LONDON'S GLOBAL UNIVERSITY

Social information processing in adolescence: Gender differences and associations with depressive symptoms

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PhD Thesis

January 2021

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Declaration

I, Jessica Bone, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in my thesis.

Signature **Date** 8th January 2021

Abstract

There is a sharp increase in depression in girls in mid-adolescence, but we do not understand why this occurs. Cognitive theories suggest that people with depression have more dysfunctional attitudes and negative biases in social information processing (perceiving, interpreting, and remembering their social environment). In my thesis, I tested the hypothesis that these negative cognitions contribute to the gender difference in depression during adolescence. I examined whether girls have more negative biases in social information processing and dysfunctional attitudes than boys, and whether biases are associated with depressive symptoms in early and mid-adolescence.

I collected data from 331 young and 268 mid-adolescents (aged 11-13 and 13-15 years). In chapter 3, I tested whether learning about social evaluation differed across genders or age groups. I then developed and validated computational models of learning about social evaluation in chapter 4. In chapter 5, I tested whether recall biases were more negative in young and mid-adolescent girls and were associated with depressive symptoms. In these chapters, I found that negative biases in learning about social evaluation and recall were associated with increased depressive symptoms in young and mid-adolescents. There was no strong evidence for gender differences in social information processing. In chapter 6, I tested whether there were gender differences in different domains of dysfunctional attitudes. I found that perfectionism was associated with more severe depressive symptoms across adolescence and girls had higher perfectionism than boys in mid-adolescence.

My findings suggest that social information processing is not more negatively biased in girls than boys during adolescence, although girls may have more perfectionism than boys. Negative biases in learning about social evaluation, recall of social evaluation, and perfectionism were associated with depressive symptoms from early adolescence. These negative biases may be a risk factor for depression and present a good candidate for future longitudinal studies.

Impact statement

Depression is twice as common in women as in men and this gender difference emerges during adolescence. Before age 11, the incidence of depression is very similar in boys and girls, but, around age 12-13, the incidence of depression increases sharply in girls. However, we do not understand why this occurs. In my thesis, I have addressed this knowledge gap, contributing to the academic literature in several ways.

I proposed a novel hypothesis to explain the gender difference in depression, linking ideas from different fields. This provides a testable theoretical model for future investigation. To test my hypothesis, I developed two cognitive tasks measuring aspects of information processing which had not previously been studied in adolescence. These novel tasks could be incorporated into future large cohort studies of adolescent mental health. I performed preliminary tests of my hypothesis, which indicated that these two aspects of social information processing did not demonstrate gender differences. However, I did find evidence for gender differences in perfectionism, providing a potential focus for future longitudinal research. Additionally, I found evidence that negative biases in perfectionism and social information processing, measured using my novel cognitive tasks, were associated with increased depressive symptoms. This extends the body of evidence on potential risk factors for depression in adolescence. Longitudinal studies which combine approaches from developmental cognitive neuroscience, epidemiology, and computational psychiatry are now needed. This would allow the application of rigorous methodology from epidemiology to test mechanistic factors which are the focus of developmental cognitive neuroscience and computational psychiatry.

My findings have implications for the treatment and prevention of depression. Cognitive vulnerability from the biases I identified might be a way to reduce and prevent depressive symptoms in adolescence. Researchers could develop interventions that directly target biases in social information processing, and clinicians could focus more specifically on perfectionism during therapy. My findings also have implications for mental health and education policy. Negative biases in learning about and recall of social evaluation and perfectionism could influence adolescents' motivation and achievement in school. Targeting these processes in

health promotion and education policies could enable improvements in future academic, social, and economic success.

I have published versions of two thesis chapters in high impact peer-reviewed journals. I am also preparing two other papers for publication. I collaborated on an additional published paper using data from my thesis (Appendix 2). I have presented findings from my thesis at international conferences including Aegina Summer School on Social Cognition (Greece) and Flux Congress (New York). I had planned presentations at the British Association for Psychopharmacology Summer Meeting (London) and European Psychiatric Association Section of Epidemiology & Social Psychiatry (Cambridge) but these were cancelled due to covid-19. I presented my research to undergraduates at UCL Summer School. Finally, I aimed to disseminate my findings more widely by: interviewing for a general interest blog; collaborating with teachers; giving assemblies in schools; and presenting at Soapbox Science (although this was cancelled due to covid-19).

Acknowledgements

I am extremely grateful to my primary and secondary supervisors, Glyn Lewis and Gemma Lewis, for their continuous support and mentorship. I appreciate the encouragement they have provided, challenging my ideas and development as a researcher, and all of the interesting discussions we have had. Thank you also to my tertiary supervisors, Sarah-Jayne Blakemore and Jonathan Roiser, for also providing consistent support, guidance, and input into my PhD. I am also grateful for having been able to attend Sarah and Jon's lab meetings throughout my PhD, which have been a useful source of discussion, ideas, and feedback.

I would like to thank everyone that I have collaborated with during my PhD. In particular, I appreciate the input of Kate Button and Katie Hobbs in the development of the social evaluation learning task and in giving me the opportunity to collaborate on their systematic review. I would also like to thank Alex Pike and Vincent Valton for their extensive support in the development of my computational models.

My PhD research was made possible through a grant from the Economic and Social Research Council. I would also like to thank my participant advisory group, who assisted with the development of my research study. I am very grateful to the teachers and administrative staff in schools who made my research feasible. I would like to sincerely thank all of the parents/carers who provided consent for their children to participate in my research study, and all of the participants for their time and contribution to my study. Thank you also to the UCL MSc and PhD students who assisted with my data collection, particularly those who stepped in at short notice.

I would also like to thank all of my colleagues at UCL. I greatly appreciate the feedback and ideas of everyone in Glyn's research group and Jon and Sarah's labs. Thanks to Maddy Moses-Payne and Jack Andrews for their support, advice, laughs, and inability to say no. Also, thanks to everyone in the Division of Psychiatry for always providing the help I need, as well as welcome distractions, tea breaks and endless snacks. In particular, thank you to Tayla McCloud, Hannah Scott, Dora Stefanidou, Jean Stafford, Tom Steare, Aaron Kandola, Daisy McInnerney, Jess Rees, and Pepsi Reilly. I am also grateful to Rick Adams for providing useful feedback as my PhD upgrade examiner.

Thanks also to my brilliant friends who have supported me throughout my PhD. To the members of UCL Boat Club, for always providing a distraction from my PhD (whether I wanted it or not), especially my Henley crew, Laura Riggall, Jazz Michalowska, and Claudia Abdallah. I would also like to thank the best friends for always being there - in alphabetical order - Caitlin Turner, Ffion Bevan, Helen Potts, Iisha Williams, Nick de Mulder, Pete Budden, Rhys Cumming, and Tom Bailey. Thank you for never doubting my productivity, this thesis would not have been possible without you. Thank you also to my wonderful flatmate Rosie Shields, for endless words of wisdom, generally being great, and keeping me going through a global pandemic and lockdown. Finally, thank you to my amazing family for their constant support and encouragement. In particular, I would like to thank my Mum and Dad for providing the best advice, whenever I need it, and never failing to believe in me.

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List of acronyms

AIC: Akaike Information Criterion
ALSPAC: Avon Longitudinal Study of Parents and Children
ARI-S: Affective Reactivity Index
BIC: Bayesian Information Criterion
CBT: Cognitive Behavioural Therapy
CFI: Comparative Fit Index
CI: Confidence Interval
CFA: Confirmatory Factor Analysis
CRSQ: Children's Rejection Sensitivity Questionnaire
DAS: Dysfunctional Attitude Scale
df: Degrees of Freedom
HSRQ: Health and Social Risks Questionnaire
ICC: Intraclass Correlation Coefficient
IQ: Intelligence Quotient
LGBTQ+: Lesbian, Gay, Bisexual, Transgender, Queer (or Questioning), and Others
M: Mean
MAP: Maximum A Posterior Estimation
MCMC: Markov Chain Monte Carlo
MLE: Maximum Likelihood Estimation
N: Number of Participants (Sample Size)
NICE: National Institute for Health and Care Excellence
OBVQ: Olweus Bully/Victim Questionnaire
PDS: Pubertal Development Scale
Q: Question
RCADS: Revised Children's Anxiety and Depression Scale
RMSEA: Root Mean Squared Error of Approximation
RSPM: Raven Standard Progressive Matrices Test
SD: Standard Deviation
SDQ: Strengths and Difficulties Questionnaire
SEM: Structural Equation Model
SMFQ: Short Mood and Feelings Questionnaire
SRMR: Standardised Root Mean Square Residual
SRQ-A: Adolescent Version of the Social Reward Questionnaire
TLI: Tucker-Lewis Index
UCL: University College London
UK: United Kingdom
WHO: World Health Organization

Chapter 1 Introduction

A modified version of this chapter was published in The Lancet Psychiatry: Bone, J.K., Lewis, G., & Lewis, G. (2020) The role of gender inequalities in adolescent depression. *The Lancet Psychiatry, 7,* 471–472. https://doi.org/10.1016/S2215-0366(20)30081-X. See Appendix 2.

1.1 Adolescence

Adolescence is a period of physical, psychological and social transition between childhood and adulthood (Spear, 2000). It can be defined as the developmental period that begins with puberty and ends with the transition to adult independence (Damon, 2004). Recently, adolescence has been described as spanning 10 to 24 years of age (Sawyer, Azzopardi, Wickremarathne, & Patton, 2018). This period is longer than previous definitions, which included individuals aged 10 to 19 or 20 years (World Health Organization, 1977, 2015). The new definition was proposed to reflect continued neurocognitive maturation and the later age at which many social indicators of adulthood, such as employment and marriage, are now reached (Sawyer et al., 2018).

Adolescence begins with the onset of puberty. Puberty is the biological process that drives sexual maturation, beginning around age 10 in girls and age 12 in boys in western countries (although this varies with time and location; Parent et al., 2003; Patton & Viner, 2007). Puberty is characterised by rapid increases in oestrogen in girls and testosterone in boys. Hormonal changes lead to a rapid set of physical transformations including pubertal growth, changes in sleep and circadian regulation, and metabolic changes. Sex-specific changes occur in voice, body hair, muscle and fat distribution, facial structure, skin and glandular secretions, and genital, breast, adrenal, and gonadal development (Patton & Viner, 2007). The physical maturation that occurs during puberty can affect the way young people see themselves, as well as the way others perceive and treat them. Adolescents who have gone through puberty look more adult and are often treated accordingly, with increased expectations of responsibility, autonomy, and accountability (Blakemore, Burnett, & Dahl, 2010; Chandra-Mouli, Plesons, & Amin, 2018; Lerner & Steinberg, 2009). These pubertal changes occur alongside other developmental changes during adolescence.

Recent large longitudinal studies have demonstrated that the brain continues developing substantially throughout adolescence and into adulthood (Foulkes & Blakemore, 2018). During adolescence, white matter volume increases, and cortical grey matter volume decreases, only stabilising in the mid-twenties (Giedd et al., 1999; Mills, Goddings, Clasen, Giedd, & Blakemore, 2014; Mills et al., 2016; Tamnes et al., 2017; Vijayakumar et al., 2016). Neural regions involved in higher level cognitive processes, such as the prefrontal, parietal, and temporal cortices, continue to develop into peoples' twenties or thirties (Giedd et al., 1999; Mills et al., 2014, 2016; Tamnes et al., 2017). Cognitive abilities such as taking another person's perspective (mentalising), planning, inhibiting inappropriate behaviour, consideration of the future, and awareness of the self become increasingly advanced during adolescence (Blakemore & Choudhury, 2006). This enables adolescents to reflect more on what they think about themselves, their future, and what others think about them, which are all necessary parts of becoming an adult.

Adolescence has also been characterised as a period of "social reorientation". Young people spend increasing time with peers relative to family members, undergoing large changes in their social networks (Burnett Heyes et al., 2015). During puberty, individuals in many countries (including the UK) move from being the oldest students in primary schools to the youngest in secondary schools. During this process, adolescents may move away from established friendships and meet a new peer group. Secondary schools are much larger and have different social hierarchies, which may be more difficult to navigate (Blakemore, 2019). Continued development may also contribute to changes in social behaviour during adolescence, with changes in brain maturation and cognition, both of which allow more complex responses to social information (Nelson, Leibenluft, McClure, & Pine, 2005). Pubertal hormones may also influence emotional and behavioural responses to social information (Nelson et al., 2005). These aspects of social reorientation will be discussed in more detail later in this chapter (section 1.9).

Becoming an independent adult puts many new pressures on young people. Alongside the profound biological, neural, and social changes, adolescence is a period of vulnerability to mental health problems (Kessler et al., 2005). There are large increases in the incidence of common mental health problems (anxiety and depression) and suicidality (suicidal thoughts, ideas and attempts), as well as increases in completed suicides (Dahl, 2004; Kessler et al.,

2005). Adolescence is therefore a key period to study the emergence of depressive symptoms.

1.2 Depression in adolescence

1.2.1 Defining depression

Depression is a common mental health problem and the leading cause of disability worldwide (World Health Organization, 2017). It is estimated to affect over 300 million people (World Health Organization, 2017). Depression can occur at any time during life, although symptoms may differ between adolescents and adults (Rice et al., 2018; Thapar, Collishaw, Pine, & Thapar, 2012). According to clinical diagnostic criteria, the key symptoms of depression are low mood (or irritability in children and adolescents) and a loss of interest and pleasure in things people used to enjoy (anhedonia; American Psychiatric Association, 2013). Other symptoms include feelings of hopelessness, excessive guilt, low self-esteem, irritability, a lack of motivation, finding it difficult to make decisions and concentrate, and recurrent thoughts of suicide or self-harm. Depression can also include physical symptoms such as a slowing down of physical movement, significant weight loss or gain, and fatigue, which may be more common in adolescents than adults (Rice et al., 2018). These symptoms may lead to social withdrawal, doing poorly at school or work, and having difficulties at home (Thapar et al., 2012).

To receive a diagnosis of major depressive disorder according to the Diagnostic and Statistical Manual criteria, adolescents must experience one of the key symptoms of depression plus four or more other symptoms during the same two-week period (American Psychiatric Association, 2013). Symptoms must cause the individual clinically significant distress or impairment in social, occupational, or other important areas of functioning. The International Classification of Diseases defines depression similarly, with depressive episodes classified as mild, moderate, or severe depending on the number and severity of symptoms experienced (World Health Organization, 2018).

Despite the use of this criteria, depression is not a discrete diagnostic category. It exists dimensionally, and severe forms of depression are best viewed as the extreme end of a continuum of depressive symptoms (Hankin, Fraley, Lahey, & Waldman, 2005; Ruscio &

Ruscio, 2000). There is often no clear point at which to treat someone who is experiencing depressive symptoms and no obvious threshold at which a diagnosis should be applied. Mild or moderate depressive symptoms share the same risk factors and characteristics as clinical diagnoses of depression, the only difference being severity (Ayuso-Mateos, Nuevo, Verdes, Naidoo, & Chatterji, 2010). Depressive symptoms (without a diagnosis of depression) are associated with impaired psychosocial functioning and increased risk of future depression and other mental health problems (Copeland, Costello, Angold, & Shanahan, 2009; Fergusson, Horwood, Ridder, & Beautrais, 2005; Gotlib, Lewinsohn, & Seeley, 1995; Lewinsohn, Solomon, Seeley, & Zeiss, 2000; Pine, Cohen, Cohen, & Brook, 1999). Depressive symptoms also have a negative impact on population health, contributing to a higher public health burden (Das-Munshi et al., 2008; World Health Organization, 2017).

1.2.2 Epidemiology of depression during adolescence

Although we know that depression becomes increasingly common during adolescence, there are very few high-quality studies demonstrating the incidence of depression in adolescence. One study using diagnostic interviews found evidence that the cumulative incidence of new depressive cases increased from 1.07% at age 11, to 2.1% at age 13, 2.5% at age 15, 15.0% at age 18 and then dropped to 10.7% at age 21 (measured as the percent of adolescents first depressed at each age; Hankin et al., 1998). Another study also demonstrated that the incidence of depressive symptoms above a clinical threshold on a questionnaire increased with age (Joinson, Kounali, & Lewis, 2017).

In the 2017 Mental Health of Children and Young People Survey in the UK, the point prevalence of depression was 0.3% in 5 to 10 year olds, 2.7% in 11 to 16 year olds, and 4.8% in 17 to 19 year olds (Vizard et al., 2018). Although these estimates were ascertained in a representative sample with a clinical diagnostic tool, they may not be comparable across age groups because teachers reported symptoms for the youngest group (compared to self-report in other groups). Other estimates of the prevalence of depression vary widely across studies, which is likely due to methodological issues. Estimates of the cumulative prevalence of depression are as high as 20% by the end of adolescence (Hankin et al., 1998; Lewinsohn, Rohde, Klein, & Seeley, 1999). This is comparable to lifetime prevalence estimates of depression in adults, indicating that adult depression often begins in adolescence (Birmaher

et al., 1996). In line with this, other evidence suggests that 75% of adult mental health problems start in adolescence (before the age of 24; Kessler et al., 2005).

Depression during adolescence is associated with physical health problems, academic problems, impaired social relationships, substance abuse, and high risk sexual behaviour (Birmaher et al., 1996; Horowitz & Garber, 2006; Thapar et al., 2012). Depression increases risk of suicide, which is one of the leading causes of death in young people aged 10 to 34 in England (Public Health England, 2017). Depression during adolescence is also strongly associated with depression during adulthood. In one study, 45% of 16 year olds with depression had a recurrence by age 23 (Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2000). Depression during adolescence is therefore an important target for interventions and a potentially modifiable risk factor for subsequent depression during adulthood. However, we can only develop effective interventions for depression when we understand the underlying mechanisms. Improved awareness of the mechanisms causing adolescent depression may not only help us to alleviate symptoms in adolescence but may also reduce the burden of the disorder across the lifespan. Preventing or delaying the first episode of adolescent depression may thus be of greater public health benefit than preventing its recurrence in adulthood.

1.2.3 Potential causes of depression during adolescence

Depression is a clinically heterogeneous disorder which is likely to have a diverse range of causes. Understanding what causes depression in adolescence is therefore challenging. It is likely a result of several risk factors which may interact to increase vulnerability to depression (Thapar et al., 2012). Examining these risk factors individually is difficult because many of them are highly correlated and may also relate to later adversities.

The strongest risk factors for depression during adolescence are exposure to psychosocial adversities and a family history of depression (Thapar et al., 2012). There is extensive evidence that stressful life events (e.g. bereavement, injury) and chronic adversity (e.g. maltreatment, abuse, negative family relationships, interpersonal stress, bullying, low socioeconomic status, poor physical health, and academic pressure) are associated with increased depressive symptoms, particularly when a number of stressors are experienced over a long period (Goodyer, Wright, & Altham, 1990; Hawker & Boulton, 2000; Lewinsohn, Allen, Seeley, & Gotlib, 1999; Pine, Cohen, Johnson, & Brook, 2002; Thapar et al., 2012).

Children of parents with depression have three to four times higher rates of depression compared to children with healthy parents (Rice, Harold, & Thapar, 2002b). This increased risk is due to both genes and non-inherited factors, with the majority of intergenerational transmission of depression due to the shared environment of children and their parents (Rice et al., 2002b). Depression is thought to become increasingly heritable from childhood (with heritability estimates as low as zero) to late adolescence, by which time heritability is modest (around 30-50%; Thapar & Rice, 2006).

Psychosocial adversities and genetic risk may also increase risk for depression by leading to changes in cognition, temperament, personality attributes (e.g. emotionality, behavioural inhibition, and neuroticism), underlying neural circuits, and endocrine systems (Thapar et al., 2012). These changes in cognition are the focus of cognitive models of depression, which I will discuss further in section 1.3 and throughout my thesis. Psychosocial adversities and genetic risk, together with developmental, hormonal and neural mechanisms, may also alter adolescents' susceptibility to stressors. Diathesis-stress models of depression propose that adolescents may have diatheses (pre-existing characteristics) which interact with stressors, such as negative life events, and increase the individual's risk of depression after stressful events (e.g. Cole et al., 2008).

1.2.4 Gender differences in the epidemiology of depression

Women are twice as likely to experience depression as men (Kessler, 2003; McManus, Bebbington, Jenkins, & Brugha, 2016; Merikangas et al., 2010). According to one review, estimates of the ratio in odds of depression between males and females range from 1.3 to 3.1, with an average of 2.1 for lifetime prevalence (Kuehner, 2003). Findings from subsequent studies have provided further evidence to support this finding (Kuehner, 2017). This gender difference in the prevalence of depression is one of the most robust findings in psychiatric epidemiology (Salk, Hyde, & Abramson, 2017). It is found whether depression is assessed using self-report symptom measures (e.g. Allgood-Merten, Lewinsohn, & Hops, 1990) or clinical interviews and diagnoses (e.g. Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993). Although there is some evidence that the gender difference in depression varies in size geographically, it has been found in high, middle and low-income countries (Bromet et al., 2011), and across many sociocultural settings (Kuehner, 2017).

It has been suggested that the gender difference in depression is a result of measurement artefacts when assessing depressive symptoms (Mirowsky & Ross, 1995; Piccinelli & Wilkinson, 2000; van Beek, Hessen, Hutteman, Verhulp, & van Leuven, 2012). Traditional questionnaires may be biased towards recognising depressive symptoms in females, meaning that the prevalence of depressive symptoms is overestimated in females. However, even after accounting for differences in the measurement of depression, there is still evidence for a gender difference (e.g. Bulhões, Ramos, Severo, Dias, & Barros, 2019; Byrne, Baron, & Campbell, 1993). There may be heterogeneous subtypes of depression, for example depression with and without comorbid conduct problems (Riglin et al., 2016), which differ in their aetiology and do not all demonstrate gender differences. However, "pure" depressive symptoms (those traditionally measured in clinical interviews and questionnaires such as low mood and anhedonia) demonstrate robust gender differences (e.g. Bulhões et al., 2019; Byrne et al., 1993; Riglin et al., 2016).

This gender difference in depression emerges during adolescence. Before age 11, the incidence of depression is similar in boys and girls, and may even be slightly higher in boys (Douglas & Scott, 2014). Around 12-13 years of age, the incidence of depression increases sharply in girls but remains similar in boys (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015; Hankin et al., 1998, 2015; Joinson et al., 2017; Kwong et al., 2019; Patton et al., 2008). Later in adolescence, the incidence of depression does start to increase for males but rates remains lower in males than females (Hankin et al., 1998). The longitudinal trajectories of depressive symptoms during adolescence were recently tested in a large UK cohort (the Avon Longitudinal Study of Parents and Children; ALSPAC), as illustrated in Figure 1.1 (Kwong et al., 2019). This demonstrates that, on average, depressive symptoms are more severe and increase more with age in girls than boys during adolescence.

One review of the gender difference in depression meta-analysed studies with nationally representative samples and validated diagnostic or measurement criteria (Salk et al., 2017). Across 65 diagnosis studies and 95 symptom studies, the gender difference peaked in adolescence and then declined and remained stable in adulthood. Age was the strongest predictor of the effect size, with the largest gender differences occurring at 13 to 15 years for depression diagnoses (odds ratio=3.02). The odds ratio decreased with age after this peak, from 16 to 19 years (odds ratio=2.69) to ages 20 to 29 years (odds ratio=1.93). There was no

evidence of decreases in the gender difference after age 29, with the odds ratio for depression diagnoses remaining between 1.71 and 2.02 in working age adults. A similar pattern was found for depressive symptoms (Salk et al., 2017).

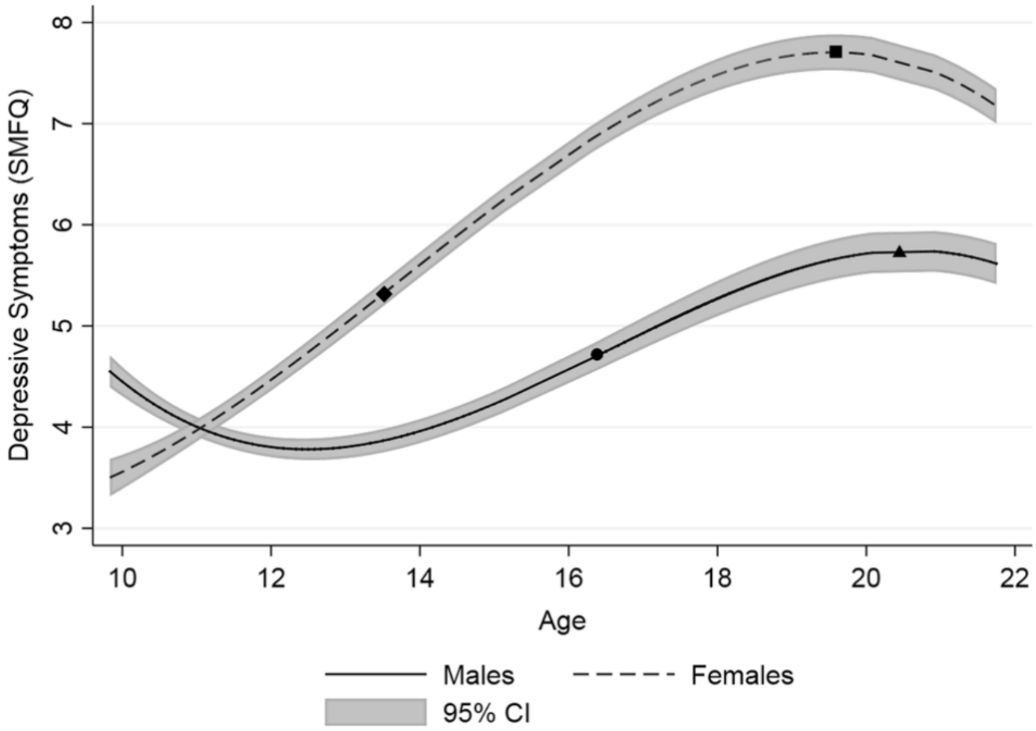

Figure 1.1 Averaged population trajectories of depressive symptoms across adolescence for males and females in the ALSPAC cohort. Figure from Kwong et al. (2019). Depressive symptoms were measured with the Short Mood and Feelings Questionnaire (SMFQ). Features of the trajectories are overlaid with the following terms: ● Male age of peak velocity of depressive symptoms. ▲ Male age of maximum depressive symptoms. ♦ Female age of peak velocity of depressive symptoms. ■ Female age of maximum depressive symptoms.

Young women (aged 16-24 years) were recognised as a group at high risk of common mental disorders, including depression, in the most recent UK Psychiatric Morbidity Survey (McManus et al., 2016). This report also found evidence that the prevalence of depression is increasing more in young women than young men in the UK (McManus et al., 2016).

Despite the wealth of evidence demonstrating a gender difference in the prevalence of depression, it is very poorly understood. We do not know what causes this gender difference. There is also very little data on changes in the incidence of depression during adolescence, despite its relevance for investigating the aetiology of depression. Understanding exactly when the incidence of depression increases in girls could provide insights into the aetiology of depression.

In order to prevent the increase in depression during adolescence, we must identify modifiable risk factors. Theories about causes of depression often do not consider the gender difference in depression, ignoring an important characteristic of this condition. If a factor cannot be related to the gender difference, it is unlikely to provide a complete explanation of the aetiology of depression. Since the gender difference in the incidence and prevalence of depression emerges in adolescence, and individuals often have multiple episodes of depression (Lewinsohn, Rohde, et al., 2000), factors contributing to the adolescent gender difference are likely to also affect the gender difference in the prevalence of depression later in life. I will therefore focus on explanations that apply to the emergence of the depression in adolescence.

1.2.5 Explanations for the gender difference in depression

Although single explanations for the gender difference have been proposed, it is likely to be a complex multifactorial process (see Kuehner, 2017 for a recent review). There are numerous factors, many of which are likely to contribute to the increase in depressive symptoms in girls during adolescence. My aim is not to review all of these explanations, which has been done many times (e.g. Hyde, Mezulis, & Abramson, 2008; Kuehner, 2003, 2017; Piccinelli & Wilkinson, 2000), but to link previous explanations in a new theory, which will be explored throughout my thesis.

Explanations of the gender difference in depression can be divided into two broad categories, internal and external factors. Internal factors refer to biological or psychological characteristics, such as puberty, sex hormones, genetics, or differences in cognitive vulnerability (Angold, Costello, & Worthman, 1998; Ge, Conger, & Elder, 2001; Keenan, Culbert, Grimm, Hipwell, & Stepp, 2014; Kuehner, 2017; Martel, 2013; Rice, Harold, & Thapar, 2002; Scourfield et al., 2003). External factors, in contrast, are environmental or societal, such as psychosocial stressors and child sexual abuse, which are often outside of an individuals' control (Dunn, Gilman, Willett, Slopen, & Molnar, 2012; Fergusson, Swain-Campbell, & Horwood, 2002; Kuehner, 2017; Lewis, McElroy, Harlaar, & Runyan, 2016). Internal factors are thought to lead to individual differences in susceptibility to external risk factors (Kuehner, 2017).

In the most recent review, internal risk factors were classified as biological or psychological (Kuehner, 2017). Key biological factors identified in this review were genetic risk, gene-environment interactions, hormones, and physiological stress responses. Psychological factors included neuroticism, rumination, and body shame and dissatisfaction. In contrast, external risk factors were either micro level or macro level environmental factors. Early adversity (such as childhood sexual abuse), interpersonal violence after childhood, and exposure to stress were micro level, whereas the macro level factors identified were societal gender inequalities (Kuehner, 2017).

However, the distinction between internal and external factors is an artificial one. The external environment in which people develop also influences their own vulnerability, thus becoming internalised. Additionally, factors labelled as external might be influenced by internal genetic factors, as our genes might influence the environment we select (Jaffee & Price, 2007; Knafo & Jaffee, 2013; Krapohl et al., 2017). Internal factors and external factors may also multiplicatively increase an individual's risk of depression. Gender differences in depression are thus likely to be a result of increased exposure to internal and external risk factors in girls compared with boys.

Gender inequalities are one external factor which could lead to increased vulnerabilities to depression in girls (Kuehner, 2017). They start early in life, are pervasive, and exist in most societies (Baunach, 2001; Heise et al., 2019; Human Development Report Office, 2020). Girls may experience more stressful life events during development than boys. For example, globally, girls are more likely than boys to experience sexual violence (Schraedley, Gotlib, & Hayward, 1999; UNICEF, 2014; Weiss, Longhurst, & Mazure, 1999). Boys and girls are often treated differently from birth, and frequently have different behaviour expected from them. Gender socialisation is the process by which people learn to behave in a certain way as male or female, dictated by societal norms and individual attitudes (Chandra-Mouli et al., 2018; Heise et al., 2019). Boys are often expected to fulfil masculine stereotypes of confidence and bravery, whereas many traditional and stereotypical characteristics of girls (passivity, emotionality, helplessness) are similar to common symptoms of depression (Chandra-Mouli et al., 2017, 2018). Many societies have norms that assign higher value to being a man than being a woman (Chandra-Mouli et al., 2017). Socialisation during childhood and early adolescence may therefore contribute to increased vulnerability to depression in girls and

lead to gender differences in depression (Heise et al., 2019; Ruble, Greulich, Pomerantz, & Gochberg, 1993).

Individuals may internalise societal gender norms when developing their identities, beliefs, and behaviours (Chandra-Mouli et al., 2018). On average, males have higher self-esteem than females (Bleidorn et al., 2016), and this gender difference is present in adolescence (Gentile et al., 2009; Robins & Trzesniewski, 2005). By early adolescence, boys take more risks than girls and there is evidence that gender differences in risk-taking during adolescence are smaller in countries with higher gender equality (Cárdenas, Dreber, von Essen, & Ranehill, 2012). Across Europe, higher national gender equality is related to a lower prevalence of depression and a smaller gender difference in depression for some groups (van de Velde, Huijts, Bracke, & Bambra, 2013). There is also evidence that the gender difference in depression decreased in US cohorts of individuals born from 1957 to 1994 (Platt, Bates, Jager, McLaughlin, & Keyes, 2020). This decrease in the gender difference was associated with the increasing ratio of college degree attainment in females to males (Platt, Bates, Jager, McLaughlin, & Keyes, 2020), further suggesting that increased gender equality may be associated with lower depressive symptoms in females. Specific mechanisms through which a lack of societal gender equity may be internalised and cause an increase in depression in females have not yet been explored in depth. In my thesis, I will propose one potential mechanism which could link gender equality to depressive symptoms. In order to explain my hypothesis, I will first describe how depressive symptoms develop according to cognitive models of depression.

1.3 Cognitive models of depression

In general, cognitive models of depression propose that individuals may have biases in their beliefs, attitudes, and the way that they process information, and these biases could increase their vulnerability to depression (Abramson, Metalsky, & Alloy, 1989; Beck, 1967; Cole, Martin, & Powers, 1997; Roiser, Elliott, & Sahakian, 2012). According to Beck's cognitive model of depression, individuals are able to make appraisals about the meaning and value of events and generate appropriate responses through the use of three schemas (Beck, 1967). These schemas consist of beliefs about the self, the world, and the future, and form the "cognitive triad" (Beck, 1967). Individuals may develop more negative (or "depressogenic")

schema as a result of adverse experiences during childhood (Beck, 1967, 1979, 2008; Beck & Bredemeier, 2016). This is a diathesis-stress model, as these depressogenic schema increase susceptibility to depression. Beck hypothesised that negative schema interact with negative life events, and then lead to increases in depressive symptoms. Once activated, these schema also influence how individuals attend to, perceive, interpret, learn from, and remember their environment (Beck, 1967, 1979, 2008; Beck & Bredemeier, 2016). These processes are described by the umbrella term "information processing". Biases in information processing also contribute to increases in depressive symptoms (Beck & Bredemeier, 2016). These biases are thought to result from early adverse experiences, stress, and genetic factors, as well as being further exacerbated by the activation of negative schema (Beck & Bredemeier, 2016).

Another cognitive model of depression has instead proposed that biases in information processing develop first, and then lead to increases in negative schema (Roiser et al., 2012). In this way, this model proposes that the lower-level processes (information processing) act as a building block for the higher-level constructs (negative schema). However, this model also suggests that, once developed, negative schema increase biases in information processing, as proposed by Beck (Beck & Bredemeier, 2016; Roiser et al., 2012). Whatever the direction of causality between these types of cognition, cognitive models propose that depression is driven by negative biases in both lower-level information processing and higher-level schema (Beck & Bredemeier, 2016; Roiser et al., 2012). These cognitive biases are likely to interact with negative life events to lead to increases in depressive symptoms.

Information processing occurs within both "hot cognition", in which information has an emotional impact on individuals, and "cold cognition", in which information has no emotional influence (so stimuli are emotionally neutral or outcomes are not motivationally relevant; Roiser & Sahakian, 2017). Both types of information processing are thought to be disrupted in depression (see Roiser & Sahakian, 2017 for a review). In my thesis, when using the term information processing, I am referring to hot cognition or "emotion processing", meaning the processing of information which has an emotional impact on individuals. This may be because the information itself is intrinsically emotionally salient (e.g. emotional faces, words, or scenes) or because processing of the information results in feedback, which then influences an individual's affective state (e.g. leading to disappointment or satisfaction; Roiser & Sahakian, 2017). This includes the processing of rewarding and punishing stimuli.

Although cognitive models were developed based on research with adults, they have generally been applied to adolescents with little consideration of developmental factors. Cognitive models propose that negative cognitions are a result of adverse experiences during childhood, which may be problematic for the extension of these models into childhood and early adolescence. If negative cognitions are still being constructed in childhood and early adolescence, then individuals may not yet have formed diatheses which interact with stressful life events to cause depression (Cole et al., 2008; Turner & Cole, 1994). It is thus currently unclear whether these cognitive models of depression are relevant to all adolescents. I will explore this issue further throughout my thesis.

Next I will review the evidence for negative schema and biased information processing, and whether these are associated with depressive symptoms, in adolescence. I will then outline my novel hypothesis, linking these cognitive models to an explanation for the emergence of the gender difference in depression.

1.4 Dysfunctional attitudes in adolescence

As discussed, higher-level negative schema may be a cause or consequence of information processing biases (Beck & Bredemeier, 2016; Roiser et al., 2012). Either way, negative beliefs about the self are central to cognitive theories of depression. Beck's cognitive model of depression describes negative schema about the self called dysfunctional attitudes. These include beliefs about how you should act and feel, what you should achieve, what others should think about you, and what others' opinions mean for your happiness (Beck, 1983; Weissman & Beck, 1978). For example, a specific dysfunctional belief is "if I fail at my work, then I am a failure as a person" (Weissman & Beck, 1978). Dysfunctional attitudes are thought to derive from early experiences throughout childhood (Beck, 1983; Weissman & Beck, 1978) or from the repeated impact of negative information processing biases (Roiser et al., 2012). They may guide interpretations of new events, shape expectations for the future, and influence memory of prior experiences (Beck & Bredemeier, 2016).

Alloy and Abramson's hopelessness theory of depression (Abramson et al., 1989) proposes a similar construct, which they call attributional style. This involves attributing negative events to causes that impact self-worth, believing that events are one's own fault, will impact all

aspects of one's world, and will also impact the future. In research studies, these attributions have been measured using the Cognitive Styles Questionnaire (CSQ; Haeffel et al., 2008; Meins et al., 2012).

In my thesis, I will focus on dysfunctional attitudes, as they are often divided into two domains, negative beliefs about achievements and beliefs about social approval (e.g. De Graaf, Roelofs, & Huibers, 2009; Rogers et al., 2009). These two domains have been labelled perfectionism (or performance evaluation) and need for approval (need for social approval, approval by others, or dependency; Barnett & Gotlib, 1990; Beck, 1983; Cane, Olinger, Gotlib, & Kuiper, 1986; De Graaf et al., 2009; Imber et al., 1990; Rogers et al., 2009; Zlotnick, Shea, Pilkonis, Elkin, & Ryan, 1996). Perfectionism relates to having high personal standards and interpreting mistakes as failure. Need for approval involves beliefs that one's own happiness and self-worth are dependent on gaining approval and support from others (De Graaf et al., 2009; Rogers et al., 2009). As I will discuss in more detail in section 1.9 and chapter 6, I was interested in negative beliefs in the social domain. In contrast to dysfunctional attitudes, attributional style may (to some extent) be determined by the nature of the negative event that attributions are made about. The CSQ does not include scenarios which are clearly social versus non-social so cannot provide a measure of negative attributions for social events in its current form.

According to cognitive models of depression, dysfunctional attitudes may make individuals more vulnerable to depression (Beck & Bredemeier, 2016; Weissman & Beck, 1978). There is strong evidence from a range of studies that dysfunctional attitudes are associated with depressive symptoms in adolescence. For example, a number of large studies (case-control and cross-sectional measuring depressive symptoms continuously) have found evidence for concurrent associations between dysfunctional attitudes and depressive symptoms in adolescence (Gotlib, Lewinsohn, Seeley, Rohde, & Redner, 1993; Lewinsohn, Gotlib, & Seeley, 1997; Lewinsohn et al., 1994; Rogers et al., 2009; Smith, Reynolds, Orchard, Whalley, & Chan, 2018; Young, LaMontagne, Dietrich, & Wells, 2012). These designs are not able to provide evidence on the temporality of associations between dysfunctional attitudes and depressive symptoms. It is thus possible that these findings are a result of reverse causality, with increased depressive symptoms leading to more dysfunctional attitudes. However, there is stronger evidence from longitudinal studies which adjust for baseline depression. Numerous

large longitudinal studies have found evidence for associations between dysfunctional attitudes and subsequent depressive symptoms in adolescence, even after adjusting for baseline symptoms (Abela & Sullivan, 2003; Hankin, Young, Gallop, & Garber, 2018; Lewinsohn, Joiner, & Rohde, 2001; Pearson et al., 2015; Pössel, 2017; Rawal, Collishaw, Thapar, & Rice, 2013b). Additionally, altering dysfunctional attitudes is one of the main aims of cognitive behavioural therapy (CBT; Beck, 1979, 1983; Clark & Beck, 1999), which is an effective therapy for some adolescents with depression (e.g. Oud et al., 2019). This suggests that dysfunctional attitudes could causally affect depressive symptoms.

1.5 Information processing in adolescence

1.5.1 Questionnaire-based versus task-based measures of cognition

Traditionally, studies of cognition used self-report questionnaires in which participants recorded hypothetical responses to events (Elliott, Zahn, Deakin, & Anderson, 2011). This restricted investigation to conscious thoughts and behaviours, such as dysfunctional attitudes, which may be susceptible to mood-congruent response biases (Colman et al., 2016). Additionally, questionnaires such as those measuring dysfunctional attitudes may overlap in content with questionnaires measuring depressive symptoms. The strong associations generally found between dysfunctional attitudes and depressive symptoms could be a result of shared, or very similar, items across questionnaires. This may artificially inflate evidence for associations between these scales rather than identifying a meaningful risk factor for depression.

There is now more focus on tasks that assess automatic cognitive processes, which may influence thoughts and behaviours without conscious awareness (Kahneman, 2011; Roiser et al., 2012). These implicit processes may demonstrate different biases to those found in explicit ratings of thoughts and behaviour (Kahneman, 2011; Roiser et al., 2012). Cognitive tasks can provide a behavioural measure of information processing, tapping into the cognitive mechanisms underlying how people think about their environment and social interactions. In comparison to explicit measures of cognition, these tasks may overlap less in content with explicit reports of symptoms, which could reduce the risk of artificially inflating associations with depressive symptoms.

1.5.2 Evidence for biases in information processing

Using cognitive tasks measuring implicit processing, there is evidence that healthy individuals generally have positively biased information processing, favouring positive information (Beck & Bredemeier, 2016; LeMoult & Gotlib, 2019; Roiser & Sahakian, 2017). This includes biases in attending to positive information, interpreting ambiguous information positively, and remembering more positive than negative information (Roiser & Sahakian, 2017). Additionally, healthy individuals may have an optimism bias, overestimating the likelihood of positive events happening in the future, and underestimating the likelihood of negative events (Sharot, 2011). This could increase resilience, decreasing the likelihood of healthy individuals experiencing depression. In contrast, there is evidence that adults with depression have reduced positive, or increased negative, information processing (Beck & Bredemeier, 2016; LeMoult & Gotlib, 2019; Roiser & Sahakian, 2017). Individuals with more severe depressive symptoms thus generally show a more negatively biased pattern of responding than individuals with less severe symptoms. These negative biases may lead to increased depressive symptoms by increasing negative affect, encouraging social withdrawal, and reducing motivation, among other mechanisms (Beck & Bredemeier, 2016; LeMoult & Gotlib, 2019; Roiser & Sahakian, 2017).

There is evidence that negative biases in attention, interpretation, learning and memory are associated with depression in adulthood (e.g. Everaert, Duyck, & Koster, 2014; Everaert, Podina, & Koster, 2017; Moore & Fresco, 2012; Roiser & Sahakian, 2017). However, the evidence in adolescents is less consistent. A recent review examined the evidence for information processing biases in adolescent depression (Platt, Waters, Schulte-Koerne, Engelmann, & Salemink, 2017). Although this review was not systematic, it took a structured approach to provide a broad overview of the literature. Quantitative synthesis of previous research would be very difficult because of the heterogeneous methods used to date. A large variety of experimental paradigms have been developed to measure automatic biases in attention, interpretation, and memory.

In this review (Platt et al., 2017), studies most often measured biases in memory by asking individuals to rate whether positive and negative personality characteristics described themselves, followed by a surprise recall or recognition test in which participants are asked

to remember as many characteristics as possible. Overall, there was no consistent evidence that adolescents with depression had reduced positive or increased negative biases in memory in comparison to healthy controls (Platt et al., 2017). In contrast, attentional biases have been measured with a variety of cognitive tasks, including the emotional Stroop, affective dot-probe, and emotional go/no-go. There was some evidence that adolescents with more depressive symptoms had biases in attention towards negative information. Finally, interpretation biases have been measured by presenting young people with ambiguous scenarios and words and asking them to choose a positive or negative solution, and these tasks have showed the strongest evidence for biases in adolescent depression. There was evidence for an association between negative interpretations and depressive symptom severity in youth with depression, community samples, and those at high risk of depression (Platt et al., 2017). These interpretation biases are also called biases in appraisal processes, and they may extend to the interpretation of ambiguous facial expressions as more negative or threatening (Lau & Waters, 2017).

Biases in other types of information processing may also contribute to adolescent depression. Although there is inconsistent evidence for biases in recall or recognition memory, there is stronger evidence that another memory phenomenon, over-general autobiographical memory, is associated with depression in adolescence (Lau & Waters, 2017). Over-general autobiographical memory has been measured by asking adolescents to give a specific memory to a positive, negative, or neutral cue word. There is strong evidence that, compared to healthy adolescents, adolescents with depression have a tendency to describe general categories of similar events when given a cue word, rather than specific memories of their own experiences (Lau & Waters, 2017; Rawal & Rice, 2012). Evidence for this difficulty in remembering specific events has also been found in adolescents at high risk for depression, indicating that it may increase adolescents' vulnerability to depression (Kuyken & Dalgleish, 2011; Rawal & Rice, 2012; Warne, Collishaw, & Rice, 2019).

There have been an increasing number of studies testing associations between information processing and depressive symptoms during adolescence, but the evidence is not as well established as in adults. Even in adults, there is much conflicting evidence. It is not yet clear whether information processing is a risk factor for, or result of, depression because most

studies are cross-sectional. Despite the lack of evidence, it is likely that negatively biased information processing is associated with more severe depressive symptoms in adolescence.

1.5.3 Effect of age on information processing

One limitation to the research with adolescents so far is the unclear effect of age on information processing. As discussed, adolescence is a developmental period involving a large number of biological, cognitive, and social changes. The continued development of these processes throughout early and mid-adolescence (Pfeifer & Blakemore, 2012) may be relevant to the development of information processing biases. Adolescents may only develop stable cognitive styles in early or mid-adolescence, once abstract thinking and operational reasoning abilities are more developed (Lakdawalla, Hankin, & Mermelstein, 2007; Platt et al., 2017). Information processing biases may thus emerge with development, increasing in prevalence with age. Alternatively, biases may be present from early adolescence, as a result of early experiences. If information processing biases are a risk factor for depression, we might expect them to be present from early adolescence. However, there is very little research testing biases developmentally, and it remains unclear whether they change with age (previous research will be reviewed in chapters 3-5). In my thesis, I will test whether the prevalence of information processing biases changes with age during adolescence.

Associations between information processing biases and depressive symptoms could also differ between adolescence and adulthood. This would be an example of effect modification, with age moderating the association between information processing biases and depressive symptoms. As individuals develop the ability for more abstract and operational thinking, the association between information processing biases and depressive symptoms may increase with age (Dearing & Gotlib, 2009; Platt et al., 2017). As outlined in section 1.3, most research has applied cognitive models of depression to adolescents without considering these developmental factors. There is some evidence that negatively biased cognitions are only associated with the emergence of depressive symptoms from mid-adolescence (Cole et al., 2008; Turner & Cole, 1994). In childhood and early adolescence, stressful life events may be directly associated with increased depressive symptoms, without any effect of negative cognitions (Cole et al., 2008; Turner & Cole, 1994). However, other studies have found no evidence for differences in the association between information processing and depressive

symptoms across age (Abela & Hankin, 2008; Platt et al., 2017). It remains possible that information processing biases are associated with depressive symptoms from early adolescence. In my thesis, I will test whether associations between information processing biases and depressive symptoms differ with age and, if so, when they emerge during development.

1.6 A novel hypothesis concerning the gender difference in depression

Despite the extensive evidence for cognitive models of depression, research testing cognitive models has not addressed the gender difference in depression. I propose that gender should be classified as an exposure variable within the causal pathway proposed by cognitive models of depression (Bone, Lewis, & Lewis, 2020). As outlined in section 1.2.5, girls may experience more negative impacts of societal gender inequality, gender stereotypes, and socialisation during childhood than boys. Being female might therefore cause a more negative environment during childhood, meaning girls experience more stressors. This environment could lead to more negative cognitions, which increase the risk of depression. By adolescence, girls might have learnt to have more negative attitudes towards themselves because of how they are represented and treated in society. Societal inequalities during development could therefore directly influence negative schema, lead to negative biases in information processing, and contribute to the increased incidence of depression in girls during adolescence.

I propose that this mechanism will contribute to depressive symptoms throughout adolescence. I expect that girls might have more negative schema and information processing biases, and this acts as a risk factor for depressive symptoms. I would expect gender differences in negative schema and information processing to be present from early adolescence, as a result of girls' exposure to a more negative environment during childhood. This would mean that age is not an effect modifier of the gender difference in negative schema and information processing (i.e. the gender difference in negative cognitions does not change with age).

It is important to note that I am not proposing that negative schema and information processing biases are more strongly associated with depressive symptoms in girls than boys.

This would be effect modification and has been tested in previous research on the gender difference in depression (see section 1.7). Instead, I am proposing a mediation hypothesis, in which negative schema and information processing biases mediate the association between gender and depressive symptoms. Being female may lead to a higher prevalence of negative schema and information processing biases, which are then associated with more severe depressive symptoms.

Next, I will briefly explore the current evidence for gender differences in information processing and negative schema in adolescence. I will then discuss the importance of the social environment during adolescence, focussing on how negative cognitions surrounding social interactions may be particularly important for the emergence of the gender difference in depressive symptoms.

1.7 Evidence for gender differences in information processing

Gender may be associated with differences in information processing biases and these may be detectable at a cognitive or behavioural level. I have proposed that negative information processing biases may be more prevalent in girls from early adolescence. However, very few studies have examined gender differences in information processing and most of them have been with adults.

Previous research on gender differences in information processing has focussed on the idea that women are more adept at understanding and processing emotions than men. For example, one review of gender differences in facial emotion recognition across childhood and adolescence suggested that females have an advantage over males in understanding other people's emotional facial expressions (McClure, 2000). More recently, others have tested whether gender differences in negative information processing biases in adults are associated with depressive symptoms (e.g. Campanella et al., 2012; Peckham, McHugh, & Otto, 2010). In adults, some small studies have found evidence that women show larger neural responses to positive and negative facial expressions than men, and these gender differences are attenuated after adjusting for depressive symptoms (e.g. Campanella et al., 2012). However, a meta-analysis found no evidence that gender moderated the association between biased attention to negative information and depression (Peckham et al., 2010). Research with

adolescents lags behind studies of adults and, to my knowledge, there is no evidence on whether there are gender differences in information processing biases in adolescence.

Additionally, much of the research on information processing biases in depression has treated gender as a moderator (e.g. Hyde et al., 2008). This approach aims to test whether gender affects the direction or strength of the association between information processing and depressive symptoms. This is usually tested as an interaction between the exposure and moderator on the outcome. In this case, information processing is the exposure, gender is the moderator, and depressive symptoms the outcome. However, interaction tests usually have low power because they involve testing whether the association between the exposure and outcome differs for each strata of the moderator (Greenland, 1983). In adolescence, girls usually have more variation in and more severe depressive symptoms (e.g. Kwong et al., 2019), meaning there is more power to test associations with information processing than for boys. Additionally, tests of interactions are model-dependent, so findings may vary depending on the way in which interactions are analysed (Greenland, 1983; Kendler & Gardner, 2010). Furthermore, analysing gender as a moderator is often not theoretically grounded. Researchers do not consider what effect modification means in terms of the association between information processing and depressive symptoms. It could be that information processing is only associated with depressive symptoms in girls, and not boys, or that it is more strongly associated in girls than boys. These possibilities do not arise from cognitive theory and it is not clear why they would occur.

An alternative approach is to treat information processing as a mediator, and test whether it can explain (part of) the association between gender and depressive symptoms. Mediation has a clear theoretical basis and can be used to make causal inferences (MacKinnon, 2008; Shrout, 2011). It allows the identification of risk factors for depression, which occur for both boys and girls, but may be more prevalent in girls. In my thesis, I aim to examine whether information processing could be a mediator of the association between gender and depressive symptoms during adolescence.

1.8 Evidence for gender differences in dysfunctional attitudes

According to my hypothesis, girls may have more dysfunctional attitudes than boys from early adolescence, making them more vulnerable to depression. A number of studies have investigated whether there are gender differences in dysfunctional attitudes during adolescence, which I will discuss in more detail in chapter 6. Briefly, the majority of previous studies have not found evidence for gender differences in overall dysfunctional attitudes during adolescence (Abela & Sullivan, 2003; Chen & Li, 2014; Gotlib et al., 1993; Hankin et al., 2018; Lewinsohn et al., 2001; Meiser & Esser, 2017, 2019; Rawal et al., 2013b; Young et al., 2012). However, as outlined in section 1.4, dysfunctional attitudes are often separated into perfectionism and need for approval (e.g. De Graaf, Roelofs, & Huibers, 2009; Rogers et al., 2009). Consistent with traditional gender stereotypes, it has been proposed that males are more perfectionistic whereas females may have more need for approval (Barnett & Gotlib, 1990; Beck, 1983; De Graaf et al., 2009; Farmer et al., 2001; Meiser & Esser, 2019; Otani et al., 2013; Zlotnick et al., 1996). If this is the case, girls may develop more need for approval as result of gender inequality and socialisation during childhood, and this may make them more vulnerable to depression.

As with tests of information processing, I propose that we should test dysfunctional attitudes as a mediator of the association between gender and depressive symptoms, rather than treating gender as a moderator of the association between dysfunctional attitude and depressive symptoms. One longitudinal study has tested whether negative cognitions mediate the association between gender and depressive symptoms, or whether negative cognitions are a result of increased depressive symptoms (Mezulis, Funasaki, Charbonneau, & Hyde, 2010). In adolescents aged 11-15 years, gender differences in depressive symptoms emerged before gender differences in negative cognitions, indicating that cognition could not mediate the association between gender and depressive symptoms. However, this study was relatively small (n=366) and had substantial attrition, which might have caused bias, and the final sample is unlikely to be representative of adolescents in the general population. It also used the CSQ, measuring attributions of negative events, as opposed to dysfunctional attitudes (Mezulis et al., 2010). Although this study did not find evidence that negative cognitions mediated the gender difference in depression, it is possible that dysfunctional attitudes, particularly need for approval, could mediate the association between gender and

depressive symptoms. As outlined below (section 1.9), the social environment may be especially salient during adolescence. In chapter 6, I will examine the role of perfectionism and need for approval in the gender difference in depression during adolescence.

1.9 Importance of social cognition

In this section, I will explore why negative schema and information processing specifically about the social environment (social information processing) may be particularly important for the emergence of the gender difference in depression during adolescence.

1.9.1 New peer relationships

During adolescence, peers become increasingly important. Peers are people with similar life experiences or demographic traits (e.g. similar ages, interests, backgrounds, or status). Relationships change from childhood friendships to adult-like peer relationships during adolescence (Burnett Heyes et al., 2015). The amount of time spent with peers also increases, and this time may be particularly rewarding (Davey, Yücel, & Allen, 2008; Lam, McHale, & Crouter, 2014; Larson & Richards, 1991). Peers also start to surpass parents as the primary source of social support in adolescence (Furman & Buhrmester, 1992). They become more important in shaping social behaviour (Steinberg & Silverberg, 1986) and gaining their social approval becomes particularly salient (Steinberg, 2008). To become independent adults, young people must build strong affiliations with their peer group and gradually refine their social networks (Burnett Heyes et al., 2015). Thus, during adolescence, peers become more important, more influential, and more highly valued.

Managing new social relationships is a significant challenge in adolescence. As adolescents put lots of energy into forming peer networks (Steinberg & Morris, 2001), and relationships become more important, the potential negative consequences of rejection or exclusion by peers also increase. There is evidence that, in comparison to children and adults, adolescents are particularly sensitive to social rejection (see Platt, Kadosh, & Lau, 2013 for a review). Peer rejection is associated with more negative affect and increased distress and anxiety in adolescents compared to children and adults (e.g. Sebastian, Viding, Williams, & Blakemore, 2010). For example, in one study, adolescents reported more negative mood after being excluded by other players in a ball-tossing game than adults (Somerville, 2013). Adolescents

also report being more concerned about making decisions that could lead to being excluded by their peers compared to adults (Andrews, Foulkes, Bone, & Blakemore, 2020). This may be problematic as peer rejection is frequently experienced (Peake, Dishion, Stormshak, Moore, & Pfeifer, 2013; Sebastian, Roiser, et al., 2010). Adolescent relationships are very unstable, with less than half of close friendships lasting more than a year during this period (Connolly, Furman, & Konarski, 2000; Değirmencioğlu, Urberg, Tolson, & Richard, 1998).

1.9.2 A changing self-concept

Young people must also begin to develop a sense of identity during adolescence. It is a period in which the sense of self changes dramatically, as adolescents learn who they are and what they believe about themselves. Two main sources of information are used to form this self-concept (Sebastian, Burnett, & Blakemore, 2008). Firstly, appraisals of what we are like can be made by reflecting on our own reactions to and feelings about past experiences. This is how you perceive yourself (e.g. "I am shy"). Secondly, information can be gained from your beliefs about how others perceive you (e.g. "people think I am outgoing"). This has been referred to as a social self-concept or the looking glass self, as it uses the reflected appraisals of others (Gallagher, 2000; Sebastian et al., 2008).

There is evidence that self-evaluations become more comprehensive and complex throughout childhood and adolescence. By early adolescence, individuals are more likely to compare themselves to others and be aware that others are also making these judgments (Sebastian et al., 2008). Adolescents become increasingly self-conscious and concerned with other people's opinions of them (Parker, Rubin, Erath, Wojslawowicz, & Buskirk, 2006; Sebastian et al., 2008; Vartanian, 2000). In particular, the beliefs of peers are increasingly influential for adolescents' evaluation of their own social and personal worth (O'Brien & Bierman, 1988). As adolescents begin to place more value on other people's opinions, the social self-concept could gain a larger role in individuals' self-evaluations.

Self-evaluations may also become more negative during adolescence. There is evidence that older adolescents have more negative self-evaluations than early adolescents (van der Aar, Peters, & Crone, 2018). A review which included longitudinal studies found evidence that self-esteem declines during adolescence, particularly in girls (Robins & Trzesniewski, 2005). If the self-concept is increasingly based on social comparisons, and peer relationships become more

important, social interactions may influence depressive symptoms through the self-concept. There is cross-sectional evidence that having a negative self-concept is associated with more severe depressive symptoms in adolescence (Hards, Ellis, Fisk, & Reynolds, 2019; Ybrandt, 2008). Additionally, another cross-sectional study found evidence that higher self-reported peer stress was associated with a more negative self-concept and increased depressive symptoms in adolescence (Wenz-Gross, Siperstein, Untch, & Widaman, 1997). However, these changes in the self-concept could be a result of increasing depressive symptoms, rather than being driven by social development during adolescence.

1.9.3 Competency-based models of depression

Competency-based models of depression describe how children use the appraisals of others about their performance in particular domains to develop their self-schema (Cole, 1990, 1991; Cole et al., 1997). This may be similar to the development of the social self-concept in adolescence. According to these models, consistent negative feedback may lead to the development of depressive symptoms. Children are thought to seek and receive evaluation from significant others (e.g. parents, teachers, peers) in domains such as academic performance, social acceptance, athletic competence, personal conduct, and physical appearance. This feedback from others may be internalised throughout childhood. Frequent positive feedback may promote the development of complex positive self-constructs, whereby children feel competent and able to perform across a number of domains. However, if a child receives consistent negative feedback from a number of sources, they are more likely to construct a negative self-image, which may lead to a lack of confidence, low self-esteem, and feelings of helplessness (Cole, 1990, 1991; Cole et al., 1997). In support of these models, there is evidence that competence evaluations by others are associated with self-perceived competence over time, and changes in self-perceived competence are associated with subsequent changes in depressive symptoms (Cole et al., 1997). These beliefs about self competence may persist into adolescence and contribute to adolescents' vulnerability to depression.

1.9.4 Gender differences in social cognition

There is some evidence that the importance of the social environment may differ for boys and girls. Traditionally, researchers investigating gender roles have proposed that females are

more interpersonally oriented, and males are more achievement oriented (e.g. Ellemers, 2018; Kirsh & Kuiper, 2002; Stroud, Salovey, & Epel, 2002; Yang & Girgus, 2019). These stereotypes suggest that females have more communal goals, demonstrating warmth and caring for others, whereas men have greater agency, being assertive and focussed on performance (Ellemers, 2018). Gender stereotypes may be encouraged by socialisation during childhood and reinforced by increased awareness of gender norms in adolescence (Cyranowski, Frank, Young, & Shear, 2000; Kirsh & Kuiper, 2002). If so, more societal pressure on females to be social and interpersonally oriented could make them more vulnerable to the negative impacts of peer relationships.

In mid to late adolescence, girls report more fear of negative evaluation from peers, more avoidance and distress in new situations, more social support from their close friends, more intimacy in their close friendships, and perceptions of higher competency in their close friendships compared to boys (La Greca & Lopez, 1998). There is also evidence that social rejection is particularly salient for girls (Guyer, McClure-Tone, Shiffrin, Pine, & Nelson, 2009; Rudolph & Conley, 2005). For example, in one short-term longitudinal study of early adolescents, there was some evidence that social-evaluative concerns mediated the gender difference in depression (Rudolph & Conley, 2005). In this study, girls reported worrying more about social evaluation than boys. Increases in these social-evaluative concerns were then associated with more severe concurrent and subsequent depressive symptoms, even after adjusting for baseline depressive symptoms (Rudolph & Conley, 2005).

Consistent with this, in a prospective cohort of adolescents aged 9 to 13 years at baseline, girls reported more stressful interpersonal events than boys (Meiser & Esser, 2019). Interpersonal stress refers to all types of problematic social interactions, such as conflict with peers. In this study, reporting more interpersonal stress was associated with more severe depressive symptoms. There was also evidence that interpersonal stress partially mediated the association between gender and subsequent depressive symptoms, measured two years later (Meiser & Esser, 2019). Girls reported higher interpersonal stress, which was then associated with more severe depressive symptoms.

