cue words. This extends previous research by showing that not only "traumatic" events but also negative life events that are prevalent among the general population may be associated with the development of OGM. The results showed no evidence for an association between current parental depression severity and offspring OGM. This finding may be contrasted with the study of Woody et al. (2015) which found that children of mothers with a history of depression retrieved fewer specific memories in response to negative cue words compared to children of healthy mothers. It is important to note that the current study did not use a case-control design, but rather examined how current parental depression severity was associated with OGM within a high-risk sample. Therefore, a possible explanation for these findings is that the heterogeneity of parental depression severity in a high-risk sample may not be substantial enough for an effect on offspring OGM to be detected (although there is a range of symptomathology in the sample; Mars et al., 2015). It is also not possible to rule out the possibility that prior parental depressive episodes may have had earlier effects on autobiographical memory development in the child. Additional studies are needed to examine how indices of parental depression may affect the development of offspring's OGM.

Finally, it was found that overgeneral autobiographical memory retrieved in response to negative cue words mediated the association between recent negative life

events and adolescent-rated quality of peer relationships. This finding highlights a possible mechanism through which negative life events might increase risk for social impairment. Various causal models of depression posit that environmental risk factors may influence proximal mechanisms, which in turn may increase vulnerability for depression (Thapar et al., 2012). This finding provides preliminary support for a similar process for social functioning and highlights OGM as a possible pathway through which negative life events may affect social functioning. Social functioning in turn may have adverse consequences on vulnerability for depression, especially in high risk individuals (Baumeister & Leary, 1995; Garber, 2006; Hammen, Shih, Altman, & Brennan, 2003).

The age range was wide in this sample (10-18). It has been suggested that autobiographical memory continues to develop throughout adolescence (Fivush, 2011) and therefore the effect of OGM on mental health resilience and social functioning may differ for younger and older adolescents. It is also possible that age can affect performance on the AMT task. For example, it is possible that the younger participants may find it harder to retrieve memories in response to the abstract cue words or that their understanding of the cue words may be more limited. In my analyses, I controlled for age but did not examine interactions of age and OGM, meaning whether the effect of OGM on mental health resilience and social functioning may be moderated by age. I also did not examine the effect of interactions of environmental risk factors (recent negative life events/parent current depression severity) and age on OGM. It is possible that the effect of environmental risk factors on OGM may be moderated by age. For example, negative life events, specifically social stressors and behaviour-dependent life events, are more common in adolescence than in childhood (Davey et al., 2008; Rice, Harold, & Thapar, 2003; Rudolph, 2002; Silk et al., 2012). Thus, the association between negative life events and OGM may be stronger for the older participants compared to the younger participants in the sample. These questions should be investigated in future studies.

The present study is the first to examine OGM as a predictor of mental health resilience and everyday social functioning. The findings suggest that OGM affects resilience to mood symptoms and social functioning and thus may be an important cognitive mechanism to target in therapeutic and preventive interventions. Consistent with this, initial trials targeting the improvement of memory specificity in depressed adults and adolescents show promise (Dalgleish & Werner-Seidler, 2014; Neshat-Doost et al., 2012; Raes, Williams, & Hermans, 2009).

CHAPTER 4: Examining the association between familial risk for depression and reward learning in a social context in young adults

Depressed individuals show difficulties in learning from reinforcement and modulating behaviour as a function of reward. Recent evidence suggests that reward learning disruptions may also be present in individuals at high familial risk of depression. These difficulties may underlie some of main functional impairments exhibited in depressed and high risk individuals, and in particular, social impairments. However, previous research has not employed social paradigms to explore reward learning in individuals at high risk of depression, nor has it examined whether reward learning is associated with social functioning in this group. This study examined whether familial risk for depression is associated with reward learning in a social context and whether variation in anhedonia and depressive symptoms may explain heterogeneity in reward learning within high risk individuals. Furthermore, this study examined whether difficulties in reward learning that are associated with familial risk, anhedonia or depressive symptoms are also associated with adaptive social functioning. Reward learning was assessed as the ability to differentiate between social partners with different tendencies for reciprocity (fair, neutral, unfair) in the context a of a multi-round trust game. Familial risk was assessed as number of depressed parents (range 0-2). It was hypothesised that higher familial risk would be associated with reduced differentiation between the different players over time and that this may be driven by reduced learning rate particularly for the fair (rewarding) player. Results of the study indicated that familial risk for depression was associated with the degree of differentiation between social partners with varying propensities for social reciprocity. Participants at higher familial risk made a greater differentiation between the fair and neutral social players, and a smaller differentiation between neutral and unfair players compared to those at lower familial risk. Findings also indicated that higher anhedonia attenuated the differentiation between the fair and neutral player and was also associated with a slower learning rate for the fair player only. Finally, findings suggested that the interaction of familial risk and learning rate for the fair player was associated with social functioning such that higher familial risk was associated with lower social functioning only in those with a lower learning rate for the fair player. This study provided evidence of an association between familial risk and anhedonia and reward learning in a social context. Furthermore, this study suggests that difficulties in reward learning in a social context may increase social impairment in those at high familial risk.

4.1. Introduction

Depression is a common, debilitating condition and is a leading cause of burden and disability worldwide (Üstün et al., 2004). Lifetime prevalence rates peaks in early adulthood, with depression affecting 15-20% of the population at that time (Birmaher et al., 1996; Hankin et al., 1998; Lewinsohn et al., 1998; Stallard et al., 2012; Thapar et al., 2012). Depression is often chronic and recurrent (Kessler & Walters, 1998), particularly when onset is early. A chronic course with early onset is more likely in those with a family history of depression (Eaton et al., 2008; Klein et al., 1999; Levinson, 2006; Wickramaratne & Weissman, 1998).

Depressed individuals often experience difficulties in the social arena, and social maladjustment is a major contributor to the substantial societal burden caused by depression. For example, depressed individuals show impairment in the work place, experience persistent difficulties in close relationships, and have higher rates of marital discord and divorce (Barnett & Gotlib, 1988; Forkmann et al., 2014; Tweed, 1993; Wang et al., 2014; Weissman, Paykel, Siegel, & Klerman, 1971). Evidence also points to the importance of social factors in explaining vulnerability to depression. For instance, experiencing social difficulties may also be associated with the development of depressive symptoms and disorder (Baumeister & Leary, 1995; Santini et al., 2015; Teo et al., 2013). Thus, studies indicate that higher social support, large and diverse social networks, and good quality of social relationships protect against future depression (Santini et al., 2015; Teo et al., 2013), whereas negative social interactions and loneliness are associated with higher rates of depressive symptoms and disorder (Cacioppo et al., 2006; Teo et al., 2013). Finally, a group at high familial risk for depression - the offspring of depressed parents - have been found to experience poorer social functioning compared to offspring of healthy parents (Weissman et al., 1997; Weissman et al., 2006), regardless of their own psychopathology (Lewinsohn, Olino, & Klein, 2005; Weissman et al., 2006). This suggests that social functioning may be associated with depression, given that rates of depression are increased in the offspring of depressed parents.

The detrimental effects of social impairment on wellbeing and vulnerability for depression highlight the need to explore factors that may underlie social impairment, especially in those at high risk of depression. Previous studies have shown that depressed individuals have impairments in reward processing, show blunted reward sensitivity and difficulties in learning from reinforcement and modulating behaviour as a function of reward (Cella, Dymond, & Cooper, 2010; Henriques & Davidson, 2000;

Pizzagalli et al., 2005). Reward processing difficulties also seem to be present in individuals at high familial risk of depression (Gotlib et al., 2010; Liu et al., 2016; Rawal et al., 2013). It is possible that such alterations in reward processing may underlie some of the impairments in social functioning seen in depressed individuals and in individuals at high-risk of depression(Rawal et al., 2013). For instance, Rawal et al. (2013) found that measures of reward seeking in a gambling task were positively associated with indices of social functioning such as quality of peer relations, humour and engagement in extra-curricular activities in a sample of adolescents at familial risk for depression. Learning from reinforcement and rewards is suggested to be important for social functioning as it allows individuals to adapt to changing social situations and modulate behaviour according to responses from the environment, which is essential for establishing good quality, meaningful social relationships (Heerey, 2014). For example, from a very young age, individuals learn associations between their behaviour and social cues like facial expressions, such that a behaviour that elicits a smile is more likely to be repeated whereas a behaviour that elicits an angry facial expression is more likely to be altered (Ekman, 1993; Johnston, Miles, & Macrae, 2010; Kringelbach & Rolls, 2003; Tarabulsy, Tessier, & Kappas, 1996). Thus it is possible that difficulties in learning from reinforcement may impede adaptive social decision making and interfere with goal directed behaviour aimed at increasing the likelihood of positive and rewarding outcomes (Heerey, 2014; Pizzagalli et al., 2008). In line with this notion, a recent study by Heerey (2014) found that the ability to learn contingencies between social cues such as a genuine or polite smile and feedback on an experimental task was associated with self-reported social ability in adults.

Behavioural theories of depression propose an important role for diminished participation in enjoyable and rewarding activities in the aetiology and maintenance of depression (Joiner, Lewinsohn, & Seeley, 2002; Lewinsohn, 1985; Lewinsohn & Graf, 1973). Consistent with this, recent studies show that alterations in reward processing such as reduced reward seeking behaviour - especially under conditions of high probability for gain - is associated with lower social functioning and increased rates of subsequent depression (Forbes et al., 2007; Rawal et al., 2013). Thus, it is possible that social functioning may serve as a mechanism through which reward impairments increase risk for depression. Indeed, social functioning is strongly associated with wellbeing and positive emotions (Baumeister & Leary, 1995). Those with impaired reward learning may experience difficulties in social functioning due to problems with learning contingencies

between behaviour and social rewards, identifying opportunities to receive rewards, and modulating behaviour in social situations in order to increase the likelihood of receiving social rewards (Heerey, 2014). This may contribute to diminished pleasure and experience of positive emotions in the social arena and may increase risk for depression (Cacioppo et al., 2006; Santini et al., 2015; Teo et al., 2013). Exploring the association between reward learning and social functioning in high risk groups provides the opportunity to explore this possible mechanism through which reward learning alterations may confer risk for depression. However, previous research has not explored how reward learning relates to social functioning in individuals at high risk of depression. Therefore, in the current study I sought to examine how familial risk for depression relates to reward learning, and how reward learning in turn is associated with social functioning.

While several studies have examined the presence of reward learning impairments in the offspring of depressed parents, they have yielded mixed results. Behavioural studies that have examined reward learning in individuals at familial risk suggest that heterogeneity in reward processing in high- risk individuals may be associated with the severity of sub-clinical depressive symptoms or anhedonia. Two recent studies examined reward learning as measured by a reward bias (i.e. the tendency to show a behavioural preference for the more often rewarded stimulus on a visual discrimination task demonstrated in healthy individuals; Chapter 1, Table 1.2.) in children of depressed parents and children of healthy parents (Luking et al., 2015; Morris, Bylsma, Yaroslavsky, Kovacs, & Rottenberg, 2015). Luking et al. (2015) found that familial risk was not significantly associated with reduced reward bias. However, reduced reward bias was found to be associated with levels of anhedonia in the low-risk group and with negative mood in the high-risk group. Morris et al. (2015) reported that reward bias was not associated with familial risk, but with the presence of anxiety and depressive disorders in the child. Using a similar task which assessed reward bias, Liu et al. (2016) examined reward learning in adult first degree relatives of patients with MDD and in healthy controls with no family history of depression. Results of this study suggested a reduced tendency to establish a reward bias in first degree relatives with high levels of subclinical depressive symptoms compared to controls and relatives with low levels of subclinical depressive symptoms. Furthermore, within the group of first degree relatives with high depressive symptoms, levels of anhedonia were negatively associated to reward bias. As previously mentioned (Chapter 1), reward learning and hedonic capacity (i.e. the experience of pleasure while receiving a reward) are suggested to represent two distinguishable aspects

of reward processing (Berridge & Robinson, 1998, 2003; Dichter, 2010; Treadway & Zald, 2011). However, these two components might be associated, but previous studies in high risk and healthy individuals have not clarified whether those who experience anhedonia also demonstrate difficulties in reward learning.

Taken together, this evidence suggests that further studies are needed to explore the presence of reward learning impairments in those at high risk of depression, and to examine whether variation in anhedonia and depressive symptoms may explain heterogeneity within this group.

Nearly all studies of reward processing in depressed and high risk individuals have used gambling tasks or probabilistic visual discrimination tasks to assess reward sensitivity and reward learning. However, these tasks may not tap reward learning in more natural contexts, such as the social arena. Thus, there has been a call for more studies to examine reward processing in depression using social paradigms (Forbes, 2009; Mellick et al., 2015). In order to address this gap in the literature, in the present study I employed a trust game paradigm adopted from Phan et al. (2010) which entails reward learning in a social context. In this task, participants play a multi-round trust game with three players who have different tendencies for reciprocity (fair, neutral, unfair). Participants are required to learn the players' tendencies to reciprocate in order to maximize their personal gain. Thus, this task assesses reward learning in the context of social exchanges.

Previous studies that have examined reward seeking in tasks where the probability of gain varies across the task have found that reduced reward seeking when the likelihood of gain was high was prospectively associated with increased risk for depression in adolescents (Forbes et al., 2007; Rawal et al., 2013). Moreover, studies that have examined reward learning in high risk participants showed that anhedonia or depressive symptoms in those at high risk are associated with a reduced tendency to choose the more frequently rewarded stimuli in reward learning tasks (Liu et al., 2016; Luking et al., 2015). This evidence suggests that the detrimental effect of familial risk or the interaction of familial risk and anhedonia/depressive symptoms on reward learning may be driven by difficulty modulating behaviour in order to pursue reward in situations in which reward/gain is highly probable. Thus, it is plausible that in a task involving reward learning in a context of social exchanges, in which different partners have different tendencies for reciprocity (Phan et al., 2010) the effect of the familial risk, anhedonia and depressive symptoms will be most prominent in learning the tendency of the more frequently rewarding partner (i.e. the fair player; Phan et al., 2010).

In this study I aimed to examine how reward learning in a social context is associated with familial risk for depression (assessed by a history of depression in one or both parents), anhedonia, depressive symptoms and social functioning in a sample of young adults. Reward learning was assessed behaviourally and indexed by the extent to which participants altered their investment rate for the different players (fair, neutral, unfair) in the trust game task over time. I aimed to examine the following questions and made the following predictions:

1) Are familial risk, anhedonia and depressive symptoms associated with reward learning in a social context? Focusing on differential reward learning assessed by investment behaviour for the fair, neutral and unfair players in the trust game, I hypothesised that higher familial risk would be associated with reduced differentiation between the different player types over time and that this may be driven by reduced learning rate (change in investment behaviour over time) particularly for the fair (rewarding) player. Following previous studies of reward learning in high risk individuals (Liu et al., 2016; Luking et al., 2015; Morris et al., 2015), I also hypothesised that at least part of the effect of familial risk may be attributable to current symptoms of anhedonia and depression.

2) Is reward learning in a behavioural task associated with 'real-life' social functioning? Drawing on evidence indicating that depressed and high risk individuals are characterised by social impairment, I hypothesised that difficulties in reward learning that are associated with familial risk, anhedonia or current depressive symptoms would be negatively associated with adaptive social functioning.

4.2. Methods

4.2.1. Participants

Participants were recruited using an online university subject pool and advertisements in university campuses in central London, UK. Participants underwent interviews to assess familial risk of depression. For the purpose of recruitment, participants were defined as being at either low or high familial risk of depression. The inclusion criterion for low risk participants was the absence of a history of MDD in both parents. The inclusion criterion for high risk participants was the presence of a history of MDD in one or both parents. A history of MDD in the parent was established if the parent previously or currently met DSM-IV criteria for a major depressive episode as assessed by the family history method (described below) or received a diagnosis of MDD from a doctor. A

diagnosis of bipolar disorder or schizophrenia in the affected parent were exclusion criteria. Four participants who reported having a parent with a history of depression were excluded due to difficulties in recalling details about parental depression or not meeting the inclusion criteria for parental depression. The final sample included 66 subjects: 29 participants whose parents had no history of MDD and 37 participants whose parents had a history of depression. Among the high risk participants, 30 had one parent with a history of MDD and 7 had two parents with a history of MDD. Participants were not excluded based on their own current or past psychopathology. As familial risk for depression is associated significantly higher rates of MDD (Beardselee, Versage, & Giadstone, 1998), we assessed current depressive symptoms and the presence of a past MDD episode and included these as covariates in all analyses that followed, in order to examine whether any observed effect of familial risk was attributable to current or past depressive psychopathology.

4.2.2. Measures

4.2.2.1. Familial risk assessment

Assessments of parental psychopathology were obtained using the family history method, in which participants are interviewed regarding the presence of mental health disorders in first degree relatives (Andreasen, Endicott, Spitzer, & Winokur, 1977) which has been shown to be valid and reliable (Colvin, Richardson, Cyranowski, Youk, & Bromberger, 2014; Orvaschel, Thompson, Belanger, Prusoff, & Kidd, 1982; Weissman et al., 2000). The interview consisted of an adapted version of the FIGS (Family Interview for Genetic Studies; Maxwell, 1992). Participants were first asked a screening question regarding parental depression, followed by questions concerning age of onset, number of episodes, length of episodes and whether parental depression was diagnosed by a doctor. Participants were then interviewed with a depression symptoms checklist regarding the best recalled, worst episode of the affected parent(s). Additional information was collected regarding functional impairment, hospitalisation, the type of the treatments the parent received, and whether or not the parent received any other mental health diagnoses. The number of parents with a history of depression (range 0-2) was used as the measure of familial risk in this study (Milne et al., 2008).

4.2.2.2. Participant psychopathology

Previous major depressive episode

The presence of past MDD episodes was assessed using the MINI International Neuropsychiatric Interview 6.0.0 (Sheehan & Lecrubier, 2010) which assesses the presence of DSM-IV psychiatric disorders. Trained researchers administered the interview. Inter-rater agreement for the presence of a past depressive episode was excellent: $k=1$, $p<.01$.

Current depressive symptoms

Depressive symptoms were assessed using the PHQ-9 (Kroencke, Spitzer, & Williams, 2001) which is a 9-item self-report questionnaire that assesses the presence and severity of DSM-IV depressive symptoms over the past 2 weeks. Symptoms are rated on a 4 point scale from 0 (“not at all”) to 3 (“nearly every day”). The total score was used as a measure of current depressive symptoms.

4.2.2.3. Anhedonia

Anhedonia was assessed using the SHAPS (The Snaith-Hamilton Pleasure Scale; Snaith et al., 1995), a well validated 14-item questionnaire aimed at assessing the hedonic value of different pleasurable experiences. The questionnaire covers four areas of hedonic experiences: interests/hobbies, social interaction, sensory experience, and food/drink. Participants are requested to rate how much they agree with each statement regarding the hedonic value of an enjoyable experience. Responses were scored on a four point scale ("definitely agree" to "strongly disagree"). The total sum of responses was used, where higher scores reflect higher levels of anhedonia.

4.2.2.4. Reward learning

Reward learning was examined using a trust game paradigm adopted from Phan et al. (2010). In this task participants play a computerised multi-round trust game. Participants play the role of an investor who must decide whether to keep or to invest tokens in a player (‘trustee’). If the participant chooses to keep the tokens, the tokens are evenly split between the participant and the player they are playing with. If the participant chooses to invest the tokens, the number of tokens is doubled and the player (‘trustee’) either "reciprocates" by sending back half the tokens to the participant, or keeps all the

tokens and sends nothing back to the participant (See Figure 4.1.). Participants play the game with three different "simulated" human players, who have different tendencies for reciprocity: a fair player (reciprocates in 75% of trials), a neutral player (reciprocates in 50% of trials) and an unfair player (reciprocates in 25% of trials). The tendencies of the players to reciprocate are not known to participants. As in the original version of the task (Phan et al., 2010) an additional computer-player is included as a control and participants are informed that the computer will reciprocate 50% of the time. Participants were told that each human player's performance is derived from a recording of the performance of another participant who previously participated in this study, but were not told the tendency of each human player to reciprocate. This task involves the participant learning each player's tendency for reciprocity and investing accordingly to maximize personal gain. The task includes 4 blocks of 20 trials each (each block included 5 trials with each player: fair, neutral, unfair, computer). The human players were represented by photos of real people, with the face in each photo obscured by a coloured oval. Participants could differentiate between the players by the colour of the oval (See Figure 4.1.). The pictures of players and oval colours were counterbalanced between participants.

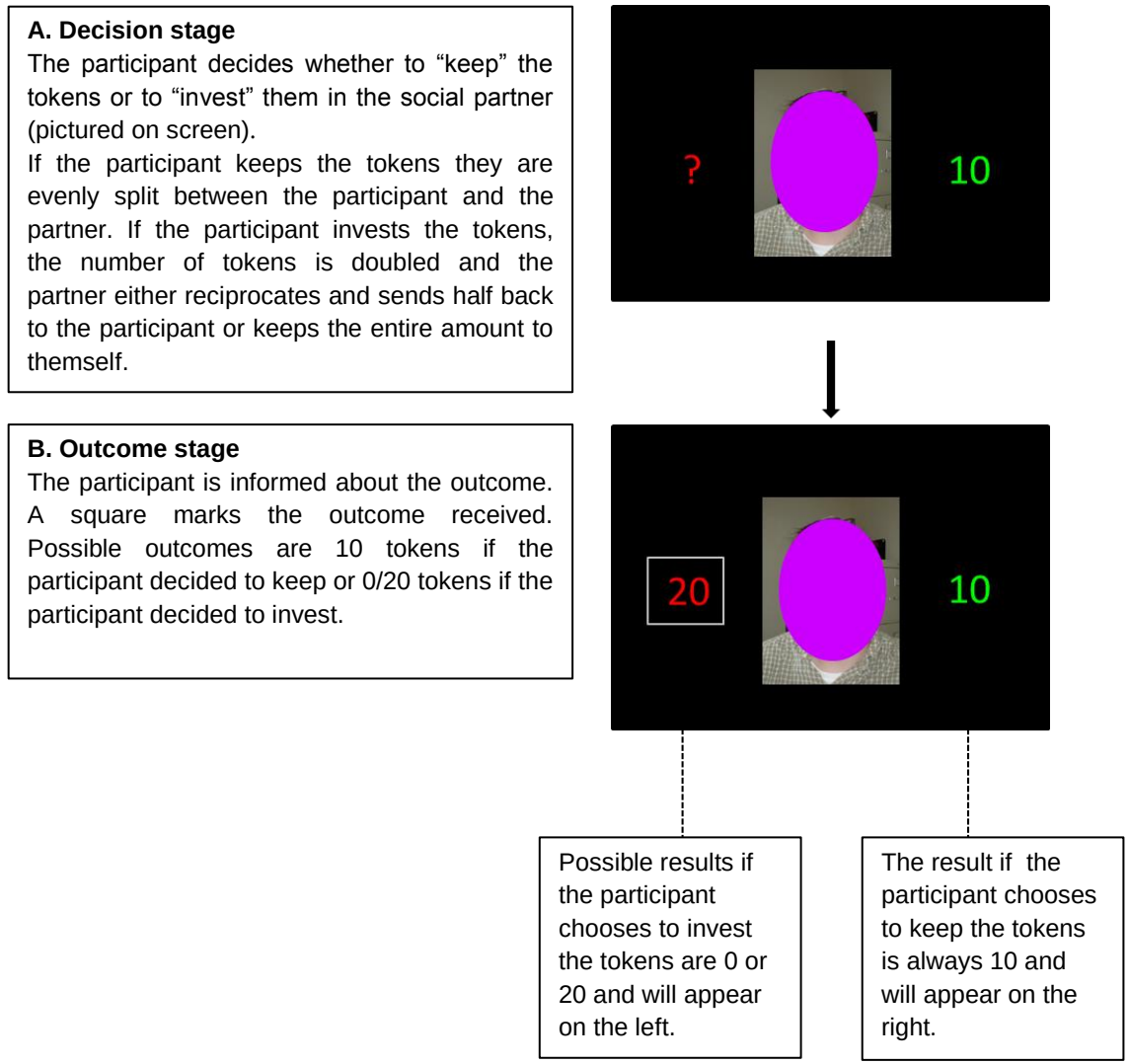
Following completion of the task, participants were presented with the images of each human player and asked: "How often did this player share the tokens with you? Response options were: more than half the time (1); half of the time (2) less than half of the time (3). They were also asked "how trustworthy they would rate the player on a scale from 1-10?" (1-"not very trustworthy", 10-"extremly trustworthy").

For comparability with previous publications (e.g. Phan et al., 2010) the original trust game task which included 3 human players and a computer player was employed. However, as the focus of this study was reward learning, only the trials played with the human players (whose tendencies for reciprocity were unknown to participants) were retained in the analyses. The following outcome measures were calculated:

1) Investment rate per player for each block. This was calculated by defining the four original blocks as four consecutive time points, then binning the 5 trials played with each player at each of the four time points into one block per time point, and calculating a score of investment rate (% of investment choices) for each block. This outcome variable was used to test the first part of research question 1 (differentiation between the players over time).
2) The second part of research question 1 predicted that any observed effects of familial risk, depressive symptoms and anhedonia on differentiation between the

players would be primarily attributable to the learning rate for the fair player. In order to test this specifically, and as a follow-up to the primary analysis, an individual learning rate for each player was calculated for each participant by regressing the percentage of investment rate in each player on the four time points (Reeb-Sutherland, Levitt, & Fox, 2012). This variable represents the rate at which individuals changed their investment in each player over time, with positive values reflecting an increase in investment rate over time and negative values reflecting a decrease in investment rate over time.

Figure 4.1. An example of a single trial in the task



Note: The players (fair, neutral, unfair) can be differentiated by the type of picture presented and colour of the oval (brown, yellow or purple).

4.2.2.5. IQ

IQ was assessed using the matrix reasoning subset of the Wechsler Abbreviated Scale for Intelligence (WASI; Wechsler, 1999) which is commonly used to assess fluid intelligence (Horn, 1991; Zook, Davalos, DeLosh, & Davis, 2004).

4.2.2.6. Social functioning

Social functioning was assessed using the Social Adaptation Self-evaluation Scale (Bosc, Dubini, & Polin, 1997). This scale assesses social motivation, behaviour and satisfaction in areas such as work, family relationships and social relationships. The questionnaire consists of 21 items, each scored on a four point scale ("very" to "not at all"). The questionnaire has been shown to be valid and reliable and showed sensitivity to change in patients with MDD (Bosc et al., 1997). The total score served as a measure of social functioning, with higher scores representing higher functioning.

4.2.3. Procedure

Written informed consent was obtained from all participants prior to participation. Participants completed the trust game computerized task, responded to interviews assessing both parental history of depression and mental health psychopathology in the participant, undertook the matrix reasoning subset, and answered questionnaires examining current depressive symptoms, anhedonia and social functioning during a single session. Ethical approval for this study was provided by UCL research department of clinical, educational and health psychology ethics committee (ethics application 2015/528).

4.2.4. Statistical analysis

First, correlations between study variables were examined to identify potential covariates. The first part of research question 1 predicted that familial risk will be associated with reduced differentiation between the players in the trust game task. In order to explore this, a multilevel regression analysis was conducted to examine the effect of time, player type, familial risk, anhedonia, depressive symptoms and the interactions between them on investment rate. A measure of investment rate for each human player at each time point was computed for every participant as described above. This resulted in 12 blocks (3 players x 4 time points) for each individual. Thus, a multilevel regression model was employed, in which blocks were nested within individuals (Hoffman & Rovine, 2007; See Figure 4.2.). Time and player type were examined as block-level variables, and

familial risk, anhedonia and depressive symptoms were examined as individual-level variables. To examine differentiation between the players, player type was dummy coded with the neutral player as the reference category. This resulted in two dummy-coded variables: one for the fair vs. neutral player, and one for the unfair vs. neutral player. IQ, gender, age, and the presence of a past MDD episode were included as individual-level control variables. Continuous variables were mean-centred prior to the analysis. Models were estimated using maximum likelihood, and successively more complex models were fit as detailed below. The fit of each model was compared to the baseline model by examining the change in -2 log-likelihood (-2LL), a likelihood ratio test based on a chi-square distribution that is used as an index of model fit (Field, 2009). Potential predictors, random effects and covariates were added gradually in successive models. If a potential predictor, random effect or covariate did not significantly improve model fit (in terms of -2LL change for the critical degree of freedom change) it was omitted from the next model. The baseline model included only a fixed intercept. The following variables were successively included in models and fit was assessed: 1) A random intercept. 2) The fixed effects of time, player type and the interaction between time and player type. 3) Individual random effects (slopes) for player type (i.e. random effects for fair vs. neutral player and for unfair vs. neutral player). 4) The main effect of familial risk, 2-way interactions of familial risk and player type variables, familial risk and time and 3-way interactions of familial risk, player type variables and time. 5) Anhedonia, 2-way interactions of anhedonia and player type variables, anhedonia and time and 3-way interactions of anhedonia, player type variables and time. 6) A 2-way interaction of familial risk and anhedonia and 3-way interactions of familial risk, anhedonia and player type variables. 7) Current depressive symptoms and interactions of current depressive symptoms with player type, time and familial risk similar to those examined for anhedonia. 8) The covariates of age, gender, IQ, and the presence of a previous MDD episode. These covariates were also examined in all analyses that followed. Significant 2-way and 3-way interactions were plotted. Significant 3-way interactions were followed up by simple slopes analysis (Dawson, 2014).

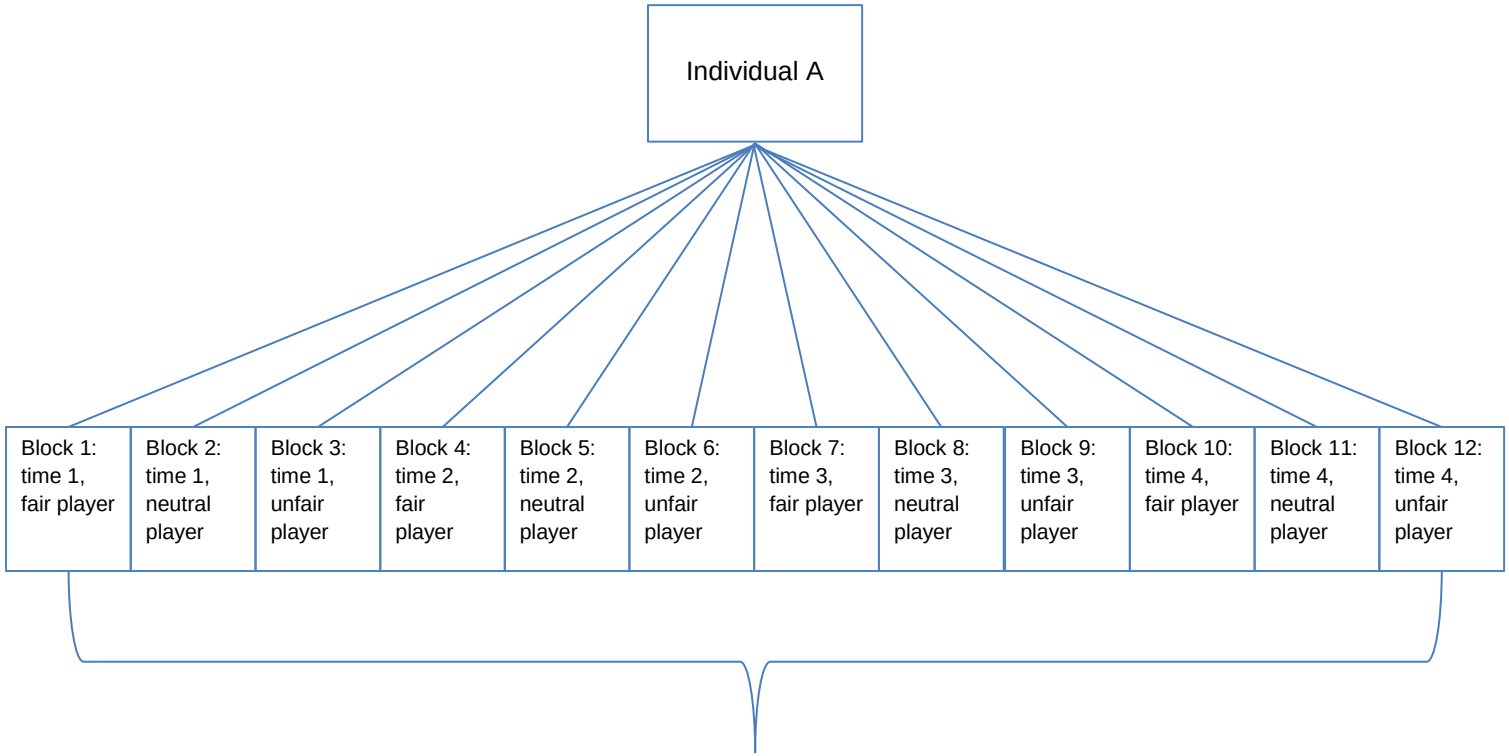
Next, I examined whether the differentiation between players and any effects of familial risk, anhedonia, and current depressive symptoms on differentiation between players found in the behavioural performance were replicated in the subjective ratings of players (i.e. identification of players' reciprocity and ratings of players' trustworthiness). First, I examined whether ratings of identification of players' reciprocity in the sample as a whole indicated that participants managed to differentiate between the players. Second, I

examined whether familial risk, anhedonia and current depressive symptoms were associated with ratings of players' trustworthiness. In order to examine these, a multilevel regression analysis was conducted for each question, in which players were nested within individuals. The analysis for identification ratings examined the effects of player type (dummy coded) on identification ratings. The analysis of ratings of trustworthiness examined, as before, the main effect of player type variables (dummy coded) and their interactions with familial risk, anhedonia, and current depressive symptoms as predictors of trustworthiness ratings.

