Neighbourhood Disadvantage and Internalising Symptoms in Adolescents: The Mediating Role of Stressful Life Events, Temperament, and Maternal Aggression

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Submitted in total fulfillment of the requirements of the degree of Doctor of Philosophy

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DECLARATION

This is to certify that

i. the thesis comprises only my original work towards the PhD,
ii. due acknowledgement has been made in the text to all other material used,
iii. the thesis is less than 100,000 words in length, exclusive of tables, references and appendices, and
iv. the research reported in this thesis was conducted in accordance with the principles of the ethical treatment of human subjects as approved for this research by Human Research Ethics at the University of Melbourne.

.........................................................
Owen Spear               Date
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ABSTRACT

Purpose of the study: Disadvantaged neighbourhoods are associated with increased risk for anxiety and depression in adolescents. However the mechanisms for this relationship are not fully understood. Using a longitudinal design, I investigated whether several potential mediators, including stressful life events, maternal aggressive, dysphoric and positive behaviour, and adolescent temperament (Surgency, Negative affectivity, Effortful Control, Affiliation), could help explain the relationship between neighbourhood disadvantage and symptoms of anxiety and depression in early- to mid-adolescence.

Method: A community sample of 245 adolescents and their parents participated in a range of assessments at baseline (age approximately 12-13 years old), including an observational assessment of parent-adolescent interactions, and a battery of adolescent-rated questionnaires. Neighbourhood disadvantage was assessed by combining Postal Area data collected during this first wave of assessment with a measure of disadvantage called the Socio-Economic Indexes For Areas (SEIFA) developed by the Australian Bureau of Statistics. Adolescents were followed-up approximately 4 years later and completed questionnaires assessing depressive and anxious symptoms.

Results: Analyses revealed that adolescents from disadvantaged neighbourhoods were more likely to report a greater number of stressful life events, and depressive and anxious symptoms. They were also more likely to score higher on temperament measures of Negative Affectivity, and lower on measures of Surgency and Effortful control. Mothers from disadvantaged neighbourhoods were more likely to display aggressive and dysphoric behaviour for longer periods, and positive behaviour for shorter periods, however no differences were detected in regard to the frequency of these behaviours.

Mediation analyses using a bootstrapping approach determined that stressful life events and three temperament dimensions (low Surgency, low Effortful Control, high Negative Affectivity) significantly mediated the relationship between neighbourhood disadvantage and symptoms of anxiety and depression at baseline. Stressful life events and maternal aggression significantly mediated the relationship between neighbourhood disadvantage and change in depressive and anxious symptoms from baseline to follow-up.
Additional analyses were performed controlling for family socioeconomic status (SES). In terms of direct effects of neighbourhood disadvantage, analyses controlling for SES found that neighbourhood disadvantage remained a significant predictor in eight of the ten analyses. In terms of mediating relationships, four of fourteen relationships remained significant after controlling for SES.

Conclusion: The research reported in this thesis provides evidence that disadvantaged neighbourhoods differ from less disadvantaged neighbourhoods in several different ways. In addition, various factors were found to partially mediate the relationship between neighbourhood disadvantage and anxiety and depression at different periods during adolescence. Temperament appears to be important earlier in adolescence, maternal affective behaviour seems to be important during mid- to later-adolescence, while stressful life events appear to act throughout adolescence. These findings suggest that the neighbourhood environment is likely to influence adolescents both directly, and indirectly through its effects on more proximal and individual risk factors. It was concluded that prevention and intervention programs targeting a range of risk factors in adolescents from disadvantaged neighbourhoods could be particularly effective at reducing the prevalence of internalising disorders in adolescents.
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OVERVIEW

This section aims to provide the reader with an overview of this thesis, addressing the key points in each chapter and summarizing the main findings. The overall aim of this thesis was to investigate the influence of neighbourhood disadvantage on adolescents’ risk for depression and anxiety. This aim was divided into three specific aims. Firstly, this research aimed to determine whether neighbourhood disadvantage was associated with increased rates of depression and anxiety in adolescents. The second aim was to explore the ways in which the personal and psychosocial characteristics of adolescents who live in disadvantaged neighbourhoods differ from less disadvantaged neighbourhoods. Specifically, I explored whether adolescents’ temperaments, parent affective behaviour, and rates of stressful life events differ between disadvantaged and less disadvantaged neighbourhoods. The third aim was to identify which of these differences may mediate or link these disadvantaged neighbourhoods and increased risk for depression and anxiety in adolescents both cross-sectionally and longitudinally.

The first six chapters review the relevant literature. Chapter 1 provides the reader with an overview of adolescent depression and anxiety. Prevalence data reviewed in this chapter indicate that depression and anxiety are a significant problem for many adolescents, indicating that these bouts of depression and anxiety are associated with poorer outcomes later in life. It is also clear that there is a multitude of factors that play a role in the development of depression and anxiety in adolescents. Neighbourhood disadvantage appears to be an important, but relatively poorly understood factor that increases risk for internalising disorders in adolescents.

Chapter 2 reviewed literature surrounding the effect of neighbourhoods on risk for depression and anxiety. This chapter identifies that neighbourhood disadvantage is associated with increased risk for depression and anxiety in adults, children, and adolescents. Differences in neighbourhood effects between children and adolescents versus adults are reviewed, with evidence of a stronger effect of neighbourhood on children and adolescents, than adults. Theories for the effect of neighbourhood disadvantage on internalising disorders are also summarised. Researchers have proposed various theories to explain the relationship between neighbourhood disadvantage and mental health, including those focusing on neighbourhood disorder, stress, parenting, quality of the built environment, and other social factors. Researchers are beginning to empirically examine the mechanisms by which neighbourhoods influence risk.
for anxiety and depression, with formal mediation analyses being one way in which to gain further understanding of the way in which neighbourhoods influence mental health outcomes in adolescents.

Chapter 3 highlighted the importance of research using formal statistical mediation analyses to examine the mechanisms for the effects of neighbourhood disadvantage on adolescent mental health, and describes the specific mediational methods used in the current thesis.

Chapter 4 reviewed evidence for the hypothesised role of stress as a mediator between neighbourhood disadvantage and internalising disorders in adolescents. Specifically, this chapter summarised research that suggests that disadvantaged neighbourhoods are more stressful places in which to live, and this, in turn, is likely to lead to increased risk for depression and anxiety.

Chapter 5 explored research suggesting that parenting may also mediate the relationship between neighbourhood disadvantage and internalising disorders. It also outlines the specific aspect of parenting examined in the current thesis: parental affective behaviour (aggressive, dysphoric and positive), which is thought to have a significant impact on children’s emotional functioning.

Chapter 6 reviewed the possibility that adolescent temperament may act as a mediator between neighbourhood disadvantage and internalising symptoms in adolescents. While temperament is generally viewed as relatively stable throughout life, some evidence suggests that it is influenced by environmental factors such as stress, parenting and neighbourhood social influences. Some researchers have hypothesised that disadvantaged neighbourhoods may be one factor that could lead to changes in temperament. While there is no research examining whether temperament mediates the relationship between neighbourhood disadvantage and internalising symptoms there is evidence suggesting that (1) temperaments are modifiable, particularly earlier in life, and (2) that certain temperaments are associated with risk for depression and anxiety.

Chapter 7 brings together key aspects of the research reviewed in the previous chapters and outlines a theoretical model for the thesis. This model proposes that neighbourhoods influence risk for anxiety and depression both directly, and through indirect, or mediating pathways. The relationships examined in the current thesis are summarised in figure 1 below. A number of specific predictions regarding the influence of neighbourhood disadvantage on internalising symptoms are described. The first set of predictions relate to the way in which disadvantaged
neighbourhoods may differ from less disadvantaged neighbourhoods. I predicted that disadvantaged neighbourhoods have (1) adolescents with higher rates of depression and anxiety; (2) parents who, when interacting with their children, display more frequent and longer periods of negative affective behaviour, and shorter periods of positive behaviour (3) adolescents who display higher levels of maladaptive temperament dimensions (such as Negative Affectivity), and; (4) higher rates of stressful life events in children. I then hypothesised that stress, mothers’ affective behaviour, and adolescent temperament would mediate the relationship between neighbourhood disadvantage and internalising symptoms. If significant mediation was detected, then this could suggest that these factors represent some of the possible links between the neighbourhood environment and internalising disorders in adolescents.

Figure 1: Relationships examined in the current thesis

Chapter 8 outlined the methodology used in the thesis, and includes descriptions of the sample population, key study measures, assessment protocol, and relevant data collection points. The current study involved a community sample of 245 adolescents and their parents, who participated in a range of assessments at baseline, including an observational assessment of parent-adolescent interactions, an interview assessing stressful life events, and a battery of adolescent-rated questionnaires. Adolescents were followed-up approximately 4½ years later and completed a questionnaire assessing depressive and anxious symptomatology. Neighbourhood disadvantage was assessed by combining Postal Area data collected during the first wave of assessment with a measure of disadvantage called the Socio-Economic Indexes For Areas (SEIFA) developed by the Australian Bureau of Statistics (Pink, 2006).
Chapter 9 outlined the statistical method used to analyse data, including descriptions of the linear regression procedure to analyse main effects, and the mediation analytic tool used to analyse mediating pathways. The chapter also summarises the data imputation technique used for dealing with missing data, and provides detailed descriptive statistics on variables.

Chapter 10 outlined the results of direct relationships between neighbourhood disadvantage and various outcome variables. Analyses identified that neighbourhood disadvantage was associated with increased rates of depressive and anxious symptoms at baseline, but not associated with differences in the change in symptoms from baseline to follow-up. Adolescents from disadvantaged neighbourhoods were more likely to demonstrate lower Surgency, Effortful Control, and higher Negative Affectivity, and experienced a greater number of stressful life events. Parents from disadvantaged neighbourhoods were more likely to express longer periods of aggressive and dysphoric behaviour and shorter periods of positive behaviour whilst interacting with their children.

Chapter 11, 12 and 13 outlined the results pertaining to mediating relationships. Analyses determined that the temperament dimensions Surgency, Negative Affectivity and Effortful Control mediated the relationship between neighbourhood disadvantage and internalising symptoms at baseline, but not the change in symptoms from baseline to follow-up. Conversely, maternal aggression mediated the relationship between neighbourhood disadvantage and change in symptoms from baseline to follow-up, but not baseline symptomatology. Stress mediated the relationship between internalising symptoms at baseline, and the change in these symptoms through adolescence.

The final chapter of the thesis, Chapter 14, summarises and discusses each of the findings and subsequently outlines a model explaining the relationship between neighbourhood disadvantage and internalising disorders. This model proposed that neighbourhood disadvantage influences risk for internalising not only directly, but also indirectly through its influence on maternal aggression, risk for stressful life events, and adolescent temperament. Temperament appears to be important earlier in adolescence, maternal aggression seems to be important during mid- to later-adolescence, while stressful life events appear to act throughout adolescence. The chapter summarises limitations and strengths of the research, and considers implications of the findings for clinical interventions and future research.
CHAPTER 1
THE DEVELOPMENT OF DEPRESSION AND ANXIETY IN ADOLESCENCE

Depression is the second most disabling disorder in the world amongst 15 to 44 year-olds (World Health Organization, 2008), and anxiety disorders are thought to cause similar levels of disability (Ezpeleta, Keeler, Erkanli, Costello, & Angold, 2001). Adolescence is a period of heightened risk for anxiety and mood disorders; approximately 10-20% of adolescents will experience depression, and 12-20% will experience an anxiety disorder (Boyd, Kostanski, Gullone, Ollendick, & Shek, 2003). Given the high prevalence rates and associated adverse outcomes, there is a great need to prevent these disorders in children and adolescents. Accordingly, researchers have begun to investigate the mechanisms through which adolescents develop depression and anxiety. Understanding these mechanisms has the potential to increase our understanding of who, and what, to target in early intervention and prevention strategies for adolescents at high risk for depression and anxiety disorders. This task is of considerable importance, given that only a quarter to a third of adolescents and children with mental health problems actually receive treatment, with many of those that do, terminating the treatment prematurely, or failing to respond to the treatment offered (Essau, 2005). Prevention may be the only sustainable method to decrease population levels of depression and anxiety (Organization, 2004).

This chapter will first discuss the prevalence of anxiety and depression in Australia in section 1.1. The epidemiology of anxiety and depression will then be discussed separately. Section 1.2 will discuss the prevalence and impact of anxiety during adolescence. Section 1.3 will discuss the prevalence and impact of depression during adolescence. Section 1.4 will then discuss the various advantages and disadvantages of diagnostic versus symptom-level approaches when researching internalising disorders. Section 1.5 discusses comorbidity between depression and anxiety, while section 1.6 gives a brief outline of the various factors that are thought to increase risk for depression and anxiety in adolescents. The importance of longitudinal designs – one of the strengths of the current study – will be discussed in section 1.8, before a summary of the chapter is provided in section 1.9.
1.1 Anxiety and depression in Australia

The current thesis is novel in that it is one of few studies to examine neighbourhood effects on psychopathology in Australian adolescents. A recent national survey estimated that approximately 20% of Australians experience a mental illness in a 12-month period (Australian Bureau of Statistics, 2007), with Australian prevalence rates for adolescent anxiety and depression standing at 14% and 13%, respectively (Boyd et al., 2000). Mental health problems are now considered the largest population wide cause of disability in Australia (Begg et al., 2007), with expenditure on mental disorders expected to increase from $5.1 billion in 2003 to $12.1 billion in 2033 (Goss, 2008). Despite an increased focus on treatment of mental illness (Butler, Chapman, Forman, & Beck, 2006), the majority of those with mental illness do not receive appropriate treatment (Nehmy, 2010). A comprehensive survey of Australians aged 4-17 years revealed that only a third who were deemed to have a dysfunction mental health problem actually attended a mental health service in the previous six months (Sawyer, Arney, & Baghurst et al., 2008). As Nehmy (2010, p. 74) states “The prevalence, burden, and under-treatment of mental health problems indicate that it is of great importance to explore various means of preventing and treating depression and anxiety in adolescence”. Understanding the impact of Australian neighbourhood environments is one way to increase our understanding of this complex issue. Examining the impact of neighbourhood effects in the Australian context is also important given that the dynamics of neighbourhood disadvantage and its relationship to anxiety and depression might be very different in Australia, especially compared to the USA which has different racial factors not present in the Australian context, and which provides much less welfare than Australia and some European countries (Gough, Bradshaw, Ditch, Eardley, & Whiteford, 1997; Whiteford, 2005). As such, if effects can be demonstrated in an Australian sample, then this represents a very important cross validation of the proposition that neighbourhood effects per se can increase mental health problems.

1.2 Anxiety

1.2.1 What is anxiety?

Anxiety is defined concisely by Barlow (2002) as ‘a future-oriented emotion, characterized by perceptions of uncontrollability and unpredictability over potentially aversive events and a rapid shift in attention to the focus of potentially dangerous events or one’s own affective response to these events’ (p. 104). When individuals are faced with anxiety provoking situations, they
experience elevated physiological arousal, such as shallow breathing or a racing heart rate. This elevated physiological arousal is one of three response systems that are triggered in individuals when they are anxious (Lang, 1968). The other two include (1) a behavioural or motor response system, such as behavioural avoidance and (2) a cognitive response system, such as thoughts about impending danger. When anxiety interferes significantly in daily functioning and/or causes significant distress, it can be classed as a disorder. The disorder approach refers to anxiety as a group of symptoms that commonly cluster together.

The most widely used classification system for anxiety disorders is described by the Diagnostic and Statistical Manual IV – Text Revision (American Psychological Association, 2000). This manual outlines adolescent anxiety disorders, which include both adolescent and child specific disorders (e.g. Separation Anxiety Disorder) and those found throughout life (Generalized Anxiety Disorder, Social Phobia, Specific Phobia, Obsessive–Compulsive Disorder, Panic Disorder, and Post-traumatic Stress disorder). These classifications have changed from previous DSM classifications, which included Overanxious Disorder and Avoidant Disorder, and are likely to change again with the introduction of the DSM-5 (Regier, Narrow, First, & Marshall, 2002).

1.2.2 Prevalence and impact

Anxiety disorders comprise the most common disorders of adolescence (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). Between 2.5% and 5% of children and adolescents suffer from an anxiety disorder at any given time (Costello et al., 2003), with many more suffering from sub-threshold anxiety. Reported prevalence rates vary greatly; Boyd and colleagues (2000) reviewed the anxiety prevalence literature and determined that reported rates of anxiety symptoms in adolescents and children varied between 3.8% and 25%. Childhood anxiety is thought to cause as much disability as other psychological and physical childhood disorders (Ezpeleta et al., 2001), impacting significantly on family processes (Ezpeleta et al., 2001), and functioning with peers, school, and recreation (Essau, Conradt, & Petermann, 2000). Aside from the distress caused by anxiety disorders, these disorders interfere with child and adolescent development, and can lead to suicide, drug and alcohol abuse, depression, bipolar mood disorders, and loss of work days (Greenberg, Sisitsky, & Kessler et al., 1999; Pine, 1997; Lehman, Brown, & Barlow, 1998). Children and adolescents with anxiety disorders are also more likely to be unpopular, victimized and less socially competent (Giora, Gega, Landau, & Marks, 2005). Longitudinal studies indicate
that anxious children maintain higher levels of life interference into early adulthood (Caspi, Elder, & Bem, 1988; Last, Hansen, & Franco, 1997), and are more likely to suffer from further anxieties later in life (Pine, Cohen, Gurley, Brook, & Ma, 1998).

1.2.3 Adolescence and increased risk for anxiety disorders

Adolescence is a period of heightened risk for anxiety disorders, with several studies indicating higher rates of anxiety disorders in adolescence than pre-adolescence (Kessler, Avenevoli, & Ries Merikangas, 2001). For example, one study found the prevalence of anxiety disorders in a birth cohort increased from 7.5% at 11 years of age to 20.3% at 21 years of age (Newman, Moffitt, Caspi, & Magdol, 1996). Some anxiety disorders are less prevalent in adolescence relative to childhood, such as Separation Anxiety, while other disorders, such as Social Anxiety Disorder and Generalized Anxiety Disorder are more common in adolescence (Rapee, 1991 Westenberg, Gullone, Bokhorst, Heyne, & King, 2007).

There are thought to be several reasons for the increased rates of certain anxiety disorders in adolescence. Adolescence is a challenging period, characterised by transition and change. These changes occur in many different areas, including emerging puberty, increased neighbourhood and peer involvement, school transitions, and increased family conflict (Eccles et al., 1993). Adolescence is also a period of substantial biological, social-emotional, psycho-social, and cognitive development (Holmbeck, Friedman, Abad, & Jandasek, 2006), which can create challenges and stress for adolescents and consequently increase risk for the development and persistence of anxiety disorders (Clark, Smith, Neighbors, Skerlec, & Randall, 1994). For instance, rates of social anxiety increase during adolescence, with peak incidence coinciding with increases in fears of social judgement and evaluation (Ollendick & Hirshfeld-Becker, 2002). Moving towards greater independence from family also creates new challenges, which may encourage the avoidance behaviours commonly associated with anxiety disorders (Rao et al., 2007). Aside from the greater risk for anxiety associated with adolescence and puberty, increased rates during adolescence may also be due to the fact that many children who experience anxiety disorders are at moderate to high risk of continuing to experience these disorders during adolescence (Bittner et al., 2007).
1.2.4 Gender, puberty and anxiety

Females are almost twice as likely to suffer from anxiety as males (Costello et al., 2003; Essau et al., 2000). However, this is not found in all studies, with a minority of studies finding no significant sex differences in rates of anxiety disorders (Canino et al., 2004; Ford, Goodman, & Meltzer, 2003). These inconsistencies are likely to be due to differences between samples in age or proportion of specific disorders. There is some evidence that among girls, but not boys, a more advanced pubertal status (controlling for age) is associated with higher reported anxiety symptoms (Deardorff et al., 2007). It should be noted, however, that pubertal development was not be measured in the current study.

1.3 Depression

1.3.1 What is depression?

Depression is a disorder characterised by lack of motivation, sadness and withdrawal. Adolescent depression does not vary a great deal from adult depression (Lewinsohn, Rohde, & Seeley, 1998). According to the DSM-IV-TR (American Psychological Association, 2000), a diagnosis of depression requires five or more symptoms to be experienced over a period of at least two weeks. Symptoms of depression include low mood, lack of pleasure, poor sleep, fatigue, under- or over-eating, psychomotor agitation or retardation, feelings of worthlessness or inappropriate guilt, diminished ability to concentrate or make decisions, and suicidal thoughts and behaviours. Low mood and/or lack of pleasure are required for a diagnosis of depression.

1.3.2 Prevalence and impact

Between 20-28% of people will report experiencing at least one episode of depression by their early 20s (Costello et al., 2002), with the peak onset of depression occurring between 15-19 years (Hankin et al., 1998). Adolescent depression is associated with a range of concurrent impairments including low self-esteem, low perceived social competence, dysfunctional levels of emotional reliance and self-consciousness, poor coping skills, and poor physical health (Lewinsohn et al., 1998). In addition to these functional impairments, adolescent depression is also associated with a range of adverse outcomes during adulthood. For example many of those who experience depression during their teenage years report subsequent episodes later in life - it
has been estimated that 25% to 69% of adolescents who have experienced depression will experience another episode or more of depression by their early 20s (Hankin et al., 1998; Lewinsohn, Allen, Seeley, & Gotlib, 1999). Adolescents who have experienced depression are also more likely to have impaired educational and occupational performance, poorer health, early parenting, lower life satisfaction and increased rates of substance abuse (Fergusson & Woodward, 2002; Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2003).

1.3.3 Adolescence, puberty and increased risk for depression

Adolescence is a period of greatly increased risk for depression, where the 1-year prevalence has been estimated to be 4% to 7%, compared to 2% in childhood (Costello et al., 2002). Risk for depression increases dramatically in early adolescence, and then rises further through adolescence until late adolescence and early 20s (Kessler et al., 2001; Costello et al., 2002). In early adolescence, rates of depression are twice as high in females as in males (Hankin et al., 1998; Lewinsohn et al., 1998).

There are many different theories as to the increased rates of depression that occur during adolescence. Puberty, and its associated biological, emotional and psychological changes, is thought to be a time of greater risk for depression. One study suggests that it may be a more important developmental risk factor than age per se (Angold, Costello, & Worthman, 1998; Patton et al., 1996). In addition, the development of cognitive functions associated with affect regulation lags behind the development of emotion-related systems in the brain that occurs during adolescence (Kesek, Zelazo, & Lewis, 2008). This means that while adolescents are experiencing a range of strong emotions associated with the adolescent period, they are not as able to effectively regulate and understand these emotions, and thus they are more vulnerable to depression (Kesek et al., 2008). Again, it is worth noting that pubertal timing is not measured in the current study.