Girls may therefore have more negative social experiences than boys in adolescence, increasing their risk of developing depressive symptoms. In line with competency-based

models of depression (Cole, 1990, 1991; Cole et al., 1997), if girls receive more negative feedback from various sources across different domains during childhood, this might cause them to have more negative cognitions than boys. However, if girls have more dysfunctional attitudes, or more negative biases in the way that they process social information, this may lead them to perceive that they have more negative social experiences than boys, regardless of whether there are gender differences in the occurrence of these experiences. Either way, both negative social experiences and negative cognitions could lead to increased depressive symptoms for both boys and girls.

Overall, it is clear that adolescence is a distinctly social period. Interacting with peers requires lots of social information processing. Adolescents must attend to a variety of information, interpret and remember it, and then use this information to support their beliefs and decisions and develop a self-concept. This may be particularly difficult as social interactions are often ambiguous, requiring use of prior knowledge and learning to gain an understanding of new information. Negative schema and negative biases in processing social information may be particularly important for the emergence of the gender difference in depression during adolescence.

1.10 Underlying neural mechanisms

To examine the neurobiology underlying behaviour, many studies use neuroimaging. Although many types of neuroimaging exist, the most commonly used is functional magnetic resonance imaging (fMRI). This involves measuring blood oxygen levels in the brain, which are used to indicate activation in different brain regions and systems, whilst participants perform a task. Using this method, researchers can identify neural activity which may be associated with behaviour. They can then study individual differences in this neural activation, and test whether differences in activity are associated with depressive symptoms.

There have been many studies investigating the neural mechanisms underlying how adolescents think about their social relationships (e.g. Burnett, Bird, Moll, Frith, & Blakemore, 2009; Burnett, Sebastian, Cohen Kadosh, & Blakemore, 2011; Choudhury, Blakemore, & Charman, 2006; Kilford, Garrett, & Blakemore, 2016; Sebastian et al., 2011; Sebastian, Viding, et al., 2010). However, little is known about gender differences in neural development, and

this is a controversial field of research (Blakemore, Winston, & Frith, 2004). There is some evidence that there may be gender differences in the neural processing of social information (Guyer et al., 2009), but no clear patterns have yet been identified (Bolling et al., 2011; Pfeifer & Blakemore, 2012; Sebastian et al., 2011).

Neuroimaging studies generally have small samples because of high costs. This is an issue for investigating gender differences because, to test these gender differences, we need larger samples with equal proportions of boys and girls. Imaging studies are also very susceptible to selection bias. They are rarely representative of community or clinical populations as many people cannot participate or may not volunteer, for example those with severe anxiety. This may bias the findings from these studies. There is evidence that non-representative sampling in neuroimaging studies influences findings on associations between age and brain structure (LeWinn, Sheridan, Keyes, Hamilton, & McLaughlin, 2017). Many imaging measures also have low reliability (Nord, Gray, Charpentier, Robinson, & Roiser, 2017; Plichta et al., 2012). In contrast, many behavioural measures have good reliability, some of which have been established in large samples (e.g. Bland et al., 2016).

In order to understand task-related activation in functional imaging studies, we must first understand the behavioural association between task performance and depressive symptoms. If there is no association, it becomes difficult to interpret imaging results. It is therefore important to first understand associations between social information processing and depressive symptoms at a behavioural level, providing the groundwork for more hypothesis-driven neuroimaging studies.

1.11 Computational modelling

Computational psychiatry is a relatively new field which enables more in-depth investigation of behaviour and provides a potential way of more closely linking behaviour to underlying neural mechanisms. In this approach, researchers specify potential mechanisms causing task performance using precise mathematical algorithms (Adams, Huys, & Roiser, 2016; Browning et al., 2020; Maia, Huys, & Frank, 2017). A number of hypothesised cognitive processes can be tested and compared to determine which provide the best model of behaviour. In this way, computational models can potentially provide a mechanistic understanding of behaviour

on cognitive tasks. Parameters describing these underlying processes can then be included in analyses, testing whether they are associated with demographic factors (such as age and gender) and depressive symptoms, as well as neural activity. Computational modelling is therefore another useful approach to analysing social information processing, which will be explored in further detail in chapter 4.

1.12 Importance of identifying risk factors for the gender difference in depression during adolescence

Overall, my thesis aims to provide insights into the cognitive mechanisms underlying the emergence of the gender difference in depression during adolescence. At the moment, there are no widely accepted methods for preventing depression in boys or girls. One large barrier to the development of preventative interventions for depression is that the aetiology is poorly understood, as discussed throughout this chapter. We are still lacking robust causal evidence on risk factors for depression, as well as knowledge on how to mitigate them and thus prevent depression (Lewis, Jones, & Goodyer, 2016).

In this chapter, I have proposed that negative schema and social information processing biases are risk factors for depression which may be more prevalent in girls. This psychological vulnerability from negative schema and information processing biases might be a way to address the emergence of depressive symptoms in adolescence. It may be more easily modified than external causes of depression traditionally identified in epidemiological research. Interventions that reduce negative schema and biases in information processing might increase resilience to stressors and reduce depressive symptoms in boys and girls. Public health strategies could also target more basic information processing in order to successfully change behaviour and prevent depression (as recommended by Marteau, Hollands, & Fletcher, 2012). The ongoing neural development in the adolescent brain may make this a particularly good time for interventions designed to modify cognition.

Reducing the incidence of depression in adolescence may have large public health benefits. It could potentially lead to reductions in physical health problems, academic problems, substance abuse, high risk sexual behaviour, and suicide, as well as improving social relationships and reducing the risk of subsequent recurrences of depression (Birmaher et al.,

1996; Horowitz & Garber, 2006; Lewinsohn, Rohde, et al., 2000; Public Health England, 2017; Thapar et al., 2012).

1.13 Aims and hypotheses in my thesis

In this chapter, I have provided an overview of the literature on the emergence of the gender difference in depression. This highlighted our lack of understanding of factors which are associated with the gender difference in depressive symptoms during adolescence. I have proposed a novel hypothesis (reiterated below), describing one potential mechanism which may contribute to the gender difference in depression during adolescence. Research is required to test this hypothesis and identify potential mediators of the gender difference in depression. This could provide psychological targets for interventions and prevention strategies to reduce the incidence of depression during adolescence.

1.13.1 My novel hypothesis

Throughout this chapter, I have discussed the hypothesis that negative schema and social information processing biases mediate the gender difference in depression. According to my hypothesis, negative schema and social information processing biases are a risk factor for depressive symptoms, and this risk factor may be more prevalent in girls from early adolescence. I have thus proposed that gender should be classified as an exposure variable within the causal pathway described by cognitive models of depression (Bone et al., 2020). In this causal pathway, being female is associated with more negative schema and social information processing biases, which are then associated with increased depressive symptoms. I have hypothesised that this causal pathway will be present from early adolescence, presenting a risk factor for depressive symptoms which does not change with age during adolescence.

1.13.2 Testing mediation and moderation

My hypothesis that negative schema and social information processing may mediate the association between gender and depressive symptoms has implications for the temporal order of these events. If negative schema and social information processing are mediators, differences must occur as a result of gender, and changes should precede increases in depressive symptoms. In order to ultimately test whether this is a valid model, longitudinal

data are required. However, tests of mediation only indicate if a specified model is plausible, whether analyses are implemented in cross-sectional or longitudinal data (Maxwell & Cole, 2007; Maxwell, Cole, & Mitchell, 2011; Shrout, 2011). Cross-sectional data can thus be used as a first step in testing whether a mediation model is plausible.

As no cohort studies have included measures of social information processing during adolescence, and conducting a prospective cohort study of adolescents was not feasible within my PhD, I collected cross-sectional data. Using cross-sectional data allowed me to test whether gender was associated with the negative cognitions outlined above (which cannot be a result of reverse causation). It also enabled tests of the associations between negative cognitions and depressive symptoms in a population-based sample, which indicates whether negative cognitions could be a mediator of the association between gender and depressive symptoms in adolescence (although associations between negative cognitions and depressive symptoms could be a result of reverse causation). In each chapter, I have tested these associations but have not tested a full mediation model. The only exception is in chapter 6, testing dysfunctional attitudes, where the nature of analyses (structural equation modelling) lent itself to specifying and testing a mediation model. My aim was to test whether my proposed mediation model may be plausible and the limitations of this approach are discussed in more detail in chapter 6 and the general discussion. I have not aimed to provide any evidence for causal associations in my cross-sectional study.

Additionally, throughout this chapter, I have argued that testing information processing as a mediator is a better approach than treating gender as a moderator. However, as most previous studies have tested gender as a moderator, I have also used this approach. My overarching hypothesis suggests that I would not find any evidence that age or gender modify the associations between negative cognitions and depressive symptoms during adolescence. In order to compare my findings to previous research, I tested whether age and gender moderated associations between negative cognitions and depressive symptoms, despite the limitations of this approach. Finding no evidence of effect modification would be consistent with my hypothesis.

1.13.3 Approach used in my thesis

In order to outline my aims and hypotheses, I will first briefly describe the data used in my thesis. I took an epidemiological approach to examine cognitive neuroscience risk factors for the development of depressive symptoms in adolescence. I conducted a cross-sectional study measuring performance on computerised cognitive tasks and traditional self-report measures. I used a developmental approach to investigate whether these processes change across adolescence. Participants were selected from two age groups, young adolescents (aged 11-12 years) and mid-adolescents (aged 13-15 years). This recruitment strategy aimed to span the age at which rates of depression start increasing (Kwong et al., 2019; Merikangas et al., 2010), and to capture adolescents' transition from early to late puberty (Parent et al., 2003; Patton & Viner, 2007).

1.13.4 Aims of each chapter in my thesis

Throughout this chapter, I have mentioned a number of aims for my thesis, which I will now describe in more detail. The main aim of my thesis was to investigate an explanation for the emergence of the gender difference in depressive symptoms during adolescence. I aimed to test three different aspects of negative cognitions - learning about social evaluation, recall of social evaluation, and dysfunctional attitudes. Within each type of cognition, I aimed to test whether there were gender differences and if these gender differences changed with age. I also tested whether these negative cognitions were associated with depressive symptoms in adolescence. The specific aims of each study are listed below, with the hypotheses described in more detail in the next section.

Study 1 aim: To investigate learning about social evaluation, examine whether there are gender differences in this learning, explore whether these gender differences change with age, and test whether learning about social evaluation is associated with depressive symptoms in adolescence (chapter 3).

Study 2 aim: To investigate potential processes underlying learning about social evaluation, examine whether there are gender or age differences in these processes, and test whether the processes underlying learning about social evaluation are associated with depressive symptoms in adolescence (chapter 4).

Study 3 aim: To investigate recall of self-referential and other-referential social evaluation, examine whether there are gender differences in this recall, explore whether these gender differences change with age, and test whether recall of social evaluation is associated with depressive symptoms in adolescence (chapter 5).

Study 4 aim: To investigate whether there are gender differences in perfectionism and need for approval, examine whether these gender differences change with age, and test whether perfectionism and need for approval are associated with depressive symptoms in adolescence (chapter 6).

1.13.5 Hypotheses of each chapter in my thesis

Overall, I hypothesised that girls would have more negatively biased social information processing and dysfunctional attitudes than boys. I hypothesised that these negative biases in social information processing and dysfunctional attitudes would be present from early adolescence, so would not differ across young and mid-adolescents. I also hypothesised that more negative biases in social information processing and dysfunctional attitudes would be associated with more severe depressive symptoms. The specific hypotheses for each study are justified in more detail in each chapter. These hypotheses are numbered consecutively throughout my thesis and are outlined below according to chapter.

1.13.5.1 Chapter 3: Learning about social evaluation during adolescence: gender differences and associations with depressive symptoms

In my first study, I used a social evaluation learning task, in which participants learnt whether a person was liked or disliked by a computer character. After learning, participants rated each character's overall opinion of the person. I examined whether learning differed according to whether social evaluation described the self (self-referential) or another person (other-referential) and whether the person was liked or disliked. Adults demonstrate a positive bias on this task, as they are better at learning that they are liked relative to disliked (Button, Browning, Munafò, & Lewis, 2012; Button et al., 2015). This positive bias is reduced with increased depressive symptoms (Hobbs, Sue, Kessler, Munafò, & Button, 2018). This task has never been used with adolescents.

Hypothesis 1.1: I hypothesised that, as in adults, adolescents would demonstrate a positive self-referential bias. This would be reflected in choosing the positive response option more often when learning about the self than other people, demonstrating poorer learning that someone disliked the self than another person. It would also be evident in rating the character's opinion of the self as more positive than their opinion of the other after learning.

Hypothesis 1.2: I hypothesised that this positive self-referential bias would be smaller in girls than boys.

Hypothesis 1.3: I also hypothesised that this gender difference would be present from early adolescence, so the influence of gender would not differ across age groups.

Hypothesis 1.4: I hypothesised that this positive self-referential bias would be negatively associated with depressive symptoms.

Hypothesis 1.5: Finally, I hypothesised that the association between the positive self-referential bias and depressive symptoms would not differ across genders or age groups.

1.13.5.2 Chapter 4: Computational mechanisms underlying social evaluation learning during adolescence

In my second study, I extended my findings from chapter 3 by examining potential processes underlying learning about social evaluation in adolescence, investigating how social feedback influences learning and future decisions. To do this, I developed and validated a computational model describing how adolescents learnt about social evaluation. I aimed to develop reinforcement learning models to describe trial-by-trial patterns of behaviour, parameterising the processes involved in learning about social evaluation during adolescence.

Hypothesis 2.1: Based on my findings in chapter 3, I hypothesised that a number of parameters would be necessary for reinforcement learning models to adequately describe adolescents' behaviour, including separate learning rates for self-referential and other-referential information and parameters modelling a positive self-referential bias.

Hypothesis 2.2: I hypothesised that none of these parameters would change with age.

Hypothesis 2.3: I also hypothesised that parameters relating to the positive self-referential bias would be smaller in girls than boys, in both young and mid-adolescents.

Hypothesis 2.4: Finally, I hypothesised that the positive self-referential bias parameter(s) and self-referential learning rate(s) would be associated with depressive symptoms, across both genders and age groups.

1.13.5.3 Chapter 5: Recall bias during adolescence: gender differences and associations with depressive symptoms

In my third study, I focussed on another aspect of social information processing, recall biases. Negative memory biases are thought to be associated with depressive symptoms, but evidence from studies with adolescents has been inconsistent to date (Platt et al., 2017). I developed a test of recall biases, which measured recall of social evaluation (positive and negative personality traits). This novel task allowed me to examine whether recall differed according to whether words were seen describing the self (self-referential) or another person (other-referential) and word valence (positive or negative).

Hypothesis 3.1: I hypothesised that, overall, adolescents would have a self-referential bias, recalling more self-referential than other-referential words.

Hypothesis 3.2: I also hypothesised that adolescents' self-referential bias would be positive, as demonstrated by recall of more self-referential positive than self-referential negative words.

Hypothesis 3.3: I hypothesised that girls would demonstrate less positive self-referential recall biases than boys, recalling fewer self-referential positive and more self-referential negative words.

Hypothesis 3.4: I hypothesised that this gender difference in recall biases would be present from early adolescence, so would not differ across age groups.

Hypothesis 3.5: I also hypothesised that self-referential recall biases would be associated with depressive symptoms. Specifically, I predicted that self-referential positive recall would be negatively associated with depressive symptoms, and self-referential negative recall would be positively associated with depressive symptoms.

Hypothesis 3.6: Finally, I hypothesised that the association between self-referential recall biases and depressive symptoms would be consistent across genders and age groups.

1.13.5.4 Chapter 6: Dysfunctional attitudes during adolescence: gender differences and associations with depressive symptoms

In my fourth and final study I used a different approach to measure another related aspect of cognition. I investigated dysfunctional attitudes using the Dysfunctional Attitude Scale (De Graaf et al., 2009; Weissman, 1979; Weissman & Beck, 1978), a self-report questionnaire. Dysfunctional attitudes are higher-level negative schema, which individuals can reflect upon when asked, and are thus usually tested via self-report. To my knowledge, it is not possible to assess these overarching negative schemas through cognitive tasks.

Hypothesis 4.1: I hypothesised that perfectionism would be higher in boys and need for approval would be higher in girls.

Hypothesis 4.2: I expected dysfunctional attitudes to be present from early adolescence, and thus hypothesised that there would be no association between age group and dysfunctional attitudes.

Hypothesis 4.3: I hypothesised that perfectionism and need for approval would both be positively associated with depressive symptoms.

Hypothesis 4.4: I also hypothesised that dysfunctional attitudes would mediate the association between gender and depressive symptoms. I expected girls to have higher need for approval, which would then be associated with more severe depressive symptoms.

Hypothesis 4.5: Finally, I hypothesised that these associations between gender, perfectionism, need for approval, and depressive symptoms would be present from early adolescence, so would not differ across the two age groups.

1.14 Conclusion

In this chapter, I have presented a novel hypothesis to explain the gender difference in depression during adolescence. Next, I will describe the methods I used to test this

overarching hypothesis (chapter 2), before testing the specific hypotheses outlined above (chapters 3-6). I then conclude with an overall discussion of my findings (chapter 7).

Chapter 2 General methods

2.1 Participants

I recruited a population-based sample of adolescents from mixed gender secondary schools across London. All adolescents in Year 7 (11-12 years old) and Years 9-10 (13-15 years old) were eligible to participate. I had planned to recruit only Year 10s into the older group (who were aged 14-15) but experienced difficulties with recruitment. Teachers of Year 10 had very little free time and schools were reluctant for these students to miss any lessons. Therefore, instead of recruiting Year 10s in one of the participating schools, Year 9s who were right at the end of the school year (so very nearly Year 10s) were eligible to participate.

I chose these two age groups to span the age at which rates of depression start to increase (Kwong et al., 2019; Merikangas et al., 2010), and to capture adolescents' transition from early to late puberty (Parent et al., 2003; Patton & Viner, 2007). This meant I could study gender differences before and after the age at which depression starts increasing. In order to increase the generalisability of my findings, I did not impose any restrictions on whether adolescents had any mental or physical health problems or were receiving psychotropic medication or psychological therapy.

To show a difference of 0.4 standard deviations in the outcomes between boys and girls, at an alpha of 0.05 and a power of 0.80, I needed a sample of 320 adolescents. I determined this effect size based on findings from a previous study which used the social evaluation learning task in adults (Button et al., 2015). I was interested in testing gender differences in both young and mid-adolescents, so doubled this sample size, aiming to recruit a total of 640 participants. I also intended to perform subgroup analyses, so aimed to include approximately 160 adolescents of each gender at each age.

2.2 Ethical approval

I obtained ethical approval from University College London (project ID 3453/001). Informed assent was provided by all participants. Participants' parents/carers provided informed opt-in or opt-out consent, dependent on the school their child was attending. Of the eight schools I recruited for the study, seven required opt-in parental consent and one allowed opt-out

parental consent. Only seven parents/carers chose to opt-out (2% of those contacted). All procedures complied with the ethical standards of the relevant committees on human experimentation, the Helsinki Declaration (2008 revision), and the General Data Protection Regulation.

2.3 Social information processing tasks

2.3.1 Social Evaluation Learning task

The social evaluation learning task is a two-alternative forced choice task based on probabilistic stimulus-reward learning tasks (Button et al., 2012; Button, Karwatowska, Kounali, Munafò, & Attwood, 2016; Button et al., 2015; Chamberlain et al., 2006). It uses pseudo-social interactions to assess participants' ability to learn whether people are liked or disliked, based on feedback. I adapted the task developed by Button and colleagues (Button et al., 2012, 2016, 2015) to be more appropriate for use with adolescents.

Before starting the task, participants are told that they will meet different computer characters. For each character, one of two social rules was learnt: the person is liked by the character or the person is disliked by the character. Participants are not aware of these rules, but the rules are learnt in one of two conditions: about the participant themselves (self-referential) or about another person called Taylor (other-referential). There are thus four different blocks in this task: self like, self dislike, other like, and other dislike.

At the start of each block, the computer character introduces themselves and tells the participant that their task is to decide what they (the character) thinks of either the participant themselves (self-referential) or Taylor (other-referential). After a fixation cross, a positive and negative word pair is presented (e.g. good/bad, funny/grumpy). Participants are asked to choose the word which best corresponds to what the character thinks about them (self-referential blocks) or Taylor (other-referential blocks). No time limit is imposed upon word selection. They then receive probabilistic feedback on the screen about whether their choice was correct (green tick) or incorrect (red cross). Feedback was always shown for 500ms. From this feedback, participants are asked to use trial and error to learn whether the computer character likes or dislikes them (or Taylor) over 20 trials. See Figure 2.1 for a depiction of two trials in this task.

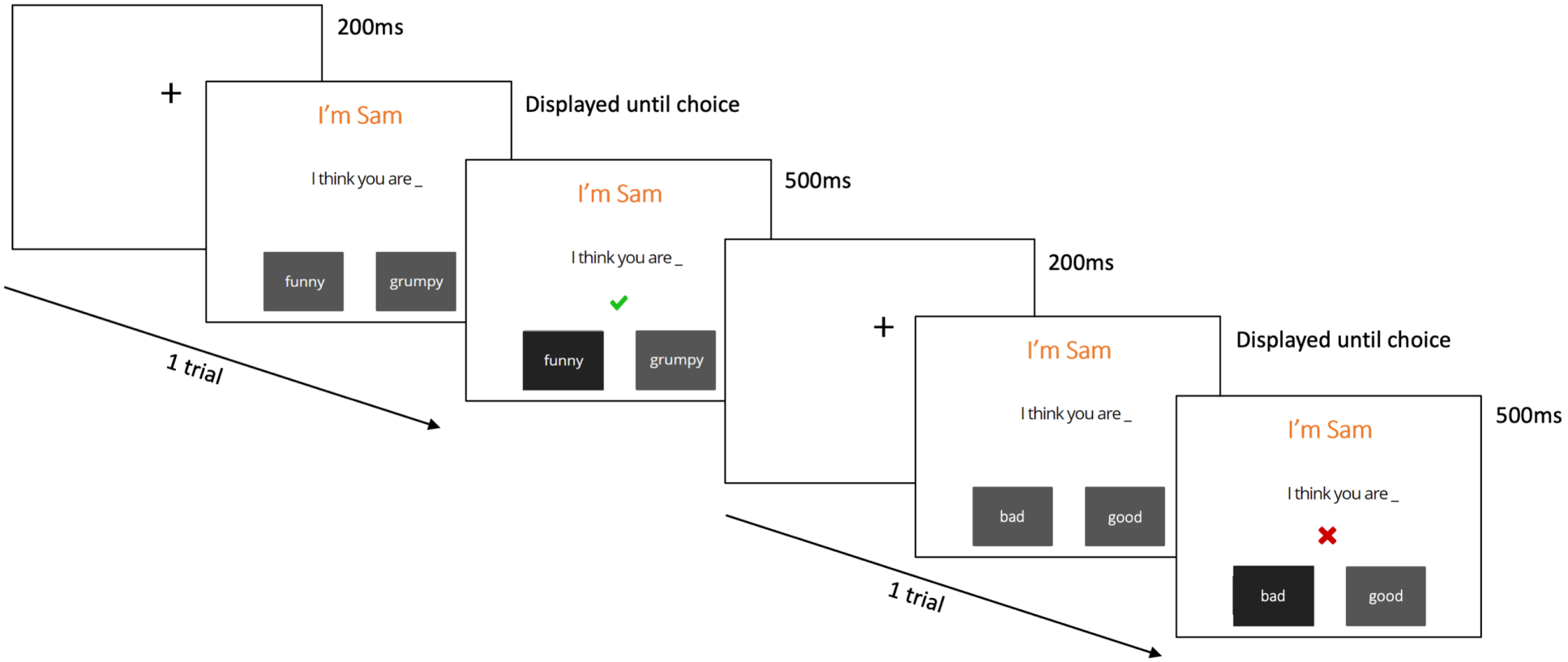

Figure 2.1 Social evaluation learning task. An example of two trials from a self-referential block, in which the computer character is called Sam and the participant is learning what Sam thinks of them. After viewing a fixation cross, the participant is presented with a positive and negative word pair and instructed to choose the word which best corresponds to what Sam thinks about them. No time limit is imposed upon word selection. Participants then receive feedback about whether their choice is correct (green tick) or incorrect (red cross). Participants use trial and error to learn whether the character likes or dislikes them over 20 trials. In the first trial shown here, the participant selected the positive word, which was correct. In the second trial, the participant chose the negative word, which was incorrect. Both trials show true (not misleading) feedback.

To prevent ceiling effects, I set feedback contingency to 80%, with feedback for each block corresponding to the two rules. The positive word was correct in 80% of like trials and the negative word was correct in 80% of dislike trials. These contingencies were implemented so that "correct" responses received an 8:2 ratio of positive to negative feedback and "incorrect" responses received an 8:2 ratio of negative to positive feedback. Misleading feedback was therefore given on 20% of trials.

In each block, the participant is introduced to a new character. The participant "interacts" with this character for 20 trials (20 word pairs presented) in the learning phase of this task. The block then ends with a global rating, where the participant is asked to rate how much they thought the character liked them (self-referential) or Taylor (other-referential). Ratings are made by moving a pointer on a sliding scale (0% = dislike, 100% = like). This global rating requires participants to reflect on their learning in the previous 20 trials.

I counterbalanced the order of presentation of self/other conditions, and the like/dislike rules within conditions, across participants. Other-referential learning is always about Taylor, but participants "meet" a new computer character on each block. I called the characters Charlie, Sam, Jo, and Alex, and I counterbalanced these character names across conditions and rules. I chose unisex character names to prevent any influence of the characters' sex on learning about social evaluation, as participants could have demonstrated biases towards same-sex characters (Benenson & Christakos, 2003; Buhrmester & Prager, 1995). In order to use this task with adolescents, I reduced the number of trials in each block from 32 to 20 and replaced the original word pairs with personality trait descriptors suitable for this age group (see section 2.3.1.1).

There are 40 word pairs presented in the task in total (Appendix 3). Twenty word pairs are seen for the self, and 20 for the other person, with each word pair seen twice (once each in the like and dislike blocks). I counterbalanced word pairs across blocks, and positive and negative words appeared randomly on the left or right of the screen.

Several outcome variables can be analysed from this task including positive responses, bias scores, errors, and errors to criterion during learning (Button et al., 2016, 2015; Hobbs et al., 2018). I chose to analyse the number of positive responses during learning as they have been

tested previously (Button et al., 2016, 2015) and are equivalent across task conditions. In contrast, number of errors refers to different actions in like rules (error = choosing negative word) and dislike rules (error = choosing positive word). Additionally, positive responses do not rely on meeting an arbitrary criterion, such as a certain number of correct responses in a row (as used in the errors to criterion variable; Button et al., 2012). I recorded positive responses as the number of times participants chose the positive word, which could range from 0 to 20 for each block. I also measured the global rating after each block, which could range from 0 to 100 (0%=dislike, 100%=like). Global ratings required participants to sum information across their recent interactions to provide an explicit measure of overall learning. Choices (positive or negative word selected) and feedback given by the computer on each trial were also recorded for use in reinforcement learning models.

A prior study demonstrated that this task has moderate test-retest reliability in adults. In 144 adults who completed two sessions approximately one week apart, the intraclass correlation coefficient (ICC) was calculated in a linear multilevel model with time points clustered within individual. The ICC for self-referential learning was 0.61 (95% CI 0.46 to 0.72; Hobbs, personal communication). Additionally, the task demonstrated stable associations with depressive symptoms over time. In the same sample, over approximately one week, an ICC of 0.88 was observed between task performance and depressive symptom severity (measured using the Patient Health Questionnaire - PHQ-9; Button & Hobbs, 2020).

2.3.1.1 Stimuli presented

Personality descriptors were emotive adjectives describing trait characteristics (e.g. cool/boring, funny/grumpy, generous/greedy). I selected positive and negative words from databases according to their age of acquisition (Brysbaert & New, 2009; Grühn, 2016; Kučera & Francis, 1967; Leech, Rayson, & Wilson, 2014; Warriner, Kuperman, & Brysbaert, 2013). The oldest mean age of acquisition of any included word was 8.78 years (SD=1.99). I prioritised words with more psycholinguistic data available and semantically categorised words as positive and negative. I then paired words, matched firstly on age of acquisition. I also aimed to pair semantically linked words, minimise differences in psycholinguistic parameters (number of syllables, usage frequency, meaningfulness, familiarity, arousal), and maximise differences in likeableness, valence, and desirability ratings (Brysbaert & New, 2009; Grühn, 2016; Kučera & Francis, 1967; Warriner et al., 2013).

2.3.2 Surprise recall task

I assessed incidental memory for the personality descriptors presented in the social evaluation learning task using a surprise recall task. After a delay of approximately four minutes, participants were asked to remember as many of the personality descriptors as possible. They were given two minutes to perform this free recall task, typing responses on the computer. A countdown timer appeared when participants had 30 seconds remaining. I recorded any misspelled words that resembled correct responses as correct to ensure that spelling errors did not bias accuracy rates. The number of self-referential and other-referential positive and negative words accurately recalled (hits), and the number of positive and negative incorrect responses (false alarms) were calculated. The key outcome variables from this task were therefore self-referential positive hits, self-referential negative hits, other-referential positive hits, other-referential negative hits, positive false alarms, and negative false alarms.

2.4 Questionnaires

I asked participants to complete a battery of self-report questionnaires online. I chose all of these questionnaires to be age appropriate. Where possible, I selected brief or short forms of measures due to the time-constraints imposed by classroom testing. Next, I will describe all of the questionnaires included in this battery in the order in which they were completed.

2.4.1 Raven's Standard Progressive Matrices

Participants completed a nine-item abbreviated version of the Raven Standard Progressive Matrices Test (RSPM; based on Bilker et al., 2012). This multiple-choice test of abstract reasoning requires participants to select the missing item in nine black and white matrices. It was a brief measure of non-verbal fluid intelligence. The psychometric properties associated with this short-form are comparable with the full-length Raven's Progressive Matrices test and a 30-item version, with time savings of over 75% for administration (Bilker et al., 2012). On average, participants took approximately four minutes to complete these matrices.

2.4.2 Mood and Feelings Questionnaire

The Mood and Feelings Questionnaire (short version; SMFQ) is a 13-item self-report measure of depressive symptoms over the last two weeks, developed for children and adolescents

(Angold, Costello, Messer, & Pickles, 1995; Thapar & McGuffin, 1998; Turner, Joinson, Peters, Wiles, & Lewis, 2014). Each item was rated on a scale of 0-2 (possible total scores 0-26), with higher scores indicating greater severity. Although the SMFQ is not a diagnostic measure, scores of 12 or higher indicate the possible presence of depression (Angold et al., 1995).

2.4.3 Affective Reactivity Index

The Affective Reactivity Index (ARI-S) is a self-report measure of irritability over the last 6 months (Stringaris et al., 2012). Total score was summed from six individual items (each item 0-2; possible total 0-12), with higher scores indicating more irritability. A seventh impairment item (rated 0-2) indicates how much participants' irritability causes problems.

2.4.4 Revised Child Anxiety and Depression Scale

The Revised Children's Anxiety and Depression Scale (RCADS) is a self-report questionnaire designed for 8-18 year olds, with subscales measuring separation anxiety disorder, social phobia, generalized anxiety disorder, panic disorder, obsessive compulsive disorder, and low mood (Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000). Only the generalised anxiety disorder subscale of the RCADS child self-report questionnaire was used, consisting of 5 questions about how often participants worry about things. No time frame was specified. I removed an additional item from the original generalised anxiety disorder subscale of the RCADS ('I think about death'), as the questionnaire would be used in a classroom setting. Each item was rated on a scale from 0-3, giving a total possible score of 0-15, with higher scores indicating more severe generalised anxiety symptoms.

2.4.5 Dysfunctional Attitude Scale

The Dysfunctional Attitude Scale (Form A Revised; DAS) is a 17-item self-report measure of the presence and intensity of dysfunctional attitudes (De Graaf et al., 2009; Weissman, 1979; Weissman & Beck, 1978). Each item consists of a statement and a seven-point Likert scale (1=fully disagree, 7=fully agree). The total score is the sum of the seventeen items (possible range 17-119), with a higher score indicating more dysfunctional attitudes. This version of the DAS has previously been divided into two subscales, with 11 items measuring perfectionism and the other six items measuring need for approval (De Graaf et al., 2009). The items

included in this questionnaire are appropriate for adolescents and have high internal consistency (Rogers et al., 2009).

2.4.6 Health and Social Risks Questionnaire

The Health and Social Risks Questionnaire (HSRQ; Andrews et al., 2020) assesses the degree to which adolescents and adults are concerned about engaging in health and social risk behaviours. I collected data on a preliminary version of this questionnaire, which was still in development. In this initial version, participants were given a list of 16 risky actions, including eight health risks (e.g. pick up broken glass with bare hands) and eight social risks (e.g. argue with a popular friend in front of a group of people). They were asked to rate how worried they would feel doing this behaviour on a scale from 0 (not worried at all) to 100 (very worried). After data collection, I combined responses on this questionnaire in my sample with data from other samples, collected by colleagues, in a validation study of this questionnaire (see Appendix 2 for more information). After validation, the final version of this questionnaire included 11 risky actions (five health risks and six social risks; Appendix 2).

2.4.7 Social Reward Questionnaire (Adolescent Version)

The Adolescent Version of the Social Reward Questionnaire (SRQ-A) was developed for 11 to 16 year olds (Foulkes, Neumann, Roberts, McCrory, & Viding, 2017). Twenty statements describe what participants may enjoy when they spend time with other people in their life (friends, classmates, strangers, etc.). Each statement was scored on a seven-point Likert scale from strongly disagree (1) to strongly agree (7).

I calculated mean scores for each of five subscales of the SRQ-A (admiration, negative social potency, passivity, prosocial interactions, sociability). In this questionnaire, four items measure admiration, the enjoyment of being flattered and gaining positive attention. Five items measure negative social potency, the enjoyment of being cruel, antagonistic and using others. Three items measure passivity, the enjoyment of giving others control and allowing them to make decisions. Five items measure prosocial interactions, the enjoyment of having kind and reciprocal relationships. Finally, three items measure sociability, the enjoyment of engaging in group interactions.

2.4.8 Children's Rejection Sensitivity Questionnaire

The Children's Rejection Sensitivity Questionnaire (CRSQ) is a six-item self-report measure of individuals' disposition to defensively expect, readily perceive, and overreact to social rejection (Downey, Lebolt, Rincón, Freitas, & Freitas, 1998). It consists of six social scenarios, in which participants imagine themselves. For example, one scenario is "Imagine you had a really bad fight the other day with a friend. Now you have a serious problem and you wish you had your friend to talk to. You decide to wait for your friend after class and talk with him/her. You wonder if your friend will want to talk to you."

Following each vignette, participants are asked to respond to three questions. The first two questions assess anxious and angry responses by asking how nervous and how mad they would feel in this situation. Responses to these two items range from 1 (not nervous/mad) to 6 (very very nervous/mad). In the third question, participants reported the likelihood of an accepting versus a rejecting response from 1 (Yes!) to 6 (No!). Scoring of the CRSQ weights participants' expectation of acceptance versus rejection by their anxious and angry responses. Two scores for each vignette are calculated by multiplying the response to the expectation item by individuals' responses regarding anxiety and anger. Responses are then summed to produce cross-situational anxiety and anger scores. Finally, scores across the six vignettes can be averaged to provide a total rejection sensitivity score. Higher scores indicate more rejection sensitivity.

2.4.9 Olweus Bully/Victim Questionnaire (Revised)

I derived a version of the Olweus Bully/Victim Questionnaire (OBVQ), which first gives participants a definition of bullying. The questionnaire then has two global questions on victimisation "how often have you been bullied at school in the past couple of months?" and bullying behaviour "how often have you taken part in bullying another student(s) at school in the past couple of months?" There are then nine additional questions on different types of victimisation, which can be clustered into four factors: verbal, indirect, physical, and cyberbullying. These items were taken from the Revised OBVQ (Kyriakides, Kaloyirou, & Lindsay, 2006). I took an additional question, which asks specifically about experiences of cyberbullying (bullying behaviours performed over the phone or internet), from another version of the Olweus Bully/Victim Questionnaire (Bevans, Bradshaw, & Waasdorp, 2013).

Each item is rated on a five-point scale, based on frequency in the past couple of months, ranging from never (0) to several times a week (4). Three different scores can be calculated from this questionnaire. Firstly, global victimisation is based on the single question of whether participants have been bullied in the past couple of months (possible total 0-4). Secondly, number of victimisation behaviours experienced is the sum of the nine items on different types of bullying, including cyberbullying (possible total 0-36). Finally, global bullying is based on the single question of whether participants have bullied other students in the past couple of months (possible total 0-4). Higher scores indicate more frequent victimisation/bullying in the past couple of months.

2.4.10 Pubertal Development Scale

The Pubertal Development Scale (PDS) is a self-report measure of pubertal status (Petersen, Crockett, Richards, & Boxer, 1988). It consists of three questions about the development of body hair, the occurrence of a growth spurt, and changes in complexion plus two sex-specific items. Girls are asked about breast development and the onset of menstruation whereas boys are asked about changes in voice and growth of facial hair. Responses are coded on 4-point scales (1 = no development, 4 = completed development) except for the question about onset of menarche, which is answered with the options yes or no.

Pubertal development, as measured on the PDS, can be classified into the five Tanner stages - prepubertal, early pubertal, mid-pubertal, late pubertal, and post-pubertal (Carskadon & Acebo, 1993; Crockett, 1988; Norris & Richter, 2008). However, in my study, very few participants completed this measure (see section 2.9.4) and there was a low and unbalanced number of participants in each Tanner stage, particularly in prepubertal and early pubertal stages. I therefore created a binary pubertal stage variable, categorising participants as either early/mid puberty (which corresponded to Tanner stages 1-3) or late/post puberty (corresponding to Tanner stages 4-5). Girls in the early group were pre-menarche and girls in the late group were post-menarche. Boys in the early group had low individual ratings on growth of body hair, voice change, and growth of facial hair growth compared to boys in the late group.

2.4.11 Strengths and Difficulties Questionnaire

The Strengths and Difficulties Questionnaire (SDQ) is a self-report measure suitable for 11-16 year olds (Goodman, Meltzer, & Bailey, 1998). it measures emotional and behavioural problems over the last six months. Twenty-five items are rated on a scale of 0-2. These items are divided equally between five subscales - emotional symptoms, conduct problems, hyperactivity/inattention, peer relationship problems, and prosocial behaviour. A total difficulties score is generated as the sum of the first four of these subscales (excluding prosocial behaviour responses) and total possible scores range from 0 to 40, with higher scores indicating more difficulties.

2.5 Information from parents

I developed a parental questionnaire for participants' parents/carers. I asked parents/carers to report their relationship to the participant and participants' age, ethnicity, first language, whether the participant had been diagnosed with a mental health problem (yes/no), and diagnoses if applicable (options: anxiety, conduct disorder, depression, eating disorder, generalised anxiety disorder, panic attacks or panic disorder, post-traumatic stress disorder, social phobia or social anxiety, substance misuse disorder, other). I asked whether the participant had ever been seen by mental health services, was currently taking antidepressants, or was receiving psychological therapy for depression. I also asked whether the participant had any special educational needs and, if so, whether they had attention deficit/hyperactivity disorder (ADHD), autistic spectrum disorder (ASD), dyslexia, emotional and behavioural difficulties, epilepsy, or any other needs. Finally, I asked parents/carers about the participants' mother and father's highest qualification and history of mental health problems (depression, anxiety, stress, none of the above), where known.

I grouped ethnicity into 5 categories, based on the most common ethnicities – White, Mixed, Asian/Asian British, Black/Black British, and Other. I also created a binary first language variable, indicating whether the participant's first language was English (0=no, 1=yes). As an indicator of socioeconomic status, I took the highest reported parental qualification for each participant (across both mother and father). I then split parental education into low (0=highest qualification GCSE or lower) and high (1=A Levels or higher). As my social information processing tasks involved skills that are known to be affected in dyslexia and ASD,

I also created binary variables to indicate whether participants had been diagnosed with dyslexia or ASD (0=no, 1=yes), as reported by parents.

2.6 Participant advisory group

During the development phase of my study, I recruited a group of five Year 10 students to create a participant advisory group. These students were from an eligible secondary school which intended to participate in my study. I had several meetings with the group whilst planning and designing my study. They assisted with the design of the social information processing tasks, checked the language of all study materials, and provided feedback on and suggestions for my proposed procedures for the study.

2.7 Piloting

I piloted the social information processing tasks and questionnaires initially with my colleagues, and then with my participant advisory group. Following this, I also asked a group of undergraduates to complete the tasks (during a lecture) to assess the feasibility of classroom data collection. After collecting feedback from this piloting and checking the task data, I made a number of changes to the tasks, including adding colour to the characters' names in the social evaluation learning task, making the font larger, making the instructions clearer, and generally improving the layout of the tasks for compatibility with different sized screens. I also added more instructions and checkpoints throughout the questionnaires, as well as splitting some measures across separate pages.

2.8 Procedure

I collected data between November 2018 and July 2019, recruiting a range of mixed secondary schools from across London. In schools requiring opt-in consent (seven schools), I sent information sheets and opt-in consent forms (Appendix 4) to parents/carers of all Year 7 and 10 students. I asked parents/carers to return a completed consent form to the school or complete it online via Gorilla (www.gorilla.sc). I attached the parental questionnaire to the consent form (Appendix 4) and asked parents/carers to either return the paper version or complete it online whilst providing consent.

In the one school which agreed to use opt-out consent, I automatically enrolled all students in Years 7 and 9 in the study (see section 2.1 for an explanation of why Year 9s were included). I sent participants' parents/carers information sheets and a form to opt their child out of completing the study (Appendix 4) which they could return to the school, post back to me, or complete online (via Gorilla). I also emailed the parental questionnaire (Appendix 4) to all parents/carers for completion.

After obtaining parental consent, I collected data with groups of between two and 31 adolescents using computers, laptops, or tablets. Exact procedures varied across schools, but I did most data collection in classrooms during a lesson. At least one member of the research team was present alongside the teacher in all classrooms. My research term consisted of me and several UCL MSc and PhD students, who I trained on all of the measures included in my study as well as general data collection procedures. All researchers had enhanced DBS checks. During data collection, my research team explained procedures to participants, answered questions, and provided support where needed.

All data collection was computerised and completed online using Gorilla. Gorilla is a cloud software platform developed specifically for the behavioural sciences. It is hosted on Microsoft Azure within the European Union. All traffic to and from Gorilla is encrypted (TLS/SSL) and the database is encrypted using industry-standard cryptography. Gorilla is fully compliant with data protection legislation and British Psychological Society guidelines.

After reading participant information sheets and providing informed assent (Appendix 4), participants were asked basic demographic questions about their age, gender, school, and year group. They then completed the social evaluation learning task, followed by the RSPM, and the surprise recall task. The SMFQ was then completed, followed by other study questionnaires in the following order: ARI-S, RCADS, DAS, HSRQ, SRQ-A, and CRSQ. The SMFQ, ARI-S, RCADS and DAS were labelled as key questionnaires, and participants were offered the option to stop data collection after completing them (without completing the HSRQ, SRQ-A, or CRSQ). I did this to allow for different data collection time limits across schools (based on lesson lengths) as well as the large range in the time it took to participants to complete these measures (between 15 and 40 minutes).

Within a week of classroom data collection, I emailed participants (via Gorilla) asking them to complete additional questionnaires at home. I sent participants who did not complete these additional questionnaires two further reminder emails over the following two months. Additional questionnaires were the OBVQ, PDS, and SDQ, completed in this order. I did not ask participants to complete these questionnaires in the classroom due to time constraints and the sensitive content on bullying and pubertal development. After completing these questionnaires, participants could opt-in to a prize draw to win a £50 Amazon voucher. I had one voucher available for approximately every 50 students. I held prize draws periodically, and emailed vouchers to winning participants. I aimed to improve response rates for the additional questionnaires using this prize draw.

2.9 Data collected

2.9.1 Participating schools

I recruited participants from eight diverse mixed gender secondary schools across London. These schools varied in size (range 132 to 1397 pupils, M=777, SD=468), funding status (state versus independent/fee-paying), location (Central London versus Greater London), and proportion of students receiving free school meals (range 0% to 28%, M=8%, SD=12%). Class sizes in each school also varied greatly (from 2 to 31), so I created a variable indicating the size of the group in which each participant completed the study (referred to as testing group size). I did this in case the number of peers surrounding participants influenced their responses to any tasks or questionnaires.

2.9.2 Consent rates

See Figure 2.2 for a flow diagram of participant recruitment. Across the eight schools, parental informed consent was provided for 687 adolescents. Parental consent rates varied from 7% to 98% in each school (M=39%, SD=33%). In total, 606 adolescents with parental consent then provided informed assent to participate (88% of those with parental consent). In each school, the percentage of adolescents with parental consent who then provided informed assent ranged from 68% to 95% (M=88%, SD=10%).

Of the 606 participants who assented to participate, 7 (1%) participants were excluded from the final sample (final n=599). Reasons for exclusion of these participants were: did not fully

complete any tasks or questionnaires (n=4); did not have sufficient understanding of English to complete the study measures (n=1); or identified by researchers as misbehaving and not correctly completing study measures during classroom testing (n=2). The final sample (n=599) consisted of 33% of the eligible population (n=1829), and this ranged from 6% to 89% of the total adolescents eligible in each school (M=35%, SD=31%).

In my final sample, 141 (24%) adolescents were recruited from five schools with low parental consent rates (under 30%) and 458 (76%) adolescents were recruited from three schools with high parental consent rates (over 60%). Participants recruited from schools with low versus high consent did not differ in terms of age in years (mean diff=0.13, 95% CI=-0.01 to 0.52, p=0.06), gender ($\chi 2(1)=2.12$, p=0.15) or depressive symptoms (SMFQ score; mean diff=0.23, 95% CI=-0.85 to 1.31, p=0.68). However, participants from schools with low consent had higher non-verbal IQ score than participants from schools with high consent (mean diff=1.29, 95% CI=0.92 to 1.66, p<0.001).

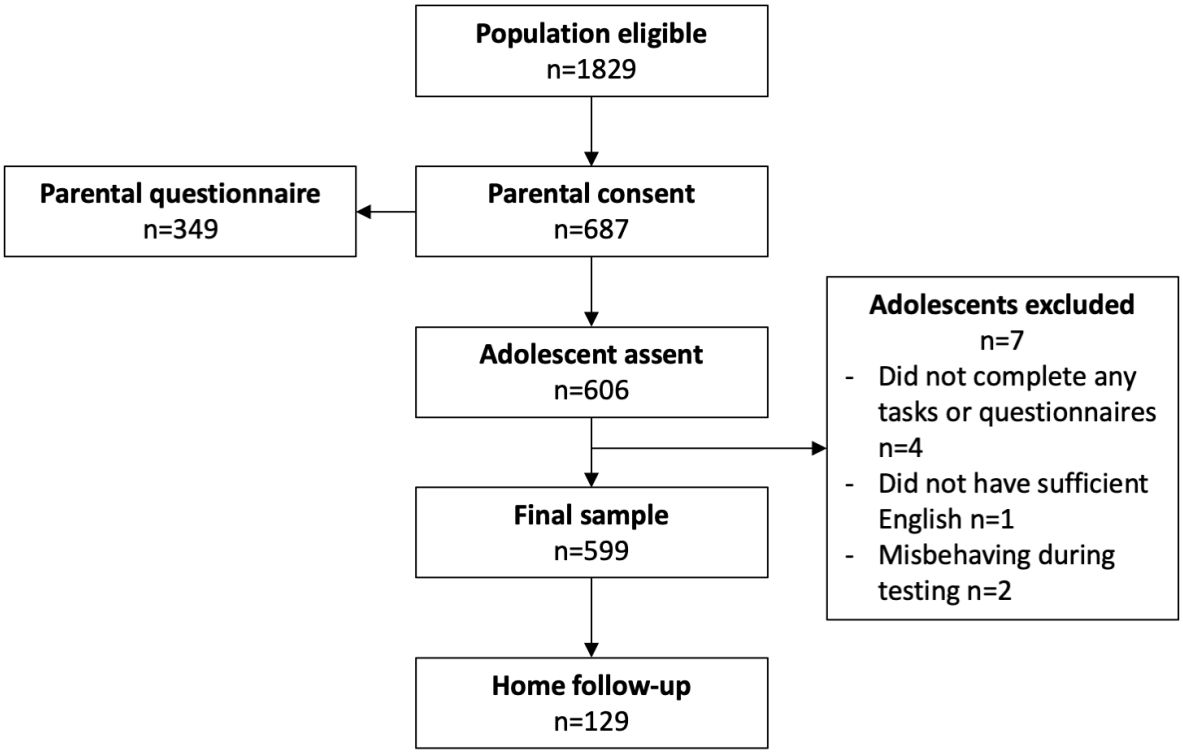

Figure 2.2 Flow diagram of participant recruitment from the eligible population of Years 7 and 9 or 10 in the eight participating schools.

2.9.3 Person-mean imputation

I replaced missing responses on self-report questionnaires using person-mean imputation where possible (Table 2.1). I used person-mean imputation for the SMFQ, DAS, RCADS, ARI-S, SRQ-A, and SDQ. I replaced missing responses for participants who responded to 70% or more of the questions, using each individual's mean score on that questionnaire. I was therefore able to calculate total scores on these questionnaires for participants who responded to 10 or more SMFQ questions (up to three missing), 12 or more DAS questions (up to five missing), four or more RCADS questions (up to one missing), and five or more ARI-S questions (up to one missing). Following guidance for the SDQ (Goodman et al., 1998), I calculated total scores for participants who completed 12 or more questions in the four subscales used to compute the overall score (emotional symptoms, conduct problems, hyperactivity/inattention, peer relationship problems; up to eight responses missing). As recommended for the SRQ-A (Foulkes et al., 2017), I replaced missing items by subscale, with scores imputed if participants responded to at least 50% of questions on that subscale. Thus, for subscales with four or five questions (admiration, negative social potency, and prosocial interactions), I calculated total scores for participants with three or more valid responses (up to two missing). For the other SRQ-A subscales (passivity and sociability), I calculated total scores for participants who responded to two or more questions (up to one response missing).

Table 2.1 Proportion of participants for whom missing data was replaced with person-mean imputation.

Measure	Complete	Imputed
	n (%)	
Depressive symptoms (SMFQ)	480 (80%)	115 (20%)
Irritability (ARI-S)	520 (88%)	73 (12%)
Generalised anxiety symptoms (RCADS)	536 (91%)	50 (9%)
Dysfunctional attitudes (DAS)	381 (64%)	187 (36%)
Social reward value (SRQ-A)	442 (86%)	74 (14%)
Emotional and behavioural problems (SDQ)	106 (88%)	15 (12%)

Note. SMFQ: Short Mood and Feelings Questionnaire. ARI-S: Affective Reactivity Index. RCADS: Revised Child Anxiety and Depression Scale. DAS: Dysfunctional Attitude Scale. SRQ-A: Social Reward Questionnaire (Adolescent Version). SDQ: Strengths and Difficulties Questionnaire.

Person-mean imputation has been criticised because summary scores are computed from different subsets of items for each participant, which can lead to problems if items are not similarly distributed (Lee, Bartholow, McCarthy, Pedersen, & Sher, 2015). If items with higher

means are more often missing, then summary scores for participants with more missing data will be based on items with lower means, and summary scores will be lower for participants with more missing data (Lee et al., 2015). This leads to a systematic downward bias in questionnaire summary scores. Additionally, by treating the imputed dataset as real values in subsequent analyses, this will attenuate standard errors, because it underestimates sampling error (Lee et al., 2015). These limitations can also introduce bias when testing associations between the questionnaire and other measures (Lee et al., 2015). Using multiple imputation avoids this issue as it involves adjusting standard errors for missing data.

However, person-mean imputation has been recommended when at least half of the questionnaire items are complete, as it performs well and is computationally simple and efficient (Bono, Ried, Kimberlin, & Vogel, 2007; Hawthorne & Elliott, 2005). When the percentage of items missing are low, person-mean imputation provides a good representation of the original data (Bono et al., 2007; Downey & King, 1998; Roth, Switzer, & Switzer, 1999). Additionally, not replacing missing items would have led to lots of missing data in my study, which can cause a number of other problems. I thus decided that using person-mean imputation for the amount of missing data in my study was the best solution, and I have performed a number of checks to ensure that this method was appropriate.

The main questionnaires of interest in my thesis were the SMFQ and DAS. On each questionnaire, the items with missing data varied across participants (i.e. there was not a single item with a high mean which was consistently missing). Data could have been missing completely at random (MCAR), missing at random (MAR), or missing not at random (MNAR). Missingness on the SMFQ and DAS was associated with a number of observed variables (e.g. age group, continuous age in years, non-verbal IQ score, and school), indicating they were MAR (or MNAR, which cannot be ruled out) and listwise deletion would not be appropriate.

Using person-mean imputation for the SMFQ and DAS did not substantially alter the distribution of summary scores. The mean and standard deviation of the SMFQ remained similar before (M=7.65, SD=5.78) and after (M=7.59, SD=5.69) imputation, as did the mean and standard deviation of the DAS (before M=51.21, SD=18.58; after M=50.90, SD=19.06). On both scales, the majority of participants had only one item missing (93 of 115 participants whose data were imputed in on the SMFQ and 117 of 187 on the DAS). I also tested the

association between DAS (exposure) and SMFQ (outcome) scores, using univariable linear regression, before and after including participants with missing items replaced. The evidence for the association between these measures did not change before including participants with missing items replaced (n=330, coef=0.18, 95% CI=0.16 to 0.21, p<0.001) compared to after (n=567, coef=0.19, 95% CI = 0.17 to 0.20, p<0.001). I thus deemed person-mean imputation suitable given its efficiency, the frequency of its use in previous research, the missingness mechanism in my data, the restrictions I imposed on the number of items allowed to be missing, and the relatively low proportion of participants with more than one item missing on each questionnaire.

I did not replace missing data for the PDS, OBVQ, HSRQ, CRSQ, and the parental questionnaire because of the nature of these questionnaires. They do not use Likert scales and questions were designed to measure more than one construct. For these measures, I only included participants with complete responses in analyses.

2.9.4 Other missing data

Not all participants were able to complete all measures as a result of time constraints on classroom testing. Also, very few participants completed the additional questionnaires at home (n=129, 22%), and response rates on the parental questionnaire were relatively poor (n=349, 58%). The completion rate for each measure in my study is shown in Table 2.2.

2.10 Key sample characteristics

My final sample consisted of 599 participants (48% female). The majority of participants were of white ethnicity (78%), had English as their first language (89%), and did not have a mental health problem (95%) or special educational needs (86%). Parental education was generally high (88%) and 44% of participants' parents reported a history of maternal depression anxiety or stress, alongside 34% reporting a history of paternal depression anxiety or stress (Table 2.3).

The sample consisted of two age groups, 331 (55%) young adolescents recruited from Year 7 and 268 (45%) mid-adolescents recruited from Years 9-10. Young adolescents' ages ranged from 11 to 13 years (M=11.56, SD=0.50) and mid-adolescents' ages ranged from 13 to 15 years (M=14.18, SD=0.51). I had expected young adolescents to be aged 11 to 12 years, but

there was one participant in Year 7 aged 13 years. Compared to young adolescents, mid-adolescents had higher non-verbal IQ scores and depressive symptoms and were more likely to have been diagnosed with a mental health problem, have used mental health services, and be receiving psychological therapy (Table 2.3).

Table 2.2 Number of complete responses on each measure, after person-mean imputation, by age group (with measures presented in order of completion).

Measure	**Young adolescents** n=331	**Mid-adolescents** n=268	**Overall** n=599
	n (%)		
Adolescent-reported (in class)			
Age (years)	329 (99%)	268 (100%)	597 (100%)
Gender	321 (97%)	267 (100%)	588 (98%)
Social evaluation learning task	330 (100%)	268 (100%)	598 (100%)
Non-verbal IQ score (RSPM)	331 (100%)	268 (100%)	599 (100%)
Recall task	319 (96%)	263 (98%)	582 (97%)
Depressive symptoms (SMFQ)	327 (99%)	268 (100%)	595 (99%)
Irritability (ARI-S)	326 (98%)	267 (100%)	593 (99%)
Generalised anxiety symptoms (RCADS)	324 (98%)	262 (98%)	586 (98%)
Dysfunctional attitudes (DAS)	304 (92%)	264 (99%)	568 (95%)
Health and social risk concerns (HSRQ)	258 (78%)	243 (91%)	501 (84%)
Social reward value (SRQ-A)	267 (81%)	248 (93%)	515 (86%)
Rejection sensitivity (CRSQ)	147 (44%)	177 (66%)	324 (54%)
Adolescent-reported (home follow-up)			
Bullying and victimisation (OBVQ)	63 (19%)	65 (24%)	128 (21%)
Pubertal stage (PDS)	58 (18%)	61 (23%)	119 (20%)
Emotional and behavioural problems (SDQ)	56 (17%)	65 (24%)	121 (20%)
Parent/carer reported			
Ethnicity	195 (59%)	151 (56%)	346 (58%)
English as first language	192 (58%)	150 (56%)	342 (57%)
Mental health problem	195 (59%)	151 (56%)	346 (58%)
Used mental health services	195 (59%)	151 (56%)	346 (58%)
Taking antidepressants	195 (59%)	152 (57%)	347 (58%)
Receiving psychological therapy	195 (59%)	152 (57%)	347 (58%)
Special educational needs and disabilities	194 (59%)	150 (56%)	344 (58%)
Parental education	186 (56%)	141 (53%)	327 (55%)
Maternal depression anxiety or stress	183 (55%)	145 (54%)	328 (55%)
Paternal depression anxiety or stress	167 (50%)	125 (47%)	292 (49%)

Note. RSPM: Raven's Standard Progressive Matrices. SMFQ: Short Mood and Feelings Questionnaire. ARI-S: Affective Reactivity Index. RCADS: Revised Child Anxiety and Depression Scale. DAS: Dysfunctional Attitude Scale. HSRQ: Health and Social Risks Questionnaire. SRQ-A: Social Reward Questionnaire (Adolescent Version). CRSQ: Children's Rejection Sensitivity Questionnaire. OBVQ: Olweus Bully/Victim Questionnaire (Revised). PDS: Pubertal Development Scale. SDQ: Strengths and Difficulties Questionnaire.

Table 2.3 Key demographic and clinical characteristics according to age group.

Measure	Young adolescents n=331	Mid-adolescents n=268	Overall n=599
	Mean (SD)		
Age (years)	11.56 (0.50)	14.18 (0.51)	12.74 (1.40)
Non-verbal IQ score (RSPM)	4.04 (1.99)	4.88 (2.02)	4.42 (2.04)
Depressive symptoms (SMFQ)	7.04 (5.48)	8.25 (5.88)	7.59 (5.69)
Generalised anxiety symptoms (RCADS)	6.28 (3.86)	6.81 (3.66)	4.42 (2.04)
	N (%)		
Gender			
Male	169 (53%)	129 (48%)	298 (51%)
Female	152 (47%)	138 (52%)	290 (49%)
Ethnicity			
White	155 (79%)	116 (77%)	271 (78%)
Mixed	9 (5%)	17 (11%)	26 (8%)
Asian/Asian British	5 (3%)	2 (1%)	7 (2%)
Black/Black British	7 (4%)	3 (2%)	10 (3%)
Other	19 (10%)	13 (9%)	32 (9%)
English as first language	173 (90%)	132 (88%)	305 (89%)
Mental health problem	6 (3%)	13 (9%)	19 (5%)
Used mental health services	12 (6%)	16 (11%)	28 (8%)
Receiving psychological therapy	0	5 (3%)	5 (1%)
Special educational needs and disabilities	24 (12%)	23 (15%)	47 (14%)
High parental education	165 (89%)	124 (88%)	289 (88%)
Maternal depression anxiety or stress	81 (44%)	64 (44%)	145 (44%)
Paternal depression anxiety or stress	56 (34%)	42 (34%)	98 (34%)
Pubertal stage			
Early	49 (85%)	13 (21%)	62 (52%)
Late	9 (16%)	48 (79%)	57 (48%)

Note. RSPM: Raven's Standard Progressive Matrices. SMFQ: Short Mood and Feelings Questionnaire. RCADS: Revised Child Anxiety and Depression Scale. See Table 2.2 for missing data on each measure.

Throughout my thesis, my primary outcome is depressive symptoms, measured using the SMFQ. In young adolescents, SMFQ score ranged from 0 to 23 (M=7.04, SD=5.48). SMFQ score was positively skewed (skewness=1.00) with a slightly heavier tail than a normal distribution (kurtosis=3.29). Depressive symptoms were slightly higher in female (M=7.80, SD=5.85) than male (M=6.40, SD=5.09) young adolescents. The SMFQ threshold for depression was met by 59 (18%; 58% of whom were female) young adolescents. In mid-adolescents, SMFQ score ranged from 0 to 26 (M=8.25, SD=5.88). SMFQ score in mid-adolescents was also positively skewed (skewness=0.88) with a slightly heavier tail than a normal distribution (kurtosis=3.37). Depressive symptoms were higher in females (M=9.70, SD=6.30) than males (M=6.65, SD=4.94). The SMFQ threshold for depression was met by 61 (23%; 70% of whom were

female) mid-adolescents. See Figure 2.3 for a plot of the mean SMFQ according to gender and age group. The prevalence of depressive symptoms in my sample was similar to the prevalence of depressive symptoms in adolescents aged 14 in the Millennium Cohort Study (Patalay & Gage, 2019) and adolescents aged 11 to 15 in ALSPAC (Kwong et al., 2019).