The second part of research question 1 predicted that any effects of familial risk, anhedonia or depressive symptoms on differentiation between the players will be attributable to a reduced learning rate, particularly for the fair player. As the analysis conducted for the first part of research question 1 allowed testing for differentiation between players (by employing dummy-coded variables with the neutral player as reference category) but not for the learning rate for each player separately, to follow up the first analysis and examine the effects of familial risk, anhedonia and depressive symptoms on the learning rate for each player, separate hierarchical regression analyses were conducted, with learning rates for each player as outcome measures. In the first step, the main effects of familial risk and anhedonia were entered as predictors. In the second step the interaction between familial risk and anhedonia was examined. In subsequent steps, the main effect of current depressive symptoms, the interaction of familial risk and current depressive symptoms and covariates of past depressive episode, IQ, age and gender were examined separately. Only predictors which improved the model's fit, as assessed by change in R^2 (Field, 2009), were retained in the final model.

Research question 2 predicted that indices of reward learning showing association with familial risk, anhedonia or current depressive symptoms would also be associated with social functioning. To examine this, Pearson's correlation coefficients were first examined. Significant correlations were then followed up with a hierarchical multiple regression analysis that controlled for covariates and interactions as previously described.

Figure 4.2. Blocks nested within individuals in the main multilevel analysis



4.3. Results

4.3.1. Preliminary analysis

Correlations between study variables and descriptive statistics are presented in Table 4.1. Pearson's correlation coefficients between study variables were explored to identify important covariates for the main analyses. Familial risk was not significantly associated with age, gender or IQ, indicating that low risk and high risk participants did not differ on these variables (Table 4.1.). As would be expected, familial risk was positively associated with the presence of a past depressive episode ($r=.45$, $p<.01$) and current depressive symptoms ($r=.30$, $p<.05$). No significant association was found between familial risk and anhedonia ($r=.07$, $p=.57$) or social functioning ($r=-.10$, $p=.41$). Anhedonia was negatively associated with social functioning ($r=-.66$, $p<.01$). Current depressive symptoms were also negatively associated with social functioning ($r=-.44$, $p<.01$). Age, gender, IQ, the presence of a past depressive episode were included as control variables in all the analyses that follow, given their association with familial risk or their potential role in reward processing (Funder & Block, 1989; Lighthall et al., 2012; Van Leijenhorst, Westenberg, & Crone, 2008; Whelan & McHugh, 2009).

4.3.2. Differentiation between player types

Table 4.2. illustrates results of the final multilevel analysis of influences on the differentiation between player types, as measured by investment rate. First, the results for the full sample over time are described. Next, the roles of familial risk, anhedonia and current depressive symptoms on task performance are investigated, specifically the ability of participants to differentiate between the fair, neutral and unfair players. As described, variables were only retained in the final model if including them improved model fit. The covariates of past depressive episode, age, IQ and gender did not improve the model's fit and therefore were not retained in the final model.

4.3.2.1. Effects of player type and Time

As presented in Table 4.2., for the sample as a whole, there was a main effect of player type on investment rate, where a higher investment rate was found for the fair player compared with the neutral player ($B=11.41$, $p<.01$) and a lower rate of investment was found for the unfair player compared with the neutral player ($B=-17.04$, $p<.001$). As presented in Figure 4.3., the sample showed discrimination of the players (fair, neutral, unfair) over time as indicated by a greater increase in investment rate for the fair player

compared with the neutral player over time (interaction term of the variable fair vs. neutral player and time: B=6.24, p<.001) and a greater decrease in investment rate for the unfair player compared with the neutral player over time (interaction term of the variable unfair vs. neutral player and time: B=-4.33, p<.05).

The subjective ratings for identification of players' reciprocity also showed that in the sample as a whole, participants correctly identified the players. Thus, there was a main effect of player type on the ratings of identifications, indicating that participants were more likely to rate the fair player as sharing the tokens more than half the time compared to the neutral player (B=-0.52, p<.001) and rated the unfair player as sharing the tokens less than half the time compared to the neutral player (B=0.41, p<.01).

4.3.2.2. Familial risk

There was no main effect of the degree of familial risk on the total investment rate (B=-4.14, p=.33). However, there were significant 2-way interactions between familial risk and the two variables of player type (fair vs. neutral and unfair vs. neutral; Figure 4.4.), suggesting that participants with higher familial risk showed a greater differentiation between the fair and neutral player compared to those with lower familial risk (B=10.18, p<.05), and a smaller differentiation between the neutral and unfair player compared to those at lower familial risk (B=9.42, p<.05).

4.3.2.3. Anhedonia

There was no evidence of a main effect of anhedonia on the total investment rate (B=.65, p=.19). As presented in Figure 4.5., there was a significant 2-way interaction between anhedonia and the variable of fair vs. neutral player type, suggesting that those with higher levels of anhedonia showed a smaller differentiation between the fair and the neutral player compared to those with lower levels of anhedonia (B=-1.49, p<.05). There was a significant three-way interaction between the variable of fair vs. neutral player, time and anhedonia (B =-.57, p<.05; Figure 4.6.). A simple slopes analysis revealed that there was no significant association between time and investment rate for the neutral player for those with low (b=-2.08, z=-1.43, p=0.15) or high anhedonia (b=-0.23, z=-0.16, p=0.88). This indicates that subjects did not significantly change their investment in the neutral player over time. There was however a significant positive association between time and investment rate in the fair player for those with low anhedonia (b=7.33, z=4.38, p<.001), but not for those with high anhedonia (b=2.85, z=1.7, p=0.09). This suggests that only

those with a low level of anhedonia significantly increased the investment rate in the fair player over time and that high levels of anhedonia attenuated participants' learning rates for the fair player.

4.3.2.4. Current depressive symptoms

No significant main effect or interactions with player type, time or familial risk were observed for current depressive symptoms.

4.3.2.5. Interactions involving familial risk, anhedonia, depressive symptoms

Three way interactions that included the primary variables of interest (familial risk, anhedonia, depressive symptoms and player type variables) were examined. There was a trend level three-way interaction effect between the variable of fair vs. neutral player, familial risk and anhedonia (B= 1.59, p=.07) that was not investigated further as it was not significant at the p<.05 level. All other three way interactions did not improve the model's fit and therefore were not retained in the final model.

4.3.2.6. The effects of familial risk, anhedonia and current depressive symptoms on subjective ratings of players' trustworthiness

Results suggested that participants significantly rated the fair player as more trustworthy than the neutral player (B=1.02, p<.01) and the unfair player as less trustworthy than the neutral player (B=-1.48, p<.001).

There was a negative main effect of familial risk (B=-.63, p<.05), suggesting that higher familial risk was associated with lower trustworthiness rating for the neutral player (which served as the reference category). There was also an interaction between familial risk and the fair vs. neutral player (B=1.29, p<.05), similar to the effect revealed in the behavioural analysis. This indicates that those at higher familial risk showed a greater differentiation between the fair and neutral player in trustworthiness ratings compared to those at lower familial risk. Furthermore, there was a significant interaction effect between current depressive symptoms and the variable unfair vs. neutral player (B=.25, p<.01). As portrayed in Figure 4.7., this indicates that those with higher depressive symptoms showed a smaller differentiation between the unfair and neutral player in trustworthiness ratings compared to those with lower depressive symptoms. Anhedonia and the covariates were not associated with differentiation between players in trustworthiness ratings.

Table 4.1. Descriptive statistics and correlations between study variables

	Mean (SD) or N%	1	2	3	4	5	6	7	8
1. Age	26.77 (8.11)								
2. Gender (%male, %female)	25.8, 74.2	0.01							
3. IQ	58.12 (5.51)	0.17	-0.03						
4. Number of depressed parents	.67 (.66)	-0.03	0.08	-0.07					
5. Anhedonia	20.32 (5.56)	0.07	-0.18	-0.12	0.07				
6. Past MDD episode (%No ,%yes)	51.5, 48.5	-0.10	0.09	-0.16	0.45**	0.08			
7. Current depressive symptoms	4.54 (5.02)	-0.09	0.01	-0.27*	0.30*	0.46**	0.16		
8. Social functioning	41.39 (5.73)	-0.01	0.17	0.10	-0.10	-0.66**	-0.20	-.44**	

Note: Pearson's correlations were conducted to examine associations between continuous variables and Spearman correlations were conducted to examine associations between categorical variables and continuous variables; *p<.05, **p<.01

Table 4.2. Examining the effect of familial risk, anhedonia and depressive symptoms on differentiation between the players

	Final Model	
Fixed effects	**Estimates(SE)**	**p**
Intercept	50.57 (2.79)	<.001
Time	-1.15 (1.18)	.33
Fair vs. neutral	11.41 (3.23)	<.01
Unfair vs. neutral	-17.04 (2.77)	<.001
(Fair vs. neutral)*time	6.24 (1.67)	<.001
(Unfair vs. neutral)* time	-4.33 (1.67)	<.05
Number of depressed parents	-4.14 (4.25)	.33
(Fair vs. neutral)* Number of depressed parents	10.18 (4.91)	<.05
(Unfair vs. neutral)* Number of depressed parents	9.42 (4.20)	<.05
Anhedonia	.65 (.50)	.19
(Fair vs. neutral)*Anhedonia	-1.49 (.65)	<.05
Number of depressed parents*Anhedonia	-1.03 (.66)	.13
Time*Anhedonia	.17 (.15)	.27
Time* (Fair vs. neutral)* Anhedonia	-.57 (.26)	<.05
(Fair vs. neutral)*Anhedonia* Number of depressed parents	1.59 (.86)	.07
Random effects	**Variance(SD)**	**p**
Intercept (variance)	397.87 (90.16)	<.001
Fair player vs. neutral player variance	454.93 (120.17)	<.001
Unfair player vs. neutral player variance	275.20 (89.12)	<.01
Intercept and (Fair player vs. neutral player) covariance	-167.40 (81.41)	<.05
Intercept and (Unfair player vs. neutral player) covariance	-120.38 (70.28)	.09
(Fair player vs. neutral player) and (Unfair player vs. neutral player) covariance	-158.28 (74.33)	<.05

Note: The table presents the best fitting final regression model**.**

Figure 4.3. Investment rate by player and time

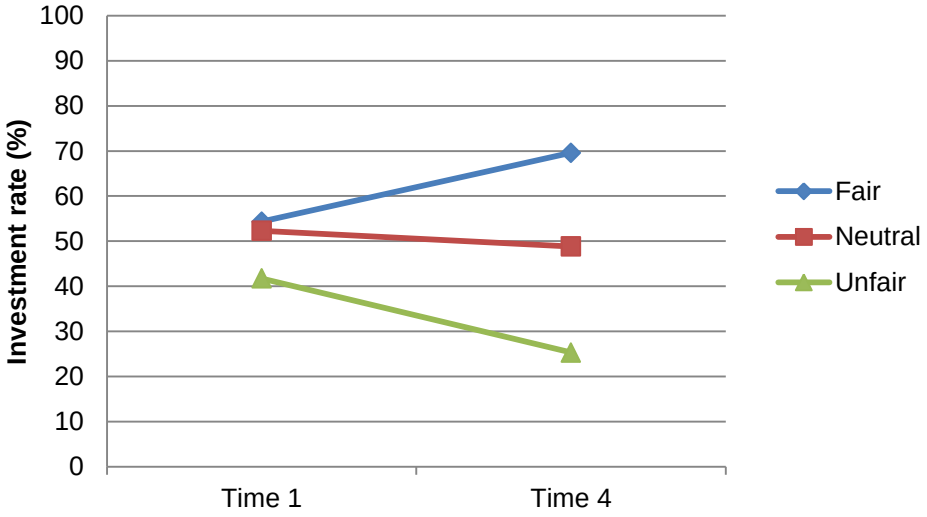
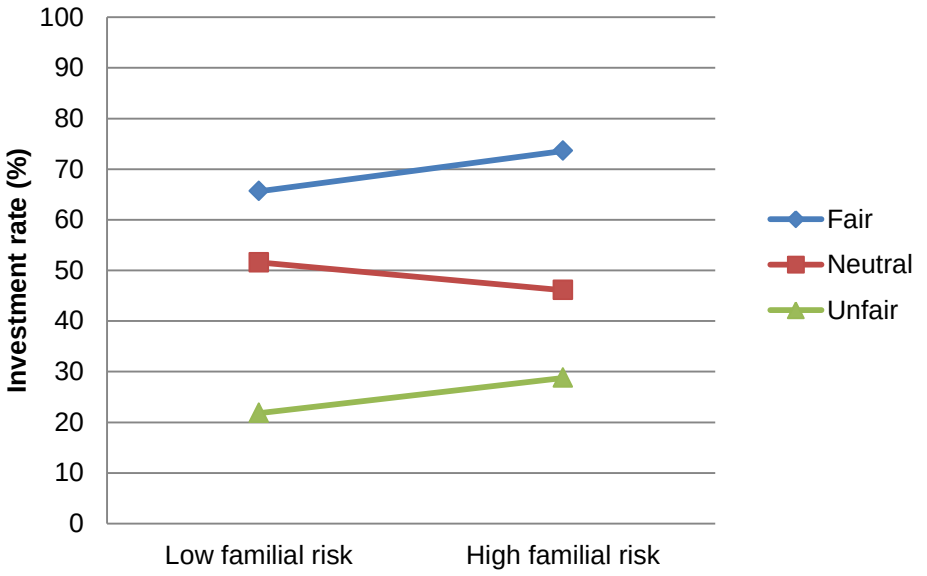
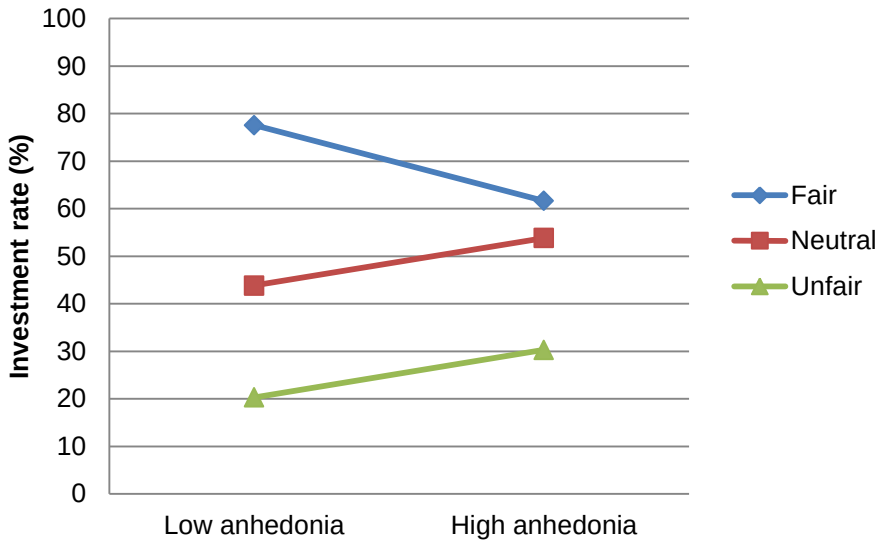


Figure 4.4. Investment rate by player and number of depressed parents



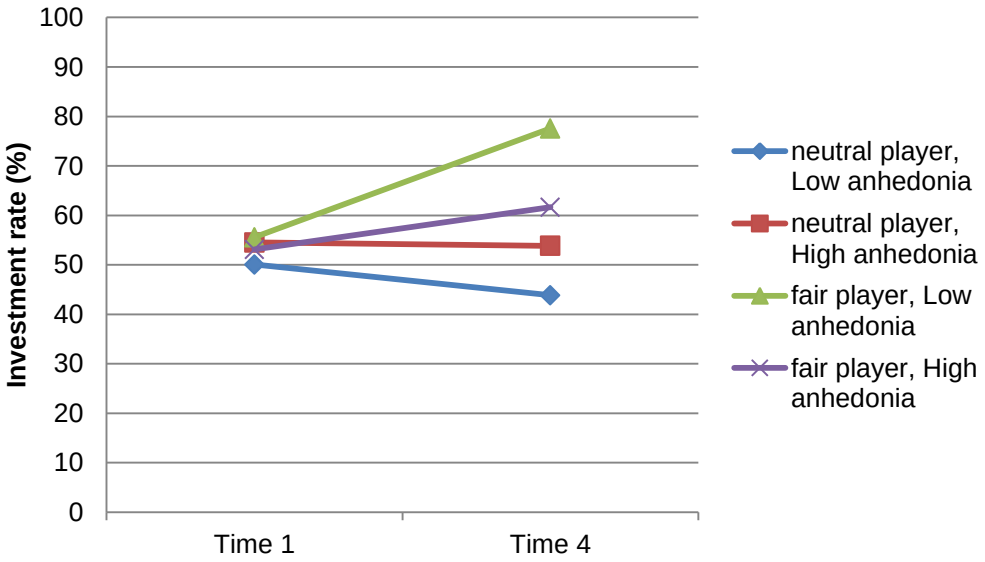
Note: number of depressed parents was estimated at ± 1 SD from the mean; time was estimated at the final block (time=4).

Figure 4.5. Investment rate by player and anhedonia



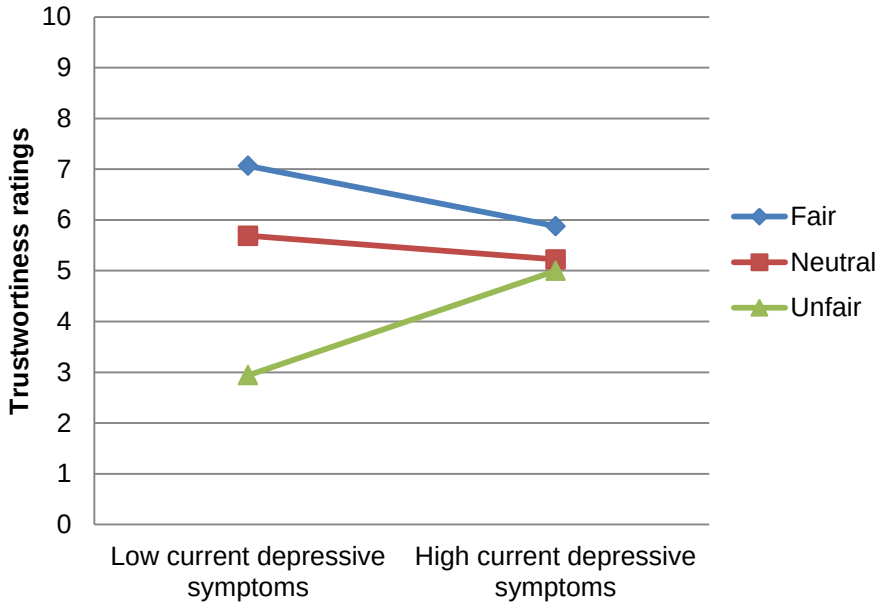
Note: Anhedonia was estimated at ± 1 SD from the mean; time was estimated at the final block (time=4).

Figure 4.6. Investment rate by time, fair vs. neutral player and anhedonia



Note: Anhedonia was estimated at ± 1 SD from the mean

Figure 4.7. Trustworthiness ratings by player and current depressive symptoms



Note: Current depressive symptoms were estimated at ± 1 SD from the mean

4.3.3. The effect of familial risk, anhedonia and current depressive symptoms on learning rate for each player

To explore the second part of research question 1, separate hierarchical multiple regression analyses were conducted with learning rate for each player type as the outcome measure, with the expectation that any effects of familial risk, anhedonia and current depressive symptoms would be more pronounced for the fair player (Table 4.3.). Results of the analysis for the fair player indicated that there was a main effect of anhedonia (step 1; B =-.26 p<.05), suggesting that those with higher levels of anhedonia had a lower rate of learning for the fair player. Anhedonia was not associated with learning rate for the neutral or unfair player. No significant main effect of familial risk, an interactive effect of familial risk and anhedonia, a main effect for current depressive symptoms, an interactive effect of current depressive symptoms and familial risk or any main effects of the covariates were observed for the learning rate of any of the players.

4.3.4. Reward learning and social functioning

To test the second research question, I examined associations between indices of reward learning that were associated with familial risk, anhedonia, or current depressive symptoms and social functioning. Following initial findings suggesting that differentiation between players was associated with familial risk, anhedonia and current depressive symptoms, I first calculated differences scores between the fair and neutral player, and between the unfair and neutral player, in order to examine the association between these indices of differentiation and social functioning. Differences scores were calculated as follows: 1) The difference score of the fair and neutral players was calculated by subtracting the total percentage of investment in the neutral from the percentage of investment in the fair player (for all blocks) 2) The difference score of the neutral and unfair players was calculated by subtracting the total percentage of investment in the unfair player from the percentage of investment in the neutral player (for all blocks). The correlations between the two differences scores, learning rate of the fair player and social functioning were then examined. Social functioning was not associated with the difference score of the fair and neutral player (r=.14, p=.25) or with the difference score of the unfair and neutral player (r=-.08, p=.55). There was however, a significant positive association between learning rate for the fair player and social functioning (r=.26, p<.05). I next examined whether the association between learning rate for the fair player and social

functioning persisted when controlling for familial risk, anhedonia, current depressive symptoms and the covariates and also whether the association was modified by interactions with familial risk, anhedonia and depressive symptoms. As detailed in Table 4.4. (step 3), familial risk for depression was not significantly associated with social functioning (β=-.07, p=.47), whereas anhedonia was negatively associated with social functioning [β=-.59, p<.001]. In the final regression model, there was no significant main effect of learning rate for the fair player on social functioning (β=.07, p=.49). There was however a significant interaction between learning rate and familial risk (β=.22, p<.05; Figure 4.8.). A simple slopes analysis suggested that for those with a high learning rate for the fair player, familial risk was not significantly associated with social functioning (b=1.36, t=1.19, p=.24), whereas for those with low learning rate for the fair player, higher familial risk was associated with lower social functioning (b=-2.50, t=-2.15, p<.05).

No significant effects were observed for the interaction of learning rate for the fair player and anhedonia, the 3-way interaction of learning rate for the fair player, the number of depressed parents and anhedonia, the main effect, or interactions of current depressive symptoms or the covariates.

Table 4.3. Examining the effect of familial risk, anhedonia and depressive symptoms on rate of learning for each player

Variable/outcome	**Learning rate: fair player**				**Learning rate: neutral player**				**Learning rate: unfair player**			
	∆ R^2	**p**	**β**	**p**	**∆ R^2**	**p**	**β**	**p**	**∆ R^2**	**p**	**β**	**p**
Step 1	.07	.12			.02	.50			.03	.34		
Number of depressed			-.01	.91			-.13	.29			.14	.25
Anhedonia			-.26	<.05			.07	.58			.11	.40

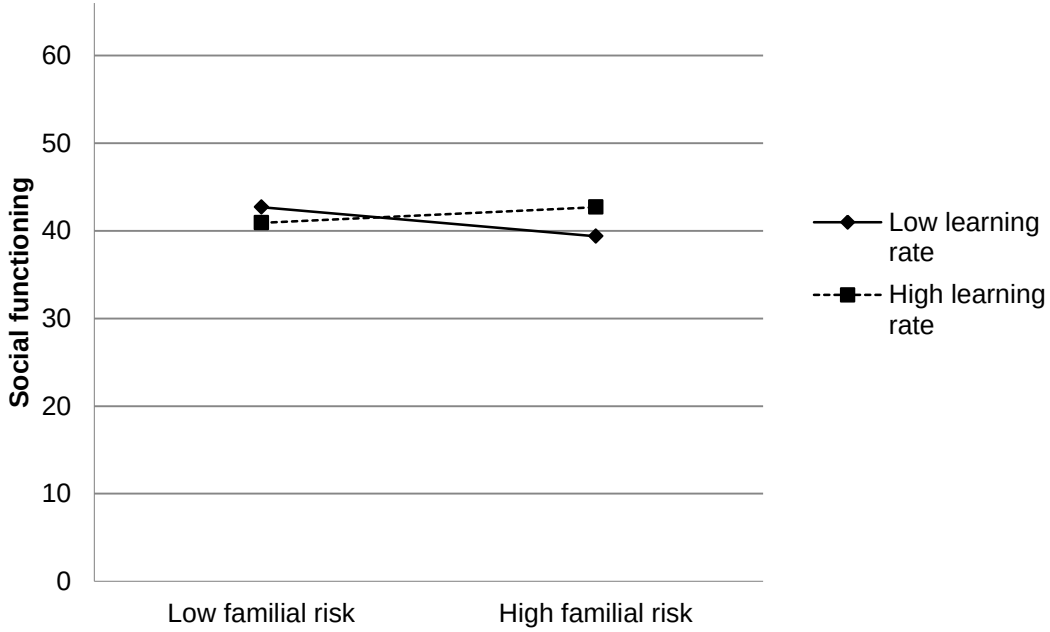
Note: The table presents the best fitting final regression model.

Table 4.4. The association between learning rate for the fair player and social functioning

	ΔR^2	p	β	p
Step 1	.07	<.05		
Learning rate: fair player			.26	<.05
Step 2	.38	<.001		
Learning rate: fair player			.09	.34
Number of depressed parents			-.05	.57
Anhedonia			-.64	<.001
Step 3	.05	<.05		
Learning rate: fair player			.07	.49
Number of depressed parents			-.07	.47
Anhedonia			-.59	<.001
Learning rate: fair player x number of depressed parents			.22	<.05

Note: The table presents the best fitting final regression model.

Figure 4.8. Social functioning by learning rate for the fair player and familial risk



Note: number of depressed parents and learning rate for the fair player were estimated at ± 1 SD from the mean**.**

4.4. Discussion

In the current study I aimed to examine how reward learning in a social context is associated with familial risk for MDD in a sample of young adults. I further examined whether an effect of familial risk on reward learning may be attributable to anhedonia or current depressive symptoms. More specifically, I examined whether familial risk for depression, anhedonia and current depressive symptoms are associated with the ability to differentiate and modulate behaviour for social partners with varying tendencies of social reciprocity. I also examined whether reward learning patterns that were associated with familial risk, anhedonia or current depressive symptoms were also associated with social functioning. I specifically hypothesised that diminished reward learning in behavioural interactions with the fair social partner would be associated with increased familial risk for depression, anhedonia and depressive symptoms given evidence that low reward seeking in highly favourable conditions is associated with the subsequent development of depressive symptoms and disorder (Forbes et al., 2007; Rawal et al., 2013) and that difficulties in showing a preference to a more often rewarded stimulus are associated with anhedonia and depressive symptoms in individuals at high familial risk (Liu et al., 2016; Luking et al., 2015; Morris et al., 2015).

Results of the main analyses indicated that familial risk for depression was associated with the degree of differentiation between social partners with varying propensities for social reciprocity. Participants at higher familial risk made a greater differentiation between the fair and neutral social players compared to those at lower familial risk. Those at higher familial risk also made a smaller differentiation between neutral and unfair players. A similar pattern with regards to the differentiation between the fair and neutral player also emerged in participant ratings of trustworthiness of the different players.

This finding suggests that individuals at low familial risk of depression may have demonstrated a positive bias toward to neutral player whereas those with high familial risk perceived the neutral player's tendency to reciprocate as more similar to that of the unfair player. This pattern of behaviour is in line with research suggesting that healthy people are characterized by a positive bias in attentional processes and in expectations regarding future events, whereas people with depression show either no positive bias or display a negative bias in attention or expectations regarding future events (Dalgleish et al., 2003;

Korn, Sharot, Walter, Heekeren, & Dolan, 2014; Ladouceur et al., 2005; Mezulis, Abramson, Hyde, & Hankin, 2004; Sharot, 2011). Thus it is possible that a positive bias is reduced in those at familial risk for depression (Joormann, Talbot, et al., 2007) and may affect reward learning processes. This pattern of behaviour demonstrated by those at high familial risk of depression is also consistent with cognitive theories of depression, which suggest that a difficulty to tolerate ambiguity and a tendency to interpret ambiguous situations as negative or threatening serves as a cognitive risk factor for depression (Andersen & Schwartz, 1992). An alternative way of characterising this pattern of findings is that individuals at high familial risk may require "stronger" evidence that a person is trustworthy or rewarding in order to trust them. In line with this interpretation, a recent study by Gradin (2015) found that depressed individuals reported lower subjective ratings of happiness compared to controls in response to fair offers but not in response to unfair offers in an ultimatum game, which may suggest that depressed individuals have a different "threshold" for estimating a situation as fair or rewarding. The present results suggest that such biases may also extend to those at high familial risk of depression as well as those who are overtly depressed.

Findings related to the trustworthiness ratings further suggested that higher depressive symptoms were associated with lower trustworthiness ratings for the neutral player, and with a smaller differentiation between the neutral and unfair player. This is consistent with findings of a negative bias or a lack of a positive bias in depressed individuals (Dalgleish et al., 2003; Korn et al., 2014; Ladouceur et al., 2005; Mezulis et al., 2004; Sharot, 2011). However, it is important to mention that these effects of current depressive symptoms were only observed for the trustworthiness ratings, and not for the behavioural outcomes.

Findings also indicated that anhedonia attenuated the differentiation between the fair and neutral player and but not between the neutral and unfair player. Furthermore, higher anhedonia was also associated with a slower learning rate for the fair player only. This suggests that anhedonia may be associated with the ability to modulate behaviour adaptively, especially in social contexts in which the likelihood of reward is high (as in the case of interactions with the fair player). A previous imaging study that employed the same paradigm as that used in this study found a pattern of activation in reward-related brain areas that was specific to reciprocity behaviour of the fair player, suggesting that reciprocal interactions with the fair player may be especially rewarding (Phan et al., 2010).The findings of the current study with regard to the association between anhedonia and reward

learning are in line with previous behavioural studies that found an association between anhedonia and reward learning in high risk and depressed individuals (Chase et al., 2010; Liu et al., 2016; Morris et al., 2015; Pizzagalli et al., 2008; Vrieze et al., 2013). However, as this is a cross sectional study, a cautious interpretation of the association between reward learning and anhedonia is warranted. It is possible that anhedonia affects reward learning, that impairments in reward learning affects hedonic capacity, or that both are driven by a more general deficit in reward processing or sensitivity.

The possibility of interactive effects of familial risk and anhedonia or familial risk and current depressive symptoms on reward learning cannot be ruled out, as trend level three-way interactions between familial risk, anhedonia and player type emerged. Thus, it is possible that due to power limitations, such interactive effects could not be identified in the current study and additional studies that employ larger samples are needed to explore these potentially important interactions.

Anhedonia was associated with social functioning in this study. Among indices of reward learning that were associated with familial risk or anhedonia, only the learning rate for the fair player was positively associated with social functioning. Familial risk for depression was not associated with social functioning, but there was an interaction between familial risk and learning rate for the fair player which revealed that familial risk was not significantly associated with social functioning in those with a high learning rate for the fair player, whereas higher familial risk was associated with lower social functioning in those with low learning rate for the fair player. This finding is intriguing and may suggest some sort of buffering effect of the ability to modify behaviour in response to rewarding situations, but requires replication. This finding that reward learning for the fair player (where the probability of reward is high) was associated with social functioning is consistent with one longitudinal high-risk study that employed a probabilistic decision making paradigm and showed that behavioural responses at high reward probability conditions were associated with depressive symptoms, new onset depressive disorder and functional impairment (Rawal et al., 2013). Although additional studies are needed to examine the specific effects of reward learning on social functioning and risk for depression, these findings suggest that reward learning in situations where the likelihood of reward is high, and the ability to modulate behaviour in response to rewards, might be especially important for adaptive functioning and serve as a protective factor for those at high familial risk.

Somewhat different effects of familial risk and anhedonia on reward learning patterns were observed: familial risk was associated with a greater differentiation between the fair and neutral player and a smaller differentiation between the neutral and unfair player, while anhedonia was associated with reduced differentiation between the fair and neutral player and a "blunting" effect for learning rate for the fair player. Familial risk was not associated with social functioning, but anhedonia and the interaction of familial risk and learning rate for the fair player were associated with social functioning. These findings support previous research which suggests that there is heterogeneity in outcomes for those with high familial risk for depression (Hammen & Brennan, 2003; Mars et al., 2012) and indicate that elevated familial risk on its own may not necessarily be associated with difficulties in social functioning, but rather that it may be important to identify the presence of specific difficulties in reward processing (such as anhedonia and reward learning) in order to understand difficulties in social functioning in both low and high risk individuals.

To the best of my knowledge, this is the first study to employ a social paradigm to assess reward learning in individuals at familial risk of depression. Examining reward learning using tasks that simulate social exchanges may have greater ecological validity such that the findings translate more readily to reward learning in real life situations. It is important to note that in the present study, reward learning was assessed in a context of social exchanges, but did not directly employ social rewards. Using the accumulation of points as a reward permitted comparison with previous reward tasks (which have also used money or points as rewards), and enabled the quantification of participant behaviour under different probabilities of reinforcement. However, as pursuing social rewards serves as an important motivation for human behaviour and is strongly implicated in emotional well-being (Baumeister & Leary, 1995; Forbes, 2009), it may be beneficial for future studies to examine reward learning for social rewards.