One other factor that appears to increase risk for depression in adolescence is the increased exposure to stress; both in the form of stressful life events (Compas, Davis, & Forsythe, 1985; Larson and Ham, 1993) and heightened levels of chronic stress resulting from physical, cognitive, and psychosocial changes associated with pubertal onset (Larson and Ham, 1993). Stress, in turn, contributes to negative affect in young adolescents (Brooks-Gunn & Warren, 1989) and increases the risk of developing psychological, behavioural, and somatic disorders.
Adolescence is also a time where children gain more independence from their parents, and begin to engage more with their environment and community (Massey, 1998). This means they are more likely to be exposed to the various neighbourhood risk factors that will be discussed in Chapter 2.

Obviously not every adolescent who experiences the changes associated with puberty and adolescence goes on to develop anxiety and depression. It is through an interplay of many different factors that certain individuals become vulnerable to the development of these disorders (Shortt & Spence, 2006). The current thesis aims to explore this interplay, by determining the way in which distal factors (such as neighbourhood and community) affect more proximal factors (such as parenting style) and individual factors (such as temperament) to increase risk for depression and anxiety. Details of these interactions will be explored in much more detail in the following four chapters.

1.4 Should anxiety and depression be viewed as a set of symptoms or in terms of diagnoses?

While anxiety has often been studied in terms of diagnostic categories, there has been considerable research that has highlighted the importance of examining anxiety dimensionally (Werry, 1992). The dimensional approach focuses on the level of symptomatology irrespective of whether a certain threshold has been met. In the case of depression this simply refers to the number of depressive symptoms. In the case of anxiety, a dimensional approach refers to the common factors that apply to all anxiety disorders, such as increased heart rate, muscle tension, agitation and insomnia. This approach has the advantage of reducing a large number of categories of anxiety to a smaller number of dimensions of psychological, emotional and behavioural dysfunction (Werry, 1992). Several well-known and widely used dimensional measures of anxiety and depression include the Depression, Anxiety, and Stress Scales (DASS) (Lovibond & Lovibond, 1995), the Beck Depression Inventory (BDI) (Beck, Ward, & Mendelson, 1961) and the Four Systems Anxiety Questionnaire (FSAQ) (Koksal & Power, 1990). Two particularly well validated measures of anxiety and depression are the Beck Anxiety inventory (BAI) (Beck, Steer, & Carbin, 1988) and the Center for Epidemiologic Studies Depression Scale (CESD-R) (Eaton, Smith, Ybarra, Muntaner, & Tien, 2004) respectively, both of which are used in the current thesis.
The benefits of a dimensional approach for anxiety and depression are that it provides a clearer view of the severity of anxiety and depression, rather than requiring a specific (and necessarily arbitrary) cut-off point to be met (Hudziak, Achenbach, Althoff, & Pine, 2007). This approach is also perhaps more applicable to the general population, as it allows for measurement and assessment of sub-threshold anxiety and depression, which is likely to be common in a community sample (Preisig, Merikangas, & Angst, 2003). A dimensional approach also aids in the screening and prevention of depression and anxiety; adolescents with elevated symptoms of depression (i.e., ‘sub-threshold depression’) have been found to resemble adolescents with diagnoses of depression on most measures of psychosocial dysfunction, including elevated risk for future depression (Secley & Lewinsohn, 2009) and face increased risk for suicidal ideation and attempts (Fergusson, Horwood, Ridder, & Beautrais, 2005). This suggests that it may be equally important for interventions and research to target subsyndromal adolescents as it is to target adolescents meeting full diagnostic criteria. Cross-sectional studies comparing sub-threshold and fully-diagnosed depression indicate a continuum of risk, whereby the same factors are associated with both symptoms and diagnoses (Cuijpers, de Graaf, & van Dorsselaer, 2004). Given that functional impairment and risk factors exist on a continuum, it would seem valid to study the symptoms of depression and anxiety on a continuum, rather than as a dichotomous phenomenon.

1.5 Comorbidity

It is worth noting that while anxiety and depression, both being forms of internalising disorders (Zahn-Waxler, Klines-Dougan, & Slattery, 2000), have clear differences in diagnostic features, and can occur in isolation, they are highly comorbid (Brady & Kendall, 1992). For instance, anxious children face 8 to 29 times the risk of suffering from depression (Costello et al., 2003; Ford et al., 2003). The highly comorbid rates of depression and anxiety are likely due to two main factors. The first reason is that anxiety and depression appear to have fairly similar risk and maintenance factors. Secondly they also share many symptoms, such as poor sleep, fatigue, and agitation.

Clearly there is significant overlap between anxiety and depression, with some researchers suggesting that the two be studied together as a single measure of internalising. However, there are enough differences in timing, outcomes, and aetiology, to justify studying them separately. For instance, anxiety is often found to precede the development of depression (e.g., Avenevoli, Stolar, Li, Dierker, & Ries Merikangas, 2001; Pine et al., 1998). Most existing neighbourhood
studies have focused on only one outcome measure (i.e., depression or anxiety). I considered that it is important to examine both anxiety and depression so that the specificity of the role of neighbourhood and associated mediating variables in the development of specific disorders can be evaluated.

1.7 Risk factors for anxiety and depression

Unsurprisingly given the range of adverse outcomes associated with depression early in life (e.g., Birmaher et al., 1996), understanding risk factors and how they relate to treatment has generated a great deal of research. From this literature, it is clear that a large number of factors influence risk for depression in childhood and adolescence. These factors include individual risk factors (genetics, temperament, cognition, behaviour), proximal risk factors (family, peers, school) and distal factors (e.g., neighbourhood, media, ethnicity) (Shortt & Spence, 2006). One model that attempts to describe these risk and protective factors and their interrelation is shown in figure 2, and is an adaptation of Bronfenbrenner’s Ecological Systems Theory (1979) as developed by Shortt and Spence (2006).

![Figure 2: An ecological–developmental model showing factors associated with depression in youth (From Shorrt & Spence, 2006). Arrows indicate interactions between various factors and levels.]
Shortt and Spence (2006) define a risk factor as a condition, characteristic, or event that (i) exists prior to an unhealthy outcome, (ii) increases the probability of that outcome, and (iii) significantly differentiates populations with a high versus low prevalence of that outcome. There are a range of factors that appear to increase risk for anxiety and depression. These include:

1. Temperament (Clark et al., 1994).
2. Parenting style (Ge et al., 1994).
3. Life events (particularly stressful life events) (Pine, Cohen, Johnson, & Brook, 2002).
7. Broader environmental factors, such as neighbourhood disadvantage (Mair, Roux, & Galea, 2008).

A detailed exploration of all the risk factors for anxiety and depression is outside the scope of the current thesis. However, three pertinent risk factors will be discussed later in the thesis. These include stressful life events (chapter 4), temperament (chapter 5), and parenting style (chapter 6).

Shortt and Spence (2006) argue that it is not enough to identify risk factors independently. What is needed is a deeper understanding of the interplay between various distal and proximal factors. Through building up our knowledge of this nature I am likely to gain further insight into the mechanisms through which adolescents develop anxiety and depression, which will allow us to more effectively prevent these disorders. The current thesis aims to examine the way in which neighbourhoods affect proximal factors (stress, parenting and temperament) to increase risk for depression and anxiety in adolescents. One important feature of the current study was the measurement of symptoms of depression and anxiety at two time points spaced four years apart. The following section will outline the importance of this design feature.

1.8 The importance of prospective longitudinal designs

The research designed in this thesis was prospective in relation to the onset of symptoms of depression and anxiety. Internalising symptoms were assessed at baseline, and participants were excluded if screening revealed a lifetime history of case level depression at this assessment.
Further assessment of symptoms of depression and anxiety were conducted approximately 4 years after the baseline assessment. This allowed for the examination of whether the neighbourhood environment and associated mediating factors (discussed in the following four chapters) prospectively predicted changes in the severity of symptoms of depression and anxiety in adolescents between the baseline and follow-up assessments.

Prospective longitudinal designs provide stronger evidence regarding causality than cross-sectional design, allowing for researchers to more confidently infer directionality of influence between constructs (Kim, 2008). However, perhaps due to the complications involved in conducting longitudinal research, there is a relative paucity of longitudinal studies examining the relationship between internalising disorders and neighbourhood factors thought to increase risk for these disorders. The design of this thesis allows for a much-needed, more rigorous examination of these relationships.

1.9 Summary and implications

Adolescent depression and anxiety represent a significant health problem, and are both associated with a range of significant impairments. These impairments are found in adolescents suffering from fully-diagnosed and sub-threshold anxiety and depression. Understanding the various risk factors for these mental health issues and their interplay is therefore of great importance.

This thesis aims to identify prospective risk in the onset of adolescent depression and anxiety, with a particular focus on the role of neighbourhood disadvantage. It also aims to explore the effect of the neighbourhood environment on mediating risk factors including stressful life events, poor parenting and maladaptive adolescent temperaments. It is hoped that this study will aid in the development of preventative programs aimed at reducing the prevalence of anxiety and depression in adolescents, especially the increased prevalence amongst more disadvantaged or vulnerable groups. The next chapter deals with neighbourhood disadvantage, an area of research that has recently begun to receive increased research attention, and has been recognised as an important risk factor for child and adolescent mental health.
CHAPTER 2
THE INFLUENCE OF NEIGHBOURHOOD DISADVANTAGE ON CHILD AND ADOLESCENT DEVELOPMENT

Children and adolescents are affected by the neighbourhood environments in which they grow up. Disadvantaged neighbourhoods (commonly defined as areas of concentrated poverty and associated characteristics such as high levels of unemployment or high crime rates; Kim, 2008) are associated with increased risk for a range of mental health issues. This chapter will review research investigating the role of neighbourhood disadvantage on child development, with a particular focus on the influence of neighbourhood disadvantage on the development of depression and anxiety in adolescence. The discussion will begin by outlining an argument for the importance of neighbourhood research (section 2.1). Section 2.2 will discuss definitions of ‘neighbourhood’ while section 2.3 reviews the development of research examining the effect of neighbourhood disadvantage on various outcomes in adults. The chapter then reviews neighbourhood effects in children and adolescents (section 2.4). Section 2.5 and 2.6 outline the way in which neighbourhoods are thought to differentially influence the mental health of different genders and different ethnic groups, respectively. Section 2.7 explores the various means used to measure neighbourhood quality. Section 2.8 outlines some of the difficulties with conducting neighbourhood research, while section 2.9 discusses the various theories regarding the effect of neighbourhood disadvantage on risk for internalising disorders. Section 2.10 outlines research into individual sensitivities to neighbourhood disadvantage and other variables that might potentially moderate these effects, while section 2.11 discusses current gaps in neighbourhood research.

2.1 Importance of neighbourhood research

Interest in neighbourhood research has increased significantly over the past thirty years and is driven by several important motivators. First, this research can be used by policy planners to help determine which neighbourhoods to target in prevention and early intervention programs. Further, neighbourhood research is able to give direction as to what aspects of these
neighbourhoods to target. That is, it can help to inform which specific factors are likely to reduce the incidence of adverse outcomes in children, adolescents and adults. Understanding the influence of neighbourhoods on risk for depression and anxiety is also of particular importance for several reasons outlined by Cutrona and colleagues (2006). They reason that people do not often realise the impact of neighbourhood environments and thus unjustifiably blame others (or may blame themselves), when neighbourhood factors may in fact be influencing them. They also argue that when threats to public health are influenced by characteristics of neighbourhoods, it is more efficient to address these threats at the neighbourhood level rather than to treat each affected individual separately. Therefore, it is important to understand and raise public awareness of the influence of disadvantaged neighbourhoods on risk for depression and anxiety.

2.2 What constitutes a neighbourhood?

Definitions of neighbourhood and the size of a neighbourhood vary widely across studies. Neighbourhood research, which has predominantly been carried out on North American populations, most often defines neighbourhoods as census tracts. Census bodies will work with residents to identify meaningful boundaries to divide up neighbourhoods. Census tracts are typically made up of between 4,000 to 6,000 people and include approximately nine or ten city blocks (Cutrona et al., 2006). Some researchers work with larger units, such as clusters of Small Area Market Statistics (in Sweden (Lofors & Sundquist, 2007)) or townships (in Taiwan (Yen, Rebok, & Yang, 2008)). Others use smaller units within census tracts called block groups made up of about 1000 people, and a few studies even use areas called face blocks, which include only single streets. All Australian studies of neighbourhood have used postcodes, with an average of approximately 11,000 people per postcode (in Melbourne).

Several studies tailor their definitions of neighbourhood to the participants in their study. For example, two studies have used circular “buffer zones” around each participant’s residence, using geographic information systems (GIS) methods to accurately map locations (Kruger, Reischl, & Gee, 2007; Berke, Gottlieb, Moudon, & Larson, 2007). Many studies have even allowed participants to provide their own definition of neighbourhood (e.g., Piko et al., 2005; Greiner, Li, Kawachi, Hunt, & Ahluwalia, 2004).

Despite the variety in definitions of neighbourhood areas, the impact of neighbourhood characteristics on mental health does not appear to depend on the neighbourhood unit that is used by any particular study (Sampson et al., 2002). According to a review article by Kim (2008),
all but three studies that combined multiple indicators as measures of the same neighbourhood-level construct reported reasonably high estimates of internal consistency reliability, kappa (j) statistics for assigning indicators to the same category, or results of factor analysis that indicated loading of indicators onto the same factor.

2.3 Neighbourhood disadvantage and negative outcomes and adults

In recent years, there has been a significant body of literature in the peer-reviewed medical and public health literature exploring the effect of neighbourhood environments on a variety of physical health outcomes in adults. For instance, Townsend and colleagues (1988) observed associations between neighbourhood disadvantage and patterns of mortality in small areas independently of the effects of occupational social class. Other studies have supported these findings, with neighbourhood disadvantage prospectively associated with cardio-vascular disease and stroke (Sundquist, Winkleby, Ahlén, & Johansson, 2004) and long-term illness (Curtis, Dooley, & Phipps, 2004). These differences in risk for various health problems are associated with the systematic distribution of health-related risk factors for morbidity and mortality by neighbourhood disadvantage. These factors include smoking (Duncan, Jones, & Moon, 1999), alcohol use (Pollack, Cubbin, Ahn, & Winkleby, 2005), physical activity (Yen & Kaplan, 1998) and diet (Forsyth, Macintyre, & Anderson, 1994). Other research has found links between neighbourhood disadvantage and problem behaviours such as delinquency, crime, and drug use (Leventhal & Brooks-Gunn, 2000).

Despite there being relatively fewer peer-reviewed papers on neighbourhood and mental health, it has long been thought that the two were linked. Even as early as 1939, Faris and Dunham (1939) determined that schizophrenia and substance abuse rates were highest amongst individuals from socially disorganised neighbourhoods in Chicago. In 1959 another researcher, Leighton (1939), explored how the expression of mental illness was influenced by local context and proposed that sociocultural disintegration of neighbourhoods was an important factor in the development of mental illness.

Early empirical studies into neighbourhood and mental health identified that low Socioeconomic Status (SES) neighbourhoods were associated with increased risk for mental illness. Measures of neighbourhood SES were single indicators based on census data, such as the percentage of households living in poverty, or multiple indicators such as single-parent households and mean household income, whose standardized values were averaged and combined. These studies
found that residents from lower-SES neighbourhoods were more likely to suffer from mental illness (e.g., Galea et al., 2007).

Most research into neighbourhoods and mental health has focused on depression over other forms of psychopathology. Of this research, the majority (over 80%) has found significant associations between at least one measure of neighbourhood disadvantage and prevalence of depression, after controlling for individual characteristics such as individual SES, race and gender (Mair et al., 2008). One powerful longitudinal study examined prospective associations between neighbourhood disadvantage and mental health (Stafford, Gimeno, & Marmot, 2008). The researchers found evidence of a dose-response relationship between the length of exposure to neighbourhood disadvantage and mental health problems, suggesting a partially causal role of neighbourhood deprivation. One survey using the same neighbourhood disadvantage data as the current thesis (Index of Relative Advantage and Disadvantage; Pink, 2006) determined that the prevalence of high mental distress is much greater in the most disadvantaged decile (22%) than the most advantaged decile (6%) (Pink, 2006). Figure 3 below shows the prevalence of ‘high’ or ‘very high’ mental distress deciles for respondents in this study.

![Graph showing prevalence of high or very high mental distress by relative advantage and disadvantage deciles](image)

Figure 3: Proportion of adults with ‘high’ or ‘very high’ mental distress, as a function of Relative Advantage and Disadvantage, adapted from Pink (2006)
Neighbourhood effects are generally found to be weaker than other factors such as familial factors or peer influences (Pickett & Pearl, 2001). There are thought to be several reasons for this. One possibility is that the influence of neighbourhoods is actually less important than other, more proximal effects. Neighbourhood effects are thought to be generally subtle, but pervasive: despite being a more distal influence, people are exposed to them on a daily basis.

Even if neighbourhood effects are modest, however, many researchers argue that interventions targeted at whole areas could improve the mental health for the whole population in those neighbourhoods (Sorensen, Emmons, Hunt, & Johnston, 1998). Further, as will be discussed below, neighbourhood effects are found to be much stronger in children and adolescents than in adults.

There are other explanations for the relatively modest neighbourhood effects found in research. One proposed reason is that distal effects are generally more difficult to measure, and thus they are likely to contain more data error or ‘noise’. In addition, most studies control for family SES. While this isolates the variance due to neighbourhood effects alone, doing so means that the variance shared by neighbourhood and individual SES (which is likely to be substantial) is also removed, potentially weakening the perceived effect of neighbourhood. For example, it is likely that someone in a poor neighbourhood is going to be poorer partly because their neighbourhood offers them fewer high-paying job opportunities and fails to provide them with the support needed to gain higher paid work. This neighbourhood effect is factored out when family SES is controlled.

In summary, it is clear that disadvantaged neighbourhoods are associated with a variety of negative outcomes in adults.

2.4 Neighbourhood research with children and adolescents

In recent years, neighbourhood research has begun to focus on the effect of neighbourhoods on child and adolescent development. Neighbourhoods affect many different aspects of adolescent and child well-being. For example, literature surrounding this issue has established that disadvantaged neighbourhoods are associated with increased risk for teenage pregnancy and juvenile delinquency (Brooks-Gunn & Duncan, 1997; Sampson, Morenoff, & Gannon-Rowley, 2002). In disadvantaged neighbourhoods there is also twice the risk of being born with low birth weight, being injured, and experiencing child abuse (Sellström & Bremberg, 2006; Brooks-Gunn
& Duncan, 1997). It has been estimated that approximately 5–10% of the variation in children’s and adolescents’ well-being and development is explained by neighbourhood factors (Leventhal & Brooks-Gunn, 2000).

The effect of neighbourhoods on adolescent mental health has been demonstrated in some powerful studies, which have observed neighbourhood effects longitudinally while controlling for individual factors. For instance, one longitudinal study demonstrated a prospective effect of neighbourhood disadvantage on depression and anxiety, observing a change over a two-year period in adolescent internalising disorders (Schneiders et al., 2003). These effects were found for both parent- and adolescent-reported mental health.

Perhaps the most compelling evidence for the relationship between neighbourhood disadvantage and internalising symptoms in adolescents comes from pseudo-experimental studies. One of the first of its kind was the Moving to Opportunity (MTO) program, which randomly moved low-income families residing in housing projects in high-poverty neighbourhoods of Baltimore, Boston, Chicago, Los Angeles, and New York City. A third of participants were given vouchers for middle-class neighbourhoods, a third were given vouchers to be used anywhere, and a third were given no vouchers at all (see Goering & Feins, 2003). Families who moved to more affluent areas experienced improved neighbourhood socioeconomic conditions, higher levels of social cohesion and less danger and violence in comparison to families who stayed living in high-poverty neighbourhoods (Katz, Kling, & Liebman, 2001; Leventhal and Brooks-Gunn, 2003). Parents and children also reported large reductions in anxiety and depressive symptoms, and short-term improvements in physical health (Katz et al., 2001; Leventhal and Brooks-Gunn, 2003).

There were, however, several limitations noted with research associated with the MTO program. Gennetian and colleagues (2012) note that one of the main weaknesses was that it was unable to answer how or why MTO produced the effects it did, for instance the differences between females and males. The current thesis has the opportunity to overcome this weakness by investigating some of the reasons for the relationship between neighbourhood disadvantage and internalising symptoms. Another limitation noted by Lundquist and Massey was that compliance with the terms of the program was very selective, despite the efforts of program managers to carry out random assignment of households to treatment groups. Further, many of those who were relocated did not remain in these areas for extended periods. Many researchers consider the
effects of neighbourhood disadvantage to be cumulative, influencing individuals over time. Thus, it was argued the program may have been able to measure the effect of the policy initiative, but not the effects of neighbourhood disadvantage per se. This would have meant, presumably, that the effect of neighbourhood disadvantage determined in the study may have been weaker than the genuine effect. In response to this, researchers reanalysed the data, measuring the cumulative amount of time spent in different neighbourhood environments. With this method, evidence was found for a stronger, cumulative effect of neighbourhood disadvantage in areas such as employment and earnings, indicating that the effect of neighbourhood disadvantage may have been underestimated in initial studies using data from the MTO. In terms of adolescent mental health, the most recent studies examining the long-term outcomes of the MTO program have found that the benefits of the MTO remained for girls, but not for boys (or parents) (Osypuk, Tchetgen Tchetgen, Acevedo-Garcia, et al., 2012; Gennetian, Sciandra, Sanbonmatsu et al., 2012). This research also found more muted effects of the MTO program in domains including mental and physical health.

The Yonkers program was another pseudo-experimental study that allowed for the investigation of the effect of neighbourhood disadvantage on well-being. It was created in response to a federal court order to remedy the issue of racial segregation in public housing and schools (U.S. vs. City of Yonkers, 1985). In this program 184 low-income African American and Latino families were moved from poor neighbourhoods to newly built units in more affluent white, middle-income neighbourhoods between 1992 and 1994. The project aimed to examine the impact of neighbourhood change on several key outcomes including job attainment, education, job stability, parenting, health status, family functioning, and mental health. The families who were moved were assessed at several time points after the move, and their wellbeing was compared with 149 families who stayed in their low-income neighbourhoods. Families who changed neighbourhoods experienced a range of improvements in well-being. Seven years after they had moved, parents reported experiencing less distress, obtained higher SES ratings, and were more likely to work and less likely to receive welfare than non-movers (Fauth, Leventhal, & Brooks-Gunn, 2007). They also reported better physical health than adults living in poor neighbourhoods, but mental health did not vary by neighbourhood.