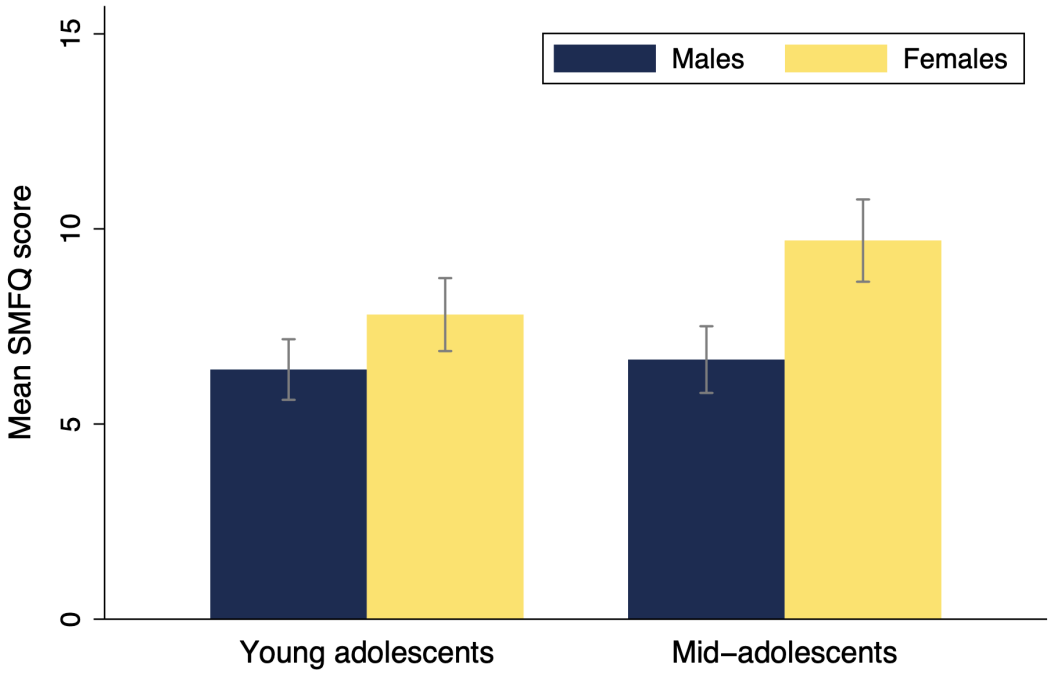

Figure 2.3 Mean depressive symptoms according to gender within each age group in my sample. SMFQ: short Mood and Feelings Questionnaire. 95% confidence intervals plotted.

2.11 Statistical analyses

I used this data throughout my thesis. I have addressed a different, but related, aspect of my research question in each chapter, focussing on the associations between gender, age group, depressive symptoms, and: learning about social evaluation (chapter 3); the processes underlying learning about social evaluation (chapter 4); recall of social evaluation (chapter 5); and dysfunctional attitudes (chapter 6). In every chapter, analyses are explained in detail and sample size is reported for that specific study. I collected data on several questionnaires (ARI-S, RCADS, HSRQ, SRQ-A, CRSQ, OBVQ, and SDQ) for use in other research, so have not analysed responses on these questionnaires in any chapters within my thesis.

2.11.1 Confounders

Throughout my thesis, data were hierarchical because participants were clustered within schools. However, given that there were only eight schools, I have adjusted for school as a

fixed effect in standard (not multilevel) regression models. Using this approach has been shown to perform best in data with few clusters and a moderate number of exposures (McNeish & Stapleton, 2016). Other potential confounders included age (measured continuously in years), testing group size, and non-verbal IQ score (measured using the RSPM; section 2.4.9). In each chapter, continuous age was mean centred within each age group in order to include both age group and continuous age in the same models (by reducing the correlation between them).

IQ is an important potential confounder of associations between negative cognitions and depressive symptoms. However, IQ may also be on the causal pathway between gender and negative cognitions. Girls may have higher IQ on average than boys in adolescence (Bilker et al., 2012; Deary, Strand, Smith, & Fernandes, 2007; Deary, Thorpe, Wilson, Starr, & Whalley, 2003; Strand, Deary, & Smith, 2006), and IQ could cause differences in negative cognitions. Additionally, IQ may also be on the causal pathway between age group and negative cognitions, because non-verbal IQ score increases with age during adolescence (Chierchiaa et al., 2019; Crone et al., 2009; Richland, Morrison, & Holyoak, 2006), and again IQ could cause differences in negative cognitions. However, given the importance of IQ as a potential confounder of associations between negative cognitions and depressive symptoms (Glaser et al., 2011; van Os, Jones, Lewis, Wadsworth, & Murray, 1997; Zammit et al., 2004), I decided to include it in all adjusted analyses. I tested the associations between IQ score and exposures and outcomes and compared models before and after adjusting for IQ score. This is discussed where relevant in each chapter and the general discussion (section 7.3.3).

As there was a large proportion of missing data on home follow-up and parent/carer reported measures (Table 2.2), I did not adjust for these variables in my main analyses. Where possible, I performed sensitivity analyses adjusting for these measures. Again, limitations of this approach are outlined in each chapter and in the general discussion.

2.11.2 Terminology: exposures and outcomes

In my thesis, I have combined approaches from epidemiology and cognitive neuroscience. Different terminology is used to refer to variables across these fields, so I have chosen consistent terms to use throughout my thesis. I will refer to exposures (to mean predictors or independent variables) and outcomes (to mean dependent variables).

2.11.3 Standardised estimates

In chapters 3 to 5 of my thesis, I have analysed performance on social information processing tasks. I have not transformed or standardised any variables or estimates. To facilitate interpretation and comparison of these findings, I have included standardised estimates for these analyses in Appendix 1. Chapter 6 of my thesis only presents standardised analyses.

2.12 Strengths and limitations of my data

There are a number of strengths and limitations of my data, which are discussed in detail in chapter 7 (general discussion). However, it is important that these factors are considered when interpreting the findings from each study within my thesis. I will therefore outline some key relevant limitations within the discussion section of each study (chapters 3-6). These discussions are not intended to be exhaustive, and more in-depth critical appraisal will follow in the final chapter of my thesis (chapter 7).

2.13 Conclusion

In this chapter, I have provided an overview of the methods used to collect data for my thesis. In the following chapters (chapters 3-6), I will present and discuss the findings of four studies using this data. I will then provide an overview of my findings and discuss the general implications of the work in my thesis (chapter 7).

Chapter 3 Learning about social evaluation during adolescence: gender differences and associations with depressive symptoms

3.1 Introduction

When interacting with other people, we make automatic judgements about them. For successful social interactions, we also need to understand what others believe about us. These judgements about a person's character, worth, and status are types of social evaluation. Learning about social evaluation may be particularly important during adolescence. Adolescents spend increasing amounts of time with their peers (Lam et al., 2014) and gaining the approval of peers becomes particularly salient (Steinberg & Silverberg, 1986). A key challenge in adolescence is to understand social evaluation, incorporate it into the developing self-concept (as outlined in section 1.9.2), and adjust behaviour to navigate changing social environments (Nelson et al., 2005). This becomes especially important as the perceived beliefs of peers strongly influence adolescents' appraisal of their own social and personal worth (O'Brien & Bierman, 1988).

Learning about social evaluation may be difficult, and subject to biases, because social interactions are often ambiguous. Individuals have to build an understanding of what others believe, but this may well be influenced by what they themselves believe. Healthy adults have an optimism bias, making assumptions that others have positive views of them and that they are liked, which may be protective (Button et al., 2012, 2015; Roiser & Sahakian, 2017; Sharot, 2011). Automatically discounting negative information in social interactions may help individuals to perceive their interactions as more positive, allowing them to maintain positive views of the self and to be more self-confident. It also means less negative information is available for later rumination, leading to better outcomes and more future social success. According to cognitive models, these positive biases are reduced in depression. Individuals with more depressive symptoms may make more realistic, or even negative, interpretations of their social interactions (Beck & Bredemeier, 2016; LeMoult & Gotlib, 2019; Moore & Fresco, 2012; Roiser et al., 2012; Roiser & Sahakian, 2017; Silk, Davis, McMakin, Dahl, & Forbes, 2012).

Using a social evaluation learning task which simulates social interactions and measures responses when learning whether individuals are liked or disliked, there is evidence that healthy adults are better at learning they are liked relative to disliked (Button et al., 2012). This positive bias is specific to social evaluation about the self and does not generalise to learning about another person (Button et al., 2016, 2015). As proposed in cognitive models, this positive self-referential bias decreased with increasing social anxiety and depressive symptoms (Button et al., 2012, 2015; Hobbs et al., 2018). Individuals with more severe depression were worse at learning that they were liked by a computer character (Hobbs et al., 2018). This bias may contribute to the development or maintenance of depressive symptoms.

It is unclear whether adolescents demonstrate the same positive biases in learning about social evaluation as adults. The ability to use social information to infer what others believe may still be developing in early adolescence. Adolescents' capacity to represent abstract social goals and the mental states of others improves during adolescence (Parker et al., 2006). Development of the prefrontal cortex may make it possible to pursue the more complex and distal rewards gained from peer relationships (Davey et al., 2008). Additionally, self-evaluations become more negative in early adolescence, and mid-adolescents are more negatively influenced by comparing themselves to peers than children or late adolescents (van der Aar et al. 2018). Adolescents may react more negatively to peer rejection and evaluation than adults (Sebastian, Viding, et al., 2010; Silk et al., 2014; Somerville, 2013). Coupled with the continuing development of the self-concept during adolescence (Sebastian et al., 2008), this may mean that adolescents do not demonstrate robust positive biases in learning during social interactions but are more vulnerable to the negative evaluations of peers.

In order to measure learning about social evaluation in adolescence, several computerised tasks have been developed. For example, in the Chatroom Task (Guyer et al., 2008), adolescents are told that they are meeting peers online to chat. They view photos of peers, judge how interested the peer would be in interacting with them, and then receive rigged acceptance and rejection feedback supposedly from these peers. Similarly, in the Social Judgment Task (Somerville, Heatherton, & Kelley, 2006), participants view photos of peers whom they are told have previously judged their photo. Participants rate whether they think

these peers liked or disliked them, and then receive rigged feedback supposedly from these peers. However, studies generally measure adolescents' predictions about social evaluation before interactions with peers, rather than assessing how adolescents learn about social evaluation during interactions. This is an important distinction, because automatic processing of social evaluation may involve different cognitive mechanisms to more effortful conscious opinions about evaluation (Kahneman, 2011; Roiser et al., 2012). Dual process models distinguish the automatic processes involved in learning from a more reflective process, which is involved in anticipation and post-event rumination (implicit versus explicit cognitions; e.g. Strack & Deutsch, 2004). In adults, there is evidence for a distinction between these processes on a social evaluation learning task. Although healthy adults showed a positive self-referential bias during implicit learning, there was no evidence for this bias when asked to explicitly reflect on what the character thought about them after learning (Button et al., 2015). Studies thus need to test automatic processing of social evaluation as well as conscious judgments of this evaluation. However, this social evaluation learning task has never been used with adolescents.

There is some evidence that older adolescents (aged 18-25) have positive biases in predicting social evaluation (Caouette & Guyer, 2016; Guyer, Benson, et al., 2014; Somerville, Kelley, & Heatherton, 2010; Van der Molen et al., 2014). Young adolescents, in contrast, may make fewer predictions that they will be liked than older adolescents and young adults, demonstrating a smaller positive self-referential bias (Gunther Moor, van Leijenhorst, Rombouts, Crone, & van der Molen, 2010; Rodman, Powers, & Somerville, 2017). However, these studies have all been small (largest n=107) and have recruited samples of volunteers, who are unlikely to be representative of the general population. To my knowledge, only one study has tested associations between predictions about social evaluation and depressive symptoms. This small study (n=60) found evidence that undergraduates (aged 18-26 years) with more severe depressive symptoms were more likely to state that peers would not be interested in chatting with them (Caouette & Guyer, 2016). Other small studies (n=28 to 42) have found evidence that late adolescents with lower self-esteem expected that peers would not like them more often than those with higher self-esteem (Guyer et al., 2008; Somerville et al., 2010).

Overall, research to date suggests that less positive predictions about social evaluation may be associated with depressive symptoms during late adolescence. However, there is no strong evidence for whether adolescents have implicit biases in learning about social evaluation, and whether these biases are associated with depressive symptoms. Additionally, very few studies have tested whether there are gender differences in expectations about social evaluation. Those which have were small and generally found no evidence that expectations of, or reactions to, evaluation differed between boys and girls during adolescence (Guyer, Caouette, Lee, & Ruiz, 2014; Guyer, Choate, Pine, & Nelson, 2012; Guyer et al., 2009). However, as proposed in section 1.6, if biases in learning about social evaluation are associated with depressive symptoms, these biases may be more prevalent in girls (Bone et al., 2020).

In this study, I aimed to test learning about social evaluation in a large (n=598) cross-sectional study. Adolescents were recruited from two age groups (young and mid-adolescents aged 11-13 and 13-15 years) to study social evaluation learning before and after the gender difference in depression emerges. Depressive symptoms ranged from mild to severe. In a social evaluation learning task, participants learnt whether a person was liked or disliked by a computer character. Participants chose positive or negative personality traits and received probabilistic feedback on whether their choice reflected what a computer character thought of them (self-referential) or another person (other-referential). After interacting with this computer character, adolescents were asked to explicitly reflect on and report what the character thought of them. I tested associations between learning during the task, reflective ratings after completing the task, age group, gender, and depressive symptom severity. My main aim was to investigate learning about social evaluation, examine whether there are gender differences in this learning, and explore whether these gender differences change with age. I also aimed to test whether learning about social evaluation was associated with depressive symptoms.

To investigate my aims, I tested hypotheses relating to task performance, gender differences, and associations with depressive symptoms. I hypothesised that, as in adults, adolescents would demonstrate a positive self-referential bias (hypothesis 1.1). This would be reflected in choosing the positive personality trait more often when learning about the self than other people, demonstrating poorer learning that someone disliked the self than another person. It would also be evident in rating the character's opinion of the self as more positive than

their opinion of the other after learning. I hypothesised that this positive self-referential bias would be smaller in girls than boys (hypothesis 1.2). I also hypothesised that this gender difference would be present from early adolescence, so the influence of gender would not differ across age groups (hypothesis 1.3). I hypothesised that this positive self-referential bias would be negatively associated with depressive symptoms (hypothesis 1.4). Finally, I hypothesised that the association between the positive self-referential bias and depressive symptoms would not differ across genders or age groups (hypothesis 1.5).

3.2 Methods

3.2.1 Participants

Complete data on the social evaluation learning task was missing for 1 participant (final n=598). See section 2.9.4 for an overview of all missing data in this study. As described in section 2.1, my power calculation indicated that a sample of 320 adolescents was needed in each age group to show a difference of 0.4 standard deviations in task outcomes between boys and girls. My sample consisted of 330 young adolescents and 268 mid-adolescents.

3.2.2 Measures

3.2.2.1 Social evaluation learning task

The social evaluation learning task was a two-alternative forced choice task based on probabilistic stimulus-reward learning tasks (adapted from Button et al., 2015). For full details of the task, see section 2.3.1.

Briefly, participants learnt whether a person was liked or disliked by a computer character. Learning occurred in two conditions: about the participant themselves (self-referential) or about another person (Taylor; other-referential). There were thus four blocks in this task: self like, self dislike, other like, and other dislike. Learning occurred over 20 trials in each block. The block ended with a global rating, in which participants rated how much they thought the character liked them (self-referential) or Taylor (other-referential) on a rating scale (0% = dislike, 100% = like). This global rating required participants to reflect on their learning in the previous 20 trials.

As described in section 2.3.1, I recorded the number of positive responses during learning in each block, and the global rating after each block, for use in analyses. I will also analyse choices (positive versus negative word selected) and feedback on each trial in reinforcement learning models in chapter 4.

3.2.2.2 Depressive symptoms

The short Mood and Feelings Questionnaire (SMFQ; Angold et al., 1995) measured depressive symptoms over the last two weeks. I replaced missing responses using person-mean imputation (see section 2.9.3) for those who responded to 10 or more questions using each individual's mean SMFQ score (116 participants, 19% of this sample).

3.2.2.3 Confounders

Participants completed an abbreviated nine-item version of the Raven Standard Progressive Matrices Test (non-verbal IQ score; Bilker et al., 2012). I measured additional potential confounders (ethnicity, English as a first language, dyslexia, autism spectrum disorders, parental education, maternal depression, paternal depression) with my parental questionnaire.

I intended to include pubertal stage as a potential confounder because it is strongly associated with depressive symptoms (Angold et al., 1998). Pubertal hormones and age may have functionally dissociable effects on neural activity during social information processing (Goddings, Burnett Heyes, Bird, Viner, & Blakemore, 2012). There is also evidence that puberty is more strongly associated with performance on a self-referential encoding task than age (Ke, Wu, Willner, Brown, & Crowley, 2018). Following classroom data collection, I sent participants the Pubertal Development Scale (PDS; Petersen et al., 1988) to complete at home. Participants were divided into two groups: early/mid puberty (equivalent to Tanner stages 1-3) and late/post puberty (equivalence to Tanner stages 4-5; see section 2.4.10 for further details).

3.2.3 Procedure

After providing informed assent, participants completed the social evaluation learning task, followed by the RSPM Test, and the SMFQ. After classroom data collection, I sent participants a link to complete the PDS. See chapter 2 for a more detailed description of study methods.

3.2.4 Statistical analyses

I performed analyses using Stata 16 (StataCorp, 2019). As my sample consisted of two age groups, and one of my aims was to investigate the influence of gender in each group, I have presented all descriptive statistics separately according to gender for each age group. I then used linear regression to test the association between age group and gender (binary exposures), and SMFQ score (continuous outcome). I also included an interaction between gender and age group in this model to test whether the gender difference in depressive symptoms increased with age.

3.2.4.1 Positive responses

Positive responses were made in four blocks: *self like*, *self dislike*, *other like*, and *other dislike*. If participants learnt these rules, I would expect positive responses to be higher in *like* than *dislike* blocks. Perfect performance would consist of 20 positive responses in *like* blocks and zero positive responses in *dislike* blocks. However, as feedback contingency was set at 80%, performance may be expected to peak at 16 positive responses in *like* blocks and four positive responses in *dislike* blocks.

Given that the four categories of hits were clustered within each individual, I used linear multilevel models. Total number of positive responses was the outcome, and I included random intercepts for participant to account for this clustering. I estimated the task conditions (self/other, like/dislike), demographic variables of interest (age group, gender) and potential confounders (continuous age within each age group, school, testing group size, and non-verbal IQ score) as fixed effects. All models are presented before and after adjustment for confounders.

I first tested whether positive responses differed according to condition (self/other) and rule (like/dislike). I included condition and rule as exposures with positive responses as the outcome. I then added an interaction between condition and rule, to test whether the association between rule and positive responses differed in self-referential versus other-referential blocks (hypothesis 1.1).

Next, I examined whether positive responses differed according gender (hypothesis 1.2). I first tested gender as an exposure with positive responses as the outcome, as well as testing

a three-way interaction between gender, condition and rule with positive responses as the outcome. I also report the two-way interactions between these variables.

To assess whether gender differences were consistent across age groups (hypothesis 1.3), I tested a four-way interaction between age group, gender, condition and rule with positive responses as the outcome. I also report the two-way and three-way interactions between these factors. As my aim was to compare the influence of gender in each age group, I only report interactions which include age group and gender. Where there was evidence of an interaction, I examined associations with positive responses separately for each subgroup.

I then tested whether positive responses were associated with depressive symptoms (hypothesis 1.4). I used linear regression to test whether positive responses in each block (four separate exposures) were associated with depressive symptoms (SMFQ score; continuous outcome). For this analysis, I included responses in all blocks in a single model to adjust for overall performance. I then adjusted this model for age group, gender, and potential confounders.

Finally, for each type of positive responses, I tested whether the association with depressive symptoms differed according to age group and gender (hypothesis 1.5). To do this I added a three-way interaction between positive responses in each condition, age group, and gender to the linear regression model with depressive symptoms as the outcome.

3.2.4.2 Global ratings

Global ratings were made on a scale from 0 to 100, whereby 0 represented the person being disliked by the computer character and 100 meant the person was liked by the computer character. Participants made one rating at the end of each block, resulting in four global ratings: self like, self dislike, other like, and other dislike. If participants learnt the rules in each block, I would expect global ratings to be close to 0 in dislike blocks and 100 in like blocks. Each 1-point increase in global rating represented a 1% increase in the participant believing that they were liked. I repeated all analyses performed with positive responses for global ratings (hypotheses 1.1-1.4), investigating whether biases during learning were also evident in subsequent explicit ratings of social evaluation.

3.2.4.3 Sensitivity analyses: additional confounders

I asked all parents/carers to complete the parental questionnaire, but response rates were low (n=348, 58%). I intended to repeat analyses controlling for additional potential confounders measured via this parental questionnaire (ethnicity, English as a first language, dyslexia, autism spectrum disorders (ASD), parental education, maternal depression, paternal depression). I first explored the distribution of these variables by age group and tested whether they were associated with task performance. I then repeated primary analyses for the subsample whose parents/carers completed the questionnaire. Results are presented before and after controlling for the additional confounders available in this subsample. I used linear multilevel models testing the associations between condition, rule, age group, and gender (exposures) and positive responses or global ratings (outcomes, tested in separate models). I also repeated analyses using linear regression models to test associations between positive responses and global ratings (exposures, tested in separate models) and depressive symptoms (outcome).

3.2.4.4 Sensitivity analyses: pubertal stage

I asked all participants to complete the PDS, but response rates were also low (n=119, 20% of total sample). I intended to include pubertal stage as a confounder, so first explored whether pubertal stage was associated with task performance. If there was evidence that it may be a confounder in this subsample, I planned to repeat all primary analyses adjusted for pubertal stage.

Standardised estimates for all of the above analyses are included in Appendix 1.

3.3 Results

The sample consisted of 598 adolescents (49% female). Of these, 330 (55%) were young adolescents from Year 7, and 268 (45%) were mid-adolescents from Years 9-10. Young adolescents' ages ranged from 11 to 13 years (M=11.56, SD=0.50) and mid-adolescents' ages ranged from 13 to 15 years (M=14.18, SD=0.51). Table 3.1 shows sample characteristics and social evaluation learning task performance according to age group and gender.

In young adolescents, SMFQ score ranged from 0 to 23 (M=7.05, SD=5.48). The SMFQ threshold for depression was met by 61 (19%) young adolescents. In mid-adolescents, SMFQ

score ranged from 0 to 26 (M=8.25, SD=5.88). The SMFQ threshold for depression was met by 62 (23%) mid-adolescents.

There was evidence that depressive symptoms were higher in mid- than young adolescents (coef=1.19, 95% CI=0.28 to 2.11, p=0.01), and higher in females than males (coef=2.19, 95% CI=1.28 to 3.10, p<0.001). There was no evidence of an interaction between age group and gender on depressive symptoms (interaction p=0.07). Although the evidence for this interaction narrowly missed statistical significance, I conducted the planned linear contrasts because of my a priori hypotheses. As predicted, depressive symptoms were higher in females in both age groups, but the gender difference was numerically larger in the older group (young adolescents coef=1.39, 95% CI=0.17 to 2.60, p=0.03; mid-adolescents coef=3.05, 95% CI=1.68 to 4.42, p<0.001).

Table 3.1 Demographic characteristics and task performance of adolescents who completed all blocks of the social evaluation learning task.

	Young adolescents		**Mid-adolescents**		**Overall**	
	Male (n=168)	**Female** (n=152)	**Male** (n=129)	**Female** (n=138)	**Skewness** (n=598)	**Kurtosis** (n=598)
	Mean (SD)				Statistic	
Age (years)	11.57 (0.50)	11.55 (0.51)	14.19 (0.47)	14.16 (0.56)	0.17	1.55
Non-verbal IQ score	3.82 (2.05)	4.41 (1.87)	4.81 (2.02)	4.97 (2.03)	-0.07	2.20
Depressive symptoms	6.42 (5.10)	7.80 (5.85)	6.65 (4.94)	9.70 (6.30)	0.95	3.36
Positive responses						
Self like	14.73 (4.23)	15.95 (3.29)	15.74 (3.52)	15.47 (3.38)	-0.98	3.75
Self dislike	7.05 (4.32)	6.58 (4.22)	6.23 (3.64)	5.98 (4.50)	0.60	2.72
Other like	14.93 (3.95)	14.82 (3.83)	15.54 (3.40)	15.57 (3.72)	-0.77	2.92
Other dislike	5.72 (3.69)	5.63 (3.70)	5.20 (3.80)	5.22 (3.77)	0.78	3.22
Global ratings						
Self like	64.19 (23.57)	64.70 (21.79)	68.50 (20.35)	66.41 (20.83)	-0.80	3.75
Self dislike	28.92 (24.72)	25.76 (21.97)	26.84 (19.28)	24.72 (18.46)	1.11	4.39
Other like	65.75 (24.25)	66.46 (20.61)	67.57 (19.38)	68.12 (19.75)	-0.72	3.54
Other dislike	27.66 (23.00)	27.12 (18.01)	27.02 (19.69)	26.01 (17.68)	1.18	4.93

Note. Positive responses could range from 0 to 20 in each condition, with 20 positive responses representing perfect performance in like blocks and 0 positive responses indicating perfect performance in dislike blocks. Global ratings ranged from 0 to 100, where 0 means dislike and 100 means like. Gender was missing for 10 young adolescents and 1 mid-adolescent. Age (years) was missing for 1 young adolescent male. Depressive symptoms (SMFQ score) was missing for 4 young adolescents (3 males, 1 female).

3.3.1 Positive responses

There was evidence that the number of positive responses differed according to rule (like/dislike). There were 9.36 (95% CI=-9.67 to -9.05, p<0.001 adjusted for confounders)

fewer positive responses in dislike compared to like rule blocks, demonstrating that participants acquired the rule contingencies as expected.

Hypothesis 1.1: adolescents would demonstrate a positive self-referential bias

There was evidence of a positive self-referential bias. Participants made 0.64 (95% CI=0.33 to 0.95, p<0.001 adjusted for confounders) more positive responses in self-referential than other-referential blocks. There was also evidence for an interaction between condition and rule on positive responses (adjusted interaction p=0.02; Table 3.2). There was a smaller difference in the number of positive responses made in self-referential dislike compared to like blocks (coef=-8.97, 95% CI=-9.42 to -8.52 adjusted for confounders) than in the other-referential condition (coef=-9.74, 95% CI=-10.17 to -9.32 adjusted for confounders).

Table 3.2 Linear multilevel models testing the associations between age group, gender, condition (self-referential/other-referential), rule (like/dislike; exposures), and positive responses or global ratings (continuous outcomes, tested in separate models).

	Unadjusted models (n=587)		**Adjusted models** (n=586)	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Outcome: Positive responses				
Condition	0.64 (0.33 to 0.95)	<0.001	0.64 (0.33 to 0.95)	<0.001
Rule	-9.33 (-9.65 to -9.02)	<0.001	-9.36 (-9.67 to -9.05)	<0.001
Age group	-0.05 (-0.37 to 0.26)	0.73	-0.15 (-0.51 to 0.22)	0.43
Gender	0.02 (-0.29 to 0.33)	0.90	0.01 (-0.30 to 0.33)	0.94
Outcome: Global ratings				
Condition	-0.72 (-2.34 to 0.90)	0.38	-0.72 (-2.34 to 0.90)	0.38
Rule	-39.52 (-41.13 to -37.90)	<0.001	-39.50 (-41.12 to -37.88)	<0.001
Age group	0.58 (-1.38 to 2.54)	0.56	-0.03 (-2.28 to 2.23)	0.98
Gender	-0.87 (-2.82 to 1.08)	0.38	-1.01 (-2.98 to 0.97)	0.32

Note. Separate models were tested for each outcome (positive responses/global ratings). Adjusted models also adjusted for continuous age within each age group, school, testing group size, and non-verbal IQ score. Positive responses ranged from 0 to 20. For global ratings, 0 represented dislike and 100 represented like.

Hypothesis 1.2: the positive self-referential bias would be smaller in girls than boys

There was no evidence for an association between gender and total positive responses (p=0.94 adjusted for confounders; Table 3.2; Figure 3.1). There was also no evidence for interactions between gender, condition, and rule on positive responses (Table 3.3).

Hypothesis 1.3: the gender difference in positive self-referential bias would not differ across age groups

I then tested whether the association between gender and positive responses differed according to age group. There was no evidence for a two-way interaction between gender and age group on positive responses (interaction p=0.39 adjusted for confounders; Table 3.3). There was also no evidence for an association between age group and positive responses (p=0.43 adjusted for confounders; Table 3.2), or interactions between age group, gender, condition, and rule on positive responses (Table 3.3; Figure 3.1).

Table 3.3 Interaction term p values from linear multilevel models testing the interactions between age group, gender, condition (self-referential/other-referential) and rule (like/dislike) on positive responses or global ratings (continuous outcomes).

	Unadjusted models (n=587)	**Adjusted models** (n=586)
Outcome: Positive responses		
Condition x rule	0.01	0.02
Gender x condition	0.71	0.72
Gender x rule	0.12	0.16
Gender x condition x rule	0.15	0.15
Age group x gender	0.42	0.39
Age group x gender x condition	0.23	0.23
Age group x gender x rule	0.19	0.24
Age group x gender x condition x rule	0.17	0.17
Outcome: Global ratings		
Condition x rule	0.64	0.64
Gender x condition	0.33	0.33
Gender x rule	0.26	0.25
Gender x condition x rule	0.83	0.83
Age group x gender	0.79	0.98
Age group x gender x condition	0.89	0.89
Age group x gender x rule	0.61	0.60
Age group x gender x condition x rule	0.55	0.55

Note. Each interaction was tested in a separate model. All models included condition, rule, age group, and gender. Adjusted models also adjusted for continuous age within each age group, school, testing group size, and non-verbal IQ score.

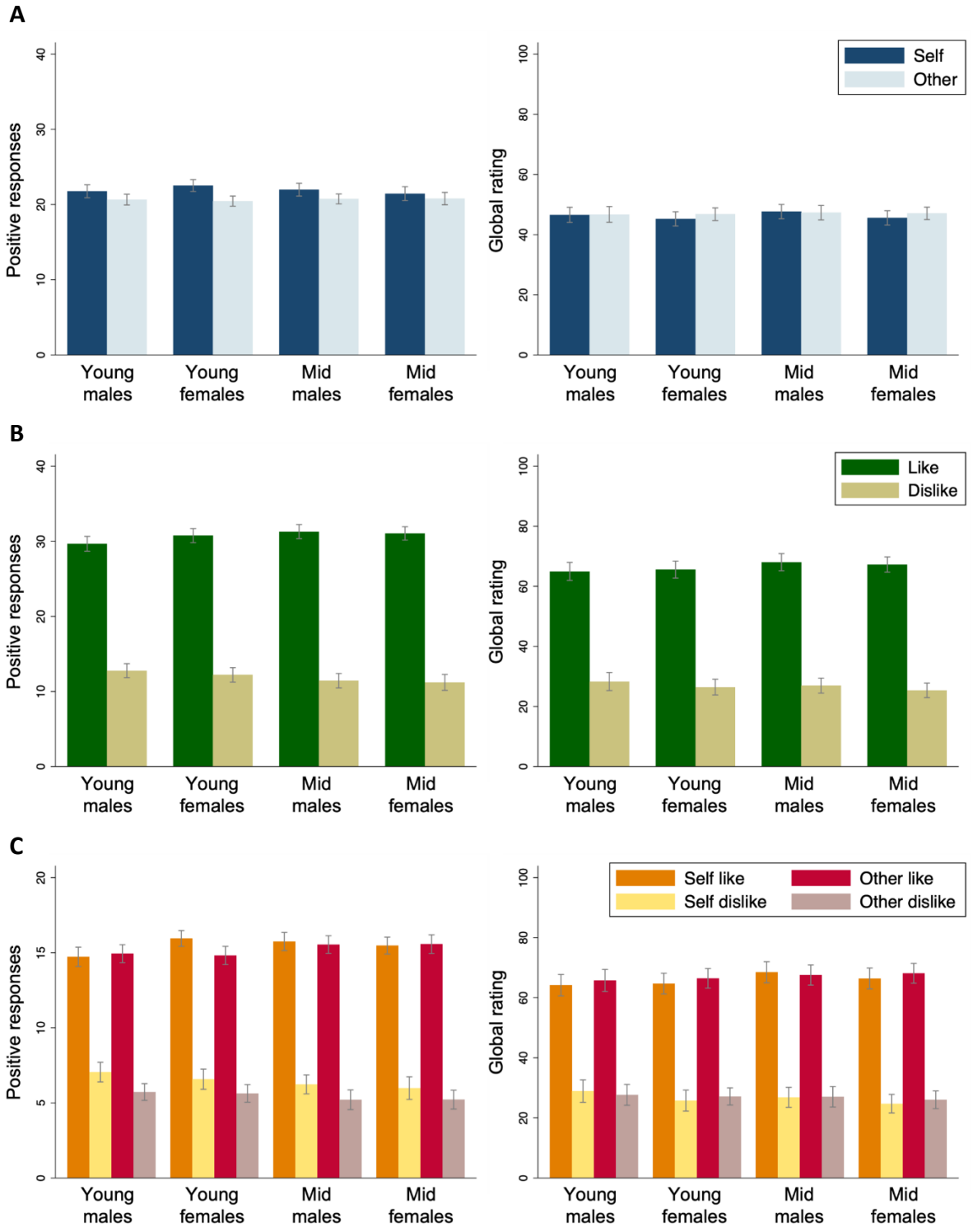

Figure 3.1 A) Three-way interaction between age group, gender, and condition (self/other) on positive responses (left) and global ratings (right), responses collapsed across like/dislike rule blocks. B) Three-way interaction between age group, gender, and rule (like/dislike) on positive responses (left) and global ratings (right), responses collapsed across self-/other-referential blocks. C) Four-way interaction between age group, gender, condition and rule on positive responses (left) and global ratings (right). All plotted using raw data with 95% confidence intervals. Young: young adolescents. Mid: mid-adolescents.

Hypothesis 1.4: the positive self-referential bias would be negatively associated with depressive symptoms

Next, I tested whether positive responses within each condition were associated with depressive symptoms. There was strong evidence that the number of positive responses in self-referential blocks was negatively associated with depressive symptoms (Table 3.4; Figure 3.2). In the self-referential like block, for each additional positive word chosen, SMFQ score was 0.24 points lower (95% CI=-0.37 to -0.12, p<0.001 adjusted for confounders). Similarly, in the self-referential dislike block, for each additional positive word chosen, SMFQ score was 0.14 points lower (95% CI=-0.25 to -0.03, p=0.02 adjusted for confounders). There was also strong evidence that, for each additional positive word chosen in the other-referential like block, SMFQ score was 0.20 points lower (95% CI=-0.33 to -0.07, p=0.003 adjusted for confounders). There was no evidence for an association between positive responses and depressive symptoms in the other-referential dislike block (p=0.24; Table 3.4; Figure 3.2).

Table 3.4 Linear regression models testing change in depressive symptoms (SMFQ score; continuous outcome) for each one unit increase in positive responses or global ratings (continuous exposures, tested in separate models) in each condition.

	Model 1: unadjusted (n=594)		**Model 2: adjusted** (n=582)	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Exposures: Positive responses				
Self like	-0.21 (-0.33 to -0.08)	0.001	-0.24 (-0.37 to -0.12)	<0.001
Self dislike	-0.16 (-0.27 to -0.05)	0.01	-0.14 (-0.25 to -0.03)	0.02
Other like	-0.19 (-0.32 to -0.06)	0.003	-0.20 (-0.33 to -0.07)	0.003
Other dislike	-0.07 (-0.19 to 0.06)	0.29	-0.08 (-0.21 to 0.05)	0.24
Exposures: Global ratings				
Self like	-0.04 (-0.06 to -0.02)	<0.001	-0.05 (-0.07 to -0.02)	<0.001
Self dislike	-0.02 (-0.05 to -0.001)	0.04	-0.02 (-0.04 to 0.001)	0.07
Other like	-0.01 (-0.03 to 0.01)	0.42	-0.01 (-0.03 to 0.01)	0.42
Other dislike	-0.01 (-0.03 to 0.02)	0.52	-0.01 (-0.03 to 0.02)	0.67

Note. Both models included all four types of hits as exposures. Model 2 was adjusted for age group, gender, continuous age within each group, non-verbal IQ score, school, and testing group size. Positive responses ranged from 0 to 20. For global ratings, 0 represented dislike and 100 represented like.

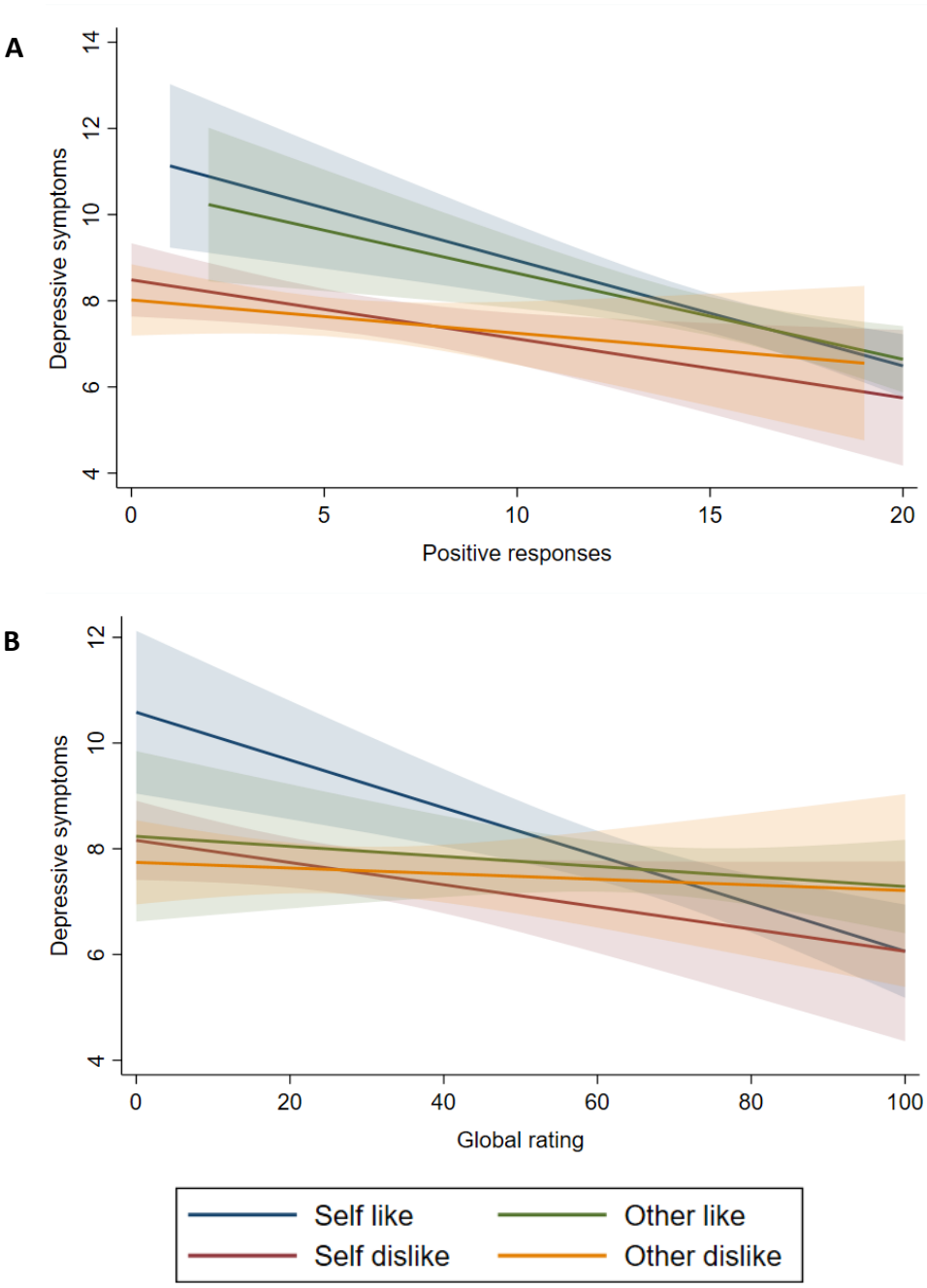

Figure 3.2 A) Expected depressive symptoms (SMFQ score) from the fully adjusted linear regression model with number of positive responses in each block as the exposures. B) Expected depressive symptoms (SMFQ score) from the fully adjusted linear regression model with global rating after each block as the exposures. Both graphs adjusted for age group, gender, continuous age within each age group, school, testing group size, and non-verbal IQ score. Shaded area shows 95% confidence intervals.

Hypothesis 1.5: the association between the positive self-referential bias depressive symptoms would not differ across genders or age groups

Finally, I tested whether associations between positive responses and depressive symptoms differed according to age group and gender. There was no evidence that the associations between positive responses and depressive symptoms differed according to age group (adjusted two-way interaction between age group and positive responses on depressive symptoms: self like p=0.73; self dislike p=0.97; other like p=0.81; other dislike p=0.18) or gender (adjusted two-way interaction between gender and positive responses on depressive symptoms: self like p=0.69; self dislike p=0.66; other like p=0.16; other dislike p=0.06).

There was also no evidence that the associations between positive responses and depressive symptoms differed according to both age group and gender (adjusted three-way interaction between positive responses, age group and gender on depressive symptoms tested separately for all conditions: self like p=0.74; self dislike p=0.76; other like p=0.25; other dislike p=0.55 adjusted).

3.3.2 Global ratings

Global ratings could range from 0 to 100 (where 0 = dislike, 100 = like). There was evidence that global ratings differed according to rule (like/dislike; Table 3.2). Participants rated the character's opinion 39.50 (95% CI=-41.12 to -37.88, p<0.001 adjusted for confounders) points lower in dislike than like blocks, demonstrating that they could accurately reflect on the rule in each block.

Hypothesis 1.1: adolescents would demonstrate a positive self-referential bias

There was no evidence that global ratings differed overall across self- or other-referential conditions (p=0.38 adjusted for confounders; Table 3.2). There was also no evidence for an interaction between condition and rule on global ratings (adjusted interaction p=0.64).

Hypothesis 1.2: the positive self-referential bias would be smaller in girls than boys

There was no evidence for an association between gender and overall global ratings (p=0.32 adjusted for confounders; Table 3.2). There was also no evidence for interactions between gender, condition, and rule on global ratings (Table 3.3).

Hypothesis 1.3: the gender difference in positive self-referential bias would not differ across age groups

I then tested whether the association between gender and global ratings differed according to age group. There was no evidence for a two-way interaction between gender and age group on global ratings (adjusted interaction p=0.83; Table 3.3). There was also no evidence for an association between age group and global rating (p=0.98 adjusted for confounders; Table 3.2), or any of the interactions between age group, gender, condition, and rule on global ratings (Table 3.3; Figure 3.1).

Hypothesis 1.4: the positive self-referential bias would be negatively associated with depressive symptoms

Next, I tested whether global ratings within each condition were associated with depressive symptoms. There was evidence that global ratings in self-referential blocks were negatively associated with depressive symptoms, but no evidence for associations between global ratings in other-referential blocks and depressive symptoms (Table 3.4; Figure 3.2). There was strong evidence that, in the self-referential like block, for each one-point increase in global rating, SMFQ score was 0.05 points lower (95% CI=-0.07 to -0.02, p<0.001 adjusted for confounders). Before adjusting for confounders, there was also evidence that global ratings in the self-referential dislike block were negatively associated with depressive symptoms (coef=-0.02, 95% CI=-0.05 to -0.001, p=0.04). However, after adjusting for confounders, this evidence was attenuated and no longer reached significance (coef=-0.02, 95% CI=-0.04 to 0.001, p=0.07 adjusted for confounders).

Hypothesis 1.5: the association between the positive self-referential bias depressive symptoms would not differ across genders or age groups

There was no evidence that associations between global ratings and depressive symptoms differed according to age group (adjusted two-way interaction between age group and positive responses on depressive symptoms: self like p=0.52; self dislike p=0.16; other like p=0.59; other dislike p=0.90) or gender (adjusted two-way interaction between gender and positive responses on depressive symptoms: self like p=0.69; self dislike p=0.33; other like p=0.21; other dislike p=0.76).

There was also no evidence that the associations between global ratings and depressive symptoms differed according to both age group and gender (adjusted three-way interaction between global ratings, age group and gender on depressive symptoms tested separately for all conditions; self like p=0.51; self dislike p=0.63; other like p=0.48; other dislike p=0.39 adjusted for confounders).

3.3.3 Sensitivity analyses: additional confounders

All parents/carers were asked to complete the parental questionnaire, but response rates were low (n=348, 58%). In this subsample, 78% of participants were of white ethnicity and 89% had English as their first language. According to parents, 5% of the subsample had a mental health problem, with 1% reporting depression. Use of mental health services by their child was reported by 8% of parents, and 1% of participants were reported to be currently receiving psychological therapy for depression. Only 1 participant was currently using antidepressants according to parental report. In total, 14% of parents/carers reported that their child had special educational needs and disabilities, with 7% reporting dyslexia and 1% reporting ASD. Parent education was high for 88% of the subsample. In terms of parental mental health, 15% of mothers and 9% of fathers had experienced depression. The two age groups were similar except that mid-adolescents had higher rates of mental health problems, use of mental health services, and receipt of psychological therapy (Table 3.5).

Table 3.5 Characteristics of the subsample with data on additional confounders according to age group.

	Young adolescents	Mid-adolescents	Overall
	N (%)		
Ethnicity			
White	155 (79%)	116 (77%)	271 (78%)
Mixed	9 (5%)	17 (11%)	26 (8%)
Asian/Asian British	5 (3%)	2 (1%)	7 (2%)
Black/Black British	7 (4%)	3 (2%)	10 (3%)
Other	19 (10%)	13 (9%)	32 (9%)
Total n	*195*	*151*	*346*
English as first language	173 (90%)	132 (88%)	305 (89%)
Total n	*192*	*150*	*342*
Mental health problem	6 (3%)	13 (9%)	19 (6%)
Total n	*195*	*151*	*346*
Used mental health services	12 (6%)	16 (11%)	28 (8%)
Total n	*195*	*151*	*346*
Receiving psychological therapy	0 (0%)	5 (3%)	5 (1%)
Total n	*195*	*152*	*347*
Special educational needs and disabilities	24 (12%)	23 (15%)	47 (14%)
Total n	*194*	*150*	*344*
High parental education	165 (89%)	124 (88%)	289 (88%)
Total n	*186*	*141*	*327*
Maternal depression anxiety or stress	81 (44%)	64 (45%)	145 (44%)
Total n	*183*	*145*	*328*
Paternal depression anxiety or stress	56 (34%)	42 (34%)	98 (34%)
Total n	*167*	*125*	*292*
Pubertal stage			
Early	49 (84%)	13 (21%)	62 (52%)
Late	9 (16%)	48 (79%)	57 (48%)
Total n	*58*	*61*	*119*

Note. Mental health problem denotes whether participant had ever been diagnosed with any mental health problem. Highest reported parental education for each participant (across both mother and father) was split into low (highest qualification GCSE or lower) and high (A Levels or higher). Pubertal stage was split into early (pre-pubertal, early pubertal, and mid-pubertal) and late (late pubertal and post-pubertal). See sections 2.4.10 and 2.5 for more details.

3.3.3.1 Positive responses

There was no evidence that ethnicity (p=0.21), English as a first language (p=0.18), dyslexia (p=0.57), parental education (p=0.11), maternal depression (p=0.81), or paternal depression (p=0.13) were associated with the total number of positive responses on the social evaluation learning task. There was evidence that ASD was associated with more positive responses overall (coef=10.28, 95% CI=3.43 to 17.14, p=0.003). I repeated the main analyses adjusting for these additional confounders. As shown in Table 3.6 and Table 3.7, this did not

substantially alter the evidence for any associations between condition, rule, age group, gender, and positive responses in this subsample whose parents/carers completed the parental questionnaire.

Table 3.6 Linear multilevel models testing associations between age group, gender, condition (self-referential/other-referential), rule (like/dislike; exposures), and positive responses or global ratings (continuous outcomes, tested in separate models). Models include only the subsample of participants for whom additional confounders were available.

	Adjusted models		**Additionally adjusted models**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Outcome: Positive responses				
Condition	0.44 (0.02 to 0.86)	0.04	0.44 (0.02 to 0.86)	0.04
Rule	-10.34 (-10.76 to -9.92)	<0.001	-10.34 (-10.76 to -9.92)	<0.001
Age group	-0.19 (-0.68 to 0.30)	0.45	-0.25 (-0.74 to 0.24)	0.32
Gender	0.12 (-0.32 to 0.55)	0.59	0.13 (-0.31 to 0.57)	0.56
Outcome: Global ratings				
Condition	-0.09 (-2.30 to 2.11)	0.94	-0.09 (-2.30 to 2.11)	0.94
Rule	-42.57 (-44.77 to -40.37)	<0.001	-42.57 (-44.78 to -40.37)	<0.001
Age group	0.49 (-2.35 to 3.33)	0.73	0.29 (-2.51 to 3.10)	0.84
Gender	-0.36 (-2.89 to 2.16)	0.78	-0.23 (-2.75 to 2.29)	0.86

Note. N=283. All models included condition, rule, age group, and gender and were adjusted for continuous age within each age group, school, testing group size, and non-verbal IQ score. Additionally adjusted models were also adjusted for ethnicity, English as a first language, dyslexia, autism spectrum disorders, parental education, maternal depression, paternal depression. Positive responses ranged from 0 to 20. For global ratings, 0 represented dislike and 100 represented like.

In the subsample of participants with data on additional confounders, there was no evidence that positive responses in self dislike (p=0.26) or other like (p=0.10) blocks were associated with depressive symptoms (Table 3.8). Adjusting for additional confounders did not alter this evidence. The lack of evidence is likely due to the reduced sample size or selection bias in participants whose parents/carers completed additional questions. Consistent with the main analysis, there was strong evidence for an association between positive responses in the self like block and depressive symptoms, and no evidence that positive responses in the other dislike block were associated with depressive symptoms, before and after adjusting for additional confounders in this subsample (Table 3.8). As in the main analyses, after adjusting for additional confounders, there was no evidence for a three-way interaction between age group, gender, and positive responses in any block on depressive symptoms (adjusted interactions: self like p=0.26; self dislike p=0.87; other like p=0.27; other dislike p=0.80).

Table 3.7 Interaction term p values from linear multilevel models testing the interactions between age group, gender, condition (self-referential/other-referential) and rule (like/dislike) on positive responses or global ratings (continuous outcomes). Models include only the subsample of participants for whom additional confounders were available.

	Adjusted models	Additionally adjusted models
Outcome: Positive responses		
Condition x rule	0.05	0.05
Gender x condition	0.18	0.18
Gender x rule	0.50	0.50
Gender x condition x rule	0.51	0.51
Age group x gender	0.74	0.79
Age group x gender x condition	0.47	0.46
Age group x gender x rule	0.64	0.64
Age group x gender x condition x rule	0.11	0.11
Outcome: Global ratings		
Condition x rule	0.23	0.23
Gender x condition	0.69	0.69
Gender x rule	0.14	0.14
Gender x condition x rule	0.82	0.82
Age group x gender	0.48	0.56
Age group x gender x condition	0.47	0.47
Age group x gender x rule	0.07	0.07
Age group x gender x condition x rule	0.90	0.90

Note. N=283. Each interaction was tested in a separate model. All models included condition, rule, age group, and gender and were adjusted for continuous age within each age group, school, testing group size, and non-verbal IQ score. Additionally adjusted models were also adjusted for ethnicity, English as a first language, dyslexia, autism spectrum disorders, parental education, maternal depression, paternal depression.

3.3.3.2 Global ratings

There was no strong evidence that ethnicity (p=0.06), English as a first language (p=0.42), dyslexia (p=0.81), ASD (p=0.16), parental education (p=0.14), maternal depression (p=0.62), or paternal depression (p=0.63) were associated with average global ratings on the social evaluation learning task. Adjusting analyses of global ratings for these additional confounders did not substantially alter the evidence for any associations between condition, rule, age group, gender, and global ratings in this subsample (Table 3.6 and Table 3.7).

As with positive responses, in this subsample, there was only evidence for an association between global ratings and depressive symptoms in the self like block, before and after adjusting for additional confounders (Table 3.8). There was no evidence that global ratings in other blocks were associated with depressive symptoms in this subsample. There was also no evidence for a three-way interaction between global ratings after each block, age group, and

gender on depressive symptoms (adjusted interactions: self like p=0.30; self dislike p=0.92; other like p=0.29; other dislike p=0.17).

Table 3.8 Linear regression models testing change in depressive symptoms (SMFQ score; continuous outcome) for each one unit increase in positive responses or global ratings (exposures, tested in separate models) in each condition, including only the subsample of participants for whom additional confounders were available.

	Model 1: adjusted		**Model 2: additionally adjusted**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Exposures: Positive responses				
Self like	-0.27 (-0.47 to -0.08)	0.006	-0.28 (-0.47 to -0.09)	0.004
Self dislike	-0.10 (-0.26 to 0.07)	0.26	-0.11 (-0.27 to 0.06)	0.20
Other like	-0.18 (-0.39 to 0.04)	0.10	-0.17 (-0.39 to 0.04)	0.11
Other dislike	0.03 (-0.15 to 0.22)	0.71	0.07 (-0.12 to 0.25)	0.47
Exposures: Global ratings				
Self like	-0.04 (-0.08 to -0.003)	0.04	-0.04 (-0.07 to 0.0001)	0.05
Self dislike	-0.01 (-0.05 to 0.02)	0.52	-0.01 (-0.05 to 0.02)	0.50
Other like	-0.004 (-0.04 to 0.03)	0.86	-0.004 (-0.04 to 0.03)	0.85
Other dislike	-0.003 (-0.04 to 0.03)	0.88	0.003 (-0.03 to 0.04)	0.86

Note. N=281. Both models included all four types of hits as exposures and were adjusted for age group, gender, continuous age within each group, non-verbal IQ score, school, and testing group size. Additionally adjusted models were also adjusted for ethnicity, English as a first language, dyslexia, autism spectrum disorders, parental education, maternal depression, paternal depression. Positive responses ranged from 0 to 20. For global ratings, 0 represented dislike and 100 represented like.

3.3.4 Sensitivity analyses: pubertal stage

The PDS was fully completed by a small proportion of participants (n=119, 20% of total sample). Of the subsample that completed this measure, 62 (52%) were in early pubertal stages (pre-pubertal, early pubertal, and mid-pubertal) and 57 (48%) were in late pubertal stages (late pubertal and post-pubertal). Mid-adolescents were generally in later stages of puberty than young adolescents (Table 3.5).

There was no evidence that pubertal stage was associated with the total number of positive responses on the social evaluation learning task (coef=-0.49, 95% CI=-2.84 to 1.87, p=0.68), or the average global ratings (coef=-0.58, 95% CI=-4.19 to 3.04, p=0.75). There was also only weak evidence that pubertal stage was associated with depressive symptoms in this subsample (coef=2.18, 95% CI=0.12 to 4.25, p=0.04). Given this, and the small proportion of the sample who completed the PDS, I did not further adjust analyses for pubertal stage.

3.4 Discussion

In this study, I investigated whether there are biases in implicitly learning about and explicitly reflecting on social evaluation during adolescence. I hypothesised that, as in adults, adolescents would demonstrate a positive self-referential bias (hypothesis 1.1). I found evidence to support this hypothesis during learning. Adolescents were worse at learning that a character disliked them than learning that a character disliked another person. I also hypothesised that this positive self-referential bias would be smaller in girls than boys (hypothesis 1.2). Contrary to my hypotheses, I found no evidence for a gender difference in this positive self-referential bias in learning in either age group (hypothesis 1.3).

As hypothesised, I did find evidence that the positive self-referential learning bias was negatively associated with depressive symptoms (hypothesis 1.4). Participants who chose the positive word more often when learning that they were liked and disliked had less severe depressive symptoms. I hypothesised that this association between the positive self-referential bias depressive symptoms would not differ across genders or age groups (hypothesis 1.5) and, consistent with this, I did not find evidence that gender or age group moderated the association between positive self-referential bias and depressive symptoms. There was also some evidence that other-referential learning was negatively associated with depressive symptoms, which was not expected. Participants who chose the positive word more often when learning that another person was liked had fewer depressive symptoms.

After each block, participants were asked to reflect on their learning and rate whether the characters liked or disliked them and the other person. I hypothesised that biases in these ratings would be similar to positive responses during learning, with adolescents rating the character's opinion of the self as more positive than their opinion of the other after learning (hypothesis 1.1). Contrary to this hypothesis, I did not find evidence that participants rated the character's opinion of them differently to the character's opinion of another person. The effect size for the difference between self-referential and other-referential global ratings was very small, particularly in comparison to the difference between global ratings for like and dislike blocks. This suggests that participants more accurately reflected on the social evaluation presented by each character when making global ratings about both the self and other person. There was also no evidence that these ratings differed with gender in either

age group (contrary to hypothesis 1.2, but consistent with hypothesis 1.3). As hypothesised, there was evidence that ratings of whether the self was liked were associated with depressive symptoms (hypothesis 1.4). Adolescents with more severe depressive symptoms rated themselves as less liked. This association was specific to learning about the self, as ratings of whether another person was liked or disliked were not associated with depressive symptoms. Also as hypothesised, there was no evidence that this association with depressive symptoms differed across genders or age groups (hypothesis 1.5).

3.4.1 Strengths and limitations

Here I will outline some strengths and limitations of my data, which are also applicable to the data used in chapters 4-6. This discussion is not exhaustive, and more critical appraisal will follow in chapter 7 (section 7.3).

My sample was population-based and included the full range of depressive symptoms (from none to severe), which I analysed continuously. This should have increased my statistical power to detect any associations between learning about social evaluation and depressive symptoms (Button et al., 2013). The sample was recruited from eight diverse schools, making it more representative than many previous studies of biases in predicting social evaluation.

This study was novel in testing learning about social evaluation in adolescence. I adapted the social evaluation learning task for use with adolescents, ensuring that appropriate personality descriptors were presented. I also updated the character names from previous versions of this task to gender-neutral names. This aimed to remove the confound of gender differences in interacting with same- versus other-sex peers (Benenson & Christakos, 2003; Buhrmester & Prager, 1995). This task allowed me to test automatic processing of social evaluation as well as more explicit opinions about evaluation, which may involve different processes (Kahneman, 2011; Roiser et al., 2012; Strack & Deutsch, 2004).

There were also some limitations of the social evaluation learning task. Performance may be due to reverse causation, as those with more severe depressive symptoms might feel reluctant to select positive words. The task aimed to mimic a social interaction, but participants were aware that they were interacting with computer characters rather than their peers. I cannot be sure that I was measuring social information processing, as I did not

measure the extent to which adolescents felt socially engaged during the task. However, adolescents do react similarly to computer-based and real-life interactions. For example, peer rejection is distressing even when it is by a computer (e.g. Sebastian, Viding, et al., 2010; Zadro, Williams, & Richardson, 2004). Future work could incorporate real social feedback from known peers, such as classmates, to allow for more ecologically valid inferences from this task.

The poor parental consent rates in several schools was a limitation. As described in section 2.9.2, 24% of my sample were recruited from five schools with low parental consent (under 30% of eligible parents/carers consented) and 76% of my sample were recruited from three schools with high parental consent (over 60% of eligible parents/carers consented). Selection bias may have occurred, as participants had higher non-verbal IQ in schools with low parental consent rates. However, the majority of the sample were from schools with high consent and opt-out consent was used to recruit nearly half of the sample. I do not think that the factors influencing parental consent would alter associations between learning about social evaluation and depressive symptoms, so selection bias should not have influenced my findings.

In this study, I hypothesised that I would find no evidence for effect modification. I predicted that age group would not moderate the association between gender and positive self-referential bias (hypothesis 1.3), and also expected that neither gender nor age group would moderate the association between positive self-referential bias and depressive symptoms (hypothesis 1.5). In order to test these hypotheses of no association, I need to determine whether I had sufficient power to reject the null hypothesis. My sample was powered to detect a difference of 0.4 standard deviations in outcomes within each age group, which is equivalent to approximately 1.5 positive responses during learning or 8.5 points in the global rating. This is a relatively large difference in comparison to the effects that I did find, meaning that my study may not have been sufficiently powered to detect small effects. Therefore, although I did not find evidence for effect modification by age group or gender, this may have been because the study was underpowered. In this statistical framework, demonstrating that an association does not exist is difficult (Amrhein, Greeenland, & McShane, 2019; Hurlbert, Levine, & Utts, 2019).

Additionally, to study the effects of age group and gender on task performance, I tested a number of two, three-, and four-way interactions. My sample is likely to have been underpowered to test two-, three-, and four-way interactions, and this may increase the possibility that findings are due to chance. The low power for testing these interactions, which is likely to be below 50% for four-way interactions, could result in absence of evidence for the effects of age group and gender, even if these effects do exist. My findings should therefore be confirmed in a sufficiently powered sample before concluding that there are no effects of age group or gender on task performance. I also have not corrected for multiple comparisons, further increasing the likelihood of a Type 1 error (false positive). I made this decision because adjustments such as using a Bonferroni correction are often too conservative, increasing the risk of Type 2 errors (false negatives; Perneger, 1998; Rothman, 1990; Streiner & Norman, 2011). I planned analyses *a priori* and only conducted the analyses directly relevant to my hypotheses to reduce the number of exploratory analyses and multiple comparisons in this study.