It should also be noted that although the reward task employed in this study is suggested to assess reward learning, it may also involve other cognitive or social processes. For example, performance in this task may be affected by not only by reward learning but also by reward seeking behaviour, meaning that after learning the players' tendencies to reciprocate, participants may nonetheless vary on how they choose to invest in the players. The performance in the task may also be affected by working memory as learning the players' tendencies for reciprocity requires integrating reinforcement history over time and "holding" it in mind. Finally, as this task examines reward processing in a social

context, performance may also involve social/ interpersonal processes such as attachment style (Strathearn, Fonagy, Amico, & Montague, 2009).

It is worth mentioning that in this study parental depression was assessed by interviewing the offspring using the family history method (Andreasen et al., 1977) and not by direct interviews with the parents. Although the family history method has been found to be valid and reliable in previous studies (Colvin et al., 2014; Orvaschel et al., 1982; Weissman et al., 2000), it is possible that recall difficulties affected how participants reported parental depression. It is also possible that current depressive symptomatology may have influenced the recall of parental depressive episodes. Finally, it is worth mentioning that this type of assessment allowed including only high risk participants who were aware of parental depression in the present sample.

Another possible limitation of this study is that participants were not excluded based on current or previous psychopathology. As offspring of depressed parents are characterised by increased rates of depression (Rice et al., 2002; Weissman et al., 2006), excluding those with previous or current psychopathology might result in a resilient sample and limit the ability to generalise results to a typical sample of offspring of depressed parents (Luking et al., 2015). I did, however examine the contribution of a past and current depressive psychopathology as control variables in the analyses.

It has been suggested that the reduced engagement in pleasurable activities present in depressed individuals is not necessarily related only to diminished enjoyment from such activities, but may also occur due to impairments in earlier stages of reward processing such as decreased reward anticipation, and difficulties in integrating reinforcement history and cost-benefit information in decision making (Sherdell et al., 2012; Treadway et al., 2012; Treadway & Zald, 2011). It has also been hypothesised that in situations that include uncertainty about the outcome and risk of loss, depressed individuals might overestimate the probability of an averse outcome and this may override motivation to pursue rewards and result in diminished reward seeking (Sherdell et al., 2012). This study suggests that altered patterns of reinforcement learning might be present in individuals with high familial risk for depression and those with high levels of anhedonia, even when adjusting for current and previous depressive symptomatology. There was also evidence of an association between reward learning and social functioning.

The effectiveness of therapeutic interventions for depressed people that target reduced motivation to engage in rewarding activities, such as behavioural activation, has

been shown in previous studies (Dichter et al., 2009; Dimidjian et al., 2006; Dobson et al., 2008). However, reward processing deficits have not been sufficiently addressed in prevention programmes. A recent novel classroom-based prevention program that targeted reward-seeking decision making (i.e. identifying risks and rewards in decision making) was effective in reducing depressive symptoms and improving reward seeking in a sample of adolescents (Rice et al., 2015). Altogether, evidence suggests that future studies should examine how disruptions in reward learning may increase risk for depression, for instance by establishing the temporal relationship between reward learning, social functioning and depressive symptomatology.

CHAPTER 5: The development and validation of a new measure of sensitivity to positive and negative social situations for adolescents

Adolescence is characterized by an increase in rates of depressive symptoms and disorder, especially among females. Although it is not yet clear why this increase in prevalence occurs during adolescence, it has been suggested that increased reward sensitivity and heightened saliency of the social arena during that period may be important. Theoretical models suggest that heightened sensitivity to negative social experiences may interfere with social reward seeking behavior and increase risk for depression, especially among females, who develop a stronger need for affiliation during adolescence. However, there is a lack of studies examining associations between sensitivity to social rewards and negative social experiences, social functioning and depressive symptoms in adolescents, which may stem in part from a lack of available measures assessing sensitivity to social rewards and to negative social experiences in this age group. This study aimed to develop a new measure assessing sensitivity to positive and negative social situations suitable for adolescents and to examine associations between these constructs and indices of social functioning, engagement in extracurricular activities and depressive symptoms. Potential gender differences in sensitivity to positive and negative social situations and in their associations with depressive symptoms were also examined. These associations were explored in a cross sectional design in a community sample of adolescents aged 11-15. The new questionnaire had good internal reliability and construct validity. The main findings indicated that adolescents who showed greater sensitivity to positive social situations, also showed greater sensitivity to negative social situations, better social functioning as assessed by greater self-reported satisfaction of friendships and higher engagement in extra-curricular activities. There was no evidence of associations between sensitivity to positive or negative social situations and depressive symptoms. There was evidence of gender differences in sensitivity to positive social situations and sensitivity to negative social situations such that females showed greater sensitivity to both. Females also reported higher rates of depressive symptoms than males. Finally, there was evidence that sensitivity to positive social situations moderated the association between gender and depressive symptoms such that among those with high sensitivity to positive social situations there was no significant association between gender and depressive symptoms, whereas among those with low sensitivity to positive social situations, females had higher rates of depressive symptoms.

5.1. Introduction

Early onset depression is characterised by a chronic and impairing symptom course and is associated with a range of negative outcomes such as impairment in academic performance and social functioning, substance abuse, and suicidal behavior (Birmaher et al., 1996; Fergusson & Woodward, 2002; Thapar et al., 2012). Whereas depression in childhood is uncommon, rates of depressive disorder and depressive symptoms increase significantly during adolescence. Thus, the one year prevalence rate of depressive disorder is 0.4-2.8% in childhood, and 4-8% percent in adolescence (Birmaher et al., 1996; Costello et al., 2006; Rice & Rawal, 2010; Thapar et al., 2012). Around 1 in 5 young people will experience a depressive episode by the end of adolescence (Hankin et al., 1998; Stallard et al., 2012; Thapar et al., 2012). This increase in the prevalence of depressive disorder and symptoms is especially prominent among girls, who are twice as likely to develop depression compared to boys during adolescence. Previous studies have shown that the gender differences in the prevalence of depression evident in adult samples first emerge during adolescence (Angold, Costello, & Worthman, 1998; Cyranowski, Frank, Young, & Shear, 2000; Davey et al., 2008; Hankin et al., 1998; Piccinelli & Wilkinson, 2000; Silk et al., 2012). Thus, adolescence serves as a key developmental period to explore mechanisms related to onset of depression.

Although it is not yet clear why this increase in prevalence occurs during adolescence, it has been suggested that biological, cognitive and environmental changes act together to increase risk for depression during that time (Davey et al., 2008; Silk et al., 2012). Reward sensitivity, especially in a social context, has been suggested as an important cognitive/behavioral mechanism related to the onset of depression during adolescence (Davey et al., 2008; Forbes & Dahl, 2005, 2012). Adolescence is characterised by changes in both reward sensitivity and social behavior. Adolescents show greater reward seeking and higher risk taking behaviour compared to children and adults; this is expressed as enhanced sensitivity to rewards and the pursuit of novel and exciting experiences (Spear, 2011; Steinberg, 2007). Social behavior also changes substantially during adolescence, with peer acceptance and social affiliation becoming increasingly important (Davey et al., 2008; Foulkes & Blakemore, 2016; Silk et al., 2012), friendships becoming closer, social groups that include both genders beginning to develop, and romantic relationships emerging (Davey et al., 2008; Silk et al., 2012). It has been suggested that during adolescence, abstract and complex social goals such as ‘belonging’ and ‘attaining social status’ become increasingly salient. Biological changes that occur

during adolescence such as the maturation of prefrontal cortex are thought to contribute to this (Davey et al., 2008). However, such social goals are not always easily obtained and adolescent relationships are often unstable (Brown, 2004; Hardy, Bukowski, & Sippola, 2002) and this is suggested to be related to the high rates of interpersonal stress experienced during adolescence (Davey et al., 2008; Silk et al., 2012). It has also been suggested that girls develop a stronger need for affiliation, especially in the form of close confiding relations, and may also demonstrate greater sensitivity to social stress (Davey et al., 2008; Kendler & Gardner, 2014; London, Downey, Bonica, & Paltin, 2007; Rudolph, 2002) and that this may contribute to the increase in depressive symptomatology seen in girls around adolescence (Thapar et al., 2012). Thus for adolescents, reward sensitivity - especially in the social arena - may play an important role in social functioning and depressive symptoms and disorder. Indeed, several models have been suggested to explain the associations between social relationships, reward seeking and depression in adolescence (Silk et al., 2012; Davey et al., 2008). For example, Silk et al. (2012) suggested that heightened sensitivity to social evaluation or to negative interpersonal experiences may interfere with social reward-seeking behaviour and lead to depression. Davey et al. (2008) suggest that experiencing negative interpersonal events, and specifically social disappointment, may suppress reward-seeking behaviour and increase risk for depression, especially in vulnerable individuals such as those with a genetic predisposition to depression, or those with a family environment that is less capable of providing support following an experience of disappointment. Thus, it seems that sensitivity to social rewards may be important to assess in the context of adolescent depression.

Indeed, as described in chapter 1, previous research has shown that depressed individuals are characterised by reward processing disruptions, and these are suggested to underlie the diminished positive affect, anhedonia and social withdrawal observed in depression (Alloy, Olino, Freed, & Nusslock, 2016; Forbes & Dahl, 2005, 2012). Several lines of evidence suggest that depression is associated with lower sensitivity to rewards as expressed by lower enjoyment from pleasurable activities and difficulties in modulating behavior in order to pursue rewards. For instance, several studies in adults have shown that depressed individuals rate positive stimuli (such as pleasant pictures or films) in experimental tasks less positively than healthy controls (B. D. Dunn, Dalgleish, Lawrence, Cusack, & Ogilvie, 2004; Rottenberg, Kasch, Gross, & Gotlib, 2002; Sloan, Strauss, Quirk, & Sajatovic, 1997; Sloan, Strauss, & Wisner, 2001), and show a lower tendency to

modulate behavior as a function of probability or magnitude of reward compared to healthy controls (Murphy et al., 2001; Pizzagalli et al., 2008). Fewer studies have examined reward processing in adolescents. However, initial evidence suggests that difficulties in adapting behavior in response to the presence of reward, or in order to pursue rewards of greater value in experimental tasks, are associated with current depression as well as with risk for developing depression in adolescence (Forbes et al., 2007; Hardin, Schroth, Pine, & Ernst, 2007; Jazbec et al., 2005; Morris et al., 2015; Rawal et al., 2013). Together, this evidence suggests that depressed individuals may have reduced sensitivity to the rewarding value of positive stimuli or experiences. Nonetheless, there is a lack of studies examining the importance of social reward sensitivity in adolescence despite the consensus that this may account for the increase in prevalence of depression in adolescents and especially in adolescent females. The few studies that have examined this give initial support for the association between social reward sensitivity and risk for depression. For example, Joiner et al. (2002) showed that low social pleasurable engagement as assessed by adolescent self-report was associated over time with depression in a longitudinal study of adolescents . Furthermore, a study by Olino et al. (2015) showed that adolescent offspring of depressed parents demonstrated lower levels of social reward-seeking behaviour (as assessed by parent report on adolescent social affiliation) compared to adolescent offspring of healthy parents. Finally, reward seeking in a gambling (non-social) task was associated with both current depression and indices of social functioning in a sample of adolescents at familial risk for depression(Rawal et al., 2013).

The lack of studies examining associations between reward sensitivity, particularly social reward sensitivity, and depression in adolescents may stem in part from a lack of available measures assessing these constructs in this age group. For example, to date only one measure aimed at assessing aspects of the experience of pleasure /anhedonia in children, *the Pleasure Scale for Children*(Kazdin, 1989), has been developed. The majority of self-report measures aimed at assessing the experience of pleasure/anhedonia (*the Temporal Experience of Pleasure Scale*; Gard, Gard, Kring, & John, 2006; *the Snaith-Hamilton Pleasure Scale*; Snaith et al., 1995; *the Sensitivity to Punishment and Sensitivity to Reward Questionnaire*; Torrubia, Avila, Moltó, & Caseras, 2001), sensitivity to social reward (*The Social Reward Questionnaire*; Foulkes, Viding, McCrory, & Neumann, 2014) or social anhedonia (*the Revised Social Anhedonia Scale*; Eckblad, Chapman, Chapman, & Mishlove, 1982; *the Anticipatory and Consummatory Interpersonal Pleasure Scale*;

Gooding & Pflum, 2014) have been developed for use in adults. Among these, only one scale, aimed at assessing social anhedonia, *the Anticipatory and Consummatory Interpersonal Pleasure Scale-Adolescent version* (ACIPS-A; Gooding, Pflum, Fonseca-Pedero, & Paino, 2016) has been recently validated in adolescents[1]. Developing measures aimed at assessing sensitivity to socially rewarding experiences as well as sensitivity to negative social experiences can be useful for examining associations between these constructs and depression in adolescents.

The hypothesised associations between sensitivity to negative social experiences, reward sensitivity and depression in adolescence (Alloy et al., 2016; Davey et al., 2008; Silk et al., 2012) have also not been directly examined. Previous studies of depressed adults have indicated that depression is associated with enhanced sensitivity to negative events and risk aversion (Grosscup & Lewinsohn, 1980; Joffe, Bagby, Levitt, Regan, & Parker, 1993; Lewinsohn, Lobitz, & Wilson, 1973; Lewinsohn & Talkington, 1979; Rawal et al., 2013; Schless, Schwartz, Goetz, & Mendels, 1974; Smoski et al., 2008). The literature examining the relationship between sensitivity to negative events, particularly negative social events, and adolescent depressive symptomatology is much more limited. For instance, it has been shown that sensitivity to rejection was linked with depressive symptoms in adolescents (Harper, Dickson, & Welsh, 2006; McDonald, Bowker, Rubin, Laursen, & Duchene, 2010). However, previous studies have not examined how sensitivity to a wide range of negative social situations may be associated with reward sensitivity, social functioning and depression in adolescents.

The goal of this exploratory study was to develop a new measure assessing sensitivity to positive and negative social situations suitable for adolescents and to examine associations between these constructs and indices of social functioning, engagement in extracurricular activities and depressive symptoms. I aimed mostly to explore sensitivity to positive social situations while examining sensitivity to negative social situations as a potentially related construct. Self-report measures assessing the subjective experience of pleasure and enjoyment in rewarding activities usually employ a Likert response scale that requires participants to rate how much they would enjoy a certain activity or to what extent a certain activity will make them happy (Gooding et al., 2016; Kazdin, 1989; Snaith et al., 1995). These types of response scales are likely to be affected by factors such as current mood (Dichter, 2010; Willner & Healy, 1994) or a social

1 Published after the planning and data collection of the current study.

desirability bias (Van de Mortel, 2008). Furthermore, previous studies found that associations between these measures and laboratory based measures of hedonic capacity vary substantially such that some studies found no association (Berenbaum, Snowhite, & Oltmanns, 1987; Willner & Healy, 1994) and others found these to be only moderately associated (Leventhal, Chasson, Tapia, Miller, & Pettit, 2006). In this study, I aimed to create a measure that employs some of the qualities of performance-based measures of reward processing, which usually assess reward sensitivity by examining how subjects' propensities to gamble points or money are modified by rewards of different magnitude. Previous research has suggested that it is possible to assess the rewarding value of a reward with an unknown subjective value using another reward with a known value such as money (Deaner, Khera, & Platt, 2005; Hayden, Parikh, Deaner, & Platt, 2007; Tamir & Mitchell, 2012). Thus, it was hypothesised that adolescents would be able to quantify the rewarding value of each social situation using points which are often used in games as a measure of reward. It has been suggested that using points may be preferable to using money to assess reward in young people as the perceived value of money may be different in young people compared to adults (Casey, 2015; S.J. Blakemore;personal communication, July 30, 2014). This is the first study to employ this strategy for assessing the rewarding value of positive social situations and the aversive value of negative social situations by asking participants to choose how many points they were willing to "invest" in order to experience positive situations or to avoid experiencing negative situations. Thus, although based on subjective ratings, this type of assessment was proposed to be a slightly more indirect way of assessing the rewarding value of social situations compared to other self-report measures assessing similar constructs and also allowed using a similar response scale to "quantify" the value of positive and negative social situations.

The current study aims to pursue the following specific goals:

1) To assess the internal reliability and construct validity of the new measure. Construct validity will be assessed by examining: A) the internal structure of items assessing sensitivity to positive social situations and items assessing sensitivity to negative social situations. B) The association between these two constructs. C) Associations between these two constructs and one established measure of the experience of pleasure/anhedonia in young people and 2 other established measures of the experience of pleasure/anhedonia that were developed for adults. It was hypothesised that sensitivity to positive social situations will be inversely associated with sensitivity to negative social situations. It was also hypothesised

that sensitivity to positive social situations will be positively associated with related measures of the experience of pleasure, and that an inverse association will be observed for sensitivity to negative social situations and related measures of the experience of pleasure (Davey et al., 2008; Silk et al., 2012).

2) To examine associations between sensitivity to positive social situations, sensitivity to negative social situations, and indices of social functioning and engagement in extra-curricular activities. Following literature suggesting that reward processing is important for adaptive social functioning (Forbes, 2009; Rawal et al., 2013) and that sensitivity to negative social experiences may suppress reward-seeking behaviour in the social arena in adolescents (Davey et al., 2008; Silk et al., 2012) it was hypothesised that sensitivity to positive social situations will be positively associated with adaptive social functioning and engagement in extra-curricular activities, whereas sensitivity to negative social situations will be negatively associated with adaptive social functioning and engagement in extra-curricular activities.

3) To examine associations between sensitivity to positive social situations, sensitivity to negative social situations and depressive symptoms, and to explore potential gender differences in these constructs and their associations with depressive symptoms. Following previous research linking reward sensitivity and sensitivity to negative events with depression (Alloy et al., 2016; Forbes & Dahl, 2005, 2012; Grosscup & Lewinsohn, 1980; Joffe et al., 1993; Smoski et al., 2008) it was hypothesised that greater sensitivity to positive social situations will be associated with lower rates of depressive symptoms and that greater sensitivity to negative social situations will be associated with higher rates of depressive symptoms. Following previous research suggesting the existence of gender differences in social relationships in adolescents, and linking these with evidence of gender differences in depression rates during adolescence (Davey et al., 2008; London et al., 2007; Rudolph, 2002; Thapar et al., 2012), it was hypothesised that: A) females would show greater sensitivity to both positive and negative social situations compared to males. B) Associations between sensitivity to positive social situations, sensitivity to negative social situations and depressive symptoms will be more pronounced for females.

4) To examine which social situations adolescents found to be the most rewarding, and which the most aversive.

5.2. Method

5.2.1. Sample

Participants were adolescents aged 11-15 (years 8-10) recruited from a Church of England secondary school in the South of England. Parents were given information sheets about the study and had the choice to opt their child out of the study. Adolescents were given information sheets and a verbal explanation about the study and had the opportunity to withdraw from the study themselves. A total of 170 families were approached to participate in the study. Three parents withdrew their children from the study and two adolescents chose to withdraw from the study themselves. Thus, a final sample of 165 adolescents (ages 11-15) completed the study. The study was approved by the UCL Research Ethics Committee (Ethics application 1522/004).

5.2.2. Development of the new measure: The Social Situations Questionnaire

5.2.2.1. Item selection

The Positive Social Situations Scale

The first section of the questionnaire (the Positive Social Situations Scale) aimed to assess sensitivity to socially rewarding situations by measuring the rewarding value of a range of hypothetical positive social situations. To develop the items, relevant theoretical and empirical literature examining social goals and rewards in adolescence and adulthood (Anderman & Anderman, 1999; Baumeister & Leary, 1995; Buss, 1983; Gable, 2006; Jarvinen & Nicholls, 1996; McCollum, 2005) as well as items from related measures such as *the Children's Pleasure Scale* (Kazdin, 1989), *the Pleasant Events Schedule* (MacPhillamy & Lewinsohn, 1982), *the Social Reward Questionnaire* (Foulkes et al., 2014), *the Snaith-Hamilton Pleasure Scale* (Snaith et al., 1995) and the *Anticipatory and Consummatory Interpersonal Pleasure Scale* (Gooding & Pflum, 2014) were reviewed. This process served to identify important classes of positive social situations and experiences that are hypothesised to be salient in adolescence and associated with emotional well-being and depression. The following social rewards were consistently identified as important for adolescents: intimacy (close relationships), nurturance (helping others and receiving help), peer acceptance, popularity/status, leadership, and being perceived as attractive. Following this process, the questionnaire items were developed with the aim of tapping a wide range of social situations associated with the above social rewards.

The Negative Social Situations Scale

The second section of the questionnaire (the Negative Social Situations Scale) aimed at assessing sensitivity to negative social situations by measuring the aversive value of a range of hypothetical negative social situations. To develop the items, relevant theoretical and empirical literature examining salient social stressors in adolescence and adulthood and negative social experiences associated with depression (Baumeister & Leary, 1995; Cacioppo et al., 2006; Compas, Malcarne, & Fondacaro, 1988; Gable, 2006; Hammen, 2005; Rudolph, 2002; Santini et al., 2015; Teo et al., 2013) as well as related measures such as *the Fear of Negative Evaluation Scale* (Watson & Friend, 1969), *the Sensitivity to Rejection Scale* (Mehrabian, 1972, 1976) and *the Consequences of Negative Social Events Questionnaire* (Wilson & Rapee, 2005) were reviewed. The following negative social experiences were consistently identified as important in adolescence and associated with depression: interpersonal loss, interpersonal conflict, peer rejection, social exclusion and low social support. Following this process, the questionnaire items were developed with the aim of tapping a wide range of negative social situations associated with these types of experiences.

5.2.2.2. Response scale

Participants were instructed to imagine that they have an allowance of 100 points for each question and were requested to decide how many points they were willing to invest either to experience a positive social situation (Positive Social Situations scale) or to avoid experiencing a negative social situation (Negative Social Situations Scale). The instructions given to participants for the Positive Social Situations Scale were to give more points to social situations that they found more desirable. Thus, all items began with the words: "how many points would you spend to…" followed by the description of the social situation. For example, one of the items that assessed the rewarding value of nurturance was: "how many points would you spend to make a friend who is sad feel better?" and one of the items that assessed peer acceptance was "how many points would you spend to feel that your classmates like you?" The instructions given to participants for the Negative Social Situations Scale were to give more points to social situations that they found more aversive. Thus, all items began with the words: "how many points would you spend to avoid…" followed by the description of the negative social situation. For example, one of the items that assessed the value of social exclusion was: "how many points would you spend to avoid not being invited to a party that all your friends have been invited to?" and

one of the items that assessed interpersonal conflict was "how many points would you spend to avoid having an argument with a close friend?".

5.2.2.3. Review and piloting

Following the development of the questionnaire items, two educational psychologists and a senior researcher, experienced at working with adolescents reviewed and commented on the questionnaire. The questionnaire was then piloted with two adolescents to ascertain the clarity of instructions and items. Amendments were made following these revisions and piloting. The final version of the questionnaire included 20 items referring to positive social situations and 20 items referring to negative social situations. The instructions included an example item for each section and an illustration of a visual rating scale (for the full questionnaire, see Appendix 5.1.).

5.2.3. Procedure

Data collection was conducted by a practicing educational psychologist in a class during a school day. The instructions for the new questionnaire were first read to the participants. Participants then completed an example item for each section and had the opportunity to ask questions prior to completing the full questionnaire. Following completion of the new questionnaire, participants completed additional measures assessing the experience of pleasure/anhedonia, depressive symptoms, engagement in extra-curricular activities and social functioning. All measures took approximately 45 minutes to complete.

5.2.4. Measures

5.2.4.1. The Social Situations Questionnaire

As described, a 40-item questionnaire was completed to assess sensitivity to positive social situations (20 items) and sensitivity to negative social situations (20 items). Total scores for both scales were computed as the mean number of points spent for positive social situations and mean number of points spent for negative social situations.

5.2.4.2. The Snaith-Hamilton Pleasure Scale (SHAPS; Snaith et al., 1995)

This is a well validated 14-item questionnaire used to assess the hedonic value of different pleasurable experiences. The questionnaire covers four domains of hedonic experiences: interest/hobbies, social interaction, sensory experience, and food/drink.

Participants are requested to rate how much they agree with each statement regarding the hedonic value of a pleasurable experience. Responses were scored on a four point scale ("definitely agree" to "strongly disagree"). Higher scores reflect lower levels of pleasure (higher levels of anhedonia).

5.2.4.3. The Children's Pleasure Scale (Kazdin, 1989)

This is a self-report measure aimed at assessing anhedonia in children aged 6-13. The original 39-item questionnaire was shortened such that old fashioned or age-inappropriate items were removed. Thus, a modified shortened version that included 18 items was used in this study. In the questionnaire participants were asked to rate how enjoyable they would find different rewarding situations on a five point scale ("not at all" to "very much"). Higher scores reflect higher levels of enjoyment (lower levels of anhedonia).

5.2.4.4. The Pleasant Events Schedule (PES; MacPhillamy & Lewinsohn, 1982)

This is a self-report measure aimed at assessing the frequency and enjoyability of common rewarding experiences. For each rewarding experience the participant is requested to rate how often this experience happened over the past month on a 3 point scale as well as how pleasant was it on a 3 point scale. The original scale was very long and included 320 items. This was shortened to 39 items to exclude old fashioned or age-inappropriate items and to reduce length. Furthermore, in the version included in this study some items have been modified or added to reflect current rewarding activities for young people. Examples of such items are: “Browsing the internet or playing computer games”, “Being liked on Facebook or Instagram”(See appendix 5.2.).

Two scores were derived from the measure: an enjoyability score, which is computed as the sum of enjoyability scores for all the rewarding experience that occurred over the past month, and a combination score which is computed as the sum of the product of the frequency and enjoyability ratings for all items.

5.2.4.5. Activities

A checklist of 4 items was used to assess the frequency of engagement in extra-curricular activities such as sports, exercise and participation in clubs/classes (Appendix 5.3.; Rawal et al., 2013). Frequency of participation was measured on a 5 point scale (0=never or less than once a month, 1=at least once a month, 2=once every 2 weeks, 3= at least once a week, 4=more than once a week). The total score was computed as the

sum of the item scores. Higher scores reflect a greater engagement in extra-curricular activities. Cronbach's α=.65.

5.2.4.6. Strengths and Difficulties Questionnaire: peer problems subscale (SDQ; R. Goodman, 1997)

The SDQ is a widely used questionnaire aimed at assessing positive and negative attributes of behavior, emotional problems and peer relationships. In this study the 5-item peer problems subscale was used. Higher scores reflect fewer problems with peers and better peer relationships.

5.2.4.7. Friendship Quality Questionnaire (Bukowski, Hoza, & Boivin, 1994)

A shortened version of this self-report questionnaire was used to assess the quality of friendship with the person the participant regarded as their best friend. The participants were requested to rate how much they agree with statements assessing companionship, closeness, help, security and conflict. Higher scores represent a higher quality of friendship.

5.2.4.8. Mood and Feelings Questionnaire (MFQ; Angold, Costello, Messer, & Pickles, 1995)

The MFQ is a widely used self-report questionnaire which assesses depressive symptoms in children and adolescents. A 34-item version of the MFQ was used in this study. Higher scores represent higher depressive symptoms.

5.2.5. Statistical analysis

First, simple descriptive statistics of the new measure were examined. A paired t-test was conducted to compare the participants' sensitivity to positive versus negative social situations.

Internal reliability of the two sections of the questionnaire was assessed using Cronbach's alpha. In order to assess construct validity, first the internal structure of the two scales (both sections of the new questionnaire) was examined by conducting an exploratory Principle Component Analysis with Oblimin rotation separately for each scale. Second, the associations between the total scores on both scales and between these and related measures of the experience of pleasure and anhedonia were assessed using Pearson's correlation coefficients.

The associations between the total scores on both scales and measures of social functioning, engagement in extra-curricular activities and depressive symptoms were then examined using Pearson's correlation coefficients.

To examine gender differences in sensitivity to positive and negative social situations, paired t-tests were conducted to examine differences in investment in the experience of positive social situations and the avoidance of negative social situations for each gender group separately. Following this, independent t-tests were conducted to compare males and females on the total score for each of the two scales of the questionnaire. Finally, to examine whether the associations between the total scores of the two scales and depressive symptoms were moderated by gender, Pearson's correlation coefficients were examined for males and females separately, and a hierarchical multiple regression was conducted for each scale to examine the interactive effect of sensitivity to positive/negative social situations and gender on depressive symptoms. Age was included in regression analyses as a covariate, as previous research suggests that depression rates increase throughout adolescence (Hankin et al., 1998; Thapar et al., 2012). Variables were entered to the regression analyses in the following order: 1) gender, sensitivity to positive/negative social situations, age; 2) the interaction of gender and sensitivity to positive/negative social situations. Significant interactions were followed up by simple slope analysis (Cohen et al., 2003; Dawson, 2014).

Finally, to explore which social situations adolescents found to be the most rewarding and which the most aversive, the five highest rated items for each scale were examined.

5.3. Results

5.3.1. Descriptive statistics and reliability

Means, standard deviations and ranges of the total scores of both sections of the questionnaire as well as other measures employed in the study are presented in Table 5.1. The mean score of the Positive Social Situations Scale was 61.05, standard deviation was 17.46 and the range was 86.25. The mean score of the Negative Social Situations Scale was 58.28, standard deviation was 21.69 and the range was 97.89. This suggests that there was substantial heterogeneity in participants' ratings in both scales. In the entire sample, participants were likely to invest more points to experience socially rewarding situations than to avoid negative social situations ($t(163) = 2.38$, $p < .05$; Table 5.1.). Mean ratings and standard deviations for each questionnaire item are presented in Table 5.2.

(Positive Social Situations Scale) and Table 5.3. (Negative Social Situations Scale). Both the Positive Social Situations Scale and the Negative Social Situations Scale had high internal reliability; Cronbach's alpha=.91 and .92 respectively.

Table 5.1. Descriptive statistics and correlations between study variables

	Descriptives		Correlations											
	Mean (sd) or N%	Range (Min,Max)	1	2	3	4	5	6	7	8	9	10	11	12
1. The Positive Social Situations Scale	61.05 (17.46)	86 (6 ,92)												
2. The negative Social Situations Scale	58.28 (21.69)	98 (0,98)	.69**											
3. SHAPS	25.66 (5.17)	28 (14,42)	-.22**	-.23**										
4. Children's pleasure scale	67.31 (11.42)	61 (29,90)	.36**	.27**	-.44**									
5. PES enjoyability	59.47 (8.30)	37 (37,74)	.26**	.20*	-.39**	.28**								
6. PES combination score (frequency x enjoyability)	70.11 (21.43)	95 (26,121)	.34**	.21*	-.43**	.40**	.62**							
7. Activities	12.79 (3.38)	16 (0,16)	.20*	.06	-.07	.18*	.04	.30**						
8. SDQ peer problems subscale	7.81 (1.75)	10 (0,10)	.12	.10	-.24**	.19*	.25**	.29**	.15[a]					
9. Friendships	57.74 (9.23)	47 (26,73)	.23**	.24**	-.40**	.34**	.37**	.47**	.18*	.26**				
10. Depressive symptoms	18.36 (13.15)	62 (0,62)	.05	.05	.32**	-.26**	-.19*	-.09	-.04	-.52**	-.04			
11. Gender (%male, %female)	52.8,47.2	-	.18[*]	.28[**]	-.11	-.09	.19[*]	.18[*]	-.07	.07	.41[**]	.21[**]		
12. Age	13.40 (1.02)	4 (11,15)	-.24[**]	-.27[**]	.23[**]	-.18[*]	-.21[*]	-.05	.05	-.14[a]	-.11	.18[*]	-.05	

Note: N ranges between 99-165 due to missing values; [a]p<.1,*p<.05, ** p<.01

5.3.2. Construct validity

5.3.2.1. Questionnaire structure

The Positive Social Situations Scale

A Principal Component Analysis (PCA) was conducted on the 20 items of the questionnaire with Oblimin rotation. The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis (KMO=.86) and all KMO values for individual items were >.74. Bartlett's test of sphericity ($\chi^2(190)=1456.81$, $p<.001$) indicated that correlations between items were sufficiently large for PCA. Four components had eigenvalues over Kaiser's criterion of 1. The scree plot showed an inflexion that would justify retaining 2 components. Thus 2 factors that explained together 47.27% of the variance were retained in the final analysis. Table 5.2. shows the item loadings on the two components after rotation. It seems that component 1 represents social status and peer acceptance, and component 2 represents nurturance and forming close relationships. Item no 8: “How many points would you spend to go to a family meal?” was the only item that did not load on either of the two components.

The Negative Social Situations Scale

A Principal Component Analysis (PCA) was conducted on the 20 items of the questionnaire with Oblimin rotation. The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis (KMO=.90) and all KMO values for individual items were >.75. Bartlett's test of sphericity ($\chi^2(190)=1434.11$, $p<.001$) indicated that correlations between items were sufficiently large for PCA. Three components had eigenvalues over Kaiser's criterion of 1. The scree plot showed an inflexion that would justify retaining 1 component. Thus 1 component that explained 41.85% of the variance was retained in the final analysis. Table 5.3. shows the item loadings on that component. It seems that this component taps various aspects of social rejection.