The Yonkers program also focused on the impact of neighbourhood change on children and adolescents, which was measured using several key outcomes, including school achievement, school engagement, peer networks, juvenile delinquency, employment and mental and physical
Research indicated that adolescents from the study experienced a range of benefits from moving to less poor neighbourhoods. For instance, 8–18-year-olds who moved to more affluent neighbourhoods experienced less disorder, victimization, and access to illegal substances than those who remained in poorer neighbourhoods (Fauth, Leventhal, & Brooks-Gunn, 2005). Greater benefit was found for children when compared to older adolescents. For instance, it was found that boys and girls under 10 years (but not 16-18 year olds) experienced fewer relationship and behavioural problems, while more problems were found for teenagers aged 16-18 who moved suburb (Fauth et al., 2005). Thus it was concluded that neighbourhood effects may be strongest in children and young adolescents, however it is possible that there are complicating factors for older adolescents, who found it more difficult to integrate into their more affluent neighbourhoods.

The effect of neighbourhood disadvantage on adolescent and child mental health appears to be even stronger than on adult mental health, and is found more consistently than in adults. For instance, according to a review paper by Mair and colleagues, 9 out of 10 studies examining the effect of neighbourhood on child and adolescent depression have found significant associations (2008). While the amount of variance shared between neighbourhood characteristics and depressive symptoms in adults ranges between 0.4–2.9%, it is estimated to be around 11% for children (Kim, 2008). Some factors that do not appear to affect adults do affect the well-being of children. For instance, neighbourhood poverty without social disorder has not been found to consistently affect risk for depression in adults (Cutrona, Russell, Hessling, Brown, & Murry, 2000), but does show consistent effects in children (Xue, Leventhal, Brooks-Gunn, & Earls, 2005).

In fact, it may be that adolescents are the most vulnerable of all age groups to the effects of disadvantaged neighbourhoods. Children appear to be more influenced by their family environment, while adolescents are beginning to interact more with the wider social context around them (Massey, 1998). As discussed in the previous chapter, they are also at a particularly vulnerable stage of life. As they are shifting towards a more independent way of being, they often look to non-familial sources to gain support and a new sense of identity. The neighbourhood is one of the potential sources of influence. On the one hand, positive supportive neighbourhoods are likely to provide support and encourage positive development during this formative period. On the other, disadvantaged neighbourhoods are associated with daily experiences of
unproductive and dangerous interactions that create an atmosphere that is highly stressful and likely to lead to negative outcomes (Aneshensel & Sucoff, 1996).

2.5 Gender and neighbourhood disadvantage

Several studies have examined whether males and females are differentially affected by their neighbourhood environments. In their review of neighbourhood and depression studies Mair and colleagues (2008) report that of the nine studies that examine gender effects, only three found differences in terms of gender. Two found stronger associations in females (Gutman & Sameroff, 2004; Piko, Fitzpatrick, & Wright, 2005) while one found a stronger association in males (Berke et al., 2007).

Research has also looked at gender differences in children and adolescents. There is some research to suggest that neighbourhood effects may be stronger in boys than girls (Greenberg et al., 1999). For instance, Leventhal and Brooks-Gunn (2011) found that internalising symptoms were worse in boys as a function of neighbourhood factors, but not girls. This gender difference is thought to be because boys interact more directly with their community environment, thus increasing exposure to neighbourhood effects (Simons, Johnson, Beaman, Conger, & Whitbeck, 1996). It is also thought that boys might be more responsive to the negative impacts of disadvantaged neighbourhoods than girls, as they report a greater number of friends in the neighbourhood, tend to play more out of the home, and report a greater affiliation with their neighbourhood (Kroneman, Loeber, & Hipwell, 2004).

2.6 Ethnicity and neighbourhood disadvantage

There have only been a few studies comparing the effect of neighbourhood in different ethnic groups (Kim, 2008). Of these studies, there do appear to be some differences, but these differences are not consistent. For instance, community cohesion was found to be associated with decreased rates of depression in Caucasian-Americans, but was not associated with depression in African-Americans (Gary, Stark, & LaVeist, 2007). Another study found that Mexican-Americans suffered from lower rates of mental illness in areas where there was a high concentration of Mexican-Americans (Ostir, Eschbach, Markides, & Goodwin, 2003) whereas another study found that African-Americans suffered from more mental illness when they resided in neighbourhoods with a higher concentration of African-Americans (Henderson et al., 2005). Whilst there appears to be differences in neighbourhood effects in different ethnic groups, this is
not as relevant to the Australian context, where the ethnic composition is different to that of North American cities, and thus will not be a focus of the current thesis.

2.7 Measures of neighbourhood quality

Measures of neighbourhood disadvantage tend to fall into one of two categories: **objective** versus **subjective** measures of neighbourhood quality.

Subjective measures assess neighbourhood conditions from the same sample of participants who provide measures of psychopathology. This allows researchers to avoid collecting data from different sources, and provides a more idiosyncratic view of ‘neighbourhood’ and neighbourhood disadvantage. This taps into the way that the neighbourhood environment is **experienced** by community members. Researchers using this approach point to the fact that residents of the same neighbourhoods interpret and react to neighbourhood conditions quite differently (Cook, Shagle, & Degirmencioglu, 1997). However, this approach introduces the complicating factor of reporting bias, or same-source bias. This may occur when people who are depressed or anxious report higher levels of disadvantage or disorder in their neighbourhood because of their low mood or anxiety, not because their neighbourhoods are actually more disadvantaged. For instance, when an individual is depressed they tend to perceive the outlook for the future and of the world in general more negatively than it might appear to outside observers (Beck & Alford, 2008). Thus an adolescent’s perceptions of their neighbourhoods might be impacted by their mental health rather than the other way round (Fagg, Curtis, Clark, Congdon, & Stansfeld, 2008). The literature using subjective measures of neighbourhood is thus prone to bidirectional associations, and confounding. This approach also presents the problem in that it is difficult for those using this research to develop prevention programs to know what to target in a community when different members of the community react in different ways to neighbourhood characteristics. Researchers using a subjective approach often do not actually know why certain individuals rate their neighbourhood as worse.

Objective measures of neighbourhood tend to be either measures of **neighbourhood SES** or **composite measures** of neighbourhood disadvantage, which encompass many different variables thought to be associated with neighbourhood disadvantage. Neighbourhood SES is easily and objectively measured by averaging the average income of neighbourhood residents. However, it only focuses on one aspect of the neighbourhood, and it is possible that a neighbourhood could be relatively poor, but not be especially disadvantaged if it has other positive characteristics such
as low crime rates, well connected transport and strong community ties. These factors are generally considered as important, if not more important, than neighbourhood SES (Krieger et al., 2003). This effect was demonstrated by one study that provided families with a variety of services similar to services that would be found in higher income communities. Obviously, neighbourhood SES itself was not altered, however it was found that these services led to educational benefits for children and adolescents, despite a lack of change in neighbourhood SES (Dobbie, Fryer, & Fryer Jr, 2011) approach to measurement of neighbourhood disadvantage was used in the Project on Human Development in Chicago Neighbourhood (PHDCN). This project asked 8,782 residents a series of demographic, social and organisational questions about their neighbourhood to gain a subjective, but independent measure of neighbourhood quality (Molnar, Buka et al., 2003). This approach has the benefit of avoiding same-source bias, but is much more labour intensive.

Composite measures of neighbourhood disadvantage provide a more comprehensive assessment of neighbourhood disadvantage, and involve the compilation of various characteristics of disadvantage (e.g., low-weight babies, high unemployment, high crime rates, high number of single parents) into a single measure of neighbourhood disadvantage. While this approach does not allow for investigation of specific neighbourhood factors, it has the advantage of being comprehensive and objective, and lacks the reporting bias that may be present in subjective measures of neighbourhood disadvantage.

The measure used in the current study is termed the *Socioeconomic Indexes for Areas* (SEIFA) compiled by the Australian Bureau of statistics (Pink, 2006). This measure examined levels of socio-economic advantage and disadvantage, which was defined loosely as people’s access to material and social resources, and their ability to participate in society. The SEIFA variables were categorised into the following dimensions: (1) income variables (2) education variables (3) employment variables (4) occupation variables (5) housing variables (6) other variables of disadvantage (see section 8.3 for a detailed list of included variables). This index can be used to measure socio-economic wellbeing in a continuum, from the most disadvantaged areas to the most advantaged areas. Given neighbourhood disadvantage and advantage are very highly negatively correlated, this measure will henceforth be referred to as neighbourhood disadvantage.
2.8 Complications with neighbourhood research: reverse causation and residual confounding

One of the major complications for neighbourhood research is the issue of selection effects or reverse causation. Families have some choice over the neighbourhood in which they reside (termed selective migration). Depressed families may be more likely to stay in or move to disadvantaged neighbourhoods, or anxious families may be more likely to move away from disordered or unsafe neighbourhoods. If this were the case, then disadvantaged neighbourhoods would not necessarily be causal of depression and anxiety, but merely correlated with these disorders. Cross-sectional studies are particularly vulnerable to reverse causation, and thus Mair et al. (2008) state, in their review of neighbourhood and depression research, that more longitudinal studies are needed to rule out reverse causation as an explanation for cross-sectional associations.

It is likely that both selective migration and neighbourhood influence each explain some proportion of the link between neighbourhood disadvantage and poor mental health. One study found evidence of selective migration: those in poor psychological health were more likely to move than other groups and were especially likely to move towards disadvantaged neighbourhoods (Curtis, Riva, & Rosenberg, 2009). This seems to provide some support for the ‘health selection’ process whereby people with mental health problems tend to ‘drift’ towards poor, socially disadvantaged areas. However, this effect seemed to operate indirectly through individual socio-economic characteristics that predicted both mental health outcomes and differential migration patterns (Curtis et al., 2009). Curtis and colleagues (2009) note that as long as individual SES is factored into any analyses, then this effect should be accounted for. Another study examined the effect of selective migration by following 25-74 year olds for 10 years. Physical health and health-related behaviour were only weakly associated with migration, and the authors concluded that selective migration would not contribute a great deal to neighbourhood inequalities in health and health-related behaviour (van Lenthe, Martikainen, & Mackenbach, 2007). This study did not, however, examine the mental health of participants.

While selective migration effects may play some role in explaining the association between neighbourhood disadvantage and mental health, it is less likely that families would shift neighbourhood due to child or adolescent mental health. Even if families did move because of their children’s’ mental health issues, this could sometimes involve a move to safer and more affluent neighbourhoods (thus weakening the perceived effect of disadvantaged neighbourhoods). For
instance, parents could be concerned for the effect of an unsafe neighbourhood environment on the well-being of their children (Clampet-Lundquist, Edin, Kling, & Duncan, 2006), or a desire to access better educational or social facilities for adolescents as they reach school age (Rabe & Taylor, 2010). In conclusion, it appears that while selective migration may explain some of the relationship between neighbourhood disadvantage and mental health issues, this is less likely in adolescent populations and can be partially taken into account by controlling for family SES.

Another complication of neighbourhood research is that of residual confounding by individual level variables. That is, there are likely to be certain unmeasured characteristics that influence families’ choice of neighbourhood and their risk for developing mental health issues. If these influential variables are omitted from analysis, the effect of neighbourhoods on risk for internalising disorders could be over- or under-estimated. Researchers generally try to overcome this issue by controlling for factors such as family SES, gender, ethnicity and a variety of other variables. This attempts to rule out within-neighbourhood homogeneity (e.g., SES) as an explanation for findings. However, there is no consensus on what the key confounders are likely to be. There is also a lack of understanding regarding the sensitivity of results to residual confounding.

Individual SES is perhaps the most important factor to control for, as it is very highly correlated with neighbourhood SES and appears to exert a separate but significant effect on physical and mental health in residents (e.g., Fryers, Melzer, & Jenkins, 2003; Kohn, Dohrenwend, & Mirotsnik, 1998). One longitudinal study tested 1075 children aged 9-17 (at time 1) over 9 years. Those from the lowest family SES suffered from a greater number of depressive/anxious symptoms and delinquent/aggressive syndromes than those from the highest SES. Further, it was found that those with psychopathology and low SES did not improve as much as those with psychopathology from middle- and high-SES backgrounds. One notable experimental study revealed the possible importance of family income in improving children’s outcomes (Morris, Gennetian, Duncan &. 2009). This study controlled the employment status and income of single mothers and observed changes in children’s school achievement. Some mothers gained employment with no change in income (above their welfare payments), some gained employment with increased income, and some remained unemployed. It was found that children’s school achievement increased only when mothers were given employment and were also given increases in income (Morris et al., 2009).
Controlling for individual-level confounding variables such as individual SES usually reduces the size of the association between neighbourhood disadvantage and internalising symptoms (although the association rarely disappears completely; Mair et al., 2008). Controlling for individual-level factors could also lead to over-controlling. For instance, factors such as family SES are in some ways inextricably linked to neighbourhood disadvantage. In particular, disadvantaged neighbourhoods may provide less access to services, such as financial services, and lower-paying jobs, which drives down family SES. Thus neighbourhood effects may be underestimated when too many individual-level factors are controlled for.

In conclusion, it is not known which factors should be controlled for. However the current thesis follows a rule of reporting analyses with and without analyses controlling for these factors, to provide a more comprehensive view of the influence of neighbourhood on mental health.

Another potential way to overcome the issue of confounding is to conduct quasi-experimental studies, as discussed previously in section 2.4. These studies, based on programs such as the Yonkers or MTO program, provide some of the most compelling evidence for the relationship between neighbourhood disadvantage and mental health, given their ‘manipulation’ of the neighbourhood environment.

The literature on neighbourhood and mental health suggests that disadvantaged neighbourhoods increase risk for internalising disorders, particularly in children and adolescents. However, this tells us very little about what it is about these neighbourhoods that increases risk. Academics have attempted to answer this question through several means. One is to propose theories as to the mechanisms through which neighbourhoods increase risk. This is often done through examining whether certain specific characteristics of neighbourhoods are associated with increased risk. Another approach is to statistically test whether there are mediating factors between neighbourhood and internalising disorders. A mediational approach is taken in this thesis, however literature that utilises the first approach will first be discussed before outlining research surrounding mediation in the next three chapters.

2.9 Theories for the effect of neighbourhood disadvantage on risk for internalising disorders

Despite neighbourhood disadvantage being an established risk factor for internalising disorders, there is no single prevalent theory regarding the mechanisms of its influence on depression and
anxiety. Theories tend to fall into three broad categories including (1) community support and cohesion, (2) neighbourhood disorder, (3) and environmental qualities such as the quality of buildings or number of green spaces.

A great deal of non-experimental literature argues that community support and cohesion accounts for neighbourhood effects on health (Fauth et al., 2007). Many different community factors appear to act as risk or protective factors, with the empirical literature demonstrating that some social factors are more important than others. There is inconclusive, but promising evidence for the relationship between social capital and depression (Fauth et al., 2007). Social capital refers to the social connections (such as those gained through sporting clubs) that facilitate further connections, co-operation, and benefits for all members (such as increased job opportunities). A neighbourhood with high social capital appears to buffer residents against the more harmful aspects of their neighbourhood. Other important community factors include turnover of residents (Mancini, Bowen, & Martin, 2005), whereas factors such as a person’s attachment to their neighbourhood appear to be less important (De Silva, McKenzie, Harpham, & Huttly, 2005). A non-cohesive and unsupportive social environment is thought to contribute to poor regulation of child behaviour, less social support, poorer role models and greater isolation (Stafford, De Silva, Stansfeld, & Marmot, 2008).

Socially disordered neighbourhoods – those that are uncivil, threatening or unsafe – are perhaps the most distressing in which to live. Measures of social disorder include public drinking, harassment, drug dealing and gangs, all of which are thought to indicate a breakdown of informal social control (Skogan, 1992). Research suggests that social disorder is the neighbourhood characteristic that is most directly linked to increased rates of depression (Ross, 2000), with 25 of the 37 factors (68%) relating to social disorder examined in studies being significantly associated with depressive symptoms (Kim, 2008). For instance, one study found that perceptions of ambient hazards, including graffiti, crime, and drug use, were prospectively related to adolescent depression (Aneshensel & Sucoff, 1996). The effect of social disorder on risk for depression and anxiety is thought to be due, at least in part, to the increased stress and anxiety from fear of victimisation, powerlessness and a lack of safety (Hill, Ross, & Angel, 2005). Neighbourhood disorder and danger may also prevent a healthy lifestyle with regular exercise (which is associated with increased risk for depression (Mead et al., 2009)), as residents may be less likely to leave their homes and use nearby green spaces and facilities for exercise (Grzywacz & Marks, 2001).
One non-social dimension that to a lesser, but still significant extent, appears to increase risk for internalising disorders is poor quality physical conditions, such as degraded buildings or few ‘green spaces’. For instance, one study found that even after accounting for median neighbourhood income, people living in neighbourhoods characterised by poorer features of the built environment were 29% to 58% more likely to have experienced depression in the previous 6 months, compared to those living in neighbourhoods with better features of the built environment (Galea, Ahern, Rudenstine, Wallace, & Vlahov, 2005). Another study found that observers’ ratings of housing quality predicted depression beyond the effects of family SES in low- and middle-income rural women (Evans, Wells, Chan, & Saltzman, 2000). Furthermore, women who moved from poor-quality apartments to single-family homes were found to experience significant decreases in depressive symptoms (Evans et al., 2000). Research also suggests that being surrounded by greenery (Takano, Nakamura, & Watanabe, 2002), and well-maintained and aesthetically pleasing buildings (Weich et al., 2002), can decrease risk for depression and anxiety, perhaps by reducing levels of stress (Galea et al., 2005). Interestingly, access to services is not often found to be associated with risk for depression or anxiety (Kubzansky et al., 2005).

2.10 Individual sensitivity to neighbourhood disadvantage and other moderating relationships

While research using ‘direct effect models’ continues to examine the relative importance of various neighbourhood characteristics, recent research indicates that disadvantaged neighbourhoods do not affect all people in the same way. That is, people with different circumstances and personal traits adjust in different ways to neighbourhood disadvantage (Cutrona et al., 2006). In accord with this, researchers have begun to find that neighbourhood factors may be moderated by various proximal factors (Roosa, Jones, Tein, & Cree, 2003). That is, these proximal factors (e.g., temperament) appear to alter residents’ sensitivity to the level of disadvantage in their neighbourhood. It has been hypothesised that some people with resilient personalities or temperaments can cope successfully, even in dangerous and disorderly neighbourhoods, while other people – for instance those high on neuroticism – appear highly vulnerable to depression and anxiety when they live in adverse surroundings (Cutrona et al., 2000). Demonstrating this was one study that found that amongst women who lived in high-social disorder neighbourhoods, levels of distress were extremely high if the women were high on the personality trait of Negative Affectivity (Cutrona et al., 2000). In contrast, women who
scored highly on Optimism and Personal Mastery were relatively immune to the negative mental health impact of neighbourhood social disorder.

In other cases, neighbourhood disadvantage may moderate the effect of certain proximal factors (e.g., trauma or parenting style) on risk for depression and anxiety. For example, the positive influence of supportive parenting is more pronounced in children from disordered neighbourhoods (Dearing, 2004). Other research indicates that disadvantaged neighbourhoods may intensify the effect of traumatic life events on depression and anxiety (Cutrona et al., 2006). That is, in a disadvantaged neighbourhood the same negative event (e.g., being physically assaulted) is more likely to lead to an internalising disorder, than it will in a relatively advantaged neighbourhood (Stockdale et al., 2007). Whilst research into moderating relationships is of great important, these relationships are not a focus of the current thesis.

2.11 Gaps in the research: How does the current study hope to further our understanding of the effect of neighbourhoods on adolescents’ risk for depression and anxiety?

While some studies have examined the mechanisms through which neighbourhood disadvantage influences risk for internalising disorders, more are needed (Mair et al., 2008). The current study aims to provide further understanding of the effect of these factors on risk for anxiety and depression. In addition, while many studies have examined neighbourhood effects on depression, few have looked at neighbourhood effects on anxiety disorders. This is problematic, given that despite being associated with similar levels of dysfunction, we do not know whether neighbours exert the same influence on risk for anxiety disorders as they do on depression (Ezpeleta et al., 2001). Thus, this thesis represents an opportunity to shed light on the issue of the effect of neighbourhood disadvantage on risk for symptoms of anxiety.

There is also a dearth of studies that examine neighbourhood effects on risk for depression and anxiety in children and adolescents, with only ten or so relevant empirical studies of this nature conducted so far (Mair et al., 2008). This is despite the fact that, as previously discussed, neighbourhoods appear to have a much greater effect on children and adolescents than adults. In fact, one might hypothesise that of any age group, adolescents are most likely to be influenced by their neighbourhood. Adolescents are perhaps more susceptible to environmental influence than adults, and are beginning to gain greater autonomy from their family, while interacting more with their neighbourhood environment (West, 1997). The neighbourhood environment is increasing in importance as a place for socialisation and social observation. Matthews and colleagues (2000),
amongst others, showed that ‘the street’, for young adolescents, was important as a place for social interaction, where adolescents perform to their peers, and ‘test’ behaviours on passers-by. Older adolescents engage even further with their neighbourhood when they are allowed access to vehicles and increasing independent engagement in their environment, including accessing alcohol and drugs, and engaging in sexual and romantic relationships (Van Der Burgt, 2008). Thus we might expect to see much larger effects of neighbourhood disadvantage on adolescents, in comparison to children and adults.