Although I adjusted for several potential confounders, residual confounding is also possible. A further limitation is that pubertal stage was measured for very few participants (20%) and was not associated with task performance in this subsample, so was not included in analyses. It is possible that this was due to a lack of power in this reduced sample. Information on demographics, special educational needs, and parental mental health were also only available for a subsample of participants (58%). In this subsample (before and after adjusting for additional confounders), there was only evidence for an association between learning that the self was liked and depressive symptoms. This was the same for the global ratings. These findings are likely due to the reduced sample size or selection bias in parents/carers who completed additional questionnaires. However, effect estimates were within the confidence intervals from the main analyses, and these findings do suggest that learning whether others like you is most robustly associated with depressive symptoms in adolescence. It is interesting that, in this subsample, I found evidence that participants with ASD made more positive responses during learning, indicating that they have more positive biases in learning whether characters like them and others. ASD is typically associated with depression (Ghaziuddin, Ghaziuddin, & Greden, 2002; Matson & Nebel-Schwalm, 2007) so, given the main findings in

this study, this association between ASD and increased positive responses was unexpected. However, the sample with ASD was very small (n=4), so this finding may not be replicable.

As this was a cross-sectional study, I cannot provide evidence of a causal effect of biases in learning about social evaluation on depressive symptoms, which is proposed by cognitive models of depression (Beck & Bredemeier, 2016; Roiser et al., 2012). My findings are consistent with such models. However, it is equally possible that changes in depressive symptoms cause changes in learning (reverse causality), or that the association is bidirectional. Longitudinal data is required to test the hypothesis that biases in learning about social evaluation lead to increased depressive symptoms during adolescence.

3.4.2 Findings in context

I found evidence that, as demonstrated in adults (Button et al., 2012, 2015), adolescents have positive biases when learning what others think about them in social interactions. This positive self-referential bias is in contrast with previous findings that young and mid-adolescents (aged 8-14) made accurate predictions about social evaluation, whereas older adolescents (aged 19-25) had more positive biases in predicting social evaluation (Gunther Moor et al., 2010; Rodman et al., 2017). This is likely due to the different nature of the tasks used. Previous research has presented adolescents with images of peers and asked them to rate whether each peer would like or dislike them. This requires explicit reflection. In contrast, in this study, I aimed to measure more implicit biases when interacting with others, recording the number of times participants chose the word indicating that the character had a positive opinion of them. Participants then received feedback and learnt whether the positive or negative word was more likely to be correct. Choices were therefore made on the basis of accruing information, as they would be in a real social interaction, rather than an explicit judgement on whether the character would like or dislike them. It may be that individuals have positive biases when integrating information in this way from an early age, but positive biases in explicit ratings of whether others like or dislike them only emerge later.

This theory is supported by adolescents' ratings after learning about social evaluation. When asked to reflect on their learning and rate what each character thought of them and another person, adolescents no longer demonstrated positive self-referential biases. This may reflect a distinction between biases in learning and more reflective global appraisals after the event,

as proposed by dual-process models (Strack & Deutsch, 2004). Reflections on learning could represent the information that is integrated into the self-concept after interactions (although we do not know if this is the case). If so, it is interesting that there may be protective biases during interactions, but positive biases are not present in the information which is used to form a self-concept.

As in studies with adults (Hobbs et al., 2018), I found evidence that adolescents with more severe depressive symptoms were less likely to think that they were liked, both during learning and in later ratings. This occurred across both like and dislike rules, indicating that adolescents with more severe depressive symptoms were both worse at learning they were liked and better at learning they were disliked. There was also some evidence that this bias transferred to other-referential learning. Adolescents with more severe depressive symptoms were worse at learning that others were liked, although they were not better at learning others were disliked. Those with more severe depressive symptoms may have a negative bias in responding, making them more likely to choose the negative word in every block across the task. This finding could also indicate that the association between depressive symptoms and learning about social evaluation is due to overlap in the content of these measures, with the choice of a negative word corresponding to reporting more depressive symptoms. However, I would then also expect to find evidence of an association with depressive symptoms in other-referential dislike blocks, which I did not. This indicates that learning about social evaluation may be meaningfully associated with depressive symptoms, and not simply the result of content overlap in the measures. It is possible that adolescents with more depressive symptoms are also less likely to believe that others are liked, in addition to their self-referential biases. In contrast to these responses during learning, global ratings of whether others were liked or disliked were not associated with depressive symptoms.

Overall, the evidence indicates that learning about and reflecting on social evaluation when the self was liked were most strongly associated with depressive symptoms. This is consistent with previous evidence that adolescents with more depressive symptoms or lower self-esteem were less likely to believe that peers will like or be interested in them (Caouette & Guyer, 2016; Guyer et al., 2008; Somerville et al., 2010). It is also consistent with cognitive models of depression which state that individuals with depression have more negative interpretations of their social interactions (Beck & Bredemeier, 2016; LeMoult & Gotlib, 2019;

Moore & Fresco, 2012; Roiser et al., 2012; Silk et al., 2012). Impairments in learning that others have a positive opinion of you may lead to a less positive view of the self, reduced self-confidence and self-esteem, more negative mood, and more negative information available for later rumination. This could result in less perceived social success and social withdrawal.

However, the mechanisms underlying learning about and reflecting on social evaluation remain unclear. Adolescents may have an initial positive bias, believing that others will like them, and this bias decreases with increasing depressive symptoms. Alternatively, adolescents could update their beliefs more based on feedback indicating that others like them, automatically discounting feedback which suggests otherwise, and this bias may be associated with depressive symptoms. There could also be a combination of these processes, or a number of other underlying mechanisms, which are associated with depressive symptoms. Although I have discussed learning throughout this study, I have relied on summary statistics from the social evaluation learning task as indicators of learning (number of positive responses). This is informative, providing an overall picture of participants' biases when learning about social evaluation. However, it does not allow me to investigate any of the processes potentially underlying learning, which could enable me to identify differences in self-referential and other-referential learning. In the next chapter, I will therefore develop and validate reinforcement learning models (Sutton & Barto, 2018), with the aim of understanding the processes by which adolescents learn about social evaluation.

3.4.3 Conclusion

In this chapter, I have described findings from a cross-sectional study providing preliminary evidence on how young and mid-adolescents learn about social evaluation. Consistent with my hypotheses, I found evidence that adolescents were better at learning that they were liked than disliked, and a reduction in this positive self-referential bias was associated with depressive symptoms. Also as hypothesised, there was no evidence for age or gender differences in this association between positive self-referential bias and depressive symptoms. Contrary to my hypotheses, I did not find evidence for a gender difference in learning about social evaluation. However, tests for age and gender differences in these processes were likely underpowered. Next, I will describe computational analyses which aim

to investigate whether any of the processes involved in social evaluation learning change with age and identify which aspects of learning are associated with depressive symptoms.

Chapter 4 Computational mechanisms underlying social evaluation learning during adolescence

Contribution declaration: The initial reinforcement learning models described in this chapter were developed in collaboration with Dr Alex Pike. I then performed all analyses and further adapted the models myself.

4.1 Introduction

In chapter 3, I examined how adolescents learn about social evaluation. Using a task which simulates social interactions, I found evidence that adolescents had a positive self-referential bias during learning. Adolescents were better at learning that computer characters liked than disliked them. They were also better at learning that characters disliked another person than learning that a character disliked them. Completing this task requires instrumental learning, as participants use probabilistic feedback to infer whether they are liked or disliked. Although participants' responses on this task indicate whether they have learnt about social evaluation, they do not reveal the specific cognitive processes involved in learning (as discussed in section 3.4.2). Adolescents' positive self-referential bias could be a result of a number of cognitive processes. For example, individuals may have pre-existing biases that others will evaluate them positively or they might ignore feedback which suggests a character does not like them.

Computational models can help to provide insight into which cognitive processes may underlie behaviour. Models contain a number of parameters, each of which specifies the influence of a hypothesised cognitive process on behaviour at a trial level (Adams et al., 2016). The computational framework of reinforcement learning describes how associations between stimuli, actions, and outcomes are learned. It can be used to model how individuals learn to gain positive feedback and avoid negative feedback, improving choices to achieve the best outcome (Rescorla & Wagner, 1972; Sutton & Barto, 2018). In reinforcement learning models, a parameterised algorithm learns the subjective expectation of reward from performing an action in a given state, and the extent to which this expectation influences the probability of making each action. The simplest reinforcement learning models assume one common mechanism for all kinds of outcomes and actions regardless of their valence or state. However, these models can be extended to describe learning in different conditions, for

example about the self and others, as well as by including biases in learning and actions. The reinforcement learning framework can thus help us to understand how people learn about social evaluation.

A recent study developed reinforcement learning models to describe how adults learn about social evaluation (Hopkins, Dolan, Button, & Moutoussis, 2019). Modelling performance on the social evaluation learning task, they found evidence that individuals updated their expectations differently for learning about the self and others. People also placed more value on positive information when choosing their actions and varied in their initial tendency to choose a positive word in this task, generally showing an initial positive bias (Hopkins et al., 2019). However, this study was not large (n=100) and the sample was recruited based on clinical characteristics, which could lead to selection bias.

Decision making and learning are complex processes which may continue developing throughout adolescence (Hauser, Will, Dubois, & Dolan, 2019; Palminteri, Kilford, Coricelli, & Blakemore, 2016). Reinforcement learning may differ in early adolescence to late adolescence and adulthood (Bolenz, Reiter, & Eppinger, 2017; Cohen et al., 2010; Davidow, Foerde, Galván, & Shohamy, 2016; Hauser, Iannaccone, Walitza, Brandeis, & Brem, 2015; Hauser et al., 2019; Palminteri et al., 2016; Van Der Schaaf, Warmerdam, Crone, & Cools, 2011). From childhood to adulthood, individuals become better at optimally weighting recent feedback and less exploratory in their decision making (Nussenbaum & Hartley, 2019). It is less clear how specific aspects of learning, for example learning from positive and negative information, differ across development (Nussenbaum & Hartley, 2019). This is partly due to heterogeneity in the tasks used across studies, which have different optimal patterns of learning. It is also possible that social reinforcement learning develops differently to non-social reinforcement learning (Bolenz et al., 2017). Positive social feedback may become increasingly rewarding throughout adolescence, and this may influence learning (Davey et al., 2008).

Although I did not find evidence for associations between age group and learning about social evaluation in chapter 3, reinforcement learning processes may still be developing in early and mid-adolescence. Very few studies have tested this to date. There is some evidence from a social reinforcement learning task, which manipulated the probability of receiving positive social feedback, that adolescents had lower learning rates for positive social feedback

compared to children and adults (sample aged 8-25; Jones et al., 2014). Whist different amounts of positive feedback enhanced learning in children and adults, all amounts of positive feedback equally motivated adolescents. This supports the idea that adolescence may be a period of particular sensitivity to social feedback. In contrast, there was no evidence for differences in learning rates for negative social feedback across development (Jones et al., 2014). Together these findings indicate that learning about social evaluation may not be explained by simple reinforcement learning theory in adolescence. However, each age group was relatively small (between 37 and 45 participants) in this cross-sectional study, so it may have been underpowered to test age differences in learning. It is therefore not clear whether adolescents learn from social feedback differently to adults. A recent review concluded that "next to nothing" is known about the development of learning about social evaluation during adolescence (Hauser et al., 2019).

In chapter 3, I also found evidence that responses when learning about social evaluation were associated with depressive symptoms. Adolescents who chose the positive word less often when learning that they were liked and disliked had more severe depressive symptoms. There was also evidence that making fewer positive responses when learning that another person was liked was associated with more severe depressive symptoms. By identifying which aspects of learning drive these associations, we can gain a better understanding of the links between learning about social evaluation and depressive symptoms. This may reveal mechanisms which cause or maintain depressive symptoms in adolescence and enable us to target more specific aspects of social information processing in interventions to reduce or prevent depressive symptoms.

Although there is evidence for impairments in reinforcement learning in depression during adulthood (see Halahakoon et al., 2020 for a review), very few studies have tested whether reinforcement learning is associated with depressive symptoms in adolescence (Hauser et al., 2019). In general, depressive symptoms may be associated with lower reward sensitivity and reduced reward-seeking behaviour in adolescence (e.g. Bress, Foti, Kotov, Klein, & Hajcak, 2013; Forbes, Shaw, & Dahl, 2007; Morgan, Olino, McMakin, Ryan, & Forbes, 2013; Olino et al., 2014; Rawal, Collishaw, Thapar, & Rice, 2013a; Rawal et al., 2014; Rice et al., 2015). However, many of these studies have used monetary reward tasks. Learning about financial

reward may differ to learning about social rewards during adolescence, as pursuing social rewards may be particularly important in this developmental period of social reorientation.

One study tested associations between self-esteem and performance on a social evaluation learning task in late adolescents (Will et al., 2020). This task involved participants predicting whether other people liked them and receiving social feedback. There was evidence that late adolescents with low (compared to high) self-esteem had a lower expectancy of being liked, and also had lower learning rates for social evaluation, indicating a reduced tendency to update their expectations in response to social feedback (mean age 21; Will et al., 2020). Although there was also some evidence that these aspects of learning were associated with depressive symptoms in canonical correlation analyses, specific associations between each parameter and depressive symptoms were not tested (Will et al., 2020). Additionally, this was a small cross-sectional study (n=61), recruited from relatively affluent families on the basis of high and low self-esteem, meaning it is unlikely to be representative of adolescents in the general population. Based on this evidence, it is not clear whether specific biases in learning about social evaluation are associated with depressive symptoms in adolescence. If these associations do exist, social reinforcement learning could contribute to the increased incidence of depression in girls (as outlined in section 1.6). However, no research has tested gender differences in social reinforcement learning during adolescence to date.

In this study, I aimed to investigate the processes underlying learning about social evaluation in adolescence. Reinforcement learning may be important for maintaining a positive self-referential bias during and after social interactions, and disruptions in specific processes could lead to increases in depressive symptoms. I therefore developed and validated reinforcement learning models to describe trial-by-trial patterns of behaviour, parameterising the processes involved in learning about social evaluation during adolescence. I tested a range of reinforcement learning models which formalised the processes I hypothesised to be involved in learning. I also aimed to examine whether there are gender or age differences in these processes and test whether the processes underlying learning about social evaluation are associated with depressive symptoms in adolescence. I therefore tested whether the parameters describing learning were associated with age group, gender, and depressive symptoms.

Based on my findings in chapter 3, I hypothesised that a number of parameters would be necessary for reinforcement learning models to adequately describe adolescents' behaviour, including separate learning rates for self-referential and other-referential information and parameters modelling a positive self-referential bias (hypothesis 2.1). I hypothesised that none of these parameters would change with age (hypothesis 2.2). I also hypothesised that parameters relating to the positive self-referential bias would be smaller in girls than boys, in both young and mid-adolescents (hypothesis 2.3). Finally, I hypothesised that the positive self-referential bias parameter(s) and self-referential learning rate(s) would be associated with depressive symptoms, across both genders and age groups (hypothesis 2.4).

4.2 Methods

For this study, I used the same data as in chapter 3 (see section 3.3 for descriptive statistics). All reinforcement learning analyses were performed using R version 3.6.0 (R Core Team, 2019) and RStudio version 1.2.5001 (RStudio Team, 2020), with packages bbmle (Bolker & R Development Core Team, 2020), boot (Canty & Ripley, 2019; Davison & Hinkley, 1997), reshape2 (Wickham, 2007), Hmisc (Harrell, 2019), ggplot2 (Wickham, 2016), and RStan (Stan Development Team, 2019).

On each trial of the social evaluation learning task, participants were asked to click on either a positive or negative personality characteristic, selecting the word they believed best matched the computer character's opinion of them (or Taylor in other-referential blocks). Participants then received probabilistic feedback indicating whether this choice was right or wrong, and learnt what the character thought of them through trial and error. I aimed to model how participants learnt about this social evaluation using adaptions of Rescorla-Wagner reinforcement learning models (Rescorla & Wagner, 1972), thus testing hypothesis 2.1. Each reinforcement learning model consists of two parts (the learning model and action model) which formalise how participants use feedback to choose between the two possible actions in this task. The learning model defines how participants learn the value of each action, with values updated on each trial as participants get feedback. The action model then describes how those values are turned into choices.

In the learning model, participants learn the value of the actions *choose the positive word* and *choose the negative word*. The value of action *a* on trial *t*, in the current state s_t, is updated based on feedback after every outcome (giving the action value $Q_t(s_t, a_t)$). The state s_t denotes which condition the trial was in (self-referential versus other-referential). On each trial, the action value is updated by adding the prediction error (δ), scaled by the learning rate (α), to the existing action value. The prediction error is the discrepancy between the expected reward value (the value estimate from the previous trial $Q_{t-1}(s_t, a_t)$) and the actual reward on the trial (r_t). This is multiplied by the learning rate (α), which determines the extent to which the update is influenced by the new evidence. When the learning rate is low, value estimates update slowly, as a recency-weighted average of many outcomes associated with that action. A high learning rate corresponds to new evidence having a strong impact, quickly replacing old learning. This is the Rescorla-Wagner (or delta) rule:

$$Q_t(s_t, a_t) = Q_{t-1}(s_t, a_t) + \alpha \cdot \delta$$

$$\delta = r_t - Q_{t-1}(s_t, a_t)$$

In the action model, actions are chosen probabilistically. The propensity to choose each action is the action value $Q_t(s_t, a_t)$, entered into a softmax function, weighted by an inverse temperature parameter (β > 0). This parameter β models the degree to which actions are guided by action values:

$$Probability(s_t, a_t) = \frac{\exp\left(Q_t(s_t, a_t) \cdot \beta\right)}{\mathrm{sum}(\exp(Q'(s, a) \cdot \beta))}$$

Lower values of β indicate a high level of behavioural stochasticity, meaning choices are barely controlled by action values and are selected with equal probability (exploratory choices). Action selection becomes more deterministic with increasing values of β, meaning the agent is more likely to select the action with the highest action value.

To avoid numerical underflow or overflow (values smaller or larger in magnitude than can be represented by the computer) after taking exponents, I used inverse temperature parameters (β) instead of temperature parameters (1/β) in the softmax function (Clarke et al., 2014). Additionally, I used stabilisation, subtracting the largest possible action value for the trial from the actual action value ($Q_t(s_t, a_t)$) before entering it into the softmax function. As action

values were calculated for the two possible actions (choose positive versus negative word), this stabilisation was:

$$Q_t(s_t, a_{t\,pos}) = Q_t(s_t, a_{t\,pos}) - \max\left(Q_t(s_t, a_{t\,pos}), Q_t(s_t, a_{t\,neg})\right)$$

$$Q_t(s_t, a_{t\,neg}) = Q_t(s_t, a_{t\,neg}) - \max\left(Q_t(s_t, a_{t\,pos}), Q_t(s_t, a_{t\,neg})\right)$$

In order to modify these reinforcement learning models to best describe social evaluation learning, I focussed on learning rates, as these describe which conditions have the largest impact on learning. Four different classes of models were tested (Table 4.1). Firstly, a *uniform model* was considered, which used the classic reinforcement learning model formulation. This included one learning rate, updated on all trials. Given that previous analyses have demonstrated differences in learning about the self and other (chapter 3; Button et al., 2015; Hobbs et al., 2018; Hopkins et al., 2019), I also considered a *condition model* with two separate learning rates for self-referential and other-referential blocks. Additionally, as learning may differ according to whether the positive or negative word was chosen, a *valence model* was tested, with two separate learning rates for positive and negative word choices. Finally, the most complex *condition-valence model* included four learning rates, for positive and negative choices and about the self and other.

Table 4.1 Reinforcement learning model families, grouped according to their defining core parameters.

Model family	Number of parameters	Core parameters
Uniform	2-6	α β
Condition	3-7	α_{self} α_{other} β
Valence	3-7	$\alpha_{positive}$ $\alpha_{negative}$ β
Condition-valence	5-9	$\alpha_{self\ positive}$ $\alpha_{self\ negative}$ $\alpha_{other\ positive}$ $\alpha_{other\ negative}$ β

Note. α: learning rate. β: inverse temperature. Initially, four possible combinations of additional parameters were tested: no bias parameters, one start bias (γ), one positivity bias (δ), or one start and one positivity bias (γ + δ).

Based on prior modelling of this task (Hopkins et al., 2019), I also tested an *outcome model* which included separate learning rates for trials with a positive and negative outcome word (regardless of which word was chosen or the feedback provided). I also checked a more complex version of this model, with four separate learning rates for positive and negative outcomes about the self and other (*condition-outcome model*). Finally, I checked one other type of model, because of previous findings that adolescents may learn differently from positive and negative feedback (Christakou et al., 2013; Jones et al., 2014; Palminteri et al., 2016; Van Den Bos, Cohen, Kahnt, & Crone, 2012). This model included two learning rates for positive and negative feedback (*feedback model*), or four learning rates for positive and negative feedback about the self and other (*condition-feedback model*). These models were tested as a check for other mechanisms potentially underlying behaviour.

As I observed that participants learnt very quickly, and then appeared to exhibit stable responses in each block (Figure 4.1), I also considered whether learning rates should decrease across trials in each block. I tested this by using the trial number in the learning model, with two separate approaches. I tried dividing the learning rate by the trial number ($\frac{\alpha}{trial}$) as well as using the trial number as an exponent (α^{trial}) in separate models. As the learning rate varied between 0 and 1, both of these approaches meant that the learning rate decreased as trial number increased.

I also considered additional bias parameters. Firstly, I added a start bias parameter (γ) to the action model, which defined starting values for the first trial of each block $Q(s_1, a_1)$. This was intended to reflect an initial bias towards choosing the positive word (as can be seen in Figure 4.1), independent of learning. This parameter was allowed to vary across participants but was constant across all blocks within participants. I also included two start bias parameters (γ_{self} and γ_{other}), estimated separately for self-referential and other-referential blocks. These allowed the initial values on the first trial of each block to reflect different biases towards choosing the positive word in self-referential and other-referential conditions.

Secondly, I added positivity bias parameters to the action value to quantify a general positive bias, independent of learning (as done by Hopkins et al., 2019). As with the start bias parameter, I tested two types of positivity bias. The first positivity bias parameter (δ) was implemented across all blocks. This positivity bias was applied for every action selection,

weighting the action, and was constant across all trials. I also considered two separate parameters for positivity biases in self and other blocks ($\delta_{self} + \delta_{other}$), allowing the action value to be biased by separate parameters in self-referential and other-referential blocks.

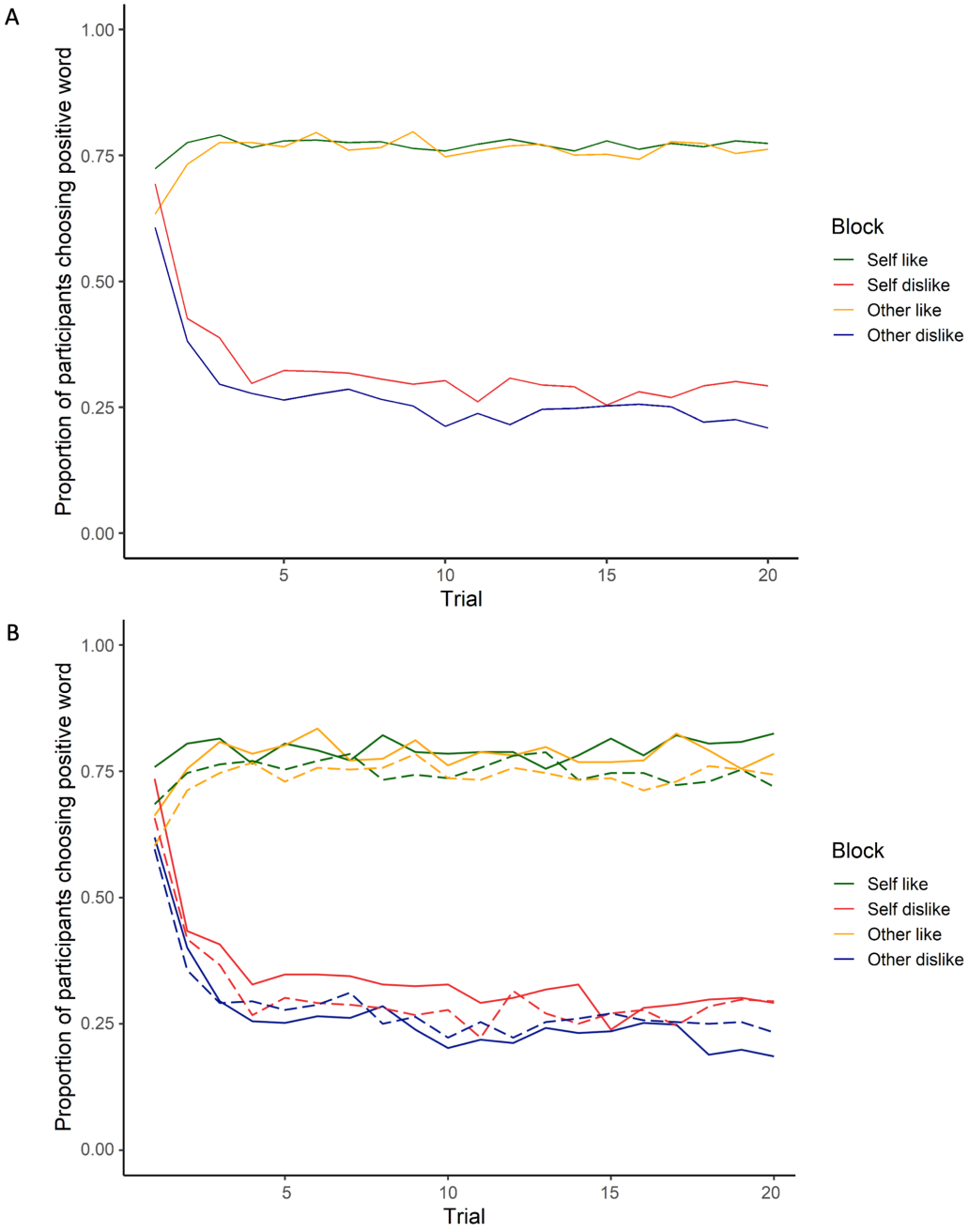

Figure 4.1 Proportion of participants who chose the positive word on each trial of each block (observed performance). A) Proportion of the whole sample. B) Proportion of participants choosing the positive word according to low versus high depressive symptoms. In order to illustrate this, a median split of depressive symptoms was performed. The solid line indicates participants with low depressive symptoms (SMFQ score ≤ 6; n=302). The dashed line indicates participants with high depressive symptoms (SMFQ score > 6; n=292).

4.2.1 Parameter estimation

I estimated individual parameters in a hierarchical model using maximum a posterior estimation (MAP). This procedure involves a number of steps. Firstly, I estimated each model using maximum likelihood estimation (MLE), generating the best set of parameters to maximise the likelihood of predicting each participant's choices. The set of parameters with the smallest negative log likelihood was retained for each participant. Multiple random initial values were used at each interaction to help avoid local minima. This procedure was repeated with several different ranges of random initial values (-5 to 5, -10 to 10, -15 to 15), and numbers of iterations (10, 20). This did not substantially alter parameter estimates, so initial values between -10 and 10 with 20 iterations are reported.

To reduce noise inherent to MLE, I used estimates from the first round of MLE to calculate a hierarchical prior representing the normal distribution of each parameter across the sample. This assumes that the sample all come from the same population and their learning is similar. After calculating these distributions, I re-estimated parameters using MAP, which takes the sample summary statistic as a prior for each parameter, pulling parameter estimates towards the sample mean. This helps to remove any outliers after MLE (which are unlikely under the prior normal distributions) and reflects the fact that data for the rest of the sample are relevant for estimating each individual's parameters (Daw, 2011).

During model fitting, I constrained parameters to within meaningful ranges. I applied exponential transforms to ensure that beta parameters were between 0 and 10. I applied inverse logit transforms to ensure that learning rate parameters were constrained between 0 and 1, and tanh transforms to constrain start bias parameters between -1 and 1.

After identifying a winning model, I re-estimated this model using hierarchical Markov Chain Monte Carlo (MCMC) sampling to provide the best estimate of parameters for further analyses (Valton, Wise, & Robinson, 2020). I fitted this model using RStan (Carpenter et al., 2017). A total of 6000 MCMC samples (1000 warmup) were drawn in 4 chains, resulting in an average effective sample size of 14,278. I determined convergence by visually inspecting the trace plots and monitoring the Gelman-Rubin statistic for each parameter, with values close to 1.00 implying convergence (Gelman & Rubin, 1992).

4.2.2 Model selection

In the following steps, I followed guidelines recommended by Palminteri and colleagues (2017) for model selection which consider both model parsimony and model falsification. The fit of each model to the observed data was compared using the Bayesian information criterion (BIC; Schwarz, 1978) and Akaike information criterion (AIC; Akaike, 1998) at an individual level:

$$BIC = -2\ln(L) + k\ln(n)$$

$$AIC = -2\ln(L) + 2k$$

Where k is the number of free parameters, n is the number of trials, and L is the likelihood of the model (the probability of the observed data at the parameter values that maximise the likelihood function). Each of these were then summed over participants, and the lowest BIC and AIC indicated the winning model (in terms of model parsimony).

4.2.3 Model recovery

To check the parameter estimation process, I generated synthetic parameters and tested their recovery across the range of likely parameter values for each model. To do this, 100 synthetic parameters were drawn from a Gaussian distribution with mean and standard deviation equal to the distribution of the parameters in each model (estimated from the observed data using MAP). A synthetic dataset was generated using these parameters for each model. I then attempted to recover the synthetic parameters in each model using MAP. I compared the synthetic parameters with those recovered from the models using Pearson correlations, thus testing parameter recovery.

4.2.4 Generative performance

I aimed to establish that the best-fitting models did not just predict the observed data, but also reproduced the effects of interest, as shown in chapter 3 (Palminteri et al., 2017). To do this, I simulated data for each participant given the version of the task that they saw, using the parameters produced by each model. I then compared this simulated data to the observed data by plotting the proportion of positive responses on each trial according to task block. I also calculated the percentage of trials on which simulated choices matched observed

choices. To test whether the model could reproduce the associations seen in standard regression analyses, I computed the number of positive responses on each block from the simulated data, and repeated the main analyses performed with the observed data (as in chapter 3).

The winning model was selected using evidence from model fit (BIC, AIC), parameter recovery, and generative performance (Palminteri et al., 2017).

4.2.5 Associations with age group, gender, and depressive symptoms

Next, I examined whether any of the processes involved in learning were associated with age group (hypothesis 2.2), gender (hypothesis 2.3), or depressive symptom severity (hypothesis 2.4). Using estimates from the winning model, I tested whether each parameter (outcome) was associated with age group, gender, and depressive symptom severity (exposures) in unadjusted separate univariable linear regression models. I then adjusted each model for potential confounders (continuous age within each age group, school, testing group size, and non-verbal IQ score). Where there was evidence for an association between depressive symptoms and a parameter, I tested whether this association differed across genders and age groups. To do this, I added a three-way interaction between depressive symptoms, gender, and age group to the linear regression model with the parameter as the outcome. I also tested the two-way interactions including depressive symptoms. Standardised estimates for these analyses are included in Appendix 1.

4.2.6 Sensitivity analysis: effect of age group

In the preceding analyses, I assumed that all adolescents represented a single population, with inter-individual variability such that young and mid-adolescents all lie on a continuum in parameter space. This was based on finding no evidence for age differences in behavioural performance in chapter 3, and the lack of previous evidence for age differences in social reinforcement learning from early to mid-adolescence. However, very little is known about the development of social reinforcement learning. It is possible that young and mid-adolescents are two separate populations, with separate, overlapping, distributions of task performance. To investigate this, I first tested whether generative performance of the winning model differed according to age group. To do this, I used an independent samples t-

test of the percentage of trials on which simulated choice matched observed choice. I then repeated parameter fitting, using hierarchical MCMC sampling with young and mid-adolescents as two groups with separate priors. I tested whether this was a better model of task performance by using the BIC and AIC and simulating task choices, which I then compared to observed performance. Finally, I examined whether there was evidence that the parameters estimated separately for young and mid-adolescents differed across the two age groups using independent sample t-tests.

4.3 Results

4.3.1 Model development and validation

4.3.1.1 All model families

I first tested models with up to one start bias parameter and one positivity bias parameter, to determine which model type should be taken forward for further development. Comparisons using the BIC and AIC did not show consistent evidence for a winning model (Table 4.2). The winning model according to the BIC was the uniform model, with one learning rate and no additional bias parameters. According to the AIC, the condition model (separate learning rates for self-referential and other-referential blocks) with one start bias parameter was best. This inconsistency between the BIC and AIC probably occurred because the BIC penalises more complex models more heavily. It is likely that the BIC was over-penalising for additional parameters, as the simplest model had a substantially lower BIC. As the BIC and AIC were summed across participants, and sample size was large (n=598), relative BICs and AICs appeared very different, but this only represented a very small difference per participant.

Due to these limitations, and in order to consider both model parsimony and model falsification, I plotted the choice probabilities across trials generated from each model (Figure 4.2). In these plots, the uniform models did not provide a good model of behaviour (comparing Figure 4.2 to Figure 4.1). The condition and condition-valence models generated probabilities of positive choices most similar to the observed data. As the condition model family also had the lowest AIC, and second lowest BIC, this model was selected for further modification.

Table 4.2 Fit of initial models to the observed data using both the Bayesian Information Criterion (BIC) and Akaike Information Criterion (AIC).

Model	No. params	Total BIC	Relative BIC	Total AIC	Relative AIC
Uniform - α, β					
No bias parameters	2	**51759.02**	**0.00**	48910.12	200.53
1 start bias	3	53260.51	1501.49	48987.15	277.56
1 positivity bias	3	53270.15	1511.13	48996.79	287.20
1 start and 1 positivity bias	4	55007.91	3248.89	49310.10	600.51
Condition - α_{self}, α_{other}, β					
No bias parameters	3	52997.54	1238.52	48724.18	14.59
1 start bias	4	54407.40	2648.38	**48709.59**	**0.00**
1 positivity bias	4	54514.50	2755.48	48816.69	107.10
1 start and 1 positivity bias	5	55968.52	4209.50	48846.26	136.67
Valence - $\alpha_{positive}$, $\alpha_{negative}$, β					
No bias parameters	3	53238.48	1479.46	48965.13	255.54
1 start bias	4	54977.41	3218.39	49279.60	570.01
1 positivity bias	4	60684.20	8925.18	54986.39	6276.80
1 start and 1 positivity bias	5	59457.84	7698.82	52335.58	3625.99
Condition-valence - $\alpha_{self\ pos}$, $\alpha_{self\ neg}$, $\alpha_{other\ pos}$, $\alpha_{other\ neg}$, β					
No bias parameters	5	56048.36	4289.34	48926.10	216.51
1 start bias	6	57803.02	6044.00	49256.31	546.72
1 positivity bias	6	74662.27	22903.25	66115.55	17405.96
1 start and 1 positivity bias	7	103916.40	52157.38	93945.23	45235.64
Additional model checks					
Outcome [a]	3	53228.04	1469.02	48954.69	245.10
Condition-outcome [b]	5	56186.73	4427.71	49064.47	354.88
Feedback [c]	3	53579.75	1820.73	49306.39	596.80
Condition-feedback [d]	5	57046.22	5287.20	49923.96	1214.37

Note. N=598. α: learning rate. β: inverse temperature. See section 4.2.2 for details of how the total BIC and AIC were calculated. Relative BIC was calculated as the difference between that model and the lowest BIC (same procedure for AIC). Additional model checks (outcome and feedback models) were fitted as comparison models, so did not include the start and positivity bias parameters. For these models, included parameters were: [a] $\alpha_{pos\ outcome}$, $\alpha_{neg\ outcome}$, β. [b] $\alpha_{self\ pos\ outcome}$, $\alpha_{other\ pos\ outcome}$, $\alpha_{self\ neg\ outcome}$, $\alpha_{other\ neg\ outcome}$, β. [c] $\alpha_{pos\ feedback}$, $\alpha_{neg\ feedback}$, β. [d] $\alpha_{self\ pos\ feedback}$, $\alpha_{other\ pos\ feedback}$, $\alpha_{self\ neg\ feedback}$, $\alpha_{other\ neg\ feedback}$, β.

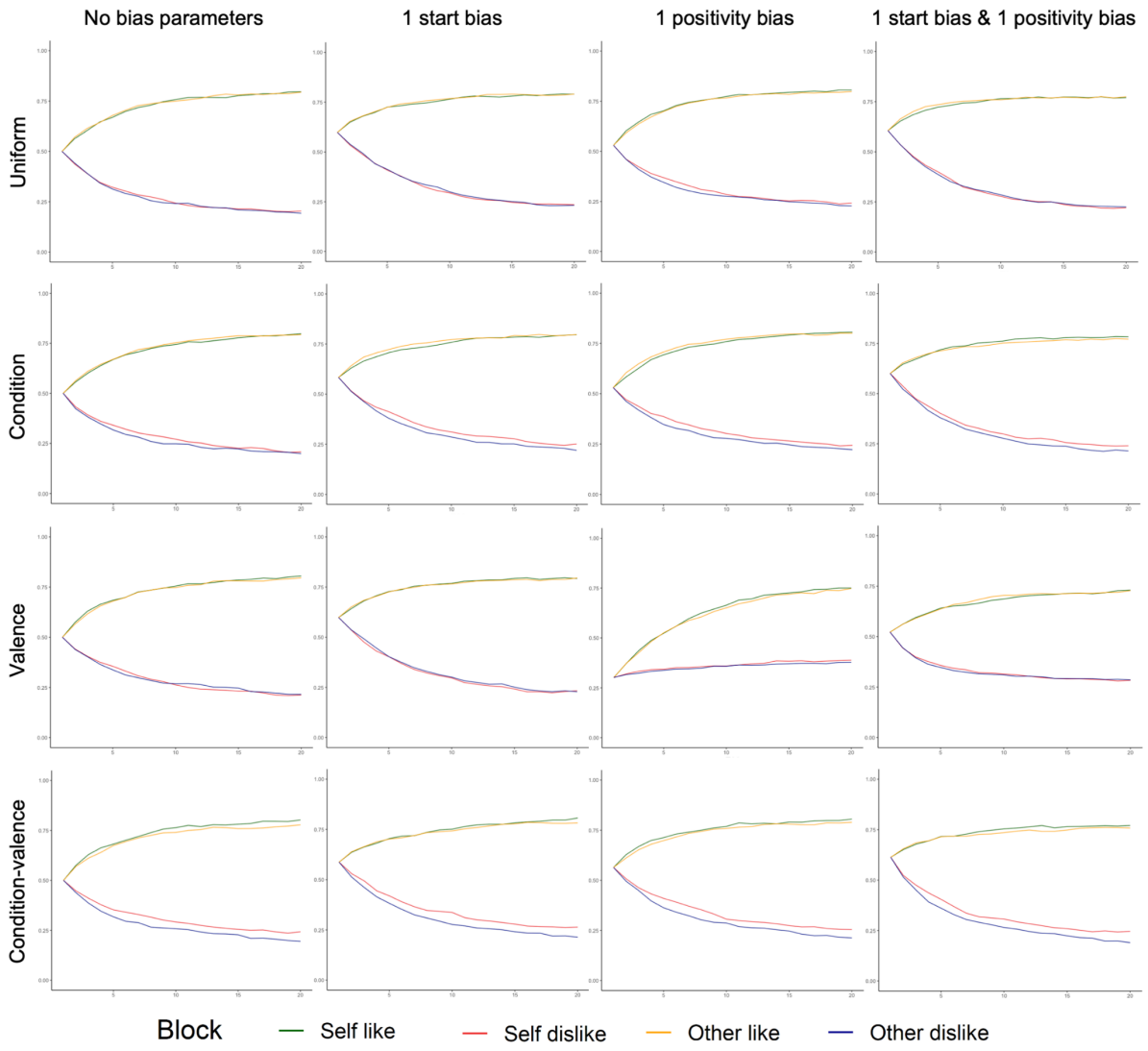

Figure 4.2 Probability of positive choices from the hypothesised initial models. In each plot, the X axis is the trial number (1 to 20) and the Y axis is the probability of a positive choice (0 to 1).

4.3.1.2 Condition models

I next considered several modifications to the condition models: decreasing learning rates, one or two positivity biases, and one or two start biases. When modelling the decreasing learning rates, dividing the learning rate by the trial number ($\frac{\alpha}{trial}$) did not fit observed performance well, and models had high BICs and AICs. Decreasing learning rates were therefore modelled using the trial number as an exponent (α^{trial}). Including two separate positivity biases for self-referential and other-referential blocks also led to poor model fit and parameter recovery, so I only included one overall positivity bias.

In this set of models, the winning model according to both the BIC and AIC had two learning rates (α_{self} and α_{other}) which decreased throughout each block (Table 4.3). However, plotting the probability of choosing the positive word from each model demonstrated that this model did not reproduce observed performance well (Figure 4.3). In these plots, models with two start bias parameters and decreasing learning rates best reproduced observed performance (Figure 4.3 versus Figure 4.1).

Table 4.3 Fit of condition models to the observed data using both the Bayesian Information Criterion (BIC) and Akaike Information Criterion (AIC).

Model	No. params	Total BIC	Relative BIC	Total AIC	Relative AIC
Fixed learning rates					
No bias parameters	3	53044.87	104.22	48771.52	104.22
1 start bias	4	54462.55	1521.90	48764.75	97.45
1 positivity bias	4	54551.31	1610.66	48853.50	186.20
1 start and 1 positivity bias	5	56040.85	3100.20	48918.59	251.29
2 start biases	5	56366.38	3425.73	49244.12	576.82
2 start and 1 positivity bias	6	58105.62	5164.97	49558.91	891.61
Decreasing learning rates					
No bias parameters	3	**52940.65**	**0.00**	**48667.30**	**0.00**
1 start bias	4	54503.01	1562.36	48805.20	137.90
1 positivity bias	4	54533.20	1592.55	48835.39	168.09
1 start and 1 positivity bias	5	56116.30	3175.65	48994.04	326.74
2 start biases	5	56579.87	3639.22	49457.61	790.31
2 start and 1 positivity bias	6	58322.39	5381.74	49775.68	1108.38

Note. N=598. See section 4.2.2 for details of how the total BIC and AIC were calculated. Relative BIC was calculated as the difference between that model and the lowest BIC (same procedure for AIC).

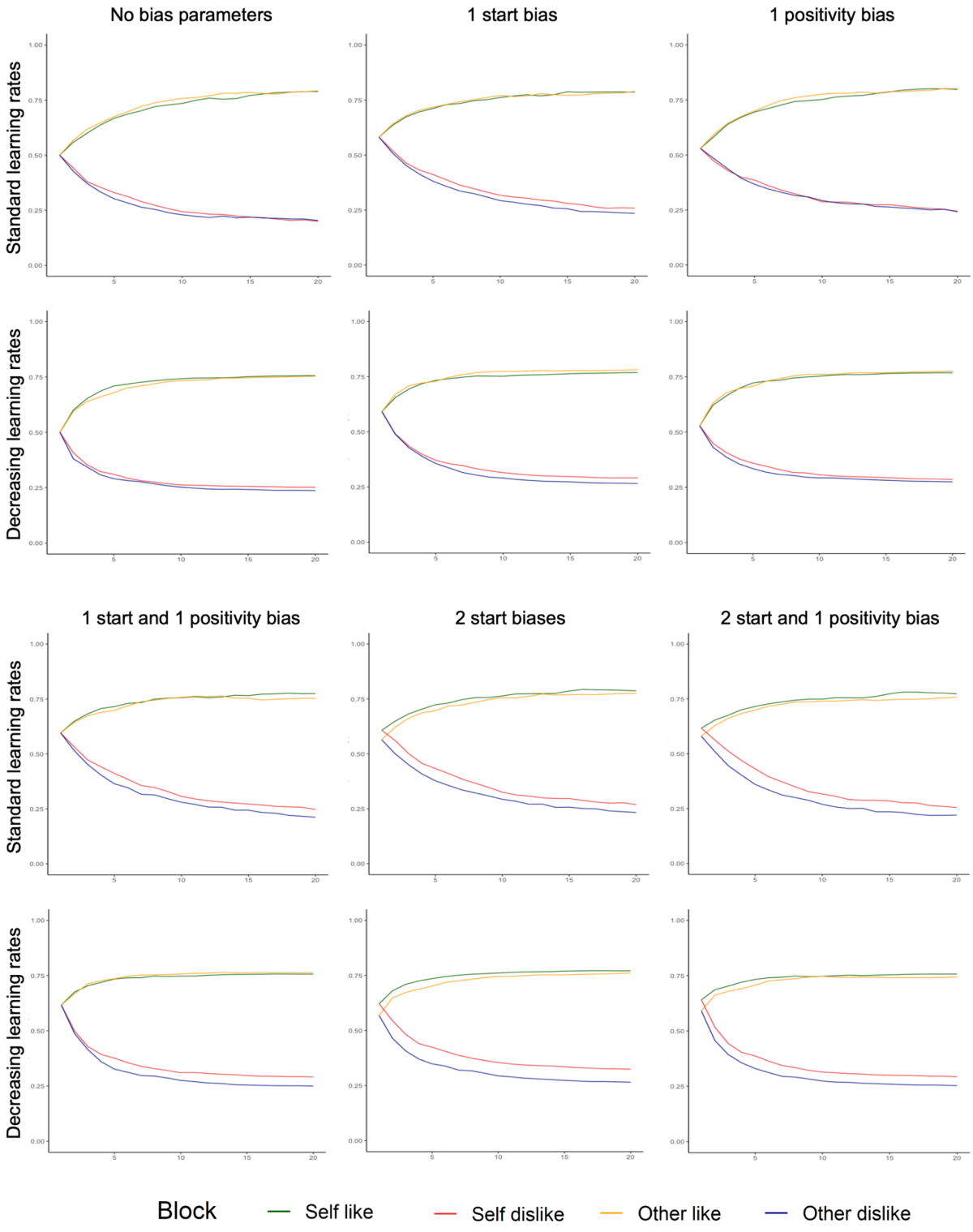

Figure 4.3 Probability of positive choices from all considered condition models, with separate learning rates for the self and other. In each plot, the X axis is the trial number (1 to 20) and the Y axis is the probability of a positive choice (0 to 1).

I also assessed parameter recovery, which was poor for the more complex models, particularly those with a positivity bias alongside start biases (Table 4.4). This poor recovery can be caused by including redundant parameters, or parameters which are highly correlated. Parameter recovery was best in models with fixed learning rates and one or two start biases or one positivity bias, as well as the two start bias model with decreasing learning rates (Table 4.4). At this stage, on the basis of all of the evidence (BIC, AIC, data visualisation, parameter recovery), I selected the three best models to examine generative performance. These models had separate learning rates for the self and other and either: one start bias, two start biases, or two start biases and decreasing learning rates.

Table 4.4 Parameter recovery for all considered condition models.

Model	Parameters	Parameter recovery
Fixed learning rates		
No bias parameters	α_{self}, α_{other}, β	0.56, 0.45, 0.75
1 start bias	α_{self}, α_{other}, β, γ	0.67, 0.7, 0.7, 0.74
1 positivity bias	α_{self}, α_{other}, β, δ	0.56, 0.7, 0.64, 0.76
1 start & 1 positivity bias	α_{self}, α_{other}, β, γ, δ	0.44, 0.44, 0.40, 0.59, 0.66
2 start biases	α_{self}, α_{other}, β, γ_{self}, γ_{other}	0.71, 0.58, 0.56, 0.69, 0.73
2 start & 1 positivity bias	α_{self}, α_{other}, β, γ_{self}, γ_{other}, δ	0.01, 0.17, 0.23, 0.39, -0.01, -0.19
Decreasing learning rates		
No bias parameters	α_{self}, α_{other}, β	0.61, 0.64, 0.57
1 start bias	α_{self}, α_{other}, β, γ	0.73, 0.57, 0.48, 0.58
1 positivity bias	α_{self}, α_{other}, β, δ	0.54, 0.47, 0.52, 0.68
1 start & 1 positivity bias	α_{self}, α_{other}, β, γ, δ	-0.07, -0.01, 0.31, 0.23, 0.05
2 start biases	α_{self}, α_{other}, β, γ_{self}, γ_{other}	0.7, 0.66, 0.37, 0.64, 0.65
2 start & 1 positivity bias	α_{self}, α_{other}, β, γ_{self}, γ_{other}, δ	-0.05, -0.03, 0.03, 0.11, -0.02, 0.03

Note. N=100. Parameter recovery is the correlation between the generated and recovered parameter estimates for 100 synthetic datasets. The parameters were: self-referential learning rate (α_{self}), other-referential learning rate (α_{other}), inverse temperature (β), self-referential start bias (γ_{self}), and other-referential start bias (γ_{other}).

4.3.2 Generative performance

In order to select the final model, I simulated task performance using the parameters produced by each model, in combination with each participants' condition, choices, and feedback, for the three chosen models. There was little difference in the models in this posterior predictive check, but the model with two start biases and decreasing learning rates performed best, correctly predicting 70.46% of participants' choices. For models with a stable learning rate, the model with two start biases predicted 70.22% of participants' choices correctly and the model with one start bias correctly predicted 69.62%.

Plotting the predicted choices on each trial also demonstrated that the model with decreasing learning rates and two start biases performed best, as it most closely resembled the observed data (Figure 4.4). The winning model therefore had separate decreasing learning rates for self-referential and other-referential blocks, an inverse temperature parameter, and separate start bias parameters for self-referential and other-referential blocks.

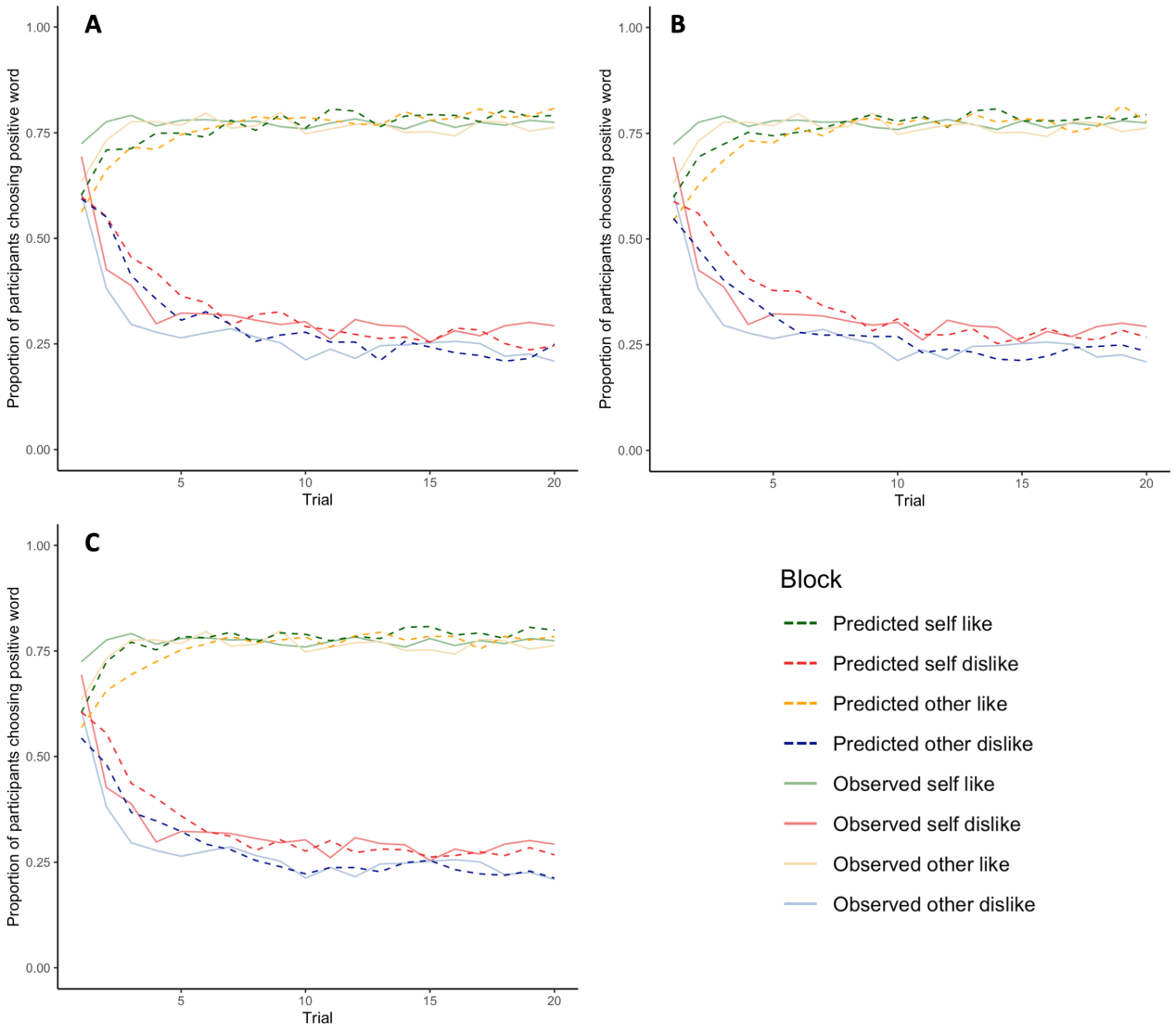

Figure 4.4 Observed and simulated task performance (using parameters from MAP), averaged across participants, for the three best-fitting models. A) Condition model with fixed learning rates and 1 start bias. B) Condition model with fixed learning rates and 2 start biases. C) Condition model with decreasing learning rates and 2 start biases.

I then refitted the winning model using sampling to obtain the best parameter estimates. In this model, the average learning rate for other-referential information (M=0.82, SD=0.11) was

larger than for self-referential information (M=0.77, SD=0.17). This indicates that, on average, participants updated their value estimates more for other-referential than self-referential feedback. Additionally, the average start bias on self-referential blocks (M=0.12, SD=0.07) was larger than the start bias for other-referential blocks (M=0.06, SD=0.07), indicating that participants had a larger initial positive bias when learning social evaluation about themselves than others. The inverse temperature parameter was relatively high (M=2.43, SD=0.75), suggesting that participants' choices were quite deterministic.

I then checked whether simulated data from the winning model reproduced the effects of interest found in standard regression analyses (chapter 3). I calculated the number of simulated positive responses on each block and repeated the regression analyses. As in the raw data, there was evidence that the number of positive choices differed according to condition and rule. There were 0.68 (95% CI=0.41 to 0.94, p<0.001) more positive choices in self-referential than other-referential blocks, and 9.62 (95% CI=-9.88 to -9.35, p<0.001) fewer positive choices in dislike compared to like rule blocks. There was also evidence for an interaction between condition and rule on positive choices (interaction p=0.002). As in the raw data, there was a smaller difference in the number of positive choices in self-referential dislike compared to like blocks (coef=-9.20, 95% CI=-9.59 to -8.81) than in other-referential blocks (coef=-10.04, 95% CI=-10.39 to -9.68). Also consistent with previous analyses, positive choices were not associated with age group (coef=0.10, 95% CI=-0.37 to 0.57, p=0.67) or gender (coef=0.08, 95% CI=-0.39 to 0.55, p=0.74). There was similar evidence that positive choices in the self like, self dislike, and other like blocks, but not other dislike, were negatively associated with depressive symptoms (self like coef=-0.19, 95% CI=-0.31 to -0.06, p=0.003; self dislike coef=-0.12, 95% CI=-0.22 to -0.01, p=0.03; other like coef=-0.12, 95% CI=-0.24 to 0.004, p=0.06; other dislike coef=-0.05, 95% CI=-0.17 to 0.07, p=0.41)

4.3.3 Associations between model parameters and age group, gender, and depressive symptoms

Using parameter estimates from the winning model, I tested whether any of the processes involved in learning differed with age or gender (hypothesis 2.2 and 2.3). In unadjusted analyses, there was evidence that the inverse temperature parameter increased with age (Table 4.5). It was 0.19 (95% CI=0.07 to 0.31, p=0.002) points higher in mid-adolescents than

young adolescents, indicating that older participants' choices were more deterministic. However, after adjusting for potential confounders, there was no evidence that this parameter was associated with age group (Table 4.5). This is a result of confounding by non-verbal IQ score, which was positively associated with the inverse temperature parameter (no other potential confounders were associated with this parameter). There was evidence that having higher non-verbal IQ was associated with making more deterministic choices. However, it is possible that I have adjusted for a variable on the causal pathway, as age is likely to be causally related to non-verbal IQ score. If so, I may be over adjusting and obscuring the association between age group and the inverse temperature parameter, with the estimate biased towards the null (Schisterman, Cole, & Platt, 2009).

Table 4.5 Linear regression models testing associations between age group, gender, and depressive symptoms (exposures) and model parameters (outcomes, tested in separate models).

	Unadjusted models		**Adjusted models**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Outcome: self-referential learning rate (α_{self})				
Age group	0.02 (-0.01 to 0.04)	0.25	-0.01 (-0.04 to 0.02)	0.70
Gender	0.02 (-0.01 to 0.05)	0.17	0.01 (-0.01 to 0.04)	0.30
Depressive symptoms	0.0005 (-0.002 to 0.003)	0.71	0.0002 (-0.002 to 0.003)	0.84
Outcome: other-referential learning rate (α_{other})				
Age group	0.01 (-0.003 to 0.03)	0.11	0.01 (-0.01 to 0.03)	0.26
Gender	0.02 (-0.002 to 0.04)	0.07	0.02 (-0.001 to 0.04)	0.06
Depressive symptoms	-0.001 (-0.003 to 0.0002)	0.09	-0.001 (-0.003 to 0.0002)	0.08
Outcome: inverse temperature (β)				
Age group	0.19 (0.07 to 0.31)	0.002	0.09 (-0.04 to 0.22)	0.18
Gender	0.07 (-0.05 to 0.19)	0.26	0.03 (-0.09 to 0.14)	0.66
Depressive symptoms	-0.01 (-0.02 to 0.003)	0.19	-0.01 (-0.02 to 0.002)	0.10
Outcome: self-referential start bias (γ_{self})				
Age group	-0.005 (-0.02 to 0.01)	0.42	-0.01 (-0.03 to 0.001)	0.07
Gender	0.01 (-0.01 to 0.02)	0.39	0.005 (-0.01 to 0.02)	0.42
Depressive symptoms	-0.002 (-0.003 to -0.001)	<0.001	-0.002 (-0.003 to -0.001)	<0.001
Outcome: other-referential start bias (γ_{other})				
Age group	0.001 (-0.003 to 0.004)	0.69	0.0001 (-0.004 to 0.004)	0.98
Gender	0.001 (-0.002 to 0.005)	0.52	0.001 (-0.003 to 0.005)	0.60
Depressive symptoms	-0.001 (-0.001 to -0.0002)	0.001	-0.001 (-0.001 to -0.0002)	0.001

Note. N=598. In unadjusted models, each exposure was tested in a separate univariable linear regression with the model parameter as the outcome. Each model was then adjusted for potential confounders (continuous age within each age group, school, testing group size, and non-verbal IQ score). Gender was missing for 10 participants and depressive symptoms (SMFQ score) were missing for 4 participants.

There was no evidence for associations between age group and any other parameters. Similarly, there was no evidence for associations between gender and any parameters (Table 4.5). There was also no evidence for an interaction between age group and gender on any parameters (adjusted interactions: α_{self} p=0.42; α_{other} p=0.81; β p=0.33; γ_{self} p=0.18; γ_{other} p=0.36).

I then examined whether any parameters were associated with depressive symptoms (hypothesis 2.4). There was no evidence that the learning rates or inverse temperature parameter were associated with depressive symptoms, before or after adjustment for confounders (Table 4.5). In contrast, there was strong evidence that both start bias parameters were negatively associated with depressive symptoms. For each one-point increase in SMFQ score, self-referential start bias decreased by 0.002 (95% CI=-0.003 to -0.001, p<0.001 adjusted for confounders). Similarly, for each one-point increase in SMFQ score, other-referential start bias was 0.001 lower (95% CI=-0.001 to -0.0003, p=0.001 adjusted for confounders). This suggests that adolescents with more severe depressive symptoms had a smaller start bias, meaning they were less likely to choose the positive word on early trials of both self- and other-referential blocks. This can be seen in the difference between the proportion of participants with low versus high depressive symptoms choosing the positive word on the first trial in Figure 4.1.

Next, I tested whether the associations between depressive symptoms and start biases differed according to gender or age group. There was no evidence for a two-way interaction between gender and depressive symptoms on either self-referential (adjusted interaction p=0.77) or other-referential (adjusted interaction p=0.96) start bias. There was also no evidence that the association between depressive symptoms and start biases differed according to age group (adjusted interactions: self-referential p=0.91; other-referential p=0.27). There was also no evidence that the associations between depressive symptoms and other-referential start bias differed according to gender and age group (three-way interaction between age group, gender, and depressive symptoms on γ_{other} p=0.47 adjusted for confounders).

However, there was some evidence that the association between self-referential start bias and depressive symptoms differed across genders and age groups (three-way interaction

between age group, gender, and depressive symptoms on γ_{self} p=0.04 adjusted for confounders). Examining this interaction, it appeared that the self-referential start bias parameter was more strongly associated with depressive symptoms for young adolescent males (coef=-0.003, 95% CI=-0.01 to -0.001) and mid-adolescent females (coef=-0.003, 95% CI=-0.005 to -0.001), compared to young adolescent females (coef=-0.001, 95% CI=-0.003 to 0.001) and mid-adolescent males (coef=-0.001, 95% CI=-0.004 to 0.002). However, coefficients for each subgroup were all in the same direction and did not differ substantially numerically.

4.3.4 Sensitivity analyses: effect of age group

There was some evidence that the final model better predicted the behaviour of mid-adolescents than young adolescents (mean diff=-1.79, 95% CI=-3.55 to -0.03, p=0.05), although the difference was not substantial. The model correctly predicted 68.11% (SD=11.47%) of young adolescents' choices, compared to 69.89% (SD=10.14%) of mid-adolescents' choices.