5.3.2.2. The association between sensitivity to positive social situations and sensitivity to negative social situations

The total scores of the Positive Social Situations Scale and the Negative Social Situations Scale were positively correlated ($r=.69$, $p<.01$; Table 5.1.).This unexpected

positive association suggests that participants who were likely to invest a higher number of points in experiencing positive social situations were also likely to invest a higher number of points in avoiding negative social situations.

5.3.2.3. *Associations with related measures of the experience of pleasure/ anhedonia*

Pearson's coefficients of correlations between the two scales' total scores and related measures of the experience of pleasure/anhedonia are presented in Table 5.1.

The Positive Social Situations Scale

The total score of the Positive Social Situations Scale was associated with scores on established measures of the experience of pleasure/anhedonia in the expected direction (SHAPS: $r=-.22$, $p<.01$; Children's Pleasure Scale: $r=.36$, $p<.01$; PES enjoyability: $r=.26$, $p<.01$; PES combination score: $r=.34$, $p<.01$). This suggests that those who were likely to invest a higher number of points to experience positive social situations were less anhedonic, and reported higher levels of pleasure in rewarding experiences.

The Negative Social Situations Scale

The total score of the Negative Social Situations Scale was associated with scores on measures of anhedonia/experience of pleasure in the unexpected direction (SHAPS: $r=-.23$, $p<.01$; Children's Pleasure Scale: $r=.27$, $p<.01$; PES enjoyability: $r=.20$, $p<.05$; PES combination score: $r=.21$,$p<.05$), which suggests that those who were likely to invest a higher number of points to avoid negative social situations were less anhedonic, and reported higher levels of pleasure in rewarding experiences.

Table 5.2. The Positive Social Situations Scale: Item loadings on questionnaire components and means for individual items

Item (All items begin with "How many points would you spend")	Component 1: Social status and peer acceptance	Component 2: Forming close relationships and nurturance	Mean rating for item (sd)
14. for someone to compliment you on how you look?	.88		49.72 (30.40)
16. to be praised by someone you admire?	.80		60.77 (31.60)
19. to get praised by a teacher?	.77		43.70 (31.02)
3. for people at your class to look up to you?	.71		45.74 (30.60)
15. to make a new friend?	.65		54.29 (30.37)
13. to win a competition in something that you are interested in at school?	.65		60.08 (30.86)
18. to feel that your classmates like you?	.64		57.41 (29.99)
2. to think you are an interesting person?	.62		49.39 (27.36)
17. to talk on the phone with a friend?	.57		46.17 (28.73)
20. to get the highest grade in your class in an important test?	**.55**		**77.01 (29.19)**
8. to go to a family meal?			65.44 (28.81)
4. to make someone that you care about feel happy?		**.84**	**81.67 (23.12)**
5. to have a heart to heart conversation with someone close?		.74	61.79 (28.44)
12. to make a friend who is sad feel better?		**.74**	**84.06 (20.18)**
11. for someone that you care about to tell you that they love you?		**.60**	**67.85 (30.11)**
6. to have a friend suggest you go out together to do something fun?		.58	66.39 (26.38)
9. to feel really understood by someone else when you are upset about something that happened to you?		**.54**	**72.30 (26.92)**
10. for a close friend to ask you for your advice?		.50	58.09 (28.70)
7. for someone you like to call and ask you out on a date?		.49	57.52 (33.82)
1. to go out to a party with your friends?		.44	60.54 (27.77)

Note: only loadings above .4 are presented. The five items with the highest mean ratings are in bold.

Table 5.3. The Negative Social Situations Scale: Item loadings on questionnaire components and means for individual items

Item(All items begin with "How many points would you spend to avoid")	Component: Social rejection	Mean rating for item(sd)
17. a situation where someone is being disrespectful towards you?	.76	52.84 (34.31)
13. having a friend turn you down when you invite them to go see a movie?	.76	48.38 (30.71)
14. a situation where a good friend of yours chooses to go out with someone else instead of you on a	.74	56.12 (31.05)
7. a situation where you ask a friend to help you with a difficult task and they say no?	.74	49.90 (32.38)
15. a situation where a friend stops returning your calls?	.74	48.57 (32.28)
10. being criticized by a classmate in front of others?	.73	65.44 (33.68)
8. a situation where other people ignore your opinion in a conversation?	.72	58.64 (31.10)
16. feeling that somebody that you know doesn't like you?	.68	50.28 (34.27)
18. losing a competition in school about something you are interested in?	.66	55.07 (32.80)
19. getting teased over your physical appearance?	**.66**	**69.27 (34.67)**
11. a situation where someone that you have been dating for a couple of weeks breaks up with you?	.65	56.15 (34.7)
12. a situation where you get the lowest grade of all your friends in a test you did?	.63	62.14 (34.41)
20. getting told off by a teacher over something bad that you did?	.62	53.09 (34.16)
6. overhearing somebody say something bad about you?	.60	47.6909 (37.51)
9. going to a party and not finding anyone to talk with?	**.60**	**68.53 (29.52)**
1. a situation where a friend tells you that they don't want to see you anymore?	**.55**	**75.12 (29.44)**
3. not being invited to a party that all your friends have been invited to?	.55	57.99 (31.19)
4. being a part of a conversation on a topic you know nothing about?	.48	40.03 (31.96)
2. a situation where you ask out someone that you like and they turn you down?	**.48**	**72.86 (30.88)**
5. having an argument with a close friend?	**.47**	**75.15 (28.15)**

Note: only loadings above .4 are presented. The five items with the highest mean ratings are in bold.

5.3.3. Associations with measures of social functioning and engagement in extra-curricular activities

Pearson's coefficients of correlations between the questionnaire total scores and measures of social functioning and engagement in extra-curricular activities are presented in Table 5.1.

The Positive Social Situations Scale

The total score of the Positive Social Situations Scale was correlated in the expected direction with friendship quality (r=.23, p<.01) and engagement in extra-curricular activities (r=.20, p<.05) but not with SDQ peer problems subscale (r=.12, p=.13). This indicates that participants with higher scores on the Positive Social Situations Scale were more likely to report greater satisfaction with their friendships and display greater engagement in extra-curricular activities, but did not report fewer peer relationship problems.

The Negative Social Situations Scale

The total score of the Negative Social Situations Scale was correlated in the unexpected direction with friendship quality (r=.24, p<.01). No significant associations were observed between the total score on this scale and engagement in extracurricular activities (r=.06, p=.46) or SDQ peer problems subscale (r=.10, p=.20). This suggests that participants who invested more points to avoid negative situations were also likely to report greater satisfaction with their friendships and did not report lower engagement in extra-curricular activities or greater problems in peer relationships.

5.3.4. Associations with depressive symptoms and gender differences

5.3.4.1. Associations with depressive symptoms

For the entire sample, the total score on the Positive Social Situations Scale was not significantly associated with depressive symptoms (r=.05, p=.52; Table 5.1.). This indicates that in contrast to my hypothesis, participants with greater sensitivity to positive social situations did not report fewer depressive symptoms. For the entire sample, the total score on the Negative Social Situations Scale was not significantly associated with depressive symptoms (r=.05, p=.55). This indicates that in contrast to my hypothesis,

participants with greater sensitivity to negative social situations did not report a higher rate of depressive symptoms.

5.3.4.2. Gender differences

Gender differences in sensitivity to positive social situations and sensitivity to negative social situations

Examining the difference between the investment in experiencing positive social situations and the investment in avoiding negative social situations separately for males and females, revealed that males were likely to invest more points to experience positive social situations (M= 57.77, SE= 2.05) than to avoid negative social situations (M= 52.26, SE= 2.32), t(84)=3.11, p<.05. In contrast, females invested a similar number of points to experience positive social situations (M= 64.42, SE= 1.74) and to avoid negative social situations (M= 64.70, SE= 2.32), t(78)=-.16, p=.87. Females (M= 64.42, SE= 1.74) were likely to invest a higher number of points to experience positive social situations compared to males (M= 57.77, SE=2.05, t (160) =-2.45, p<.05) and to avoid negative social situations compared to males (females: M= 64.70, SE= 2.32; males: M= 52.68, SE= 2.33; t(161) = -3.64, p<.001).

Following the finding suggesting two underlying components for sensitivity to positive social situations (i.e. forming close relationships and nurturance, social status and peer acceptance) and based on previous research suggesting that affiliation in the form of close interpersonal relations may be of greater importance to females than males (Davey et al., 2008; Kendler & Gardner, 2014; Rudolph, 2002), it was hypothesised post hoc that females will have higher scores than males on the component of forming close relationships and nurturance, but not necessarily on the component of social status and peer acceptance. A comparison of mean component scores for males and females revealed that females (M= .26,SE= .09) had significantly higher component scores of forming close relationships and nurturance than males (M= -.23,SE=.12; t(147.56)=-3.26, p<.01), but not significantly higher scores than males on the component of social status and peer acceptance (females: M= .10, SE= .11; males: M= -.10; SE= .11; t(157)=-1.24, p=.22).

Gender differences in the associations of sensitivity to positive social situations and sensitivity to negative social situations with depressive symptoms

The Positive Social Situations Scale

When examining the association between sensitivity to positive social situations and depressive symptoms by gender results indicated that for males, there was a positive, non-significant association (r=.17, p=.11) whereas for females there was a negative, non-significant association (r=-.17, p=.14). To examine the effect of gender further, the interaction between sensitivity to positive social situations and gender was tested in a hierarchical linear regression. Results of the regression analysis are presented in Table 5.4.A. As presented in Table 5.4.A, step 2, there was a significant positive main effect of gender on depressive symptoms (β=.22, p<.01) indicating that females reported a higher number of depressive symptoms. There was also a significant positive main effect of age (β=.21, p<.05), suggesting that older participants reported a higher number of depressive symptoms. There was a significant interaction effect of the total score on the Positive Social Situations scale and gender (β=-.20, p<.05). Simple slopes analysis was used to follow up the significant interactive effect between the Positive Social Situations scale total score and gender on depressive symptoms. As illustrated in Figure 5.1., this analysis revealed that sensitivity to positive social situations moderated the association between gender and depressive symptoms such that gender and depressive symptoms were positively associated in those with lower sensitivity to positive social situations (b= 10.09, t= 3.31, p<.01), but no significant association between gender and depressive symptoms was observed in those with higher sensitivity to positive social situations (b= 1.32, t= 0.46, p= 0.64). This suggests that females were more likely have more depressive symptoms than males only among participants who showed lower sensitivity to positive social situations.

The negative Social Situations Scale

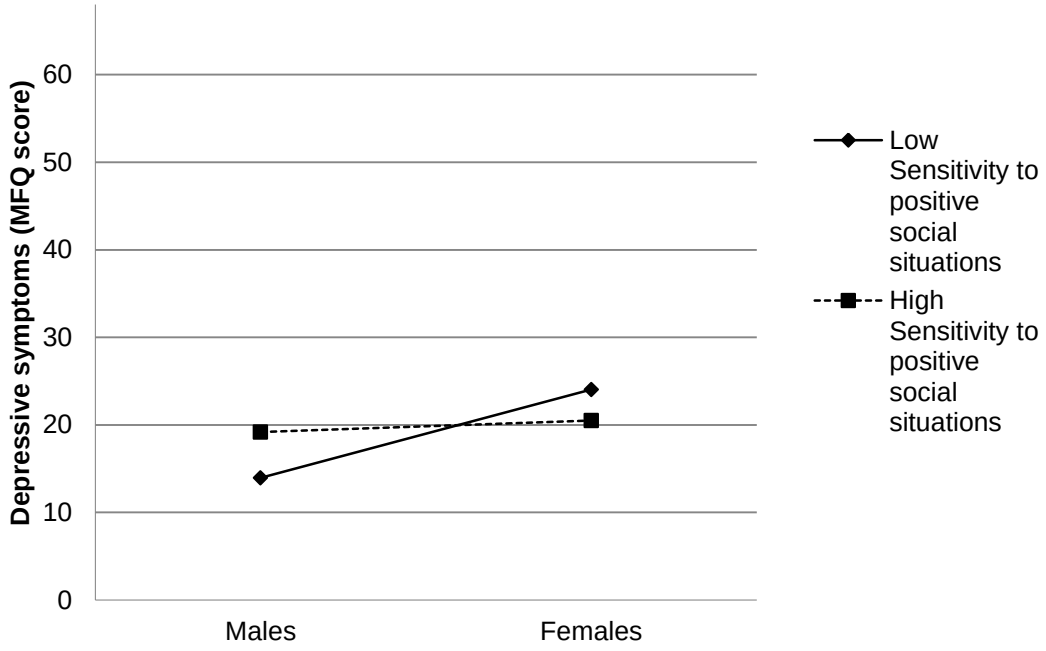
When examining the association between sensitivity to negative social situations scores and depressive symptoms by gender, results indicated that for males, there was a positive, non-significant association (r=.14, p=.21) whereas for females there was a negative, non-significant association (r=-.16, p=.16). To examine the effect of gender further, the interaction between sensitivity to negative social situations and gender was tested in a hierarchical linear regression. Results of the regression analysis are presented

in Table 4B. As presented in Table 5.4.B, step 2, similar to the regression analysis for the Positive Social Situations Scale, there was a significant positive main effect of gender on depressive symptoms (β=.22, $p<.01$) and a significant positive main effect for age (β=.20, $p<.05$). There was also a trend level interaction between the total score on the Negative Social Situations scale and gender (β=-.20, $p=0.06$).

Table 5.4. The main effects of total scores on the Positive/Negative Social Situations Scale, gender and their interactive effect on depressive symptoms

A. Sensitivity to positive social situations as a predictor				B. Sensitivity to negative social situations as a predictor			
	B (S.E.)	**β**	**p**		**B (S.E.)**	**β**	**p**
Step 1				**Step 1**			
Gender	5.49 (2.11)	.21	<.05	Gender	5.61 (2.16)	.21	<.05
The Positive Social Situations Scale	.94 (1.08)	.07	.39	The Negative Social Situations Scale	.59 (1.11)	.04	.59
Age	2.81 (1.08)	.21	<.05	Age	2.62 (1.08)	.20	<.05
Step 2				**Step 2**			
Gender	5.72 (2.09)	.22	<.01	Gender	5.76 (2.15)	.22	<.01
The Positive Social Situations Scale	2.61 (1.35)	.20	.06	The Negative Social Situations Scale	2.36 (1.45)	.18	.11
Age	2.74 (1.06)	.21	<.05	Age	2.60 (1.07)	.20	<.05
The Positive Social Situations Scale x Gender	-4.37 (2.15)	-.20	<.05	The Negative Social Situations Scale x Gender	-3.97 (2.13)	-.20	.06

Figure 5.1. The interactive effect of the total score on the Positive Social Situations Scale and gender on depressive symptoms



Note: Low and high scores on The Positive Social Situations Scale were plotted as the sample mean ± one standard deviation.

Males were coded as 0, females were coded as 1.

5.3.5. Social situations rated as the most rewarding and the most aversive

Mean ratings and standard deviations for each positive social situation are resented in Table 5.2. The 5 top rated items are marked bold. An examination of top rated items suggests that the majority of social situations that participants rated as most rewarding were related to forming close relationships and nurturance. Mean ratings and standard deviations for each negative social situation are resented in Table 5.3. The 5 top rated items are marked bold. Although the PCA conducted for the Negative Social Situations Scale suggested that items loaded on one component representing various aspects of social rejection, an examination of the top rated items on this scale somewhat mirrored the preferences for the positive social situations in the sense that a large proportion of the situations participants found to be the most aversive are related to experiencing conflict or rejection by someone close or desirable.

5.4. Discussion

This study aimed to develop and validate a new measure assessing sensitivity to positive and negative social situations in adolescents. The reliability and validity of the measure were assessed by examining: the internal reliability of each of the two scales, their internal structure, the association between sensitivity to positive social situations and sensitivity to negative social situations, and the associations of both with related measures used to assess anhedonia and the experience of pleasure that have been used in previous studies of children and adults. I then examined whether these two constructs are associated with social functioning, engagement in extra-curricular activities and depressive symptoms. Gender differences in sensitivity to positive social situations and negative social situations and in the association of these constructs with depressive symptoms were also examined.

This is the first measure developed to assess the rewarding value of positive social situations and the aversive value of negative social situations by asking participants to choose how many points they were willing to "invest" in order to experience positive social situations or to avoid experiencing negative social situations. It was hypothesised that this method would allow "quantification" of the value of a range of positive and negative social situations, in a similar way to the assessment conducted by performances based reward measures. The response scale of the new measure (which focuses on quantifying the

rewarding value of social situations using points) was designed to be less subjective than the response scales used in existing questionnaires of the experience of pleasure/anhedonia and so might be less susceptible to mood and social desirability biases. This type of assessment also allowed quantification of the value of positive and negative social situations on an equivalent scale.

Both questionnaire scales had excellent internal reliability, as indexed by Cronbach's alpha which provided evidence of high internal consistency. An examination of the structure of the Positive Social Situations Scale suggested two underlying components for this scale: 1) social status and peer acceptance 2) forming close relationships and nurturance. This structure is consistent with several previous studies which suggest that two main dimensions or goals underlie positive affect in interpersonal relations: agency and affiliation/intimacy. Agency involves the enjoyment derived by exerting influence over others and demonstrating leadership in social contexts, whereas affiliation/intimacy involves having close affectionate relationships with others (Davey et al., 2008; McAdams, 1985, 1988). Examination of the structure of the Negative Social Situations Scale suggested one main component underlying the items of this scale. This component is suggested to represent various aspects of social rejection. Furthermore, an examination of the items showing the highest ratings for each scale suggested that the social situations participants found to be the most rewarding were mostly related to forming close relationships, and the social situations participants found to be the most aversive were mostly related to experiencing conflict or rejection in close or important relationships.

With regard to the hypothesis of an inverse association between sensitivity to positive social situations and sensitivity to negative social situations, findings actually revealed a strong positive association between the two constructs in this sample. Although this finding may be contrasted with theoretical models suggesting that increased sensitivity to negative social experiences may interfere with reward seeking (Davey et al., 2008; Silk et al., 2012), it is consistent with literature suggesting that adolescence is characterised by high salience of the social arena, which may be manifested in enhanced sensitivity to social rewards as well as in enhanced sensitivity to social stressors (Davey et al., 2008; Foulkes & Blakemore, 2016; Silk et al., 2012). Alternatively this association may also be related to a response bias of the participants, meaning that some participants may have a tendency to provide consistently high or low ratings in questions using a Likert scale.

There were significant associations in the expected direction between sensitivity to positive social situations and related measures of the experience of pleasure/anhedonia

(SHAPS, Children's Pleasure Scale and PES enjoyability and combination scores). These associations further establish the construct validity of the newly developed questionnaire. There were also significant associations between sensitivity to negative social situations and measures of the experience of pleasure/anhedonia (SHAPS, Children's Pleasure Scale and PES enjoyability and combination scores), suggesting that those who invested more points to avoid negative social situations were also likely to report lower levels of anhedonia and higher levels of pleasure from rewarding experiences. Although this result may be surprising as it stands in contrast with previous studies showing associations between sensitivity to negative events and depression (Grosscup & Lewinsohn, 1980; Joffe et al., 1993; Schless et al., 1974), as mentioned, in this sample, sensitivity to positive social situations and sensitivity to negative social situations were strongly associated and thus sensitivity to negative situations may represent an aspect of general sensitivity to social experiences which would not necessarily be associated with anhedonia.

The findings mostly supported the hypothesised association between sensitivity to positive social situations and social functioning and engagement in extra-curricular activities. Findings indicated that investment in positive social situations was significantly associated with satisfaction with friendships and engagement in extracurricular activities but not with ratings of peer problems. This suggests that, as expected, those with greater sensitivity to positive social situations may be more likely engage more frequently in extra-curricular activities and experience greater satisfaction with their friendships. However, sensitivity to positive social situations was not associated with peer problems contrary to expectations. It may be beneficial in future studies to examine further how sensitivity to rewarding social experiences may be associated with distinct aspects of social functioning.

In contrast to my hypothesis regarding the association between sensitivity to negative social situations and social functioning, the findings indicated that higher sensitivity to negative social situations was associated with greater satisfaction with friendships. Again, this may be explained by the strong association found in this study between sensitivity to positive social situations and sensitivity to negative social situations. Moreover, it is reasonable to assume that in typically developing adolescents, trying to avoid negative social experiences to some extent may be adaptive and contribute to positive social functioning. This suggestion is supported by a study by Jarvinen & Nicholls (1996) that examined social goals in adolescents and found that avoidance of negative experiences, such as being teased and being rejected by peers, served as one of the factors underlying social goals in adolescents. However, as the current study found a

different association than that suggested by previous literature (Davey et al., 2008; Silk et al., 2012), this should be examined in future studies.

An examination of associations between sensitivity to positive or negative social situations and depressive symptoms revealed no significant associations between these constructs and depressive symptoms. This was unexpected in light of previous research indicating that lower reward-seeking behaviour under conditions of high probability for gain (as assessed by performance-based measures) is associated with the presence of current depressive disorder in adolescents (Forbes et al., 2007; Rawal et al., 2013) and that greater sensitivity to negative events and risk aversion is positively associated with the presence of current depressive disorder in adults (Grosscup & Lewinsohn, 1980; Joffe et al., 1993; Lewinsohn et al., 1973; Lewinsohn & Talkington, 1979; Schless et al., 1974; Smoski et al., 2008). It is possible that the lack of associations between these constructs and depressive symptoms in the entire sample may be related to methodological issues such as the measurement strategy employed in this study. For example, it is possible that the new measure does not capture the same element of reward processing as that assessed by performance-based measures. Furthermore, in light of the lack of association shown in this study between the scores in the negative social situations scale and depressive symptoms and the positive association found between scores on this scale and friendship quality, it is possible that this measure may not have tapped heightened sensitivity to negative social situations, which is suggested to be associated with depression, but perhaps tapped indifference to negative social situations. It may be beneficial in future studies to examine which aspects of sensitivity to negative social situations may be associated with depression. Another possible explanation for these findings is that higher sensitivity to positive social situations and lower sensitivity to negative social situations as assessed here may not have a linear association with depression but may act as a protective factor under conditions of stress or adversity. Finally, sensitivity to positive social situations or to negative social situations may not have concurrent effects on depressive symptoms, but may instead be associated with change in depressive symptoms over time.

Substantial gender differences were observed in sensitivity to positive social situations, sensitivity to negative social situations and the association between sensitivity to positive social situations and depressive symptoms. First, an examination of gender differences in sensitivity to positive social situations and to negative social situations indicated that males were likely to invest more points to experience positive social

situations than to avoid negative social situations, whereas females were likely to invest approximately the same number of points in both. When comparing males and females, females were likely to invest more points than males both to experience positive social situations and to avoid negative social situations. These findings are somewhat in line with previous research which suggests that during adolescence, females develop stronger affiliation needs than males (Cyranowski et al., 2000) and are also more sensitive to interpersonal conflict and social stress (Rudolph, 2002). Thus, as portrayed by this study, compared to males, females may attribute greater importance to experiencing positive social experiences as well as to avoiding negative social experiences. An examination of gender differences in the association between sensitivity to positive social situations and depressive symptoms revealed a main effect for gender (females had more symptoms) as well as a significant interaction between gender and sensitivity to positive social situations. The observation that females had higher rates of depressive symptoms in this study is consistent with a large body of research on adolescent depression (Piccinelli & Wilkinson, 2000; Rudolph, 2002; Thapar et al., 2012). An investigation of the observed interactive effect suggested that sensitivity to positive social situations moderated the association between gender and depressive symptoms such that higher rates of depressive symptoms in females compared to males were observed among participants with low sensitivity to positive social situations but not in those with high sensitivity to positive social situations. One possible explanation for this finding is that, due to the pivotal role social affiliation plays in females' wellbeing, reduced sensitivity to positive social situations or impairment in acknowledging or identifying opportunities to pursue and experience social rewards may put females at increased risk for developing depressive symptoms. However, as this was a cross sectional study it is also possible that reduced sensitivity to positive social situations may be a result of depressive symptoms, although it is not clear why this effect may be more prominent in females than males.

Some limitations of this study are worth noting. Firstly, as mentioned, this was a cross sectional study. Thus, it did not assess longitudinal associations between sensitivity to positive social situations or to negative social situations and future anhedonia, social functioning, engagement in extra-curricular activities and depressive symptoms, as has been shown in previous experimental studies of reward processing (Chapter 1). Thus, additional studies are needed to examine such longitudinal associations and investigate whether reduced sensitivity to positive social situations or increased sensitivity to negative social situations over a period of time may serve as a risk factor for social impairment and

depressive symptoms. It is important also to note some characteristics of the new measure that may have an impact on the interpretation of results.

During the development of the measure, I aimed to represent a broad range of positive and negative social situations that were suggested by the literature to be important for adolescents. However, no direct comparison of the affective value of the positive and negative social situations was conducted, meaning that it was not specifically examined whether the positive items were as "positive" as the negative items were "negative" in terms of the affective magnitude of items.

This is the first study that used a paradigm aimed at quantifying the rewarding/aversive value of a range of positive and negative social situations with the investment of points. In terms of the specific aspects of reward processing that were assessed here, it is plausible to assume that this measure mostly taps the rewarding/hedonic value of positive situations and the aversive value of negative situations (Deaner et al., 2005; Hayden et al., 2007; Tamir & Mitchell, 2012). The request to invest points according to the rewarding/aversive value of situations may have also tapped some aspect of motivation to experience or to avoid certain social situations. However, this measure relies on self-report and is not a purely behavioral measure of motivation or willingness to exert effort to experience positive social situations or avoid negative social situations (Sherdell et al., 2012; Treadway et al., 2012). Some studies have suggested that depressed individuals may not necessarily differ from non-depressed individuals in their ability to experience pleasure, but rather in their motivation and willingness to exert effort to pursue rewarding experiences (Sherdell et al., 2012; Treadway et al., 2012). Thus, it seems that examining motivation and the ability to exert effort to experience positive social situations in future studies may be beneficial for research of depression in adolescents.

Although assessing reward processing using performance-based measures may provide a more objective measure of real life behaviour, I chose to employ in this study a measure that relies on self-report. Beside the lack of reliable measures to assess social reward processing in adolescents, this type of assessment was also chosen because it is plausible to assume that subjective reports are important when assessing the rewarding/aversive value of social situations (Foulkes & Blakemore, 2016). Furthermore, a self-report measure allows a wide range of social situations to be assessed, whereas a performance-based measure would usually require focusing on a small number of specific stimuli to represent social rewards. However, it should be noted that subjects may have interpreted the instructions or items of the new measure in different ways. For example, in

item 16 of the negative social situations scale ("How many points would you spend to avoid feeling that somebody that you know doesn't like you?"), it is possible that there is room for interpretation with regards to whether the subject is "paying" for this not to happen or for him/her not to know about it. Furthermore, this measure employed points to quantify the subjective importance of social situations, as explained in the instructions. However, it is possible that some subjects may have interpreted the use of points in a different way. For example, in item 15, in the positive social situations scale ("how many points would you spend to make a new friend?"), some subjects may have interpreted this as "paying" for someone to be your friend. Thus, different interpretations of the questionnaire may have affected the subject's responses and therefore the pattern of associations between the study variables. It may be beneficial in future studies to discuss with participants the interpretation of the instructions and items following the completion of the questionnaire in order to assess the validity of using a points-based approach as a method of quantifying the subjective importance of social situations.

This measure may have utility in identifying adolescents with low sensitivity to socially rewarding situations and can help to delineate reward-based difficulties in young people. Such low sensitivity may be driven by low hedonic capacity, by difficulty in identifying opportunities for rewarding experiences, or by reduced expectations of experiencing pleasure in social situations. Either way, low sensitivity to social reward may influence adolescents' abilities to use social opportunities to experience positive affect and may increase risk for anhedonia, social impairment and depressive symptoms. Recent evidence suggests that behavioral interventions for depression, such as behavioral activation (which encourages patients to engage in rewarding activities) may be as effective as cognitive interventions (which aim to change maladaptive cognitions) in treating depression in adults and young people (Hetrick et al., 2015; Richards et al., 2016). Focusing on behavioral treatments may be especially beneficial in young people as they may struggle with the metacognitive demands of cognitive therapy-based approaches for treating depression (Siegle et al., 2007; Stott & Salkovskis, 2010).

CHAPTER 6: General discussion

6.1. Summary of main findings

This thesis aimed to examine the role of three cognitive processes (i.e. executive functioning, specificity of autobiographical memory, and reward processing) as risk and protective factors for the development of depression, primarily in offspring of depressed parents. Here I describe the main research aims and the key findings of the thesis.

Chapter 2 aimed to explore the buffering role of two components of executive functioning, inhibitory control and mental flexibility, in the association between a current parental depressive episode and adolescent depressive symptoms. This was examined in a high risk sample of adolescent offspring of parents with a history of MDD. First, findings of this study suggested that the presence of a current parental depressive episode was positively associated with adolescent depressive symptoms, meaning that adolescents exposed to a current episode in the parent had higher rates of depressive symptoms. The presence of a current parental depressive episode was not associated with adolescent executive functioning. Second, the main findings of the study indicated that in the presence of a current depressive episode in the parent, adolescents with better inhibitory control and mental flexibility had fewer depressive symptoms compared to those with poorer inhibitory control and mental flexibility. Hence, this study suggested that executive functions may serve as a protective factor for high risk adolescents exposed to a current parental depressive episode.

The main aim of Chapter 3 was to examine whether the level of specificity of autobiographical memory, assessed as OGM, is prospectively associated with mental health resilience and social functioning in adolescents. As a secondary aim, this study examined whether the influence of OGM was specifically observed for resilience to mood symptoms compared to resilience related to behavioural symptoms. As a third aim, this study examined whether proximal environmental risk factors, such as current severity of parental depressive symptoms and recent negative life events are associated with OGM. Finally, this study explored whether OGM served as a mediator of the association between the aforementioned environmental risk factors and mental health resilience/social functioning. This was examined using a longitudinal design in a high risk sample of adolescent offspring of parents with a history of MDD. Mental health resilience was assessed as better than expected mood/behavioural symptoms given exposure to familial risk.

The main findings from this chapter indicated that OGM in response to negative cues predicted resilience to mood symptoms, but not resilience to behavioural symptoms over time, such that adolescents that retrieved more overgeneral memories in response to cue words had lower resilience to mood symptoms. OGM, primarily in response to negative cues, also predicted social functioning over time, such that adolescents that retrieved more overgeneral memories in response to negative cue words had lower quality of peer relationships. Furthermore, the findings of this study suggested that adolescents who experienced more recent negative life events retrieved more overgeneral memories, primarily in response to negative cues. No association was observed between current severity of parental depressive symptoms and adolescent OGM. Finally, it was revealed that OGM in response to negative cues partially mediated the association between recent negative life events and social functioning. Therefore, this study suggested that OGM may affect resilience to mood symptoms and social functioning and thus that high specificity of autobiographical memory may serve as a protective factor for high risk adolescents. Furthermore, the study suggested that exposure to recent negative life events may be associated with the emergence of OGM in high risk adolescents and that overgeneral memories in response to negative cues, may mediate the effect of negative life events on later social functioning.

Chapters 4 and 5 focused on exploring social reward processing in the context of familial risk for depression, depressive symptoms and anhedonia.The first aim of chapter 4 was to examine whether familial risk for depression is associated with reward learning in a social context in young adults and whether any association between familial risk and reward learning may be attributable to anhedonia/current depressive symptoms. The second aim of the study was to examine whether reward learning in a social context is associated with social functioning. This was examined in a cross sectional experimental design in a sample of young adult offspring of parents with a history of MDD and parents with no history of MDD. Familial risk was defined as the number of depressed parents. Reward learning was assessed as the ability to differentiate between partners with different tendencies for reciprocity in the context of social exchanges in an experimental task. Findings of this study indicated that familial risk for depression and anhedonia were associated with different patterns of reward learning. Specifically, greater familial risk was associated with a larger differentiation between a fair (rewarding) and a neutral player, and a smaller differentiation between a neutral and an unfair player. Anhedonia, on the other hand, was associated with a learning pattern characterised by a smaller differentiation

between the fair and neutral players and a slower learning rate for the fair player, expressed as a slower change in investment behaviour when playing with the fair player. Furthermore, findings suggested that a lower learning rate for the fair player was associated with poorer social functioning for those with greater familial risk. In summary, this study provided evidence of associations between familial risk for depression and anhedonia and reward learning in a social context. Additionally, findings also demonstrated that difficulties in reward learning in a social context may increase social impairment in those at high familial risk. Thus, adaptive reward learning may have a protective role on social functioning for those at high familial risk.