Another lacuna in the literature to date is that there are few studies of this type conducted outside the USA. A recent systematic review of neighbourhood effects and depression found that 34 out of 45 studies were conducted in the USA (Mair et al., 2008). This is a significant problem, as findings cannot always be accurately generalised to all countries. This can be seen in studies that examine the importance of neighbourhood SES on risk for depression; those from countries that provide higher levels of welfare, such as some European countries, less reliably find significant results than studies conducted in the United States (Mair et al., 2008). This may occur for several reasons. It is possible that higher levels of welfare buffer residents against factors such as social disorder and low neighbourhood social capital (Kim, 2008). Demonstrating this is the fact that, in Kim’s review of neighbourhood literature, for social capital, all three of the null studies were carried out in countries that are relatively more egalitarian than the United States. Another possibility is that the difference between the poorest and richest neighbourhoods is greater in countries such as the United States than in countries such as Sweden, which is likely to lead to greater differences in rates of depression and anxiety between disadvantaged and less disadvantaged neighbourhoods in these countries. Oreopolous argues that the level of distress within high-poverty neighbourhoods in certain countries is not the same as the levels of distress in other countries. For instance, Canadian low-income neighbourhoods experience much lower levels of crime and segregation of minorities than in low income-neighbourhoods in the US (Oreopoulos, 2008). Wacquant examined differences between ‘ghettos’ in France and the United States and also found very different patterns of disadvantage in each (2008). For instance, many of the ghettos in the United States appear to be more homogenous in the make up of race and social class. In contrast, in France ghettos appear to be becoming less and less racially homogenous, with a wide variety of ethnicities present in these ghettos. Thus the current study will provide a much-needed examination of neighbourhood effects in Australia – particularly neighbourhood effects on children and adolescents. This will help to determine whether disadvantaged neighbourhoods in Australia influence residents in the same ways as the United
States. Indeed, Edwards (2005) states that further data about how neighbourhoods affect Australian children is urgently needed.

Only two Australian studies have examined neighbourhood effects on risk for depression and anxiety in children, and despite higher investment in welfare and infrastructure in neighbourhoods than in the USA (Gough et al., 1997), both these studies found associations between neighbourhood disadvantage and internalising disorders. The first – Homel and Burns’s (1989) study – found that children with lower levels of social adjustment and higher levels of unhappiness, worry, fear and anger lived in disadvantaged neighbourhoods. The second, more recent study found that, even when control variables were introduced, neighbourhood disadvantage was associated with decreased social and emotional functioning in children (measured through five scales including Prosocial, Peer Problems, Emotional Symptoms, Conduct Problems and Hyperactivity; Edwards, 2005).

The current study is innovative in that it uses relatively independent measures. That is, the various measures do not rely on reporting from the individual, as discussed in section 2.7. This lessens the problem of confounding. It is also relatively unique in that the population is representative of families of many different levels of SES. Many of the studies conducted so far (e.g., those resulting from the Yonkers study) observed neighbourhood effects in predominantly low-income families, which could bias results, and may prevent these studies from being as generalizable to more affluent families.

Of great advantage to the current study is its ability to study neighbourhood effects longitudinally. The majority of the studies conducted so far have been cross-sectional. According to Mair’s 2008 review article, only 10 of the 45 neighbourhood papers used any kind of follow-up or prospective analysis. Wheaton and Clarke (2003) argue that current individual status is reflective of cumulative influences of past life experiences. Thus the real impact of community context are likely to be found in lagged, longitudinal relations between early neighbourhood environment and individual-level outcomes in later stages of life. In fact, some studies, such as Whaten and Clarke (2003), found only long-term cross-level effects in children (ages 6-11) when assessing the mental health effects of current and childhood neighbourhoods. Longitudinal studies have the benefit of being able to rule out reverse causation. They are also needed to investigate time lags and cumulative effects of neighbourhoods on depression.
The current study is also important in its examination of mediating relationships in regard to early adolescence. Compared to direct effects (which study the direct influence of an independent variable on a dependent variable), indirect effects observe the relationship between an independent variable and a dependent variable via the inclusion of a third explanatory variable. Indirect effects of neighbourhood disadvantage are thought to be more influential in children, with diminishing influence throughout older adolescents and adulthood (Ingoldsby & Shaw, 2002), for instance through the impact of disadvantaged neighbourhoods on stress in parents (Klebanov, Brooks-Gunn, & Duncan, 1994). It might be expected then that these indirect effects play more of a role in adolescents than they do in adults. Examination of mediating pathways at both the neighbourhood and individual levels could allow for the establishment of plausibility of associations and the relative importance of multiple pathways. This could contribute to the development of more effective and targeted prevention and intervention programmes. A summary of mediation will be provided in Chapter 3, while research surrounding the mediating pathways examined in the current thesis will be discussed in Chapters 4, 5 and 6.

2.12 Summary and conclusions

The influence of neighbourhood on the development of internalising disorders in adolescents is beginning to receive attention in both theoretical and empirical literature. This thesis focuses on the influence of neighbourhood disadvantage (measured as a composite measure of characteristics of neighbourhood disadvantage) on risk for depression and anxiety in adolescents. Neighbourhood disadvantage has been found to be associated with depression and depressive symptoms, even when controlling for a variety of individual-level factors. The research reported in this thesis primarily aims to examine the mechanisms through which neighbourhood disadvantage influences risk for anxiety and depression, which is the focus of the next three introductory chapters. That is, I attempted to examine important family and adolescent characteristics and experiences that might be reactive to neighbourhood context. This thesis utilises a prospective longitudinal design in which neighbourhood location was assessed at baseline and used to predict the development of symptoms of depression and anxiety in a sample of adolescents with no history of depression or anxiety at baseline.
CHAPTER 3

MEDIATIONAL ANALYSES

As discussed earlier, it is thought that children and adolescents develop in the presence of a range of different factors – some proximal, such as parenting or stress, and some more distal, such as neighbourhoods. These factors are unlikely to operate in isolation. That is, it is likely that proximal and distal factors interact throughout the course of development to influence mental health. Understanding some of these complex interactions is possible using the class of statistical techniques referred to as mediational analyses. The following chapter will summarise mediational analysis as a prelude to the following three chapters, which detail the specific mediational hypotheses examined in the current thesis. Section 3.1 will provide a justification for the use of mediational analyses in regard to neighbourhood research. Section 3.2 gives a general overview of mediation, before describing the specific techniques used for mediation analyses in the current thesis. Furthermore, there are several complications involved in mediation research. Section 3.3 will outline these complications, with an explanation of the ways in which the current thesis intends to overcome them.

3.1 Justification for mediational research

Mediational analyses investigate the possibility that factors may not directly influence an outcome, but instead indirectly influence outcomes through ‘messenger’ or mediating factors. Because neighbourhood context is a distal risk factor for adolescent and child anxiety and depression, some researchers consider that it may only have a weak direct effect on internalising disorders (Ingoldsby & Shaw, 2002). Instead, these researchers propose that neighbourhood context may influence proximal factors (i.e., mediators), such as stress or parenting, that ultimately influence individual adjustment (Ingoldsby & Shaw, 2002). Understanding these mediating relationships may give insight into the mechanisms through which neighbourhood influences risk for depression and anxiety in adolescence. Appropriate use of mediation analysis has the potential to better establish the plausibility of associations and the relative importance of multiple pathways. This could thereby allow for the development of more targeted clinical and public health interventions (Colder, Lengua, Fite, Mott, & Bush, 2006), where interventions aim to change the
outcome of interest by targeting mediating variables that are hypothesized to be more proximally related to the outcome.

The model under examination in this thesis is based on the integration of the bodies of literature concerning internalising disorders, neighbourhood disadvantage, stress, temperament and parenting behaviours (reviewed in Chapters 1, 2, 4, 5, and 6). In particular, this thesis proposes that disadvantaged neighbourhoods predict baseline and prospective development of symptoms of depression and anxiety in adolescents, and that this relationship may be partially mediated by adolescent temperament, parental expression of emotion and stressful life events.

3.2 Overview of mediation analyses

Mediation refers to the process in which a particular variable represents the mechanism through which an independent variable influences an outcome variable (Baron & Kenny, 1986) (see figure 4). This represents a ‘chain’ of causal variables, whereby the independent variable predicts the mediating variable, which then predicts the outcome variable, given that the independent variable does not predict the dependent variable in the presence of the mediating variable (MacKinnon, Fairchild, & Fritz, 2007). Partial mediation refers to the process whereby the mediating variable does not completely account for the relationship between the independent variable and the outcome variable (MacKinnon et al., 2007; Baron & Kenny, 1986).

Figure 4: A simple mediation model

The most widely used method to assess mediation is detailed in the classic paper by Baron and Kenny (1986), which outlines four steps that they claim are needed to establish mediation. In regard to the current thesis – using stress as an example – Baron and Kenny would suggest that it is necessary to demonstrate the following steps (which are represented diagrammatically in figure 5 below):
1) The significant direct effect of neighbourhood disadvantage on internalising symptoms (i.e., path ‘c’ in figure 5);
2) A significant relation between neighbourhood disadvantage and the mediating variable (e.g. the ‘a’ path between neighbourhood and stress);
3) The significant direct effect of the mediating variable on internalising symptoms (e.g. the ‘b’ path between stress and internalising), with neighbourhood disadvantage also included as a predictor; and
4) That the effect of neighbourhood disadvantage on internalising symptoms is significantly reduced when the mediator variable is included as a predictor in step 3, compared to when neighbourhood disadvantage is the only predictor in step 1. That is, that the coefficient relating the independent variable to the dependent variable must be larger in absolute value than the coefficient relating the independent variable to the dependent variable in the regression model with both the independent variable and the mediating variable predicting the dependent variable.

Figure 5: Mediating relationships examined in the current thesis

In complete mediation, the effect of neighbourhood disadvantage on internalising symptoms would become non-significant after accounting for stressful life events. However, this complete mediation rarely occurs in psychological sciences (Zhao, Lynch, & Chen, 2010). It is far more common to find evidence of partial mediation, where, in this example, neighbourhood disadvantage would remain a significant predictor of internalising symptoms, however the size of its effect would be significantly reduced after accounting for the influence of stress.
Whilst Baron and Kenny’s formulation of mediation has been the established norm for many years, more recent research has suggested that there are various limitations with this approach. While Baron and Kenny propose that a direct relationship from the independent variable to the dependent variable is required, several statistical analysts have suggested that it is not necessary to have a significant path from the independent variable to the dependent variable in order for mediation to occur (Zhao et al., 2010; MacKinnon et al., 2007). In addition, the Baron and Kenny approach has been found to severely reduce the power to detect mediation, and produces an unacceptable proportion of false-negative results (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). One research paper determined that in order to detect a mediated effect with 0.8 power and a small effect size in the ‘a’ and ‘b’ pathway, a sample size of 21,000 subjects was required when using the Baron and Kenny approach (Fritz & MacKinnon, 2007). Mackinnon and colleagues suggest that mediation can be calculated using a method in which the product of paths $a$ and $b$ ($a \times b$) is calculated, termed the indirect effect. The indirect effect refers to the prediction of the dependent variable by the independent variable, via a third mediating variable. The equation $a \times b$ has been found to be equivalent to the fourth step in the Baron and Kenny approach described above (MacKinnon et al., 2002). This suggests that that it might not be necessary to consider path $c$ when investigating mediating relationships.

Research has indicated that there are many cases where significant mediation exists, but where there is not a significant direct effect of the independent variable on the dependent variable. This is termed ‘indirect-only mediation’, by Zhou and colleagues (2010). In order to test for indirect mediation, along with other forms of mediation, it is now commonly recommended that researchers use a bootstrapping statistical method, which has more statistical power and more accurate confidence intervals when testing indirect effects (MacKinnon, Lockwood, & Williams, 2004; Pituch, Stapleton, & Kang, 2006). Bootstrapping involves the generation of an empirical sampling distribution of $a \times b$, repeatedly drawing $N$ values to create new samples. Baron and Kenny’s equations 1 and 3 are estimated for each bootstrap sample, which allows for the estimation of $a$, $b$ and $a \times b$. The statistical program being used is able to estimate the indirect effect as the mean of these estimates. Bootstrapping relies on the 95% confidence intervals from the empirical distribution of the $a \times b$ estimates. The current thesis uses a bootstrapping approach for all mediational analyses.
### 3.3 Issues with mediational analysis

Despite being a powerful statistical tool, some research indicates that there are several limitations regarding mediation analyses. This section will outline these limitations, and suggest several ways in which these limitations can be overcome.

One of the major issues with mediation analysis is the problem of ‘equivalent models’. Some statisticians argue that if the IV, mediator and DV are measured simultaneously, there are other models that could explain the data equally well (for instance, rather than stress mediating the relationship between neighbourhood disadvantage and internalising symptoms, internalising symptoms could mediate the relationship between neighbourhood disadvantage and stressful life events) (Shrout & Bolger, 2002). In many situations, it is not possible to distinguish between these competing pathways without more information (Spirtes, Glymour, & Scheines, 2001). In order to deal with this issue, some researchers propose several steps to better deal with mediating relationships (Wu & Zumbo, 2008; MacKinnon et al., 2007). First, they suggest that mediation models be treated as descriptive, not causal, information. Second, mediating relationships should be supported by a strong theoretical basis. Finally, it is suggested that longitudinal mediation models be used to help investigate temporal-precedence or causal-ordering assumptions by quantifying mediation relations among variables over time. Longitudinal models also help to establish whether a mediated effect is stable over time. Thus, the current thesis examined cross-sectional mediation, but also investigated whether the three variables in question mediate the relationship between neighbourhood disadvantage and change in symptoms of anxiety and depression over 4 years. This allowed for a more thorough investigation of the mediating influence of stress, parenting, and adolescent temperament between neighbourhood disadvantage and adolescent internalising disorders.

### 3.4 Summary

This chapter outlined the importance of understanding mediating factors between neighbourhood disadvantage and internalising disorders. The current trend in statistical research indicates that, in comparison to the Baron and Kenny approach, bootstrapping techniques are a more powerful way to examine mediating relationships. While there are several limitations with mediational research, the current thesis has proposed a variety of ways in which to overcome these limitations. The following three chapters will outline research surrounding each of the specific mediating relationships examined in the current thesis.
CHAPTER 4

STRESSFUL LIFE EVENTS AS A MEDIATING FACTOR BETWEEN NEIGHBOURHOOD DISADVANTAGE AND INTERNALISING DISORDERS

The following chapter will explore research surrounding the hypothesis that the effect of neighbourhood disadvantage on internalising disorders is partly mediated by stressful life events. That is, neighbourhoods could alter the risk of adolescents experiencing stressful life events, which in turn could alter risk for depression and anxiety. The chapter will begin by providing a description of stressful life events in section 4.1, and will outline the various ways in which stress has been conceptualised. Section 4.2 will summarise the ways in which stress is measured in the empirical literature. The chapter will then detail the evidence supporting the hypothesis outlined above, including research suggesting that (1) neighbourhoods increase risk for stressful life events (section 4.3) and (2) that stressful life events increase risk for internalising disorders, including a review of empirical research (section 4.4) and theoretical explanations for this relationship (section 4.5). Section 4.6 summarises the small number of studies that directly examine the possibility that stress is a mediator between neighbourhood disadvantage and internalising disorders. Section 4.7 then examines the impact of neighbourhood stress of adolescent boys versus girls.

4.1 What is a stressful life event?

Stress is defined as a psychological and physiological response and adaptation to stressors (which are perceived threats to one’s well-being) (Maio-Esteves, 1990). The specific stress response depends on the individual’s perception of the stressors, which is influenced by factors such as an individual’s age, past experience, and gender (Donatelle, 2009; Maio-Esteves, 1990). The stress response is also influenced by the nature of the stressor, such as its frequency or predictability (Maio-Esteves, 1990).

Chronic stress refers to the constant exposure to various stressors or repeated stressful events (Baum, Garofalo, & Yali, 2006). Even if there is only one source of stress or danger, perception
of this stressor can become a chronic stressor. So, for instance, chronic stress may result from an individual exposed to the threat of violence from a neighbourhood gang (even if this occurs very infrequently), or from the experience of many separate stressful life events. Chronic stress has been referred to as the “disease of prolonged arousal” (Donatelle, 2009, p.65), and is associated with a range of negative outcomes in individuals (discussed in section 4.4).

The current thesis explores the relationship between stressful life events (one potential source of chronic stress), neighbourhood disadvantage, and internalising symptoms in adolescence. Stressful life events are discrete (time-limited) occurrences with a clearly identifiable onset and ending (Goodyer, 2001). They include events such as the loss of a job, the death of a neighbour, a motorcycle accident, or admittance to hospital. Early adolescence is accompanied by increasing exposure to major life events, many of these quite stressful (Larson & Ham, 1993). Stressful life events are more impactful than daily hassles (which include mildly stressful events that occur on a regular basis), but generally not as extreme as traumatic life events, which refer to extremely stressful or life-threatening situations that result in emotional or psychological injury. Stressful life events are also far more common than traumatic events and are thus perhaps more relevant to a community sample. They are also relevant in that they are highly predictive of the outcome measures used in the current thesis (depression and anxiety). Details of this relationship will be discussed in section 4.4. The following section will discuss the various measures of stress, comparing the merits of each approach.

4.2 How is stress measured?

The experience of stress is assessed using a variety of different measures, ranging from minimal questionnaires to comprehensive interviews. Each of these approaches has strengths and weaknesses.

The questionnaire approach tends to ask participants to note if they have experienced any of the stressful life events provided to them in a list of various categories of events. They may also be asked to rate the impact of these stressful life events. Compared to interviews, questionnaires are much easier to administer and are less taxing on individuals, thus can be administered to a larger number of participants. Some well-validated and reliable measures of stressful life events include the Social Readjustment Rating Scale (SRRS) (Holmes and Rahe, 1967), which is a list of 43 stressful life events that can contribute to illness, and the Stressful Life Events Screening Questionnaire (SLESQ) (Goodman, Corcoran, Turner, Yuan, & Green, 1998), which is a 13-
item self-report measure. Measures of chronic stress include the Childhood Adversity Checklist (CAC), a 16-item checklist modified from a questionnaire developed by Kupfer and colleagues (Kupfer & Detre, 1974; Cohen, Coyne, & Duvall, 1993), which is used to determine the occurrence of specific circumstances contributing to chronic stress during childhood. Stressful life events and chronic stress measures tend to overlap, given that stressful events will often lead to the experience of chronic stress.

While questionnaires tend to be cheaper and less time-consuming, they have several disadvantages (Lewinsohn et al., 2003). One problem is that respondents may interpret the definition of specific stressful life events in a different manner to the researcher. This approach may also result in more discrepancies when scoring intrinsically-related life events. For instance, when a person loses their job, they may also experience loss of contacts and a change in financial status, and it may not be clear to the individual whether they should count these events as a single or multiple stressors. Demonstrating this is evidence showing that test-retest reliability for questionnaire assessments covering the same period is often poor (Jenkins, Hurst, & Rose, 1979).

The interview approach involves a more comprehensive and higher quality assessment of stressful life events. These interview measures were developed to overcome some of the issues discussed previously. These approaches provide a protocol for a detailed investigation of the circumstances and timing of each stressful life event. The interviewer then uses this information to make judgments about effect of the event on the person. Two examples of these kinds of assessments are Brown's Life Events and Difficulties Schedule (1981) and the Bedford Life Events and Difficulties Schedule (LEDS), which is a semi-structured interview of life stress (Brown & Harris, 1989) in adulthood. This interview has also been adapted for use with adolescents (Monck and Dobbs, 1985). While these assessments are generally considered to have preferable validity when compared to questionnaires (e.g., Monroe, Kupfer, & Frank, 1992), they are time-consuming (taking several hours to administer), and costly.

The current thesis has taken an intermediate approach, with a combination of a comprehensive questionnaire and brief interview to follow up on the events reported in the questionnaire. The questionnaire lists 33 life events, selected from an initial set of 59 events culled from various measures such as the Schedule of Recent Experiences (Holmes & Rahe, 1967) and Life Events Schedule (Sandler & Block, 1979). The brief stressful life events interview was closely adapted by
Lewinsohn and colleagues (2003) from the LEDS and other stress interviews such the Structured Event Probe and Narrative Rating Method for Measuring Stressful Life Events (Dohrenwend, Raphael, Schwartz, Stueve, & Skodol, 1993). It provides some degree of assessment of the fidelity of the event, where the interviewer probes to find out whether events meet criteria for a stressful life event. This overcomes the issue that stressful life events tend to be associated with a multitude of other stressful life challenges. For instance, a fight with friends may be associated with feelings of rejection and loneliness. Or the divorce of parents may be associated with life challenges, such as moving house, dealing with parents with their own mental health issues, family conflict, and financial difficulties. Thus, it is very difficult to pinpoint the exact cause of the relationship between stressful life events and symptoms of depression and anxiety, and the exact number of stressful events.

A structured interview is one way to provide a way to a better assessment of the exact number of stressful life events, according to criteria stipulated by researchers. Lewinsohn and colleagues (2003) found that approximately two thirds of the events reported by questionnaire were verified by the interview. While the interview also allows for assessment of the subjective reaction of the individual to these stressors, only objective counts of the frequency of these stressful life events was examined. While this is limited in that it does not measure individual differences in response to stressors, it avoids the issue of confounding, whereby subjective ratings of stressful life events are biased by current depression or anxiety (Kim, Conger, Elder Jr, & Lorenz, 2003). It is worth noting that the approach taken in the current thesis minimises ‘false positives’, but does have the possibility of missing ‘true positives’, as the interview does not ask the respondent for further stressful life events. Thus this approach is likely to underestimate the number of stressful life events. However, it provides a compromise between quality of data and efficiency of delivery.

The following section reviews evidence for the relationship between neighbourhood disadvantage and stress in adults and adolescents.

4.3 How do disadvantaged neighbourhoods increase risk for stressful life events?

Disadvantaged neighbourhoods are more stressful places in which to live. Broadly speaking, these neighbourhoods are thought to increase stress for residents through two different sources: (1) the physical characteristics of the neighbourhood, such as dilapidated buildings or a lack of resources, and (2) the people in the neighbourhood, who may pose threats to physical safety (Kim, 2008).
In terms of physical characteristics, disadvantaged neighbourhoods are more likely to have low-quality housing, high traffic density, and undesirable commercial operations, such as adult bookstores (Sampson et al., 2002). These neighbourhoods often lack many resources, including health care, retail stores, and recreational facilities. This can prevent residents from meeting their daily needs and can be demoralizing (Sampson et al., 2002), thus leading to a build-up of stress over time.

In terms of people in the neighbourhood, disadvantaged neighbourhoods are more dangerous, have higher rates of crime, and are more disordered and unpredictable (Hill et al., 2005). Thus, people living in disadvantaged neighbourhoods are much more likely to experience stressful life events, fear of victimization and chronic stress in comparison to those from less disadvantaged neighbourhood (Warr, Feldman, Tacticos, & Kelaher, 2009; Hill et al., 2005). Several studies have found a relationship between neighbourhood crime rates and perceptions of stress for adults and adolescents (e.g., Lewis & Riger, 1986). The effect of dangerous neighbourhoods may be particularly potent for adolescents as they spend a great deal of time in their neighbourhood (Morrow, 2000), which may result in increased exposure to these neighbourhood stressors. In fact, adolescents do not even have to have been exposed to these stressful or traumatic events in order to feel stressed; higher stress can occur in both victims themselves, and those who hear about the experiences of nearby victims (Lewis & Riger, 1986).