I re-estimated the final model with separate priors, allowing for different means and distributions of parameters in each age group. This marginally improved model fit (total BIC=57928.13, AIC=50805.87) compared to estimation with priors for the whole sample (total BIC=57976.42, AIC=50854.16). However, the model still better predicted the observed choices of mid-adolescents 70.56% (SD=9.87%) than young adolescents 68.33% (SD=11.65%) after simulating choices (mean diff=-2.23, 95% CI=-3.96 to -0.50, p=0.01).

Using separate priors for each age group, there was no evidence that estimates of the self-referential learning rate (t-test p=0.21) or other-referential start bias (t-test p=0.44) parameters were different in young versus mid-adolescents. However, there was evidence that the other-referential learning rate was lower in young adolescents (M=0.81, SD=0.13) than mid-adolescents (M=0.83, SD=0.11; mean diff=-0.02, 95% CI=-0.04 to -0.004, p=0.02). There was also evidence that the inverse temperate parameter was lower in young adolescents (M=2.33, SD=0.84) than mid-adolescents (M=2.58, SD=0.57; mean diff=-0.25, 95% CI=-0.37 to -0.13, p<0.001). Finally, there was evidence that the self-referential start bias parameter was higher in young adolescents (M=0.13, SD=0.08) than mid-adolescents (M=0.10, SD=0.06; mean diff=0.03, 95% CI=0.02 to 0.04, p<0.001). This indicates that in

comparison to young adolescents, mid-adolescents updated their value estimates more for other-referential feedback, made more deterministic choices, and had a less positive initial self-referential bias.

4.4 Discussion

In this study, I examined potential processes underlying learning about social evaluation in adolescence, investigating how social feedback influences learning and future decisions. To do this, I developed and validated a reinforcement learning model describing how adolescents learnt about social evaluation. As hypothesised (hypothesis 2.1), adolescents started with a positive expectation that both they and others would be liked, shown by their bias towards choosing the positive word on the first trial of each block. This positive bias was larger for the self than others (i.e. participants were more likely to initially choose the positive word in self-referential than other-referential blocks). During learning, adolescents used feedback to update their expectations about social evaluation more for others than for themselves. This suggests that adolescents have a positive self-referential bias in learning about social evaluation, which is more resistant to feedback than learning about others.

Consistent with hypothesis 2.4, I found evidence that adolescents with more severe depressive symptoms had a less positive start bias, meaning that they were less likely to choose the positive word when they first met each character (i.e. on the first trial of each block). This occurred both for learning both about the self and another person, and there was no evidence that the association with depressive symptoms differed according to gender or age group. Contrary to hypothesis 2.4, there was no evidence that other aspects of learning were associated with depressive symptoms. In contrast to my hypothesis that parameters relating to the positive self-referential bias would be smaller in girls than boys (hypothesis 2.3), and despite evidence for the association between start biases and depressive symptoms, there was also no evidence for gender differences in any parameters.

Finally, I did not expect any of these parameters to differ across age groups (hypothesis 2.2). Initially, there was evidence that mid-adolescents were more deterministic (less exploratory) in their choices as they got older, and this may be a result of increases in non-verbal IQ score. After modelling the age groups as two separate populations, with different priors, I found

evidence that mid-adolescents updated their estimates more for other-referential feedback, made more deterministic choices, and had a less positive initial self-referential bias than young adolescents.

4.4.1 Strengths and limitations

General strengths and limitations of the data and task included in this study were outlined in chapter 3 (section 3.4.1) and will be discussed in detail in the general discussion (section 7.3).

Computational psychiatry is growing in popularity as it allows the mathematical specification of potential mechanisms underlying task performance (Adams et al., 2016; Browning et al., 2020; Maia et al., 2017). I compared models representing assumptions about different mechanisms underlying behaviour. Parameters captured patterns which were not apparent in standard analyses (chapter 3), such as the differential learning from self-referential and other-referential feedback, and initial positive biases. However, there are some limitations of the winning model. Despite the use of decreasing learning rates, initial learning was still too slow in comparison to observed behaviour. Start biases from the model were smaller than in observed behaviour and, for like blocks, the asymptote of positive choices was a little high. The winning model was the best of those I tested but, like any model, it does not provide a perfect explanation of performance. There may also be other models, which I did not explore, that could fit the data better.

I used a hierarchical approach to parameter fitting. Using group level information when fitting individual parameters greatly improves estimates, minimising extreme or incorrect parameter assumptions (Daw, 2011; Valton et al., 2020). However, it also requires specification of the population structure of the data. For the main analyses, I assumed that adolescents represented a single population, but it is possible that young and mid-adolescents are two separate populations. These contrasting assumptions can affect parameter estimation, underestimating age differences when assuming one population distribution (Valton et al., 2020). In simulation studies, modelling groups separately provides a closer (although slightly overestimated) recovery of true effect sizes. However, simulation studies were based on diagnostic groups (patients versus controls; Valton et al., 2020), whose task performance may differ more substantially than adolescents aged 11 to 13 versus 13 to 15 years. Sensitivity analyses did indicate some age differences, including a larger inverse

temperature parameter in mid-adolescents. This may be due to differences in the extent to which the model accurately captured participants' learning (Palminteri et al., 2016). In addition to describing the extent to which behaviour is deterministic, inverse temperature parameters might capture noise in parameter estimates, leading to lower inverse temperatures when there is a larger mismatch between participants' performance and model algorithms (Palminteri et al., 2016). This could have occurred, as model simulations better predicted choices for mid-adolescents than young adolescents. A different model may be a better fit for young adolescents. Alternatively, differences may be due to some young adolescents not reading instructions or completing the task properly, leading to additional noise.

In this study, I decided against incorporating depressive symptoms into the model-fitting procedure (using clinically informed model fitting). This approach would have meant individual parameters were estimated by taking into account the population distribution of depressive symptoms (Moutoussis, Hopkins, & Dolan, 2018). Instead, I estimated model parameters and tested parameter-symptom associations separately. Clinically informed model fitting has been recommended to reduce Type 2 errors compared to using single group priors (Moutoussis et al., 2018). Although theoretically this approach should be superior, it is a new method and has relatively poor parameter recovery. In simulations, models using clinically informed priors were unable to recover even simple correlations in synthetic data (unpublished data but see Valton et al., 2020). My traditional approach is more conservative, so provides strong evidence for associations between initial positive biases and depressive symptoms.

The social evaluation learning task had a limited number of trials and blocks. I reduced the number of trials when adapting the task for use with adolescents in a classroom setting. From previous data and piloting, I knew that most learning occurs over the first five trials and performance then asymptotes. This is a result of the blocked design of the task. The low number of trials should therefore not have influenced reinforcement learning modelling. However, there was only one block of each condition (self like, self dislike, other like, other dislike) in this task. Start bias parameters were therefore estimated from only two trials per participant. However, I still found robust evidence for associations between start biases and depressive symptoms.

4.4.2 Findings in context

My winning model was similar to the reinforcement learning model of this task developed in adults as it had separate learning rates for self-referential and other-referential evaluation and an initial positive bias parameter (Hopkins et al., 2019). However, my model was simpler, as the previous model also included separate learning rates for positive or negative outcome words and a general positivity bias describing the value individuals put on positive information when choosing their actions (Hopkins et al., 2019). I did not find evidence to support including these parameters. In contrast to adults, there was no evidence that adolescents learnt differently from trials which had a positive or negative outcome or varied in the value they placed on positive information. Adolescents may learn about social evaluation more optimally than adults, learning equally well from positive and negative outcomes.

The lack of evidence for differential learning from positive and negative feedback also differs to previous evidence for a valence-induced bias, whereby adolescents learn preferentially from positive, relative to negative, feedback (Christakou et al., 2013; Palminteri et al., 2016; Van Den Bos et al., 2012). However, these studies did not use social reinforcement learning paradigms. One study of social reinforcement learning found lower learning rates for positive social feedback in adolescents than children and adults, but did not report whether learning from positive versus negative social feedback differed during adolescence (Jones et al., 2014). The highly rewarding nature of peer interactions during adolescence may increase the impact of all aspects of interactions, leading to equal learning from positive and negative feedback.

This study refined analyses of responses when learning about social evaluation (chapter 3). The association between positive responses and depressive symptoms was likely a result of participants' initial positive biases, believing that the computer character would like them and the other person. This is consistent with previous evidence that adolescents with higher self-esteem have a higher expectancy of being liked in another social reinforcement learning task (Will et al., 2020). The initial bias corresponds to predictions about whether peers will like or dislike you in previous behavioural studies (e.g. Caouette & Guyer, 2016; Gunther Moor et al., 2010; Guyer et al., 2008; Rodman et al., 2017; Somerville et al., 2010). I also found some evidence that this positive self-referential bias was smaller in mid-adolescents than young adolescents (c.f. Gunther Moor et al., 2010; Rodman et al., 2017). A less positive initial bias

when learning about social evaluation may therefore emerge alongside the increase in depressive symptoms later in mid-adolescence, rather than being a vulnerability for depression present from early adolescence (c.f. Bone et al., 2020). Longitudinal studies are required to test the direction of this association.

Contrary to my hypotheses, and some previous evidence (Will et al., 2020), I did not find any evidence that other aspects of learning were associated with depressive symptoms. This suggests that prior beliefs about social evaluation are most important, and impact learning by reducing adolescents' biases towards positive information. Also contrary to my hypotheses, there was no evidence that any aspects of learning about social evaluation were more negative in girls than boys during adolescence. This is consistent with studies which have found no evidence that expectations of, or reactions to, social evaluation differed between boys and girls during adolescence (Guyer, Caouette, et al., 2014; Guyer et al., 2012, 2009).

These findings have a number of implications. Cohort studies should start to include social reinforcement learning tasks, such as the social evaluation learning task, which is easy to administer online. This would allow longitudinal investigation of whether adolescents' learning does become less exploratory and more negative with age. If my findings are replicated in longitudinal studies, prevention or treatment strategies for depression could target reinforcement learning processes, such as adolescents' beliefs about what people will think of them and others, aiming to instil more positive biases. In a previous trial, an intervention which aimed to enhance reward-processing in adolescents reduced subsequent depressive symptoms (Rice et al., 2015). Additionally, my reinforcement learning model is well-suited for integration into tests of underlying neural activity. For example, a previous study found that expectations about receiving positive social feedback correlated with medial prefrontal cortex and ventral striatum activation, and adolescents had different neural activity to children and adults (Jones et al., 2014).

4.4.3 Conclusion

In the last two chapters, I have investigated learning about social evaluation in adolescence, examined potential underlying mechanisms, and tested whether these are associated with depressive symptoms. I hypothesised that adolescents would have a positive self-referential bias in learning, and this bias would decrease with increasing depressive symptoms. I found

evidence that adolescents have positive biases in learning what others think about both themselves and others, and reductions in these biases are associated with depressive symptoms. In contrast to my hypotheses, I have shown that there is no evidence for gender differences in learning about social evaluation. Finally, also inconsistent with my expectations, I found some evidence for developmental differences in learning about social evaluation. This included a decrease in positive self-referential biases with age, which could be associated with the increase in depressive symptoms in mid-adolescence. Future studies should examine these processes longitudinally and study developmental differences over a wider age range.

Chapter 5 Recall bias during adolescence: gender differences and associations with depressive symptoms

A modified version of this chapter was published in The Journal of Affective Disorders: Bone, J.K., Lewis, G., Roiser, J.P., Blakemore, S.-J., & Lewis, G. (2020) Recall bias during adolescence: gender differences and associations with depressive symptoms. *Journal of Affective Disorders.* https://doi.org/10.1016/j.jad.2020.12.133. See Appendix 2.

5.1 Introduction

Memory is an important aspect of social information processing. To date, research has investigated two main types of biases in memory, as outlined in section 1.5.2. There is strong evidence that over-general autobiographical memory biases are associated with depressive symptoms in adolescence, and may increase vulnerability to depression (Kuyken & Dalgleish, 2011; Lau & Waters, 2017; Rawal & Rice, 2012; Warne et al., 2019). In contrast, there is less consistent evidence for the role of biases in recalling positive and negative information about the self in adolescent depression (Lau & Waters, 2017; Platt et al., 2017). Therefore, in this study, I chose to focus on the latter form of bias in memory, self-referential recall biases.

Self-referential memory is often tested by presenting individuals with positive and negative personality characteristics and asking whether the words describe them (in a self-referential encoding task). This is followed by a surprise recall test in which participants are asked to remember as many characteristics as possible. Recall biases may be consistent with schema about the self, as information about the self is usually preferentially remembered compared to information about others (the self-reference effect; Rogers, Kuiper, & Kirker, 1977; Symons & Johnson, 1997). In recall tasks, healthy adolescents generally remember more positive than negative self-referential information, which may reduce their risk of depression (Auerbach et al., 2016; Cole et al., 2014; Connolly, Abramson, & Alloy, 2016; Dainer-Best, Lee, Shumake, Yeager, & Beevers, 2018; Fattahi Asl, Ghanizadeh, Mollazade, & Aflakseir, 2015; Hammen & Zupan, 1984; Kuiper & MacDonald, 1982; Prieto, Cole, & Tageson, 1992; Taylor & Ingram, 1999; Timbremont & Braet, 2004).

Self-referential recall may be particularly important in adolescence because the social self-concept develops during this period. Adolescents become more aware of, and concerned with, other people's opinions of them (Parker et al., 2006; Sebastian et al., 2008). Self-evaluations become more negative and self-esteem declines sharply, particularly in girls (Robins & Trzesniewski, 2005; van der Aar et al., 2018). Negative self-referential recall biases may lead to increased depressive symptoms in adolescence, and this risk factor may be more prevalent in girls (Bone et al., 2020). It is unclear whether this risk factor would be present from early adolescence or emerge during adolescence.

However, a recent review did not find strong evidence of reduced positive or increased negative recall biases in adolescents with depression (Platt et al., 2017). In an updated review (included in Appendix 5), I also found inconsistent evidence for associations between recall biases and depressive symptoms in adolescence. There was evidence that depressive symptoms are associated with poorer recall of positive information, greater recall of negative information, or a combination of both biases (Alloy et al., 2012; Asarnow, Thompson, Joormann, & Gotlib, 2014; Fattahi Asl et al., 2015; Gençöz, Voelz, Gençöz, Pettit, & Joiner, 2001; Orchard & Reynolds, 2018; Speed, Nelson, Auerbach, Klein, & Hajcak, 2016; Woolgar & Tranah, 2010). In contrast, others have found no evidence for an association between recall biases and depressive symptoms (Dainer-Best et al., 2018; Holt et al., 2016; Reid, Salmon, & Lovibond, 2006).

Additionally, very few studies have tested whether negative recall biases are more prevalent in girls during adolescence (Appendix 5). A longitudinal cohort study found evidence that girls had more positive recall than boys around age 13, but there were no gender differences in negative recall (McArthur et al., 2019). Changes in recall biases (from 13 to 19 years) did not differ according to gender. This study did not measure depressive symptoms so could not test whether they were associated with recall biases (McArthur et al., 2019).

It is also unclear whether recall biases change developmentally. Social information processing biases may emerge during adolescence due to development of cognitive abilities and increasing experiences of adversity with age (Cole et al., 2008; Jacobs, Reinecke, Gollan, & Kane, 2008; Turner & Cole, 1994). Some studies have found evidence that negative recall biases increase across adolescence (Cole & Jordan, 1995; McArthur et al., 2019; Neshat-

Doost, Taghavi, Moradi, Yule, & Dalgleish, 1998; Speed et al., 2016). However, others have found no evidence for changes with age (Holt et al., 2016; Taylor & Ingram, 1999).

In addition to the lack of research on gender differences in recall bias, and the inconsistent associations with age and depressive symptoms, previous studies have methodological limitations. Many have used small samples and divided participants into groups according to presence or absence of depressive symptoms or risk of depression (Appendix 5), which limits statistical power. It is generally accepted that depression is a continuum, ranging from mild to severe symptoms (Hankin et al., 2005). Using depressive symptoms continuously in analyses should increase the sensitivity to detect any associations with recall bias.

In this study, I addressed these issues by using a novel recall task in a cross-sectional study (n=578). I recruited adolescents from two age groups (young adolescents aged 11-13 years, mid-adolescents aged 13-15 years) to study recall biases before and after the gender difference in depression emerges. Depressive symptoms ranged from mild to severe. As findings with the traditional recall task are inconsistent, I developed a novel test of recall of social evaluation. Social evaluation was positive and negative personality traits, seen in a task where participants learned whether they or another person were liked or disliked. I examined whether recall differed according to whether words were seen describing the self (self-referential) or another person (other-referential) and word valence (positive/negative). My aims were to investigate recall of self-referential and other-referential social evaluation, examine whether there are gender differences in this recall, explore whether these gender differences change with age, and test whether recall of social evaluation is associated with depressive symptoms in adolescence.

I hypothesised that, overall, adolescents would have a positive self-referential bias, recalling more self-referential than other-referential words (hypothesis 3.1). I also hypothesised that adolescents' self-referential bias would be positive, as demonstrated by recall of more self-referential positive than self-referential negative words (hypothesis 3.2). I hypothesised that girls would demonstrate less positive self-referential recall biases than boys, recalling fewer self-referential positive and more self-referential negative words (hypothesis 3.3). I hypothesised that this gender difference in recall biases would be present from early adolescence, so would not differ across age groups (hypothesis 3.4). I also hypothesised that

self-referential recall biases would be associated with depressive symptoms (hypothesis 3.5). Specifically, I predicted that self-referential positive recall would be negatively associated with depressive symptoms, and self-referential negative recall would be positively associated with depressive symptoms. Finally, I hypothesised that the association between self-referential recall biases and depressive symptoms would be consistent across genders and age groups (hypothesis 3.6).

5.2 Methods

5.2.1 Participants

Data on depressive symptoms and/or the recall task was missing for 21 (4%) participants (final n=578). See section 2.9.4 for an overview of all missing data in this study.

5.2.2 Measures

5.2.2.1 Incidental recall

I assessed incidental memory using my novel surprise recall test (described in section 2.3.2). This differed to standard self-referential recall tasks which present participants with personality characteristics, ask whether they describe the self, and then often only measure recall of words originally classified as self-referential. In contrast, in this novel task, I tested recall of all personality descriptors previously seen as social evaluation in a learning task. This method allowed me to differentiate recall of self-referential and other-referential information from social interactions. I could thus test if recall of all social evaluation was associated with gender and depressive symptoms, or whether associations were specific to self-referential information.

I asked participants to remember as many of the personality descriptors presented in the social evaluation learning task (completed approximately 4mins earlier) as they could. They were given 2mins to perform this free recall task, typing responses on the computer. A countdown timer appeared when participants had 30s remaining. Any misspelled words that resembled correct responses were recorded as correct to ensure that spelling errors did not bias accuracy rates. The number of self-referential and other-referential positive and negative words accurately recalled (hits), and the number of positive and negative incorrect responses (false alarms) were calculated.

5.2.2.2 Depressive symptoms

The Mood and Feelings Questionnaire (short version; SMFQ) measured depressive symptoms over the last two weeks (Angold et al., 1995). I replaced missing responses using person-mean imputation (see section 2.9.3) for those who responded to 10 or more questions using each individual's mean SMFQ score (111 participants, 19% of this sample).

5.2.2.3 Confounders

Participants completed an abbreviated nine-item version of the Raven Standard Progressive Matrices Test (non-verbal IQ score; Bilker et al., 2012). Additional potential confounders were collected through a parental questionnaire (ethnicity, English as a first language, dyslexia, ASD, parental education, maternal depression, paternal depression). I also intended to include pubertal stage as a confounder, for the same reasons outlined in chapter 3 (section 3.2.2.3). Briefly, pubertal stage is associated with depressive symptoms and may be associated with social information processing, with potentially dissociable effects to age (Angold et al., 1998; Goddings et al., 2012; Ke et al., 2018). Following classroom data collection, participants were emailed the Pubertal Development Scale (PDS; Petersen et al., 1988) to complete at home. Participants were divided into two groups: early/mid puberty (Tanner stages 1-3) and late/post puberty (Tanner stages 4-5). Girls in the early group were pre-menarche and girls in the late group were post-menarche. Boys in the early group had low individual ratings on growth of body hair, voice change, and growth of facial hair growth compared to boys in the late group.

5.2.3 Procedure

After providing informed assent, participants completed the social evaluation learning task, followed by the measure of non-verbal IQ as a distractor task, and then the surprise recall task. They then completed the SMFQ. After classroom data collection, participants were sent a link to complete the PDS. See chapter 2 for a more detailed description of study methods.

5.2.4 Statistical analyses

I performed analyses using Stata 16 (StataCorp, 2019). As my sample consisted of two age groups, and one of my aims was to investigate the influence of gender in each group, I have presented all descriptive statistics separately according to gender for each age group. At this

stage, there was no evidence that false alarms differed according to age group or gender, and false alarms were not associated with depressive symptoms, I did not analyse them further (Table 5.1).

5.2.4.1 Negative binomial mixed models

There were four types of hits: self-referential positive, self-referential negative, other-referential positive, and other-referential negative. In order to analyse such data, analysis of variance (ordinary least squares, under Gaussian assumptions) or linear regression would often be used, testing whether number of hits differed according to various factors. However, given that hits were count variables which were positively skewed and over-dispersed, negative binomial mixed models were more appropriate. The four categories of hits were clustered within each individual, with the total number of hits as the outcome, and a random intercept for participant to account for this clustering. The task conditions (self-/other-referential, positive/negative), demographic variables of interest (age group, gender) and potential confounders (continuous age within each age group, school, testing group size, non-verbal IQ score, and positive and negative false alarms) were estimated as fixed effects. I adjusted for false alarms to account for potential biases in participants' responses. Participants could have guessed lots of words, which could make them erroneously appear to have correctly recalled more words.

I used the negative binomial mixed models to calculate a hits ratio as the effect estimate, which represents the number of hits in one category relative to another (e.g. the ratio of negative to positive hits). A hits ratio larger than one meant that hits were lower in the reference category (e.g. more negative than positive hits). All models are presented before and after adjustment for confounders.

5.2.4.2 Recall biases

My first question was whether hits differed according to word valence (positive/negative) and the condition in which words were learned (self-referential/other-referential; hypothesis 3.1). I included condition and valence as exposures with hits as the outcome. Next, I added an interaction between condition and valence, to test whether the association between valence and recall differed for self-referential versus other-referential words (hypothesis 3.2).

5.2.4.3 Gender differences

Next, I examined gender differences in recall (hypothesis 3.3). I tested a three-way interaction between gender, condition and valence with hits as the outcome, and also report the two-way interactions between these variables. To assess whether gender differences were consistent across age groups, I tested a four-way interaction between age group, gender, condition and valence with hits as the outcome (hypothesis 3.4). As my aim was to compare the influence of gender in each age group, I only report the lower level (two-way and three-way) interactions which include gender. Where there was evidence of an interaction, I examined associations with hits separately for each subgroup.

5.2.4.4 Associations with depressive symptoms

My third question was whether recall was associated with depressive symptoms (hypothesis 3.5). Linear regression was used to test whether self-referential positive, self-referential negative, other-referential positive, and other-referential negative hits were associated with depressive symptoms (SMFQ score; continuous outcome). For this analysis, all task parameters (i.e. all types of hits) were included in a single model to adjust for overall performance. This model was adjusted for age group and gender in addition to other confounders.

For each type of hit associated with depressive symptoms, I tested whether the association differed according to age group and gender (hypothesis 3.6). I added a three-way interaction between hits, age group, and gender to the linear regression model with depressive symptoms as the outcome. I also included two-way interactions between hits and age group, and hits and gender.

5.2.4.5 Sensitivity analyses: depressive symptoms

To check that gender differences in recall were not explained by depressive symptoms, I added SMFQ score to the negative binomial mixed models testing associations between age group, gender and recall. I first tested a three-way interaction between gender, condition and valence with hits as the outcome, and also report the two-way interactions between these variables. I then tested a four-way interaction between age group, gender, condition and valence with hits as the outcome, and report the two-way and three-way interactions between these factors. As my aim was to compare the influence of gender in each age group,

I only report the interactions which include gender. I adjusted all models for depressive symptoms, condition, valence, continuous age within each group, school, testing group size, non-verbal IQ score, and positive and negative false alarms

5.2.4.6 Sensitivity analyses: additional confounders

I asked all parents/carers to complete my parental questionnaire, but response rates were low (n=340, 59%). I first explored the distribution of these potential confounders by age group and tested whether they were associated with task performance and depressive symptoms. I then repeated primary analyses for the subsample whose parents/carers completed the questionnaire. Results are presented before and after controlling for the additional confounders (ethnicity, English as a first language, dyslexia, ASD, parental education, maternal depression, paternal depression) in this subsample. I used negative binomial mixed models testing the associations between condition, valence, age group, and gender (exposures) on hits (outcome). I also tested associations between the four types of hits (exposures) and depressive symptoms (outcome) in the linear regression models.

5.2.4.7 Sensitivity analyses: pubertal stage

Only 117 (20%) participants completed the PDS. I first explored whether pubertal stage was associated with task performance and depressive symptoms. I then repeated all primary analyses in this subsample, with and without adjusting for pubertal stage. Negative binomial mixed models would not converge when pubertal stage was included (likely due to overspecification in the reduced sample size) so I used Poisson mixed models.

Standardised estimates for all of the above analyses are included in Appendix 1.

5.3 Results

My final sample consisted of 578 adolescents (49% female). Of these, 315 (54%) were young adolescents from Year 7, and 263 (46%) were mid-adolescents from Years 9-10. Young adolescents' ages ranged from 11 to 13 years (M=11.56, SD=0.50) and mid-adolescents' ages ranged from 13 to 15 years (M=14.18, SD=0.51). Table 5.1 shows sample characteristics and task performance according to age group and gender.

In young adolescents, SMFQ score ranged from 0 to 23 (M=7.14, SD=5.49). The SMFQ threshold for depression was met by 60 (18%; 58% of whom were female) young adolescents. In mid-adolescents, SMFQ score ranged from 0 to 26 (M=8.27, SD=5.86). The SMFQ threshold for depression was met by 61 (23%; 67% of whom were female) mid-adolescents.

There was evidence that depressive symptoms were higher in mid- than young adolescents (coef=1.13, 95% CI=0.20 to 2.05, p=0.02), and depressive symptoms were higher in females than males (coef=2.22, 95% CI=1.30 to 3.15, p<0.001). There was no evidence of an interaction between age group and gender on depressive symptoms (interaction p=0.10). Although the evidence for this interaction missed statistical significance (p=0.05), I conducted the planned linear contrasts because of my a priori hypotheses. As predicted, depressive symptoms were higher in females in both age groups, and the gender difference was larger in the older group (young adolescents coef=1.47, 95% CI=0.23 to 2.70, p=0.02; mid-adolescents coef=3.03, 95% CI=1.65 to 4.41, p<0.001).

Table 5.1 Demographic characteristics and task performance of adolescents who completed the recall task.

	Young adolescents		**Mid-adolescents**		**Overall**	
	Male (n=159)	**Female** (n=146)	**Male** (n=128)	**Female** (n=134)	**Skewness** (n=582)	**Kurtosis** (n=582)
	Mean (SD)				Statistic	
Age (years)	11.56 (0.50)	11.56 (0.51)	14.20 (0.47)	14.17 (0.56)	0.16	1.54
Non-verbal IQ	3.94 (2.02)	4.42 (1.87)	4.80 (2.03)	4.96 (2.05)	-0.08	2.21
Dep symptoms	6.47 (5.06)	7.94 (5.90)	6.69 (4.93)	9.73 (6.30)	0.95	3.36
Recall task performance						
Self-ref pos hits	2.03 (1.60)	2.05 (1.83)	2.38 (1.71)	2.95 (2.01)	0.83	3.50
Self-ref neg hits	2.32 (1.82)	2.34 (1.82)	2.54 (1.72)	3.13 (1.91)	0.47	2.66
Other-ref pos hits	1.74 (1.45)	2.10 (1.71)	2.00 (1.61)	2.62 (1.73)	0.69	3.17
Other-ref neg hits	1.95 (1.64)	2.60 (1.94)	2.45 (1.67)	2.96 (1.85)	0.56	2.86
Pos false alarms	0.55 (0.89)	0.64 (0.93)	0.69 (0.99)	0.70 (0.94)	1.69	6.07
Neg false alarms	0.67 (1.05)	0.64 (1.03)	0.59 (0.80)	0.71 (1.01)	2.03	8.47

Note. Young adolescents were recruited from Year 7 (11-13 years old) and mid-adolescents were recruited from Years 9-10 (13-15 years old). Age in years was missing for n=2 young adolescents. Gender was missing for n=10 young adolescents and n=1 mid-adolescent.

5.3.1 Recall biases

Overall, there was evidence that participants made 14% more negative than positive hits (95% CI=1.08 to 1.20, p<0.001 adjusted for confounders). Additionally, there was evidence that females made 18% (95% CI=1.09 to 1.28, p<0.001 adjusted for confounders) more hits than

males and mid-adolescents made 43% (95% CI=1.13 to 1.81, p=0.003 adjusted for confounders) more hits than young adolescents.

Hypothesis 3.1: Adolescents would recall more self-referential than other-referential words

There was evidence for a self-referential bias. Participants made 7% (95% CI=1.02 to 1.13, p=0.01 adjusted for confounders) more self-referential than other-referential hits.

Hypothesis 3.2: Adolescents would recall more self-referential positive than self-referential negative words

There was no evidence for a positive self-referential bias (adjusted interaction between valence and condition p=0.25). Participants made more negative than positive hits in both self-referential (hits ratio=1.10, 95% CI=1.02 to 1.19, p=0.01 adjusted for confounders) and other-referential (hits ratio=1.18, 95% CI=1.09 to 1.27, p<0.001 adjusted for confounders) conditions.

5.3.2 Gender differences

Hypothesis 3.3: Girls would demonstrate less positive self-referential recall biases than boys

There was evidence for a two-way interaction between gender and condition (adjusted interaction p=0.04). Males made more self-referential than other referential hits, whereas females did not show this self-reference effect (Table 5.2). There was no evidence for a two-way interaction between gender and valence (adjusted interaction p=0.99), as both males and females made more negative than positive hits (Table 5.2). There was also no evidence for a three-way interaction between gender, condition, and valence on hits (adjusted interaction p=0.87; Table 5.2).

Table 5.2 Negative binomial mixed models testing associations between age group, gender, condition (self-/other-referential) and valence (positive/negative; exposures) and total hits (outcome).

	Unadjusted models (n=567)		**Adjusted models** (n=566)		**Additionally adjusted for depressive symptoms** (n=566)	
	Interaction p value	Subgroups hits ratio (95% CI)	Interaction p value	Subgroups hits ratio (95% CI)	Interaction p value	Subgroups hits ratio (95% CI)
Gender x condition	**0.03**		**0.04**		**0.04**	
Males: condition		1.14 (1.05 to 1.23)		1.14 (1.05 to 1.23)		1.14 (1.05 to 1.23)
Females: condition		1.02 (0.94 to 1.09)		1.02 (0.94 to 1.09)		1.02 (0.94 to 1.09)
Gender x valence	**1.00**		**0.99**		**0.99**	
Males: valence		1.14 (1.05 to 1.23)		1.14 (1.05 to 1.23)		1.14 (1.05 to 1.23)
Females: valence		1.14 (1.06 to 1.22)		1.14 (1.06 to 1.22)		1.14 (1.06 to 1.22)
Gender x condition x valence	**0.88**		**0.87**		**0.87**	
Males self-ref: valence		1.11 (1.00 to 1.24)		1.11 (1.00 to 1.24)		1.11 (1.00 to 1.24)
Males other-ref: valence		1.17 (1.04 to 1.31)		1.17 (1.04 to 1.31)		1.17 (1.04 to 1.31)
Females self-ref: valence		1.10 (0.99 to 1.22)		1.10 (0.99 to 1.22)		1.10 (0.99 to 1.22)
Females other-ref: valence		1.18 (1.06 to 1.31)		1.18 (1.06 to 1.31)		1.18 (1.06 to 1.31)
Age group x gender	**0.22**		**0.10**		**0.13**	
Young: gender		1.13 (0.99 to 1.29)		1.07 (0.95 to 1.20)		1.04 (0.93 to 1.17)
Mid: gender		1.26 (1.12 to 1.41)		1.25 (1.12 to 1.40)		1.26 (1.12 to 1.41)
Age group x gender x condition	**0.05**		**0.05**		**0.05**	
Young males: condition		1.18 (1.05 to 1.31)		1.18 (1.05 to 1.31)		1.18 (1.05 to 1.31)
Young females: condition		0.93 (0.83 to 1.04)		0.93 (0.83 to 1.04)		0.93 (0.83 to 1.04)
Mid males: condition		1.10 (0.99 to 1.24)		1.10 (0.99 to 1.24)		1.10 (0.99 to 1.24)
Mid females: condition		1.09 (0.99 to 1.20)		1.09 (0.99 to 1.20)		1.09 (0.99 to 1.20)
Age group x gender x valence	**0.42**		**0.43**		**0.43**	
Young males: valence		1.13 (1.02 to 1.27)		1.14 (1.02 to 1.27)		1.14 (1.02 to 1.27)
Young females: valence		1.19 (1.06 to 1.33)		1.19 (1.06 to 1.33)		1.19 (1.06 to 1.33)
Mid males: valence		1.14 (1.02 to 1.28)		1.14 (1.02 to 1.28)		1.14 (1.02 to 1.28)
Mid females: valence		1.10 (0.99 to 1.21)		1.10 (0.99 to 1.21)		1.10 (0.99 to 1.21)
Age group x gender x condition x valence	**0.43**		**0.42**		**0.42**	

Note. All models included condition and valence. Adjusted models also adjusted for continuous age within each age group, school, testing group size, non-verbal IQ score, and positive and negative false alarms. Additionally adjusted models also adjusted for depressive symptoms. For gender, male was the reference group. For condition, other-referential was the reference group. For valence, positive was the reference group. Self-ref: self-referential. Other-ref: other-referential. Young: young adolescent. Mid: mid-adolescent.

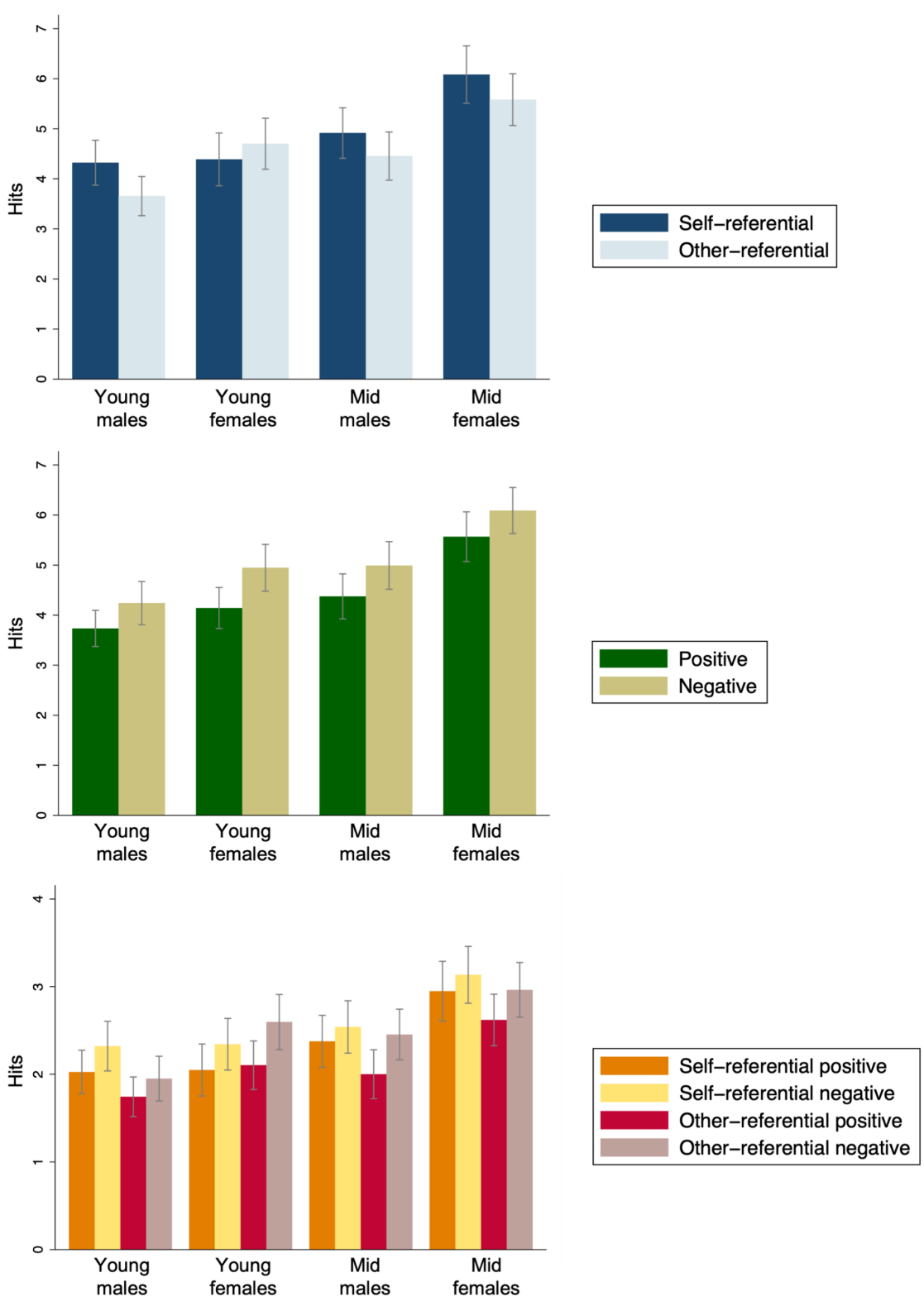

Figure 5.1 A) Three-way interaction between age group, gender, and word condition (self-referential or other-referential) on hits, collapsed across word valence. B) Three-way interaction between age group, gender, and word valence (positive or negative) on hits, collapsed across word condition. C) Four-way interaction between age group, gender, condition and valence on hits. All plotted using raw data with 95% confidence intervals. Young: young adolescents. Mid: mid-adolescents.

Hypothesis 3.4: The gender difference in positive self-referential recall biases would not differ across age groups

Next, I tested whether the gender differences in recall differed across age groups. There was no evidence for a two-way interaction between age group and gender on hits (adjusted interaction p=0.10; Table 5.2). In both age groups, females made more hits than males (Table 5.2). However, there was weak evidence for a three-way interaction between age group, gender and condition (adjusted interaction p=0.05). Young adolescent females made slightly fewer self-referential than other-referential hits but, in all other groups, more self-referential than other-referential hits were made (Table 5.2; Figure 5.1). There was no evidence that the number of positive versus negative hits differed according to age group and gender (adjusted three-way interaction p=0.43). All groups made more negative than positive hits (Table 5.2; Figure 5.1). Finally, there was no evidence of a four-way interaction between age group, gender, condition and valence (adjusted interaction p=0.42; Figure 5.1)

5.3.3 Associations with depressive symptoms

Hypothesis 3.5: Positive self-referential recall biases would be negatively associated with depressive symptoms

There was evidence for an association between positive and negative self-referential hits and depressive symptoms (Table 5.3; Figure 5.2). For each additional self-referential negative hit, SMFQ score increased by 0.45 points (95% CI=0.15 to 0.75, p=0.003 adjusted for confounders). In contrast, self-referential positive hits were negatively associated with depressive symptoms. For each additional self-referential positive hit, SMFQ score decreased by 0.38 points (95% CI=-0.69 to -0.08, p=0.01 adjusted for confounders).

Before adjusting for confounders, there was evidence that other-referential negative hits were positively associated with depressive symptoms (coef=0.34, 95% CI=0.05 to 0.62, p=0.02). However, this no longer achieved significance after adjustment for confounders (coef=0.24, 95% CI=-0.05 to 0.54, p=0.11 adjusted for confounders). There was no evidence that other-referential positive hits were associated with depressive symptoms (Table 5.3; Figure 5.2).

Table 5.3 Linear regression models testing change in depressive symptoms (SMFQ score; outcome) for each additional self-referential positive, self-referential negative, other-referential positive, and other-referential negative hit (exposures).

	Model 1: Unadjusted (n=578)			**Model 2: Adjusted (n=566)**		
	Coef	95% CI	p value	Coef	95% CI	p value
Self-referential hits						
Positive	-0.34	-0.63 to -0.04	0.02	-0.38	-0.69 to -0.08	0.01
Negative	0.47	0.17 to 0.76	0.002	0.45	0.15 to 0.75	0.003
Other-referential hits						
Positive	0.10	-0.23 to 0.41	0.57	0.04	-0.28 to 0.37	0.79
Negative	0.34	0.05 to 0.62	0.02	0.24	-0.05 to 0.54	0.11

Note. Both models included all four types of hits as exposures. Model 2 was adjusted for age group, gender, continuous age within each age group, school, testing group size, non-verbal IQ score, and positive and negative false alarms.

Hypothesis 3.6: The association between positive self-referential recall biases and depressive symptoms would be consistent across genders and age groups

Associations between self-referential hits and depressive symptoms did not differ across age groups (adjusted interactions: positive hits p=0.57; negative hits p=0.41). The association between self-referential positive hits and depressive symptoms also did not differ according to gender (adjusted interaction p=0.47). However, the association between self-referential negative hits and depressive symptoms was larger in females (coef=0.85, 95% CI=0.36 to 1.34 adjusted for confounders) than in males (coef=0.27, 95% CI=-0.13 to 0.67 adjusted for confounders). There was weak evidence for this interaction between gender and self-referential negative hits (adjusted interaction p=0.04). This gender difference in the association between self-referential negative hits and depressive symptoms was present across age groups. There was no evidence for three-way interactions between age group, gender and self-referential hits on depressive symptoms (adjusted interactions: positive hits p=0.52; negative hits p=0.30).

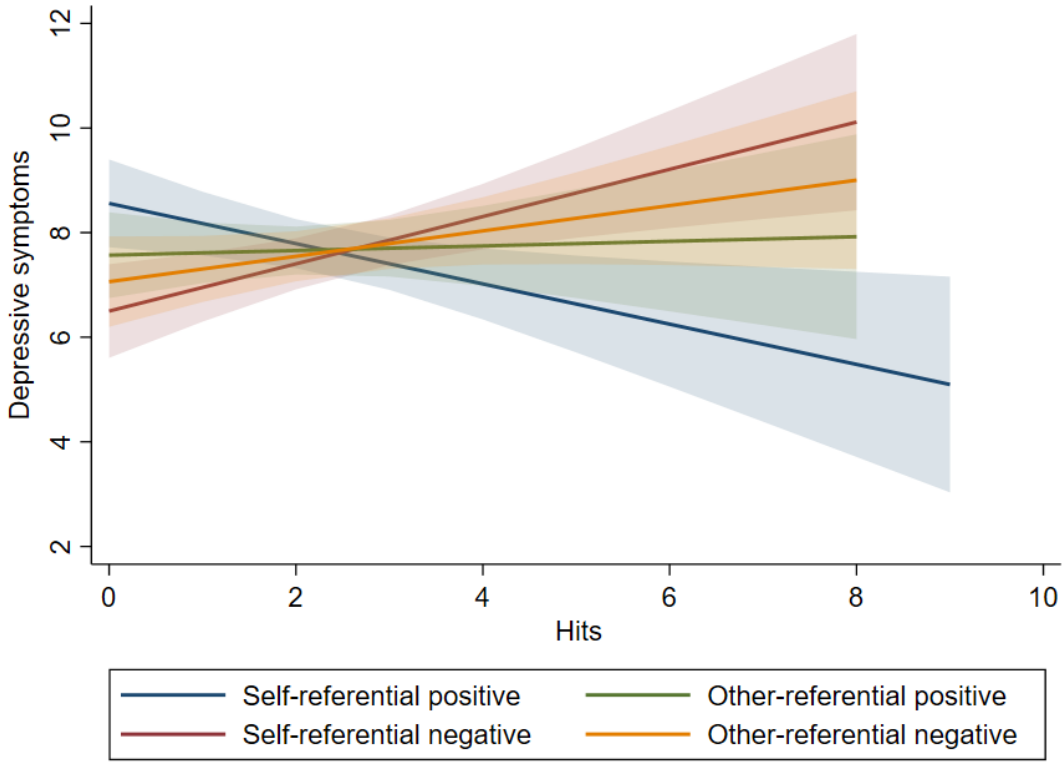

Figure 5.2 Expected depressive symptoms (SMFQ score) from the fully adjusted linear regression model for each type of hit (adjusted for age group, gender, continuous age within each age group, school, testing group size, non-verbal IQ score, and positive and negative false alarms). Plotted for range of hits achieved. Shading shows 95% confidence intervals.

5.3.4 Sensitivity analyses: depressive symptoms

I then added depressive symptoms to the original models testing gender differences in recall. This did not alter the evidence for any of the associations between condition, valence, gender, age group and hits (Table 5.2).

5.3.5 Sensitivity analyses: additional confounders

Demographic information was only available for a subsample of participants (n=340, 59%; Table 5.4). In this subsample, 78% were of white ethnicity and 89% had English as their first language. According to parents, 6% of the subsample had a mental health problem, with 1% reporting depression. Parents/carers reported that 8% of participants had used mental health services and 1% of participants were currently receiving psychological therapy for depression. Only 1 participant (0%) was currently using antidepressants according to parental report. In total, 14% of parents/carers reported that their child had special educational needs and disabilities, with 7% reporting dyslexia and 1% reporting ASD. Parent education was high for

88% of the subsample. In terms of mental health, 44% of mothers and 34% of fathers had experienced depression, anxiety or stress; 16% of mothers and 9% of fathers had depression specifically.

The two age groups were similar except that mid-adolescents had higher parent-reported rates of mental health problems, use of mental health services, and receipt of psychological therapy (Table 5.4).

Table 5.4 Characteristics of the subsample with data on additional confounders according to age group.

	Young adolescents	Mid-adolescents	Overall
	N (%)		
Ethnicity			
White	150 (79%)	115 (77%)	265 (78%)
Mixed	9 (5%)	16 (11%)	25 (7%)
Asian/Asian British	5 (3%)	2 (1%)	7 (2%)
Black/Black British	7 (4%)	3 (2%)	10 (3%)
Other	18 (10%)	13 (9%)	31 (9%)
Total n	*189*	*149*	*338*
English as first language	168 (90%)	130 (88%)	298 (89%)
Total n	*186*	*148*	*334*
Mental health problem	6 (3%)	13 (9%)	19 (6%)
Total n	*190*	*149*	*339*
Used mental health services	12 (6%)	16 (11%)	28 (8%)
Total n	*190*	*149*	*339*
Receiving psychological therapy	0 (0%)	5 (3%)	5 (1%)
Total n	*190*	*150*	*340*
Special educational needs and disabilities	24 (13%)	23 (16%)	47 (14%)
Total n	*188*	*148*	*336*
High parental education	159 (88%)	122 (88%)	281 (88%)
Total n	*180*	*139*	*319*
Maternal depression anxiety or stress	78 (44%)	64 (45%)	142 (44%)
Total n	*177*	*143*	*320*
Paternal depression anxiety or stress	54 (34%)	42 (34%)	96 (34%)
Total n	*161*	*123*	*284*
Pubertal stage			
Early	48 (84%)	13 (22%)	61 (52%)
Late	9 (16%)	47 (78%)	56 (48%)
Total n	*57*	*60*	*117*

Note. Mental health problem denotes whether participant had ever been diagnosed with any anxiety, conduct disorder, depression, eating disorder, generalized anxiety disorder, panic disorder, PTSD, social anxiety, substance misuse, or other disorder. Highest reported parental education for each participant (across both mother and father) was split into low (highest qualification GCSE or lower) and high (A Levels or higher). Pubertal stage was split into early (pre-pubertal, early pubertal, and mid-pubertal) and late (late pubertal and post-pubertal).

Associations with recall

In separate negative binomial mixed models, there was no evidence that ethnicity (p=0.33), English as first language (p=0.33), parental education (p=0.64), maternal depression (p=0.93), or ASD (p=0.12) were associated with hits. However, there was evidence that paternal depression (hits ratio = 0.82, 95% CI = 0.69 to 0.97, p=0.02) and parent-reported diagnoses of dyslexia (hits ratio = 0.65, 95% CI = 0.53 to 0.79, p<0.001) were associated with fewer hits.

I first repeated the main analyses in the subsample for whom additional confounders were not missing. In this subsample there was only evidence that valence and gender were associated with total hits (Table 5.5). Participants made 10% (95% CI=1.03 to 1.19, p<0.001 adjusted for confounders) more negative than positive hits. There was evidence that females made 18% (95% CI=1.08 to 1.30, p<0.001 adjusted for confounders) more hits than males. In contrast to analyses in the whole sample (reported in section 5.3.1), there was no evidence that condition or age group were associated with total hits in this subsample (Table 5.5). Adjusting for additional confounders did not alter the evidence for any of these associations (Table 5.5). Also, in this subsample, there was no evidence for any interactions between age group, gender, condition, or valence on total hits, before and after adjusting for additional confounders (Table 5.6).

Table 5.5 Negative binomial models testing associations between age group, gender, condition (self-/other-referential) and valence (positive/negative; exposures) and total hits (outcome), in subsample with additional confounders reported.

	Model 1: Adjusted		**Model 2: Additionally adjusted**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Condition	1.06 (0.99 to 1.14)	0.10	1.06 (0.99 to 1.14)	0.10
Valence	1.10 (1.03 to 1.19)	0.01	1.10 (1.03 to 1.19)	0.01
Age group	1.31 (0.96 to 1.79)	0.09	1.34 (0.99 to 1.81)	0.06
Gender	1.18 (1.03 to 1.19)	<0.001	1.18 (1.08 to 1.30)	<0.001

Note. N=275. Both models were adjusted for condition, valence, gender, age group, continuous age within each group, school, testing group size, non-verbal IQ score, and positive and negative false alarms. Model 2 was additionally adjusted for ethnicity, English as a first language, dyslexia, autism spectrum disorders, parental education, maternal depression, and paternal depression.

Table 5.6 Negative binomial models testing interactions between age group, gender, condition (self-/other-referential) and valence (positive/negative; exposures) on total hits (outcome), in subsample with additional confounders reported.

	1: Adjusted	**2: Additionally adjusted**
	Interaction term p value	
Gender x condition	0.56	0.56
Gender x valence	0.90	0.90
Gender x condition x valence	0.67	0.67
Age group x gender	0.23	0.29
Age group x gender x condition	0.15	0.15
Age group x gender x valence	0.16	0.16
Age group x gender x condition x valence	0.91	0.91

Note. N=275. Each interaction term was tested in a separate model. All models adjusted for condition, valence, gender, age group, continuous age within each group, school, testing group size, non-verbal IQ score, and positive and negative false alarms. Column 2 was additionally adjusted for ethnicity, English as a first language, dyslexia, autism spectrum disorders, parental education, maternal depression, and paternal depression.

Associations with depressive symptoms

In separate linear regression models, there was no evidence that English as a first language (p=0.24), parental education (p=0.60), maternal depression (p=0.70), paternal depression (p=0.14), dyslexia (p=0.22), or ASD (p=0.62) were associated with depressive symptoms. There was evidence that ethnicity was associated with depressive symptoms (p=0.02). Compared to White participants (M=7.34, SD=5.39), Mixed (M=9.11, SD=7.69), Asian/Asian British (M=10.21, SD=8.75), and other ethnicities (M=10.24, SD=5.30) had higher depressive symptoms. The Black/Black British group had lower depressive symptoms (M=5.83, SD=3.32).

In this subsample, there was only evidence that self-referential negative hits were associated with depressive symptoms (coef = 0.65, 95% CI = 0.20 to 1.10, p=0.005 adjusted for additional confounders). Before and after adjusting for additional confounders, there was no evidence that self-referential positive hits were associated with depressive symptoms (Table 5.7). Consistent with the main analysis, there was no evidence that other-referential positive or negative hits were associated with depressive symptoms (Table 5.7).

For all analyses in this subsample, before and after adjusting for additional confounders, the effect estimates were similar to the coefficients, and within the confidence intervals, from the primary analyses with the whole sample. The lack of evidence could be due to the reduced sample size or selection bias in those whose parents/carers completed additional questions.

Table 5.7 Linear regression models testing change in depressive symptoms (SMFQ score; outcome) for each additional self-referential positive, self-referential negative, other-referential positive, and other-referential negative hit (exposures), in subsample with additional confounders reported.

	Model 1: Adjusted		**Model 2: Additionally adjusted**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Self-referential hits				
Positive	-0.35 (-0.75 to 0.04)	0.08	-0.29 (-0.69 to 0.11)	0.16
Negative	0.66 (0.23 to 1.09)	0.003	0.65 (0.20 to 1.10)	0.005
Other-referential hits				
Positive	-0.01 (-0.45 to 0.42)	0.95	-0.01 (-0.45 to 0.43)	0.96
Negative	0.06 (-0.37 to 0.49)	0.79	0.05 (-0.39 to 0.49)	0.84

Note. N=275. Both models included all four types of hits as exposures and were adjusted for age group, gender, continuous age within each group, school, testing group size, non-verbal IQ score, and positive and negative false alarms. Model 2 was additionally adjusted for ethnicity, English as a first language, dyslexia, autism spectrum disorders, parental education, maternal depression, and paternal depression.

5.3.6 Sensitivity analyses: pubertal stage

The PDS was fully completed by a small proportion of participants (n=117, 20% of total sample). Of the subsample that completed this measure, 61 (52%) were in early pubertal stages (pre-pubertal, early pubertal, and mid-pubertal) and 56 (48%) were in late pubertal stages (late pubertal and post-pubertal). Mid-adolescents were generally in later stages of puberty than young adolescents (Table 5.4).

Associations with recall

In an unadjusted Poisson mixed model, there was no evidence that pubertal stage was associated with total hits (hits ratio = 1.14, 95% CI 0.99 to 1.32, p=0.07). However, given that this evidence for an association between pubertal stage and hits narrowly missed statistical significance (p=0.05), I repeated the main analyses adjusting for pubertal stage.

In the subsample of participants who completed the PDS, there was only evidence that valence and gender were associated with total hits (Table 5.8). Participants made 13% (95% CI=1.02 to 1.26, p=0.02 adjusted for confounders) more negative than positive hits. Females made 18% (95% CI=1.08 to 1.30, p<0.001 adjusted for confounders) more hits than males. In contrast to analyses in the whole sample, there was no evidence that condition or age group were associated with total hits (Table 5.8). Adjusting for pubertal stage did not alter the

evidence for any of these associations (Table 5.8). In this subsample, there was also no evidence for any interactions between age group, gender, condition, or valence on total hits, before and after adjusting for pubertal stage (Table 5.9).

Table 5.8 Poisson mixed models testing associations between age group, gender, condition (self-/other-referential) and valence (positive/negative; exposures) and total hits (outcome), in subsample with pubertal stage reported.

	Model 1: Adjusted		**Model 2: Additionally adjusted**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Condition	1.02 (0.91 to 1.13)	0.76	1.02 (0.91 to 1.13)	0.76
Valence	1.13 (1.02 to 1.26)	0.02	1.13 (1.02 to 1.26)	0.02
Age group	1.09 (0.72 to 1.64)	0.69	1.17 (0.76 to 1.79)	0.47
Gender	1.18 (1.04 to 1.34)	0.01	1.22 (1.06 to 1.40)	0.004

Note. N=117. Both models were adjusted for condition, valence, gender, age group, continuous age within each group, school, testing group size, non-verbal IQ score, and positive and negative false alarms. Model 2 was additionally adjusted for pubertal stage.

Table 5.9 Poisson mixed models testing interactions between age group, gender, condition (self-/other-referential) and valence (positive/negative; exposures) on total hits (outcome), in subsample with pubertal stage reported.

	1: Adjusted	**2: Additionally adjusted**
	Interaction term p value	
Gender x condition	0.10	0.10
Gender x valence	0.95	0.95
Gender x condition x valence	0.83	0.83
Age group x gender	0.15	0.12
Age group x gender x condition	0.09	0.09
Age group x gender x valence	0.41	0.41
Age group x gender x condition x valence	0.79	0.79

Note. N=117. Each interaction term was tested in a separate model. All models adjusted for condition, valence, gender, age group, continuous age within each group, school, testing group size, non-verbal IQ score, and positive and negative false alarms. Column 2 was additionally adjusted for pubertal stage.

Associations with depressive symptoms

There was evidence that pubertal stage was positively associated with depressive symptoms in a linear regression model. Adolescents in late, compared to early, pubertal stages scored 2.30 (95% CI 0.23 to 4.38, p=0.03) points higher on the SMFQ. In this subsample, there was no evidence that any type of hits was associated with depressive symptoms, before and after adjusting for pubertal stage (Table 5.10).

As with the previous sensitivity analyses, in the subsample who reported pubertal stage, coefficients were similar to (and within the confidence intervals from) findings in the primary analysis. The lack of evidence could be due to the reduced sample size or selection bias in participants who completed the PDS.

Table 5.10 Linear regression models testing change in depressive symptoms (SMFQ score; outcome) for each additional self-referential positive, self-referential negative, other-referential positive, and other-referential negative hit (exposures), in subsample with pubertal stage reported.

	Model 1: Adjusted		**Model 2: Additionally adjusted**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Self-referential hits				
Positive	-0.27 (-0.96 to 0.42)	0.44	-0.27 (-0.96 to 0.42)	0.44
Negative	0.51 (-0.23 to 1.24)	0.18	0.52 (-0.22 to 1.26)	0.17
Other-referential hits				
Positive	-0.02 (-0.80 to 0.75)	0.95	-0.06 (-0.84 to 0.72)	0.88
Negative	0.48 (-0.26 to 1.23)	0.20	0.55 (-0.21 to 1.31)	0.16

Note. N=117. Both models included all four types of hits as exposures and were adjusted for age group, gender, continuous age within each group, school, testing group size, non-verbal IQ score, and positive and negative false alarms. Model 2 was additionally adjusted for pubertal stage.

5.4 Discussion

Following a self- and other-referential social evaluation learning task, adolescents completed a surprise recall test. Consistent with my hypothesis (hypothesis 3.1), I found evidence that most adolescents better recalled self-referential than other-referential words, demonstrating a self-referential bias. However, there was evidence that young adolescent girls (11-13 years) recalled fewer self-referential than other-referential words, which was unexpected. I found no evidence for the hypothesised positive self-referential recall bias (hypothesis 3.2), as adolescents recalled more negative than positive words in both self-referential and other-referential conditions. Although I expected girls to demonstrate less positive self-referential recall biases (hypothesis 3.3), I found no evidence for this gender difference in positive or negative recall biases in either age group (hypothesis 3.4).

As hypothesised (hypothesis 3.5), more severe depressive symptoms were associated with a decrease in self-referential positive recall and an increase in self-referential negative recall. There was no evidence that these associations differed by age group, as predicted (hypothesis

3.6). However, contrary to my hypothesis (hypothesis 3.6), the association between self-referential negative recall and depressive symptoms was more pronounced in girls than boys.

5.4.1 Strengths and limitations

General strengths and limitations of the data included in this study were outlined in chapter 3 (section 3.4.1) and will be discussed in detail in the general discussion (section 7.3). In this study, my sample was recruited from eight diverse schools, making it more representative than many previous studies of recall biases (Appendix 5). I also modified the traditional recall task, allowing the differentiation of self-referential and other-referential recall bias.

However, this novel recall task had some limitations. Its reliability and validity are unknown, although tasks assessing memory and emotional biases are generally reliable (Bland et al., 2016). The nature of the encoding task may have influenced recall. Traditional tasks measure recall of words describing how participants see themselves, rather than how another individual sees them. Whilst it is likely that information consistent with the self-concept was preferentially recalled, I did not measure participants' self-concepts. Words incongruent with the self-concept may have been better recalled, as another person describing participants in this way could be more memorable. Additionally, adolescents with more depressive symptoms could have been differentially affected by the idea of someone liking or disliking them, altering reactions to the words, and influencing recall.

In this study, I did not find evidence for an effect of gender on positive or negative recall biases. As outlined in section 3.4.1, this could have been due to a lack of power to detect small effects in this study. My sample was powered to detect a difference of 0.4 standard deviations in outcomes within each age group, which is a relatively large difference in comparison to the effects that I did find. In order to study the effects of gender and age group, I tested two, three-, and four-way interactions, for which my power was likely to be below 50%. This may have resulted in a lack of evidence for associations which could exist. My findings should therefore be confirmed in a larger sample before concluding that there are no effects of gender on positive or negative recall biases.

Adjusting for potential confounders (continuous age within each group, school, testing group size, non-verbal IQ score, and positive and negative false alarms) did not alter the evidence

for any of the associations between age group, gender, and recall biases. I found evidence that recall was better in girls (compared to boys) and in mid-adolescents (compared to young adolescents). There was also evidence that IQ score was higher in girls than boys and higher in mid- than young adolescents. IQ score was also positively associated with better recall. It is thus possible that IQ was on the causal pathway between age group or gender and recall. As discussed in chapter 2 and 4 (sections 2.11.1 and 4.3.3), this could attenuate the evidence for associations between age group or gender and recall. However, in this study, there was still evidence for associations between age group, gender, and recall after including IQ score.

Information on demographics, special educational needs, and parental mental health were only available for a subsample of participants (59%). In this subsample, there was no longer evidence for the association between self-referential positive hits and depressive symptoms. Although there was some evidence that having dyslexia influenced task performance (adolescents with dyslexia recalled fewer words), adjusting for additional potential confounders did not alter the evidence for associations within this subsample. In the 20% of the sample who reported pubertal stage, there was no evidence that self-referential positive or negative hits were associated with depressive symptoms (before and after adjusting for pubertal stage). As discussed, these findings are likely due to the reduced sample size or selection bias in participants and parents/carers who completed additional questionnaires. Effect estimates were within the confidence intervals from the main analyses.