Chapter 5 focused on exploring sensitivity to social rewards in adolescents and examining sensitivity to negative social situations as a potentially related construct. Given the lack of measures assessing sensitivity to positive (rewarding) and negative social situations in adolescents, this study first aimed to develop and validate a questionnaire measure to assess these constructs in adolescents. Second, this study aimed to examine whether sensitivity to positive social situations is associated with sensitivity to negative social situations and whether these constructs are associated with indices of social functioning, engagement in extra-curricular activities and depressive symptoms in adolescents. Additionally this study aimed to examine gender differences in sensitivity to positive and negative social situations, as well as gender differences in the associations between these constructs and depressive symptoms, following previous research suggesting gender differences in social relationships in adolescents and associating these with gender differences in depression rates during adolescence (Davey et al., 2008; London et al., 2007; Rudolph, 2002; Thapar et al., 2012).This was examined in a cross sectional design in a community sample of adolescents aged between 11 and 15. This questionnaire aimed to assess individual differences in the rewarding value of positive social situations and the aversive value of negative social situations using points. First, the findings of this study indicated that the questionnaire developed had good reliability and construct validity, as assessed by examining the internal structure of the questionnaire and associations with established measures of the experience of pleasure/anhedonia. Second, the findings suggested that adolescents who showed greater sensitivity to positive situations, also showed greater sensitivity to negative social situations, better social functioning as assessed by greater self-reported satisfaction of friendships and higher engagement in extra-curricular activities. Furthermore, findings indicated that gender was associated with both sensitivity to positive social situations and sensitivity to negative

social situations such that females showed greater sensitivity to both according to the questionnaire measure developed. As expected in an adolescent sample, gender was also associated with depressive symptoms, such that females reported higher rates of depressive symptoms than males. Sensitivity to positive social situations was not significantly associated with depressive symptoms for the entire sample but served as a moderator of the association between gender and depressive symptoms, such that among those with high sensitivity to positive social situations there was no significant association between gender and depressive symptoms, whereas among those with low sensitivity to positive social situations, females had higher rates of depressive symptoms. Thus, this study highlighted that the measure developed to assess sensitivity to social rewards showed convergent validity as it was associated with other well-established measures of anhedonia and that sensitivity to social rewards may be positively associated with adaptive social functioning. Results also suggested that low sensitivity to social rewards may be particularly associated with depressive symptoms in adolescent females.

6.2. Limitations

This thesis contributes to a greater understanding of cognitive processes that may serve as risk and protective factors for the development of depression. Nevertheless, there are a number of limitations to this work that are discussed in the following section.

This thesis employed a combination of data collection methods such as clinical interviews, performance based measures, and questionnaire data. Familial risk and indices of psychopathology were assessed using clinical interviews or questionnaires (depending on the sample) whereas cognitive processes were assessed primarily through performance based measures. This is an important strength of the thesis as more objective assessments of cognitive processes were employed which helped reduce the risk of common method bias that may occur, for example, when using self-report measures to assess both the risk factor and outcome (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003). However, it is important to acknowledge that some elements of cognitive processing (such as the rewarding value of social experiences) may require subjective report and therefore employing a combination of performance based measures and self-report measures is likely to be the most useful for exploring cognitive processes in the context of risk for depression(Foulkes & Blakemore, 2016; Jacobs et al., 2008).Furthermore, some limitations of performance based measures are worth noting. Specifically, information about reliability and validity of experimental tasks that are

commonly used to assess cognitive processes is often lacking. In term of construct validity, it is not always clear which theoretical construct each task taps into. Moreover, associations between these tasks and related constructs are not always established (Jacobs et al., 2008; Vasey, Dalgleish, & Silverman, 2003). For example, although I assessed associations between anhedonia and performance in the trust game task that was used to assess reward learning in a social context (chapter 2), it is not clear how performance in this task is associated with reward learning in a non-social context or with other components of reward processing. However it is worth mentioning that a previous neuroimaging study which employed this task (Phan et al., 2010) showed that activation in some reward related brain areas occurred specifically in response to reciprocal interactions with the fair player which suggests some evidence of convergent validity for the task. Furthermore, another possible limitation of performance based measures is that in order to ensure reliability and validity of such measures, the conditions of administration need to be carefully controlled and standardized (Vasey et al., 2003). As previously described, assessments of the EPAD sample (employed in chapters 2 and 3) were conducted in families' homes and although this is common practice in cohort studies, it may have introduced factors (i.e. noise or distractions) that may have affected the reliability of the tasks administered.

Another methodological limitation that is important to mention is related to the assessment of parental depression. This thesis employed two methods to assess parental depression: clinical interviews with parents (chapters 2 and 3) and the family history method (Andreasen et al., 1977) in which participants were interviewed about their parents (chapter 4). Although receiving direct diagnostic information from parents is obviously a preferable method of assessing parental depression, as it was not feasible to directly assess parents in the sample of young adults employed in chapter 4, the family history method was chosen to assess familial risk for depression. Although this method is often used to assess family history of mental health disorders in adults (Colvin et al., 2014; Klein et al., 2005; Lieb et al., 2002; Weissman et al., 2000) and was found to be valid and reliable (Andreasen et al., 1977; Weissman et al., 2000), studies report varying rates of sensitivity and specificity for identification of psychopathology in first degree relatives (Klein et al., 2005; Weissman et al., 2000). Furthermore, with regard to parental depression, sensitivity and specificity seem to be affected by the gender of the parent reported on and the presence of a history of MDD in the offspring. Thus, in a community study which assessed sensitivity and specificity of this method by comparing information

from offspring interviews to direct interviews with parents, Klein et al. (2005) reported that sensitivity was .45 for mothers and .32 for fathers and specificity was .85 for mothers and .90 for fathers , indicating greater sensitivity for mothers. Also, similar to results of other studies (Chapman, Mannuzza, Klein, & Fyer, 1994; Kendler et al., 1991), Klein et al. (2005) found that sensitivity tended to be higher and specificity tended to be lower in subjects with a history of MDD themselves. In the study described in chapter 4, high risk participants were recruited based on their awareness of parental depression which may have biased the sample. Furthermore, some of the high risk subjects had a history of MDD themselves. Thus it is possible that a mood-related rater bias affected how subjects reported parental depression and that a history of MDD in the subjects themselves reduced specificity of identification of parental depression. However, it is not clear whether this may have influenced the findings of the study in any consistent way.

This thesis focused on offspring of depressed parents, with the aim of increasing current understanding of risk and protective factors in this high risk group. As previously mentioned (chapter 1), such a focus is warranted given that offspring of depressed parents are three-to four times more likely to develop depression themselves compared to offspring of healthy parents and that depression in this high risk group is characterized by an earlier onset and a more recurrent and severe course compared to depression in community samples(Garber, 2006; Kovacs & Lopez-Duran, 2010; Lieb et al., 2002; Rice et al., 2002; Rice & Rawal, 2010; Weissman et al., 1997).However, this may limit the ability to generalize findings to other populations. Thus, some cognitive mechanisms may act as risk factors in vulnerable individuals, but not in general community samples. For example, whereas several studies found that overgeneral memory predicted future depression in high risk samples (Hipwell et al., 2011; Rawal & Rice, 2012) or predicted the course of disorder in depressed individuals (Sumner, Griffith, & Mineka, 2010),OGM did not predict depression over time, nor did it moderate the effect of life events on depression in a community sample of adolescents(Crane et al., 2016). Hence, Crane et al. (2016) have suggested that the presence of OGM may reflect different mechanisms in high risk and community samples and may not serve as vulnerability factor in the general population. However, it is also possible that the lack of a significant association between OGM and depression in such community samples is due to issues related to restricted range such as low risk exposure or low rates of psychopathology, and may not necessarily reflect a "true" lack of effect.

It is worth noting that as recommended by Rothman (1990, 2014), I did not correct for multiple comparisons in the thesis. Rothman has suggested that correcting for multiple comparisons may reduce the likelihood of type I errors at the cost increasing the likelihood of type II errors, which means failing to detect real associations. Thus, the strategy of correcting for multiple comparisons is not recommended unless it is likely to assume that the data represents random numbers rather than associations that exist in nature and are studied through observations (Rothman, 1990, 2014). The approach taken here is consistent with that taken in previous published studies in this area (Forbes et al., 2007; Hipwell et al., 2011; Kilford et al., 2015; Kyte et al., 2005). Nevertheless, replication of findings in independent data sets is likely to be important.

It is also important to note the limitations of study designs employed in this thesis, which limit the extent to which inferences about causality can be made based on these findings. It has been suggested by Mill (1843) that 3 conditions need to be met in order to infer causality: 1) a statistical association between a potential cause and outcome 2) the cause temporally precedes the outcome 3) alternative explanations for the association can be ruled out. It is important to note that the risk factors that I have chosen to examine in this thesis have been shown to precede the onset of depression in previous studies. However, chapters 2, 4 and 5 employ cross sectional designs which preclude temporal precedence of the risk factors examined and thus limits inferences about causality. Thus, for example, it is not possible to make a causal inference about the association between current parental depression and adolescent depressive symptoms in chapter 2. However, any theoretical inferences about causality in the association between parental depression and offspring depression were made based on a large body of previous research establishing parental depression as a prominent risk factor for offspring depression.

Chapter 3, on the other hand, employed a longitudinal design to assess associations between OGM and mental health resilience/social function, which allowed establishing temporal precedence of OGM to outcomes of mental health resilience/social functioning and controlling for baseline levels of outcome measures. However, the observational nature of this study (and the studies in chapters 2, 4 and 5) does not allow one to completely rule out the possibility that unmeasured factors (environmental or genetic) account for observed associations. Random allocation to experimental conditions is usually the common strategy used to rule out alternative explanations caused by unmeasured factors. However, it is not possible to conduct random allocation for high and low familial risk groups or for high and low performance groups in various cognitive

processes. Thus, this was addressed in all chapters by statistically controlling for variables that may be associated with predictors and outcomes.

Finally, it is worth referring to the findings of this thesis with regard to associations between indices of parental depression and offspring cognitive processes. In chapter 2, there was no evidence of an association between a current parental depressive episode and adolescent executive functioning and in chapter 3 there was no evidence of an association between severity of parental depressive symptoms and offspring OGM. It is important to mention that these associations were examined within a high risk sample where adolescents were exposed to parental depression during their lifetime and therefore low heterogeneity in exposure to parental depression may have reduced the power to detect such an effect. Thus, in order to establish whether parental depression affects children's cognitive processing, associations should also be examined in the context of community samples. Furthermore, these associations were examined cross-sectionally in a sample of adolescents and it is possible that parental depression had earlier effects on these cognitive processes or that such associations would be observed when examined over time.

6.3. Implications and future research

The findings of this thesis have a number of theoretical and clinical implications. In this section, I will review the implications of the results and provide recommendations for future research related to the specific cognitive processes explored in this thesis. I will then move onto discussing the broader implications related to the study of cognitive risk and protective factors for depression.

The findings with regard to executive functioning indicated that better executive functioning may serve as a protective factor for adolescents exposed to a current episode of parental depression. This finding was consistent with a large body of research, which suggests that executive functions are involved in emotion regulation and the control of negative emotions (Diamond, 2013; Joormann & Quinn, 2014; Roiser et al., 2012). Furthermore, it was consistent with previous studies which suggested that the protective effect of executive functioning may be especially observable when demands for emotional regulation are high, such as under conditions of stress or exposure to adversity (Bakker et al., 2011; Muris et al., 2008). As this study focused on a specific population and on exposure to a specific form of risk/adversity it may be beneficial to examine in future studies if the protective effects observed in this study can be generalized to other

populations, to other forms of exposure to adversity/stress and whether they may persist over time.

It will also be helpful to explore how executive functions may serve as a protective factor for high risk individuals and which mechanisms may mediate such protective effects. For example, do better executive functions convey their protective effect through increased use of adaptive mood regulation strategies, such as reappraisal or through reduced levels of rumination, or both? This has not been systematically examined by previous studies. Examining this may have important clinical implications as specific components of executive functioning may be differentially associated with other cognitive processes related to emotion regulation. Thus, elucidating such associations may further the understanding of the clinical importance of executive functions and clarify which components of executive functioning may be the most important to focus on targeting in preventive interventions.

The findings with regard to OGM extended previous research by showing that OGM not only predicts depressive disorder in high risk populations but may also predict positive outcomes such as resilience to mood symptoms and social functioning. As social difficulties have been found to be strongly associated with increased risk for depression (Baumeister & Leary, 1995; Feder et al., 2009; Rutter, 2013; Southwick et al., 2005), this suggests that effects of autobiographical memory on social functioning might be a mechanism through which OGM may influence resilience to depression. However, that possibility was not specifically tested in this study. Several potential mechanisms have been suggested by previous literature: using autobiographical memory to reflect on past experiences in order to cope with negative situations, using positive memories to repair mood, allowing better social problem solving or supporting concrete versus abstract thinking about problems (Dalgleish & Werner-Seidler, 2014; Rusting & DeHart, 2000; Watkins et al., 2008; Williams et al., 2007). Thus, similarly to the suggestions made with regard to executive functions, it may be beneficial to explore which of these possible mechanisms may mediate the effect of OGM on resilience to mood symptoms.

Furthermore, as it has been shown that specificity in the retrieval of autobiographical memories may rely on executive functioning (Dalgleish et al., 2007; Williams et al., 2007), future investigations may benefit from examining whether the specificity of autobiographical memory mediates the protective effect of executive functioning observed in depressive symptoms. This may have important clinical

implications, as enhancing executive functioning in interventions for high risk individuals could also have a beneficial effect on specificity of autobiographical memory.

The studies exploring reward processing in this thesis aimed to address a specific gap in this area: the lack of studies exploring associations between impairments in social reward processing and depression (Forbes, 2009; Mellick et al., 2015).The findings from Chapter 4 showed that familial risk for depression and anhedonia may have independent contributions in explaining individual differences in reward processing in a social context. Moreover, findings provided preliminary support for the role of adaptive reward learning in a social context as being associated with social functioning, which evidence suggests could act as a protective factor for high risk individuals (Feder et al., 2009; Rutter, 2013; Southwick et al., 2005). The study in chapter 5 contributed to existing research as it involved developing and validating a new measure assessing sensitivity to positive social situations and to negative social situations suitable for adolescents. Furthermore, this highlighted the potential importance of sensitivity to social rewards due to its associations with anhedonia and social functioning, and its moderating role in the association between gender and depressive symptoms. Whilst the cross-sectional design of this study requires findings to be interpreted with caution, these findings may provide initial support for the protective role of sensitivity to socially rewarding situations in adolescents, particularly in adolescent females. However, longitudinal studies are needed to establish that reduced sensitivity to social rewards indeed precedes the onset of depression and is not a consequence of depressive symptoms or anhedonia. Together, these two studies extended previous findings by examining elements of reward processing using social paradigms. As the pursuit of social rewards are inherently important for adaptive functioning, and have been shown to be important in depression, exploring reward processing using social paradigms in future studies is paramount to furthering the understanding of how reward processing may be involved in risk for depression.

With regard to all three cognitive mechanisms explored in this thesis (i.e. executive functioning, specificity of autobiographical memory and reward processing) it has been suggested throughout the thesis that there may be clinical potential in incorporating strategies to boost these processes in preventive interventions (Garber, 2006; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001; Rice & Rawal, 2010). However, additional support is needed to establish these as protective factors in future studies that: 1) explore the presence of difficulties related to these cognitive processes in high risk individuals 2)establish that these are not a proxy for other known vulnerability factors 3) employ

longitudinal designs and provide additional evidence to demonstrate that such impairments precede the onset of depression in both high risk groups and community samples (Kraemer et al., 2001; Mill, 1843). Furthermore, it may be beneficial to examine the potential of addressing such impairments related to these cognitive processes in clinical outcome trials and to examine whether change in these cognitive processes mediate change in depressive symptoms in preventive trials (Rice et al., 2015).

Another important issue related to the study of cognitive risk and protective factors for depression is worth noting. Studies in cognitive research of depression usually focus on a specific cognitive mechanism and explore its association with risk for depression. However, the large body of studies exploring different cognitive mechanisms separately does not clarify their relative contribution to increasing risk for depression. Thus, it may be beneficial to explore different cognitive mechanisms that are suggested to be causally involved in the development of depression in a risk prediction model that may help to assess the relative importance of various cognitive mechanisms in predicting depression (Moons et al., 2012) . This may assist in determining which cognitive mechanisms may be the most important to target in preventive interventions.

As discussed in chapter 1, this thesis focused on exploring three cognitive processes as risk and protective factors for the development of depression. These cognitive processes were chosen as the focus of this thesis as they are suggested to be involved in emotion regulation and have been shown by previous studies to precede the onset of depression, are also suggested to be modifiable and have been targeted by interventions that are effective in preventing and treating depression (Diamond & Lee, 2011; Forbes et al., 2007; Forkmann et al., 2014; Friedberg & McClure, 2015; Garber, 2006; Giesen et al., 2007; Jacobs et al., 2008; Kilford et al., 2015; Rawal & Rice, 2012).Thus, there is good reason to justify the focus on cognitive processes. However, it is important to consider that other familial and social factors that have not been examined in this thesis, such maladaptive parenting and poverty, are thought to be involved in the etiology of depression and in intergenerational transmission of depression (S. H. Goodman & Gotlib, 1999). For example, maladaptive parenting has been shown by previous research to increase risk for future depression in children and might be important to consider in the offspring of depressed parents (S. H. Goodman & Gotlib, 1999; Lovejoy et al., 2000; Lyons-Ruth, Wolfe, & Lyubchik, 2000; McLeod, Weisz, & Wood, 2007). It has been suggested by models of intergenerational transmission of depression that depressed parents may have difficulties meeting their children's emotional needs at different

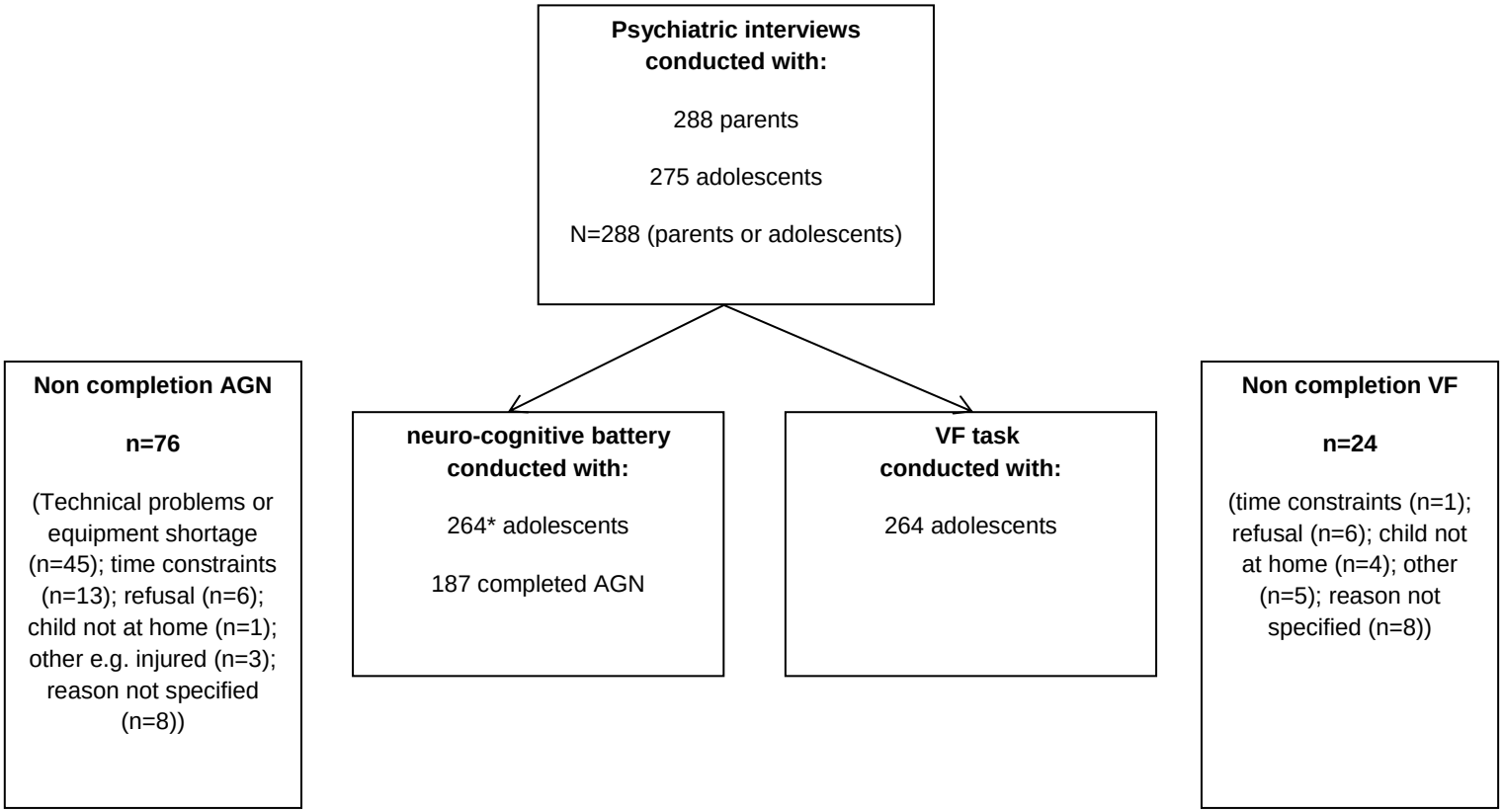
developmental stages, and this likely to affect the development of emotion regulation skills in offspring and potentially increase risk for future depression (Beardslee et al., 2011; S. H. Goodman & Gotlib, 1999; Rutter, 1985). Previous studies (mostly in depressed mothers) have indeed shown that depressed parents exhibit more depressive affect and behaviour, display patterns of inadequate parenting, such as lower responsiveness or reduced scaffolding, and engage in more negative, punitive and conflictual interactions with their children compared to healthy parents (Beardslee et al., 2011; S. H. Goodman & Gotlib, 1999). Thus, while familial and social factors such maladaptive parenting were not examined in this thesis, it is possible that some of the adverse effects of such factors on children's risk for depression could act via cognitive processes in the child. Hence, it may be beneficial to explore in future studies of offspring of depressed parents how parenting behaviours or elements related to parent-child interaction are associated with cognitive processes that are involved in emotion regulation, such as executive functions, the development of overgeneral memory and reward processing. For example, it is likely that attachment style may be involved in reward processing of social rewards (Ainsworth, 1979; Hervas & Vazquez, 2013; Strathearn et al., 2009). However, this has not been examined in the current thesis and has hardly been considered in theoretical literature of reward processing or examined by previous empirical studies in offspring of depressed parents.

6.4. Conclusion

Existing research suggests that executive functions, specificity of autobiographical memory and reward processing may be involved in the etiology of depression. This thesis focused on exploring these processes as risk and protective factors in the offspring of depressed parents, a group at high risk for developing depression. This thesis contributed to existing research by demonstrating that these processes have a protective role with regard to outcomes such as depressive symptoms, "better than expected" resilience to depressive symptoms and social functioning. It has also addressed a specific methodological gap with regard to reward processing, by employing social paradigms to explore reward processing in young adults at familial risk for depression and adolescents. Findings of this thesis have clinical implications and may assist in furthering the research and knowledgebase required to improve preventive interventions for high risk groups.

Appendices

Appendix 2.1. Participation rates and reasons for non-completion of executive functioning tasks



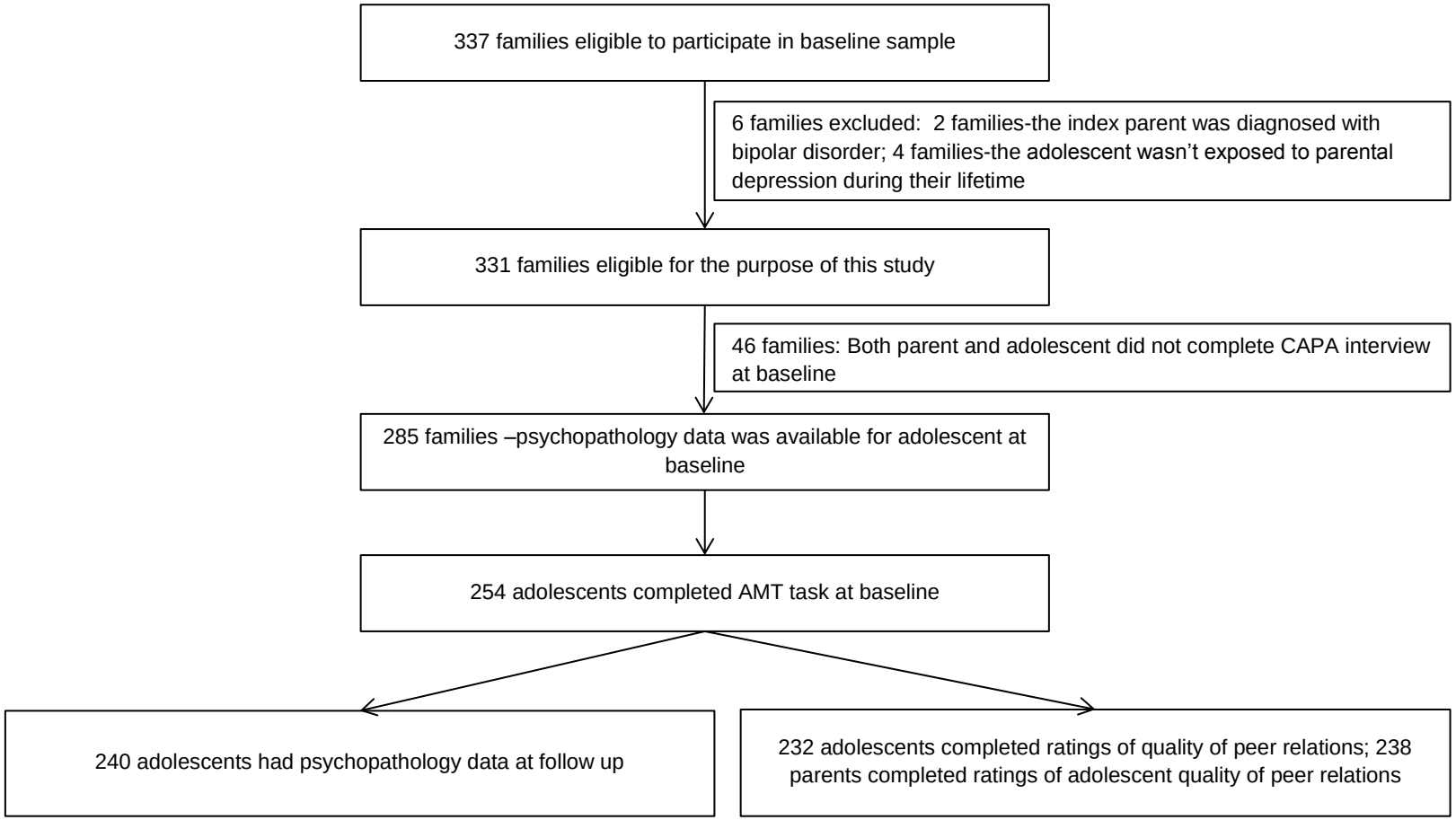
Note: One of these subjects only completed the verbal fluency task and was unable to complete the battery in full due to time constraints.

Appendix 2.2. Current parental depression, offspring executive functioning and their interaction predicting offspring depressive symptom count while controlling for additional parental depression variables

	AGN Inhibitory control errors (N=167)			VF Mental generativity (N=231)			AGN Shifting costs (N=166)		
	B(S.E.)	**β**	**p**	**B(S.E.)**	**β**	**p**	**B(S.E.)**	**β**	**p**
Gender of adolescent	.51 (.29)	.13	.08	.48 (.25)	.12	.06	.33 (.29)	.08	.27
Age of adolescent	.45 (.16)	.22	<.01	.34 (.13)	.17	<.05	.29 (.15)	.14	.06
IQ of adolescent	-.32 (.16)	-.15	<.05	-.25 (.15)	-.12	.09	-.34 (.16)	-.16	<.05
Current PD	1.72 (.38)	.33	<.001	.98 (.33)	.19	<.01	1.72 (.39)	.33	<.001
EF measure (adolescent)	.30 (.17)	.15	.07	.04 (.15)	.02	.77	-.18 (.15)	-.09	.23
Parent previous severe episode	.15 (.34)	.03	.65	.25 (.29)	.06	.39	.30 (.34)	.07	.37
Parent age of onset	-.17 (.33)	-.04	.60	-.01 (.29)	-.002	.98	-.01 (.34)	-.002	.98
Adolescent exposure to previous parent episodes	.38 (.54)	.05	.47	.29 (.53)	.04	.59	.33 (.54)	.04	.54
EF measure (adolescent) x current PD	.74 (.44)	.13	.09	-.68 (.31)	-.16	<.05	.91 (.38)	.18	<.05

Note: The table presents the final regression model; PD= Parental depression, EF=Executive functioning.

Appendix 3.1. Participation rates



Note: N may vary between analyses due to missing values in other variables/covariates.

Appendix 3.2. Quality of peer relationships- adolescent version

Instructions:
Below is a list of statements related to friendships. Please read each statement carefully and cross the reply that best applies to you.

	Strongly agree	Agree	Neutral/ mixed	Disagree	Strongly disagree
1. Children in my class at school are friendly to me.					
2. Children who are not in my class are friendly to me.					
3. Other children often tease me.					
4. Other children often exclude me from things (e.g. games, parties, chats).					
5. Other children think I am nice looking					
6. Other children think I am 'cool'					
7. Other children think I am a nice person					
8. Other children think I am good at my work					
9. Other children think I am good at sport					
10. Other children want to be my friend					

Appendix 3.3. Quality of peer relationships- parent version

Instructions: We would like to know a bit about your child's friends. Please cross the box that best suits your child.		
1. About how many times does your child do things with friends outside of school?		
	Less than once a week	
	1 to 2 times a week	
	3 to 4 times a week	
	5 or more times a week	
2. Would you agree or disagree that your child is one of the more popular kids in his or her school		
	Strongly agree	
	Agree	
	Neutral or mixed	
	Disagree	
	Strongly disagree	
3. Would you agree or disagree that your child is disliked or rejected by other kids		
	Strongly agree	
	Agree	
	Neutral or mixed	
	Disagree	
	Strongly disagree	

Appendix 3.4. OGM variables predicting adolescent-rated quality of peer relationships while controlling for baseline mood symptoms

OGM measure	OGM total			OGM negative			OGM positive		
	B (S.E.)	β	p	B (S.E.)	β	p	B (S.E.)	β	p
Step 1									
OGM measure	1.26	.20	<.01	1.29 (.45)	.20	<.01	.80 (.44)	.12	.07
Step 2									
OGM measure	.77 (.33)	.12	<.05	.82 (.334)	.12	<.05	.45 (.33)	.07	.17
B adolescent-rated quality of peer relationships	4.45	.66	<.001	4.45(.34)	.66	<.001	4.50 (.35)	.67	<.001
Step 3									
OGM measure	.78 (.33)	.12	<.05	.85 (.33)	.13	<.05	.44 (.33)	.07	.18
B adolescent-rated quality of peer relationships	4.48	.67	<.001	4.48 (.34)	.67	<.001	4.54 (.34)	.68	<.001
Age	.22 (.33)	.04	.49	.22 (.32)	.04	.49	.18 (.33)	.03	.59
IQ	-.50 (.36)	-.07	.16	-.53 (.36)	-.07	.14	-.48 (.36)	-.07	.19
Gender	1.43	.11	<.05	1.44 (.66)	.11	<.05	1.47 (.66)	.12	<.05
Step 4									
OGM measure	.78 (.33)	.12	<.05	.85 (.34)	.13	<.05	.44 (.33)	.07	.18
B adolescent-rated quality of peer relationships	4.47	.67	<.001	4.49 (.37)	.67	<.001	4.51 (.37)	.67	<.001
Age	.22(.33)	.04	.51	.23 (.33)	.04	.49	.16 (.33)	.03	.62
IQ	-.50 (.37)	-.07	.17	-.53 (.37)	-.08	.15	-.47 (.37)	-.07	.21
Gender	1.427	.11	<.05	1.45 (.67)	.11	<.05	1.44 (.67)	.11	<.05
B mood symptoms	.03 (.37)	.004	.94	-.02 (.37)	-.003	.96	.08 (.37)	.01	.82

Note: B=Baseline

Appendix 3.5. OGM variables predicting parent-rated quality of peer relationships while controlling for baseline mood symptoms

OGM measure	OGM total			OGM negative			OGM positive		
	B (S.E.)	β	p	B (S.E.)	β	p	B (S.E.)	β	p
Step 1									
OGM measure	.34 (.16)	.15	<.05	.25 (.16)	.11	.11	.31 (.16)	.13	.05
Step 2									
OGM measure	.19 (.11)	.08	.09	.10 (.11)	.04	.36	.21 (.11)	.09	.06
B parent- rated quality of peer relationships	1.71 (.11)	.71	<.001	1.72 (.12)	.71	<.001	1.72 (.11)	.71	<.001
Step 3									
OGM measure	.17 (.11)	.07	.12	.09 (.11)	.04	.44	.19 (.11)	.08	.08
B parent- quality of peer relationships	1.70 (.11)	.71	<.001	1.71 (.12)	.71	<.001	1.71 (.11)	.71	<.001
Age	-.19 (.11)	-.08	.09	-.20 (.11)	-.09	.07	-.19 (.11)	-.08	.09
IQ	-.05 (.12)	-.02	.67	-.05 (.12)	-.02	.69	-.04 (.120)	-.02	.72
Gender	.30 (.22)	.06	.18	.30 (.22)	.06	.18	.30 (.22)	.07	.17
Step 4									
OGM measure	.17 (.11)	.07	.14	.08 (.11)	.03	.49	.19 (.11)	.08	.08
B parent-rated- quality of peer relationships	1.69 (.12)	.70	<.001	1.69 (.12)	.70	<.001	1.68 (.12)	.70	<.001
Age	-.20 (.11)	-.09	.08	-.21 (.11)	-.09	.07	-.20 (.11)	-.09	.08
IQ	-.04 (.12)	-.02	.72	-.04 (.12)	-.02	.74	-.03 (.12)	-.01	.79
Gender	.29 (.22)	.06	.20	.28 (.22)	.06	.20	.29 (.22)	.06	.20
B mood symptoms	.05 (.12)	.02	.66	.06 (.12)	.02	.64	.07 (.12)	.03	.56

Note: B=Baseline

Appendix 3.6. Recent negative life events predicting baseline OGM while controlling for baseline mood symptoms

Outcome variable/ predictors	OGM total			OGM negative			OGM positive		
	B (S.E.)	β	P	B (S.E.)	β	P	B (S.E.)	β	P
Step 1									
Recent negative life events	.18 (.13)	.09	.15	.16 (.08)	.14	<.05	.02 (.08)	.01	.83
Step 2									
Recent negative life events	.21 (.13)	.11	.10	.183 (.08)	.16	<.05	.03 (.08)	.02	.71
Age	-.20 (.12)	-.110	.10	-.10 (.07)	-.09	.17	-.10 (.08)	-.09	.19
IQ	.10 (.13)	.05	.48	.09 (.08)	.07	.27	.005 (.08)	.004	.95
Gender	-.06 (.25)	-.02	.80	-.05 (.15)	-.02	.74	-.01 (.15)	-.006	.92
Step 3									
Recent negative life events	.13 (.14)	.07	.34	.13 (.09)	.11	.13	.002 (.09)	.001	.98
Age	-.22 (.12)	-.12	.07	-.12 (.08)	-.11	.12	-.11 (.08)	-.10	.16
IQ	.12 (.13)	.06	.39	.10 (.08)	.08	.21	.01 (.08)	.01	.88
Gender	-.09 (.25)	-.02	.71	-.07 (.15)	-.03	.65	-.02 (.15)	-.01	.87
B mood symptoms	.19 (.14)	.10	.18	.12 (.09)	.10	.16	.06 (.09)	.06	.45

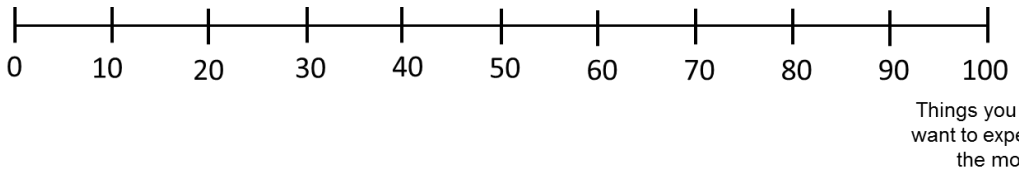
Note: B=Baseline

Appendix 5.1. The Social Situations Questionnaire

The Positive Social Situations Scale

Instructions:
Below is a list of positive social situations. Imagine yourself in each situation and think how significant this situation is for you. Try to imagine that you have an allowance of 100 points and that you can choose to spend any amount of points (up to 100) on each situation. **Your job is to decide how many points you would spend to experience that situation.** Some situations you would want to experience more than others so you can give them more points.