Increased rates of crime and disorder can also lead to a sense of isolation for neighbours as they may distrust their fellow residents (Stockdale et al., 2007). When residents distrust one another and isolate themselves, they can often fail to develop positive relationships. This can then affect the social cohesion of the neighbourhood - one protective factor for stress (Stockdale et al., 2007). Thus disadvantaged neighbourhoods are also stressful in that they lack social support and social resources (Cutrona et al., 2006). The current thesis potentially taps into risk factors associated with exposure to victimisation and violence through specific questions in the questionnaire including those related to being a victim of, or witnessing, a crime, violence or assault.

In conclusion, it appears that individuals in disadvantaged neighbourhoods are exposed to a significantly greater number of stressful life events, which may translate into the experience of chronic stress (Frydenberg, 2008). It is worth noting that the current thesis does not differentiate between stressors originating in the neighbourhood and other stressors. Whilst this could be
considered a limitation, I am simply interested in whether there is a systematic link between disadvantaged neighbourhoods and the experience of a range of stressful life events. As will be discussed in the following section, there is evidence that the increased stress experienced by those living in disadvantaged neighbourhoods is likely to increase risk for depression and anxiety.

4.4 Stress increases risk for depression and anxiety

Having established evidence for the relationship between neighbourhood disadvantage and increased stress in adolescents, the following section will outline evidence for the relationship between stress and internalising disorders.

A large body of literature suggests that high levels of stress are associated with a variety of negative outcomes in individuals. While a low level of stress is not problematic, repeated stressful life events and/or chronic stress can increase risk for health problems such as heart disease, diabetes, cancer, fatigue, and headaches (Bendelow, 2009; Donatelle, 2009) as well as psychological issues such as depression, anxiety, addiction to alcohol or other substances, homicide, and suicide (Stockdale et al., 2007). Stressful life events are a significant contributor to risk for internalising disorders: one study of a clinical population found that stressful life events accounted for approximately 9% of depressive symptoms in children (Luby, Belden, & Spitznagel, 2006). The relationship between stress and internalising disorders has even been shown to be independent of genetics, with one study finding that anxious children who are part of a monozygotic twin pair are more likely to report independent negative events than is their non-anxious twin (Eley & Stevenson, 2000).

Many of the studies conducted so far have been conducted with clinical and/or cross sectional samples, who provide data on both their current symptomatology and retrospective ratings of the number of stressful life events they have experienced. This presents the problem that responses given by those currently suffering from an internalising disorder are more likely to retrospectively report more stressful events due to mood congruent memory (Dalgleish & Watts, 1990), whereby individuals are more easily able to recall events that occurred while they were in a similar mood to their current mood. Researchers have begun to prospectively study the effects of stressful life events to overcome this issue. This approach minimises the influence of current mood on the reporting of stress and anxiety (and vice versa), to provide a clearer perspective of whether stressful life events are predicting later psychopathology.
A large number of prospective studies of children have found a significantly increased number of life events were reported prior to the onset of mental health problems such as anxiety and depression (Goodyer, 1996; Hillegers et al., 2004; Kendler, Karkowski, & Prescott, 1999), as well as externalizing behaviours, suicidal tendencies, and anorexia nervosa (Jensen, Richters, Ussery, Bloedau, & Davis, 1991). One of the most powerful studies of stress and mood disorders examined the cumulative effect of stressful life events on internalising symptoms in 451 adolescents over five data collection periods spaced one year apart (Kim et al., 2003). These researchers found that life stresses experienced by adolescents impacted significantly on their emotional distress. That is, the higher the number of stressful life events, the higher the levels of anxious and depressive symptomatology. They concluded that stressful life events increase risk for depression and anxiety in adolescents. Interestingly, they also found a reciprocal relationship between these two constructs, with internalising symptoms increasing risk for stressful life events. For example, this may occur in situations where adolescents who are sad or withdrawn may have poor social relationships because they are unrewarding companions. Anxiety and depression could also interfere in academic studies, which require focus, energy, and effective regulation of emotions. Thus, it appears likely that there is a bi-directional relationship between stressful life events and internalising symptomatology and associated behaviour in adolescents.

Research also indicates that cumulative exposure to stressors is predictive of a range of psychological and behavioural problems in adolescents (e.g., Compas, 1987). Thus the more stressors experienced by an individual, the greater the chance that they will experience greater levels of anxiety and depression. Indeed the presence of one stressor is often not enough to lead to significant maladjustment. A series of stressful life events are required for the development of serious emotional or behavioural problems (Forehand, Middleton, & Long, 1987). One study by Garmezy (1987) found that the effect of stressors on psychological well-being was not just additive but multiplicative. In this study it was found that two risk factors increased risk by fourfold for a psychological disorder, while four factors increase risk 10-fold.

In conclusion, it appears that stressful life events are an important risk factor in adolescent psychopathology. This research also points to the importance of measurement of internalising symptoms separately (in time) from measurement of stressful life events. In order to avoid the effect of confounding, the current thesis observes change in internalising symptoms in adolescents over four years. In addition, measurement of stressful life events is spaced at least 1.5 years apart from measurement of internalising symptoms.
4.5 Theories for the relationship between stress and anxiety

There are various theories linking stress to internalising disorders, and these will be discussed in the following section. The most well established theory to explain the link between stress and psychopathology is the diathesis-stress model (see Belsky & Pluess (2009) for a review). This model proposes that while certain individuals are predisposed to various forms of psychopathology (due to both genetic and environmental factors), it is often only under stressful life circumstances that individuals develop these disorders, through an interaction of predisposing factors and stress. However, there is not a consensus on the exact mechanisms through which this occurs, and various different pathways have been proposed. Some of the psychological theories propose that stress increases negative affect, depletes existing social support resources, erodes self-concept (e.g., mastery), and minimizes options for coping behaviour (Kessler, Price, & Wortman, 1985; Mirowsky & Ross, 1986), all factors thought to increase risk for depression and anxiety. A neurobiological mechanism proposed by Heim and Nemeroff (2001) suggests that stressful life events may induce ongoing hyperactivity of the hypothalamic pituitary axis as a result of the initiation of the fight or flight response. This leads to increased endocrine, autonomic and behavioural stress responses (including the release of adrenaline and cortisol), which can lead to deterioration of the immune system and physical problems, such as chronic illness, exhaustion, unhealthy eating and sleeping habits, hyperventilation, muscle tension and headaches (Donatelle, 2009; Goodman, Huang, Schafer-Kalkhoff, & Adler, 2007; Pickett and Pearl, 2001). When stressful life events and associated physical problems accumulate, this vulnerability is thought to result in symptoms of internalising disorders (Heim & Nemeroff, 2001). There is even evidence that stress can cause changes in the brain that predispose adolescents to increased risk for depression and anxiety. For instance, one study found that the amygdala increases in size in response to stress, which may encourage behaviours and emotions associated with anxiety and depression (Lupien, McEwen, Gunnar, & Heim, 2009).

4.6 Stressful life events as a mediator between neighbourhood disadvantage and internalising disorders

The previous sections have outlined research presenting strong evidence that neighbourhood disadvantage is associated with increased risk for stressful life events, and also prospective evidence for a relationship between stressful life events and internalising disorders in adolescence. Thus it appears plausible that stressful life events act as a mediator between
neighbourhood disadvantage and internalising disorders. However, to date there has been limited research directly examining this hypothesis, and findings have been mixed. The following section reviews the small number of studies that have examined the role of stressful life events in mediating the relationship between neighbourhood disadvantage and internalising disorders, and explains how the current thesis will add to this literature.

One study examined the relationship between neighbourhood disadvantage, stressful life events and internalising and externalizing symptoms in 384 primary school children from the first, second and fourth grades (Attar et al., 1994). The authors found that in the most disadvantaged neighbourhoods, the mean number of stressful life events (measured using a questionnaire) was nearly double that found in neighbourhoods of more moderate neighbourhood disadvantage. This increased number of stressful life events was found to be associated with increased aggressive behaviour in children one year after stressful life events were assessed. However, no formal mediation analyses were used to test these relationships. This study also examined symptoms of anxiety and depression, but, contrary to predictions, stressful life events were not associated with these measures. Researchers hypothesised that internalising behaviour may not be adaptive in a harsher environment, where it may be more effective to engage in aggressive behaviour in response to stress. Another reason for the lack of relationship between stress and internalising symptoms could also relate to the fact that measures of internalising disorders were provided by the children’s teachers, who may not have a detailed understanding of these children’s symptoms, especially in the internalising domain. In addition, this study was conducted in younger children (6-9 years old), and most research demonstrating a link between neighbourhood stressors and internalising symptoms has been conducted in samples of older children and adolescents (10-15 years old) (e.g. Compas, Howell, Phares, Williams, & Giunta, 1989; Larson and Ham, 1993). The current thesis will aim to improve upon this study by using formal mediation analyses, by using a questionnaire and interview approach for measurement of stressful life events, and by obtaining detailed symptom data directly from a sample of adolescents.

Another study failed to find a consistent mediating effect of stress for the relationship between neighbourhood disadvantage and internalising symptoms. Researchers used structural equation modelling to analyse data from 738 Mexican American adolescents and found that stressful life events mediated the relationship between neighbourhood disadvantage and internalising symptoms in fourth generation Mexican children, but not in first generation children (Roosa et
al., 2010). They also failed to find a direct relation between neighbourhood disadvantage and stressful life events. Researchers were unsure why this finding might have occurred, and concluded that additional research was needed to corroborate these results and determine why neighbourhood disadvantage may have different relationships to adjustment for Mexican American early adolescents than for others. They also suggested that these findings may not be generalizable to other ethnic populations. Furthermore, this study used questionnaires, not interviews, to measure stressful life events, and an idiosyncratic measure of neighbourhood disadvantage, which included only three indicators of neighbourhood disadvantage (percent of female headed households with children, percent of families with incomes below the poverty line, and percent of unemployed males). The current thesis aimed to improve upon this study by using a longitudinal design and more comprehensive measures of stressful life events and neighbourhood disadvantage.

Finally, one longitudinal research paper used data from the National Survey of Children (N = 1423) to simultaneously assess the effects of current and past neighbourhood on internalising symptoms (Wheaton & Clarke, 2003). The researchers found a lagged effect of childhood neighbourhood socioeconomic disadvantage on early adult mental health, while accounting for initial mental health status. They also found that life course stress and ambient neighbourhood stress mediated the relationship between neighbourhood and change in internalising symptoms over time (note that path analysis, not direct mediation analyses, were used to measure these relationships). This was found even when individual- and family-level variables were taken into account. Researchers concluded that most of the effect of the neighbourhood environment on mental health over time is a combined function of the accumulation of first discrete, and then chronic stressors at the individual level. Interestingly, the immediate effects of neighbourhood at both points in time were weaker than the lagged effect.

In conclusion, there are very few studies examining whether stressful life events mediate the relationship between neighbourhood disadvantage and internalising disorders in adolescents. Of those that have been conducted, there have been very mixed findings. This is surprising, given that there is strong evidence that disadvantaged neighbourhoods increase risk for stressful life events, and strong evidence that stressful life events increase risk for depression and anxiety. The current thesis will hopefully shed some light on this issue by improving on some of the shortcomings of the previously mentioned research by using a longitudinal design, a comprehensive measure of neighbourhood disadvantage, and a combination of interview and
questionnaire to measure stressful life events. It will also use a more contemporary approach to mediation analysis (i.e., direct mediation analyses using bootstrapping), which overcomes issues noted in chapter 3.

4.7 Impact of neighbourhood stress on adolescent girls versus boys

Research suggests that there are differences between males and females in terms of their experiences with, and reactions to, stress (Sigfusdottir & Silver, 2009). Overall, it appears that boys tend to report more stressors associated with their neighbourhoods, such as violence, accidents and illnesses (Sigfusdottir & Silver, 2009). Thus it might be expected that boys report greater stressful events as a result of neighbourhood disadvantage. However, girls tend to report stress resulting from relationship-based stressors such as those associated with friendships, family and intimate relationships (Morrow, 2000; Sigfusdottir & Silver, 2009), which may be increased in disadvantaged neighbourhoods with poor social cohesion. In addition, girls report more fears and experiences of neighbourhood-level stressors that relate to rape, sexual assault, and other types of violence targeting females (Cicognani et al., 2008; Morrow, 2000). Thus it is unclear whether boys or girls are likely to experience greater stress as a result of neighbourhood disadvantage.

Girls and boys also tend to react to their stressors differently, with evidence that adolescent girls report more stress and tend to be more sensitive to stressors than boys (Coleman, 2007; Frydenberg, 2008). Whilst girls tend to internalize their feelings in response to stress, boys tend to externalize them (Sigfusdottir & Silver, 2009). Stress-associated internalising tends to lead to an increased risk for depression and anxiety (Sigfusdottir & Silver, 2009). Thus it appears that on average, stress impacts upon girls more than boys. One longitudinal study examined this issue by measuring change in depressive symptoms and stressful life events in 191 girls and 185 boys over 4 years. The trajectories of depressive symptoms differed between boys and girls. While depressive symptoms were related to the level of stressful life events in both girls and boys, only girls experienced changes in depressive symptoms associated with changes in stressful life events (Ge, Lorenz, Conger, Elder, & Simons, 1994). Results regarding neighbourhood stress and gender are not always consistent. For instance, another study did not find random variation in depression by gender across neighbourhoods of differing levels of stress (Matheson et al., 2006). Given the weight of evidence, it could be expected that females might suffer more internalising symptoms as a result of stress induced as a result of neighbourhood disadvantage.
4.8 Summary and review

This chapter has reviewed evidence concerning the influence of neighbourhood disadvantage on stress in adolescents, as well as evidence concerning the influence of stress on internalising disorders in adolescents. When considered together, these two bodies of literature suggest a plausible causal chain in which neighbourhood disadvantage influences risk for stressful life events, which in turn influences the development of internalising disorders in adolescents. There is very little research examining this model using formal mediational analyses. Filling this gap in the literature is one of the central goals of this thesis, and the specific model, research questions and hypotheses under investigation will be detailed in Chapter 7.
CHAPTER 5
TEMPERAMENT AS A MEDIATING FACTOR BETWEEN NEIGHBOURHOOD DISADVANTAGE AND INTERNALISING DISORDERS

The following chapter will explore research surrounding the hypothesis that the effect of neighbourhood disadvantage on internalising disorders could be partly mediated by temperament. That is, neighbourhoods could alter the development of temperament, which could then alter risk for depression and anxiety. The chapter will begin by providing a definition of temperament and will outline the specific dimensions of temperament examined in the current thesis (section 5.1). The chapter will then detail evidence suggesting that temperaments are malleable and subject to various environmental influences including parenting, stress and neighbourhood social influences (section 5.2.). Section 5.3 explains how these changes in temperament may lead to increased risk for internalising disorders. The chapter will then conclude by summarising the mechanism proposed in the current thesis.

5.1 What is temperament?

Affective temperament refers to biologically based individual differences in emotional reactivity and regulation that emerge early in development, remain relatively constant across the lifespan, and vary normatively across the healthy population (Rothbart & Derryberry, 1981).

Various temperament dimensions have been proposed throughout the past sixty years, with many seemingly different theories of temperament. One of the earliest theories of temperament originated in ancient Greece, which included the black bile or melancholic type, sanguine or cheerful type, choleric or irritable type, and phlegmatic or calm type (Kagan, Snidman, Arcus, & Reznick, 1994). More recently, research in the area received a significant boost with Thomas and Chess’s influential theory that featured nine subcategories of temperament and three temperament constellations including Easy, Difficult, and Slow-to-warm-up children (Chess, Thomas, Birch, & Hertzig, 1960). Various other theories of temperament were proposed after
this, including theories by Kegan (1996), Streleu & Zawadzki (1983) and Buss and Plomin (1997).

The large number of theories of temperament could be interpreted to suggest a lack of consensus in the temperament literature. However, researchers will often rename temperament variables even when the content of the previous and renamed temperament constructs is similar (Caspi, Moffitt, Newman, & Silva, 1996; Rothbart, Ahadi, & Evans, 2000). In fact, most of the theories mentioned previously contain several similar higher order constructs, with three overarching constructs being common to most theories of temperament. These include dimensions relating to (1) Negative affectivity, neuroticism, or harm avoidance (2) novelty seeking, extraversion and impulsivity and (3) behavioural and emotional regulation. Table 1 shows the higher-order factors of the various theories of temperament described in the current chapter. These temperament factors are grouped according to the Rothbart model of temperament (Rothbart & Derryberry, 1981), which is the model used in the current thesis and is described in detail below. Note that some of these temperament models include sub-factors that constitute higher-order factors. However, for the sake of simplicity, only higher order factors have been shown in the table.

<table>
<thead>
<tr>
<th>Broad Factor</th>
<th>Thomas &amp; Chess</th>
<th>Kagan</th>
<th>Rothbart &amp; Derryberry</th>
<th>Buss &amp; Plomin</th>
<th>Strelau &amp; Zawadzki</th>
</tr>
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<tr>
<td>Negative Affectivity</td>
<td>Quality of Mood Intensity of Reaction</td>
<td>Inhibited</td>
<td>Negative Affectivity</td>
<td>Emotionality</td>
<td>Emotionality</td>
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<tr>
<td>Surgency</td>
<td>Approach-Withdrawal Activity</td>
<td>Uninhibited</td>
<td>Surgency</td>
<td>Activity</td>
<td>Activity</td>
</tr>
<tr>
<td>Effortful Control</td>
<td>Distractibility Adaptable Attention Span Persistence</td>
<td>Effortful Control</td>
<td>Impulsivity</td>
<td>Perseveration Briskness</td>
<td></td>
</tr>
<tr>
<td>Affiliation</td>
<td>Affiliation (a separate factor only in 9-16 year olds)</td>
<td>Sociability</td>
<td></td>
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</tr>
<tr>
<td>Other factors</td>
<td>Rhythmicity Threshold Responsiveness</td>
<td>Sensory Sensitivity Endurance</td>
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</table>
It is worth noting that there has been disagreement in the literature about the difference between temperament and personality. Temperament is generally considered to involve endogenous basic tendencies that emerge in infancy, while personality has been conceived as a broader set of behavioural and cognitive preferences that are influenced by social processes and emerge later in development (Strelau, 1983; Thomas & Chess, 1977). It has also been suggested that temperament is a lifelong component of personality (Cloninger, 2000), and that personality develops from the basis of temperamental traits (Clark et al., 1994). However, it has also been argued that many of the basic components of temperament and personality are so closely related that previous distinctions are inadequate. Personality and temperament overlap in several key ways: they are both measurable early in life, they are heritable, relatively stable across situational contexts and time (particularly later in life), and are related to emotional and motivational behaviour (Nigg, 2006). This overlap has led some researchers to propose that the distinction is not justified (Rettew & McKee, 2005), and researchers are beginning to conceptually and empirically integrate these concepts (Nigg, 2006). Thus, although the distinction between temperament and personality is still a matter of debate, for the remainder of this thesis, research from both domains will be discussed concurrently.

One of the most extensively researched theories of temperament is that proposed by Rothbart and Derryberry (1981), which focuses on the biological basis of temperament. Rothbart and Derryberry’s approach highlights individual differences in reactivity and self-regulation, where reactivity refers to individual differences in arousal of negative emotions including fear and frustration and positive emotions including joy and pleasure, and self-regulation refers to processes that modulate reactivity and can facilitate or inhibit the affective response (Rothbart et al., 2000). This theory of temperament is generally considered one of the most comprehensive as it encompasses behavioural styles, predispositions to particular reactions (e.g., smiling), psychophysiological functioning and phenomenological experience. To develop their theory of temperament Rothbart used statistical, observational, and laboratory evidence from a large range of fields including personality, animal, behaviour genetics, psychophysiological, and temperament literature. On the basis of this literature they have developed a range of measurement instruments that cover the entire developmental range, including a parent-report and self-report questionnaire tailored to children and early adolescents between the ages of 9-16 (Rothbart et al., 2000).
Despite emphasising the biological aspects of temperament, Rothbart and colleagues recognise the influence of environmental and social factors on temperament. Thus, their theory acknowledges the changing nature of temperament over time. In accord with this, they have created distinct measures for different age periods that more accurately reflect the changes in the expression of temperament through childhood and adolescence. In order to reflect these changes they have allowed for the emergence of a fourth factor – Affiliation – during adolescence. Affiliation refers to the desire for closeness with others, and is independent of shyness or extraversion. Those high on this factor are also sensitive to low intensity perceptual and pleasurable stimuli. In younger children this is merged with Effortful Control, but is considered separate during adolescence as social connections and intimacy become more important, with behaviours such as sex and bonding occurring more frequently (Putnam, Ellis, & Rothbart, 2001). The current thesis examines all four higher order temperament dimensions of the Rothbart model of temperament, which are described below.

**Surgency** is characterised by high activity levels, rapid approaches to novel stimuli, impulsivity and sensation seeking, tendencies to smile and vocalise, and enjoyment of intense stimulation. This temperament dimension is similar to the personality dimension of Extraversion (Putnam et al., 2001). In adolescence, it refers to the seeking out of excitement in the form of risky sports or parties. A lack of shyness and fear are also added in adolescence to further define this factor (Putnam et al., 2001).

**Negative Affectivity** is characterised by the experience and expression of frustration or anger in response to limitations and can be observed from infancy to adolescence. It also encompasses the experience and expression of fear and shyness, which is negatively related to Surgency in adolescence (Putnam et al., 2001). Children who are high on this temperament dimension tend to take longer to communicate with or approach strangers, show signs of distress or withdrawal in response to novel stimuli, and are restricted or inhibited in the way they interact socially (Hirshfeld et al., 1992).

**Effortful Control** is defined as the ability to direct attention, and to regulate emotion and behaviour. It is seen as the product of the executive attention system that develops primarily throughout infancy and the first couple of years of life. However it continues to mature throughout adolescence and its maturation is likely to be influenced by environmental factors.
In adolescence, Effortful Control is related to inhibitory control, which refers to the capacity to suppress inappropriate actions or responses.