In this cross-sectional study, I cannot provide evidence of a causal effect of recall bias on depressive symptoms. My findings are consistent with this causal hypothesis, which is proposed by cognitive models of depression (Beck & Bredemeier, 2016; Roiser et al., 2012). However, it is also possible that changes in depressive symptoms cause changes in recall biases, or that the association is bidirectional. Longitudinal data is required to test the hypothesis that negatively biased recall leads to increased depressive symptoms.

5.4.2 Findings in context

I found evidence of enhanced memory for self-referential information during adolescence, as shown in children (Cunningham, Brebner, Quinn, & Turk, 2014) and adults (Symons & Johnson, 1997). It is unclear why there was evidence for this self-reference effect in all groups except young adolescent girls. In contrast to studies with healthy adults (Denny & Hunt, 1992;

Sanz, 1996; Sedikides & Green, 2000) and adolescents (Auerbach et al., 2016; Cole et al., 2014; Connolly et al., 2016; Dainer-Best et al., 2018; Fattahi Asl et al., 2015; Hammen & Zupan, 1984; Kuiper & MacDonald, 1982; Prieto et al., 1992; Taylor & Ingram, 1999; Timbremont & Braet, 2004), I did not find evidence for a difference in the self-reference effect between positive and negative information.

Adolescents recalled more negative than positive words in all conditions. This may be because self-evaluations become more negative and self-esteem declines during adolescence (Robins & Trzesniewski, 2005; van der Aar et al., 2018). However, this explanation would account for biases only in *self*-referential recall. The generalisation of this negative bias to other-referential recall could be due to the nature of my encoding task. Words were initially viewed as social evaluation, which may make negative words more salient and boost memory, regardless of whether words refer to the self or others. Consistent with this explanation, another study using a social evaluative encoding task (participants imagined overhearing others describing them) also found that adolescents remembered more negative than positive words (Holt et al., 2016).

Contrary to my hypotheses and previous evidence (McArthur et al., 2019), I did not find evidence for a gender difference in positive or negative recall. If gender inequality causes females to have more negative cognitions, I might expect more negative recall biases in girls from early adolescence, before the increase in depression (Bone et al., 2020). I did find some evidence that the association between self-referential negative recall and depressive symptoms was larger in girls than boys, across early and mid-adolescence. This was unexpected as I anticipated that recall would be similarly associated with depressive symptoms across genders. If self-referential negative recall is a risk factor for depressive symptoms, this could mean it is more important for girls than boys.

Both increased negative and reduced positive self-referential recall were associated with depressive symptoms, as found in some previous studies (Cole et al., 2014; Cole & Jordan, 1995; Connolly et al., 2016; Fattahi Asl et al., 2015; Moilanen, 1995; Timbremont & Braet, 2004; Zupan, Hammen, & Jaenicke, 1987). This finding differs to a recent review, which did not find consistent evidence for memory biases in adolescent depression (Platt et al., 2017). This could be because previous studies have generally assessed the proportion of words

previously endorsed as self-referential that are recalled. Testing recall of social evaluation may provide a more nuanced measure of memory biases.

In this study, effect estimates and confidence intervals for the associations between self-referential positive hits and depressive symptoms were clearly different from the corresponding association with other-referential positive hits, potentially suggesting a specific role of poorer self-referential positive recall in vulnerability to depressive symptoms. It is less clear whether there is a specific role of self-referential negative recall. In unadjusted analyses, self-referential and other-referential negative hits were associated with depressive symptoms. After adjusting for confounders, evidence for the association between other-referential negative hits and depressive symptoms was attenuated, but the coefficient and confidence interval were not clearly different from those for the corresponding association with self-referential negative hits. I cannot rule out that the association between self-referential negative hits and depressive symptoms reflects a general negative bias. However, self-referential negative recall was most strongly associated with depressive symptom severity, as previously found (Dainer-Best et al., 2018).

5.4.3 Conclusion

As hypothesised, and consistent with cognitive models of depression (Beck & Bredemeier, 2016; Roiser et al., 2012), I found evidence that adolescents who had more self-referential negative and less self-referential positive recall had more severe depressive symptoms. Contrary to my hypotheses, there was no evidence for gender or age differences specific to these types of recall, although this could be due to a lack of power to detect these associations. There was some evidence that the association between negative recall and depressive symptoms was stronger in girls. As expected, there was no evidence that the association between recall biases and depressive symptoms differed across early and mid-adolescence, despite the increase in depressive symptoms in older adolescents. This could be because negative recall biases are a risk factor for depressive symptoms from early adolescence. By having negatively biased self-referential recall, adolescents may have more negative memories of social interactions and more negative self-concepts, which could encourage social withdrawal and increase depressive symptoms. Longitudinal evidence is required to test whether these processes are causal. Negative self-referential bias may be a

risk factor for the emergence of depressive symptoms during adolescence and is a good candidate for future longitudinal studies.

Chapter 6 Dysfunctional attitudes during adolescence: gender differences and associations with depressive symptoms

6.1 Introduction

Dysfunctional attitudes are negative beliefs about the self, the world, and the future, which are associated with depressive symptoms in adolescence (as described in section 1.4; Beck, 1967, 1983; Beck & Bredemeier, 2016). As proposed by cognitive models of depression (Beck & Bredemeier, 2016; Roiser et al., 2012), and supported by a range of evidence (see section 1.4), dysfunctional attitudes may be a risk factor for depression. If this is the case, we might expect dysfunctional attitudes to be more common in girls than boys from early adolescence. As discussed in section 1.6, socialisation and gender inequality may cause girls to have more negative thoughts and beliefs about themselves, which could increase their risk of developing depressive symptoms (Bone et al., 2020). Girls may therefore have more dysfunctional attitudes than boys before the increase in the incidence of depressive symptoms occurs.

A number of studies have investigated whether there are gender differences in dysfunctional attitudes during adolescence. Dysfunctional attitudes are measured with the Dysfunctional Attitude Scale (DAS; Weissman, 1979; Weissman & Beck, 1978). The majority of cross-sectional and longitudinal studies have not found evidence for gender differences in total DAS scores (Abela & Sullivan, 2003; Chen & Li, 2014; Gotlib et al., 1993; Hankin et al., 2018; Lewinsohn et al., 2001; Rawal et al., 2013b; Young et al., 2012). Two relatively large longitudinal studies did find evidence that boys had more dysfunctional attitudes than girls (n=889 and 924 respectively; Meiser & Esser, 2017, 2019). However, studies showing no evidence for a gender difference have also been large (n=111 to 1710) and have recruited diverse samples, using both case-control and population-based designs. From this evidence, it therefore seems unlikely that there is an overall gender difference in dysfunctional attitudes.

However, dysfunctional attitudes are very idiosyncratic, and measuring a single continuum of dysfunctional attitudes may not represent how they exist in the general population (Beck, 1983; Burrage et al., 2016). Most studies which have tested the presence of subscales in the DAS have fitted a two-factor solution, with factors labelled perfectionism (or performance

evaluation) and need for approval (need for social approval, approval by others, or dependency; Barnett & Gotlib, 1990; Cane et al., 1986; De Graaf et al., 2009; Imber et al., 1990; Rogers et al., 2009; Zlotnick et al., 1996). Perfectionism relates to having high personal standards and interpreting mistakes as failure. Need for approval involves beliefs that one's own happiness and self-worth are dependent on gaining approval and support from others. Beck originally hypothesised that perfectionism is higher in males and need for approval is higher in females (Beck, 1983), and this idea has repeatedly been proposed (Barnett & Gotlib, 1990; De Graaf et al., 2009; Farmer et al., 2001; Meiser & Esser, 2019; Otani et al., 2013; Zlotnick et al., 1996). This is similar to traditional gender role theories, which suggest that females are more interpersonally oriented (as discussed in section 1.9.3). In contrast, males may be more achievement oriented (e.g. Ellemers, 2018; Kirsh & Kuiper, 2002; Stroud et al., 2002).

To my knowledge, only one study has tested gender differences in perfectionism and need for approval using the DAS in adolescence (Marcotte, Lévesque, & Fortin, 2006). There was no evidence for gender differences in need for approval but, consistent with Beck's (1983) proposal, perfectionism (combined with a self-control subscale) was consistently higher in boys (Marcotte et al., 2006). However, this longitudinal study had a moderately-sized convenience sample (n=644), recruited from two high schools in an upper middle-class urban area, meaning the findings may not be generalisable to the general population.

Within the field of perfectionism research, it has been hypothesised instead that girls are more perfectionistic than boys (e.g. Jaradat, 2013; Rice, Kubal, & Preusser, 2004; Starley, 2019). However, using specific perfectionism questionnaires, all but one of the studies in this field find no evidence for gender differences in perfectionism (Asseraf & Vaillancourt, 2015; Hewitt et al., 2002; Jaradat, 2013; Rice et al., 2004; Rice, Leever, Noggle, & Lapsley, 2007; Soenens et al., 2008). The only study to find evidence for higher perfectionism in girls than boys was cross-sectional and included a moderate sample (n=419) of Jordanian high school students (Jaradat, 2013). These adolescents may differ to adolescents in higher-income countries (such as the US and UK) where most other research has been conducted. Some studies have instead found evidence that the association between perfectionism and depressive symptoms is moderated by gender, with a stronger association in girls than boys (Dogan, 2019; Rice et al., 2007). However, these cross-sectional studies were small (n=244

and 145 respectively; Dogan, 2019; Rice et al., 2007) and may have been underpowered to test effect modification (Button et al., 2013; Greenland, 1983). Other larger longitudinal studies (n=653, 144, and 434 respectively) have found no evidence for this effect modification (Asseraf & Vaillancourt, 2015; Hewitt et al., 2002; Soenens et al., 2008).

Across research using the DAS and other perfectionism questionnaires, it remains unclear whether there are gender differences in the prevalence of perfectionism and need for approval during adolescence. If there are gender differences in these factors, it is possible that they could mediate the gender difference in depression. For example, being female could lead to increased need for approval, which is then associated with increased depressive symptoms. In this way, dysfunctional attitudes could contribute to the emergence of the gender difference in depression.

Dysfunctional attitudes are thought to be stable traits in adulthood which are applicable across situations and increase an individual's vulnerability to depression (Abela & Hankin, 2008). However, it is unclear whether they are established by early adolescence, or if they are still developing during adolescence (Nolen-Hoeksema, Girgus, & Seligman, 1992; 1994). If dysfunctional attitudes develop as a result of negative childhood experiences (Beck & Bredemeier, 2016), we might expect them to be present from early adolescence. However, the maturational changes in higher-order cognitive processes that occur throughout adolescence may be necessary for individuals to develop stable cognitive styles (Cole et al., 2008; Turner & Cole, 1994). There is evidence from relatively large population-based studies, with representative samples, that dysfunctional attitudes increase across a range of ages in adolescence (15-17 years in Hankin, 2008; 9-18 and 9-13 years in Meiser & Esser, 2017, 2019). However, other studies (also with representative population-based samples) have found no evidence for an association between age and dysfunctional attitudes (11-17 years in Chen & Li, 2014; 12-17 years in Rogers et al., 2009; 12-15 years in Young et al., 2012) or even that they decrease with age (8-14 years in D'Alessandro & Burton, 2006). It is therefore unclear whether dysfunctional attitudes are stable throughout adolescence or change with age.

In addition to examining whether the prevalence of dysfunctional attitudes differs with age, studies have tested whether the association between dysfunctional attitudes and depressive symptoms changes with age. If dysfunctional attitudes are causally related to depressive

symptoms, we would expect them to be associated with depressive symptoms at all ages. There is some evidence that dysfunctional attitudes are only associated with subsequent depressive symptoms in older adolescents (14-18 years), and not younger adolescents (10-13 years; Rawal et al., 2013b). However, this prospective cohort included only 121 adolescents. Larger cross-sectional and longitudinal studies have not found any evidence that age moderates the association between dysfunctional attitudes and depressive symptoms in participants aged 9-18 years (D'Alessandro & Burton, 2006; Hankin, Wetter, Cheely, & Oppenheimer, 2008; Meiser & Esser, 2017, 2019). Dysfunctional attitudes may therefore increase vulnerability to depression from early adolescence and could contribute to the emergence of the gender difference in depression.

In this study, I tested whether there were gender differences in dysfunctional attitudes in a cross-sectional study (n=567) of adolescents in two age groups (young and mid-adolescents, aged 11 to 13 and 13 to 15 years). I recruited these age groups in order to study dysfunctional attitudes before and after the gender difference in depression emerged. I aimed to investigate whether there are gender differences in perfectionism and need for approval, examine whether these gender differences change with age, and test whether perfectionism and need for approval are associated with depressive symptoms in adolescence.

I hypothesised that perfectionism would be higher in boys and need for approval would be higher in girls (hypothesis 4.1). I expected dysfunctional attitudes to be present from early adolescence, and thus hypothesised that there would be no association between age group and dysfunctional attitudes (hypothesis 4.2). I hypothesised that perfectionism and need for approval would both be positively associated with depressive symptoms (hypothesis 4.3). Although using cross-sectional data, I also hypothesised that dysfunctional attitudes would mediate the association between gender and depressive symptoms (hypothesis 4.4). I expected girls to have higher need for approval, which would then be associated with more severe depressive symptoms. Finally, I hypothesised that these associations between gender, perfectionism, need for approval, and depressive symptoms would be present from early adolescence, so would not differ across the two age groups (hypothesis 4.5).

6.2 Methods

6.2.1 Participants

In my final sample, 31 (5%) participants were missing data on dysfunctional attitudes and 1 (<1%) participant was missing data on depressive symptoms (final n=567). See section 2.9.4 for an overview of all missing data in this study.

6.2.2 Measures

6.2.2.1 Dysfunctional attitudes

The 17-item Dysfunctional Attitude Scale (Form A) Revised (DAS) measures dysfunctional attitudes (De Graaf et al., 2009). I replaced missing responses using person-mean imputation (see section 2.9.3) for those who responded to 12 or more questions using each individual's mean DAS score (n=187 participants, 36% of sample). This version of the DAS has previously been divided into two subscales, with 11 items measuring perfectionism and the other six items forming the need for approval subscale (De Graaf et al., 2009).

6.2.2.2 Depressive symptoms

The 13-item short Mood and Feelings Questionnaire (SMFQ) measures depressive symptoms over the last two weeks (Angold et al., 1995). I replaced missing responses using person-mean imputation for those who responded to ten or more questions using each individual's mean SMFQ score (n=106 participants, 19% of this sample).

6.2.2.3 Confounders

Participants' age in years, school, and testing group size were all recorded as potential confounders. Participants also completed an abbreviated version of the Raven Standard Progressive Matrices Test (non-verbal IQ score; Bilker et al., 2012). As described in the general methods (section 2.5 and 2.8), additional potential confounders (ethnicity, English as a first language, dyslexia, autism spectrum disorders, parental education, maternal depression, paternal depression, pubertal stage) were collected through participant follow-up and parental questionnaires.

6.2.3 Statistical analysis

I performed all analyses using Stata 16 (StataCorp, 2019). As my sample consisted of two age groups, and one of my aims was to investigate the influence of gender in each age group, I have presented all descriptive statistics separately according to gender for each age group. I then performed some preliminary analyses of dysfunctional attitudes and depressive symptoms. Firstly, I used linear regression to test the association between age group and gender (binary exposures), and SMFQ score (continuous outcome). Next, I examined whether the gender difference in depressive symptoms increased with age by testing an interaction between age group and gender. I then used linear regression to test whether gender and age group were associated with total DAS score (continuous outcome). Finally, I added an interaction between age group and gender to test whether the gender difference in total DAS score changed with age.

6.2.3.1 Confirmatory factor analysis

I used confirmatory factor analysis (CFA) to test the fit of one-factor and two-factor models to DAS responses. The one-factor model included all DAS items loading onto one latent variable. The two-factor model specified perfectionism and need for approval as latent variables, with factor loadings based on subscales previously used in this version of the DAS (De Graaf et al., 2009). I estimated standardised factor loadings using sufficient-statistic maximum-likelihood estimation.

I then assessed model fit using the χ^2 test statistic, the traditional measure for evaluating model fit, which tests the discrepancy between the observed sample and the fitted covariance matrices (Kline, 2015). As the χ^2 statistic is affected by sample size (trivial differences may be significant with large samples), I also used other model fit indices: the Root Mean Squared Error of Approximation (RMSEA) with 90% confidence intervals (as typically used); Standardised Root Mean Square Residual (SRMR); Comparative Fit Index (CFI); and the Tucker-Lewis index (TLI; Jackson, Gillaspy, & Purc-Stephenson, 2009; Kline, 2015). Cut-offs to evaluate acceptable fit on each of these measures were RMSEA<0.08, SRMR<0.08, CFI>0.9, and TLI>0.9 (Hu & Bentler, 1999; MacCallum, Browne, & Sugawara, 1996). I chose to use multiple indices as they provide a more comprehensive evaluation of model fit, testing both relative (CFI and TLI) and absolute (RMSEA and SRMR) model fit. I tested internal consistency

of each factor using Cronbach's alpha and calculated the Pearson product-moment correlation between latent factors.

6.2.3.2 Structural equation models

I then used the better fitting two-factor model of dysfunctional attitudes in full structural equation models (SEM). I extended the measurement model from the above CFA, adding a structural model with parameters specifying associations between gender, age group, dysfunctional attitudes, and depressive symptoms. In SEM, the measurement model is the relation of latent variables to the observed items (as in CFA) and the structural model is the associations among latent variables and between latent variables and any exposures (as in path analysis or regression; Bollen, 1989).

This model is shown in Figure 6.1 (Model 1). In this model, I tested whether gender and age group were associated with perfectionism and need for approval (hypothesis 4.1 and 4.2), and whether perfectionism and need for approval were associated with depressive symptoms (hypothesis 4.3). In order to test whether dysfunctional attitudes could mediate the gender difference in depression (hypothesis 4.4), I tested indirect associations in the model. Indirect associations are the part of the association between one variable and another that passes through a specific intervening variable. I tested indirect associations from both age group and gender to depressive symptoms, through two different intervening variables, perfectionism and need for approval. For indirect associations, I estimated 95% confidence intervals using percentiles from bootstrapping with 1000 replications. Using this method avoids the assumption that indirect effects have normal and symmetric sampling distributions, neither of which are typically the case (MacKinnon, 2008).

I have presented this model before (Model 1; Figure 6.1) and after (Model 2; Figure 6.2) adjustment for potential confounders (continuous age within each age group, school, testing group size, and non-verbal IQ score). Before adjusting for potential confounders, this model included 72 free parameters (33 residual variances, 30 factor loadings, 8 structural associations, and 1 covariance between perfectionism and need for approval) and, after adjusting for confounders, it had 102 free parameters. Adding confounders meant estimating 30 additional structural associations. In SEM, there is a rule of thumb that a sample should have 10 participants for each model parameter estimated (Kline, 2015). However, a sample

of 567 participants for estimating the 72 parameters in the adjusted model is acceptable, and I have compared findings from the adjusted and unadjusted models to assess whether the adjusted model may be underpowered.

6.2.3.3 Differences across age groups

I was also interested in whether the associations between gender, perfectionism, need for approval, and depressive symptoms differed in young versus mid-adolescents (hypothesis 4.5). I replaced the age group and gender variables in the adjusted model (Model 2) with an age group by gender interaction term. I examined the global p value to indicate whether the association between gender, perfectionism, need for approval, and depressive symptoms differed across age groups. As there was evidence for this interaction, I re-estimated this model across the two age groups.

I first tested a model with all parameters constrained across age groups (Model 3). From this model, I examined which structural paths should be constrained versus allowed to differ between young and mid-adolescents. All other parameters were constrained across age groups, including the measurement model and associations between confounders and latent variables. I used score tests (also called Lagrange multiplier tests) to evaluate whether constraints on the structural parameters of interest should be relaxed across groups. Where there was evidence that a parameter should not be constrained, I estimated it separately across groups (Model 4). This model was nested within the previous model because the simpler model (Model 2) could be derived by imposing constraints on Model 4 (i.e. constraining all parameters across age groups). I compared the relative fit of this nested model to the model where all parameters were constrained using a likelihood ratio test (chi-square difference test; Model 4 versus Model 3). For model comparison, I adjusted models for all confounders except school. If school was included then there were empty cells in the covariance matrix, meaning the saturated model (and thus model fit indices) could not be estimated. After selecting a winning model, I also adjusted it for school.

6.2.3.4 Sensitivity analysis: improving parameter estimation

In a sensitivity analysis, I used a data-driven approach to improve parameter estimation in the adjusted model for the whole sample (Model 2). I used modification indices to add covariance parameters between the residual variances of items loading onto the same latent factor

(Model 5; Figure 6.3). I calculated modification indices for every parameter that was restricted (set to zero) in the model. These indicated the expected change in model fit that would result if the restriction on that parameter was removed (Sörbom, 1989). This approach is often not recommended because it is data-driven, as opposed to theory-driven (Curran & Bauer, personal communication). Estimating more parameters improves fit to the data but also makes the model more complex. Despite these limitations, I aimed to improve model fit, resulting in a properly specified model with valid estimates of the parameters and standard errors. If a path exists in the observed data, but is omitted from the model, this can bias other estimated paths (Bollen, 1989; Kline, 2015).

I used modification indices iteratively, adding a covariance parameter for the pair of residual variances with the highest modification index, rerunning the model, and then identifying the pair with the next highest index (Curran & Bauer, personal communication; Kline, 2015). Covariance parameters were added between DAS subscale and SMFQ item residuals: DAS Q2 with DAS Q7; DAS Q3 with DAS Q12; DAS Q4 with DAS Q5; DAS Q4 with DAS Q8; DAS Q5 with DAS Q8; DAS Q6 with DAS Q8; DAS Q7 with DAS Q12; DAS Q10 with DAS Q17; DAS Q13 with DAS Q14; DAS Q14 with DAS Q15; DAS Q15 with DAS Q17; SMFQ 2 with SMFQ Q3; SMFQ Q4 with SMFQ Q7; SMFQ Q7 with SMFQ Q8; SMFQ Q8 with SMFQ Q11; SMFQ Q9 with SMFQ Q13; and SMFQ Q10 with SMFQ Q11 (Model 5; Figure 6.3). Results from this model should be interpreted with caution because 17 covariance parameters were added, resulting in a total of 119 parameters estimated in a sample of 559 adolescents. This was much lower than the recommended 10:1 ratio of participants to estimated parameters (Kline, 2015). I compared the relative fit of this nested model to the model without additional covariance parameters estimated using a likelihood ratio test (Model 5 versus Model 2). I then examined associations between age group, gender, perfectionism, need for approval, and depressive symptoms in this model.

6.2.3.5 Sensitivity analyses: additional confounders

I had also planned to adjust for additional potential confounders reported by participants' parents/carers (ethnicity, English as a first language, dyslexia, autism spectrum disorders, parental education, maternal depression, paternal depression). However, as outlined previously (section 2.9.4), parental response rates were low (n=336, 59% of those who also completed the DAS and SMFQ). Adjusting for additional confounders would have added

another 21 parameters to the full model (seven exposures, each associated with perfectionism, need for approval, and depressive symptoms), meaning a total of 123 parameters would have to be estimated. Given the recommended ratio of participants to parameters (Kline, 2015), I decided that it was not appropriate to estimate an SEM with 123 parameters in a sample of 336 participants. This could have led to biased estimates and increased the probability of Type 1 errors. Similarly, adjusting for pubertal stage in the subsample of 116 participants who completed the measure of puberty (as described in section 2.9.4) was not feasible.

6.3 Results

My final sample consisted of 567 adolescents (49% female). Of these, 303 (53%) were young adolescents from Year 7 and 264 (47%) were mid-adolescents from Years 9-10. Young adolescents' ages ranged from 11 to 13 years (M=11.57, SD=0.50) and mid-adolescents' ages ranged from 13 to 15 years (M=14.17, SD=0.52). Table 6.1 shows sample characteristics and dysfunctional attitudes according to age group.

Table 6.1 Demographics characteristics and dysfunctional attitudes of adolescents who completed the Dysfunctional Attitude Scale.

	Young adolescents		**Mid-adolescents**		**Overall**	
	Males (n=158)	**Females** (n=139)	**Males** (n=127)	**Females** (n=136)	**Skewness** (n=567)	**Kurtosis** (n=567)
	Mean (SD)				Statistic	
Age	11.57 (0.50)	11.56 (0.51)	14.19 (0.47)	14.16 (0.56)	0.12	1.54
Non-verbal IQ	3.86 (2.06)	4.50 (1.85)	4.80 (2.01)	4.99 (1.99)	-0.11	2.24
Dep symptoms	6.32 (5.05)	7.98 (5.83)	6.54 (4.89)	9.78 (6.31)	0.94	3.37
DAS total	46.51 (18.22)	50.85 (20.64)	52.14 (18.27)	54.82 (18.02)	0.36	2.62
Perfectionism	26.43 (11.63)	28.91 (13.25)	30.25 (12.18)	31.72 (11.72)	0.68	2.98
Need for approval	20.08 (8.01)	21.94 (8.74)	21.89 (8.21)	23.10 (8.27)	0.02	2.91

Note. DAS: Dysfunctional Attitude Scale. DAS, perfectionism, and need for approval were raw total scores, calculated as the sum of each participant's responses. Gender missing for n=7 and continuous age missing for n=1 young adolescent male.

In young adolescents, SMFQ score ranged from 0 to 23 (M=7.11, SD=5.47). The SMFQ threshold for depression was met by 58 (19%) young adolescents. In mid-adolescents, SMFQ score ranged from 0 to 26 (M=8.24, SD=5.89). The SMFQ threshold for depression was met by 61 (23%) mid-adolescents. There was evidence that SMFQ score was 1.13 (95% CI 0.20 to 2.07, p=0.02) points higher in mid-adolescents than young adolescents, and strong evidence

that SMFQ score was 2.45 (95% CI = 1.53 to 3.38, p<0.001) points higher in females than males. There was no evidence of an interaction between age group and gender on depressive symptoms (interaction p=0.10). Depressive symptoms were higher in females in both age groups. Although the evidence for this interaction did not meet the threshold for statistical significance (p=0.05), this gender difference did increase with age (young adolescents coef=1.67, 95% CI=0.42 to 2.91; mid-adolescents coef=3.24, 95% CI=1.86 to 4.62).

Total DAS score ranged from 17 to 104 (M=50.87, SD=19.06). There was evidence that age group and gender were associated with overall dysfunctional attitudes. DAS score was 5.46 (95% CI = 1.86 to 9.06, p=0.003 adjusted for confounders) points higher in mid-adolescents than young adolescents and 3.57 (95% CI=0.42 to 6.73, p=0.03 adjusted for confounders) points higher in females than males. There was no evidence for an interaction between age group and gender on DAS score (adjusted interaction p=0.62).

6.3.1 Confirmatory factor analysis

In the CFA, a two-factor model adequately fit the data, and was a better fit than a one-factor model (Table 6.2). In the two-factor model, which specified perfectionism and need for approval as latent factors, the SRMR (0.06) indicated an acceptable model fit and the RMSEA (0.09), CFI (0.89) and TLI (0.88) were very close to meeting acceptable fit criteria. As the perfectionism and need for approval subscales in a similar version of the DAS have previously been validated in adolescents (Rogers et al., 2009), and two factors better fit the data than one factor, I retained the two-factor model. Factor loadings were medium to high for all items (0.54 to 0.82; Table 6.3). Internal consistency was high for the perfectionism (α=0.90) and need for approval (α=0.86) factors. There was also evidence for a strong positive correlation between the perfectionism and need for approval factors (r(565)=0.82, p<0.001).

Table 6.2 Model fit indices for the two confirmatory factor analyses (measurement models) and five structural equation models (models 1-5) of dysfunctional attitudes.

Model	N	RMSEA (90% CI)	SRMR	CFI	TLI	χ^2	df
Whole sample							
Measurement model *1 latent factor*	567	0.11 (0.11 to 0.12)	0.07	0.82	0.79	996.91	119
Measurement model *2 latent factors*	567	0.09 (0.08 to 1.00)	0.06	0.89	0.88	634.64	118
Model 1 *Unadjusted*	560	0.06 (0.05 to 0.06)	0.05	0.89	0.88	1306.27	456
Model 2 *Adjusted* [a]	559	0.05 (0.05 to 0.05)	0.05	0.88	0.87	1694.53	726
Separate by age group							
Model 3 *All parameters constrained* [a]	559	0.06 (0.06 to 0.06)	0.08	0.86	0.86	2240.40	1125
Model 4 *Unconstrained association between gender & perfectionism* [a]	559	0.06 (0.06 to 0.06)	0.08	0.86	0.86	2233.83	1124
Sensitivity analysis							
Model 5 *Adjusted model with added covariances* [b]	559	0.04 (0.04 to 0.04)	0.04	0.93	0.92	1289.23	709

Note. RMSEA: Root Mean Squared Error of Approximation. 90% CI: 90% confidence interval. SRMR: Standardised Root Mean Square Residual. CFI: Comparative Fit Index. TLI: Tucker-Lewis index. df: degrees of freedom. Cut-offs for acceptable fit on these measures were RMSEA<0.08, SRMR<0.08, CFI>0.9, and TLI>0.9. All χ^2 p values were <0.001. [a] *Adjusted for continuous age within each age group, school, testing group size, and non-verbal IQ score.* [b] *Adjusted and added covariance included between DAS subscale and SMFQ item residuals (see statistical analysis or Figure 6.3).*

6.3.2 Structural equation models

I then tested hypotheses 4.1 to 4.4 by assessing the associations between age group, gender, perfectionism, need for approval, and depressive symptoms in a full SEM (Figure 6.1; Table 6.4). This model did not include confounders. It fitted the data acceptably according to the RMSEA (0.06) and SRMR (0.05), and the CFI (0.89) and TLI (0.87) were very close to meeting the acceptable model fit criteria (Table 6.2).

Table 6.3 Factor loadings for one and two factor models of dysfunctional attitudes in confirmatory factor analyses.

DAS question	Factor loadings		
	One factor	Two factors	
		Perf	NFA
1. It is difficult to be happy, unless one is good looking, intelligent, rich and creative.	0.60	0.60	
2. If I do not do well all the time, people will not respect me.	0.70	0.71	
3. If a person asks for help, it is a sign of weakness.	0.51	0.56	
4. If I do not do as well as other people, it means I am an inferior human being.	0.77	0.82	
5. If I fail at my work, then I am a failure as a person.	0.71	0.76	
6. If you cannot do something well, there is little point in doing it at all.	0.64	0.66	
7. If someone disagrees with me, it probably indicates that he does not like me.	0.56	0.54	
8. If I fail partly, it is as bad as a complete failure.	0.73	0.77	
9. If other people know what you're really like, they will think less of you.	0.68	0.66	
10. My value as a person depends greatly on what others think of me.	0.72		0.82
11. If I am to be a worthwhile person, I must be truly outstanding in at least one major respect.	0.57	0.71	
12. If I ask a question, it makes me look inferior.	0.75	0.59	
13. It is awful to be disapproved of by people important to you.	0.57		0.63
14. If you don't have other people to lean on, you are bound to be sad.	0.54		0.61
15. If others dislike you, you cannot be happy.	0.61		0.72
16. My happiness depends more on other people than it does on me.	0.62		0.68
17. What other people think about me is very important.	0.64		0.78

Note. N=567 in both models. Perf: Perfectionism. NFA: Need for approval.

Hypothesis 4.1 Perfectionism would be higher in boys and need for approval would be higher in girls

There was evidence for a direct association between gender and perfectionism. Females had higher perfectionism than males (coef=0.10, 95% CI=0.01 to 0.18, p=0.03). There was no evidence for an association between gender and need for approval (p=0.10; Table 6.4).

Hypothesis 4.2 There would be no association between age group and dysfunctional attitudes

There was evidence that mid-adolescents had higher perfectionism than young adolescents (coef=0.14, 95% CI=0.06 to 0.23, p=0.001). There was similar, but weaker, evidence for an association between age group and need for approval. Mid-adolescents had higher need for approval than young adolescents (coef=0.10, 95% CI=0.01 to 0.18, p=0.03).

Table 6.4 Associations between age group and gender (exposures) and perfectionism, need for approval, and depressive symptoms (latent variables) in the full structural equation models.

	Model 1: Unadjusted (n=560)		**Model 2: Adjusted[a]** (n=559)	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Direct association with perfectionism				
Age group	0.14 (0.06 to 0.23)	0.001	0.16 (0.06 to 0.25)	0.002
Gender	0.10 (0.01 to 0.18)	0.03	0.09 (0.005 to 0.18)	0.04
Direct association with need for approval				
Age group	0.10 (0.01 to 0.18)	0.03	0.13 (0.03 to 0.22)	0.01
Gender	0.07 (-0.01 to 0.16)	0.10	0.08 (-0.001 to 0.17)	0.05
Direct association with depressive symptoms				
Age group	-0.01 (-0.08 to 0.06)	0.78	-0.04 (-0.11 to 0.04)	0.37
Gender	0.17 (0.11 to 0.24)	<0.001	0.18 (0.11 to 0.25)	<0.001
Perfectionism	0.57 (0.45 to 0.69)	<0.001	0.56 (0.43 to 0.70)	<0.001
Need for approval	0.12 (-0.01 to 0.24)	0.08	0.14 (-0.01 to 0.29)	0.07
Indirect association with depressive symptoms				
Age group - perfectionism	0.06 (0.02 to 0.11)		0.07 (0.02 to 0.12)	
Age group - need for approval	0.01 (-0.002 to 0.02)		0.01 (-0.002 to 0.04)	
Gender - perfectionism	0.04 (0.01 to 0.08)		0.04 (-0.001 to 0.09)	
Gender - need for approval	0.01 (-0.002 to 0.02)		0.01 (-0.002 to 0.03)	

Note. Coefficients were standardised. Indirect associations were estimated using bootstrapping (1000 replications) and 95% percentile confidence intervals. [a] Adjusted for continuous age within each age group, school, testing group size, and non-verbal IQ score. Model 1 is represented in Figure 6.1 and Model 2 in Figure 6.2.

Hypothesis 4.3 Perfectionism and need for approval would both be positively associated with depressive symptoms

There was strong evidence that higher perfectionism was associated with more severe depressive symptoms (coef=0.57, 95% CI=0.45 to 0.69, p<0.001). However, there was no evidence that higher need for approval was associated with more severe depressive symptoms (p=0.08; Table 6.4).

Hypothesis 4.4 Dysfunctional attitudes would mediate the association between gender and depressive symptoms

There was strong evidence from this SEM that depressive symptoms were higher in females than males (coef=0.17, 95% CI=0.11 to 0.24, p<0.001). I tested whether gender was indirectly associated with depressive symptoms through perfectionism or need for approval. There was no evidence for an association between gender and depressive symptoms through need for

approval (Table 6.4). However, there was evidence for an indirect association between gender and depressive symptoms through perfectionism (coef=0.04, 95% CI=0.01 to 0.08; Table 6.4). Females had more perfectionism than males, and perfectionism was positively associated with depressive symptoms (Figure 6.1).

Although I did not make hypotheses about indirect associations between age group and depressive symptoms, these were specified in this model. There was no evidence for a direct association between age group and depressive symptoms (p=0.78; Table 6.4). However, there was evidence for an indirect association between age group and depressive symptoms through perfectionism (coef=0.06, 95% CI=0.02 to 0.11). Mid-adolescents had higher perfectionism than young adolescents, and higher perfectionism was associated with more severe depressive symptoms (Figure 6.1). There was no evidence for an indirect association between age group and depressive symptoms through need for approval (Table 6.4).

I then added confounders to this SEM (Model 2; Figure 6.2; Table 6.4). This improved model fit in terms of the RMSEA and χ^2 statistic and did not substantially alter the CFI and TLI indices (Table 6.2). After adjusting for confounders, there was weak evidence for an association between gender and need for approval, as females had higher need for approval than males (coef=0.08, 95% CI=-0.001 to 0.17, p=0.05; *hypothesis 4.1*). However, the evidence for an indirect association between gender and depressive symptoms through perfectionism was weaker than in the unadjusted model (coef=0.04, 95% CI=-0.001 to 0.09; *hypothesis 4.4*). Evidence for all other associations was similar (Table 6.4; Figure 6.2).

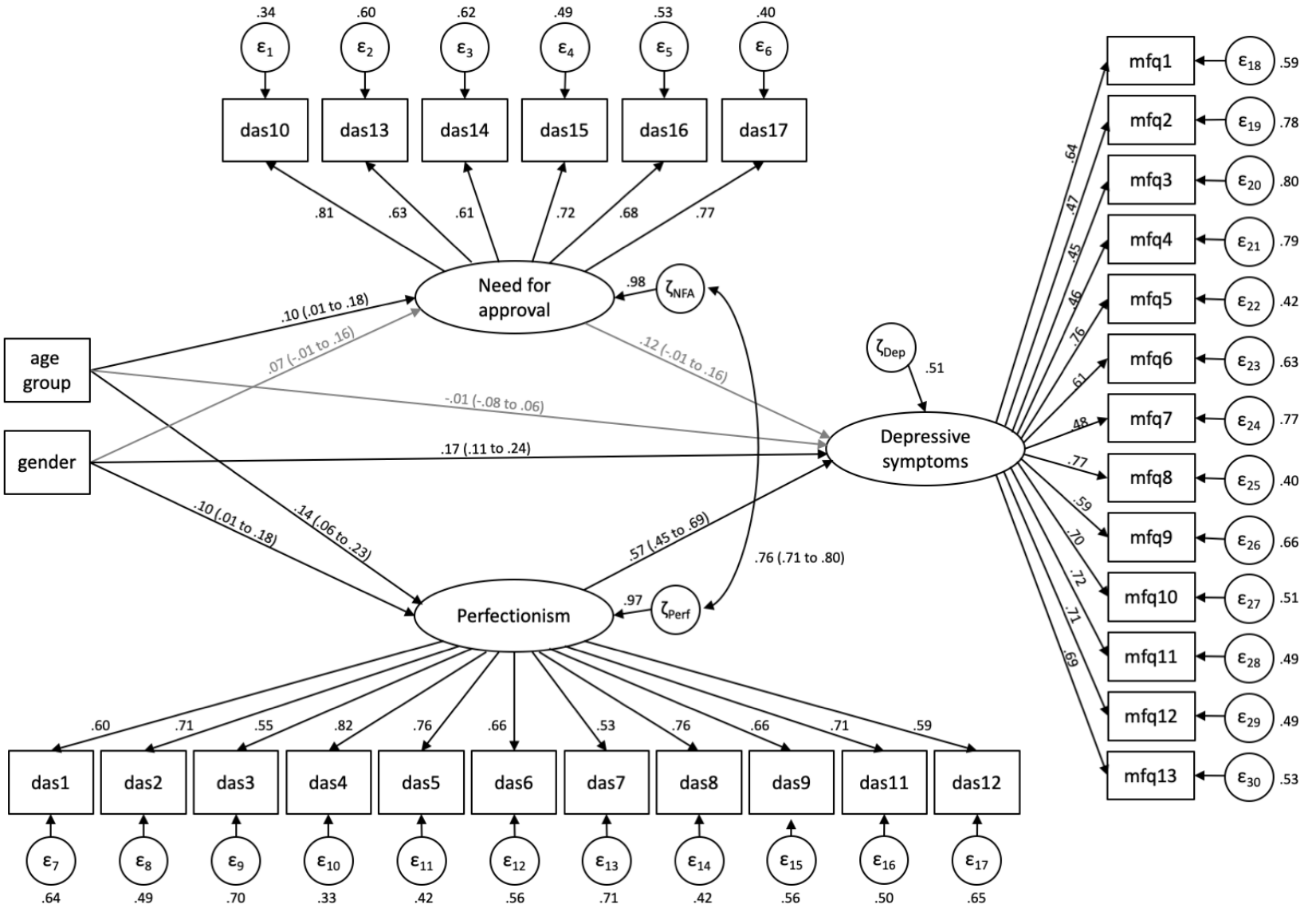

Figure 6.1 Unadjusted structural equation model (n=560). Coefficients were standardised and 95% confidence intervals are given in parentheses for structural associations. Paths which are not significant (p<0.05) are grey.

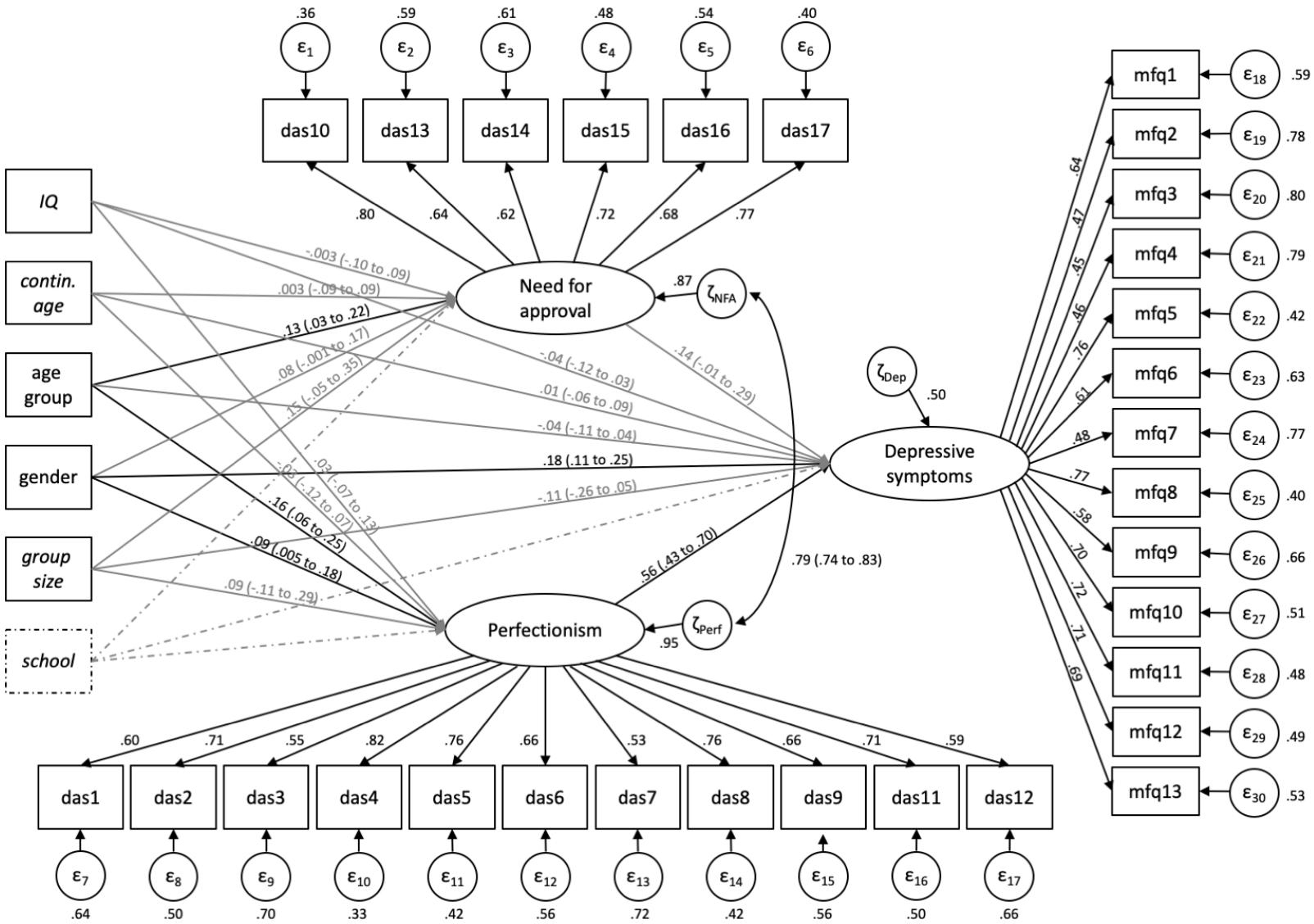

Figure 6.2 Structural equation model, adjusted for continuous age within each age group, school, testing group size, and non-verbal IQ score (n=559). Coefficients were standardised, and 95% confidence intervals are in parentheses for structural associations. Paths which are not significant (p<0.05) are grey. School is shown with a dotted line as it was included using 7 dummy variables.

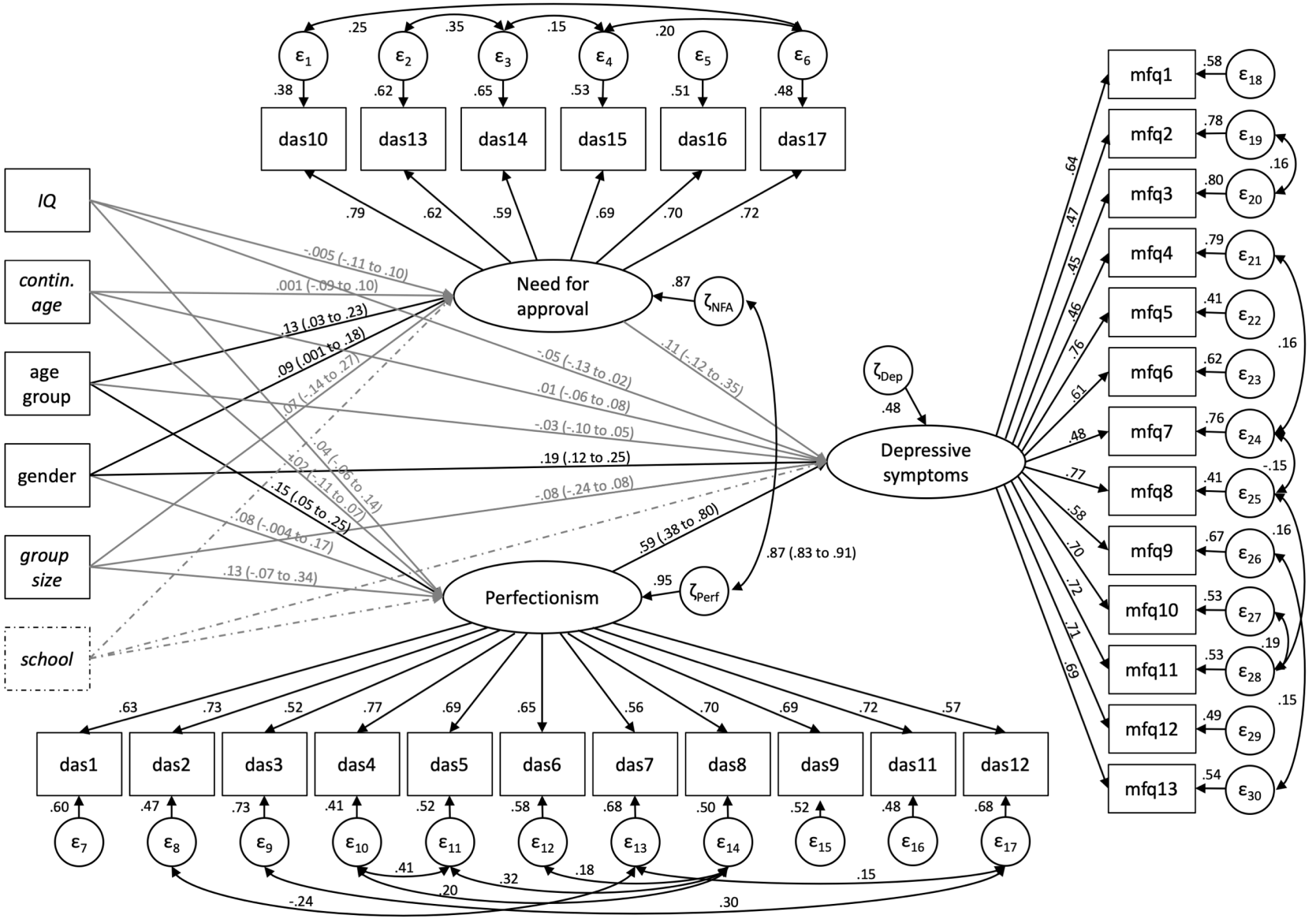

Figure 6.3 Structural equation model adjusted for confounders (as in Figure 6.2) with covariance parameters between DAS subscale and SMFQ item residuals (n=559). Coefficients were standardised and 95% confidence intervals are in parentheses for structural associations. Paths with p<0.05 are grey.

6.3.2.1 Differences across age groups

Hypothesis 4.5 Associations between gender, perfectionism, need for approval, and depressive symptoms would not differ across age groups

I then tested whether the associations between gender and the latent variables differed in young versus mid-adolescents. There was evidence for an interaction between age group and gender on perfectionism (adjusted interaction global p<0.001), need for approval (adjusted interaction global p=0.001), but no evidence for an interaction on depressive symptoms (adjusted interaction global p=0.08). I therefore tested whether the associations between gender, dysfunctional attitudes, and depressive symptoms differed across age groups.

The first model, with all parameters constrained across groups, had adequate model fit (Model 3; Table 6.2). Testing which structural paths should be unconstrained in this model, there was only evidence that the association between gender and perfectionism differed across age groups (score test p=0.01; all other parameters p>0.05). A likelihood ratio test provided evidence ($\chi^2(1) = 6.57$, p=0.01) that allowing this parameter to be estimated separately across age groups (Model 4) better fit the data than the fully constrained model (Model 3).

After determining that Model 4 best fitted the data, I then added school as a confounder. In this final model, there was still strong evidence for a positive association between gender and depressive symptoms (coef=0.18, 95% CI=0.11 to 0.25, p<0.001) and perfectionism and depressive symptoms (coef=0.57, 95% CI=0.45 to 0.70, p<0.001). There was no evidence that need for approval was associated with either gender or depressive symptoms (Table 6.5).

In young adolescents, there was no evidence for an association between gender and perfectionism, or an indirect association between gender and depressive symptoms through perfectionism (Table 6.5). However, in mid-adolescents, there was evidence that females had higher perfectionism than males (coef=0.16, 95% CI=0.06 to 0.25, p=0.002) and evidence for an indirect association between gender and depressive symptoms through perfectionism (coef=0.05, 95% CI=0.003 to 0.11). Females had higher perfectionism than males, which was then associated with more severe depressive symptoms (Table 6.5).

Table 6.5 Associations from the structural equation model, adjusted for confounders, separated by age group. Only the parameter for the association between gender and perfectionism was allowed to vary across age groups.

	Model 4: Adjusted, separate age groups (n=559)	
	Coef (95% CI)	p value
Direct association with perfectionism		
Gender		
Young adolescents	0.04 (-0.06 to 0.14)	0.46
Mid adolescents	0.16 (0.06 to 0.25)	0.002
Direct association with need for approval		
Gender	0.07 (-0.02 to 0.16)	0.11
Direct association with depressive symptoms		
Gender	0.18 (0.11 to 0.25)	<0.001
Perfectionism	0.57 (0.45 to 0.70)	<0.001
Need for approval	0.11 (-0.02 to 0.25)	0.10
Indirect association with depressive symptoms		
Gender - perfectionism		
Young adolescents	0.03 (-0.02 to 0.08)	
Mid adolescents	0.05 (0.003 to 0.11)	
Gender - need for approval	0.01 (-0.002 to 0.03)	

Note. Coefficients were standardised. Indirect associations were estimated using bootstrapping (1000 replications) and 95% percentile confidence intervals. All parameters constrained across groups except the association between gender and perfectionism. Model adjusted for continuous age within each age group, testing group size, school, and non-verbal IQ score.

6.3.2.2 Sensitivity analysis: improving parameter estimation

Finally, I used modification indices to obtain the best estimates of parameters for associations in the whole sample in (Model 5; Figure 6.3). Adding covariance parameters between the residuals of some DAS subscale items, as well as some SMFQ items, improved model fit. All model fit indices met the acceptable cut-off criteria (Table 6.2). A likelihood ratio test indicated that this nested model was a better fit than Model 2 ($\chi^2(17) = 405.30$, $p<0.001$).

In this model, evidence was very similar to Model 2 (Table 6.6). However, there was weaker evidence for an association between gender and perfectionism (coef=0.08, 95% CI=-0.004 to 0.17, p=0.06; *hypothesis 4.1*). The evidence for an indirect association between gender and depressive symptoms through perfectionism was also weaker (coef=0.04, 95% CI=-0.01 to 0.09; *hypothesis 4.4*).

Table 6.6 Associations from the structural equation model, adjusted for confounders, with added covariance parameters in the whole sample.

	Model 5: Adjusted with covariances (n=559)	
	Coef (95% CI)	p value
Direct association with perfectionism		
Age group	0.15 (0.05 to 0.25)	0.003
Gender	0.08 (-0.004 to 0.17)	0.06
Direct association with need for approval		
Age group	0.13 (0.03 to 0.23)	0.01
Gender	0.09 (0.001 to 0.18)	0.05
Direct association with depressive symptoms		
Age group	-0.03 (-0.10 to 0.05)	0.51
Gender	0.19 (0.12 to 0.25)	<0.001
Perfectionism	0.59 (0.38 to 0.80)	<0.001
Need for approval	0.11 (-0.12 to 0.35)	0.33
Indirect association with depressive symptoms		
Age group - perfectionism	0.07 (0.02 to 0.13)	
Age group - need for approval	0.01 (-0.02 to 0.05)	
Gender - perfectionism	0.04 (-0.01 to 0.09)	
Gender - need for approval	0.01 (-0.01 to 0.04)	

Note. Coefficients were standardised. Indirect associations were estimated using bootstrapping (1000 replications) and 95% percentile confidence intervals. Model adjusted for continuous age within each age group, school, testing group size, and non-verbal IQ score, with covariance parameters included between DAS subscale and SMFQ item residuals (see statistical analysis or Figure 6.3).

Thus, in this sensitivity analysis, there was evidence that females had higher perfectionism, need for approval, and depressive symptoms than males (*hypothesis 4.1*). Mid-adolescents had higher perfectionism and need for approval than young adolescents (*hypothesis 4.2*). There was also evidence that perfectionism, but not need for approval, was positively associated with depressive symptoms (*hypothesis 4.3*). There was very weak evidence for an indirect association between gender and depressive symptoms, as females had higher perfectionism than males, which was positively associated with depressive symptoms (*hypothesis 4.4*). However, there was also strong evidence for a direct association between gender and depressive symptoms, as females had more severe depressive symptoms than males. Finally, there was no evidence for a direct association between age group and depressive symptoms, but there was evidence for an indirect association between age group and depressive symptoms through perfectionism (but not need for approval). Mid-

adolescents had higher perfectionism than young adolescents, which was then associated with more severe depressive symptoms.

6.4 Discussion

In this study, I aimed to test whether there were gender differences in dysfunctional attitudes in young and mid-adolescents. I found evidence that dysfunctional attitudes were best modelled with two separate factors, perfectionism and need for approval. In line with previous research using the DAS, I hypothesised that perfectionism would be higher in boys and need for approval would be higher in girls (hypothesis 4.1). I also expected dysfunctional attitudes to be present from early adolescence, and thus hypothesised that there would be no association between age group and dysfunctional attitudes (hypothesis 4.2). Contrary to these hypotheses, I found evidence that girls (compared to boys) and mid-adolescents (compared to young adolescents) had higher perfectionism and higher need for approval.

As hypothesised, I found strong evidence for a positive association between perfectionism and depressive symptoms, but I did not find evidence for the expected association between need for approval and depressive symptoms (hypothesis 4.3). Need for approval was also positively associated with depressive symptoms but the effect size was much smaller than for perfectionism, and the confidence intervals were wider, meaning that this association was not statistically significant. I also hypothesised that dysfunctional attitudes would mediate the association between gender and depressive symptoms (hypothesis 4.4). There was weak evidence that perfectionism mediated the association between gender and depressive symptoms. Girls had higher perfectionism than boys, which was associated with more severe depressive symptoms. There was no evidence that need for approval mediated the gender difference in depression.

Additionally, I hypothesised that associations between gender, perfectionism, need for approval, and depressive symptoms would not differ across the two age groups (hypothesis 4.5). In contrast, I found evidence that the association between gender and perfectionism differed across age groups. In young adolescents, there was no evidence for an association between gender and perfectionism. However, in mid-adolescents, there was evidence that girls had higher perfectionism than boys, and this contributed to the association between

gender and depressive symptoms. In both age groups, perfectionism was strongly positively associated with depressive symptoms, but there was no evidence for an association between need for approval and depressive symptoms.

6.4.1 Strengths and limitations

General strengths and limitations of the data included in this study were outlined in chapter 3 (section 3.4.1) and will be discussed in detail in the general discussion (section 7.3).

In this study, I used SEM to test age and gender differences in the latent constructs of perfectionism and need for approval and their associations with depressive symptoms. This approach assumes that the observed information reflects unmeasurable constructs and accounts for measurement error in latent variables by simultaneously estimating measurement and structural models (Bollen, 1989; Kline, 2015). It also allowed me test mediation hypotheses, estimating indirect and direct associations simultaneously, despite using cross-sectional data.

The 17-item DAS has been validated in adults in the general population (De Graaf et al., 2009) and is very similar to another version of the DAS validated in adolescents with depression (Rogers et al., 2009). It had eleven items measuring perfectionism and six items measuring need for approval. It is therefore possible that it measured perfectionism more accurately than need for approval. This could explain why perfectionism was more strongly associated with depressive symptoms than need for approval. It is possible that perfectionism and need for approval are similarly associated with depressive symptoms, but I found more evidence for associations with perfectionism because I measured it with less error. However, more items load onto the perfectionism than need for approval factors in all versions of the DAS (Barnett & Gotlib, 1990; Cane et al., 1986; De Graaf et al., 2009; Imber et al., 1990; Rogers et al., 2009; Zlotnick et al., 1996). Additionally, using SEM should have reduced the impact of measurement error on these estimates of associations with depressive symptoms (Bollen, 1989; Kline, 2015).

However, in this study, I have not assessed measurement invariance by gender, meaning that dysfunctional attitudes and depressive symptoms may not be comparable across males and females. This could occur if gender alters the processes by which latent factors

(perfectionism, need for approval, depressive symptoms) produce differences in item responses (individual questions on the DAS and SMFQ). In CFA, these processes are represented by the item parameters (factor loadings and intercepts; Curran, Cole, Bauer, Hussong, & Gottfredson, 2016). The same CFA can be applied to the data separately for males and females in a multiple-group analysis, and different sets of model parameters constrained to be equal across genders to test whether this alters model fit (e.g. Byrne et al., 1993). If there is evidence that parameters are equivalent across males and females, this indicates measurement invariance, and suggests that the SMFQ and DAS are measuring the same processes in males and females. Testing measurement invariance thus allows us to examine whether males and females interpret the same measure in a conceptually similar way (Bialosiewicz, Murphy, & Berry, 2013).

Although I did not assess measurement invariance, previous studies have found evidence that the two factor model of dysfunctional attitudes is invariant across genders during adolescence (McWhinnie, Abela, Knäuper, & Zhang, 2009; Rogers et al., 2009), and associations between perfectionism and depressive symptoms are gender invariant (Asseraf & Vaillancourt, 2015). However, in most validations of the SMFQ, gender invariance has not been assessed, despite the evidence for gender differences in depressive symptoms. There is evidence that gender has very little impact on the discriminatory validity of the SMFQ, so cut-offs to indicate diagnoses of depression are valid across males and females (McKenzie et al., 2011; Thabrew, Stasiak, Bavin, Frampton, & Merry, 2018; Turner et al., 2014). However, females may overreport the number of depressive symptoms that they are experiencing on the SMFQ compared to males (Turner et al., 2014). This could have led to an overestimation of symptoms among females. Future work should thus assess whether the measurement model included in this study is invariant across males and females for both dysfunctional attitudes and depressive symptoms. If it is not, then further investigation would be required, and scores on the SMFQ and DAS may need to be considered separately for males and females.

It is also possible that this study was underpowered for testing more complex models. A large number of parameters were estimated in models including confounders, meaning the sample did not meet the recommended 10 participants per parameter (Kline, 2015). However, there are no definitive rules on sample size for SEM, and this is a rule of thumb. Results from the

simplest models were very similar to the more complex models. After adding confounders to the model, there was weak evidence for an association between gender and need for approval, which could be a false positive. However, this seems unlikely given that the coefficient and confidence intervals were very similar to the unadjusted analyses. The sample was as large as reasonably possible and was also large enough for models to properly converge. Given the likelihood of even more complex models being underpowered, I did not further adjust analyses for additional confounders.

Although this study was cross-sectional, there is strong evidence from previous longitudinal studies that dysfunctional attitudes are associated with subsequent depressive symptoms in adolescence, even after accounting for baseline depression (Abela & Sullivan, 2003; Hankin et al., 2018; Lewinsohn et al., 2001; Pearson et al., 2015; Pössel, 2017; Rawal et al., 2013b). In this study, I found evidence that gender and age group were associated with dysfunctional attitudes, which cannot be due to reverse causation. Despite being cross-sectional, my findings therefore suggest that dysfunctional attitudes could mediate the association between age or gender and depressive symptoms in adolescence. Longitudinal data would allow the test of a full mediation model, controlling for baseline depressive symptoms.

6.4.2 Findings in context

Some of the strongest evidence in this study was for the association between age group and dysfunctional attitudes. Perfectionism and need for approval were higher in mid-adolescents (13 to 15 years) than young adolescents (11 to 13 years). This was unexpected, as I did not hypothesise that dysfunctional attitudes would increase with age. Previous evidence has been inconsistent, although my findings are in line with some other population-based studies of adolescents aged 9-18 (Hankin, 2008; Meiser & Esser, 2017, 2019). Increases in dysfunctional attitudes during adolescence may be a result of continued cognitive development, allowing individuals to develop stable cognitive styles (Nolen-Hoeksema et al., 1992; 1994). However, I found evidence for associations between perfectionism and depressive symptoms in young and mid-adolescents, in line with most previous studies (D'Alessandro & Burton, 2006; Hankin et al., 2008; Meiser & Esser, 2017, 2019). Thus, even in early adolescence, cognitive styles have developed sufficiently for dysfunctional attitudes to be associated with depressive symptoms. Adolescents may therefore develop more dysfunctional attitudes as their schema

about the self become more negative, alongside the increasingly negative self-evaluations and drop in self-esteem that have been observed during this period (Robins & Trzesniewski, 2005; van der Aar et al., 2018). Alternatively, this increase in dysfunctional attitudes with age could be a result of reverse causation, as it may be driven by increases in depressive symptoms.