Here is an example of the rating scale:



If the situation described is something you would really want to experience, you would write a 100.

For example,

How many points would you spend to have a newspaper write about something important that you did? _____ (0-100 points).

Remember, there is no right or wrong answer. Different people might give a different amount of points to each situation.

List of positive social events	0-100 points
1. How many points would you spend to go out to a party with your friends?	
2. How many points would you spend for people around you to think you are an interesting person?	
3. How many points would you spend for people at your class to look up to you?	
4. How many points would you spend to make someone that you care about feel happy?	
5. How many points would you spend to have a heart to heart conversation with someone close?	
6. How many points would you spend to have a friend suggest you go out together to do something fun?	
7. How many points would you spend for someone you like to call and ask you out on a date?	

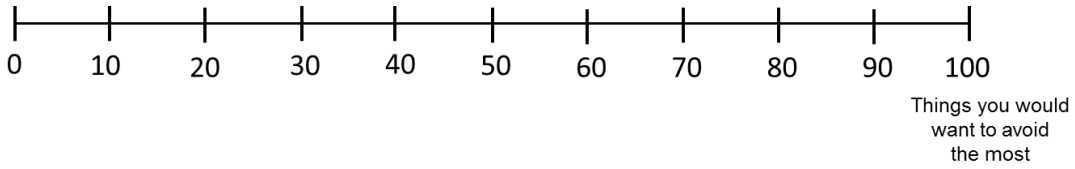
List of positive social events	0-100 points
8. How many points would you spend to go to a family meal?	
9. How many points would you spend to feel really understood by someone else when you are upset about something that happened to you?	
10. How many points would you spend for a close friend to ask you for your advice?	
11. How many points would you spend for someone that you care about to tell you that they love you?	
12. How many points would you spend to make a friend who is sad feel better?	
13. How many points would you spend to win a competition in something that you are interested in at school?	
14. How many points would you spend for someone to compliment you on how you look?	
15. How many points would you spend to make a new friend?	
16. How many points would you spend to be praised by someone you admire?	
17. How many points would you spend to talk on the phone with a friend?	
18. How many points would you spend to feel that your classmates like you?	
19. How many points would you spend to get praised by a teacher?	
20. How many points would you spend to get the highest grade in your class in an important test?	

The Negative Social Situations Scale

Instructions:

Below is a list of negative social situations. Imagine yourself in each situation and think how significant this situation is for you. Try to imagine that you have an allowance of 100 points and that you can choose to spend any amount of points (up to 100) on each situation. **Your job is to decide how many points you would spend to avoid that situation.** Some situations you would want to avoid more than others so you can give them more points.

Here is an example of the rating scale:



If the situation described is something you would really want to avoid, you would write a 100.

For example,

How many points would you spend to avoid making a bad impression on someone that you have just met? _____ (0-100 points).

Remember, there is no right or wrong answer. Different people might give a different amount of points to each situation.

List of negative social events	0-100 points
1. How many points would you spend to avoid a situation where a friend tells you that they don't want to see you anymore?	
2. How many points would you spend to avoid a situation where you ask out someone that you like and they turn you down?	
3. How many points would you spend to avoid not being invited to a party that all your friends have been invited to?	
4. How many points would you spend to avoid being a part of a conversation on a topic you know nothing about?	
5. How many points would you spend to avoid having an argument with a close friend?	
6. How many points would you spend to avoid overhearing somebody say something bad about you?	
7. How many points would you spend to avoid a situation where you ask a friend to help you with a difficult task and they say no?	

List of negative social events	0-100 points
8. How many points would you spend to avoid a situation where other people ignore your opinion in a conversation?	
9. How many points would you spend to avoid going to a party and not finding anyone to talk with?	
10. How many points would you spend to avoid being criticized by a classmate in front of others?	
11. How many points would you spend to avoid a situation where someone that you have been dating for a couple of weeks breaks up with you?	
12. How many points would you spend to avoid a situation where you get the lowest grade of all your friends in a test you did?	
13. How many points would you spend to avoid having a friend turn you down when you invite them to go see a movie?	
14. How many points would you spend to avoid a situation where a good friend of yours chooses to go out with someone else instead of you on a Saturday night?	
15. How many points would you spend to avoid a situation where a friend stops returning your calls?	
16. How many points would you spend to avoid feeling that somebody that you know doesn't like you?	
17. How many points would you spend to avoid a situation where someone is being disrespectful towards you?	
18. How many points would you spend to avoid losing a competition in school about something you are interested in?	
19. How many points would you spend to avoid getting teased over your physical appearance?	
20. How many points would you spend to avoid getting told off by a teacher over something bad that you did?	

Appendix 5.2. The Pleasant Events Schedule (shortened version)[2]

Instructions: This questionnaire is designed to find out about the things you have done during the last month, and how much you enjoyed doing them. For each activity please rate how many times this activity has happened. Then, rate how pleasant each activity was.

	How many times did this happen in the last 30 days?			If this happened how pleasant was it?		
	0 times	1 time	2 or more	Not pleasant	Somewhat pleasant	Very Pleasant
1. Playing team sports (soccer, rugby, baseball, basketball, etc.)						
2. Outdoor activities (nature walks, mountaineering, camping, hiking, fishing, birdwatching, etc.)						
3. Reading (books, poems, newspapers, magazines)						
4. Going to a fun party						
5. Watching television						
6. Working on machines (cars, bikes, motorcycles, tractors, etc.) or doing woodwork, carpentry or repairing things						
7. Writing (essays, stories, novels, plays, poetry, a diary)						
8. Going to or organising social club meetings/ youth club, scouts, guides, etc.						
9. Playing a musical instrument						
10. Cooking or making snacks						
11. Doing your hair/putting on makeup/using perfume or aftershave/ making yourself look nice for an event						
12. Acting/singing/performing						
13. Being with friends						

[2] Adapted from MacPhillamy & Lewinsohn(1982).

	How many times did this happen in the last 30 days?			If this happened how pleasant was it?		
	0 times	1 time	2 or more	Not pleasant	Somewhat pleasant	Very Pleasant
14. Being with family or relatives						
15. Doing crafts or art-work (painting, pottery, jewellery, beads, weaving, sewing, etc.)						
16. Being popular at a gathering or social event						
17. Dancing						
18. Dating, etc.						
19. Taking pictures or “selfies”						
20. Helping someone or giving someone advice						
21. Making a new friend						
22. Getting a good mark						
23. Hearing jokes, telling jokes or laughing						
24. Eating a good meal						
25. Going shopping						
26. Being noticed as attractive						
27. Browsing the internet or playing computer games						
28. Chatting online or texting friends						
29. Being “liked” on Facebook or Instagram						
30. Talking on the telephone with friends						
31. Going to the movies						
32. Going to a restaurant						
33. Being invited out						

	How many times did this happen in the last 30 days?			If this happened how pleasant was it?		
	0 times	1 time	2 or more	Not pleasant	Somewhat pleasant	Very Pleasant
34. Getting an award or prize						
35. Doing exercise (running, jogging, swimming, cycling, going to the gym or doing gymnastics, etc.)						
36. Listening to music						
37. Being with my girlfriend or boyfriend						
38. Being complimented or told I have done well						
39. Being told I am loved						

Appendix 5.3. Activities scale

Instructions: These questions are about the activities you do. For each item please tick the relevant box.					
1. How often do you exercise (intense enough to be out of breath and have an increased heart rate)?	Never or less than once a month	At least once a month	Once every 2 weeks	At least once a week	More than once a week
2. How often do you exercise (not enough to be out of breath and have an increased heart rate) e.g. walking, cycling, kicking a ball about?	Never or less than once a month	At least once a month	Once every 2 weeks	At least once a week	More than once a week
3. How often do you play sport?	Never or less than once a month	At least once a month	Once every 2 weeks	At least once a week	More than once a week
4. How often do you go to meetings of clubs or other groups (e.g. football, Scouts, Guides, yoga, evening class)?	Never or less than once a month	At least once a month	Once every 2 weeks	At least once a week	More than once a week

References

Abela, J. R., & Hankin, B. L. (2008). Cognitive vulnerability to depression in children and adolescents. In J. R. Abela & B. L. Hankin (Eds.), *Handbook of depression in children and adolescents*. New York: Guilford Press.

Abela, J. R., & Hankin, B. L. (2011). Rumination as a vulnerability factor to depression during the transition from early to middle adolescence: a multiwave longitudinal study. *Journal of abnormal psychology, 120*(2), 259.

Abramson, L. Y., Alloy, L. B., Hogan, M. E., Whitehouse, W. G., Donovan, P., Rose, D. T., . . . Raniere, D. (1999). Cognitive vulnerability to depression: Theory and evidence. *Journal of Cognitive Psychotherapy, 13*(1), 5-20.

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological review, 96*(2), 358.

Abramson, L. Y., Seligman, M. E., & Teasdale, J. D. (1978). Learned helplessness in humans: critique and reformulation. *Journal of abnormal psychology, 87*(1), 49.

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.

Ainsworth, M. S. (1979). Infant–mother attachment. *American psychologist, 34*(10), 932.

Alloy, L. B., Abramson, L. Y., Whitehouse, W. G., Hogan, M. E., Tashman, N. A., Steinberg, D. L., . . . Donovan, P. (1999). Depressogenic cognitive styles: Predictive validity, information processing and personality characteristics, and developmental origins. *Behaviour research and therapy, 37*(6), 503-531.

Alloy, L. B., Olino, T., Freed, R. D., & Nusslock, R. (2016). Role of Reward Sensitivity and Processing in Major Depressive and Bipolar Spectrum Disorders. *Behavior Therapy*.

American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders: DSM-IV (4th ed.). Washington, DC.

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Washington, DC: APA.

Amstadter, A. B., Myers, J. M., & Kendler, K. S. (2014). Psychiatric resilience: longitudinal twin study. *The British Journal of Psychiatry, 205*(4), 275-280.

Anderman, L. H., & Anderman, E. M. (1999). Social predictors of changes in students' achievement goal orientations. *Contemporary Educational Psychology, 24*(1), 21-37.

Andersen, S. M., & Schwartz, A. H. (1992). Intolerance of ambiguity and depression: A cognitive vulnerability factor linked to hopelessness. *Social Cognition, 10*(3), 271.

Andreasen, N. C., Endicott, J., Spitzer, R. L., & Winokur, G. (1977). The family history method using diagnostic criteria: reliability and validity. *Archives of General Psychiatry, 34*(10), 1229.

Angold, A., & Costello, E. (1993). Depressive comorbidity in children and adolescents. *American Journal of Psychiatry, 150*(12), 1779-1791.

Angold, A., & Costello, E. J. (2000). The child and adolescent psychiatric assessment (CAPA). *Journal of the American Academy of Child & Adolescent Psychiatry, 39*(1), 39-48.

Angold, A., Costello, E. J., Farmer, E. M., Burns, B. J., & Erkanli, A. (1999). Impaired but undiagnosed. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(2), 129-137.

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*.

Angold, A., Costello, E. J., & Worthman, C. M. (1998). Puberty and depression: the roles of age, pubertal status and pubertal timing. *Psychological Medicine, 28*(01), 51-61.

Angold, A., Prendergast, M., Cox, A., Harrington, R., Simonoff, E., & Rutter, M. (1995). The child and adolescent psychiatric assessment (CAPA). *Psychological medicine, 25*(04), 739-753.

Avenevoli, S., Swendsen, J., He, J.-P., Burstein, M., & Merikangas, K. R. (2015). Major depression in the National Comorbidity Survey–Adolescent Supplement: prevalence, correlates, and treatment. *Journal of the American Academy of Child & Adolescent Psychiatry, 54*(1), 37-44. e32.

Bakker, M. P., Ormel, J., Verhulst, F. C., & Oldehinkel, A. J. (2011). Adolescent family adversity and mental health problems: the role of adaptive self-regulation capacities. The TRAILS study. *Journal of Abnormal Child Psychology, 39*(3), 341-350.

Baldo, J. V., Shimamura, A. P., Delis, D. C., Kramer, J., & Kaplan, E. (2001). Verbal and design fluency in patients with frontal lobe lesions. *Journal of the International Neuropsychological Society, 7*(05), 586-596.

Barnett, P. A., & Gotlib, I. H. (1988). Psychosocial functioning and depression: distinguishing among antecedents, concomitants, and consequences. *Psychological bulletin, 104*(1), 97.

Basso, M. R., & Bornstein, R. A. (1999). Relative memory deficits in recurrent versus first-episode major depression on a word-list learning task. *Neuropsychology, 13*(4), 557.

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: desire for interpersonal attachments as a fundamental human motivation. *Psychological bulletin, 117*(3), 497.

Beardselee, W. R., Versage, E. M., & Giadstone, T. R. (1998). Children of affectively ill parents: a review of the past 10 years. *Journal of the American Academy of Child & Adolescent Psychiatry, 37*(11), 1134-1141.

Beardslee, W. R., Brent, D. A., Weersing, V. R., Clarke, G. N., Porta, G., Hollon, S. D., . . . Iyengar, S. (2013). Prevention of depression in at-risk adolescents: Longer-term effects. *JAMA psychiatry, 70*(11), 1161-1170.

Beardslee, W. R., Gladstone, T. R., & O'Connor, E. E. (2011). Transmission and prevention of mood disorders among children of affectively ill parents: a review. *Journal of the American Academy of Child & Adolescent Psychiatry, 50*(11), 1098-1109.

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

Beck, A. T. (1976). *Cognitive therapy and emotional disorders*. New York: International University Press.

Beck, A. T. (2008). The evolution of the cognitive model of depression and its neurobiological correlates. *American Journal of Psychiatry, 165*(8), 969-977.

Beck, A. T., Brown, G., Steer, R. A., & Weissman, A. N. (1991). Factor analysis of the Dysfunctional Attitude Scale in a clinical population. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 3*(3), 478.

Beck, A. T., Rush, A., Shaw, B., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford Press.

Benton, A. L. (1968). Differential behavioral effects in frontal lobe disease. *Neuropsychologia, 6*(1), 53-60.

Berenbaum, H., Snowhite, R., & Oltmanns, T. F. (1987). Anhedonia and emotional responses to affect evoking stimuli. *Psychological Medicine, 17*(03), 677-684.

Berking, M., & Wupperman, P. (2012). Emotion regulation and mental health: recent findings, current challenges, and future directions. *Current Opinion in Psychiatry, 25*(2), 128-134.

Berridge, K. C., & Robinson, T. E. (1998). What is the role of dopamine in reward: hedonic impact, reward learning, or incentive salience? *Brain Research Reviews, 28*(3), 309-369.
Berridge, K. C., & Robinson, T. E. (2003). Parsing reward. *Trends in neurosciences, 26*(9), 507-513.
Best, J. R., & Miller, P. H. (2010). A developmental perspective on executive function. *Child development, 81*(6), 1641-1660.
Bird, A., & Reese, E. (2006). Emotional reminiscing and the development of an autobiographical self. *Developmental psychology, 42*(4), 613.
Birmaher, B., Ryan, N. D., Williamson, D. E., Brent, D. A., Kaufman, J., Dahl, R. E., . . . 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*(11), 1427-1439.
Bolger, N., DeLongis, A., Kessler, R. C., & Schilling, E. A. (1989). Effects of daily stress on negative mood. *Journal of personality and social psychology, 57*(5), 808.
Bosc, M., Dubini, A., & Polin, V. (1997). Development and validation of a social functioning scale, the Social Adaptation Self-evaluation Scale. *European Neuropsychopharmacology, 7*(1), S57-S70.
Brennan, P. A., Hammen, C., Andersen, M. J., Bor, W., Najman, J. M., & Williams, G. M. (2000). Chronicity, severity, and timing of maternal depressive symptoms: relationships with child outcomes at age 5. *Developmental psychology, 36*(6), 759.
Brennan, P. A., Le Brocque, R., & Hammen, C. (2003). Maternal depression, parent–child relationships, and resilient outcomes in adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry, 42*(12), 1469-1477.
Brent, D., & Weersing, V. R. (2009). Depressive disorders in childhood and adolescence. *Rutter's Child and Adolescent Psychiatry, Fifth Edition*, 587-612.
Brent, D. A., Kolko, D. J., Birmaher, B., Baugher, M., Bridge, J., Roth, C., & Holder, D. (1998). Predictors of treatment efficacy in a clinical trial of three psychosocial treatments for adolescent depression. *Journal of the American Academy of Child & Adolescent Psychiatry, 37*(9), 906-914.
Brown, B. B. (2004). Adolescents' relationships with peers. *Handbook of adolescent psychology, 2*, 363-394.
Bukowski, W. M., Hoza, B., & Boivin, M. (1994). Measuring friendship quality during pre-and early adolescence: The development and psychometric properties of the Friendship Qualities Scale. *Journal of social and Personal Relationships, 11*(3), 471-484.
Burcusa, S. L., & Iacono, W. G. (2007). Risk for recurrence in depression. *Clinical psychology review, 27*(8), 959-985.
Burke, J. D., Loeber, R., & Birmaher, B. (2002). Oppositional defiant disorder and conduct disorder: a review of the past 10 years, part II. *Journal of the American Academy of Child & Adolescent Psychiatry, 41*(11), 1275-1293.
Buss, A. H. (1983). Social rewards and personality. *Journal of personality and social psychology, 44*(3), 553.
Cacioppo, J. T., Hughes, M. E., Waite, L. J., Hawkley, L. C., & Thisted, R. A. (2006). Loneliness as a specific risk factor for depressive symptoms: cross-sectional and longitudinal analyses. *Psychology and aging, 21*(1), 140.
Campos, J. J., Mumme, D. L., Kermoian, R., & Campos, R. G. (1994). A functionalist perspective on the nature of emotion. *Monographs of the society for research in child development, 59*(2-3), 284-303.
Casey, B. (2015). Beyond simple models of self-control to circuit-based accounts of adolescent behavior. *Annual review of psychology, 66*, 295-319.

Caspi, A., Moffitt, T. E., Thornton, A., Freedman, D., Amell, J. W., Harrington, H., . . . Silva, P. A. (1996). The life history calendar: a research and clinical assessment method for collecting retrospective event-history data. *International journal of methods in psychiatric research, 6*(2), 101-114.

Castaneda, A., Suvisaari, J., Marttunen, M., Perälä, J., Saarni, S., Aalto-Setälä, T., . . . Tuulio-Henriksson, A. (2008). Cognitive functioning in a population-based sample of young adults with a history of non-psychotic unipolar depressive disorders without psychiatric comorbidity. *Journal of affective disorders, 110*(1), 36-45.

Cavanaugh, S. V. (1984). Diagnosing depression in the hospitalized patient with chronic medical illness. *Journal of Clinical Psychiatry*.

Cella, M., Dymond, S., & Cooper, A. (2010). Impaired flexible decision-making in major depressive disorder. *Journal of Affective Disorders, 124*(1), 207-210.

Chan, S. W., Goodwin, G. M., & Harmer, C. J. (2007). Highly neurotic never-depressed students have negative biases in information processing. *Psychological Medicine, 37*(09), 1281-1291.

Chapman, T. F., Mannuzza, S., Klein, D. F., & Fyer, A. J. (1994). Effects of informant mental disorder on psychiatric family history data. *The American journal of psychiatry, 151*(4), 574.

Chase, H., Frank, M., Michael, A., Bullmore, E., Sahakian, B., & Robbins, T. (2010). Approach and avoidance learning in patients with major depression and healthy controls: relation to anhedonia. *Psychological Medicine, 40*(03), 433-440.

Chisholm, D., Sweeny, K., Sheehan, P., Rasmussen, B., Smit, F., Cuijpers, P., & Saxena, S. (2016). Scaling-up treatment of depression and anxiety: a global return on investment analysis. *The Lancet Psychiatry, 3*(5), 415-424.

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

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). Mahwah, NJ, US: Lawrence Erlbaum Associates Publishers.

Cole, D. A., Ciesla, J. A., Dallaire, D. H., Jacquez, F. M., Pineda, A. Q., LaGrange, B., . . . Felton, J. W. (2008). Emergence of attributional style and its relation to depressive symptoms. *Journal of abnormal psychology, 117*(1), 16.

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child development, 75*(2), 317-333.

Collishaw, S., Hammerton, G., Mahedy, L., Sellers, R., Owen, M. J., Craddock, N., . . . Thapar, A. (2016). Mental health resilience in the adolescent offspring of parents with depression: a prospective longitudinal study. *The Lancet Psychiatry, 3*(1), 49-57.

Colvin, A., Richardson, G. A., Cyranowski, J. M., Youk, A., & Bromberger, J. T. (2014). Does family history of depression predict major depression in midlife women? Study of Women's Health Across the Nation Mental Health Study (SWAN MHS). *Archives of women's mental health, 17*(4), 269-278.

Compas, B. E., Connor-Smith, J., & Jaser, S. S. (2004). Temperament, stress reactivity, and coping: Implications for depression in childhood and adolescence. *Journal of Clinical Child and Adolescent Psychology, 33*(1), 21-31.

Compas, B. E., Malcarne, V. L., & Fondacaro, K. M. (1988). Coping with stressful events in older children and young adolescents. *Journal of consulting and clinical psychology, 56*(3), 405.

Conrad, M., & Hammen, C. (1993). Protective and resource factors in highand low-risk children: A comparison of children with unipolar, bipolar, medically ill, and normal mothers. *Development and psychopathology, 5*(04), 593-607.

Conway, M. A., & Pleydell-Pearce, C. W. (2000). The construction of autobiographical memories in the self-memory system. *Psychological review, 107*(2), 261.

Cook, J., Spring, B., McChargue, D., & Doran, N. (2010). Effects of anhedonia on days to relapse among smokers with a history of depression: a brief report. *Nicotine & Tobacco Research*, ntq118.

Copeland, W. E., Shanahan, L., Worthman, C., Angold, A., & Costello, E. J. (2012). Cumulative depression episodes predict later C-reactive protein levels: a prospective analysis. *Biological psychiatry, 71*(1), 15-21.

Costello, E. J., 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., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of general psychiatry, 60*(8), 837-844.

Cragg, L., & Nation, K. (2008). Go or no-go? Developmental improvements in the efficiency of response inhibition in mid-childhood. *Developmental Science, 11*(6), 819-827.

Crane, C., Heron, J., Gunnell, D., Lewis, G., Evans, J., & Williams, J. M. G. (2016). Adolescent over-general memory, life events and mental health outcomes: Findings from a UK cohort study. *Memory, 24*(3), 348-363.

Cyranowski, J. M., Frank, E., Young, E., & Shear, M. K. (2000). Adolescent onset of the gender difference in lifetime rates of major depression: a theoretical model. *Archives of general psychiatry, 57*(1), 21-27.

Dalgleish, T., Spinks, H., Yiend, J., & Kuyken, W. (2001). Autobiographical memory style in seasonal affective disorder and its relationship to future symptom remission. *Journal of abnormal psychology, 110*(2), 335.

Dalgleish, T., Taghavi, R., Neshat-Doost, H., Moradi, A., Canterbury, R., & Yule, W. (2003). Patterns of processing bias for emotional information across clinical disorders: A comparison of attention, memory, and prospective cognition in children and adolescents with depression, generalized anxiety, and posttraumatic stress disorder. *Journal of clinical child and adolescent psychology, 32*(1), 10-21.

Dalgleish, T., & Werner-Seidler, A. (2014). Disruptions in autobiographical memory processing in depression and the emergence of memory therapeutics. *Trends in cognitive sciences, 18*(11), 596-604.

Dalgleish, T., Williams, J. M. G., Golden, A.-M. J., Perkins, N., Barrett, L. F., Barnard, P. J., . . . Tchanturia, K. (2007). Reduced specificity of autobiographical memory and depression: the role of executive control. *Journal of Experimental Psychology: General, 136*(1), 23.

Davey, C. G., Yücel, M., & Allen, N. B. (2008). The emergence of depression in adolescence: Development of the prefrontal cortex and the representation of reward. *Neuroscience & Biobehavioral Reviews, 32*(1), 1-19.

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

Davis, R. N., & Nolen-Hoeksema, S. (2000). Cognitive inflexibility among ruminators and nonruminators. *Cognitive Therapy and Research, 24*(6), 699-711.

Dawson, J. F. (2014). Moderation in management research: What, why, when, and how. *Journal of Business and Psychology, 29*(1), 1-19.

De Lissnyder, E., Koster, E. H., Derakshan, N., & De Raedt, R. (2010). The association between depressive symptoms and executive control impairments in response to emotional and non-emotional information. *Cognition and Emotion, 24*(2), 264-280.

Deaner, R. O., Khera, A. V., & Platt, M. L. (2005). Monkeys pay per view: adaptive valuation of social images by rhesus macaques. *Current Biology, 15*(6), 543-548.

Deveney, C. M., & Deldin, P. (2006). A preliminary investigation of cognitive flexibility for emotional information in major depressive disorder and non-psychiatric controls. *Emotion, 6*(3), 429.

Diamond, A. (2013). Executive functions. *Annual review of psychology, 64*, 135.

Diamond, A., & Lee, K. (2011). Interventions shown to aid executive function development in children 4 to 12 years old. *Science, 333*(6045), 959-964.

Dichter, G. S. (2010). Anhedonia in unipolar major depressive disorder: A review. *The Open Psychiatry Journal, 4*, 1-9.

Dichter, G. S., Felder, J. N., Petty, C., Bizzell, J., Ernst, M., & Smoski, M. J. (2009). The effects of psychotherapy on neural responses to rewards in major depression. *Biological psychiatry, 66*(9), 886-897.

Dimidjian, S., Hollon, S. D., Dobson, K. S., Schmaling, K. B., Kohlenberg, R. J., Addis, M. E., . . . Gollan, J. K. (2006). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. *Journal of consulting and clinical psychology, 74*(4), 658.

Dobson, K. S., Hollon, S. D., Dimidjian, S., Schmaling, K. B., Kohlenberg, R. J., Gallop, R. J., . . . Jacobson, N. S. (2008). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the prevention of relapse and recurrence in major depression. *Journal of consulting and clinical psychology, 76*(3), 468.

Dodge, K. A. (1993). Social-cognitive mechanisms in the development of conduct disorder and depression. *Annual review of psychology, 44*(1), 559-584.

Dos Santos Silva, I. (1999). *Cancer Epidemiology: Principles and Methods*. Lyon, France: World Health Organiztion, International Agency for Research on Cancer.

Dougherty, L. R., Klein, D. N., Durbin, C. E., Hayden, E. P., & Olino, T. M. (2010). Temperamental positive and negative emotionality and children's depressive symptoms: A longitudinal prospective study from age three to age ten. *Journal of Social and Clinical Psychology, 29*(4), 462.

Dunlop, B. W., & Nemeroff, C. B. (2007). The role of dopamine in the pathophysiology of depression. *Archives of general psychiatry, 64*(3), 327-337.

Dunn, B. D., Dalgleish, T., Lawrence, A. D., Cusack, R., & Ogilvie, A. D. (2004). Categorical and dimensional reports of experienced affect to emotion-inducing pictures in depression. *Journal of abnormal psychology, 113*(4), 654.

Dunn, V., & Goodyer, I. M. (2006). Longitudinal investigation into childhood-and adolescence-onset depression: psychiatric outcome in early adulthood. *The British Journal of Psychiatry, 188*(3), 216-222.

Eaton, W. W., Shao, H., Nestadt, G., Lee, B. H., Bienvenu, O. J., & Zandi, P. (2008). Population-based study of first onset and chronicity in major depressive disorder. *Archives of General Psychiatry, 65*(5), 513-520.

Eckblad, M., Chapman, L., Chapman, J., & Mishlove, M. (1982). The revised social anhedonia scale. *Unpublished test*.

Ekman, P. (1993). Facial expression and emotion. *American psychologist, 48*(4), 384.

Ellis, H. C. (1991). Focused attention and depressive deficits in memory. *Journal of experimental psychology. General, 120*(3), 310-312.

Erber, R., & Erber, M. W. (1994). Beyond mood and social judgment: Mood incongruent recall and mood regulation. *European Journal of Social Psychology, 24*(1), 79-88.
Eshel, N., & Roiser, J. P. (2010). Reward and punishment processing in depression. *Biological psychiatry, 68*(2), 118-124.
Evans, J., Williams, J., O'loughlin, S., & Howells, K. (1992). Autobiographical memory and problem-solving strategies of parasuicide patients. *Psychological Medicine, 22*(02), 399-405.
Evans, L. D., Kouros, C. D., Samanez-Larkin, S., & Garber, J. (2016). Concurrent and short-term prospective relations among neurocognitive functioning, coping, and depressive symptoms in youth. *Journal of Clinical Child & Adolescent Psychology, 45*(1), 6-20.
Favre, T., Hughes, C., Emslie, G., Stavinoha, P., Kennard, B., & Carmody, T. (2008). Executive functioning in children and adolescents with major depressive disorder. *Child Neuropsychology, 15*(1), 85-98.
Feder, A., Nestler, E. J., & Charney, D. S. (2009). Psychobiology and molecular genetics of resilience. *Nature Reviews Neuroscience, 10*(6), 446-457.
Feng, X., Keenan, K., Hipwell, A. E., Henneberger, A. K., Rischall, M. S., Butch, J., . . . Babinski, D. E. (2009). Longitudinal associations between emotion regulation and depression in preadolescent girls: moderation by the caregiving environment. *Developmental psychology, 45*(3), 798.
Fergusson, D. M., Boden, J. M., & Horwood, L. J. (2007). Recurrence of major depression in adolescence and early adulthood, and later mental health, educational and economic outcomes. *The British Journal of Psychiatry, 191*(4), 335-342.
Fergusson, D. M., & Woodward, L. J. (2002). Mental health, educational, and social role outcomes of adolescents with depression. *Archives of general psychiatry, 59*(3), 225-231.
Field, A. (2009). *Discovering statistics using SPSS* (third ed.): Sage publications.
Fivush, R. (2011). The development of autobiographical memory. *Annual review of psychology, 62*, 559-582.
Forbes, E. E. (2009). Where's the fun in that? Broadening the focus on reward function in depression. *Biological psychiatry, 66*(3), 199.
Forbes, E. E., & Dahl, R. E. (2005). Neural systems of positive affect: relevance to understanding child and adolescent depression? *Development and psychopathology, 17*(03), 827-850.
Forbes, E. E., & Dahl, R. E. (2012). Research Review: altered reward function in adolescent depression: what, when and how? *Journal of Child Psychology and Psychiatry, 53*(1), 3-15.
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.
Ford, T., Goodman, R., & Meltzer, H. (2003). The British child and adolescent mental health survey 1999: the prevalence of DSM-IV disorders. *Journal of the American Academy of Child & Adolescent Psychiatry, 42*(10), 1203-1211.
Forkmann, T., Scherer, A., Pawelzik, M., Mainz, V., Drueke, B., Boecker, M., & Gauggel, S. (2014). Does cognitive behavior therapy alter emotion regulation in inpatients with a depressive disorder? *Psychology research and behavior management, 7*, 147.
Fossati, P., Bastard Guillaume, L., Ergis, A.-M., & Allilaire, J.-F. (2003). Qualitative analysis of verbal fluency in depression. *Psychiatry research, 117*(1), 17-24.
Foster, C. E., Webster, M. C., Weissman, M. M., Pilowsky, D. J., Wickramaratne, P. J., Rush, A. J., . . . Cerda, G. (2008). Course and severity of maternal depression: Associations with family functioning and child adjustment. *Journal of Youth and Adolescence, 37*(8), 906-916.
Foulkes, L., & Blakemore, S.-J. (2016). Is there heightened sensitivity to social reward in adolescence? *Current Opinion in Neurobiology, 40*, 81-85.