Affiliation refers to the desire for closeness with others, independent of extraversion or shyness. Those high on affiliation are also sensitive to low intensity perceptual and pleasurable stimuli. As mentioned previously, although considered a separate dimension in adolescence, this factor is merged with Effortful Control in childhood, as factor analysis has revealed one factor with positive loadings from scales indexing both aspects of Effortful Control and Affiliation. It is thought that a system underpinning Affiliation is activated during puberty, serving to encourage maternal behaviours, attachment, pair bonding, and sexual behaviours (Putnam et al., 2001).

5.2 Environmental factors influence temperament

This section will discuss research surrounding the various environmental influences on temperament including parenting, stress and neighbourhood social influence.

Temperament has generally been represented in the research literature as a stable, biologically-based construct (e.g., Buss & Plomin, 1975). Any change in temperament that was observed in studies was generally interpreted as measurement error. However, recent research has demonstrated that temperament does change over time, and that this change can be distinguished from measurement error (Roberts, Caspi, & Moffitt, 2001; Vaidya, Gray, Haig, & Watson, 2002). The actual influence of genetic factors on dimensions of temperament is estimated to be between 0.20 and 0.60 (Saudino, 2005), leaving variance to be explained by other factors, including environmental influence. Further, while it is true that temperament is relatively stable later in life, it is less so during childhood and adolescence. Roberts and Del Vecchio conducted a meta-analysis of more than a hundred studies of personality traits in childhood, adolescence, and adulthood (2000). They determined that rank-order stability (corrected for measurement error) ranged from .35 in infancy to .72 for adults aged 60 years and older. Thus, stability of personality appears to increase with age, with a relatively higher degree of malleability during childhood and adolescence. While this evidence suggests that temperaments can change over time, particularly during childhood and adolescence, it does not tell us which factors might influence such changes. The following section will discuss some of the factors that may be involved in the ongoing development of temperament traits during adolescence, including parenting, stress and neighbourhood social influence. Whilst the influence of these various factors will not be directly empirically examined in the current thesis, they are important in
understanding possible links between neighbourhood disadvantage and change in temperament, and provide a theoretical framework for the discussion of the findings of the current thesis.

5.2.1 Parenting and temperament

Perhaps one of the strongest influences on temperament is early child-rearing practices. A large number of studies have found links between various parenting styles and differences in Negative Affectivity. For instance, one study found evidence that inconsistent maternal discipline prospectively predicted sub-factors of Negative Affectivity, including irritability and fearfulness, even after controlling for prior levels of temperament and parenting (Lengua & Kovacs, 2005). Other studies have found that maternal involvement and contingency responding (i.e., actively responding to a baby’s attempts at communication) predicted increases in positive emotionality in infants (Belsky, Fish, & Isabella, 1991; Malatesta & Haviland, 1982), while maternal negative emotionality predicted increases in negativity in infants over time (Malatesta & Haviland, 1982). Children with complementary, responsive, and sensitive maternal parenting were found to be more likely to shift from a high-reactive temperament, to a low-reactive temperament (Belsky et al., 1991). Two recent studies found that negative emotionality was related to more negative and less positive parenting (Bridgett et al., 2009; Paulussen-Hoogeboom, Stams, Hermanns, & Peetsma, 2007).

Parenting also appears to influence self-control in children. For instance, studies have found that children with more responsive mothers are less likely to be impulsive (Olson, Bates, & Bayles, 1990) and are more likely to have higher effortful control (Kochanska, Murray, & Harlan, 2000). Another study found that the use of rewards predicted self-regulation in 3- to 5-year-old children (Kyrios & Prior, 1990). One longitudinal study determined that positive parenting attributes predicted increases in persistence in children from ages 4 to 7 years (Halverson & Deal, 2001).

Of course, the relationship between parenting and temperament is likely to be bi-directional, with child characteristics also influencing parenting behaviours. Evidence for this was found in a longitudinal study by Lengua and Kovacs (2004), which demonstrated that inconsistent discipline increased negative emotionality in children, while child irritability evoked inconsistent discipline in parents. Despite evidence for the influence of temperament on parenting, the weight of evidence suggests that parenting is likely to be a significant influencer of many aspects of child temperament, including self-regulation, and positive and negative emotionality.
5.2.2 Stress and temperament

Researchers have also begun to explore the influence of stress on temperament. Several studies indicate a link between stressful life events and increased Negative Affectivity. One study observed changes in the temperaments of older people in response to a stressful life event. Mroczek and Spiro (2003) measured temperamental change over a 12-year period and found that participants who experienced the death of a spouse became more neurotic (similar to Negative Affectivity) relative to those not bereaved. They also found that those who were unmarried showed no change in neuroticism, while those who married became less neurotic. While the magnitude of these changes was small, they occurred later in life when temperament is thought to be relatively stable.

Further evidence for a link between stress and change in Negative Affectivity has also come from several studies looking at younger populations. One study demonstrated that boys who lived in stressful disorganised noisy environments became more emotionally negative (similar to Negative Affectivity) with age compared with boys in calm environments (Matheny Jr & Phillips, 2001). This effect was not found for girls, however the sample size was relatively small in this study. Another study, by Vaidya and colleagues (2002) examined stress-related temperament change in college students. Over 2.5 years they observed changes in their temperament in response to positive and stressful life events. Negative life events measured at Time 1 were associated with Time 2 neuroticism even after controlling for neuroticism at Time 1. These researchers also found evidence to suggest that certain events could lead to increases in Surgency. Positive life events measured at Time 1 were found to predict higher scores on measures of extraversion (similar to Surgency) at Time 2, even after controlling for extraversion at Time 1. The researchers concluded that changes in the experience of stress resulting from positive or negative life events predict change in temperament. Further studies have found links between job loss and divorce and increases in neuroticism (e.g., Costa Jr & McCrae, 1997).

Stress may also be linked to changes in Effortful Control. One study found that children living in families with various risk factors such as low parental education, poor family functioning, or low work satisfaction, became less persistent (a component of Effortful Control) between the ages of 4-7 years (Halverson & Deal, 2001). This change was found to be fairly large ($r = .40$). Thus stressful home environments may encourage maladaptive changes in Effortful Control. Conscientiousness (similar to Effortful Control) has been found to decrease in individuals who
have been fired from their jobs (Costa Jr & McCrae, 1997), and divorced men also show decreases in conscientiousness, while those who have recently begun dating show increases in this factor (Neyer & Asendorpf, 2001). Subfactors of Extraversion, similar to Surgency, seemed to decrease in response to divorce in men, and increase in men and women after they began dating (Costa Jr & McCrae, 1997).

It appears cumulative stress may even cause children to ‘shift’ into different temperament categories over time. One study used measures of temperament provided by mothers to assign children at 3-4 years old then 5-6 years old to three classes of temperament: resilient, overcontrolled, and undercontrolled (Hart, Atkins, & Fegley, 2003). Children of the resilient type tended to be self-confident, high academic achievers and successful in relationships (this type is likely to have some overlap with high Surgency and Effortful Control); those assigned to the overcontrolled group were shy and socially withdrawn (similar to low Surgency and high Negative Affectivity); while those in the undercontrolled group tended to be delinquent, aggressive, and physically active (Similar to high Negative Affectivity and low Effortful Control) (Hart, et al., 2003). Hart and colleagues (2003) examined change in temperament type over a 2-year period. Surprisingly, only 50% of the sample remained in the same temperament class over this period. It was found that children from homes with many risk factors (e.g. low income, single parent, poor home environment) were more likely to move out of the resilient class to another class over the two-year period. Researchers hypothesised that stress had had a maladaptive effect on children’s resilience. These changes in temperament were also shown to have real-world implications: children who were assigned to the undercontrolled class during the second data wave showed less academic growth over the next 6 years, compared to children from the resilient or overcontrolled class. While the stress originated from a more proximal cause, this research suggests that neighbourhood stress could influence various temperament dimensions.

5.2.3 Regional social influence and temperament

Another pertinent factor thought to influence temperament and personality is regional social influence, which refers to the social norms and interactions of a neighbourhood (or an even wider area measure such as a city or state) that influence personality, temperament and behaviour styles of individuals (Bourgeois & Bowen, 2001). While the characteristics and social make-up of certain regions may have initially been set through differences in intellectual histories and daily practices of early residents, these characteristics are likely to establish social norms, which then
exert social influences on inhabitants (Kitayama, Ishii, Imada, Takemura, & Ramaswamy, 2006). After establishing these differences, it is possible that they persist and may even become more pronounced as geographic differences in personality feed into differences in typical behaviours and environments, which then encourage further changes in personality. Thus temperament or personality traits common to a particular region may mutually reinforce each other. For instance, communities higher on Negative Affectivity, where people are more likely to be frustrated and anxious, may influence those who are lower on Negative Affectivity to be more anxious or frustrated, which is likely to encourage the development of neurotic traits. Conversely, there is evidence that communities high on Agreeableness (similar to Affiliation) demonstrate more community involvement, social connectedness, and prosocial behaviour (Rentfrow, Gosling, & Potter, 2008). This in turn may encourage more agreeableness in community members. This research might suggest that disadvantaged neighbourhoods, where social connectedness tends to be lower, are likely to influence residents to have temperaments lower on Affiliation.

The theory of regional social influences is supported by a wide variety of research indicating that there are regional differences in personality and temperament (e.g., Steel & Ones, 2002; Van Hemert, Van de Vijver, Poortinga, & Georgas, 2002). McCrae and colleagues have published several studies examining the prevalence of five factor personality traits (Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Openness) across nations and across regions within nations (e.g. Allik & McCrae, 2004; Hofstede & McCrae, 2004; McCrae, Terracciano, Realo, & Allik, 2007). These personality traits correspond to four fundamental temperament dimensions (see Table 2 for details). McCrae determined that the prevalence of these personality traits varied across nations. For instance, Norwegian women were found to be higher on Extraversion and Openness than women from the USA (McCrae, 2001). Interestingly, these differences in personality across nations are also associated with meaningful difference in geographic social indicators such as cancer, obesity, life expectancy, and substance abuse (McCrae & Terracciano, 2008). For instance, women are more likely to smoke in countries where people tend to be higher on Extraversion.
Table 2
Approximate Correspondence Between Rothbart’s Temperament Dimensions and Dimensions Identified in the 5 Factor Model of Personality (Costa & McCrae, 1992)

<table>
<thead>
<tr>
<th>Temperament</th>
<th>5 Factor model of personality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative Affectivity</td>
<td>Neuroticism</td>
</tr>
<tr>
<td>Surgency</td>
<td>Extraversion</td>
</tr>
<tr>
<td>Effortful Control</td>
<td>Conscientiousness</td>
</tr>
<tr>
<td></td>
<td>Agreeableness</td>
</tr>
<tr>
<td>Affiliation</td>
<td>Openness</td>
</tr>
<tr>
<td></td>
<td>Agreeableness</td>
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</tbody>
</table>

A recent study surveyed over half a million North Americans and found that five-factor personality traits (as well as associated attitudes and behaviours) varied significantly depending on the region within the USA from which participants came (Rentfrow et al., 2008). Interestingly, state-level personality means were clustered, with nearby states having fairly similar scores of personality. More specifically, researchers found that Extraversion was higher in the central states, while Agreeableness was higher in the Midwest and Southern states. There appeared to be a ‘stress belt’ running from the central Southern regions of the USA, through to North-East USA. This is illustrated in figure 6. Interestingly, these differences in personality were related to clear differences in social indicators, such as crime, religiosity, values, health behaviour and mortality. For instance, states higher on Neuroticism were also more likely to have individuals who were less healthy and more socially withdrawn. Taken together, this research suggests that personality and temperament do vary across geographic areas and suggests that the wider environmental context is likely to play a significant role in the development of temperament.
Of course, it is difficult to disentangle cause and effect: social influence is likely to play a role, but so too is selective migration (similar in principle to downward drift), which refers to the idea that individuals high in a certain temperament dimension move to areas that are more accepting or encouraging of their personalities (Rentfrow et al., 2008). This theory of selective migration is supported by research indicating that people will move towards environments that support their attitudes, beliefs and personalities (Ickes, Snyder, & Garcia, 1997; McCrae, 2001). However, this effect is likely to be less applicable to adolescents, who have less control over the neighbourhood in which they reside.

One study has examined neighbourhood effects longitudinally to investigate whether neighbourhood may have a causal influence on temperament. This study used the same data as Hart and colleagues (2003) to examine whether personality change (in regard to resilience and overcontrol) in childhood was associated with neighbourhood economic disadvantage (Hart et al., 2008). Data were collected in two waves (3–4 years of age, and 5–6 years of age). Only children
remaining in the same neighbourhood during the two waves of data collection were included, and analyses controlled for family-level variables including maternal education, family income, and cognitive and emotional support in the home environment. It was found that, compared to children from affluent neighbourhoods, those living in economically disadvantaged neighbourhoods were more likely to demonstrate maladaptive personality change. While this effect was relatively small, children from poorer neighbourhoods decreased in resiliency (self-confident and high achieving) and overcontrol (associated with shy and anxious behaviour), and increased in problem behaviour. This effect was more pronounced in the highest poverty areas, with 44% of children from these areas experiencing maladaptive personality change. The decreased levels of resiliency and overcontrol found in children from poorer areas were then found to predict lower rates of academic achievement and higher levels of delinquency 6 years later. This study also looked for possible mediators between neighbourhood poverty and negative personality change, including maternal depression, child attendance at a Head Start program, change in quality of the home environment, and maternal trust in the neighbourhood. However, no evidence was found for any significant mediational relationships. Thus, the authors were unsure of the mechanisms through which neighbourhood poverty influenced temperament change. However, they predicted that stress associated with poor neighbourhoods is likely to be one of the major influences.

Many researchers have begun to examine regional personality differences in terms of differences in the ‘culture of honour’ (Cohen, 2001; Nisbett & Cohen, 1996), a term used to refer to the willingness of individuals to resort to violence in order to protect their reputation. Studies suggest that those from Southern U.S. states place much greater importance on personal reputation and respect than those from Northern states (Nisbett & Cohen, 1996). Thus, those from the South are more likely to react cognitively and physiologically with more anger and violence to dangerous situations or acts perceived as insults, leading to higher rates of aggression and homicide in the South. It is possible that disadvantaged neighbourhoods may develop a similar culture of honour (Cohen, 2001). This may happen for reasons associated with a poorer ability to access economic resources, and more harsh and dangerous conditions in these neighbourhoods, which may encourage theft and other illegal activities (Cohen, 2001). Aside from being more likely to take part in more aggressive and illegal activities, law-abiding citizens may have less trust in others and may need to be more vigilant and aggressive as a protective mechanism. This, in turn, may encourage certain temperament dimensions, such as low
frustration tolerance and low Effortful Control (as control of aggressive behavior is seen as less desirable).

Taken together, this evidence suggests that poor parenting, neighbourhood social influences and the experience of environmental stress during development can cause changes to temperament, including changes in temperaments related to Negative Affectivity, Surgency, Effortful Control and Affiliation. Based on this literature, it was expected that the adolescents in the current study would display differences in temperament depending upon the level of disadvantage in their neighbourhood. Specifically, it was predicted that adolescents from disadvantaged neighbourhoods would display differences in regard to several aspects of temperament: (1) they would be higher on Negative Affectivity (in line with research by Vaidya and colleagues (2002), Hart et al (2003), and Matheny and Phillips (2001)), (2) lower on Surgency (in line with Vaidya and colleagues (2002), and Hart et al (2003)), (3) lower on Effortful Control (in line with research by Halverson and Deal (2001) and Hart et al (2003)), and (4) lower Affiliation (in line with Rentfrow, Gosling & Potter (2008)).

5.3 Temperament and risk for internalising disorders

The previously discussed research suggests that neighbourhoods could alter the development of temperament. The following section reviews research that indicates a strong link between certain temperament dimensions and internalising disorders.

While *Surgency* is associated with externalizing disorders and substance abuse (Wills, Windle, & Cleary, 1998) it is generally found to be a protective factor in terms of internalising disorders. For instance, children who score low on measures of Surgency have been found to suffer significantly higher levels of depression in adolescence and adulthood (Clark, Watson, & Mineka, 1994; Forbes & Dahl, 2005). Various subcategories of Surgency, such as pleasure seeking and low-fear are also protective against depression (Clark et al., 1994). Surgency is thought to have a protective effect by making children more likely to form social connections, and more likely to demonstrate approach behaviours (Forbes & Dahl, 2005). This means they are less likely to withdraw (a hallmark of depression), and are more likely to have social supports.

Effortful control is also fairly consistently found to be a protective factor for adolescent internalising disorders (particularly depression) (Lengua, 2006). Those who are low on this temperament dimension are less able to control their impulses and emotions, and thus more
likely to engage in risky and antisocial behaviour. Low Effortful Control also reduces the ability for people to regulate their emotions, which then means that they are less able to regulate anxiety and depression, leading to a perpetuation and exacerbation of depressive and anxious symptoms.

There is a clear link between Negative Affectivity and internalising disorders. For instance, Ormel and colleagues (2005) studied 2230 preadolescents and found that Negative Affectivity predicted internalising (including symptoms of anxiety, depression and somatic complaints) and externalizing problems (aggression and rule-breaking behaviours) at 2- and 5-year follow up. While there is clear overlap between Negative Affectivity and internalising disorders, they are generally considered separable constructs on the basis that internalising disorders, but not Negative Affectivity, are associated with dysfunction and life interference as a result of symptoms (Pérez-Edgar & Fox, 2005; Rapee & Spence, 2004). Most empirical research also distinguishes Negative Affectivity from anxiety disorders and depression on a temporal basis. For instance, research has shown that children high on behavioural inhibition (similar to Negative Affectivity) during the first few years of their life are at significantly greater risk of suicide, depression and anxiety disorders later in life (Schwartz, Snidman, & Kagan, 1999).

Less is known about the association between internalising disorders and Affiliation. This is partly because many models include Affiliation with higher-order factors similar to Surgency. One longitudinal study examined Affiliation and mental health in adolescents (Ormel et al., 2005). Researchers found that high Affiliation predicted later internalising symptoms, and low Affiliation predicted later externalizing problems.

There are various theories as to why temperament is associated with increased risk for internalising disorders (Clark et al., 1994). Vulnerability models suggest that temperament plays a causal role in psychopathology by altering the risk of developing internalising disorders, while pathoplasty models suggest that temperament also affects the duration and severity of these disorders. Complication or scar models propose almost the opposite, by suggesting that internalising disorders alter temperament either temporarily or permanently. Spectrum models suggest that temperament and psychopathology are reflective of the same underlying processes, and that internalising disorders are a more dysfunctional and pronounced version of normal temperament dimensions. It is possible that all of these theories are valid to some extent, with varying processes working at different times. Temperament could predispose children and adolescents to internalising disorders, and influence their course and severity, and then be
influenced by the experience of psychopathology. At the very least, given the previously mentioned research, temperament could act as a marker for later psychopathology. Thus, in regard to the current thesis, it was predicted that if there were found to be differences in temperament as a function of neighbourhood disadvantage, these differences would also be associated with altered risk for depression or anxiety. Specifically, lower levels of Effortful Control and Surgency, and higher levels of Negative Affectivity would be associated with greater symptoms of anxiety and depression.

5.4 Summary and review

This chapter has reviewed evidence concerning the influence of environmental factors, including stress and neighbourhood, on adolescent temperament, as well as evidence concerning the influence of temperament on internalising disorders in adolescents. When considered together, these two bodies of literature suggest a possible causal chain in which neighbourhood disadvantage (including neighbourhood stress and social influence) influences temperament, which in turn influences the development of internalising disorders in adolescents. While several researchers have touched on this model (e.g. Hart et al., 2003), it has never been explicitly and empirically examined using formal statistical tests of mediation. Filling this gap in the literature is one of the central goals of this thesis, and the specific model, research questions and hypotheses under investigation will be detailed in Chapter 7.
CHAPTER 6

PARENT AFFECTIVE BEHAVIOUR AS A MEDIATING FACTOR BETWEEN NEIGHBOURHOOD DISADVANTAGE AND INTERNALISING DISORDERS

The following chapter summarises research surrounding the hypothesis that the effect of neighbourhood disadvantage on internalising disorders is partly mediated by the way in which parents express emotion during interactions with their children. That is, neighbourhoods could alter the way that parents express emotion, which in turn could alter risk for depression and anxiety in their children. Parental affective behaviour is discussed in section 6.1. The chapter then details evidence surrounding the hypothesis outlined above, including research suggesting that (1) neighbourhoods of differing levels of disadvantage are associated with differences in parental affective behaviour (section 6.2) and (2) that different parent affective behaviours alter risk for internalising disorders (section 6.3). Section 6.4 will then outline empirical research that has directly examined the in the relationship between neighbourhood disadvantage and internalising disorders in children and adolescents.

6.1 - Parent affective behaviour

Parenting quality is a broad concept that encompasses the wide variety of ways that parents interact with their children, and has been examined within a wide range of theories, including social support theory, social learning theory, interpersonal theory and attachment theory (Cowan & Cowan, 2002). The current thesis focuses on a specific aspect of parent quality - parental interaction style, which includes the behaviours that influence the emotional climate of the family (Cowan & Cowan, 2002). As will be discussed later, parent interaction style is thought to be an important risk factor for depression and anxiety in children and adolescents. The following section will provide a summary of the parent interaction styles examined in the current thesis.
One important form of parent interaction is the expression of positive and negative emotions, also termed affective behaviour. Affective behaviour style is thought to play a significant role in developing children’s socioemotional skills (Morris, Silk, Steinberg, Myers, & Robinson, 2007). These behaviours form a direct interface between adults and children, and influence children’s development through observation and modelling of parent’s behaviour. Through these processes children are likely to learn about different emotions, appropriate (or inappropriate) expression of emotion in response to various triggers, and the contextual appropriateness of emotion expression (Eisenberg, Cumberland, & Spinrad, 1998). Most studies examining parent affective behaviour tend to examine three broad types of emotion:

1. *Sad/dysphoric*, which includes expressions of anxious, pained, or whining affect, submissive behaviour, complaints and self-deprecating comments.
2. *Angry/aggressive*, which includes expressions of irritable affect, dominant or aversive behaviour, sarcastic, disapproving, disrespectful, or critical statements, commands, ridicule, or hostile tone and gestures.
3. *Positive/facilitative*, which encompasses happy, supportive, and warm affect, approving or positive statements about the self or others, playfulness, use of humour and smiling, and statements that maintain conversations.