I found strong evidence that perfectionism was associated with depressive symptoms. My findings indicate that perfectionism may be more important for depression during adolescence than need for approval. Previous research on perfectionism has also found evidence for associations with depressive symptoms during adolescence (e.g. Asseraf & Vaillancourt, 2015; Flett, Coulter, Hewitt, & Nepon, 2011; Hewitt et al., 2002; Jacobs et al., 2009; O'Connor, Rasmussen, & Hawton, 2010; Soenens et al., 2008). Having very high standards, and never believing that you have achieved enough, could rapidly lead to distress, negative mood, and a lack of motivation and self-esteem. This may be particularly relevant from ages 11 to 15, when individuals may experience increasing academic pressure at school. However, we should also consider whether the association between perfectionism and depressive symptoms is artificially inflated by overlap in the content of these measures. There are several items in the SMFQ and DAS perfectionism subscale which are very similar, including feeling like a failure, never being as good as others, and thinking that you are disliked by others. This could mean that the latent factors of perfectionism and depressive symptoms identified in this study are not independent and instead represent one overall construct. Future work should assess whether perfectionism can be measured independently of depressive symptoms and is a meaningful risk factor for depression.

In my final model, there was no evidence for an association between age group and depressive symptoms, despite preliminary evidence that depressive symptoms were higher in mid- than young adolescents. Instead, I found evidence that perfectionism might mediate the association between age group and depressive symptoms. As adolescents get older, they could become more perfectionistic, which then increases their risk of developing depressive symptoms. This cross-sectional study thus suggests that perfectionism may increase vulnerability to depression and could contribute to the emergence of the gender difference in depression. Longitudinal data is needed to confirm this finding.

However, my findings on gender differences in dysfunctional attitudes were less clear. Overall, I found evidence that perfectionism was higher in girls, and weak evidence that need for approval was also higher in girls. This finding differs to previous studies which have found no evidence for gender differences in overall DAS scores (Abela & Sullivan, 2003; Chen & Li, 2014; Gotlib et al., 1993; Hankin et al., 2018; Lewinsohn et al., 2001; Rawal et al., 2013b; Young et al., 2012), or evidence that boys had more dysfunctional attitudes than girls (Meiser & Esser, 2017, 2019). This finding also differs to one study which showed evidence that boys were more perfectionistic, but found no gender differences in need for approval (Marcotte et al., 2006). Within the dysfunctional attitudes literature, it has repeatedly been proposed that perfectionism is higher in males and need for approval is higher in females, and this has been suggested as a cause of the gender difference in depression (Barnett & Gotlib, 1990; Beck, 1983; De Graaf et al., 2009; Farmer et al., 2001; Meiser & Esser, 2019; Otani et al., 2013; Zlotnick et al., 1996).

This proposed gender difference, as measured by the DAS, may be an artificial distinction. Perfectionism in relation to achievement is often also linked to concern about rejection by others or need for approval (e.g. Burrage et al., 2016). Consistent with this, although my model fit indices indicated that a two-factor model with perfectionism and need for approval latent factors was better than a one-factor model, the correlation between perfectionism and need for approval latent factors was very high. The DAS aims to measure perfectionism about achievement or performance evaluation, but perfectionism also exists in other domains. Adolescents may have perfectionist ideas about their relationships and interactions with peers that could be reflected in higher scores on the DAS perfectionism factor. Additionally, items measuring the construct of perfectionism generally centre around having high personal standards. If these standards are not met, this may lead to feelings of failure and worthlessness. These are states often felt by females. On average, males are more confident and have higher self-esteem (Robins & Trzesniewski, 2005). My finding that girls were more perfectionistic than boys is also consistent with hypotheses within the perfectionism literature, despite the lack of previous evidence for this gender difference (Asseraf & Vaillancourt, 2015; Hewitt et al., 2002; Jaradat, 2013; Rice et al., 2004, 2007; Soenens et al., 2008).

Evidence for the gender difference in perfectionism was only present in mid-adolescents, and not young adolescents. Alongside evidence for associations between age and perfectionism, this indicates that perfectionism increases more in girls than in boys during adolescence. The mid-adolescent group in this study were a similar age (13 to 15 years) to adolescents in a previous study (13 to 16 years) which found evidence for the opposite gender difference in perfectionism (Marcotte et al., 2006). It is unclear why this previous study did not find evidence that perfectionism was higher in girls, particularly given the higher prevalence of depressive symptoms in girls, and the association between perfectionism and depressive symptoms. Marcotte and colleagues recruited a convenience sample from two high schools in an upper middle-class urban area, which could have influenced their findings. However, they did find longitudinal evidence for increases in perfectionism in girls who became depressed over the two-year follow-up (Marcotte et al., 2006). In combination with my findings, this suggests that girls may become more perfectionistic than boys in mid-adolescence, and this could increase their vulnerability to depression.

These findings could be used to further refine the targets of cognitive behavioural therapy (CBT; Beck, 1979, 1983; Clark & Beck, 1999), improving its efficacy and efficiency. Understanding the development of perfectionism may also be important in preventing depression. Prevention strategies which target negative cognitions should consider focussing on perfectionism, as this could reduce vulnerability to depressive symptoms. Although perfectionism may be resistant to change (Flett & Hewitt, 2014), using a more focussed approach to a specific target may make prevention programmes more feasible. By helping perfectionistic adolescents to understand that they are doing well, the targets they are setting for themselves are perhaps too ambitious, and their self-worth should not be defined by their achievements, we could encourage focussing on more positive aspects of the self, reduce negative cognitions, and improve mental health. It is also important to consider the impacts of perfectionism within an academic setting, where it is likely to increase stress and low mood and may reduce motivation if adolescents believe that they are underachieving, particularly in comparison to their peers.

6.4.3 Conclusion

In this chapter, I found evidence that being older and being female was associated with increased perfectionism and need for approval, which was unexpected. I also found evidence that higher perfectionism was associated with more severe depressive symptoms, as hypothesised and as proposed by cognitive models of depression (Beck & Bredemeier, 2016; Roiser et al., 2012). Also as hypothesised, I found some evidence that perfectionism may mediate the association between age and depressive symptoms, as mid-adolescents were more perfectionistic than young adolescents, and this was associated with more severe depressive symptoms. However, this must be interpreted cautiously due to the use of cross-sectional data. In contrast to my hypotheses, there was less strong evidence that need for approval was associated with depressive symptoms, and there was no evidence that need for approval mediated the gender difference in depression. Longitudinal evidence is now required to test whether perfectionism has a causal role in the emergence of depressive symptoms in adolescence.

Chapter 7 Discussion

In this chapter I first summarise the aims of my thesis (section 7.1) and then outline the main findings from my thesis (section 7.2). I then discuss the strengths and limitations of the methods and approaches used and how they may have influenced my findings (section 7.3). Next, I address how my findings relate to previous research on adolescent depression (section 7.4). Finally, I discuss potential implications for research, policy, and clinical practice (sections 7.5 & 7.6).

7.1 Summary of the main aims of my thesis

The main aim of my thesis was to investigate an explanation for the emergence of the gender difference in depressive symptoms during adolescence. I aimed to test three different aspects of negative cognitions - learning about social evaluation, recall of social evaluation, and dysfunctional attitudes. The aims of each study in my thesis were set out in the general introduction and chapters 3-6. Specific hypotheses associated with each aim will be discussed in section 7.2, with an overview of whether my findings provide evidence for each hypothesis. Briefly, the main aims of my thesis were:

1) To investigate learning about social evaluation, examine whether there are gender differences in this learning, explore whether these gender differences change with age, and test whether learning about social evaluation is associated with depressive symptoms in adolescence (chapter 3).

2) To investigate potential processes underlying learning about social evaluation, examine whether there are gender or age differences in these processes, and test whether the processes underlying learning about social evaluation are associated with depressive symptoms in adolescence (chapter 4).

3) To investigate recall of self-referential and other-referential social evaluation, examine whether there are gender differences in this recall, explore whether these gender differences change with age, and test whether recall of social evaluation is associated with depressive symptoms in adolescence (chapter 5).

4) To investigate whether there are gender differences in perfectionism and need for approval, examine whether these gender differences change with age, and test whether perfectionism and need for approval are associated with depressive symptoms in adolescence (chapter 6).

7.2 Summary of the main findings from my thesis

In order to test these aims, I collected data from 331 young adolescents (aged 11-13 years) and 268 mid-adolescents (aged 13-15 years) in a cross-sectional study. These two age groups were chosen to span the age at which rates of depression start increasing (Kwong et al., 2019; Merikangas et al., 2010), and to capture adolescents' transition from early to late puberty (Parent et al., 2003; Patton & Viner, 2007). In this sample (and consistent with prior studies; Kwong et al., 2019; Patalay & Gage, 2019), depressive symptoms were higher in girls than boys, and this gender difference increased with age. Participants completed a battery of social information processing tasks and questionnaires.

7.2.1 Chapter 3: Learning about social evaluation during adolescence: gender differences and associations with depressive symptoms

In chapter 3, I adapted a task, which assessed learning about social evaluation, for use with adolescents. I tested learning in 598 participants. I found evidence for my hypotheses that adolescents would demonstrate a positive self-referential bias in learning (hypothesis 1.1). Adolescents were better at learning that they were liked than disliked. Also as hypothesised, there was evidence that this positive bias was specific to self-referential learning and did not transfer to learning whether characters liked or disliked another person. Adolescents chose the positive personality trait more often when learning about the self than other people. I hypothesised that this positive self-referential bias would be smaller in girls than boys in both young and mid-adolescents (hypothesis 1.2 and 1.3). However, I found no evidence that learning about social evaluation differed across genders or age groups. This could have been due to a lack of power to test these associations.

In this study, I found evidence for my hypothesis that the positive self-referential bias in learning would be negatively associated with depressive symptoms (hypothesis 1.4). Adolescents who were worse at learning they were liked had more severe depressive

symptoms. However, there was some evidence that worse learning that another person was liked was also associated with depressive symptoms, which was unexpected. I hypothesised that the association between the positive self-referential bias and depressive symptoms would not differ across genders or age groups (hypothesis 1.5). Consistent with this, I did not find any evidence that gender or age group moderated the association between the positive self-referential bias and depressive symptoms (although this could also be a result of a lack of power).

I also hypothesised that I would find the same positive self-referential bias, and associations with gender and depressive symptoms, in adolescents' ratings of social evaluation after learning. Contrary to my hypotheses, when asked to reflect on their learning, adolescents did not demonstrate a positive self-referential bias. Instead, they accurately rated whether characters liked or disliked both themselves and others. As with learning, and contrary to my hypothesis, there was no evidence for gender differences in these ratings. Although there was no evidence for a positive self-referential bias, I did find evidence that adolescents who rated that characters liked them less had more severe depressive symptoms (as hypothesised). Also as hypothesised, there was no evidence that this association between ratings and depressive symptoms differed according to gender or age group.

7.2.2 Chapter 4: Computational mechanisms underlying social evaluation learning during adolescence

Building on these preliminary analyses, in chapter 4, I examined computational processes underlying learning about social evaluation in adolescence, investigating how social feedback influences learning and future decisions. To do this, I developed reinforcement learning models parameterising the processes through which adolescents may learn about social evaluation. I tested multiple reinforcement learning models to explain behaviour on the social evaluation learning task, each representing different assumptions about the involvement and interaction of specific cognitive processes.

I hypothesised that a number of parameters would be necessary for this reinforcement learning model to adequately describe adolescents' behaviour, including separate learning rates for self-referential and other-referential information and parameters modelling a positive self-referential bias (hypothesis 2.1). Consistent with this, the best fitting model

indicated that adolescents learnt differently about self-referential and other-referential social evaluation, and also had positive biases in their initial expectations of whether characters would like or dislike them and other people. This positive bias in other-referential learning was unexpected.

I hypothesised that none of these parameters would change with age (hypothesis 2.2). However, there was some evidence for developmental differences in learning about social evaluation, including smaller positive self-referential biases in mid-adolescents than young adolescents, which could be associated with the increase in depressive symptoms in mid-adolescence. I also hypothesised that parameters relating to the positive self-referential bias would be smaller in girls than boys, in both young and mid-adolescents (hypothesis 2.3). But, in this study, I did not find evidence for gender differences in any aspects of learning about social evaluation.

Finally, I hypothesised that the positive self-referential start bias parameter and self-referential learning rate would be associated with depressive symptoms, across both genders and age groups (hypothesis 2.4). Partially consistent with this, a reduction in adolescents' positive self-referential start biases was associated with more severe depressive symptoms, but there was also evidence that a reduced positive other-referential start bias was associated with depressive symptoms. Contrary to my hypothesis, there was no evidence that other processes involved in learning (e.g. self-referential learning rate) were associated with depressive symptoms. This study provides evidence of a potential computational mechanism underlying the association between behavioural performance on this task and depressive symptoms.

7.2.3 Chapter 5: Recall bias during adolescence: gender differences and associations with depressive symptoms

In chapter 5, I tested my theory using another aspect of social information processing. I tested memory for self-referential and other-referential social evaluation using a novel recall task with 578 adolescents. I found evidence to support my hypothesis that adolescents would recall more self-referential than other-referential words (hypothesis 3.1), in all groups except young adolescent girls. However, contrary to my hypothesis that adolescents would recall more self-referential positive than self-referential negative words (hypothesis 3.2), I found

evidence that adolescents had negative biases in recall, remembering more negative than positive personality characteristics in both self-referential and other-referential conditions. I also hypothesised that girls would demonstrate less positive self-referential recall biases than boys, recalling fewer self-referential positive and more self-referential negative words (hypothesis 3.3). I found no evidence for this hypothesised gender difference, overall or within each age group (hypothesis 3.4).

I had hypothesised that positive self-referential recall biases would be negatively associated with depressive symptoms (hypothesis 3.5). By having less positively biased self-referential recall, adolescents may have more negative memories of social interactions and more negative self-concepts, which could encourage social withdrawal and increase depressive symptoms. Although I did not find evidence for a positive self-referential recall bias, there was evidence that adolescents who had less self-referential positive and more self-referential negative recall had more severe depressive symptoms, consistent with my hypothesis. Finally, I hypothesised that this association with depressive symptoms would be consistent across genders and age groups (hypothesis 3.6). In line with this, there was no evidence that the association between recall biases and depressive symptoms differed in young and mid-adolescents. However, I found some evidence that the association between self-referential negative recall and depressive symptoms was stronger in girls than boys. This could mean that, if self-referential negative recall is a risk factor for depressive symptoms, it is more important for girls than boys.

7.2.4 Chapter 6: Dysfunctional attitudes during adolescence: gender differences and associations with depressive symptoms

Finally, in chapter 6, I examined another aspect of negative cognitions in adolescence. I studied dysfunctional attitudes, which are negative beliefs about the self, the world, and the future. In a sample of 567 adolescents, I used structural equation modelling to demonstrate that dysfunctional attitudes were best described by two latent factors, perfectionism and need for approval.

Based on previous research also using the Dysfunctional Attitude Scale (DAS), I hypothesised that perfectionism would be higher in boys and need for approval would be higher in girls (hypothesis 4.1). I also expected dysfunctional attitudes to be present from early adolescence,

and thus hypothesised that there would be no association between age group and dysfunctional attitudes (hypothesis 4.2). In contrast to these hypotheses, I found evidence that girls (compared to boys) and mid-adolescents (compared to young adolescents) had higher perfectionism and need for approval.

Additionally, I hypothesised that higher perfectionism and need for approval would both be associated with more depressive symptoms (hypothesis 4.3). I found evidence that more perfectionism was associated with more severe depressive symptoms, but there was no strong evidence for an association between need for approval and depressive symptoms. Although I was analysing cross-sectional data, I had also hypothesised that dysfunctional attitudes would mediate the association between gender and depressive symptoms (hypothesis 4.4). As I found no strong evidence that need for approval was associated with depressive symptoms, this was unlikely to mediate the gender difference. However, consistent with my hypothesis, there was some evidence to suggest that perfectionism is a potential mediator of the association between gender and depressive symptoms. Girls had higher perfectionism than boys, which was associated with more severe depressive symptoms. Although I did not make a mediation hypothesis about age group, I also found evidence that perfectionism potentially mediated the association between age group and depressive symptoms, albeit in cross-sectional data. Mid-adolescents were more perfectionistic than young adolescents, and higher perfectionism was associated with more severe depressive symptoms.

Finally, I hypothesised that these associations between gender, perfectionism, need for approval, and depressive symptoms would be present from early adolescence, so would not differ across the two age groups (hypothesis 4.5). Consistent with this, when modelling the age groups separately, the association between perfectionism and depressive symptoms was present from early adolescence. However, gender was only associated with perfectionism in mid-adolescents. There was no evidence for a gender difference in perfectionism in young adolescents. This could indicate that the gender difference in perfectionism in mid-adolescents was due to reverse causation, as the higher levels of perfectionism in mid-adolescent girls may have been a result of the more severe depressive symptoms in this group.

My findings from this chapter suggest that perfectionism is more strongly associated with depressive symptoms than need for approval. Perfectionism becomes more prevalent with age, particularly in girls, during adolescence. High levels of perfectionism could be a risk factor for depression, increasing girls' vulnerability to developing depressive symptoms in mid-adolescence.

7.2.5 Overview of all findings from my thesis

Overall, my findings indicate that there may not be gender differences in social information processing during adolescence. Despite this, I did find evidence that both learning about and recall of self-referential social evaluation were associated with depressive symptoms in adolescence. Associations between social information processing and depressive symptoms appeared to be present from early adolescence, so may not change with age, despite the sharp increase in depressive symptoms during this period. There was also very little evidence that the association between social information processing and depressive symptoms differed according to gender.

However, when assessing a different aspect of negative cognitions, I did find evidence for a gender difference in dysfunctional attitudes. Girls had more perfectionism and need for approval than boys. When testing young and mid-adolescents separately, my findings indicated that the gender difference in perfectionism emerged with age. There was evidence that girls had higher perfectionism than boys in mid-adolescence, but not young adolescence. As with social information processing, there was strong evidence that perfectionism was associated with depressive symptoms, and no evidence that this association differed in young and mid-adolescents. In mid-adolescents, there was cross-sectional evidence that perfectionism might mediate the association between gender and depressive symptoms.

7.3 Strengths and limitations of studies in my thesis

Before interpreting these findings further, it is important to evaluate the validity and reliability of the studies described in my thesis. In these studies, I have explored the complex issue of gender differences in depressive symptoms during adolescence, as well as the complicated topic of social information processing biases. In this section, I will discuss the

broad strengths and limitations of these studies in relation to chance, bias, confounding, and reverse causality.

7.3.1 Chance

A key strength of my thesis was the use of a population-based sample which included the full range of depressive symptom severity (from none to severe). Depressive symptoms were analysed continuously, which should have increased my statistical power to detect any associations (Button et al., 2013). Two separate age groups were recruited to study gender differences before and after the age at which depression starts increasing. I also performed a power calculation in the initial planning of my study. This indicated that a sample of 640 participants was required to detect an effect size of 0.4 standard deviations within each age group. I aimed to recruit approximately 160 adolescents of each gender at each age. However, examining the standardised estimates presented in Appendix 1, effect sizes in my studies were mostly smaller than 0.4 standard deviations. My studies may therefore have been underpowered to detect these effects. Additionally my sample size ranged from 567 to 598, so was smaller than the target sample of 640. Subgroup sizes varied, as fewer mid-adolescents participated than young adolescents. I also tested three-way and four-way interactions between task conditions, age group, and gender in chapters 3 and 5. These tests were probably underpowered (Button et al., 2013; Greenland, 1983) with my power to test four-way interactions likely to have been below 50%. By limiting statistical power, this may increase the possibility that my findings are due to chance or that I did not find evidence for an effect which does exist. However, the lack of evidence for gender differences in social evaluation learning in chapter 3 is supported by the absence of evidence for an association between gender and parameters from the reinforcement learning model in chapter 4.

Power is particularly an issue where I hypothesised that gender and age group would not moderate associations between social information processing and depressive symptoms. Using frequentist statistics, testing this hypothesis is only really meaningful when there is sufficient power to reject the null hypothesis (i.e. enough power that small effects would be detected reliably). As my power to detect small effect sizes was low for these interactions, conclusions that gender and age did not moderate associations between social information processing and depressive symptoms should be cautious. Replicating my findings in a larger

study would allow greater confidence in the results (Button et al., 2013), although it would not remove the other limitations with testing interactions, such as the model-dependence of results (Greenland, 1983; Kendler & Gardner, 2010). I did use an epidemiological approach to test cognitive hypotheses, aiming to reduce the possibility that my findings were due to chance. My sample was larger than those often recruited when testing information processing. Given the difficulty and time-consuming nature of collecting data from adolescents in schools, with the need to gain parental consent, this was the largest sample that I could feasibly recruit.

I sought to minimise the possibility of results being due to chance by defining hypotheses *a priori*. I collected data on a battery of tasks and questionnaires, but developed analysis plans and determined which measures would be included in each study before data collection. As data collection took place during lessons, and adolescents were recruited from eight schools in which lesson lengths differed dramatically, I included several additional questionnaires not required for my main analyses. These were intended to keep participants busy until the end of the lesson in all schools, accounting for large individual differences in study completion time, and reducing the likelihood of participants who finished early distracting other students. These additional questionnaires were selected for use with collaborators (e.g. Appendix 2) and in other analyses, not included in my thesis. The inclusion of additional questionnaires therefore should not have increased the probability of findings in my thesis being due to chance. However, the protocol for my cross-sectional study was not registered or made available in a public database, and I did not pre-register hypotheses for individual studies. This is not yet routine practice, but is recommended for improving replication in scientific research, preventing practices such as data-dredging and p-hacking (Gelman & Loken, 2014). Although I know that hypotheses and analysis plans were specified *a priori*, future replications of this work should be pre-registered to demonstrate this.

Additionally, some issues with replication have been attributed to over-reliance on p values (Concato & Hartigan, 2016; Gelman & Loken, 2014). Using the arbitrary cut-off of $p<0.05$ for statistical significance has a number of limitations, and some researchers have suggested that we stop using p values altogether (Kraemer, 2019). In my thesis, I have reported, and interpreted, effect estimates and 95% confidence intervals alongside p values for all analyses. However, caution may be needed when interpreting findings for which there was very weak

evidence (marginally significant findings around p=0.05). These results should be replicated in larger datasets.

A limitation of the nature of my hypotheses was the necessity for multiple comparisons (e.g. testing numerous models including different interaction terms). Throughout my thesis, I did not correct for multiple comparisons. Adjustments such as using a Bonferroni correction are often too conservative, increasing the risk of Type 2 errors, particularly in exploratory analyses (Perneger, 1998; Rothman, 1990; Streiner & Norman, 2011). However, we should be cautious about drawing definitive conclusions given the number of comparisons conducted on the same dataset in my thesis, and the increased probability of a chance finding.

7.3.2 Bias

Another key strength of my thesis was the recruitment of the sample from diverse mixed gender schools across London. This should have been more representative of the general population of adolescents than many previous studies, which rely on a select sample of adolescents from smaller independent (fee-paying) schools. By recruiting my sample from one population and analysing depressive symptoms continuously, I should have reduced bias compared to case-control designs, which are very susceptible to selection bias.

Selection bias may still be a limitation in my study as a result of non-response. I used opt-in parental consent in seven out of eight schools and response rates were poor. The final sample consisted of 33% of the eligible population. In my sample, the majority of participants (76%) were recruited from schools with high parental consent rates (where over 60% of eligible adolescents had parental consent). Participants from schools with low parental consent rates did not differ in terms of age, gender, or depressive symptoms, but did have higher non-verbal IQ score compared to participants from schools with high consent rates, indicating that selection bias may have occurred. However, I do not believe that factors influencing participation would alter associations between age group, gender, negative cognitions, and depressive symptoms, so selection bias should not be an issue. Additionally, opt-out parental consent was used to recruit nearly half of the sample, which should have reduced the likelihood of selection bias occurring.

Parents/carers were asked to complete a questionnaire on their child's demographics, mental health, and special educational needs as well as parental mental health. I intended to include these measures as confounders in all adjusted analyses, but parental report was only available for 58% of the sample. Thus, fully adjusted analyses (chapters 3 and 5) may not have included a representative sample of participants. In order to make comparisons about the effects of these potential confounders, main analyses were repeated just for the subsample of participants whose parents/carers had completed additional measures, allowing the influence of additional potential confounders to be investigated within the same sample. Additionally, information on participants' demographics, mental health, special educational needs, and parental mental health were likely available for a biased sample, meaning I cannot infer these characteristics for my whole sample. More specific implications of adjusting for these potential confounders have been discussed in each study (chapters 3 and 5).

For the subsample whose parents/carers did complete the parental questionnaire, 77% of adolescents were of white ethnicity, 88% had English as their first language, and 88% had high parental education (categorised as A levels or higher). In comparison, in the 2011 census data on adults in Greater London, 60% were of white ethnicity, 88% had English as their first language, and 53% of 25 to 64 year olds had high education (A levels or equivalent or above; Office for National Statistics, 2016). These statistics suggest that my sample was representative in terms of people's first language. However, my sample may have over-represented White participants with highly educated parents/carers (particularly as young people in London are more diverse than adults, who completed the census). However, given this was likely a biased subsample of my total sample, my overall sample should be more representative than indicated by these statistics.

The proportion of participants completing measures decreased substantially from the start to the end of the classroom testing, with completion rates ranging from 100% to 54%. This was particularly an issue in young adolescents, who were generally slower at completing measures. Thus, questionnaires included later in the battery may have been completed by a biased sample of participants who were quicker at responding. This was expected, and I included the key study questionnaires first to maximise response rates. However, I cannot rule out the possibility that this has influenced results due to different patterns of missing data related to exposure status (e.g. age, gender, depressive symptoms). Additionally, only a

small proportion of participants (21%) completed the additional questionnaires at home. I was therefore unable to include measures of puberty, bullying, and emotional and behavioural problems in most analyses. Where these measures were included, findings may have been biased due to missing data.

As all studies in my thesis used questionnaires, self-report bias may also have influenced my findings. Given the stigma associated with mental health problems, and the nature of data collection in classrooms, reporting bias may be a concern. It is possible that participants' responses were influenced by social desirability, although I would not expect this to differ by age group, gender, or other exposures. Given the cross-sectional nature of the study, and the short timeframe to which measures referred, recall bias is unlikely to be an issue. For example, the short Mood and Feelings Questionnaire (SMFQ) tests depressive symptoms over the last two weeks. Additionally, the SMFQ is a commonly used measure of depressive symptoms in epidemiological studies, has been validated for use with adolescents, and demonstrates strong validity and reliability (Angold et al., 1995; Thapar & McGuffin, 1998).

The social information processing tasks were novel as I developed them for my study. They had not previously been used with adolescents. As discussed in chapters 3-5, their psychometric properties (reliability and validity) are therefore generally unknown. The social evaluation learning task has demonstrated moderate test-retest reliability, and stable associations with depressive symptoms, over a short follow-up in adults (Button & Hobbs, 2020). However, I do not know that this reliability is also present in adolescents. As my tasks are novel, it is unclear how their nature may affect performance, and I did not do any reliability testing. It is possible that elements of these measures introduced measurement error into my findings, despite careful task development, piloting and feedback from the participant group. Given that I treated data continuously in my analyses, it is unlikely that measurement error would introduce bias into the associations I tested (as it does in binary data), but it may have increased the variance in my data.

7.3.3 Confounding

In each chapter, I accounted for a range of potentially important confounders, although it was not possible to measure all potential confounders. For example, due to time limitations and the need to test participants in groups, I was not able to include a more detailed measure of

IQ or verbal IQ. I did not ask participants' parents/carers about their finances but used parental education as a proxy for socioeconomic status. It also was not possible to collect data from schools such as whether participants had free school meals and their academic achievement. Although I adjusted for several potential confounders, residual confounding is also possible due to imperfect measurement of these variables.

Throughout my thesis, I have adjusted for non-verbal IQ score, as is common practice in developmental studies. However, as discussed in more detail in chapters 2, 4 and 5, IQ is likely to be on the causal pathway between gender or age group and social information processing. Adjusting for IQ score may therefore bias my estimates of associations between gender or age group and social information processing towards the null (as may have occurred with the associations between age group and social evaluation learning in chapter 4). Collider bias may also be an issue, even though social information processing is unlikely to cause IQ score, as there may be a common cause of both IQ score (the collider) and social information processing (the outcome). If this is the case, including IQ score in analyses may lead to finding spurious evidence for associations, or other unpredictable effects (Cole et al., 2010; Day, Loh, Scott, Ong, & Perry, 2016; Greenland, 2003). However, IQ is an important potential confounder of associations between social information processing and depressive symptoms. Adolescents with lower IQ score may perform more poorly on social information processing tasks, and there is strong evidence that low IQ score is associated with more severe depressive symptoms (Glaser et al., 2011; van Os et al., 1997; Zammit et al., 2004). On balance, I decided that IQ score should be included in analyses, even though it may lead to collider bias or act as a mediator between gender or age group and social information processing.

In developmental research, puberty is also an important potential confounder, which may be on the causal pathway. Puberty may contribute to some of the gender differences in negative cognitions and depressive symptoms. As girls generally start puberty earlier than boys, they are in more advanced stages of puberty during early adolescence (Parent et al., 2003; Patton & Viner, 2007). Pubertal stage is also strongly associated with depressive symptoms (e.g. Angold et al., 1998; Ge et al., 2001; Keenan et al., 2014). If social information processing or dysfunctional attitudes change as a result of puberty, then pubertal stage may explain some of the gender differences in these processes during adolescence.

Although pubertal stage and age are highly correlated, there is some evidence that pubertal hormones and age have functionally dissociable effects on neural activity during social information processing (Goddings et al., 2012). There is also some evidence that puberty is more strongly associated with performance on a self-referential encoding task than age (Ke et al., 2018). This could explain why I did not find evidence for an effect of age on social information processing. In contrast, I did find evidence that dysfunctional attitudes increased with age. This is consistent with a previous study which found no evidence that pubertal stage is associated with dysfunctional attitudes (Bélanger & Marcotte, 2011). Puberty may therefore be most relevant when investigating gender differences in social information processing during adolescence. Low response rates to follow-up questionnaires meant that I was only able to assess the effect of pubertal stage in sensitivity analyses (chapters 3 and 5). This is a limitation but is not uncommon, as most studies in this field measure participants' age and not pubertal status. It is also often difficult to include both of these measures in analyses as they are highly correlated.

Similarly, I had hoped to adjust for parental depression in the whole sample. In my subsample, 20% of parents who completed the questionnaire reported that they or their partner had experienced depression. There is some evidence that girls have a stronger genetic risk for depression during adolescence than boys (Flint & Kendler, 2014; Rice, Harold, & Thapar, 2002; Scourfield et al., 2003; Sullivan, Michael Neale, & Kendler, 2000). If this is the case, parental depression could be a confounder by increasing the risk of depression in girls more than boys. Genetic confounding may have occurred, whereby genetic factors causally affect both the exposure and outcome (Pingault et al., 2018), in this case social information processing and depressive symptoms. Additionally, gene-environment correlations, where the environment adolescents experience is influenced by their genotype (Pingault et al., 2018), may have added to this confounding. Genetic confounding is not something I can rule out in my thesis. Investigating the role of genetics in gender differences in negative cognitions was beyond the scope of my PhD but presents an important avenue for further investigation.

Finally, I originally intended for participants with diagnoses of dyslexia and autism spectrum disorders (ASD) to be excluded from this study, as my tasks involved skills that are known to be affected in these conditions. However, excluding these adolescents was not possible with the low parental questionnaire response rates. This meant that I did not have data on

diagnoses of dyslexia or ASD for 41% of my sample. I also did not think that it was ethical to exclude participants from whole class testing sessions or remove their data after they had participated. The presence of dyslexia or ASD could therefore have confounded associations between age, gender, negative cognitions, and depressive symptoms. I did find some evidence that ASD was associated with more positive responses when learning about social evaluation (chapter 3), and dyslexia was associated with worse recall of social evaluation (chapter 5). However, adjusting for dyslexia and ASD in the subsample whose parents/carers reported additional potential confounders did not substantially alter the evidence for my findings.

7.3.4 Reverse causation

As discussed in section 1.13.2, the studies in my thesis were intended as a first step in testing my proposed mediation model of the gender difference in depression. These studies allowed me to test whether gender was associated with negative cognitions in adolescence. These associations cannot be a result of reverse causation as negative cognitions are unlikely to causally affect participants' gender. As I did not find any evidence that gender was associated with two separate aspects of social information processing, it may not be necessary to invest resources in a prospective cohort study testing this hypothesis. However, given the preliminary evidence indicating that perfectionism may mediate associations between gender (as well as age group) and depressive symptoms, further longitudinal data on these processes would be useful.

In these cross-sectional studies, I could not provide evidence of a causal effect of social information processing or dysfunctional attitudes on depressive symptoms. This causal pathway is proposed by cognitive models of depression (Beck & Bredemeier, 2016; Roiser et al., 2012). My findings are consistent with such models. However, it is equally possible that changes in depressive symptoms cause changes in negative cognitions (reverse causality), or that the association is bidirectional. Longitudinal data is required to test the hypothesis that negatively biased social information processing and dysfunctional attitudes lead to increased depressive symptoms. Longitudinal data would also allow the testing of a full mediation model, with mediators and outcomes tested at separate time points, so that the temporal associations between variables can be specified.

7.4 Findings in context

Throughout my thesis, I have interpreted my findings in the context of cognitive models of depression, as described in chapter 1. In light of this, in the following section, I discuss the meaning of my findings in relation to previous studies of social information processing and dysfunctional attitudes during adolescence. I will also refer to the broader literature on the gender difference in depression and the importance of social cognition during adolescence.

7.4.1 Potential risk factors for depression in adolescence

A key finding from my thesis was the consistent associations between reduced positive self-referential processing and more severe depressive symptoms in a population-based sample of adolescents. In chapters 3 and 5, I found evidence that processing social information specifically about the self, compared to others, may be more strongly associated with depressive symptoms. This is consistent with cognitive models of depression in adults, which state that depressed individuals have negative thoughts and beliefs about themselves and their world (Beck & Bredemeier, 2016; Roiser et al., 2012). However, there was some evidence for associations between other-referential processing and depressive symptoms (chapters 3-5), indicating that these processes may be less clear-cut in adolescents than adults. As adolescence is such an important period of change in peer relationships (see section 1.9), biases in processing social information about others may be more strongly associated with depressive symptoms than in adulthood.

I found no evidence for age differences in the association between social information processing and depressive symptoms in young (aged 11-13 years) compared to mid-adolescents (aged 13-15 years). Although it has been proposed that the association between social information processing biases and depressive symptoms may increase with age (Cole et al., 2008; Dearing & Gotlib, 2009; Turner & Cole, 1994), as individuals develop the ability for more abstract and operational thinking, there is no consistent evidence for this hypothesis (Abela & Hankin, 2008; Platt, Waters, Schulte-Koerne, Engelmann, & Salemink, 2017). This suggests that social information processing biases are associated with depressive symptoms from early adolescence, despite ongoing cognitive development during adolescence. There was also no evidence for age differences in the association between dysfunctional attitudes and depressive symptoms (chapter 6). This finding is consistent with most previous research

on dysfunctional attitudes (D'Alessandro & Burton, 2006; Hankin et al., 2008; Meiser & Esser, 2017; but cf. Rawal et al., 2013b). Overall, it appears that the mechanisms proposed by cognitive models of depression (Beck & Bredemeier, 2016; LeMoult & Gotlib, 2019; Roiser et al., 2012) are relevant throughout adolescence, with robust associations between negative cognitions and depressive symptoms from age 11. Given this, the increase in depressive symptoms during adolescence may not be a result of changes in negative cognitions.

My findings provide strong evidence that biases in cognition are cross-sectionally associated with depressive symptoms. However, the direction of these associations remains a key question. As discussed, cognitive models propose that negative biases are a risk factor for depression, leading to increased depressive symptoms by increasing negative affect, encouraging social withdrawal, and reducing motivation (Beck & Bredemeier, 2016; LeMoult & Gotlib, 2019; Roiser et al., 2012). A review did find some evidence for a causal role of biases in attention and interpretation from studies of adolescents with elevated risk of depression as well as cognitive bias modification paradigms, although they did not find consistent evidence for a causal role of memory biases (Platt et al., 2017). It is possible that the aspects of cognition that I have studied are a risk factor for depressive symptoms, with negative biases increasing adolescents' risk of developing depression. However, even in adults, the direction of associations between negative biases and depressive symptoms remain unclear. Cohort studies of adolescents and adults should include measures of negative cognitions to determine whether biases are a cause or consequence of depressive symptoms.

In my thesis, I have focussed on three specific social information processing biases (learning about social evaluation, recall of social evaluation, and dysfunctional attitudes). There are a number of other elements of cognition which may also be risk factors for depression in adolescence. For example, over-general autobiographical memory is another social information processing bias which could increase vulnerability to depression (Kuyken & Dalgleish, 2011; Rawal & Rice, 2012; Warne et al., 2019). Adolescents' attributional style may also be a risk factor, with those who believe that negative events are one's own fault, will impact all aspects of one's world, and will impact the future as well as attributing negative events to causes that impact self-worth at higher risk of depression (Abramson et al., 1989; Lau & Waters, 2017). In addition to these cognitive risk factors for depression, other risk

factors may exist in a number of domains (such as those described in section 1.2.3), all of which are likely to cumulatively increase vulnerability to depression during adolescence.

7.4.2 Potential neural mechanisms

Once we understand associations between social information processing and depressive symptoms at the behavioural level, we can then study task-related neural activation in functional imaging studies. These should be hypothesis-driven and based on behavioural findings. Diverse changes across a number of brain systems in adolescence are likely to result in shifts in how the brain can attend to, integrate, and retain information (Dahl, Allen, Wilbrecht, & Suleiman, 2018). This may be particularly relevant to neural mechanisms underlying social cognition, as the "social brain" (a complex network of brain regions that participate in understanding and interacting with others) continues to develop throughout adolescence (Burnett et al., 2009, 2011; Sebastian, Viding, et al., 2010; Somerville, 2013). There is also rapid maturation of cortical regions involved in reward processing and emotional regulation and perception (Kerestes, Davey, Stephanou, Whittle, & Harrison, 2014).

Although beyond the scope of my thesis, potential differences in neural mechanisms in adolescents with more (versus less) severe depressive symptoms are of interest. Adolescent depression has been hypothesised to result from a temporal mismatch in the development of brain regions involved in emotional processing and reward processing (e.g. amygdala, striatum) and those involved in the cognitive regulation of emotion (e.g. prefrontal cortex). This is the dual-systems model (e.g. Casey, Jones, & Somerville, 2011; Pfeifer & Allen, 2012). However, this may be overly simplistic, and there is evidence which is inconsistent with this model (see Pfeifer & Allen, 2012 for a review). A systematic review found evidence that, across a variety of social information processing tasks, adolescents with depression most often had abnormal activation in ventromedial frontal regions, the anterior cingulate, and the amygdala (Kerestes et al., 2014). It is therefore possible that alterations in a network of medial prefrontal cortex regions, together with closely related regions, could underlie my findings (as proposed by Price & Drevets, 2010, 2012). However, the effects of age, pubertal stage, and gender on the neural mechanisms underlying adolescent depression remain unclear (Kerestes et al., 2014).

As I found strong evidence for associations between biases in self-referential information processing and depressive symptoms, investigating the neural mechanisms underlying these biases presents a useful avenue for future investigation. Understanding these mechanisms in adolescence is particularly important because it is a period of such rapid development. There are likely to be important and complex interactions between observable behavioural changes and brain development in adolescence. Learning will affect brain development, and maturational changes in the brain will also affect learning and motivation in turn (Dahl et al., 2018). We must therefore investigate both sides of these processes in order to understand what causes the increase in depressive symptoms during adolescence.

7.4.3 Explaining the gender difference in depression

In my thesis, I hypothesised that the higher incidence of depression in girls during adolescence is a result of more negatively biased cognitions in girls than boys. The only evidence I found to support this mediation hypothesis was in my study of dysfunctional attitudes (chapter 6). I found evidence that girls may be more perfectionistic than boys in mid-adolescence, and perfectionism was associated with more severe depressive symptoms (albeit in cross-sectional data). Girls may have perfectionist ideas about their relationships and interactions with peers, as well as high personal standards for their achievements. This is consistent with hypotheses in the perfectionism literature (e.g. Jaradat, 2013; Rice, Kubal, & Preusser, 2004; Starley, 2019), although previous studies have not generally found evidence for this gender difference (Asseraf & Vaillancourt, 2015; Hewitt et al., 2002; Jaradat, 2013; Rice et al., 2004; Rice, Leever, Noggle, & Lapsley, 2007; Soenens et al., 2008).

In my study, there was only evidence for the gender difference in perfectionism in mid-adolescence, which is also when depressive symptoms increase dramatically in girls. This raises the question of whether perfectionism is a cause or consequence of depressive symptoms in adolescence, which requires longitudinal evidence. As discussed, a longitudinal study tested whether negative cognitions mediate the association between gender and depressive symptoms, or whether negative cognitions are a result of increased depressive symptoms (Mezulis et al., 2010). In adolescents aged 11-15 years, gender differences in depressive symptoms emerged before gender differences in negative cognitions, indicating that cognition could not mediate the association between gender and depressive symptoms.

However, this study was relatively small (n=366) and had substantial attrition so the final sample is unlikely to be representative of adolescents in the general population. It also used the Cognitive Style Questionnaire (CSQ), measuring attributions of negative events, as opposed to dysfunctional attitudes (Mezulis et al., 2010). Higher quality longitudinal evidence is thus needed to test whether perfectionism does mediate the gender difference in depression during adolescence.

I did not find any evidence that girls had more negative social information processing than boys during adolescence (chapters 3-5). Social information processing biases were associated with depressive symptoms, but there was no evidence that they differed in girls and boys, despite girls having more severe depressive symptoms. There was some weak evidence that recall biases may be more strongly associated with depressive symptoms in girls than boys (chapter 5), but there was no evidence for a similar pattern in learning about social evaluation (chapters 3-4). To my knowledge, these are the largest tests of gender differences in social information processing in adolescence to date. Given my findings, it is unlikely that learning about or recall of social evaluation are mediators of the association between gender and depressive symptoms during adolescence. However, it is still possible that other aspects of social information processing mediate this association, or that I did not find evidence for gender differences because of a lack of power.

7.4.3.1 Lower-level versus higher-level biases in cognition

There may be a distinction in my findings between implicit biases in automatic processing and explicitly reported cognitions (lower-level and higher-level biases respectively). The social evaluation learning and recall tasks both assessed automatic cognitive processes, which may influence thoughts and behaviours without conscious awareness (Kahneman, 2011; Roiser et al., 2012). In contrast, participants self-reported dysfunctional attitudes, requiring them to consciously reflect on biases in cognition. Biases in implicit automatic processes may differ to more conscious explicit ratings of thoughts and behaviour (Kahneman, 2011; Roiser et al., 2012). I only found evidence for a gender difference in explicit ratings of negative cognitions, and not in implicit social information processing. This could indicate that girls have more negative biases when reflecting on and reporting their thoughts, but not during social interactions or when recalling information from these interactions. However, the social evaluation learning task also distinguished implicit biases in learning from explicit reflections

on learning after meeting each character (chapter 3). I did not find evidence that either of these aspects of learning about social evaluation differed between boys and girls. It therefore remains unclear why both social information processing and dysfunctional attitudes were associated with depressive symptoms in my studies, but only dysfunctional attitudes differed according to gender, even though I found evidence for a gender difference in depressive symptoms.

It is possible that the overlap in content between the explicit measure of cognition and depressive symptoms contributed to the gender difference in dysfunctional attitudes. In my thesis, the measures of dysfunctional attitudes and depressive symptoms contained several similar questions, as discussed in section 6.4.2. These similarities could lead to an artificial inflation of the association between dysfunctional attitudes and depressive symptoms, and increase the evidence for a gender difference in dysfunctional attitudes. In contrast, my measures of implicit social information processing may overlap less in content with explicit reports of depressive symptoms, which could reduce the risk of artificially inflating these associations. This could have contributed to the contrasting evidence for gender differences in implicit and explicit measures of cognition.

Testing how social information processing biases and dysfunctional attitudes are related to each other in adolescence could help us to understand these processes. It is not clear whether changes in lower-level information processing biases may lead to changes in dysfunctional attitudes (as proposed by Roiser et al., 2012), or whether changes in higher-level dysfunctional attitudes cause information processing biases (as proposed by Beck & Bredemeier, 2016). I have not tested these hypotheses in my cross-sectional data, but longitudinal studies would allow tests of how these aspects of cognition relate to each other. It is important for future studies to integrate and compare research on social information processing and dysfunctional attitudes, which are often seen as separate literatures (Jacobs et al., 2008), as I have done in my thesis.

7.4.3.2 Models of the gender difference in depression

Factors other than negative cognitions may be more important for the emergence of the gender difference in depressive symptoms. These factors are numerous and range from key biological mechanisms, such as puberty, hormones and genetics, to early adversities such as

childhood sexual abuse (Angold et al., 1998; Dunn, Gilman, Willett, Slopen, & Molnar, 2012; Fergusson, Swain-Campbell, & Horwood, 2002; Ge et al., 2001; Keenan et al., 2014; Kuehner, 2017; Lewis, McElroy, Harlaar, & Runyan, 2016; Martel, 2013; Scourfield et al., 2003). By demonstrating that biases in social information processing are unlikely to have a key role in the gender difference in depression, my findings indicate that future research should focus on other factors. For example, research could continue to investigate the role of pubertal stage and timing, rumination, genetic risk, body image and dissatisfaction, exposure and susceptibility to stress, and societal gender inequalities. However, it is unlikely that one risk factor for depression is sufficient to cause an increase in depressive symptoms in the absence of other characteristics. Providing models which integrate and describe the additive and multiplicative effects of these factors is therefore a priority.

Developing a comprehensive model of all factors which contribute to the gender difference in depressive symptoms during adolescence is beyond the scope of my thesis. However, it is clear that no adequate model yet exists. The most recent review of causes of the gender difference in depression proposed a number of gender-related subtypes of depression, with the "developmental subtype" most likely to contribute to the gender difference in depression (Kuehner, 2017). Consistent with this, it would be most beneficial for a model to address factors contributing specifically to the emergence of the gender difference in adolescence. It would need to be a biopsychosocial model, integrating risk factors across all domains.

The ABC model of gender differences in depression during adolescence includes affective, biological and cognitive factors (Hyde et al., 2008). This is a diathesis-stress model, which proposes that risk factors are vulnerabilities to depression that, in interaction with negative life events, increase girls' risk of depression from adolescence. However, this model proposes several examples of effect modification, whereby associations between risk factors and depressive symptoms are stronger in girls than in boys. It is not clear why this would occur, as opposed to risk factors being more prevalent in girls than boys. I found no evidence that associations between learning about social evaluation (chapter 3), or dysfunctional attitudes (chapter 6), and depressive symptoms varied according to gender, and only very weak evidence that the association between recall biases and depressive symptoms was stronger in girls than boys (chapter 5). Models of the gender difference in depression therefore should not rely on risk factors which supposedly act differently in boys and girls.

As demonstrated in my thesis, it is particularly important for any proposed theory to account for the role of the social environment in adolescence. Developing a model is important for integrating research to date, understanding the role of developmental factors, and making testable predictions about the gender difference in depression. An overarching model might help to identify mediators which are on the causal pathway between gender and depressive symptoms. This would allow the identification and targeting of specific factors in treatment and prevention strategies for depressive symptoms in adolescence.

7.4.3.3 The role of gender inequality

In my thesis, I outlined the hypothesis that societal gender inequality might result in more negative cognitions in girls during adolescence. However, I did not find strong evidence for more negative social information processing biases in girls than boys during adolescence. We may therefore need to consider other mechanisms through which a lack of societal gender equality may be associated with depression. There is evidence that men have higher self-esteem than women on average (Bleidorn et al., 2016), which is a concept closely related to dysfunctional attitudes. Self-esteem and dysfunctional attitudes may be on the causal pathway between being exposed to negative gender norms and developing depressive symptoms. Social beliefs resulting from gender norms may also contribute in other ways to the increased incidence of depressive symptoms in females. For example, globally, over 40% of people believe that men have more right to a job than women when employment is scarce, and a staggering 28% of people report thinking it is justified for a man to beat his wife (Human Development Report Office, 2020). This demonstrates the high levels of discrimination against females within society.

Here we might be able to learn from research on minority stress theory. This theory states that minority populations experience more stressors as a result of prejudice and discrimination, which have negative impacts on their mental health (Meyer, 2003). Minority stress research has focussed on the LGBTQ+ community, and has demonstrated that perceived discrimination, internalised prejudice, and fear of stigmatisation are associated with psychological distress, including depressive symptoms, and suicide risk (e.g. Meyer, 2003; Tebbe & Moradi, 2016). We could think of females as a more marginalised, less powerful group in society, who experience stressors in a similar manner to minority groups.

This may help us to develop further hypotheses on the role of gender inequality in the emergence of the gender difference in depression.

7.5 Implications

Despite challenges with inferring causality in my thesis, my findings may have implications for the treatment and prevention of depression and for informing policy on mental health and education. The gender difference in the prevalence of depression is one of the most robust findings in psychiatric epidemiology and has been replicated across many cultures (Salk et al., 2017). Young women (aged 16-24 years) were recognised as a group at high risk of common mental disorders, such as depression, in the 2014 UK Psychiatric Morbidity Survey (McManus et al., 2016). Despite the apparent improvements in gender norms in developed societies, with more people championing gender equality, the prevalence of depression is increasing more in young women than young men in the UK (McManus et al., 2016). Changing gender norms and reducing gender inequality is challenging and progress is slow.

Given that my findings are based on cross-sectional data from schools in and around London, we must be cautious about applying them directly to other contexts. Despite this, chapters 3 to 5 add to a body of evidence demonstrating the importance of biases in self-referential social information processing for depressive symptoms in adolescence. Cognitive vulnerability from these biases might be a way to reduce depressive symptoms in adolescence. Interventions that directly target biases in social information processing could increase resilience to stressors and reduce depressive symptoms in girls and boys. Research on cognitive bias modification (CBM) aims to do exactly this, inducing more positive biases in information processing which may then lead to reductions in depressive symptoms (Cristea, Kok, & Cuijpers, 2015; Hallion & Ruscio, 2011). There is some preliminary evidence that CBM may be effective in modifying biases in attention and interpretation during adolescence, but evidence for its efficacy in reducing symptoms remains mixed (Cristea, Mogoaşe, David, & Cuijpers, 2015; Platt et al., 2017). However, my findings could provide new targets for CBM interventions, with new strategies focussing specifically on learning about and recall of social evaluation.

Chapter 6 also provides evidence that negative schema about the self are important for depressive symptoms in adolescence. Dysfunctional attitudes are already a target of cognitive behavioural therapy (CBT; Beck, 1979, 1983; Clark & Beck, 1999), which NICE recommends as the first-line treatment for depression in young people (National Institute for Health and Care Excellence, 2019). My findings indicate that clinicians should focus more specifically on aspects of perfectionism during CBT. Improving treatments for depression such as CBM and CBT is particularly important for adolescents, as there are a limited number of antidepressants which should be prescribed to young people (National Institute for Health and Care Excellence, 2019). Additionally, the efficacy of these antidepressants may be poor (Cipriani et al., 2016), and there is debate about whether they may have adverse effects in adolescence (I. M. Goodyer, 2018).

The prevention of depression in adolescence has also received considerable attention. Adolescence is a formative period and may be a time of malleability, with increased neural plasticity (Monahan, Guyer, Silk, Fitzwater, & Steinberg, 2016). Although these developmental changes may increase the risk of depression, they also provide key potential targets for prevention strategies. In this dynamic maturational period, adolescents' lives can rapidly go in either positive or negative directions (Dahl et al., 2018). It is also a critical time for determining lifestyle trajectories, with patterns of behaviour emerging within education, nutrition, relationships, exercise, and substance use, which have long-term implications for health across the life course (Monahan et al., 2016).

As with intervening to treat adolescent depression, the findings from my thesis present potential targets for prevention strategies in late childhood or very early adolescence. For example, a previous trial of three classroom-based prevention programmes for adolescent depression demonstrated that an intervention which aimed to enhance reward-processing reduced subsequent depressive symptoms (Rice et al., 2015). This involved evaluating the potential risk and rewards of everyday decisions, as well as demonstrating how to use rewarding experiences to improve mood. This trial also tested CBT and Mindfulness-Based Cognitive Therapy interventions, but only the reward-processing program was associated with reduced depressive symptoms (Rice et al., 2015). This indicates that implicit social information processing biases, and the reinforcement learning mechanisms which I have

identified in my thesis, could be amenable to change and lead to subsequent reductions in depressive symptoms.

Adolescent depression is associated with physical health problems, academic problems, impaired social relationships, substance abuse, and high risk sexual behaviour (Birmaher et al., 1996; Horowitz & Garber, 2006; Thapar et al., 2012), as well as increased risk of depression during adulthood (Lewinsohn, Rohde, et al., 2000). Adolescence is therefore a key period in which to prevent depression. Public health strategies could target basic information processing in order to successfully change behaviour and prevent health problems (Marteau et al., 2012). As I found that the prevalence of social information processing biases does not change during adolescence, and biases are present from early adolescence, prevention efforts may need to start in childhood.

Furthermore, my thesis contributes to the evidence on the importance of social cognition during adolescence. Although dysfunctional attitudes about gaining social approval were not associated with depressive symptoms (chapter 6), biases in several aspects of processing social evaluation and perfectionism were associated with depressive symptoms (chapters 3-6). These biases have the potential to influence adolescents' social interactions and mood and should be considered in the context of changing peer relationships. Adolescents spend increasing amounts of time with their peers and, although these experiences tend to be positive, adolescents' relationships are often in a state of flux (Cairns, Leung, Buchanan, & Cairns, 1995). Interactions with parents also remain important and contribute to mental health in adolescence (Dahl et al., 2018). This developmental period thus involves a large reliance on social information processing. Social relationships have wide ranging impacts during adolescence, particularly at school, where they could have a large influence on motivation and achievement in education. Furthering understanding of adolescent development can enable improvements in policies which positively impact lifelong trajectories of health, education, and economic and social success.

7.6 Future directions

A number of key issues for further investigation have emerged from the findings in my thesis, many of which I have already discussed. One area in which research is fundamentally lacking

is longitudinal studies combining approaches from developmental cognitive neuroscience, epidemiology, and computational psychiatry. The emergence of depressive symptoms in adolescence is likely related to a number of maturational processes during development, which should be studied longitudinally. Longitudinal evidence is needed to test whether negative schema and biases in social information processing precede or follow the emergence of depressive symptoms. This should be studied in epidemiological samples which are sufficiently powered, representative, and in which depressive symptoms can be studied continuously. The cognitive tasks studied in my thesis were relatively simple and easy to administer online, so could certainly be used in future cohort studies. Additionally, researchers should consider using tasks which can be computationally modelled, enabling more in-depth investigation of the processes underlying social learning and decision making. This is vital for the field to move towards personalised treatment and prevention strategies.

There are also a number of considerations specific to studying gender differences in depression. Traditionally in psychiatric research, and in my thesis, gender has been treated as a binary variable. Whereas sex is biologically determined, gender is a social construct and is experiential (Galambos, 2004). Gender identity is often established in early childhood and can be described as extent to which people see themselves as masculine and feminine (Galambos, 2004). This is a spectrum, rather than a binary definition of male or female. Future research must consider how to study gender more fluidly, for example by determining the extent to which participants identify as male or female. This may enable more nuanced investigation of gender differences in depression as well as more sensitive research in the context of changing attitudes towards gender. Consistent with this approach, risk factors for depression are likely to be individual vulnerabilities, that vary dimensionally among individuals (girls and boys). Although some risk factors may be more prevalent in girls, they are by no means present in all girls. It would be interesting to determine whether some risk factors also vary with the extent to which adolescents identify as male or female.

We should also be aware of how gender stereotypes might influence our aims and hypotheses. For example, there is a lot of research which suggests that peer relationships are more important for girls' than boys' mental health during adolescence. It is important not to assume that peer relationships do not contribute to boys' mental health, as this is very unlikely. As demonstrated in my analysis of dysfunctional attitudes in chapter 6, some of the

traditional hypotheses about gender roles and gender differences may not be applicable to adolescents today. Although formulating hypotheses based on gender roles may be useful, we should be ready to update our beliefs when studying risk factors for depressive symptoms during adolescence.

My findings do not clearly show that girls have more negative cognitions than boys in adolescence. However, I did not test the impact of gender inequality on these cognitions. My thesis was a preliminary investigation, in which I assumed that girls would experience more negative impacts of gender inequality. I did not include a measure of gender empowerment or gender-based discrimination as I assumed that my sample would be homogenous in terms of societal gender equality. The majority of participants were of white ethnicity and all participants were recruited from Greater London. Most participants were thus likely to have had similar experiences of gender equality during childhood. Additionally, the UK has relatively high gender equality, ranking fifth in the European Union on the Gender Equality Index (European Institute for Gender Equality, 2019). This may mean that gender differences in negative cognitions are less apparent in my sample than they would be in other countries, although I did find robust evidence for a gender difference in depressive symptoms. In order to determine whether gender inequality does lead to more negative cognitions in girls, as I have proposed, cross-cultural investigations are required. Future research could test macro-level gender equality, for example by using the Gender Inequality Index (Gaye, Klugman, Kovacevic, Twigg, & Zambrano, 2010), which measures educational attainment, economic and political participation, and reproductive health issues. Researchers could then examine whether national gender equality is associated with gender differences in dysfunctional attitudes and social information processing biases.

Overall, it is clear that identifying modifiable causes of the gender difference in depression would have enormous health benefits but is still a neglected research priority. There is a crucial need for efforts at both a population and individual level to reduce the prevalence of depression in both girls and boys during adolescence.

7.7 Conclusion

My thesis has included a varied body of work testing the novel hypothesis that girls have more negative biases in social information processing and dysfunctional attitudes than boys. I also tested hypotheses that self-referential negative biases in social information processing and dysfunctional attitudes would be associated with depressive symptoms in young and mid-adolescents. I collected data in a cross-sectional study of young and mid-adolescents and tested these hypotheses using performance on cognitive tasks, computational modelling, and self-reported dysfunctional attitudes. My research addressed a substantial gap in the literature, aiming to link cognitive theories of depression to the gender difference in depressive symptoms during adolescence.

A key finding was that negative self-referential biases in learning about social evaluation and recall of social evaluation, as well as more perfectionism, were associated with more severe depressive symptoms during adolescence. This indicates that, consistent with cognitive models of depression, self-referential processing is strongly associated with depressive symptoms in adolescence and could be a risk factor for the emergence of depressive symptoms. My thesis also presented the first evidence suggesting that there may not be gender differences in some types of social information processing across early and mid-adolescence, despite the gender difference in depressive symptoms. Findings from my thesis demonstrate a number of new priorities for future research, including the importance of longitudinal evidence, and the consideration of alternative causes of the gender difference in depressive symptoms during adolescence.

Dissemination

Publications

At the time of submission, an adaptation of the following chapters have been published:

Chapter 1: Bone, J.K., Lewis, G., & Lewis, G. (2020) The role of gender inequalities in adolescent depression. *The Lancet Psychiatry, 7,* 471–472. See Appendix 2.

Chapter 5: Bone, J.K., Lewis, G., Roiser, J.P., Blakemore, S.-J., & Lewis, G. (2020) Recall bias during adolescence: gender differences and associations with depressive symptoms. *Journal of Affective Disorders.* See Appendix 2.

Two further papers are in preparation for publication:

Chapters 3-4: Bone, J.K., Pike, A., Lewis, G., Roiser, J.P., Blakemore, S.-J., & Lewis, G. Learning about social evaluation during adolescence: computational mechanisms, gender differences and associations with depressive symptoms.

Chapter 6: Bone, J.K., Lewis, G., & Lewis, G. Dysfunctional attitudes during adolescence: gender differences and associations with depressive symptoms.

Conference presentations

I have presented findings from chapter 3 as oral and poster presentations at international conferences including the following: I Aegina Summer School on Social Cognition (Greece) and Flux Congress (New York). I had planned to present findings from chapters 4-6 at the British Association for Psychopharmacology Summer Meeting (London), European Psychiatric Association Section of Epidemiology & Social Psychiatry (Cambridge), and Flux Congress (Santa Rosa) but these were cancelled due to Covid-19.

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Appendix 1 Standardised estimates for chapters 3 to 5

To facilitate interpretation and comparison of my findings, I have included standardised estimates for all analyses in chapters 3 to 5 of my thesis. All continuous variables were standardised by subtracting the sample mean and dividing by the sample standard deviation for each observed value of the variable. I then repeated all analyses using these standardised variables. In order to include standardised variables, which contain both positive and negative values, I used linear multilevel models and linear regression models. Analyses in chapters 3 and 4 were not modified but where negative binomial and Poisson mixed models were used in chapter 5, I replaced these with linear multilevel models. The table numbers in this Appendix are identical to the corresponding table in the chapters 3 to 5 of my thesis. I have not included findings from chapter 6 in this appendix as those analyses were standardised.