Foulkes, L., Viding, E., McCrory, E. J., & Neumann, C. S. (2014). Social Reward Questionnaire (SRQ): development and validation. *Frontiers in psychology, 5*, 201.

Fredrickson, B. L., & Joiner, T. (2002). Positive emotions trigger upward spirals toward emotional well-being. *Psychological science, 13*(2), 172-175.

Freedman, D., Thornton, A., Camburn, D., Alwin, D., & Young-DeMarco, L. (1988). The life history calendar: A technique for collecting retrospective data. *Sociological methodology, 18*(1), 37-68.

Friedberg, R. D., & McClure, J. M. (2002). *Clinical Practice of Cognitive Therapy with Children and Adolescents: The Nuts and Bolts*. New York, NY: Guilford Press.

Friedberg, R. D., & McClure, J. M. (2015). *Clinical practice of cognitive therapy with children and adolescents: The nuts and bolts*.

Funder, D. C., & Block, J. (1989). The role of ego-control, ego-resiliency, and IQ in delay of gratification in adolescence. *Journal of personality and social psychology, 57*(6), 1041.

Gable, S. L. (2006). Approach and avoidance social motives and goals. *Journal of personality, 74*(1), 175-222.

Garber, J. (2006). Depression in children and adolescents: linking risk research and prevention. *American journal of preventive medicine, 31*(6), 104-125.

Garber, J., Braafladt, N., & Weiss, B. (1995). Affect regulation in depressed and nondepressed children and young adolescents. *Development and psychopathology, 7*(01), 93-115.

Garber, J., Clarke, G. N., Weersing, V. R., Beardslee, W. R., Brent, D. A., Gladstone, T. R., . . . Hollon, S. D. (2009). Prevention of depression in at-risk adolescents: a randomized controlled trial. *Jama, 301*(21), 2215-2224.

Garber, J., & Little, S. (1999). Predictors of competence among offspring of depressed mothers. *Journal of Adolescent Research, 14*(1), 44-71.

Gard, D. E., Gard, M. G., Kring, A. M., & John, O. P. (2006). Anticipatory and consummatory components of the experience of pleasure: a scale development study. *Journal of Research in Personality, 40*(6), 1086-1102.

Gest, S. D., Reed, M. G. J., & Masten, A. S. (1999). Measuring developmental changes in exposure to adversity: A life chart and rating scale approach. *Development and psychopathology, 11*(01), 171-192.

Giesen, F., Searle, A., & Sawyer, M. (2007). Identifying and implementing prevention programmes for childhood mental health problems. *Journal of paediatrics and child health, 43*(12), 785-789.

Gilhooly, K., Fioratou, E., Anthony, S., & Wynn, V. (2007). Divergent thinking: Strategies and executive involvement in generating novel uses for familiar objects. *British Journal of Psychology, 98*(4), 611-625.

Gladstone, T. R., Beardslee, W. R., & O'Connor, E. E. (2011). The prevention of adolescent depression. *Psychiatric Clinics of North America, 34*(1), 35-52.

Goddard, L., Dritschel, B., & Burton, A. (1996). Role of autobiographical memory in social problem solving and depression. *Journal of abnormal psychology, 105*(4), 609.

Gooding, D. C., & Pflum, M. J. (2014). The assessment of interpersonal pleasure: introduction of the Anticipatory and Consummatory Interpersonal Pleasure Scale (ACIPS) and preliminary findings. *Psychiatry research, 215*(1), 237-243.

Gooding, D. C., Pflum, M. J., Fonseca-Pedero, E., & Paino, M. (2016). Assessing social anhedonia in adolescence: The ACIPS-A in a community sample. *European Psychiatry, 37*, 49-55.

Goodman, R. (1997). The Strengths and Difficulties Questionnaire: a research note. *Journal of Child Psychology and Psychiatry, 38*(5), 581-586.

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: a developmental model for understanding mechanisms of transmission. *Psychological review, 106*(3), 458.

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal depression and child psychopathology: A meta-analytic review. *Clinical child and family psychology review, 14*(1), 1-27.

Gorman, J. M. (1996). Comorbid depression and anxiety spectrum disorders. *Depression and anxiety, 4*(4), 160-168.

Gotlib, I. H., Hamilton, J. P., 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.

Gotlib, I. H., & Joormann, J. (2010). Cognition and depression: current status and future directions. *Annual review of clinical psychology, 6*, 285.

Gotlib, I. H., Joormann, J., & Foland-Ross, L. C. (2014). Understanding Familial Risk for Depression A 25-Year Perspective. *Perspectives on psychological science, 9*(1), 94-108.

Gradin, V., Pérez, A., MacFarlane, J., Cavin, I., Waiter, G., Engelmann, J., . . . Steele, J. (2015). Abnormal brain responses to social fairness in depression: an fMRI study using the Ultimatum Game. *Psychological medicine, 45*(06), 1241-1251.

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.

Gross, J. J. (1998). The emerging field of emotion regulation: an integrative review. *Review of general psychology, 2*(3), 271.

Gross, J. J., & Muñoz, R. F. (1995). Emotion regulation and mental health. *Clinical psychology: Science and practice, 2*(2), 151-164.

Grosscup, S. J., & Lewinsohn, P. M. (1980). Unpleasant and pleasant events, and mood. *Journal of Clinical Psychology, 36*(1), 252-259.

Haga, S. M., Ulleberg, P., Slinning, K., Kraft, P., Steen, T. B., & Staff, A. (2012). A longitudinal study of postpartum depressive symptoms: multilevel growth curve analyses of emotion regulation strategies, breastfeeding self-efficacy, and social support. *Archives of women's mental health, 15*(3), 175-184.

Haglund, M. E., Nestadt, P., Cooper, N., Southwick, S., & Charney, D. (2007). Psychobiological mechanisms of resilience: Relevance to prevention and treatment of stress-related psychopathology. *Development and psychopathology, 19*(03), 889-920.

Hale, S., Rose, N. S., Myerson, J., Strube, M. J., Sommers, M., Tye-Murray, N., & Spehar, B. (2011). The structure of working memory abilities across the adult life span. *Psychology and Aging, 26*(1), 92.

Hammen, C. (1992). Cognitive, life stress, and interpersonal approaches to a developmental psychopathology model of depression. *Development and psychopathology, 4*(01), 189-206.

Hammen, C. (2000). Interpersonal factors in an emerging developmental model of depression. In S. L. Johnson, A. M. Hayes, T. M. Field, N. Schneiderman, & P. M. McCABE (Eds.), *Stress, coping, and depression* (pp. 71-88). Mahwa, New Jersy: Lawrence Erlbaum.

Hammen, C. (2002). Context of stress in families of children with depressed parents. In S. H. Goodman & I. H. Gotlib (Eds.), *Children of depressed parents: Mechanisms of risk and implications for treatment* (pp. 175-199). Washington, DC, US: American Psychological Association.

Hammen, C. (2005). Stress and depression. *Annu. Rev. Clin. Psychol., 1*, 293-319.

Hammen, C., & Brennan, P. A. (2003). Severity, chronicity, and timing of maternal depression and risk for adolescent offspring diagnoses in a community sample. *Archives of general psychiatry, 60*(3), 253-258.

Hammen, C., Shih, J., Altman, T., & Brennan, P. A. (2003). Interpersonal impairment and the prediction of depressive symptoms in adolescent children of depressed and nondepressed mothers. *Journal of the American Academy of Child & Adolescent Psychiatry, 42*(5), 571-577.

Hammerton, G., Zammit, S., Potter, R., Thapar, A., & Collishaw, S. (2014). Validation of a composite of suicide items from the Mood and Feelings Questionnaire (MFQ) in offspring of recurrently depressed parents. *Psychiatry research, 216*(1), 82-88.

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.

Hardin, M. G., Schroth, E., Pine, D. S., & Ernst, M. (2007). Incentive-related modulation of cognitive control in healthy, anxious, and depressed adolescents: Development and psychopathology related differences. *Journal of Child Psychology and Psychiatry, 48*(5), 446-454.

Hardy, C. L., Bukowski, W. M., & Sippola, L. K. (2002). Stability and change in peer relationships during the transition to middle-level school. *The Journal of Early Adolescence, 22*(2), 117-142.

Harmer, C. J., Goodwin, G. M., & Cowen, P. J. (2009). Why do antidepressants take so long to work? A cognitive neuropsychological model of antidepressant drug action. *The British Journal of Psychiatry, 195*(2), 102-108.

Harmer, C. J., O'Sullivan, U., Favaron, E., Massey-Chase, R., Ayres, R., Reinecke, A., . . . Cowen, P. J. (2009). Effect of acute antidepressant administration on negative affective bias in depressed patients. *American Journal of Psychiatry, 166*(10), 1178-1184.

Harper, M. S., Dickson, J. W., & Welsh, D. P. (2006). Self-silencing and rejection sensitivity in adolescent romantic relationships. *Journal of Youth and Adolescence, 35*(3), 435-443.

Harrington, R. (2000). Childhood depression: is it the same disorder. *Childhood onset of "adult" psychopathology: Clinical and research advances*, 223-243.

Harvey, P., Le Bastard, G., Pochon, J., Levy, R., Allilaire, J., Dubois, B. e., . . . Fossati, P. (2004). Executive functions and updating of the contents of working memory in unipolar depression. *Journal of psychiatric research, 38*(6), 567-576.

Hasin, D. S., Goodwin, R. D., Stinson, F. S., & Grant, B. F. (2005). Epidemiology of major depressive disorder: results from the National Epidemiologic Survey on Alcoholism and Related Conditions. *Archives of general psychiatry, 62*(10), 1097-1106.

Hayden, B. Y., Parikh, P. C., Deaner, R. O., & Platt, M. L. (2007). Economic principles motivating social attention in humans. *Proceedings of the Royal Society of London B: Biological Sciences, 274*(1619), 1751-1756.

Hayes, A. F. (2009). Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. *Communication monographs, 76*(4), 408-420.

Hayes, A. F. (2012). PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling [White paper].

Heerey, E. A. (2014). Learning from social rewards predicts individual differences in self-reported social ability. *Journal of Experimental Psychology: General, 143*(1), 332.

Hendrawan, D., Yamakawa, K., Kimura, M., Murakami, H., & Ohira, H. (2012). Executive functioning performance predicts subjective and physiological acute stress reactivity: Preliminary results. *International Journal of Psychophysiology, 84*(3), 277-283.

Henriques, J. B., & Davidson, R. J. (2000). Decreased responsiveness to reward in depression. *Cognition & Emotion, 14*(5), 711-724.
Henriques, J. B., Glowacki, J. M., & Davidson, R. J. (1994). Reward fails to alter response bias in depression. *Journal of abnormal psychology, 103*(3), 460.
Hertel, P. T., & Hardin, T. S. (1990). Remembering with and without awareness in a depressed mood: Evidence of deficits in initiative. *Journal of Experimental Psychology: General, 119*(1), 45.
Hervas, G., & Vazquez, C. (2013). Low spirits keep rewards subdued: Decreases in sensitivity to reward and vulnerability to dysphoria. *Behavior therapy, 44*(1), 62-74.
Hetrick, S. E., Cox, G. R., Fisher, C. A., Bhar, S. S., Rice, S. M., Davey, C. G., & Parker, A. G. (2015). Back to basics: could behavioural therapy be a good treatment option for youth depression? A critical review. *Early intervention in psychiatry, 9*(2), 93-99.
Hipwell, A. E., Sapotichne, B., Klostermann, S., Battista, D., & Keenan, K. (2011). Autobiographical memory as a predictor of depression vulnerability in girls. *Journal of Clinical Child & Adolescent Psychology, 40*(2), 254-265.
Hirschfeld, R., Montgomery, S. A., Keller, M. B., Kasper, S., Schatzberg, A. F., Möller, H.-J., . . . Versiani, M. (2000). Social functioning in depression: a review. *Journal of Clinical Psychiatry*.
Hoffman, L., & Rovine, M. J. (2007). Multilevel models for the experimental psychologist: Foundations and illustrative examples. *Behavior Research Methods, 39*(1), 101-117.
Horn, J. L. (1991). Measurement of intellectual capabilities: A review of theory. *Woodcock-Johnson technical manual*, 197-232.
Horowitz, J. L., & Garber, J. (2006). The prevention of depressive symptoms in children and adolescents: A meta-analytic review. *Journal of consulting and clinical psychology, 74*(3), 401.
Hughes, C., Roman, G., Hart, M. J., & Ensor, R. (2013). Does maternal depression predict young children's executive function?–a 4-year longitudinal study. *Journal of Child Psychology and Psychiatry, 54*(2), 169-177.
Humphreys, K. L., Katz, S. J., Lee, S. S., Hammen, C., Brennan, P. A., & Najman, J. M. (2013). The association of ADHD and depression: Mediation by peer problems and parent–child difficulties in two complementary samples. *Journal of abnormal psychology, 122*(3), 854.
Ingram, R. E., Miranda, J., & Segal, Z. V. (1998). *Cognitive vulnerability to depression*: Guilford Press.
Jacobs, R. H., Reinecke, M. A., Gollan, J. K., & Kane, P. (2008). Empirical evidence of cognitive vulnerability for depression among children and adolescents: A cognitive science and developmental perspective. *Clinical psychology review, 28*(5), 759-782.
Jaenicke, C., Hammen, C., Zupan, B., Hiroto, D., Gordon, D., Adrian, C., & Burge, D. (1987). Cognitive vulnerability in children at risk for depression. *Journal of Abnormal Child Psychology, 15*(4), 559-572.
Jarvinen, D. W., & Nicholls, J. G. (1996). Adolescents' social goals, beliefs about the causes of social success, and satisfaction in peer relations. *Developmental psychology, 32*(3), 435.
Jazbec, S., McClure, E., Hardin, M., Pine, D. S., & Ernst, M. (2005). Cognitive control under contingencies in anxious and depressed adolescents: an antisaccade task. *Biological psychiatry, 58*(8), 632-639.
Joffe, R. T., Bagby, R. M., Levitt, A. J., Regan, J. J., & Parker, J. D. (1993). The Tridimensional Personality Questionnaire in major depression. *The American journal of psychiatry*.
Johnson, J. H., & McCutcheon, S. M. (1980). Assessing life stress in older children and adolescents: Preliminary findings with the Life Events Checklist. *Stress and anxiety, 7*, 111-125.

Johnson, M. H. (2012). Executive function and developmental disorders: the flip side of the coin. *Trends in cognitive sciences, 16*(9), 454-457.

Johnston, L., Miles, L., & Macrae, C. N. (2010). Why are you smiling at me? Social functions of enjoyment and non-enjoyment smiles. *British Journal of Social Psychology, 49*(1), 107-127.

Joiner, T. E., Lewinsohn, P. M., & Seeley, J. R. (2002). The core of loneliness: Lack of pleasurable engagement--more so than painful disconnection--predicts social impairment, depression onset, and recovery from depressive disorders among adolescents. *Journal of personality assessment, 79*(3), 472-491.

Joormann, J., & Gotlib, I. H. (2008). Updating the contents of working memory in depression: interference from irrelevant negative material. *Journal of abnormal psychology, 117*(1), 182.

Joormann, J., & Quinn, M. E. (2014). Cognitive processes and emotion regulation in depression. *Depression and anxiety, 31*(4), 308-315.

Joormann, J., & Siemer, M. (2004). Memory accessibility, mood regulation, and dysphoria: difficulties in repairing sad mood with happy memories? *Journal of abnormal psychology, 113*(2), 179.

Joormann, J., & Siemer, M. (2014). Emotion regulation in mood disorders. *Handbook of emotion regulation, 2*, 413-427.

Joormann, J., Siemer, M., & Gotlib, I. H. (2007). Mood regulation in depression: Differential effects of distraction and recall of happy memories on sad mood. *Journal of abnormal psychology, 116*(3), 484.

Joormann, J., Talbot, L., & Gotlib, I. H. (2007). Biased processing of emotional information in girls at risk for depression. *Journal of abnormal psychology, 116*(1), 135.

Karabekiroğlu, A., Topçuoğlu, V., Gimzal Gönentür, A., & Karabekiroğlu, K. (2010). Executive function differences between first episode and recurrent major depression patients. *Turk Psikiyatri Derg, 4*, 280-288.

Karbach, J., & Kray, J. (2009). How useful is executive control training? Age differences in near and far transfer of task-switching training. *Developmental Science, 12*(6), 978-990.

Kazdin, A. E. (1989). Evaluation of the Pleasure Scale in the assessment of anhedonia in children. *Journal of the American Academy of Child & Adolescent Psychiatry, 28*(3), 364-372.

Kendler, K. S., & Gardner, C. O. (2014). Sex differences in the pathways to major depression: a study of opposite-sex twin pairs. *American Journal of Psychiatry*.

Kendler, K. S., Ohlsson, H., Sundquist, J., & Sundquist, K. (2015). IQ and Schizophrenia in a Swedish National Sample: Their Causal Relationship and the Interaction of IQ With Genetic Risk. *American Journal of Psychiatry, 172*(3), 259-265.

Kendler, K. S., Silberg, J. L., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1991). The family history method: whose psychiatric history is measured. *Am J Psychiatry, 148*(11), 1501-1504.

Kessler, R. C., & Walters, E. E. (1998). Epidemiology of DSM-III-R major depression and minor depression among adolescents and young adults in the national comorbidity survey. *Depression and anxiety, 7*(1), 3-14.

Kessler, R. C., & Wang, P. S. (2009). Epidemiology of depression. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression*. New York: Guilford press.

Kilford, E. J., Foulkes, L., Potter, R., Collishaw, S., Thapar, A., & Rice, F. (2015). Affective bias and current, past and future adolescent depression: a familial high risk study. *Journal of Affective Disorders, 174*, 265-271.

King, P. M., & Kitchener, K. S. (2015). Cognitive development in the emerging adult: The emergence of complex cognitive skills. In J. J. Arnett (Ed.), *The Oxford handbook of emerging adulthood* (pp. 105). New York: Oxford University Press.

Klassen, A., Miller, A., & Fine, S. (2006). Agreement between parent and child report of quality of life in children with attention-deficit/hyperactivity disorder. *Child: care, health and development, 32*(4), 397-406.

Klein, D. N., Durbin, C. E., Shankman, S. A., & Santiago, N. J. (2002). Depression and personality.

Klein, D. N., Lewinsohn, P. M., Rohde, P., Seeley, J. R., & Olino, T. M. (2005). Psychopathology in the adolescent and young adult offspring of a community sample of mothers and fathers with major depression. *Psychological Medicine, 35*(03), 353-365.

Klein, D. N., Schatzberg, A. F., McCullough, J. P., Dowling, F., Goodman, D., Howland, R. H., . . . Rush, A. J. (1999). Age of onset in chronic major depression: relation to demographic and clinical variables, family history, and treatment response. *Journal of affective disorders, 55*(2), 149-157.

Klimes-Dougan, B., Ronsaville, D., Wiggs, E. A., & Martinez, P. E. (2006). Neuropsychological functioning in adolescent children of mothers with a history of bipolar or major depressive disorders. *Biological psychiatry, 60*(9), 957-965.

Korn, C., Sharot, T., Walter, H., Heekeren, H., & Dolan, R. (2014). Depression is related to an absence of optimistically biased belief updating about future life events. *Psychological medicine, 44*(03), 579-592.

Korszun, A., Moskvina, V., Brewster, S., Craddock, N., Ferrero, F., Gill, M., . . . Mors, O. (2004). Familiality of symptom dimensions in depression. *Archives of general psychiatry, 61*(5), 468-474.

Kovacs, M., & Lopez-Duran, N. (2010). Prodromal symptoms and atypical affectivity as predictors of major depression in juveniles: implications for prevention. *Journal of Child Psychology and Psychiatry, 51*(4), 472-496.

Kraaij, V., Pruymboom, E., & Garnefski, N. (2002). Cognitive coping and depressive symptoms in the elderly: a longitudinal study. *Aging & mental health, 6*(3), 275-281.

Kraemer, H. C., Stice, E., Kazdin, A., Offord, D., & Kupfer, D. (2001). How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *American Journal of Psychiatry, 158*(6), 848-856.

Kringelbach, M. L., & Rolls, E. T. (2003). Neural correlates of rapid reversal learning in a simple model of human social interaction. *Neuroimage, 20*(2), 1371-1383.

Kroencke, K., Spitzer, R., & Williams, J. (2001). The phq-9: Validity of a brief depression severity measure [Electronic version]. *Journal of General Internal Medicine, 16*(9), 606-613.

Kross, E., Ayduk, O., & Mischel, W. (2005). When asking "why" does not hurt distinguishing rumination from reflective processing of negative emotions. *Psychological science, 16*(9), 709-715.

Kuyken, W., & Dalgleish, T. (1995). Autobiographical memory and depression. *British Journal of Clinical Psychology, 34*(1), 89-92.

Kuyken, W., Howell, R., & Dalgleish, T. (2006). Overgeneral autobiographical memory in depressed adolescents with, versus without, a reported history of trauma. *Journal of abnormal psychology, 115*(3), 387.

Kwon, S.-M., & Oei, T. P. (1992). Differential causal roles of dysfunctional attitudes and automatic thoughts in depression. *Cognitive Therapy and Research, 16*(3), 309-328.

Kyte, Z. A., Goodyer, I. M., & Sahakian, B. J. (2005). Selected executive skills in adolescents with recent first episode major depression. *Journal of Child Psychology and Psychiatry, 46*(9), 995-1005.

Ladouceur, C. D., Dahl, R. E., Williamson, D. E., Birmaher, B., Ryan, N. D., & Casey, B. (2005). Altered emotional processing in pediatric anxiety, depression, and comorbid anxiety-depression. *Journal of Abnormal Child Psychology, 33*(2), 165-177.

LaGrange, B., Cole, D. A., Dallaire, D. H., Ciesla, J. A., Pineda, A. Q., Truss, A. E., & Folmer, A. (2008). Developmental changes in depressive cognitions: A longitudinal evaluation of the Cognitive Triad Inventory for Children. *Psychological Assessment, 20*(3), 217.

Lakdawalla, Z., Hankin, B. L., & Mermelstein, R. (2007). Cognitive theories of depression in children and adolescents: A conceptual and quantitative review. *Clinical child and family psychology review, 10*(1), 1-24.

Letkiewicz, A. M., Miller, G. A., Crocker, L. D., Warren, S. L., Infantolino, Z. P., Mimnaugh, K. J., & Heller, W. (2014). Executive function deficits in daily life prospectively predict increases in depressive symptoms. *Cognitive Therapy and Research, 38*(6), 612-620.

Leventhal, A. M., Chasson, G. S., Tapia, E., Miller, E. K., & Pettit, J. W. (2006). Measuring hedonic capacity in depression: a psychometric analysis of three anhedonia scales. *Journal of Clinical Psychology, 62*(12), 1545-1558.

Levinson, D. F. (2006). The genetics of depression: a review. *Biological psychiatry, 60*(2), 84-92.

Lewandowski, R. E., Verdeli, H., Wickramaratne, P., Warner, V., Mancini, A., & Weissman, M. (2014). Predictors of positive outcomes in offspring of depressed parents and non-depressed parents across 20 years. *Journal of child and family studies, 23*(5), 800-811.

Lewinsohn, P. M. (1985). 5. A Behavioral Approach to Depression'. In J. C. Coyne (Ed.), *Essential papers on depression* (pp. 150-180): New York University Press.

Lewinsohn, P. M., & Graf, M. (1973). Pleasant activities and depression. *Journal of consulting and clinical psychology, 41*(2), 261.

Lewinsohn, P. M., Hoberman, H., Teri, L., & Hautzinger, M. (1985). An integrative theory of depression. In S. Reiss & B. RR (Eds.), *Theoretical issues in behavior therapy*. New York: Academic Press.

Lewinsohn, P. M., Lobitz, W. C., & Wilson, S. (1973). " Sensitivity" of depressed individuals to aversive stimuli. *Journal of abnormal psychology, 81*(3), 259.

Lewinsohn, P. M., Olino, T. M., & Klein, D. N. (2005). Psychosocial impairment in offspring of depressed parents. *Psychological medicine, 35*(10), 1493-1503.

Lewinsohn, P. M., Rohde, P., & Seeley, J. R. (1998). Major depressive disorder in older adolescents: prevalence, risk factors, and clinical implications. *Clinical psychology review, 18*(7), 765-794.

Lewinsohn, P. M., & Talkington, J. (1979). Studies on the measurement of unpleasant events and relations with depression. *Applied Psychological Measurement, 3*(1), 83-101.

Lieb, R., Isensee, B., Höfler, M., Pfister, H., & Wittchen, H.-U. (2002). Parental major depression and the risk of depression and other mental disorders in offspring: a prospective-longitudinal community study. *Archives of general psychiatry, 59*(4), 365-374.

Lighthall, N. R., Sakaki, M., Vasunilashorn, S., Nga, L., Somayajula, S., Chen, E. Y., . . . Mather, M. (2012). Gender differences in reward-related decision processing under stress. *Social Cognitive and Affective Neuroscience, 7*(4), 476-484.

Liu, W.-h., Roiser, J. P., Wang, L.-z., Zhu, Y.-h., Huang, J., Neumann, D. L., . . . Chan, R. C. (2016). Anhedonia is associated with blunted reward sensitivity in first-degree relatives of patients with major depression. *Journal of Affective Disorders, 190*, 640-648.

London, B., Downey, G., Bonica, C., & Paltin, I. (2007). Social causes and consequences of rejection sensitivity. *Journal of Research on Adolescence, 17*(3), 481-506.

Lonigan, C. J., Phillips, B. M., & Hooe, E. S. (2003). Relations of positive and negative affectivity to anxiety and depression in children: evidence from a latent variable longitudinal study. *Journal of consulting and clinical psychology, 71*(3), 465.

Lovejoy, M. C., Graczyk, P. A., O'Hare, E., & Neuman, G. (2000). Maternal depression and parenting behavior: A meta-analytic review. *Clinical psychology review, 20*(5), 561-592.

Luking, K. R., Pagliaccio, D., Luby, J. L., & Barch, D. M. (2015). Child gain approach and loss avoidance behavior: relationships with depression risk, negative mood, and anhedonia. *Journal of the American Academy of Child & Adolescent Psychiatry, 54*(8), 643-651.

Lyons-Ruth, K., Wolfe, R., & Lyubchik, A. (2000). Depression and the parenting of young children: making the case for early preventive mental health services. *Harvard review of psychiatry, 8*(3), 148-153.

Maalouf, F. T., Brent, D., Clark, L., Tavitian, L., McHugh, R. M., Sahakian, B. J., & Phillips, M. L. (2011). Neurocognitive impairment in adolescent major depressive disorder: state vs. trait illness markers. *Journal of Affective Disorders, 133*(3), 625-632.

MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate behavioral research, 39*(1), 99-128.

MacLeod, C. M. (1991). Half a century of research on the Stroop effect: an integrative review. *Psychological bulletin, 109*(2), 163.

MacPhillamy, D. J., & Lewinsohn, P. M. (1982). The pleasant events schedule: Studies on reliability, validity, and scale intercorrelation. *Journal of consulting and clinical psychology, 50*(3), 363.

Mannie, Z., Williams, C., Browning, M., & Cowen, P. (2015). Decision making in young people at familial risk of depression. *Psychological Medicine, 45*(02), 375-380.

Mars, B., Collishaw, S., Hammerton, G., Rice, F., Harold, G. T., Smith, D., . . . Craddock, N. (2015). Longitudinal symptom course in adults with recurrent depression: Impact on impairment and risk of psychopathology in offspring. *Journal of affective disorders, 182*, 32-38.

Mars, B., Collishaw, S., Smith, D., Thapar, A., Potter, R., Sellers, R., . . . Thapar, A. (2012). Offspring of parents with recurrent depression: which features of parent depression index risk for offspring psychopathology? *Journal of Affective Disorders, 136*(1), 44-53.

Masten, A. S., & Coatsworth, J. D. (1998). The development of competence in favorable and unfavorable environments: Lessons from research on successful children. *American psychologist, 53*(2), 205.

Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annu. Rev. Clin. Psychol., 1*, 167-195.

Maxwell, M. E. (1992). Family Interview for Genetic Studies (FIGS): a manual for FIGS. *Clinical Neurogenetics Branch, Intramural Research Program, National Institute of Mental Health, Bethesda, MD*.

McAdams, D. P. (1985). Motivation and friendship. In S. Duck & D. Perlman (Eds.), (pp. 115-141). London: Sage.

McAdams, D. P. (1988). Personal needs and personal relationships. In S. Duck (Ed.), *Handbook of personal relationships* (pp. 7-22). New York: Wiley.

McCabe, C., Mishor, Z., Cowen, P. J., & Harmer, C. J. (2010). Diminished neural processing of aversive and rewarding stimuli during selective serotonin reuptake inhibitor treatment. *Biological psychiatry, 67*(5), 439-445.

McCollum, D. L. (2005). What Are the Social Values of College Students?: A Social Goals Approach. *Journal of College and Character, 6*(6).

McDermott, L. M., & Ebmeier, K. P. (2009). A meta-analysis of depression severity and cognitive function. *Journal of affective disorders, 119*(1), 1-8.
McDonald, K. L., Bowker, J. C., Rubin, K. H., Laursen, B., & Duchene, M. S. (2010). Interactions between rejection sensitivity and supportive relationships in the prediction of adolescents' internalizing difficulties. *Journal of Youth and Adolescence, 39*(5), 563-574.
McLeod, B. D., Weisz, J. R., & Wood, J. J. (2007). Examining the association between parenting and childhood depression: A meta-analysis. *Clinical psychology review, 27*(8), 986-1003.
McMakin, D. L., Olino, T. M., Porta, G., Dietz, L. J., Emslie, G., Clarke, G., . . . Birmaher, B. (2012). Anhedonia predicts poorer recovery among youth with selective serotonin reuptake inhibitor treatment–resistant depression. *Journal of the American Academy of Child & Adolescent Psychiatry, 51*(4), 404-411.
McRae, K., Jacobs, S. E., Ray, R. D., John, O. P., & Gross, J. J. (2012). Individual differences in reappraisal ability: Links to reappraisal frequency, well-being, and cognitive control. *Journal of Research in Personality, 46*(1), 2-7.
Mehrabian, A. (1972). *Nonverbal communication*: Transaction Publishers.
Mehrabian, A. (1976). Questionnaire measures of affiliative tendency and sensitivity to rejection. *Psychological reports, 38*(1), 199-209.
Melby-Lervåg, M., & Hulme, C. (2013). Is working memory training effective? A meta-analytic review. *Developmental psychology, 49*(2), 270.
Mellick, W., Sharp, C., & Ernst, M. (2015). Neuroeconomics for the Study of Social Cognition in Adolescent Depression. *Clinical psychology: Science and practice, 22*(3), 255-276.
Mezulis, A. H., Abramson, L. Y., Hyde, J. S., & Hankin, B. L. (2004). Is there a universal positivity bias in attributions? A meta-analytic review of individual, developmental, and cultural differences in the self-serving attributional bias. *Psychological bulletin, 130*(5), 711.
Micco, J. A., Henin, A., Biederman, J., Rosenbaum, J. F., Petty, C., Rindlaub, L. A., . . . Hirshfeld-Becker, D. R. (2009). Executive functioning in offspring at risk for depression and anxiety. *Depression and anxiety, 26*(9), 780-790.
Mill, J. S. (1843). A System of Logic. *London: Parker*.
Milne, B., Moffitt, T., Crump, R., Poulton, R., Rutter, M., Sears, M., . . . Caspi, A. (2008). How should we construct psychiatric family history scores? A comparison of alternative approaches from the Dunedin Family Health History Study. *Psychological medicine, 38*(12), 1793-1802.
Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive psychology, 41*(1), 49-100.
Moffitt, T. E. (1993). The neuropsychology of conduct disorder. *Development and psychopathology, 5*(1-2), 135-151.
Moons, K. G., Kengne, A. P., Woodward, M., Royston, P., Vergouwe, Y., Altman, D. G., & Grobbee, D. E. (2012). Risk prediction models: I. Development, internal validation, and assessing the incremental value of a new (bio) marker. *Heart, 98*(9), 683-690.
Moore, R. G., Watts, F. N., & Williams, J. M. G. (1988). The specificity of personal memories in depression. *British Journal of Clinical Psychology, 27*(3), 275-276.
Moore, S. A., & Zoellner, L. A. (2007). Overgeneral autobiographical memory and traumatic events: an evaluative review. *Psychological bulletin, 133*(3), 419.
Moretti, M., Fine, S., Haley, G., & Marriage, K. (1985). Childhood and adolescent depression: Child-report versus parent-report information. *Journal of the American Academy of Child Psychiatry, 24*(3), 298-302.