As will be discussed later, these different forms of affective behaviour are thought to have different effects on children’s risk for psychopathology. The current thesis examines the relationship between adolescent internalising symptoms and observational measures of parental affective behaviour derived from micro-social coding of parental behaviour during laboratory based parent-child interactions. This behaviour will be examined in two different ways: (1) the frequency of aggressive, dysphoric and positive behaviour, and (2) the duration that parents tend to express these affective behaviours.

The frequency of expressed emotion refers to the number of times in a given period that parents express positive or negative emotion towards their children. Frequent expression of aggressive behaviour is associated with increased conflict with children and a harsher parenting style (Davis, Hops, Alpert, & Sheeber, 1998), while frequent expression of dysphoric behaviour is likely to be associated with needy, anxious and unsupportive parenting (Middleton, Scott, & Renk, 2009). On the other hand, frequent expression of positive behaviour is likely to be associated with
supportive and warm parenting (Sanders, 1999). Further details of frequency of parental affective behaviour and its impact on children will be discussed below in section 6.4.

The other aspect of parenting that was examined in the current study was the duration of affective behaviour, which refers to the average time that parents tend to spend in any given emotional state. A high duration of negative behaviour (such as aggression or dysphoria) is thought to reflect an inability to shift out of undesirable mood states, while a low duration of positive behaviour it thought to reflect an inability to stay in desirable mood states (Bariola, Gullone, & Hughes, 2011). Thus duration of affective behaviour is thought to be a measure of emotion and behaviour regulation (Bariola et al., 2011). While low-level aggression or dysphoria may at times be appropriate (such as when a child misbehaves or when a person has made an error) (Halberstadt, Crisp, & Eaton, 1999), an extended duration of these affective behaviours may suggest maladaptive behaviour.

Having summarised the aspects of parenting style assessed in the current thesis, the following section will outline research that suggests that neighbourhood disadvantage is associated with differences in parenting. Given there are a limited number of studies on neighbourhood and parenting, the following will review both parent interaction, and more broad research on parenting quality.

6.2 Neighbourhoods and differences in parenting

Disadvantaged neighbourhoods are associated with a variety of differences in parenting styles, and parent-child affective behaviour. The following section explores the main mechanisms through which neighbourhoods are thought to influence parenting, including higher stress, increased powerlessness and helplessness, and poor community support and resources. This is followed by a discussion of empirical research examining links between disadvantaged neighbourhoods and parenting quality.

6.2.1 High stress

As discussed in Chapter 4, disadvantaged neighbourhoods are more stressful for adolescents (Cutrona et al., 2006). They are also more stressful for parents, and the constant experience of stress is thought to wear down parents and decrease their ability to parent effectively (Cutrona,
Wallace, & Wesner, 2006). Daily experience of stress is conducive to anger outbursts, irritability, and rejecting and controlling behaviour towards children (Conger, Ge, Elder Jr, Lorenz, & Simons, 1994). Stressed parents also have less capacity to provide warm and supportive parenting to their children (Conger et al., 1994). Menaghan (1991) coined the term ‘mood spillover’ to describe the way in which community disorganization and distress are characteristic of the socio-cultural context of families in disadvantaged neighbourhoods. Perhaps because of the high stress experienced in disadvantaged neighbourhoods, parents living in these neighbourhoods are more likely to have poor mental health, inadequate coping skills, and lower efficacy (Klebanov et al., 1994; Leventhal & Brooks-Gunn, 2000). These factors are associated with a decreased ability to regulate emotions, which is likely to be reflected in high duration of negative mood states and low duration of positive mood states (Gross & Muñoz, 2006).

Neighbourhood stress and associated effects on parents are thought to create a home environment where few resources are available to adolescents and where parents may be more likely to use harsh controls and verbal aggression (Earls, McGuire, & Shay, 1994). Some researchers have also hypothesised that family conflict may represent a form of stress release for parents (Duncan, Strycker, Duncan, & Okut, 2002). The danger associated with disadvantaged neighbourhoods may also cause parents to be more controlling of their children (and thus generate family conflict) as parents attempt to restrict associations with delinquent peers, because of the serious harm that could result from such activities in high-risk neighbourhoods (Duncan et al., 2002). As will be discussed in section 6.4, it has been proposed that primary stressors in parents’ lives may breed secondary stressors in children's lives over time (Pearlin, 1999). These may be of the same type, thus setting off a transfer of stress experience across generations (Menaghan, Kowaleski-Jones, & Mott, 1997).

6.2.2 Powerlessness and helplessness

Some researchers propose that parents’ feelings of powerlessness are a primary mediator of stressors originating in the neighbourhood and parenting quality (Guterman, Lee, Taylor, & Rathouz, 2009). One study found that disadvantaged neighbourhoods only had a weak direct effect on risk for abusive behaviour in parents, and instead indirectly affected this risk through the mediating factors of parental stress and lack of self-control (Guterman et al., 2009). The researchers proposed that disadvantaged neighbourhoods might create feelings of learned helplessness and powerlessness, where parents lose a sense of self-control, feeling that they
cannot escape the aversive conditions in their neighbourhood. This may spiral into a reduced sense of control in their behaviour and in their parenting role, all of which have been shown to predict coercive and disengaged parenting (Bugental & Happaney, 2004; Bugental, Lyon, Lin, McGrath, & Bimbela, 1999). This reduced sense of control in behaviour may be reflected in a longer duration of negative affective behaviour, and shorter duration of positive behaviour, which presumably will be reflected in affective behaviour evident during parent child interactions.

6.2.3 Poor community support and neighbourhood resources

Families from disadvantaged neighbourhoods are more likely to have poor access to resources such as health care, retail stores, and recreational facilities (Wickrama & Bryant, 2004). In addition, disadvantaged neighbourhoods provide poor role models and often lack community social support. Inadequate formal and informal social relations, and poor social norms within neighbourhoods are likely to hinder effective parenting (Coleman, 1994). These factors are thought to leave parents lacking the resources to effectively parent (Conger & Donnellan, 2007), leading to poor child management in terms of a lack of involvement, supervision, and effective discipline (Sroufe, Duggal, Weinfield, & Carlson, 2000). There is empirical support for this hypothesis, with evidence that social network support is directly associated with positive parenting (Crnic & Greenberg, 1990; Hashima & Amato, 1994) and also indirectly through parental depression (Simons, Lorenz, Wu, & Conger, 1993). One study by Jennings and colleagues (1991) found that mothers who were more satisfied with their personal networks were more likely to praise their children and demonstrated less intrusive controlling behaviour. The lack of helping networks associated with disadvantaged neighbourhoods may also increase stress for parents, thus further perpetuating ineffective parenting (Leventhal & Brooks-Gunn, 2000).

6.2.4 Empirical research linking neighbourhoods and parenting styles

There is strong empirical evidence for differences in parenting style across neighbourhoods of differing levels of disadvantage. However, the majority of studies are cross-sectional, thus there is very little empirical evidence for a causal relationship between neighbourhood disadvantage and parenting style. The following findings come from cross-sectional studies.
Research has found correlations between neighbourhood disadvantage and various forms of poor parenting. These include studies revealing that parents who live in disadvantaged neighbourhoods are: (1) less likely to parent in a warm manner and more likely to engage in ineffective parenting practices than parents from less disadvantaged neighbourhoods (Simons et al., 1996); (2) less likely to interact with their children in a supportive, consistent, and involved manner (Downey & Coyne, 1990); (3) more likely to use harsh and unpredictable parenting (Pinderhughes et al., 2004); and (4) more likely to use more hostile and coercive parenting including physical punishment (Downey & Coyne, 1990). However, despite the apparent strong links between neighbourhood disadvantage and various maladaptive parenting practices, there are several lacunas in the literature.

First, there is no research examining neighbourhood disadvantage and parenting where parenting is measured using direct observation. Observational research is generally considered the most reliable and valid way to measure parent affective behaviour (Zeman, Klimes-Dougan, Cassano, & Adrian, 2007). It is more objective, and provides a more ‘natural’ assessment of behaviour than questionnaire data (Morris, Robinson, & Eisenberg, 2006). Observational measures also allow for behaviour to be measured that participants may not be consciously aware of, such as non-verbal behaviour (Bakeman & Gnisci, 2006). Finally observational measures are thought to be less biased by social desirability than questionnaire methods, as participants have less control over their behaviour during tasks such as video-taped assessments (Morris et al., 2006). The current thesis will improve upon the current literature by observing whether disadvantaged neighbourhoods are associated with differences in parent affective behaviour, as measured using direct observation.

There is also a lack of longitudinal research regarding neighbourhood disadvantage and parenting, which means that it is difficult to rule out reverse causation. For instance, parents who engage in ineffective parenting with their children may be more likely to move to more disadvantaged neighbourhoods for reasons similar to those discussed in chapter 2. However, as discussed previously, there is a strong theoretical basis for believing that disadvantaged neighbourhoods could causally influence poorer parenting practices. At the very least, children living in disadvantaged neighbourhoods are more likely to experience more negative interactions with their parents than children in less disadvantaged neighbourhoods, and, as will be discussed in the following section, this could put them at increased risk for internalising problems.
Finally, there is no research examining neighbourhood disadvantage and duration of affective behaviour. It is possible that, just as children from disadvantaged neighbourhoods demonstrate poorer emotional and behavioural regulation (Halverson & Deal, 2001), parents may also demonstrate poorer regulation, which would presumably be reflected in longer duration of negative affective behaviour, and shorter duration of positive behaviour.

Based on the previously discussed theories and empirical research, it was expected that parents from disadvantaged neighbourhoods would display greater frequency and duration of aggressive and dysphoric behaviour, and lower frequency and duration of positive behaviour towards their children.

6.4 Parenting style and risk for depression and anxiety in children

The following section will provide a summary of research into supportive versus harsh parenting and their influence on risk for depression and anxiety in children. It will then discuss research regarding parents’ expression of emotion and the way this is thought to influence children and adolescents.

6.4.1 Supportive versus harsh parenting and risk for depression and anxiety in children

There is considerable evidence suggesting that warm, supportive parenting is beneficial to adolescent development (Baumrind, 1991; Lamborn, Mounts, Steinberg, & Dornbusch, 1991), while hostile and unsupportive parenting has been linked to increased risk for depression and anxiety (Ge, Conger, Lorenz, & Simons, 1994). Adolescents who are exposed to warm and supportive parenting are likely to feel more cared for and loved, and are more likely to develop higher self-esteem (Ross, Mirowsky, & Goldsteen, 1990). Children with supportive parents are offered greater opportunity to explore interests and to challenge their limits in various settings (Perry, 2000). They are provided with a more stable family environment, consistent norms, social resources and cultural capital (Perry, 2000). All of these factors are likely to have a positive influence on adolescent mental health (Ross et al., 1990). On the other hand, uninvolved parenting and parental rejection are related to low self-esteem, school failure, poor mental health in adolescence, and a disrupted transition to adulthood (Conger et al., 1994). The relationship between parenting style and internalising disorders is likely to be causal, with parenting style predicting later symptoms of these disorders (Ge, Best, Conger, & Simons, 1996).
Of the large number of family context factors thought to influence risk for internalising disorders in adolescence, it is possible that parent affective behaviour and expression of emotion may act as a conduit for these risk factors. That is, outside pressures such as neighbourhood disadvantage and stress, parental depression, low SES, marital conflict, and quality of the parent-adolescent relationship (all of which are consistently found to be associated with risk for depression and anxiety in adolescents and children; Cowan & Cowan, 2002) may exert their influence on children through the affective behaviour of parents. Through interacting with their parents, children and adolescents learn how to respond to various stimuli in their environment, and find their own way to express emotions (Halberstadt, Cassidy, Stifter, Parke, & Fox, 1995). Parent affective behaviour influences children’s feelings about themselves, the world around them, and their future (Eisenberg et al., 1998). Through this, it is believed that parent affective behaviour influences risk for depression and anxiety in children. For instance, if a parent expresses consistent negative emotion about the world, their children may feel scared, sad or rejected, which is likely to increase their risk for internalising disorders.

As mentioned earlier, the current thesis examines three broad classes of emotional behaviour and expression: aggressive, dysphoric and positive. A predominance of each of these affective behaviours in parents is likely to lead to changes in risk for depression or anxiety in their children. For instance, one of the functions of aggression is to elicit fear and submission responses in others (Keltner & Kring, 1998). Thus, it could be reasoned that children who are exposed to aggressive parents could experience more fear and be more likely to develop symptoms of anxiety. Aggressive behaviour from parents could also initiate feelings of rejection, worthlessness or helplessness, all of which are associated with depression (Rodriguez, 2003). Dysphoric behaviour from parents could alter risk for anxiety and depression in their children through modelling or emotion contagion processes, whereby expressions of dysphoric emotion can elicit reciprocal dysphoric emotion in another individual (Campos, Mumme, Kermoian, & Campos, 1994). Dysphoric/submissive emotions also aim to elicit support and reassurance from others (Keltner & Kring, 1998). Therefore, parents who express dysphoria could leave children feeling unsupported, or may encourage these children to play support roles for parents, all of which is likely to increase risk for internalising symptoms (Jacobvitz, Hazen, Curran, & Hitchens, 2004). On the other hand, adolescents who are exposed to positive parental emotions are more likely to feel secure, loved and accepted, all of which are likely to play a protective role against
depression and anxiety (Blatt & Homann, 1992). The following section will review empirical evidence that explores these hypotheses regarding parental expression of emotion and internalising symptoms (first depression, then anxiety).

6.4.2 Frequency of parent expression of emotion and depression in children

Several studies have found direct links between adolescent depression and exposure to parents who display critical, conflictual and angry emotional behaviours, and low levels of supporting emotional behaviours (Rueter, Scaramella, Wallace, & Conger, 1999; Sheeber, Hops, & Davis, 2001). However, this research is limited in that it is based primarily on parents’ and children’s self-reports of parental expressivity (e.g., Halberstadt et al., 1995). As discussed previously, perhaps a better approach is to directly and independently observe parent-child interactions (Sheeber et al., 2001).

There are several studies that have used observational measures to assess the relationship between parental expressed emotion and depression in children, and have found that parent-child interactions predict symptoms of depression and anxiety in adolescents and children. For instance, (Sheeber & Sorensen, 1998) found that, in comparison to mothers of non-depressed adolescents, mothers of clinically depressed adolescents displayed more depressive behaviours and fewer facilitative behaviours (but not less aggressive behaviours) during a problem-solving task with their child. Another study, which observed a community sample of 20 children and their families, found that parents of children with clinically diagnosed depression displayed a greater number of negative behaviours and few positive behaviours in the home, in comparison to children with low levels of depressive symptoms (Messer & Gross, 1995). Sheeber and colleagues (2007) also found that depressed adolescents and adolescents with subthreshold depression were exposed to less warm or supportive and more aggressive parental behaviours, compared to healthy adolescents. As this research is cross-sectional, however, it is difficult to disentangle whether parental expression of emotion was influencing children to develop depression, or whether they were simply responding to their children’s own expression of negative emotion (Sheeber, Davis, Leve, Hops, & Tildesley, 2007).

This issue has been addressed in a longitudinal study in which a community sample of 14 to 20 year olds were asked to interact with their mothers in a problem solving task. Supportive and conflictual maternal behaviours were assessed using direct observation, and maternal and
adolescent reports. This was done at an initial assessment and at a follow up one year later (Sheeber, Hops, Alpert, Davis, & Andrews, 1997). A multi-method factor representing supportive maternal behaviours (comprising a combination of questionnaire and observational variables) predicted decreased levels of adolescent depressive symptoms at follow-up, even after accounting for baseline levels of symptoms at Time 1. In addition to this, it was also found that a multimethod factor representing conflictual parental behaviours at Time 1 predicted increasing levels of depressive symptoms at Time 2. The longitudinal design of this study provides stronger evidence that it is not simply depressive behaviour in adolescents that elicits aggression from parents, but that parental aggression and lack of support are likely to play a role for increasing risk for depression in adolescents. Another longitudinal study directly observed depressed and non-depressed mothers interacting with their children (mean age 12) during a problem solving task (Burge & Hammen, 1991). Maternal negativity and lack of involvement predicted higher levels of depressive symptoms in children six months later. However, this study did not include baseline levels of depression, thus it is not clear whether parenting effects would have had an independent influence on symptoms at follow-up after controlling for existing symptoms.

One pertinent longitudinal paper examined the influence of parent affective behaviour on change in depression and anxiety in 194 adolescents utilising the same sample used in the current thesis (Schwartz et al., 2011). This study observed the aggressive, dysphoric and positive behaviour of parents during parent-adolescent interactions. Researchers determined that higher levels of parental aggression prospectively predicted higher levels of depressive symptomatology in adolescents over a period of 2.5 years. Lower levels of positive behaviour from parents predicted higher levels of depression. Interestingly, parental dysphoric behaviour was not related to depression. This study was particularly powerful as it used direct observation of parent-child interaction and explored the effect of parental interaction on prospective measures of symptomatology. The longitudinal design of the study also allowed for a more thorough investigation of the directionality of influence. The multi-method approach to measurement also reduced the possibility of Type 1 error due to shared method variance. Whilst it is not possible to conclusively infer a causal relationship on the basis of this research, as other unmeasured variables may have accounted for the findings, this study provided strong evidence to support a causal relationship between parent affective behaviour (most notably parent aggression) and depressive symptoms in adolescents. The current study extends upon this research by exploring duration of affective behaviour as well as frequency of expressed emotion. I also aimed to explore the way in which neighbourhood disadvantage interacts with these variables.
In conclusion, longitudinal evidence suggests a relationship between low expression of positive behaviour and high expression of aggressive behaviour from parents, and risk for depression in children. As will be discussed below, no studies have found a significant relationship between parental expression of dysphoric behaviour and depression (Schwartz et al., 2011).

6.4.3 Frequency of parental expression of emotion and anxiety in children

Most research examining the influence of parent interaction on anxiety has focused on parental control, and parental expression of positive and negative emotion (Rapee, 1997). Both cross-sectional (Gar & Hudson, 2008; Hudson & Rapee, 2001) and longitudinal (Bayer, Hiscock, Ukoumunne, Price, & Wake, 2008) studies using observational designs have consistently found that children with anxiety are more likely to have parents who demonstrate high levels of controlling behaviours. There is less consistent evidence for the relationship between child internalising and parental expression of positive and negative behaviour and emotions. Some studies have found lower levels of warmth (Hudson and Rapee, 2001) and higher levels of criticism (Gar & Hudson, 2008) in the parents of anxious children. However, other studies have found no significant relationship (Siqueland, Kendall, & Steinberg, 1996). One study examined the effect of positive and negative parental behaviours, and found that boys with an anxiety disorder (but not girls) were exposed to more negative affect and less positive affect than boys without anxiety disorders (Suveg et al., 2008).

As has previously been discussed, longitudinal studies are able to provide a more solid investigation of the effect of parenting on psychopathology. These studies indicate that parental behaviours predict change in anxiety symptoms over time. For instance, two longitudinal studies found that self-reported controlling behaviour, low warmth, and negative emotional expressivity in parents was linked to symptoms of anxiety in children (Duchesne, Larose, Vitaro, & Tremblay, 2010; Volbrecht & Goldsmith, 2010). Another study observed parents of socially inhibited children and found that when mothers of inhibited children behaved in a controlling or derisive manner when children were aged two, their children were more likely to display more social restraint 2 years later (Rubin, Burgess, & Hastings, 2003). Another longitudinal research paper found that adolescents’ symptoms of anxiety increased when they rated their parent’s behaviour as overprotective, rejecting and anxious (Lieb, Wittchen, Hofler, & Fuetsch 2000).
The longitudinal study by Schwartz and colleagues described above also examined the influence of parent affective behaviour on change in anxiety in their sample of adolescents (Schwartz et al., 2011). Researchers determined that higher levels of parental aggression prospectively predicted higher levels of anxiety in adolescents over a period of 2.5 years. However, dysphoric and positive behaviour was not predictive of higher levels of anxiety.

In conclusion, it appears that controlling parental behaviours are predictive of anxiety in children (McLeod, Wood, & Weisz, 2007). In terms of parents’ expression of emotions, research suggests that negative parental behaviours (such as criticism and hostility) are more closely linked to anxiety in children than deficits in parents’ expression of positive emotion (McLeod et al., 2007).

### 6.4.4 Duration of affective behaviour and risk for depression and anxiety in children

Interestingly, there has been no research to date examining duration of emotional behaviour in parents and outcomes in children (Bariola et al., 2011). There is, however, some evidence that parents’ difficulty reverting back to a positive emotional state during family conflict is associated with poor social, behavioural, and emotional competence in children (Carson & Parke, 1996). There is also a strong theoretical basis for believing that parents’ longer duration of negative affective behaviour and shorter duration of positive behaviour could increase risk for depression and anxiety in their children. As mentioned, duration of affective behaviour is thought to be reflective of emotional and behavioural regulation. Thus parents who display longer negative behaviour and shorter positive behaviour may be less emotionally flexible and may respond less appropriately to environmental and psychological needs. This may mean they are less able to respond appropriately to the needs of their children (Kashdan & Rottenberg, 2010), leaving children feeling unsupported. Parents who demonstrate longer periods of aggressive behaviour may induce fear, helplessness and rejection in children, all of which are linked with depression and anxiety disorders (Rodriguez, 2003). A longer duration of dysphoric and passive behaviours may model depressive behaviour, and leave children feeling unsupported and uncared for (Bylsma, Morris, & Rottenberg, 2008), all of which could induce depressive feelings. Parents who are unable to regulate negative mood states may also fail to teach their children adequate emotion regulation strategies (Lindahl & Markman, 1990; Lindahl, 1998), which are associated with risk for depression and anxiety (Gross & Muñoz, 2006). Thus parents who display high duration of aggression and dysphoria may be more likely to have children with higher levels of internalising symptoms. On the other hand, longer periods of positive emotion could create a
more positive environment for children, thus acting as a protective factor for depression and anxiety. Having summarised the ways in which parent affective behaviour may influence risk for internalising disorders in children, the following section will review studies that have directly examined parenting as a potential mediator between neighbourhood disadvantage and these outcomes.