Chapter 3: Learning about social evaluation during adolescence: gender differences and associations with depressive symptoms

Supplementary Table 3.2 Linear multilevel models testing the associations between age group, gender, condition (self-referential/other-referential), rule (like/dislike; exposures), and positive responses or global ratings (in standard deviation units).

	Unadjusted models (n=587)		**Adjusted models** (n=586)	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Outcome: Positive responses				
Condition	0.11 (0.05 to 0.16)	<0.001	0.11 (0.06 to 0.16)	<0.001
Rule	-1.54 (-1.60 to -1.49)	<0.001	-1.55 (-1.60 to -1.50)	<0.001
Age group	-0.01 (-0.06 to 0.04)	0.73	-0.02 (-0.08 to 0.04)	0.43
Gender	0.003 (-0.05 to 0.05)	0.90	0.002 (-0.05 to 0.05)	0.94
Outcome: Global ratings				
Condition	-0.02 (-0.08 to 0.03)	0.38	-0.02 (-0.08 to 0.03)	0.38
Rule	-1.37 (-1.42 to -1.31)	<0.001	-1.37 (-1.42 to -1.31)	<0.001
Age group	0.02 (-0.05 to 0.09)	0.56	-0.001 (-0.08 to 0.08)	0.98
Gender	-0.03 (-0.10 to 0.04)	0.38	-0.03 (-0.10 to 0.03)	0.32

Supplementary Table 3.4 Linear regression models testing change in depressive symptoms (in standard deviation units) for each standard deviation increase in positive responses or global ratings in each condition.

	Model 1: unadjusted (n=594)		**Model 2: adjusted** (n=582)	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Exposures: Positive responses				
Self like	-0.13 (-0.22 to -0.05)	0.001	-0.16 (-0.24 to -0.08)	<0.001
Self dislike	-0.12 (-0.20 to -0.04)	0.01	-0.10 (-0.18 to -0.02)	0.02
Other like	-0.13 (-0.21 to -0.04)	0.003	-0.13 (-0.22 to -0.05)	0.003
Other dislike	-0.04 (-0.13 to 0.04)	0.29	-0.05 (-0.13 to 0.03)	0.24
Exposures: Global ratings				
Self like	-0.16 (-0.25 to -0.08)	<0.001	-0.17 (-0.26 to -0.09)	<0.001
Self dislike	-0.09 (-0.17 to -0.005)	0.04	-0.08 (-0.16 to 0.01)	0.07
Other like	-0.04 (-0.12 to 0.05)	0.42	-0.04 (-0.12 to 0.05)	0.42
Other dislike	-0.03 (-0.11 to 0.06)	0.52	-0.02 (-0.10 to 0.07)	0.67

Supplementary Table 3.6 Linear multilevel models testing associations between age group, gender, condition (self-referential/other-referential), rule (like/dislike; exposures), and positive responses or global ratings (in standard deviation units). Models include only the subsample of participants for whom additional confounders were available (n=283).

	Adjusted models		**Additionally adjusted models**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Outcome: Positive responses				
Condition	0.07 (0.004 to 0.14)	0.04	0.07 (0.004 to 0.14)	0.04
Rule	-1.71 (-1.78 to -1.64)	<0.001	-1.71 (-1.78 to -1.64)	<0.001
Age group	-0.03 (-0.11 to 0.05)	0.45	-0.04 (-0.12 to 0.04)	0.32
Gender	0.02 (-0.05 to 0.09)	0.59	0.02 (-0.05 to 0.09)	0.56
Outcome: Global ratings				
Condition	-0.003 (-0.08 to 0.07)	0.94	-0.003 (-0.08 to 0.07)	0.94
Rule	-1.47 (-1.55 to -1.40)	<0.001	-1.47 (-1.55 to -1.40)	<0.001
Age group	0.02 (-0.08 to 0.12)	0.73	0.01 (-0.09 to 0.11)	0.84
Gender	-0.01 (-0.10 to 0.07)	0.78	-0.01 (-0.10 to 0.08)	0.86

Supplementary Table 3.8 Linear regression models testing change in depressive symptoms (in standard deviation units) for each standard deviation increase in positive responses or global ratings in each condition, including only the subsample of participants for whom additional confounders were available (n=281).

	Model 1: adjusted		**Model 2: additionally adjusted**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Exposures: Positive responses				
Self like	-0.18 (-0.30 to -0.05)	0.006	-0.18 (-0.31 to -0.06)	0.004
Self dislike	-0.07 (-0.19 to 0.05)	0.26	-0.08 (-0.20 to 0.04)	0.20
Other like	-0.12 (-0.26 to 0.02)	0.10	-0.11 (-0.25 to 0.03)	0.11
Other dislike	0.02 (-0.10 to 0.14)	0.71	0.04 (-0.08 to 0.16)	0.47
Exposures: Global ratings				
Self like	-0.15 (-0.29 to -0.01)	0.04	-0.14 (-0.28 to 0.001)	0.05
Self dislike	-0.04 (-0.17 to 0.09)	0.52	-0.04 (-0.17 to 0.09)	0.50
Other like	-0.01 (-0.16 to 0.13)	0.86	-0.01 (-0.16 to 0.13)	0.85
Other dislike	-0.01 (-0.14 to 0.12)	0.88	0.01 (-0.12 to 0.14)	0.86

Chapter 4: Computational mechanisms underlying social evaluation learning during adolescence

Supplementary Table 4.5 Linear regression models testing associations between age group, gender, and depressive symptoms and model parameters (in standard deviation units, tested in separate models). N=598.

	Unadjusted models		**Adjusted models**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Outcome: self-referential learning rate (α_{self})				
Age group	0.09 (-0.07 to 0.26)	0.25	-0.04 (-0.22 to 0.15)	0.70
Gender	0.11 (-0.05 to 0.27)	0.17	0.09 (-0.08 to 0.25)	0.30
Depressive symptoms	0.02 (-0.07 to 0.10)	0.71	0.01 (-0.07 to 0.09)	0.84
Outcome: other-referential learning rate (α_{other})				
Age group	0.13 (-0.03 to 0.30)	0.11	0.10 (-0.08 to 0.28)	0.26
Gender	0.15 (-0.01 to 0.31)	0.07	0.16 (-0.005 to 0.32)	0.06
Depressive symptoms	-0.07 (-0.15 to 0.01)	0.09	-0.07 (-0.15 to 0.01)	0.08
Outcome: inverse temperature (β)				
Age group	0.25 (0.09 to 0.41)	0.002	0.12 (-0.05 to 0.30)	0.18
Gender	0.09 (-0.07 to 0.25)	0.26	0.03 (-0.12 to 0.19)	0.66
Depressive symptoms	-0.05 (-0.13 to 0.03)	0.19	-0.06 (-0.14 to 0.01)	0.10
Outcome: self-referential start bias (γ_{self})				
Age group	-0.07 (-0.23 to 0.09)	0.42	-0.17 (-0.36 to 0.01)	0.07
Gender	0.07 (-0.09 to 0.23)	0.39	0.07 (-0.10 to 0.24)	0.42
Depressive symptoms	-0.16 (-0.24 to -0.08)	<0.001	-0.17 (-0.25 to -0.09)	<0.001
Outcome: other-referential start bias (γ_{other})				
Age group	0.03 (-0.13 to 0.19)	0.69	0.003 (-0.19 to 0.19)	0.98
Gender	0.05 (-0.11 to 0.22)	0.52	0.04 (-0.12 to 0.21)	0.60
Depressive symptoms	-0.14 (-0.22 to -0.06)	0.001	-0.14 (-0.22 to -0.06)	0.001

Chapter 5: Recall bias during adolescence: gender differences and associations with depressive symptoms

Supplementary Table 5.2 Linear multilevel models testing associations between age group, gender, condition (self-/other-referential) and valence (positive/negative; exposures) and total hits (in standard deviation units).

	Unadjusted models (n=567)	**Adjusted models** (n=566)	**Additionally adjusted for depressive symptoms** (n=566)
	Coef (95% CI)	Coef (95% CI)	Coef (95% CI)
Gender x condition			
Males: condition	0.16 (0.07 to 0.25)	0.16 (0.07 to 0.25)	0.16 (0.07 to 0.25)
Females: condition	0.02 (-0.08 to 0.13)	0.02 (-0.08 to 0.13)	0.02 (-0.08 to 0.13)
Gender x valence			
Males: valence	0.15 (0.06 to 0.25)	0.16 (0.07 to 0.25)	0.16 (0.07 to 0.25)
Females: valence	0.19 (0.08 to 0.29)	0.19 (0.08 to 0.29)	0.19 (0.08 to 0.29)
Gender x condition x valence			
Males self-ref: valence	0.13 (0.01 to 0.25)	0.13 (0.02 to 0.25)	0.13 (0.02 to 0.25)
Males other-ref: valence	0.18 (0.06 to 0.29)	0.18 (0.06 to 0.29)	0.18 (0.06 to 0.29)
Females self-ref: valence	0.14 (0.02 to 0.25)	0.14 (0.02 to 0.25)	0.14 (0.02 to 0.25)
Females other-ref: valence	0.24 (0.11 to 0.36)	0.24 (0.11 to 0.36)	0.24 (0.11 to 0.36)
Age group x gender			
Young: gender	0.15 (0.005 to 0.29)	0.09 (-0.04 to 0.22)	0.06 (-0.07 to 0.19)
Mid: gender	0.32 (0.16 to 0.48)	0.33 (0.18 to 0.48)	0.32 (0.17 to 0.48)
Age group x gender x condition			
Young males: condition	0.18 (0.06 to 0.31)	0.18 (0.06 to 0.31)	0.18 (0.06 to 0.31)
Young females: condition	-0.09 (-0.23 to 0.06)	-0.09 (-0.23 to 0.06)	-0.09 (-0.23 to 0.06)
Mid males: condition	0.13 (-0.01 to 0.26)	0.13 (-0.01 to 0.26)	0.13 (-0.01 to 0.26)
Mid females: condition	0.14 (-0.02 to 0.29)	0.14 (-0.02 to 0.29)	0.14 (-0.02 to 0.29)
Age group x gender x valence			
Young males: valence	0.14 (0.02 to 0.26)	0.14 (0.02 to 0.27)	0.14 (0.02 to 0.27)
Young females: valence	0.22 (0.08 to 0.36)	0.22 (0.08 to 0.36)	0.22 (0.08 to 0.36)
Mid males: valence	0.17 (0.04 to 0.31)	0.17 (0.04 to 0.31)	0.17 (0.04 to 0.31)
Mid females: valence	0.15 (-0.01 to 0.30)	0.15 (-0.01 to 0.30)	0.15 (-0.01 to 0.30)

Supplementary Table 5.3 Linear regression models testing change in depressive symptoms (in standard deviation units) for each standard deviation change in self-referential positive, self-referential negative, other-referential positive, and other-referential negative hits.

	Model 1: Unadjusted (n=578)			**Model 2: Adjusted (n=566)**		
	Coef	95% CI	p value	Coef	95% CI	p value
Self-referential hits						
Positive	-0.11	-0.20 to -0.01	0.02	-0.12	-0.22 to -0.03	0.01
Negative	0.15	0.06 to 0.25	0.002	0.15	0.05 to 0.24	0.003
Other-referential hits						
Positive	0.03	-0.06 to 0.12	0.57	0.01	-0.08 to 0.11	0.79
Negative	0.11	0.02 to 0.20	0.02	0.08	-0.02 to 0.17	0.11

Supplementary Table 5.5 Linear multilevel models testing associations between age group, gender, condition (self-/other-referential) and valence (positive/negative) and total hits (in standard deviation units), in subsample with additional confounders reported (n=275).

	Model 1: Adjusted		**Model 2: Additionally adjusted**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Condition	0.09 (-0.01 to 0.20)	0.09	0.09 (-0.01 to 0.20)	0.09
Valence	0.15 (0.04 to 0.25)	0.01	0.15 (0.04 to 0.25)	0.01
Age group	0.41 (-0.05 to 0.87)	0.08	0.43 (-0.02 to 0.88)	0.06
Gender	0.26 (0.12 to 0.39)	<0.001	0.26 (0.12 to 0.39)	<0.001

Supplementary Table 5.7 Linear regression models testing change in depressive symptoms (in standard deviation units) for each standard deviation change in self-referential positive, self-referential negative, other-referential positive, and other-referential negative hits, in subsample with additional confounders reported (n=275).

	Model 1: Adjusted		**Model 2: Additionally adjusted**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Self-referential hits				
Positive	-0.11 (-0.24 to 0.01)	0.08	-0.09 (-0.22 to 0.04)	0.16
Negative	0.21 (0.07 to 0.36)	0.003	0.21 (0.07 to 0.36)	0.005
Other-referential hits				
Positive	-0.004 (-0.13 to 0.12)	0.95	-0.004 (-0.13 to 0.12)	0.96
Negative	0.02 (-0.12 to 0.16)	0.79	0.01 (-0.13 to 0.15)	0.84

Supplementary Table 5.8 Linear multilevel models testing associations between age group, gender, condition (self-/other-referential) and valence (positive/negative; exposures) and total hits (in standard deviation units), in subsample with pubertal stage reported (n=117).

	Model 1: Adjusted		**Model 2: Additionally adjusted**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Condition	0.03 (-0.13 to 0.18)	0.74	0.03 (-0.13 to 0.18)	0.74
Valence	0.20 (0.04 to 0.36)	0.01	0.20 (0.04 to 0.36)	0.01
Age group	0.12 (-0.53 to 0.76)	0.73	0.22 (-0.45 to 0.89)	0.52
Gender	0.28 (0.08 to 0.48)	0.006	0.33 (0.11 to 0.55)	0.003

Supplementary Table 5.10 Linear regression models testing change in depressive symptoms (in standard deviation units) for each standard deviation change in self-referential positive, self-referential negative, other-referential positive, and other-referential negative hits, in subsample with pubertal stage reported (n=117).

	Model 1: Adjusted		**Model 2: Additionally adjusted**	
	Coef (95% CI)	p value	Coef (95% CI)	p value
Self-referential hits				
Positive	-0.08 (-0.31 to 0.13)	0.44	-0.09 (-0.31 to 0.14)	0.44
Negative	0.16 (-0.08 to 0.40)	0.18	0.17 (-0.07 to 0.41)	0.17
Other-referential hits				
Positive	-0.01 (-0.23 to 0.22)	0.95	-0.02 (-0.24 to 0.21)	0.88
Negative	0.15 (-0.08 to 0.39)	0.20	0.17 (-0.07 to 0.42)	0.16

Appendix 2 Publications from my thesis

At the time of submission, I had published three papers using data from my thesis. The key information from these papers is included here.

Bone, J.K., Lewis, G., & Lewis, G. (2020) The role of gender inequalities in adolescent depression. *The Lancet Psychiatry, 7,* 471–472. https://doi.org/10.1016/S2215-0366(20)30081-X

THE LANCET
Psychiatry
Volume 7, Issue 6, June 2020, Pages 471-472

Comment

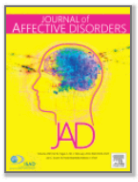
The role of gender inequalities in adolescent depression

Jessica K Bone [a] ✉, Gemma Lewis [a], Glyn Lewis [a]

Bone, J.K., Lewis, G., Roiser, J.P., Blakemore, S.-J., & Lewis, G. (2020) Recall bias during adolescence: gender differences and associations with depressive symptoms. *Journal of Affective Disorders.* https://doi.org/10.1016/j.jad.2020.12.133

ELSEVIER

Journal of Affective Disorders
Available online 28 December 2020
In Press, Journal Pre-proof



Recall bias during adolescence: gender differences and associations with depressive symptoms

Jessica K. Bone [1] ✉, Gemma Lewis [1], Jonathan P Roiser [2], Sarah-Jayne Blakemore [2, 3], Glyn Lewis [1]

Andrews, J.L., Foulkes, L.E., Bone, J.K. & Blakemore, SJ. (2020) Amplified concern for social risk in adolescence. *Brain Sciences, 10,* 397. https://doi.org/10.3390/brainsci10060397

brain sciences

MDPI

Article

Amplified Concern for Social Risk in Adolescence: Development and Validation of a New Measure

Jack L. Andrews [1,*], Lucy E. Foulkes [1], Jessica K. Bone [2] and Sarah-Jayne Blakemore [1,3]

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Received: 24 May 2020; Accepted: 17 June 2020; Published: 23 June 2020

Abstract: In adolescence, there is a heightened propensity to take health risks such as smoking, drinking or driving too fast. Another facet of risk taking, social risk, has largely been neglected. A social risk can be defined as any decision or action that could lead to an individual being excluded by their peers, such as appearing different to one's friends. In the current study, we developed and validated a measure of concern for health and social risk for use in individuals of 11 years and over (N = 1399). Concerns for both health and social risk declined with age, challenging the commonly held stereotype that adolescents are less worried about engaging in risk behaviours, compared with adults. The rate of decline was steeper for social versus health risk behaviours, suggesting that adolescence is a period of heightened concern for social risk. We validated our measure against measures of rejection sensitivity, depression and risk-taking behaviour. Greater concern for social risk was associated with increased sensitivity to rejection and greater depressed mood, and this association was stronger for adolescents compared with adults. We conclude that social risks should be incorporated into future models of risk-taking behaviour, especially when they are pitted against health risks.

Keywords: adolescence; social risk; health risk; depression; rejection sensitivity

Appendix 3 Social evaluation learning task word pairs

I selected a total of 40 positive and negative word pairs to be presented in the social evaluation learning task. These words were:

Positive	Negative
happy	sad
good	bad
nice	mad
cute	slow
friendly	angry
sweet	dumb
gentle	loud
amazing	selfish
neat	dirty
brave	lonely
charming	annoying
interesting	disrespectful
likable	unfriendly
joyful	bossy
cheery	whiny
generous	greedy
peaceful	unkind
forgiving	jealous
positive	creepy
grateful	impolite
pleasant	cruel
cheerful	strict
trustworthy	guilty
outgoing	fearful
talented	frustrating
dependable	hopeless
cool	boring
polite	trouble
kind	stupid
helpful	difficult
funny	grumpy
important	dangerous
powerful	bossy
wonderful	dreadful

Positive (continued)	Negative (continued)
hardworking	lazy
terrific	awful
lovely	rude
lovable	unpopular
agreeable	dull
encouraging	pushy

Appendix 4 Study documents

1. Parent/carer opt-in information sheet

2. Parent/carer opt-out information sheet

3. Participant information sheet

4. Parent/carer opt-in consent form (paper version)

5. Parent/carer questionnaire (paper version)

6. Participant assent form (online version)

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Jessica Bone
020 7679 9051, jessica.bone.15@ucl.ac.uk

Development of Cognitive Processing During Adolescence

Dear Parent/Carer,

We would like to invite your child to participate in our research, which is being conducted by the Developmental Cognitive Neuroscience Group at University College London. This PhD research project is being led by Professor Sarah-Jayne Blakemore and Jessica Bone, and aims to increase our understanding of how and why some young people develop mental health problems.

This study has been approved by the UCL Research Ethics Committee (Project ID 3453-001).

Participation is entirely voluntary. Before you decide whether your child may take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. A member of the research team can be contacted if you would like more information.

What is the study about?

We are interested in understanding whether the way that young people think about their social relationships is associated with their mental health.

Why are we doing this study?

Mental health problems like anxiety and depression become more common during secondary school, but we do not know why. We think that young people's relationships with their friends might be important. We are interested in whether the way that young people think about their friendships is associated with their mental health. This is important for understanding why young people develop mental health problems.

What will happen in the study?

The study involves members of our research team working in your child's school alongside their usual teacher. Researchers are fully trained with enhanced Disclosure and Barring Service (DBS) clearance.

Your child will be asked to complete some computer tasks. These will include some questionnaires about themselves and their mood, a computer game that involves learning whether a computer character likes or dislikes them, and a brief cognitive ability test. The entire session will take no more than 60 minutes and will be completed as an activity in class.

We will send your child some questions about puberty and friendships to fill in online at home. These should not take longer than ten minutes to complete. Your child's school will also provide some information directly to the research team about whether your child receives free school meals, academic attainment, and school funding.

We would like to note again that your child's participation is voluntary. Choosing not to take part will not disadvantage your child in any way and your child will still participate in usual school routines.

Who can take part in the study?

Young people in several secondary schools in London have been invited to take part in this research. You may wish to consider whether it would be distressing for your child to take part if they have been diagnosed with developmental conditions, including dyslexia and autism spectrum disorders. This is because our tasks involve skills that are known to be affected in these conditions.

What are the risks of taking part?

There are no known risks associated with taking part in this study. It is possible that your child may find the computer tasks challenging. Parts of the questionnaires may be upsetting for your child to complete. However, your child's teacher and trained members of the

research team will be present whilst these are completed. Your child will be debriefed after the session and can withdraw from the study at any time without giving a reason. If any problems do arise, we will raise these with the wellbeing and safeguarding staff at your child's school.

What are the possible benefits of taking part?

All tasks are designed to be fun and enjoyable. We hope that this work will help us understand how and why people develop depression. If your child completes all of the questionnaires, they can enter a prize draw to win a £50 Amazon voucher.

What will happen to my child's contact details and other personal information?

The data will be stored securely in compliance with the General Data Protection Regulation (GDPR). The legal basis used to process your child's personal data will be your consent. All data will be made anonymous.

What will happen to the results of this research?

The data will be stored securely at the Division of Psychiatry at University College London. The research team will take all reasonable steps to protect your child's privacy. Their data will be coded and will not be associated with their name or any other information that could reveal their identity.

The study results might be published in scientific journals and/or presented at scientific conferences. Your child's name and identity will not be revealed in any publications. Completely de-identified, anonymised data files will be shared online in accordance with open-science practices.

The recorded data will be kept for up to ten years, or longer if it is needed to fulfil scientific journal publication requirements. After this time, it will be destroyed in compliance with the GDPR.

What if something went wrong?

If you or your child have any concerns about any aspect of the study, please contact Jessica Bone (details below). If you feel the research team has not dealt with your concerns to your satisfaction you may contact the Chair of the UCL Ethics Committee at ethics@ucl.ac.uk.

What do I need to do for my child to take part?

Please complete the attached consent form and return it to your child's form tutor via your child. Alternatively, you can complete the same consent form online using this website address:
http://bit.ly/parent-carer-consent

Can I change my mind?

Participation is completely voluntary. You or your child can withdraw from the study at any time without giving a reason. If the researchers have already collected your child's data, it will not be used in the final study and will be destroyed as soon as you or your child withdraw from the study.

How do I find out more about the study?

You can contact Jessica Bone on 020 7679 9051 or jessica.bone.15@ucl.ac.uk.

Data Protection Privacy Notice

The data controller for this project will be University College London (UCL). The UCL Data Protection Office provides oversight of UCL activities involving the processing of personal data and can be contacted at data-protection@ucl.ac.uk. UCL's Data Protection Officer can also be contacted at data-protection@ucl.ac.uk. Your child's personal data will be processed for the purposes outlined in this notice. If you are concerned about how your child's personal data is being processed, please contact UCL in the first instance at data-protection@ucl.ac.uk. If you remain unsatisfied, you may wish to contact the Information Commissioner's Office (ICO). Contact details, and details of rights, are on the ICO website: https://ico.org.uk/for-organisations/data-protection-reform/overview-of-the-gdpr/individuals-rights/

Thank you for reading this information sheet and for considering your child's participation in this study.

UCL Institute of Cognitive Neuroscience
17 Queen Square,
London WC1N 3AR

Prof Sarah-Jayne Blakemore
020 7679 1131, s.blakemore@ucl.ac.uk

Jessica Bone
020 7679 9051, jessica.bone.15@ucl.ac.uk

Development of Cognitive Processing During Adolescence

Dear Parent/Carer,

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This study has been approved by the UCL Research Ethics Committee (Project ID 3453-001).

Participation is entirely voluntary. Before you decide whether your child may take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. You can contact a member of the research team for more information.

What is the study about?
We are interested in understanding whether the way that young people think about their social relationships is associated with their mental health.

Why are we doing this study?
Mental health problems like anxiety and depression become more common during secondary school, but we do not know why. We think that young people's relationships with their friends might be important. We are interested in whether the way that young people think about their friendships is associated with their mental health. This is important for understanding why young people develop mental health problems.

What will happen in the study?
The study involves members of our research team working in your child's school alongside their usual teacher. Researchers are fully trained with enhanced Disclosure and Barring Service (DBS) clearance.

Your child will be asked to complete some computer tasks. These will include some questionnaires about themselves and their mood, a computer game that involves learning whether a computer character likes or dislikes them, and a brief cognitive ability test. The entire session will take no more than 60 minutes and will be completed as an activity in class.

We will send your child some questions about puberty and friendships to fill in online at home. These should not take longer than ten minutes to complete. Your child's school will also provide some information directly to the research team about whether your child receives free school meals, academic attainment, and school funding.

We would like to note again that your child's participation is voluntary. Choosing not to take part will not disadvantage your child in any way and your child will still participate in usual school routines.

Who can take part in the study?
Young people in several secondary schools in London have been invited to take part in this research. You may wish to consider whether it would be distressing for your child to take part if they have been diagnosed with developmental conditions, including dyslexia and autism spectrum disorders. This is because our tasks involve skills that are known to be affected in these conditions.

What are the risks of taking part?
There are no known risks associated with taking part in this study. It is possible that your child may find the computer tasks challenging. It is also possible that your child might be upset by parts of the questionnaires. However, we have chosen questions that are often used in research with young people. In our experience it is very unusual for these questions or tasks to upset participants. Your child's teacher and trained members of the research team will be present whilst these are completed. Your child will be debriefed after the session and can withdraw from the study at any time without giving a reason. If any

problems do arise, we will raise these with the wellbeing and safeguarding staff at your child's school.

What are the possible benefits of taking part?
All tasks are designed to be fun and enjoyable. We hope that this work will help us understand how and why people develop depression. If your child completes all of the questionnaires, they can enter a prize draw to win a £50 Amazon voucher.

What will happen to my child's contact details and other personal information?
The data will be stored securely in compliance with the General Data Protection Regulation (GDPR). All data will be pseudonymised and made anonymous where possible. The legal basis used to process your child's personal data will be task in the public interest. This means that you do not need to provide your consent for the use of your child's personal data in this project.

What will happen to the results of this research?
The data will be stored securely at the Division of Psychiatry at University College London. The research team will take all reasonable steps to protect your child's privacy. Their data will be coded and will not be associated with their name or any other information that could reveal their identity.

The study results might be published in scientific journals and/or presented at scientific conferences. Your child's name and identity will not be revealed in any publications. Completely de-identified, anonymised data files will be shared online in accordance with open-science practices.

The recorded data will be kept for up to ten years, or longer if it is needed to fulfil scientific journal publication requirements. After this time, it will be destroyed in compliance with the GDPR.

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What if something went wrong?
If you or your child have any concerns about any aspect of the study, please contact Jessica Bone (details below). If you feel the research team has not dealt with your concerns to your satisfaction you may contact the Chair of the UCL Ethics Committee at ethics@ucl.ac.uk.

Does my child have to take part in the study?
If you DO NOT want your child to take part in this study, please contact Jessica Bone via email (jessica.bone.15@ucl.ac.uk) or telephone (020 7679 9051) or online using this website address: http://bit.ly/parent-carer-opt-out
You can also send a letter to:
Jessica Bone
UCL Division of Psychiatry,
6th Floor, Maple House
149 Tottenham Court Road, London W1T 7BN

You must indicate that you DO NOT want your child to take part in this research by [DATE].

If you do not contact Jessica Bone or complete the online form, we will assume you are happy for your child to take part. There is no obligation to take part, and your decision for your child to participate or not participate will not disadvantage your child in any way. Even if you are happy for your child to take part, they will still decide for themselves. It will be explained to your child that they can choose to withdraw from the study at any time, without giving a reason. We want to make sure that everyone who takes part in this research project is happy to do so.

Can I change my mind?
Participation is completely voluntary. You or your child can withdraw from the study at any time without giving a reason. If the researchers have already collected your child's data, it will not be used in the final study and will be destroyed as soon as you or your child withdraw from the study.

How do I find out more about the study?
You can contact Jessica Bone on 020 7679 9051 or jessica.bone.15@ucl.ac.uk.

Thank you for reading this information sheet and for considering your child's participation in this study.

UCL Institute of Cognitive Neuroscience
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Prof Sarah-Jayne Blakemore
020 7679 1131, s.blakemore@ucl.ac.uk

Jessica Bone
020 7679 9051, jessica.bone.15@ucl.ac.uk

Development of Cognitive Processing During Adolescence

Dear student,

We would like to invite you to take part in our research, which is being carried out by the Developmental Cognitive Neuroscience Group at University College London. This PhD research project is being led by Professor Sarah-Jayne Blakemore and Jessica Bone, and aims to increase our understanding of how and why some young people develop mental health problems.

This study has been approved by the UCL Research Ethics Committee (Project ID 3453-001).

Taking part is totally voluntary. Before you decide whether to take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. You can talk to a member of the research team or your teacher if you would like more information or have any questions.

What is the study about?

We are interested in understanding whether the way that young people think about their social relationships is associated with their mental health.

Why are we doing this study?

Mental health problems like anxiety and depression become more common during secondary school, but we do not know why. We think that young people's relationships with their friends might be important. We are interested in whether the way that young people think about their friendships is associated with their mental health. This is important for understanding why young develop mental health problems.

What will happen in the study?

Our research team are working in your school with your teachers. You will be asked to complete some tasks on the computer in class. These will include some questionnaires about your feelings and experiences, a short cognitive ability test, and some computer games. The entire session will not take longer than 60 minutes.

We will also send you some short questionnaires about puberty and friendships to fill in online at home. These should not take you longer than ten minutes.

Why have I been chosen?

Young people in several secondary schools in London have been invited to take part. Your parent/carer has given their consent for you to take part.

Do I have to take part?

No. It is up to you to decide whether or not you take part. Choosing not to take part will not disadvantage you in any way and you will continue to participate in the usual school routines.

What if I get upset by the research?

You might find some of the computer tasks or questionnaires difficult. You can talk to a study researcher or your teacher at any time before,

during or after taking part in the study if you have any concerns. You can drop out from the research at any time without giving a reason.

What are the benefits of taking part?

All of the computer tasks were designed to be fun and enjoyable. You will be helping us to understand how and why young people develop depression.

If you complete the questionnaires at home, you will be entered into a prize draw to win a £50 Amazon voucher.

What will happen to my information?

The data will be stored securely in compliance with the General Data Protection Regulation. The legal basis used to process your personal data will be your consent. All data will be made anonymous.

What will happen to the results of this research?

The data will be stored securely at the Division of Psychiatry at University College London. The research team will take all reasonable steps to protect your privacy. Their data will be coded and will not be associated with their name or any other information that could reveal their identity.

The study results might be published in scientific journals and/or presented at scientific conferences. Your child's name and identity will not be revealed in any publications. Completely de-identified, anonymised data files will be shared online in accordance with open-science practices.

The recorded data will be kept for up to ten years, or longer if it is needed to fulfil scientific journal publication requirements. After this time, it will be destroyed in compliance with the General Data Protection Regulation.

What if something went wrong?

If you have any concerns about any aspect of the study, please contact Jessica Bone (details below). If you feel the research team has not dealt with your concerns to your satisfaction you may contact the Chair of the UCL Ethics Committee at ethics@ucl.ac.uk.

What do I need to do to take part?

Please come to the research session and complete the assent form online.

Can I change my mind?

Participation is completely voluntary. You can withdraw from the study at any time without giving a reason. If the researchers have already collected your data, it will be destroyed as soon as you withdraw from the study.

How do I find out more about the study?

You can always talk to your teacher. If you have any other questions you can contact the study researcher, Jessica Bone:
Telephone: 020 7679 9051
Email: jessica.bone.15@ucl.ac.uk

Thank you for reading this information sheet and for thinking about taking part in this study.

UCL Institute of Cognitive Neuroscience
17 Queen Square,
London WC1N 3AR

Prof Sarah-Jayne Blakemore
020 7679 1131, s.blakemore@ucl.ac.uk

Jessica Bone
020 7679 9051, jessica.bone.15@ucl.ac.uk

Parent/Carer Consent Form: Development of Cognitive Processing During Adolescence

Please only complete this form after you have read the Parent/Carer Information Sheet.

This study is conducted by Professor Sarah-Jayne Blakemore (s.blakemore@ucl.ac.uk, 020 7679 1131) and Jessica Bone (jessica.bone.15@ucl.ac.uk, 020 7679 9051). This study was approved by the UCL Research Ethics Committee (Project ID 3453-001).

The UCL data protection officer can be contacted at pals.data.protection@ucl.ac.uk.

I confirm that by ticking each box below I am consenting to this element of the study. I understand that it will be assumed that unticked boxes mean that I DO NOT consent to that part of the study and, by not giving consent for any one element, my child may be deemed ineligible for the study.

	Tick box
I confirm that I have read and understood the Parent/Carer Information Sheet. I have had an opportunity to consider the information and what will be expected of my child. I have had the chance to ask questions which have been answered to my satisfaction.	
I consent to the processing of my child's personal information (and any information collected as part of this project) for the purposes explained to me and my child. I understand that such information will be handled in accordance with all applicable data protection legislation.	
I understand that all personal information will remain confidential and that all efforts will be made to ensure my child cannot be identified. I understand that my child's data gathered in this study will be stored anonymously and securely. It will not be possible to identify my child in any publications.	
I understand that my child's information may be subject to review by responsible individuals from University College London (UCL) and the Economic and Social Research Council for monitoring and audit purposes.	
I understand that my child's participation is voluntary and that they or I are free to withdraw at any time without giving a reason. I understand that if they or I decide to withdraw, any personal data provided up to that point will be deleted unless they or I agree otherwise.	
I understand the potential risks of participating and the support that will be available to my child should they become distressed during the course of the research.	
I understand the benefits of participating as stated in the Information Sheet.	
I understand that my child's data will not be made available to any commercial organisations.	

I understand that my child may be compensated financially for the time spent in the study, even if they or I choose to withdraw their participation.	
I am aware of whom I should contact if I wish to discuss any aspect of the study.	
I voluntarily agree for my child to take part in this study.	
I understand that the data provided in this study will be archived at UCL for 10 years in accordance to General Data Protection Regulation.	
I understand that other authenticated researchers will have access to my child's fully anonymised data.	
I agree that my child's fully anonymised research data may be used by others for future research.	

Would you like your name to be retained so that your child can be contacted by UCL researchers who would like to invite them to participate in follow up studies to this project, or in future studies of a similar nature? Please tick the appropriate box below.

Yes, I would be happy to be contacted in this way.	
No, I would not like to be contacted.	

_____ _____ _____

Parent/carer name Date Signature

_____

Your child's name

Please now complete the questions on the following page.

UCL Institute of Cognitive Neuroscience
17 Queen Square,
London WC1N 3AR

Prof Sarah-Jayne Blakemore
020 7679 1131, s.blakemore@ucl.ac.uk

Jessica Bone
020 7679 9051, jessica.bone.15@ucl.ac.uk

Questions for Parents/Carers of Participants

We will now ask you some questions about yourself, your child's parents, and your child. These questions should not take more than 2 minutes to complete.

Please only complete this form after you have read the Parent/Carer Information Sheet and completed the Parent/Carer Consent Form. Please circle the correct responses.

Please answer the following questions about **yourself**.	
What is your gender?	Male Female Other (please specify) _____
Your relationship to the child you are completing this for?	Mother Father Grandmother Grandfather Sister Brother Aunt Uncle Carer Other (please specify) _____
Please answer the following questions about the **mother of your child** (even if this is not you).	
What is their highest qualification?	No formal qualifications O-level / GCSE / equivalent AS level / A-level / equivalent Undergraduate degree (e.g. BA / BSc) Postgraduate degree (e.g. MA / MSc / PhD) I don't know Other (please specify) _____
Have they ever had any of the following problems? *(Please circle all that apply)*	Depression Anxiety Stress No – none of the above I don't know
Please answer the following questions about the **father of your child** (even if this is not you).	

What is their highest qualification?	No formal qualifications O-level / GCSE / equivalent AS level / A-level / equivalent Undergraduate degree (e.g. BA / BSc) Postgraduate degree (e.g. MA / MSc / PhD) I don't know
Have they ever had any of the following problems? *(Please circle all that apply)*	Depression Anxiety Stress No – none of the above I don't know
Please answer the following questions about **your child**.	
What school is your child attending?	
What school year is your child in?	Year 7 Year 8 Year 9 Year 10 Year 11
What is your child's gender?	Female Male Other (please specify) _____
What is your child's ethnicity?	White: British White: Irish White: Gypsy or Irish Traveller Mixed: White and Black Caribbean Mixed: White and Black African Mixed: White and Asian Asian / Asian British: Indian Asian / Asian British: Pakistani Asian / Asian British: Bangladeshi Asian / Asian British: Chinese Black / Black British: African Black / Black British: Caribbean Other: Arab Other (please specify) _____
What is your child's first language?	

Has your child ever been diagnosed with special educational needs?	Yes No I don't know
If yes, please circle all of the needs that have been diagnosed	Attention deficit (hyperactivity) disorder (ADHD/ADD) Autistic spectrum disorder (including Asperger Syndrome) Dyslexia Emotional and behavioural difficulties (EBD) Epilepsy Other (please specify) _____
Has your child ever been diagnosed with any mental health problems?	Yes No I don't know
If yes, please select all of the problems that have been diagnosed	Anxiety Conduct disorder Depression Eating disorder Generalised anxiety disorder Panic attacks or panic disorder Post-traumatic stress disorder (PTSD) Social phobia or social anxiety Substance misuse disorder Other (please specify) _____
Has your child ever been seen by mental health services?	Yes No I don't know
Is your child currently taking antidepressant medication?	Yes No I don't know
Is your child currently receiving psychological therapy for depression?	Yes No I don't know

Thank you for completing these questions. Your child will get to participate in this research study at school in the next few months.

If you have any questions or concerns, please refer to the study information sheet or contact the study researcher:

Jessica Bone
UCL Division of Psychiatry, 6th Floor, Maple House, 149 Tottenham Court Rd, London W1T 7NF
Telephone: 020 7679 9051.
Email: jessica.bone.15@ucl.ac.uk

Participant Assent Form: Development of Cognitive Processing During Adolescence

Please only complete this form after you have read the Participant Information Sheet.

This study is conducted by Professor Sarah-Jayne Blakemore (s.blakemore@ucl.ac.uk, 020 7679 1131) and Jessica Bone (jessica.bone.15@ucl.ac.uk, 020 7679 9051). This study was approved by the UCL Research Ethics Committee (Project ID 3453-001).

The UCL data protection officer can be contacted at pals.data.protection@ucl.ac.uk.

I confirm that by ticking the boxes below I am agreeing to each part of the study. I understand that un-ticked boxes mean that I do not agree to that part of the study. I know that by not agreeing with any one part, I may not be able to take part in the study.

- [ ] I have read and understood the Participant Information Sheet. I have been able to think about the information and what will be expected of me. I have also been able to ask questions and I am happy with the answers.
- [ ] I agree that my data can be used in the ways described in the Information Sheet. I understand that my information will be used according to all of the relevant laws.
- [ ] I understand that all of my personal information will remain confidential, and all efforts will be made so that I cannot be identified. My data will be stored anonymously and securely. No-one will be able to link my data to me or identify me from reports.
- [ ] I understand that my anonymous information might be looked at by responsible people from UCL or the Economic and Social Research Council to check what the researchers are doing.
- [ ] I understand that it is up to me whether I take part and I can drop out at any time without giving a reason. If I drop out, my data will be deleted unless I agree that the researchers can still use it.
- [ ] I understand the potential risks of taking part and the support that will be available to me if I were to get upset. No risks are expected for this study.
- [ ] I understand the benefits of taking part as explained in the Information Sheet.
- [ ] I know who I should contact if I want to talk about any part of the research.
- [ ] I voluntarily agree to take part in this study.
- [ ] I understand that other researchers will have access to my fully anonymous data.
- [ ] I agree that my fully anonymised research data may be used by others for future research.

Would you like your name to be kept so that you can be contacted by UCL researchers who would like to invite you to take part in follow up studies, or in similar future studies? Please tick the appropriate box below.

- ◯ Yes - I would be happy to be contacted in this way.
- ◯ No - I would not like to be contacted.

Appendix 5 Previous evidence on memory biases in adolescence

This table provides an overview of previous studies testing positive and negative memory biases in adolescence. Most studies use a standard encoding and incidental recall task. In this task, participants view positive and negative personality trait adjectives and rate whether they describe the self in a self-referential encoding task (SRET). This is followed by a surprise memory test in which participants must recall or recognise as many of the words as possible. Memory biases are measured differently across studies, and the measures used in each study are described in the relevant task column.

Study	Sample	Age	Gender	Design	Relevant task	Relevant findings
Alloy 2012	N=413 community sample depressive symptoms assessed continuously	12-13 M=13 SD=1	Mixed	Cross-sectional	SRET with self-referent and structural judgments. Incidental free recall immediately after. Measured ratio of correctly recalled negative self-referent words to the total number of self-referent words (positive recall not used because it was inverse of negative recall ratio).	Endorsing fewer positive and more negative words as self-descriptive was associated with higher depressive symptoms, controlling for anxiety symptoms. Stronger association for negative words. Females correctly recalled more words than males. Negative recall was associated with depressive symptoms. Overall correct recall and recall of negative self-referent words were associated with current depression diagnoses before but not after controlling for comorbid diagnoses. Higher recall of negative self-referent words associated with more depressive symptoms in all subgroups except African American males.
Asarnow 2014	N=91 low risk N=60 high risk	9-14 M=12 SD=2	Female	Case-control	SRET after negative mood induction. Immediate recall. Measured proportion of positive	No main effects of risk group. Evidence of an interaction for positive words (not negative): significant main effect of COMT genotype in high risk group, but not low risk. Girls with both a family

	also split by COMT genotype				and negative words endorsed as self-referent that were recalled.	history of depression and two COMT met alleles recalled a lower proportion of positive endorsed words than did high-risk girls with two COMT val alleles and low-risk girls with two COMT met alleles. High-risk girls who were homozygous val carriers recalled a higher proportion of positive endorsed words than did high-risk girls who were homozygous met carriers.
Auerbach 2016	N=31 community sample depressive symptoms assessed continuously	13-18 M=15 SD=2	Female	Longit. 3-month follow-up	SRET with EEG. Distractor task of counting backward from 50, then free recall. Surprise recall at first time. Positive processing bias score calculated by dividing the number of positive words recalled by the total number positive and negative words endorsed (negative processing bias calculated in same way).	Participants endorsed and recalled more positive than negative words at each assessment, and these effects were stable over time. Faster RTs when endorsing self-relevant positive words, as opposed to negative words, at both the initial and follow-up assessment. Did not test associations with depressive symptoms (all participants healthy and symptom scores low).
Black 2013	N=92 community sample depressive symptoms assessed continuously	13-15 M=14 SD=1	Mixed	Longit. 6-month follow-up	Dysphoric mood induction, then SRET with negative words only. Immediate free recall. Participants told that recall of the SRET words would be tested. Number of recalled self-referent negative words was divided by the total number of self-referent negative words.	Negative recall was not associated with depressive symptoms at time 2 after controlling for baseline depressive symptoms. Found evidence for an interaction between negative recall and ruminative brooding on depressive symptoms at time 2. Ruminative brooding had less influence on depressive symptoms when there was a high negative recall bias.
Cole 1995	N=87	9-15 M=12	Mixed	Cross-sectional	Modified SRET, words rated as self-referential within 5 domains	Evidence for a group x age interaction on negative recall: the difference between groups in negative

	community sample selected and split based on low or high depressive symptoms	SD=2			(academic, social, athletic, conduct, and appearance). The same words were presented in all 5 domains. Distractor game for approx. 1min then free recall. Measured number of self-referential positive/negative words recalled, and total number of positive/negative words endorsed.	recall got larger with age. At grade 4 the groups were not different, at grade 6 the high depression group recalled more negative words than the low-depression group, and this difference was even larger by grade 8. There was no evidence that this was because depressive symptom scores increased with age. Main effect of group for positive recall: the high-depression group recalled fewer positive words than the low-depression group across all 3 grades.
Cole 2014	N=214 recruited on basis of repeated or no peer victimisation	8-13 M=12 SD=1	Mixed	Case-control	Negative mood induction then modified SRET - words judged as self-descriptive, descriptive of a significant other, or valence rated. Surprise recall immediately after, then recognition test. Measured proportion of negative minus proportion of positive words recalled in each condition.	Non-victimized youths recalled more positive than negative words, but youths who experienced peer victimisation recalled positive and negative self-referential words equally well. Results for the recognition task were similar but the evidence was weaker. Peer victimisation only affected words judged as self-referential.
Connolly 2016	N=291 community sample depressive symptoms assessed continuously	12-13 M=12 SD=1	Mixed	Longit. 9-month follow-up	SRET with self-referential and structural word judgments. Incidental free recall test immediately after. Recall measured as total number of positive/negative self-referential words which were initially endorsed and recalled across both conditions.	Those with more depressive symptoms endorsed more negative and fewer positive words as self-descriptive. All participants endorsed at least one positive word as self-descriptive, but 27% did not endorse any negative words as self-referential and 43% did not endorse any positive words as not self-referential. Time 1 and 2 depressive symptoms were associated with increased recall of self-referential negative words, and decreased recall of positive words (although weaker association for positive).

						Recalling a lower proportion of positive self-referential words at time 1 was associated time 2 depression (but not negative recall).
Dainer-Best 2018	N=572 college N=293 adults N=270 adolescents depressive symptoms assessed continuously	college M=19 SD=1 adults M=38 SD=11 adols M=13 SD=1	Mixed	Cross-sectional	SRET, then participants asked to pause and relax for 1min, then surprise recall. Measured recall of self-referential words and overall recall. Also calculated number of positive/negative words endorsed, mean RT for endorsing positive/negative words, relative starting point and drift rate from drift diffusion model (using RTs and responses) for positive/negative words.	Overall, participants endorsed and recalled more positive than negative words. Drift rate showed participants easily rated positive words as self-referent and negative words as non-self-referent. Self-referential recall of negative words only (not positive words) was often a strong predictor of depression severity, although it was chosen in none of the best models. Recall metrics were worse at predicting depression severity than parameters from the encoding task.
Dalgleish 2003	N=19 with depression N=24 with PTSD N=24 with GAD N=26 controls all from clinics	7-18	Mixed	Case-control	Words (threat, depression, happy, neutral, trauma related), presented on computer screen for 7 secs and participants told to memorise them, repeat each word three times, and think about whether the word made sense to them. Participants then counted aloud in twos for 1.5mins, then did recall test. Measured number of threat- and depression-related words recalled minus number of neutral words.	Main effect of word type: more depression than threat words recalled. No evidence for a main effect of group or group x word type interaction. Depression group did recall highest number of depression words, but there was no strong evidence for this difference. Did not test performance for positive words.

Fattahi Asl 2015	N=28 high risk N=28 with MDD N=29 healthy controls	11-17	Mixed	Case-control	Participants said positive, neutral and negative words aloud and then counted backwards in 2s from 30. Then surprise recall test. Measured number of words of each valence recalled.	Evidence for an effect of group on negative recall: participants with MDD recalled most, then high risk, then healthy controls. Group effect for positive words: healthy controls recalled the most, but no difference between those with MDD or high-risk group. No group differences in neutral word recall. No correlation between neutral word recall and depressive symptoms, but positive correlation for negative words, and negative correlation for positive words.
Gencoz 2001	N=58 all psychiatric inpatients	9-17 M=14 SD=2	Mixed	Cross-sectional	SRET and then recall task. Recall measured as percent of words endorsed as self-referential that were recalled. Words that were not endorsed but were recalled were not included in calculation of recall scores.	Number of endorsed positive words correlated negatively with depressive symptoms, positive correlation for negative words. Number of recalled positive or negative words not correlated with depressive symptoms. In overall model, only positive word endorsement and positive recall were negatively associated with depressive symptoms. Findings did not change after excluding participants younger than 13.
Hammen 1984	N=61 community sample grouped by low vs high depressive symptoms	7-12 split: young M=8 old M=10	Mixed	Cross-sectional	SRET with self-referential and structural word judgments. Surprise recall immediately after. Measured proportion of words recalled out of total number endorsed for positive/negative self and structural.	High depression group judged more negative words as self-referent than low depression group, vice versa for positive words. Better recall of self-referential than structural words, and better recall of words rated as self-referent than not. Older group recalled more self-referential words than younger. Low depression group recall was facilitated for both positive words they described as like them and for negative words they said were not like them. They recalled more positive than negative words. No

						evidence for these effects for high depression group, who had about equal recall of positive and negative self-referential words.
Ho 2018	N=155 community sample depressive symptoms assessed continuously	12-16 M=14 SD=1	Mixed	Cross-sectional	Directed forgetting paradigm - participants presented with positive/negative/neutral words and told to either remember or forget them. Did a 3min distractor task and then recognition task.	More severe depression was associated with more forgetting of neutral words, particularly when instructed to do so. Overall, negative words were recognised better than positive or neutral (no difference between these), especially in the to be remembered condition. Depression was not associated with positive words. For negative and neutral words, more depressive symptoms were associated better intentional (instructed) forgetting.
Holt 2016	N=56 with MDD N=30 healthy controls	11-17	Mixed	Case-control	Social word categorisation task. Participants indicated whether they would be pleased or upset if they were to be described according to each word presented. Immediately followed by a word recognition task.	Overall accuracy was very high. No evidence for group differences in categorisation accuracy, memory sensitivity during recognition task (d'), or RTs to positive of negative words. Older participants were faster in both encoding and recognition tasks. Participants were more likely to remember negative than positive words and this difference increased with age.
Hughes 1990	N=322 community sample classified as depressed vs not using symptom cut-off	10-13 M=11	Mixed	Cross-sectional	Stories task. Participants given a story of a child's day with 10 positive and 10 negative events. Told to read along with the story and remember what they could as they would be tested. After 5min delay, did free recall of positive and negative events	In free recall, more negative events were recalled, and this did not differ according to depressive symptoms. Overall recognition was higher for positive events. Depressed group recognised fewer events overall than non-depressed group. Evidence for a group x valence interaction: depressed group recognised fewer negative events than non-depressed group. Overall more positive vs negative false alarms. Depressed group made more false

					embedded within the story, and then recognition test.	alarms overall. Weak evidence for a valence x group interaction on false alarms, as depressed group made more positive false alarms than non-depressed group.
Kelvin 1999	N=102 community sample subdivided according to level of emotionality	14-15	Mixed	Cross-sectional	Used musical mood induction before completing "assessment of mood activated latent self-schema". Questionnaire involved rating self-descriptors according to how participants thought or felt about themselves in their current frame of mind. Immediate incidental word recall.	Fewer positive and more negative words endorsed as self-referential after dysphoric mood induction vs neutral. Increased proportion of participants recalling 2+ negative words after dysphoric induction. Evidence for a correlation between depressive symptoms and negative word recall after dysphoric and neutral inductions. No correlation between depressive symptoms and positive recall for either mood state. No evidence for any gender differences.
Kuiper 1982	N=12 mildly depressed N=12 non-depressed university students	M=19	Mixed	Case-control	Participants rated “depressed content” and “non-depressed-content” words as descriptive of the self, a well-known other, and their concept of the average person. Immediately followed by incidental recall.	In ratings, self-referential positive words were processed more quickly than negative by healthy group, whereas depressed group showed no difference in ratings. Depressed group’s RTs were slower for positive words than the healthy group. Healthy participants recalled more positive than negative self-referential words, but no difference for those with mild depression. Depressed group recalled more negative words than healthy group, but no difference in positive words. Healthy participants recalled more negative words about both other-referent targets than about themselves. Depressed group recalled fewer negative words about both other-referent targets than healthy group.

McArthur 2019	N=623 community sample 80% completed at least one follow-up	M=13 SD=1	Mixed	Longit. 7-year follow-up 1 wave per year	SRET with a question prompting participants to make either a self-referent or structural judgment. Incidental free recall immediately after with a max of 5mins for recall. Positive self-schema score was the proportion of positive words both rated as self-descriptive and subsequently recalled relative to all words rated as self-descriptive (and same for negative). Task completed at every time.	Multilevel growth curve modelling results demonstrated no mean-level change for positive self-schemas over follow-up. The slope of negative self-schemas was best approximated by a quadratic growth model, suggesting that negative self-schemas increase from early adolescence (age 13) until middle adolescence (ages 16/17), decreasing thereafter. Positive and negative self-schema trajectories did not vary by gender. However, gender did significantly influence the intercept for the linear model for positive self-schemas. At age 13, girls reported higher levels of positive self-schemas compared to boys. No gender difference in intercepts for negative self-schema.
Moilanen 1995	N=79 community sample depressive symptoms assessed continuously	14-18 M=15 SD=1	Mixed	Cross-sectional	SRET with words read out and self-referential and structural word judgments. Surprise recall immediately after. Calculated proportion scores for positive/negative words recalled that had been rated self-referential.	Evidence for a negative correlation between positive recall and depressive symptoms, and positive correlation between depressive symptoms and negative recall. Positive and negative recall did not contribute to the regression model for depressive symptoms after dysfunctional attitudes and negative attitudes and expectancies were included.
Neshat-Doost 1998	N=19 with depression N=19 healthy controls	10-17 M=15 SD=2	Mixed	Case-control	Positive and negative traits and neutral words presented, and participants told to repeat each word 3 times, think about whether it made sense for them, and remember each word for the end. Counted forward aloud in	Controls recalled similar number of positive and negative words. Depressed group recalled relatively more negative words than neutral words. Relationship between depressive symptoms and recall bias for negative, relative to positive, trait words varied with age, with relationship stronger in older participants. Due to bias away from positive

					twos for 1.5min, and then free recall followed by a recognition test.	words increasing with age. No age change in relationship between depressive symptoms and negative words. No associations between recognition and depressive symptoms.
Orchard 2018	N=84 clinic sample N=212 community sample	12-18 M=16 SD=1	Mixed	Cross-sectional	Self-description questionnaire with positive, negative and neutral words rated as self-referential (very similar to SRET). Surprise free recall immediately after questionnaire. Calculated number of correct positive/negative words recalled.	Positive self-evaluation and positive recall negatively associated with depressive symptoms. Negative self-evaluation positively associated with depressive symptoms, but no evidence for association with negative recall. In predictive model (with an interpretation task too), only negative self-evaluation from this task predicted depressive symptoms. Only negative self-evaluation was associated with depression diagnosis.
Pine 2004	N=19 with MDD N=133 no MDD	9-19 M=15 SD=3	Mixed	Case-control	Recognition memory test for faces. Participants viewed (angry, fearful, happy) photos. They rated how hostile each face was, how afraid they were, and how wide the nose was. Surprise recognition test 30mins later, with recognition of same faces showing a neutral emotion.	Better recognition of angry than happy or fearful faces. Older and female participants had better recognition overall. Evidence for an interaction between face valence and group: individuals with MDD had worse recall for fearful faces vs other participants (no differences for happy or angry faces). No association with anxiety diagnosis or with parental history of MDD/anxiety.
Prieto 1992	N=15 clinic depressed N=18 clinic non-depressed N=17 community	8-12 M=10 SD=1	Mixed	Case-control	SRET completed followed by distractor game for 5mins. Free recall task and then, after another 4-5min interval, did a recognition task. Measured number of positive/negative words recalled out of the total	Main effect of valence on endorsement, as all participants responded faster to positive than negative words. For recall, evidence of a group x valence interaction: both non-depressed groups recalled more positive vs negative words, but the depressed group recalled the same number of positive vs negative words. For recognition, also

					words endorsed (both positive and negative).	evidence of a group x valence interaction: all groups recognised more positive than negative words, but this difference was smaller in the depressed group than both non-depressed groups.
Reid 2006	N=133 community sample depressive symptoms assessed continuously	8-14	Mixed	Cross-sectional	SRET with words read out and self-referential and structural word judgments. Immediate surprise recall after. Computed proportion of positive/negative words that were recalled out of those originally rated as self-referential.	Overall memory bias for negative words was accounted for by the combination of scores on anxiety, fear, depression, and aggression. No variable independently accounted for scores on the memory measure. Canonical correlation analysis supported notion of a consistent processing bias across cognitive modalities, and similarity in processing biases across anxiety, depression, and aggression. No evidence of differences across age.
Roberson-Nay 2006	N=23 healthy controls N=11 with anxiety N=10 with MDD	M=13 SD=3	Mixed	Case-control	Recognition memory test for faces. Participants viewed (angry, fearful, happy) photos. They rated how hostile each face was, how afraid they were, how wide the nose was, and then passively viewed the face. Surprise recognition test 30-40mins later, with recognition of same faces showing a neutral emotion.	Compared to controls, participants with MDD had poorer facial recognition overall (lower d'). Participants with anxiety did not differ to either group. No evidence of an interaction between groups and facial emotion. No differences in percent correct or false alarms.
Smith 2018	N=99 community sample depressive symptoms	12-18 M=15 SD=2	Mixed	Cross-sectional	SRET, immediately followed by free recall. A proportional score was calculated to account for the overall number of words recalled.	For self-referential words, more depressive symptoms were correlated with a bias for recalling negative words. For non-self-referential words, higher levels of depression and anxiety were related to recalling more positive words. Non-self-referential

	assessed continuously					recall bias was a significant predictor in the regression model of depression (including predictors from other measures and tasks), with strong positive association. Self-referential negative recall bias was positively associated with wellbeing in an overall regression model.
Speed 2016	N=121 high and low risk community sample depressive symptoms assessed continuously	8-14 M=13 SD=2	Female	Cross-sectional	SRET followed by counting backwards out loud from 60 to 1, and then free recall task. Measured number of positive/negative words endorsed and recalled out of the total positive/negative words endorsed.	Overall participants recalled more positive than negative words. Weak evidence for a valence x maternal depression interaction: high-risk participants recalled more negative words (positive words same across groups). Evidence for an interaction between valence and depressive symptoms, as higher depressive symptoms were associated with higher recall bias for negative words and lower recall bias for positive words. Age was positively associated with positive and negative recall biases. Pubertal status positively associated with negative (not positive) recall bias.
Taylor 1999	N=40 high risk N=46 low risk	8-12 M=10 SD=1	Mixed	Case-control	Neutral or negative mood induction and then SRET. Immediate free recall. Calculated proportion of endorsed words that were recalled.	Evidence for a risk status x mood x valence interaction for endorsement: positive words were endorsed least by high-risk participants in the negative mood condition. Negative words were endorsed least by low-risk participants in neutral-mood condition. Overall more positive words were recalled. Evidence for a risk status x mood x valence interaction for recall: high-risk participants in negative mood recalled a higher proportion of negative words than high-risk participants in neutral

						mood. No other group differences in recall. Age did not alter these associations.
Timbremont 2004	N=15 never depressed N=19 currently depressed N=10 remitted depressed	8-16 M=13 SD=2	Mixed	Cross-sectional	Negative mood induction then SRET. Participants told to pay attention to the words because they would have to recall as many as possible. Did 1min control task then free recall. Calculated number of positive/negative words recalled as a proportion of total words endorsed.	More positive words endorsed than negative overall. Evidence for a group x valence interaction for endorsement: never depressed participants endorsed more positive words than currently depressed. Remitted depressed did not differ from either. Current and remitted depressed endorsed more negative words than never depressed. Never and remitted depressed endorsed more positive than negative words, but there was no difference for currently depressed group. Overall, more positive than negative words recalled. Evidence for a group x valence interaction for recall: never depressed recalled more positive and fewer negative words than current and remitted depressed. Never depressed recalled more positive than negative words, but current and remitted depressed showed no difference in recall.
Timbremont 2008	N=18 currently depressed/ dysthymic N=16 previously depressed N=39 never depressed	8-18 M=14 SD=2	Mixed	Case-control	Negative mood induction then SRET. Participants told to pay attention to the words because they would have to recall as many as possible. Did 1min control task then free recall task. Calculated number of positive/negative words recalled as a proportion of total words endorsed.	Currently depressed endorsed more negative words than other groups and rated positive words as less relevant than never depressed group. Previously and never depressed endorsed more positive than negative words, but currently depressed showed no difference, endorsing fewer positive and more negative words than other groups. No evidence of interaction between group and word valence on recall. Difference between positive and negative recall not associated with depressive symptoms.

Woolgar 2010	N=38 clinical sample depressive symptoms assessed continuously	12-17 M=15 SD=1	Mixed	Cross-sectional	Neutral or negative mood induction and then self-descriptive questionnaire. Free recall immediately afterwards and participants also asked whether they had endorsed the words. Recall scores calculated as the number of endorsed words which were then recalled.	In neutral mood, depressive symptoms were correlated with higher endorsement and recall of negative words. No impact of negative mood induction on word recall.
Zupan 1987	N=41 community sample classified based on depressive symptoms	8-16 M=12 SD=3	Mixed	Case-control	SRET with self-referential and structural word judgments. Immediate surprise recall after. Calculated proportion of yes-rated words recalled in each condition out of total yes-rated words endorsed in that condition.	Depressed group endorsed more negative and fewer positive words as self-referent than non-depressed group. Overall better recall of self-referential words. Depressed group recalled more negative and fewer positive words as self-referent than non-depressed group. Non-depressed group recalled more positive self-referential than structural words, but no difference for negative words. Depressed group recalled more negative self-referential than structural words, but no difference for positive words. No differences in recall of no-rated positive words, or no-rated negative words for non-depressed group. Depressed group recalled more no-rated negative words in self-referential vs structural condition. Only current depressive symptoms were associated with recall (not history of depression).

Note: RTs: reaction times. Longit: longitudinal. MDD: Major Depressive Disorder. "High risk" and "low risk" participants were categorised into these groups according to presence of maternal depression.

Appendix 5 References

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