Morris, B. H., Bylsma, L. M., Yaroslavsky, I., Kovacs, M., & Rottenberg, J. (2015). Reward learning in pediatric depression and anxiety : preliminary findings in a high-risk sample. *Depression and anxiety, 32*(5), 373-381.

Muris, P., van der Pennen, E., Sigmond, R., & Mayer, B. (2008). Symptoms of anxiety, depression, and aggression in non-clinical children: Relationships with self-report and performance-based measures of attention and effortful control. *Child Psychiatry and Human Development, 39*(4), 455-467.

Murphy, F. C., Michael, A., & Sahakian, B. (2012). Emotion modulates cognitive flexibility in patients with major depression. *Psychological Medicine, 42*(07), 1373-1382.

Murphy, F. C., Rubinsztein, J. S., Michael, A., Rogers, R. D., Robbins, T. W., Paykel, E. S., & Sahakian, B. J. (2001). Decision-making cognition in mania and depression. *Psychological Medicine, 31*(04), 679-693.

Murphy, F. C., Sahakian, B., Rubinsztein, J., Michael, A., Rogers, R., Robbins, T., & Paykel, E. (1999). Emotional bias and inhibitory control processes in mania and depression. *Psychological Medicine, 29*(06), 1307-1321.

Murray, C. J., & Lopez, A. D. (1996). Evidence-based health policy--lessons from the Global Burden of Disease Study. *Science, 274*(5288), 740.

Neshat-Doost, H. T., Dalgleish, T., Yule, W., Kalantari, M., Ahmadi, S. J., Dyregrov, A., & Jobson, L. (2012). Enhancing autobiographical memory specificity through cognitive training an intervention for depression translated from basic science. *Clinical Psychological Science*, 2167702612454613.

Newman, D. L., Moffitt, T. E., Caspi, A., Magdol, L., Silva, P. A., & Stanton, W. R. (1996). Psychiatric disorder in a birth cohort of young adults: prevalence, comorbidity, clinical significance, and new case incidence from ages 11 to 21. *Journal of consulting and clinical psychology, 64*(3), 552.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of abnormal psychology, 100*(4), 569.

Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of abnormal psychology, 109*(3), 504.

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on psychological science, 3*(5), 400-424.

Nutt, D., Demyttenaere, K., Janka, Z., Aarre, T., Bourin, M., Canonico, P. L., . . . Stahl, S. (2007). The other face of depression, reduced positive affect: the role of catecholamines in causation and cure. *Journal of Psychopharmacology, 21*(5), 461-471.

Ochsner, K. N., & Gross, J. J. (2008). Cognitive emotion regulation insights from social cognitive and affective neuroscience. *Current Directions in Psychological Science, 17*(2), 153-158.

Office for National Statistics. (2009). *Social Trends*. (39). Newport: Palgrave Macmillan.

Olino, T. M., Silk, J. S., Osterritter, C., & Forbes, E. E. (2015). Social reward in youth at risk for depression: a preliminary investigation of subjective and neural differences. *Journal of child and adolescent psychopharmacology, 25*(9), 711-721.

Olioff, M., Bryson, S. E., & Wadden, N. P. (1989). Predictive relation of automatic thoughts and student efficacy to depressive symptoms in undergraduates. *Canadian Journal of Behavioural Science/Revue canadienne des sciences du comportement, 21*(4), 353.

Orvaschel, H., Thompson, W. D., Belanger, A., Prusoff, B. A., & Kidd, K. K. (1982). Comparison of the family history method to direct interview: factors affecting the diagnosis of depression. *Journal of Affective Disorders, 4*(1), 49-59.

Osman, A., Kopper, B. A., Barrios, F., Gutierrez, P. M., & Bagge, C. L. (2004). Reliability and validity of the Beck depression inventory--II with adolescent psychiatric inpatients. *Psychological Assessment, 16*(2), 120.

Pargas, R. C. M., Brennan, P. A., Hammen, C., & Le Brocque, R. (2010). Resilience to maternal depression in young adulthood. *Developmental psychology, 46*(4), 805.

Park, R. J., Goodyer, I., & Teasdale, J. (2002). Categoric overgeneral autobiographical memory in adolescents with major depressive disorder. *Psychological Medicine, 32*(02), 267-276.

Pearson, R. M., Evans, J., Kounali, D., Lewis, G., Heron, J., Ramchandani, P. G., . . . Stein, A. (2013). Maternal depression during pregnancy and the postnatal period: risks and possible mechanisms for offspring depression at age 18 years. *JAMA psychiatry, 70*(12), 1312-1319.

Phan, K. L., Sripada, C. S., Angstadt, M., & McCabe, K. (2010). Reputation for reciprocity engages the brain reward center. *Proceedings of the National Academy of Sciences, 107*(29), 13099-13104.

Piccinelli, M., & Wilkinson, G. (2000). Gender differences in depression. *The British Journal of Psychiatry, 177*(6), 486-492.

Pizzagalli, D. A. (2014). Depression, stress, and anhedonia: toward a synthesis and integrated model. *Annual review of clinical psychology, 10*, 393.

Pizzagalli, D. A., Iosifescu, D., Hallett, L. A., Ratner, K. G., & Fava, M. (2008). Reduced hedonic capacity in major depressive disorder: evidence from a probabilistic reward task. *Journal of psychiatric research, 43*(1), 76-87.

Pizzagalli, D. A., Jahn, A. L., & O'Shea, J. P. (2005). Toward an objective characterization of an anhedonic phenotype: a signal-detection approach. *Biological psychiatry, 57*(4), 319-327.

Podsakoff, P. M., MacKenzie, S. B., Lee, J.-Y., & Podsakoff, N. P. (2003). Common method biases in behavioral research: a critical review of the literature and recommended remedies. *Journal of applied psychology, 88*(5), 879.

Pollock, L. R., & Williams, J. M. G. (2001). Effective problem solving in suicide attempters depends on specific autobiographical recall. *Suicide and Life-Threatening Behavior, 31*(4), 386-396.

Price, J., Cole, V., & Goodwin, G. M. (2009). Emotional side-effects of selective serotonin reuptake inhibitors: qualitative study. *The British Journal of Psychiatry, 195*(3), 211-217.

Radke-Yarrow, M., & Sherman, T. (1990). Hard growing: Children who survive.

Raes, F., Hermans, D., de Decker, A., Eelen, P., & Williams, J. M. G. (2003). Autobiographical memory specificity and affect regulation: an experimental approach. *Emotion, 3*(2), 201.

Raes, F., Hermans, D., Williams, J. M. G., Demyttenaere, K., Sabbe, B., Pieters, G., & Eelen, P. (2005). Reduced specificity of autobiographical memory: A mediator between rumination and ineffective social problem-solving in major depression? *Journal of affective disorders, 87*(2), 331-335.

Raes, F., Williams, J. M. G., & Hermans, D. (2009). Reducing cognitive vulnerability to depression: A preliminary investigation of MEmory Specificity Training (MEST) in inpatients with depressive symptomatology. *Journal of behavior therapy and experimental psychiatry, 40*(1), 24-38.

Rapport, M. D., Orban, S. A., Kofler, M. J., & Friedman, L. M. (2013). Do programs designed to train working memory, other executive functions, and attention benefit children with ADHD? A meta-analytic review of cognitive, academic, and behavioral outcomes. *Clinical psychology review, 33*(8), 1237-1252.

Rawal, A., Collishaw, S., Thapar, A., & Rice, F. (2013). 'The risks of playing it safe': a prospective longitudinal study of response to reward in the adolescent offspring of depressed parents. *Psychological Medicine, 43*(1), 27-38.

Rawal, A., & Rice, F. (2012). Examining overgeneral autobiographical memory as a risk factor for adolescent depression. *Journal of the American Academy of Child & Adolescent Psychiatry, 51*(5), 518-527.

Reeb-Sutherland, B. C., Levitt, P., & Fox, N. A. (2012). The predictive nature of individual differences in early associative learning and emerging social behavior. *PloS one, 7*(1), e30511.

Regard, M., Strauss, E., & Knapp, P. (1982). Children's production on verbal and non-verbal fluency tasks. *Perceptual and motor skills, 55*(3), 839-844.

Rice, F., Harold, G., & Thapar, A. (2002). The genetic aetiology of childhood depression: a review. *Journal of Child Psychology and Psychiatry, 43*(1), 65-79.

Rice, F., Harold, G. T., & Thapar, A. (2003). Negative life events as an account of age-related differences in the genetic aetiology of depression in childhood and adolescence. *Journal of Child Psychology and Psychiatry, 44*(7), 977-987.

Rice, F., Lifford, K. J., Thomas, H. V., & Thapar, A. (2007). Mental health and functional outcomes of maternal and adolescent reports of adolescent depressive symptoms. *Journal of the American Academy of Child & Adolescent Psychiatry, 46*(9), 1162-1170.

Rice, F., & Rawal, A. (2010). Can basic risk research help in the prevention of childhood and adolescent depression? Examining a cognitive and emotional regulation approach. *Depression research and treatment, 2011*.

Rice, F., Rawal, A., Riglin, L., Lewis, G., Lewis, G., & Dunsmuir, S. (2015). Examining reward-seeking, negative self-beliefs and over-general autobiographical memory as mechanisms of change in classroom prevention programs for adolescent depression. *Journal of affective disorders, 186*, 320-327.

Richards, D. A., Ekers, D., McMillan, D., Taylor, R. S., Byford, S., Warren, F. C., . . . Kuyken, W. (2016). Cost and Outcome of Behavioural Activation versus Cognitive Behavioural Therapy for Depression (COBRA): a randomised, controlled, non-inferiority trial. *The Lancet, 388*(10047), 871-880.

Riglin, L., Collishaw, S., Shelton, K. H., McManus, I., Ng-Knight, T., Sellers, R., . . . Rice, F. (2016). Higher cognitive ability buffers stress-related depressive symptoms in adolescent girls. *Development and psychopathology, 28*(01), 97-109.

Riglin, L., Thapar, A., Shelton, K. H., Langley, K., Frederickson, N., & Rice, F. (2016). Profiling depression in childhood and adolescence: the role of conduct problems. *Journal of Child Psychology and Psychiatry, 57*(4), 481-490.

Rijsdijk, F., Riese, H., Tops, M., Snieder, H., Brouwer, W., Smid, H., & Ormel, J. (2009). Neuroticism, recall bias and attention bias for valenced probes: a twin study. *Psychological Medicine, 39*(01), 45-54.

Robinson, M. S., & Alloy, L. B. (2003). Negative cognitive styles and stress-reactive rumination interact to predict depression: A prospective study. *Cognitive Therapy and Research, 27*(3), 275-291.

Rohde, P., Lewinsohn, P. M., & Seeley, J. R. (1994). Are adolescents changed by an episode of major depression? *Journal of the American Academy of Child & Adolescent Psychiatry, 33*(9), 1289-1298.

Roiser, J. P., Elliott, R., & Sahakian, B. J. (2012). Cognitive mechanisms of treatment in depression. *Neuropsychopharmacology, 37*(1), 117-136.

Rolls, E. T. (2000). On the brain and emotion. *Behavioral and brain sciences, 23*(02), 219-228.

Rothman, K. J. (1990). No adjustments are needed for multiple comparisons. *Epidemiology, 1*(1), 43-46.

Rothman, K. J. (2014). Six persistent research misconceptions. *Journal of general internal medicine, 29*(7), 1060-1064.

Rottenberg, J., Kasch, K. L., Gross, J. J., & Gotlib, I. H. (2002). Sadness and amusement reactivity differentially predict concurrent and prospective functioning in major depressive disorder. *Emotion, 2*(2), 135.

Rudolph, K. D. (2002). Gender differences in emotional responses to interpersonal stress during adolescence. *Journal of Adolescent Health, 30*(4), 3-13.

Rusting, C. L., & DeHart, T. (2000). Retrieving positive memories to regulate negative mood: consequences for mood-congruent memory. *Journal of personality and social psychology, 78*(4), 737.

Rutter, M. (1985). Resilience in the face of adversity. Protective factors and resistance to psychiatric disorder. *The British Journal of Psychiatry, 147*(6), 598-611.

Rutter, M. (2007). Proceeding from observed correlation to causal inference: The use of natural experiments. *Perspectives on psychological science, 2*(4), 377-395.

Rutter, M. (2013). Annual research review: resilience–clinical implications. *Journal of Child Psychology and Psychiatry, 54*(4), 474-487.

Rutter, M., Kim-Cohen, J., & Maughan, B. (2006). Continuities and discontinuities in psychopathology between childhood and adult life. *Journal of Child Psychology and Psychiatry, 47*(3-4), 276-295.

Rutter, M., Pickles, A., Murray, R., & Eaves, L. (2001). Testing hypotheses on specific environmental causal effects on behavior. *Psychological bulletin, 127*(3), 291.

Sanchez, A., Vazquez, C., Marker, C., LeMoult, J., & Joormann, J. (2013). Attentional disengagement predicts stress recovery in depression: An eye-tracking study. *Journal of abnormal psychology, 122*(2), 303.

Santini, Z. I., Koyanagi, A., Tyrovolas, S., Mason, C., & Haro, J. M. (2015). The association between social relationships and depression: A systematic review. *Journal of affective disorders, 175*, 53-65.

Schartau, P. E., Dalgleish, T., & Dunn, B. D. (2009). Seeing the bigger picture: training in perspective broadening reduces self-reported affect and psychophysiological response to distressing films and autobiographical memories. *Journal of abnormal psychology, 118*(1), 15.

Scher, C. D., Ingram, R. E., & Segal, Z. V. (2005). Cognitive reactivity and vulnerability: Empirical evaluation of construct activation and cognitive diatheses in unipolar depression. *Clinical psychology review, 25*(4), 487-510.

Schless, A. P., Schwartz, L., Goetz, C., & Mendels, J. (1974). How depressives view the significance of life events. *The British Journal of Psychiatry, 125*(587), 406-410.

Schmeichel, B. J., & Tang, D. (2014). The relationship between individual differences in executive functioning and emotion regulation: A comprehensive review. *Motivation and Its Regulation: The Control Within*, 133-151.

Schultz, W. (2000). Multiple reward signals in the brain. *Nature Reviews Neuroscience, 1*(3), 199-207.

Sellers, R., Collishaw, S., Rice, F., Thapar, A. K., Potter, R., Mars, B., . . . Craddock, N. (2013). Risk of psychopathology in adolescent offspring of mothers with psychopathology and recurrent depression. *The British Journal of Psychiatry, 202*(2), 108-114.

Sellers, R., Harold, G. T., Elam, K., Rhoades, K. A., Potter, R., Mars, B., . . . Collishaw, S. (2014). Maternal depression and co-occurring antisocial behaviour: testing maternal hostility and warmth as mediators of risk for offspring psychopathology. *Journal of Child Psychology and Psychiatry, 55*(2), 112-120.

Sharot, T. (2011). The optimism bias. *Current Biology, 21*(23), R941-R945.

Sharp, C., Monterosso, J., & Montague, P. R. (2012). Neuroeconomics: a bridge for translational research. *Biological psychiatry, 72*(2), 87-92.
Sheehan, D., & Lecrubier, Y. (2010). MINI International Neuropsychiatric Interview English Version 6.0. 0. *Sheehan DV & Lecrubier Y*.
Shelton, R. C., & Tomarken, A. J. (2001). Can recovery from depression be achieved? *Psychiatric Services, 52*(11), 1469-1478.
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.
Siegle, G. J., Ghinassi, F., & Thase, M. E. (2007). Neurobehavioral therapies in the 21st century: Summary of an emerging field and an extended example of cognitive control training for depression. *Cognitive Therapy and Research, 31*(2), 235-262.
Silberg, J., Pickles, A., Rutter, M., Hewitt, J., Simonoff, E., Maes, H., . . . Eaves, L. (1999). The influence of genetic factors and life stress on depression among adolescent girls. *Archives of general psychiatry, 56*(3), 225-232.
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.
Silk, J. S., Steinberg, L., & Morris, A. S. (2003). Adolescents' emotion regulation in daily life: Links to depressive symptoms and problem behavior. *Child development, 74*(6), 1869-1880.
Singh, A., D'Onofrio, B., Slutske, W., Turkheimer, E., Emery, R., Harden, K., . . . Martin, N. (2011). Parental depression and offspring psychopathology: a Children of Twins study. *Psychological Medicine, 41*(07), 1385-1395.
Sloan, D. M., Strauss, M. E., Quirk, S. W., & Sajatovic, M. (1997). Subjective and expressive emotional responses in depression. *Journal of affective disorders, 46*(2), 135-141.
Sloan, D. M., Strauss, M. E., & Wisner, K. L. (2001). Diminished response to pleasant stimuli by depressed women. *Journal of abnormal psychology, 110*(3), 488.
Smoski, M. J., Lynch, T. R., Rosenthal, M. Z., 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.
Snaith, R., Hamilton, M., Morley, S., Humayan, A., Hargreaves, D., & Trigwell, P. (1995). A scale for the assessment of hedonic tone the Snaith-Hamilton Pleasure Scale. *The British Journal of Psychiatry, 167*(1), 99-103.
Snyder, H. R. (2013). Major depressive disorder is associated with broad impairments on neuropsychological measures of executive function: A meta-analysis and review: American Psychological Association.
Southwick, S. M., Vythilingam, M., & Charney, D. S. (2005). The psychobiology of depression and resilience to stress: implications for prevention and treatment*. *Annu. Rev. Clin. Psychol., 1*, 255-291.
Spear, L. P. (2011). Rewards, aversions and affect in adolescence: emerging convergences across laboratory animal and human data. *Developmental cognitive neuroscience, 1*(4), 390-403.
Spijker, J., Bijl, R., De Graaf, R., & Nolen, W. (2001). Determinants of poor 1-year outcome of DSM-III-R major depression in the general population: results of the Netherlands Mental Health Survey and Incidence Study (NEMESIS). *Acta Psychiatrica Scandinavica, 103*(2), 122-130.
Stahl, L., & Pry, R. (2005). Attentional flexibility and perseveration: Developmental aspects in young children. *Child Neuropsychology, 11*(2), 175-189.
Stallard, P., Sayal, K., Phillips, R., Taylor, J. A., Spears, M., Anderson, R., . . . Montgomery, A. A. (2012). Classroom based cognitive behavioural therapy in reducing symptoms of depression in high risk adolescents: pragmatic cluster randomised controlled trial.

Steinberg, L. (2007). Risk taking in adolescence new perspectives from brain and behavioral science. *Current directions in psychological science, 16*(2), 55-59.
Stice, E., Shaw, H., Bohon, C., Marti, C. N., & Rohde, P. (2009). A meta-analytic review of depression prevention programs for children and adolescents: factors that predict magnitude of intervention effects. *Journal of consulting and clinical psychology, 77*(3), 486.
Stöber, J., & Borkovec, T. (2002). Reduced concreteness of worry in generalized anxiety disorder: Findings from a therapy study. *Cognitive Therapy and Research, 26*(1), 89-96.
Stott, R., & Salkovskis, P. (2010). *Oxford guide to metaphors in CBT: Building cognitive bridges*: Oxford University Press.
Strathearn, L., Fonagy, P., Amico, J., & Montague, P. R. (2009). Adult attachment predicts maternal brain and oxytocin response to infant cues. *Neuropsychopharmacology, 34*(13), 2655-2666.
Stuss, D., Levine, B., Alexander, M., Hong, J., Palumbo, C., Hamer, L., . . . Izukawa, D. (2000). Wisconsin Card Sorting Test performance in patients with focal frontal and posterior brain damage: effects of lesion location and test structure on separable cognitive processes. *Neuropsychologia, 38*(4), 388-402.
Suchy, Y. (2009). Executive functioning: Overview, assessment, and research issues for non-neuropsychologists. *Annals of behavioral medicine, 37*(2), 106-116.
Sumner, J. A., Griffith, J. W., & Mineka, S. (2010). Overgeneral autobiographical memory as a predictor of the course of depression: A meta-analysis. *Behaviour research and therapy, 48*(7), 614-625.
Swales, M. A., Williams, J. M. G., & Wood, P. (2001). Specificity of autobiographical memory and mood disturbance in adolescents. *Cognition & Emotion, 15*(3), 321-331.
Tamir, D. I., & Mitchell, J. P. (2012). Disclosing information about the self is intrinsically rewarding. *Proceedings of the National Academy of Sciences, 109*(21), 8038-8043.
Tarabulsy, G. M., Tessier, R., & Kappas, A. (1996). Contingency detection and the contingent organization of behavior in interactions: implications for socioemotional development in infancy. *Psychological bulletin, 120*(1), 25.
Teasdale, J. D. (1988). Cognitive vulnerability to persistent depression. *Cognition & Emotion, 2*(3), 247-274.
Teo, A. R., Choi, H., & Valenstein, M. (2013). Social relationships and depression: ten-year follow-up from a nationally representative study. *PloS one, 8*(4), e62396.
Thapar, A., Collishaw, S., Pine, D. S., & Thapar, A. K. (2012). Depression in adolescence. *The Lancet, 379*(9820), 1056-1067.
Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the society for research in child development, 59*(2-3), 25-52.
Titz, C., & Karbach, J. (2014). Working memory and executive functions: effects of training on academic achievement. *Psychological research, 78*(6), 852-868.
Torrubia, R., Avila, C., Moltó, J., & Caseras, X. (2001). The Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ) as a measure of Gray's anxiety and impulsivity dimensions. *Personality and individual differences, 31*(6), 837-862.
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.
Treadway, M. T., Buckholtz, J. W., Schwartzman, A. N., Lambert, W. E., & Zald, D. H. (2009). Worth the 'EEfRT'? The effort expenditure for rewards task as an objective measure of motivation and anhedonia. *PloS one, 4*(8), e6598.

Treadway, M. T., & Zald, D. H. (2011). Reconsidering anhedonia in depression: lessons from translational neuroscience. *Neuroscience & Biobehavioral Reviews, 35*(3), 537-555.
Tully, E. C., Iacono, W. G., & McGue, M. (2008). An adoption study of parental depression as an environmental liability for adolescent depression and childhood disruptive disorders. *American Journal of Psychiatry, 165*(9), 1148-1154.
Turner, J. E., & Cole, D. A. (1994). Developmental differences in cognitive diatheses for child depression. *Journal of Abnormal Child Psychology, 22*(1), 15-32.
Turner, M. A. (1999). Generating Novel Ideas: Fluency Performance in High-functioning and Learning Disabled Individuals with Autism. *Journal of Child Psychology and Psychiatry, 40*(2), 189-201.
Tweed, D. L. (1993). Depression-related impairment: estimating concurrent and lingering effects. *Psychological medicine, 23*(02), 373-386.
Üstün, T., Ayuso-Mateos, J. L., Chatterji, S., Mathers, C., & Murray, C. J. (2004). Global burden of depressive disorders in the year 2000. *The British Journal of Psychiatry, 184*(5), 386-392.
Valentino, K. (2011). A developmental psychopathology model of overgeneral autobiographical memory. *Developmental Review, 31*(1), 32-54.
Valentino, K., Bridgett, D. J., Hayden, L. C., & Nuttall, A. K. (2012). Abuse, depressive symptoms, executive functioning, and overgeneral memory among a psychiatric sample of children and adolescents. *Journal of Clinical Child & Adolescent Psychology, 41*(4), 491-498.
Van de Mortel, T. F. (2008). Faking it: social desirability response bias in self-report research. *Australian Journal of Advanced Nursing, The, 25*(4), 40.
Van der Meer, M., Dixon, A., & Rose, D. (2008). Parent and child agreement on reports of problem behaviour obtained from a screening questionnaire, the SDQ. *European child & adolescent psychiatry, 17*(8), 491-497.
Van Leijenhorst, L., Westenberg, P. M., & Crone, E. A. (2008). A developmental study of risky decisions on the cake gambling task: age and gender analyses of probability estimation and reward evaluation. *Developmental neuropsychology, 33*(2), 179-196.
van Vreeswijk, M. F., & de Wilde, E. J. (2004). Autobiographical memory specificity, psychopathology, depressed mood and the use of the Autobiographical Memory Test: A meta-analysis. *Behaviour research and therapy, 42*(6), 731-743.
Vasey, M. W., Dalgleish, T., & Silverman, W. K. (2003). Research on information-processing factors in child and adolescent psychopathology: A critical commentary. *Journal of Clinical Child and Adolescent Psychology, 32*(1), 81-93.
Vrielynck, N., Deplus, S., & Philippot, P. (2007). Overgeneral autobiographical memory and depressive disorder in children. *Journal of Clinical Child and Adolescent Psychology, 36*(1), 95-105.
Vrieze, E., Pizzagalli, D. A., Demyttenaere, K., Hompes, T., Sienaert, P., de Boer, P., . . . Claes, S. (2013). Reduced reward learning predicts outcome in major depressive disorder. *Biological psychiatry, 73*(7), 639-645.
Wagner, C., Abramson, L. Y., & Alloy, L. B. (2015). Brief report: Executive functions in adolescent offspring of mothers with a history of depression. *Journal of adolescence, 44*, 32-36.
Wagner, S., Doering, B., Helmreich, I., Lieb, K., & Tadić, A. (2012). A meta-analysis of executive dysfunctions in unipolar major depressive disorder without psychotic symptoms and their changes during antidepressant treatment. *Acta Psychiatrica Scandinavica, 125*(4), 281-292.
Wagner, S., Müller, C., Helmreich, I., Huss, M., & Tadić, A. (2015). A meta-analysis of cognitive functions in children and adolescents with major depressive disorder. *European child & adolescent psychiatry, 24*(1), 5-19.

Wang, P. S., Beck, A. L., Berglund, P., McKenas, D. K., Pronk, N. P., Simon, G. E., & Kessler, R. C. (2014). Effects of major depression on moment-in-time work performance. *American Journal of Psychiatry*.

Watkins, E., & Brown, R. (2002). Rumination and executive function in depression: An experimental study. *Journal of Neurology, Neurosurgery & Psychiatry, 72*(3), 400-402.

Watkins, E., Moberly, N. J., & Moulds, M. L. (2008). Processing mode causally influences emotional reactivity: distinct effects of abstract versus concrete construal on emotional response. *Emotion, 8*(3), 364.

Watkins, E., & Moulds, M. (2005). Distinct modes of ruminative self-focus: impact of abstract versus concrete rumination on problem solving in depression. *Emotion, 5*(3), 319.

Watson, D., & Friend, R. (1969). Measurement of social-evaluative anxiety. *Journal of consulting and clinical psychology, 33*(4), 448.

Wechsler, D. (1999). *Wechsler abbreviated scale of intelligence*: Psychological Corporation.

Wechsler, D. (2003). Wechsler intelligence scale for children–Fourth Edition (WISC-IV). *San Antonio, TX: The Psychological Corporation*.

Weil, L. G., Fleming, S. M., Dumontheil, I., Kilford, E. J., Weil, R. S., Rees, G., . . . Blakemore, S.-J. (2013). The development of metacognitive ability in adolescence. *Consciousness and Cognition, 22*(1), 264-271.

Weissman, M. M., Paykel, E. S., Siegel, R., & Klerman, G. L. (1971). The social role performance of depressed women: Comparisons with a normal group. *American Journal of Orthopsychiatry, 41*(3), 390.

Weissman, M. M., Warner, V., Wickramaratne, P., Moreau, D., & Olfson, M. (1997). Offspring of depressed parents: 10 years later. *Archives of general psychiatry, 54*(10), 932-940.

Weissman, M. M., Wickramaratne, P., Adams, P., Wolk, S., Verdeli, H., & Olfson, M. (2000). Brief screening for family psychiatric history: the family history screen. *Archives of General Psychiatry, 57*(7), 675-682.

Weissman, M. M., Wickramaratne, P., Gameroff, M. J., Warner, V., Pilowsky, D., Kohad, R. G., . . . Talati, A. (2016). Offspring of Depressed Parents: 30 Years Later. *American Journal of Psychiatry*.

Weissman, M. M., Wickramaratne, P., Merikangas, K. R., Leckman, J. F., Prusoff, B. A., Caruso, K. A., . . . Gammon, G. D. (1984). Onset of major depression in early adulthood: increased familial loading and specificity. *Archives of general psychiatry, 41*(12), 1136-1143.

Weissman, M. M., Wickramaratne, P., Nomura, Y., Warner, V., Pilowsky, D., & Verdeli, H. (2006). Offspring of depressed parents: 20 years later. *American Journal of Psychiatry*.

Weisz, J. R., McCarty, C. A., & Valeri, S. M. (2006). Effects of psychotherapy for depression in children and adolescents: a meta-analysis. *Psychological bulletin, 132*(1), 132.

Werner-Seidler, A., & Moulds, M. L. (2011). Autobiographical memory characteristics in depression vulnerability: formerly depressed individuals recall less vivid positive memories. *Cognition & Emotion, 25*(6), 1087-1103.

Werner-Seidler, A., & Moulds, M. L. (2012). Characteristics of self-defining memory in depression vulnerability. *Memory, 20*(8), 935-948.

Whelan, R., & McHugh, L. A. (2009). Temporal discounting of hypothetical monetary rewards by adolescents, adults, and older adults. *The Psychological Record, 59*(2), 247.

Wickramaratne, P. J., & Weissman, M. M. (1998). Onset of psychopathology in offspring by developmental phase and parental depression. *Journal of the American Academy of Child & Adolescent Psychiatry, 37*(9), 933-942.

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: a meta-analytic review. *Biological psychiatry, 57*(11), 1336-1346.

Williams, J. M. G. (1996). Depression and the specificity of autobiographical memory In D. C. Rubin (Ed.), *Remembering our past: Studies in autobiographical memory* (pp. 244–267). Cambridge: Cambridge University Press.

Williams, J. M. G., Barnhofer, T., Crane, C., Herman, D., Raes, F., Watkins, E., & Dalgleish, T. (2007). Autobiographical memory specificity and emotional disorder. *Psychological bulletin, 133*(1), 122.

Williams, J. M. G., & Broadbent, K. (1986). Autobiographical memory in suicide attempters. *Journal of abnormal psychology, 95*(2), 144.

Williams, J. M. G., & Scott, J. (1988). Autobiographical memory in depression. *Psychological Medicine, 18*(3), 689-695.

Willner, P., & Healy, S. (1994). Decreased hedonic responsiveness during a brief depressive mood swing. *Journal of affective disorders, 32*(1), 13-20.

Wilson, J. K., & Rapee, R. M. (2005). The interpretation of negative social events in social phobia with versus without comorbid mood disorder. *Journal of anxiety disorders, 19*(3), 245-274.

Wing, J. K., Babor, T., Brugha, T., Burke, J., Cooper, J., Giel, R., . . . Sartorius, N. (1990). SCAN: Schedules for Clinical Assessment in Neuropsychiatry. *Archives of general psychiatry, 47*(6), 589-593.

Woody, M. L., Burkhouse, K. L., & Gibb, B. E. (2015). Overgeneral autobiographical memory in children of depressed mothers. *Cognition and Emotion, 29*(1), 130-137.

World Health Organization. (2015). *International statistical classification of diseases and related health problems* (10th ed.). Retrieved from http://apps.who.int/classifications/icd10/browse/2015/en

Yang, X.-h., Huang, J., Zhu, C.-y., Wang, Y.-f., Cheung, E. F., Chan, R. C., & Xie, G.-r. (2014). Motivational deficits in effort-based decision making in individuals with subsyndromal depression, first-episode and remitted depression patients. *Psychiatry research, 220*(3), 874-882.

Young, K. D., Bellgowan, P. S., Bodurka, J., & Drevets, W. C. (2013). Behavioral and neurophysiological correlates of autobiographical memory deficits in patients with depression and individuals at high risk for depression. *JAMA psychiatry, 70*(7), 698-708.

Young, K. D., Erickson, K., & Drevets, W. C. (2012). Match between cue and memory valence during autobiographical memory recall in depression. *Psychological reports, 111*(1), 129-148.

Zacks, R. T., & Hasher, L. (1994). Directed ignoring: Inhibitory regulation of working memory.

Zammit, S., Allebeck, P., David, A. S., Dalman, C., Hemmingsson, T., Lundberg, I., & Lewis, G. (2004). A longitudinal study of premorbid IQ score and risk of developing schizophrenia, bipolar disorder, severe depression, and other nonaffective psychoses. *Archives of general psychiatry, 61*(4), 354-360.

Zook, N. A., Davalos, D. B., DeLosh, E. L., & Davis, H. P. (2004). Working memory, inhibition, and fluid intelligence as predictors of performance on Tower of Hanoi and London tasks. *Brain and cognition, 56*(3), 286-292.