6.5 Parenting quality and affective behaviour as a mediator between neighbourhood disadvantage and internalising symptoms

Parenting style is theorised to be one of the most significant mediating factors between neighbourhood disadvantage and negative outcomes in children and adolescents (Bronfenbrenner & Morris, 1998), with research indicating it mediates the relationship between neighbourhood disadvantage and a variety of outcomes including drug involvement (Brook, Brook, & Rosa, 2001), teenage pregnancy (Hogan & Kitagawa, 1985), child behaviour problems (Kohen, Leventhal, Dahinten, & McIntosh, 2008), and adolescent externalizing behaviours (Stern & Smith, 1995). For instance, Schonberg and Shaw (2007) determined that boys from poorer neighborhoods were exposed to more familial risk factors such as maternal rejection, maternal depressive symptoms, and maternal views on physical discipline. This in turn was found to be associated with a greater number of conduct problems in boys (Schonberg & Shaw, 2007).

Throughout childhood, parents generally play the primary role in supervising children and affecting change in their environment. As such, parents largely control the activities their children participate in, both in the home and in the surrounding neighbourhood (Kim, Hetherington, & Reiss, 1999). Thus many researchers argue that neighbourhoods influence children’s development through family processes such as parents’ affective behaviour or the learning environment in the home (Kim et al., 1999; Klebanov et al., 1994). As the following section will outline, it is not clear whether parenting style continues to mediate the relationship between neighbourhood disadvantage and internalising disorders throughout adolescence. In fact only a handful of studies (most cross-sectional) have examined this relationship, with the bulk of studies suggesting that aggressive interactions play an important mediating role, while there is mixed evidence regarding positive interactions (such as family cohesion or parental warmth).

One study examined several different aspects of the family environment as potential mediators between neighbourhood disadvantage and internalising disorders (Deng et al., 2006). Researchers investigated the mediating role of mother- and child-reported family cohesion (a composite of
factors such as family support) and parent-child conflict in 189 low-income, European and Mexican American children and their mothers. It was found that families from disadvantaged neighbourhoods (measured using census data) were more likely to have low family cohesion between family members. They posited that neighbourhood disadvantage worsened the mutual trust and closeness between neighbours and among families. This lack of family cohesion was found to be associated with child internalising disorders in girls but not boys. Researchers also found that mother-reported, but not child-reported parent-child conflict significantly mediated the relationship between mother’s perceptions of their neighbourhood quality, and child internalising disorders. The differences in findings between child and parent report were thought to be due to children’s lower sensitivity to conflict, as they reported lower levels of conflict than mothers. This study was somewhat limited as it was cross-sectional, and the sample was of low-income population, and findings may not be generalizable to higher-income populations.

A similar study examined family cohesion, paternal warmth, perceived neighbourhood danger and youth internalising disorders in 463 Mexican Americans (White & Roosa, 2012). This study improved upon the previously discussed study by ascertaining measures of internalising symptoms from different reports (and thus minimizing the impact of shared method variance). Researchers found that family cohesion mediated the relationship between fathers’ perceptions that their neighbourhoods were dangerous places to live and children’s internalising symptoms. Neighbourhoods that were perceived as more dangerous were associated with lower levels of family cohesion. Low family cohesion, in turn, was associated with higher levels of internalising symptoms in children. This was found to be the case for both boys and girls, and remained significant after controlling for family income. However, paternal warmth did not mediate the association between fathers’ perceived danger and adolescent internalising symptoms. This study was limited in that it was cross-sectional, and did not provide independent measures of neighbourhoods.

A much larger study examined neighbourhood and family resources, and internalising disorders in adolescents (Wickrama & Bryant, 2004). Using questionnaire data from a community sample of 14,500 adolescents and parents, researchers found that disadvantaged neighbourhoods were associated with more hostile or harsh parenting. A pathway was found from neighbourhood disadvantage to adolescent depression, in which neighbourhood disadvantage reduced community social resources, where adults from the community were less likely to exhibit accepting and involved behaviour with their children. Conversely, parents who lived in non-
disadvantaged neighbourhoods were more likely to exhibit these behaviours, which decreased risk for depression and anxiety in children. Wickrama and Bryant proposed that neighbourhood disadvantage influences adolescent mental health both directly and indirectly through the availability of social resources. This study was limited in that it used only cross sectional data, bringing issues of causality. Reports of social resources were also measured subjectively, and thus could have been biased.

The only longitudinal study to examine parenting as a mediator examined 14,000 adolescents to see whether childhood community and family environments exert long-term influences on risk for depression in young adulthood (Wickrama & Noh, 2010). No direct effects of childhood neighbourhood disadvantage were found for depression in young adulthood. However, ineffective parenting (parental reports on uninvolved parenting and adolescents’ report on parental rejection) was directly related to depression in adulthood. Ineffective parenting also mediated the association between childhood neighbourhood disadvantage and depression in young adults. This study provides prospective evidence for a link between neighbourhood disadvantage, parenting and depression. Unfortunately it only used a 5-question parent questionnaire to measure neighbourhood quality, and as with other studies, did not provide independent measures of parenting. It also did not examine anxiety as an outcome variable.

Not all studies have found a mediating role for parenting in the relationship between neighbourhood disadvantage and outcomes in children and adolescents. For instance, one study of 1217 Mexican-American families found that parental warmth mediated the relationship between a variety of measures of neighbourhood disadvantage and externalizing disorders, but not internalising disorders (Gonzales et al., 2011). Researchers hypothesised that the timing of data collection might not have been ideal for detecting such effects. Internalising symptoms were found to decrease during the study time frame (particularly the anxiety symptoms that are more common during childhood). Other internalising symptoms, particularly depressive symptoms, begin to increase later in adolescence (Lewinsohn, Clarke, Seeley, & Rohde, 1994), outside of the timeframe with which the study could detect effects of parenting in the sample.

Interestingly, some studies suggest that harsher parenting could be beneficial in a disadvantaged neighbourhood. Qualitative parenting has suggested that harsher parenting could help to protect a child from dangers in the neighbourhood, which may be particularly helpful when positive role models in the neighbourhood are lacking (Baumrind, 1972; Furstenberg, 1993). One study found
that punitive parenting (associated with neighbourhood disadvantage) was related to greater behavioural problems in children, but was also associated with higher verbal ability scores (Kohen et al., 2008). In line with this, Beyes and colleagues (2003) found that heightened control may be protective for externalizing problems in risky neighbourhoods (Beyers, Bates, Pettit, & Dodge, 2003). Another study found that restrictive parenting had different impacts depending on neighbourhood, ethnic background (African American vs. European American), and outcome examined (Dearing, 2004). For instance, in African Americans, parenting behaviours that were restrictive were more protective in high-risk neighbourhoods (Dearing, 2004). Taken together, these studies indicate that a more controlling parenting style could be beneficial in disadvantaged neighbourhoods. However, the bulk of the research in this area suggests that warmer and more supportive parenting practises are more beneficial for children, regardless of the neighbourhood they come from.

In summary, the majority of research suggests that family conflict and parental aggression mediate the relationship between neighbourhood disadvantage and internalising symptoms. There are mixed findings regarding positive parenting as a mediator between neighbourhood disadvantage and internalising symptoms in adolescents, and no research has examined the possible mediating role of dysphoric behaviour in this relationship. The current study aims to improve upon the previously discussed research in several ways. Given the lack of longitudinal studies, it is not clear whether parenting mediates the relationship between neighbourhood disadvantage and internalising disorders only in children, or whether this mediating influence continues throughout adolescence. The longitudinal nature of the current study will allow for the investigation of whether parent interaction represents a mediating risk factor for depression and anxiety at early adolescence (approximately age 12 years), and whether parenting continues to mediate the relationship between neighbourhood disadvantage and these outcomes throughout adolescence. Many of the studies of mediating relationship described in this chapter do not use specific mediational analyses to examine relationships. Thus the current study’s use of bootstrapping will provide a more stringent test of mediating relationships. Other benefits of the current study include those discussed previously, such as the use of direct observation of parent behaviour and its exploration of duration of emotional behaviour.

### 6.6 Summary and review
This chapter has reviewed evidence concerning the influence of neighbourhood disadvantage on parenting style, as well as evidence for the relationship between parenting and internalising disorders in adolescents. When considered together, these two bodies of literature suggest a possible causal chain in which neighbourhood disadvantage influences parenting style, which in turn influences the development of internalising disorders in adolescents. There is very little research examining this model using mediational analyses. Filling this gap in the literature is one of the central goals of this thesis, and the specific model, research questions and hypotheses under investigation will be detailed in Chapter 7.
CHAPTER 7

HYPOTHESES FOR THE CURRENT THESIS

This chapter will outline the specific aims, hypotheses and the theoretical model under examination in the current thesis. In particular a partial mediation model will be proposed, in which three broad factors are predicted to partially mediate the relationship between neighbourhood disadvantage and internalising symptoms in adolescents. The rationale for this model, based on the literature reviewed in the previous chapters, will be discussed in section 7.1. The specific research aims and hypotheses under investigation in this thesis will be presented in sections 7.2 and 7.3 respectively.

7.1 Summary of the Literature review

The preceding introductory chapters have reviewed the key constructs under examination in this thesis: neighbourhood disadvantage, adolescent depression and anxiety, stress, temperament and parenting quality.

Chapter 1 highlighted the significant prevalence and morbidity associated with depression and anxiety, and noted the way in which risk factors for internalising disorders include a range of individual, proximal and distal factors that are generally thought to be complex and interconnected. It was argued that understanding the interrelationships between these factors could allow for the development of more effective programs aimed at treating and preventing the development of depression and anxiety in adolescents.

Chapter 2 reviewed literature surrounding the influence of neighbourhood disadvantage on depression and anxiety in adolescents, presenting evidence to suggest that neighbourhood effects may be strongest in adolescents in comparison to children and adolescents. The chapter also discussed the ways in which neighbourhoods have been conceptualised (including objective versus subjective measures of neighbourhood disadvantage), and outlined some of the complications of neighbourhood research including the problems of reverse causation and residual confounding. This chapter also highlighted the various limitations of the current state of research including a relative lack of longitudinal research, and few studies of neighbourhood
effects in adolescents (particularly Australian adolescents). The current thesis aims to overcome some of these shortcomings. While it appears likely that neighbourhoods have an important influence on mental health, it is not known exactly how this occurs. A mediational analysis is one way to investigate this issue.

Chapter 3 argued for the importance of research into the factors that mediate the relationship between neighbourhood disadvantage and internalising disorders. It also provided a summary of the current theories regarding mediational analysis.

Chapter 4 reviewed research addressing the possibility that stress acts as a mediator between neighbourhood disadvantage and internalising disorders in adolescents. There exists a large body of research demonstrating a strong link between neighbourhood disadvantage and increased stressful life events. There is also very strong evidence prospectively linking stress and internalising disorders in adolescents. Despite this, however, research examining stress as a mediator between neighbourhood disadvantage and internalising disorders has produced mixed findings, which may be due to the populations studied and methodologies used.

Chapter 5 reviewed research suggesting that adolescent temperament might act as a mediator between neighbourhood disadvantage and internalising symptoms in adolescents. While temperament is generally viewed as relatively stable throughout life, some evidence suggests that it is influenced by environmental factors such as social factors within neighbourhoods, stress and parenting. There is also strong evidence that temperament factors (including Surgency, Effortful Control, Negative Affectivity and Affiliation) are associated with risk for depression and anxiety. The current thesis provides the first direct investigation into whether temperament plays a mediating role between neighbourhood disadvantage and internalising symptoms in adolescents.

Chapter 6 reviewed research regarding the possibility that parenting quality acts as a mediator between neighbourhood disadvantage and internalising symptoms in adolescents. Disadvantaged neighbourhoods were shown to be linked to poorer parenting practices, and it was argued that neighbourhood disadvantage would be associated with increased frequency of expressed negative emotion and decreased frequency of positive emotion. It was also argued that these neighbourhoods would be associated with parents who expressed emotions for longer periods, reflecting a possible inability to regulate emotions and respond appropriately to their children. Research also suggests that the way parents express emotion (particularly aggressive and positive
emotion) has a significant impact on risk for internalising symptoms in their children. The chapter then reviewed research suggesting that parenting quality mediates the relationship between neighbourhood disadvantage and internalising disorders in children and adolescents. However, it was noted there is only one study to date examining this relationship longitudinally, and no studies that use direct observation of parenting style. The current thesis aimed to improve upon this by examining whether objectively measured and directly observed parent interactions mediated the relationship between objective measures of neighbourhood disadvantage and internalising symptoms in adolescents.

7.2 Research questions

A number of significant gaps in the literature have been identified in the previous chapters. In order to address some of these limitations, four key research questions were identified in this thesis:

1. Are adolescents from disadvantaged neighbourhoods more likely to suffer from greater symptoms of depression and anxiety?
2. Do adolescents from disadvantaged neighbourhoods experience greater increase (or less reduction) in their depressive and anxious symptomatology across adolescence?
3. Are disadvantaged neighbourhoods associated with differences in adolescent temperament (Surgency, Effortful Control, Negative Affectivity and Affiliation), maternal affective behaviour (aggressive, dysphoric and positive), and the frequency of stressful life events experienced by adolescents?
4. Do the variables mentioned in research question 3 mediate the relationship between neighbourhood disadvantage and symptoms of anxiety and depression in adolescents at baseline, and change in symptoms between baseline and follow-up?

7.3 Hypotheses

Based on the existing literature, nine specific hypotheses were posited in this thesis. Six hypotheses explored the direct relationship between neighbourhood disadvantage (the independent variable) and several key dependent variables: symptoms of depression and anxiety, stressful life events, adolescent temperament and parenting style. Analyses exploring these hypotheses are discussed in Chapter 10. The final three hypotheses relate to the three proposed mediators between neighbourhood disadvantage and internalising symptoms. Overall, an indirect
model is proposed in which stressful life events, parent’s affective behaviour, and adolescent temperament mediate the relationship between neighbourhood disadvantage and symptoms of depression and anxiety (represented in figure 7). All hypotheses relating to indirect pathways apply to the prediction of both baseline internalising symptoms, and change in symptoms between baseline and follow-up.

Hypotheses were defined as follows:

7.3.1 Internalising symptoms

Hypotenosis 1 (internalising symptoms): It was predicted that neighbourhood disadvantage would be associated with greater internalising symptoms at baseline, and a greater increase, or a lesser decrease, in symptoms of depression and anxiety between baseline and follow up.

7.3.2 Stress

Hypotenosis 2: It was predicted that adolescents from disadvantaged neighbourhoods would experience a greater number of stressful life events.

Hypotenosis 3: Stressful life events were predicted to partially mediate the relationship between neighbourhood disadvantage and internalising symptoms in adolescents. Specifically, neighbourhood disadvantage was hypothesised to be associated with increased stressful life
events, which would in turn be associated with increased symptoms of anxiety and depression in adolescents.

*Hypothesis 4:* It was predicted that boys from disadvantaged neighbourhoods would experience a greater number of stressful life events than girls. That is, there would be an interaction between neighbourhood and gender, whereby boys would be more exposed to stress as a result of neighbourhood disadvantage.

### 7.3.3 Temperament

*Hypothesis 5:* It was predicted that adolescents from disadvantaged neighbourhoods would score lower on measures of Effortful Control, Affiliation, and Surgency, and higher on measures of Negative Affectivity.

*Hypothesis 6:* Adolescent temperament (Surgency, Negative Affectivity and Effortful Control) was predicted to partially mediate the relationship between neighbourhood disadvantage and internalising symptoms in adolescents. Specifically, disadvantaged neighbourhoods were predicted to be associated with decreased Effortful Control, Affiliation and Surgency, and increased Negative Affectivity, which would in turn be associated with increased symptoms of depression and anxiety in adolescents.

### 7.3.4 Parent affective behaviour

*Hypothesis 7:* It was predicted that mothers from disadvantaged neighbourhoods would display a higher frequency of aggressive and dysphoric emotion, and a lower frequency of positive emotion.

*Hypothesis 8:* It was predicted that parents from disadvantaged neighbourhoods would display a longer duration of aggressive and dysphoric behaviour and shorter duration of positive behaviour.

*Hypothesis 9:* Frequency and duration of parental aggressive behaviour were predicted to partially mediate the relationship between neighbourhood disadvantage and internalising symptoms in
adolescents. Specifically, disadvantaged neighbourhoods were predicted to be associated with increased parental aggression, which in turn would be associated with increased symptoms of depression and anxiety in adolescents. It was predicted that frequency and duration of positive behaviour would mediate the relationship between neighbourhood disadvantage and internalising symptoms in adolescents. Specifically, it was predicted that disadvantaged neighbourhoods would be associated with lower frequency and shorter duration of positive emotion, and that this would lead to increased symptoms of anxiety and depression in adolescents. We also explored whether frequency and duration of parental dysphoria mediated the relationship between neighbourhood disadvantage and internalising symptoms.

Analyses examining each of these hypotheses will be discussed in Chapter 11 (Stressful life events), Chapter 12 (Temperament), and Chapter 13 (Parenting). The following chapter will discuss the methodology used in the current thesis.
CHAPTER 8

METHOD: PARTICIPANTS, MEASURES AND PROCEDURES

This chapter will detail the methodology used to address the hypotheses outlined in Chapter 7. Section 8.1 will summarise the procedure used to recruit and screen participants, including the make-up of the sample for each of the three waves of data collection. The study procedure will be discussed in section 8.2. Section 8.3 will outline the measures used for each data set, including those used to assess neighbourhood disadvantage, symptoms of depression and anxiety, stressful life events, family SES, parental affective behaviour, and adolescent temperament.

8.1 Data collection waves

The current thesis has used data collected in a large ongoing longitudinal study titled the ORYGEN Adolescent Development Study (ADS). The overall aims of this study were to assess risk and resilience factors for the development of mental illness in mid- to late-adolescence, through a longitudinal design beginning in early adolescence. The research discussed in this thesis draws on data collected during the first, second and third wave of the ADS. The waves included (1) Screening assessment - An initial screening for recruitment of participants to the study and collection of postcode data; (2) Time 1 (T1) – The first wave of intensive data collection. Measures taken include postcode, family SES, temperament, family interactions, and baseline psychopathology; (3) Time 2 (T2) – This wave occurred approximately two and a half years (2.36±0.27 years) after T1, and included the stressful life events questionnaire and interview; and (4) Time 3 (T3) – This wave occurred approximately four years (3.84±0.52 years) after wave 1 and included measures of psychopathology.

8.2 Study procedure

8.2.1 Recruitment and screening of participants

A sample of 2,479 Australian grade six students from 95 schools in metropolitan Melbourne in the state of Victoria were included in the screening stage. This sample was taken from a target
population of grade six students (most aged 11-12 years old) enrolled in primary schools in metropolitan Melbourne.

Primary schools were randomly selected with a probability proportional to the number of students enrolled in each school. An initial 175 schools were selected, providing a sample of 4587 students. The schools were a mix of Government (65%), Catholic (22.5%), and Independent Private (12.5%) schools. Of these schools, 97 (56%) agreed to participate in the study. Students who participated were provided with study information sheets and consent forms. Parental and student consent was provided for 2,453 students (53.5% of the total sampling population), comprising 52% female with a mean age of 11.62 years (SD = 0.30, range 10.24-13.15 years), with 1739 (70%), 507 (21%) and 233 (9%) from the Government, Catholic and Private Independent schools respectively. The proportions in the sampling population within each school sector were not significantly different from those found in the total intended sampling area ($\chi^2(2) = 0.81, p>0.05$). These students completed the screening measures.

The screening process involved the administration of the Early Adolescent Temperament Questionnaire (EATQ-R; Ellis & Rothbart, 2001) (detailed description below) and a brief demographic and contact information form (including postcode data used in the calculation of neighbourhood disadvantage). Study staff based at the ORYGEN Youth Health Research Centre administered the screening questionnaire to students in the classroom, and those not present were sent questionnaires to be completed at home and returned by mail. In order to maintain confidentiality, all participants were provided with ID numbers, which were used to identify their responses to the questionnaire.

The aim of the screening procedure was to ascertain a sample of adolescents who represented a range of scores across the EATQ-R temperament dimensions. Thus, equal numbers of adolescents were recruited across the following ranges of scores on the four higher order factors of the EATQ-R: 0-1 standard deviations (SD) above and below the mean, 1-2 SD above and below the mean, 2-2.5 SD above and below the mean, and greater than 2.5 SD above and below the mean. This created a sample containing even variation across each of the higher order traits of interest, with an emphasis in the distribution at the tails. This was done such that nearly all of the adolescents scoring in the extreme ranges of the four temperaments were recruited. An example of the distribution of Negative Affectivity scores for selected participants is shown below in figure 10.
Figure 8: Distribution of Negative Affectivity scores. The top image shows the frequency of scores for the broader screening sample. The bottom image shows the frequency of scores for the selected sample. Note the relatively flat distribution of Negative Affectivity scores for the selected sample.

The sampling and screening procedure resulted in the selection of 425 students who were invited to participate in the intensive assessment phase of the study. Their families were contacted by telephone and invited to an information meeting regarding the project. The initial interview with the adolescent was generally completed during the same meeting. A total of 245 of these families agreed to participate. This sample, made up of 50.6% females, is referred to in this thesis as the ADS sample. This is the sample used in the current thesis.

Given that the aim of the ADS study was to examine the development of affective mental illness in a prospective manner, adolescents were excluded from the study if they showed signs of any past or present axis I diagnosis of depressive disorder, substance use disorder, or eating disorder. In order to assess for these disorders, adolescents were administered the Schedule for Affective Disorder and Schizophrenia for School-Aged Children Epidemiological Version (K-SADS-E; Orvaschel & Puig-Antich, 1987) during an initial telephone interview.
The ADS sample were asked to take part in a variety of assessments during the baseline data collection period, and during two proceeding waves of data collection, including several home assessments, and the family interaction task. Figure 11 outlines the number of participants at each stage. Tests were run to determine whether there were any differences in temperament, clinical symptoms, neighbourhood disadvantage, stressful life events, or maternal affective behaviours between participants who consented to the follow-up assessments and participants who declined. No significant differences were found for participants who agreed to participate in the T1 family assessment in terms of clinical symptoms (all $p >0.05$), neighbourhood disadvantage (all $p >0.05$), affective temperament (all $p >0.05$), or stressful life events (all $p >0.05$). No significant differences were found for participants who agreed to participate in the T2 Stressful Life events assessment in terms of clinical symptoms at T3 and anxious symptoms at T1 (all $p >0.05$), neighbourhood disadvantage (all $p >0.05$), affective temperament (all $p >0.05$). However, there were significant differences in terms of depressive symptoms at T3 ($p <0.05$). There were no significant differences found for participants who agreed to participate in the T3 assessment (measuring clinical symptoms) in regard to clinical symptoms at T1 and anxious symptoms at T3 (all $p >0.05$), affective temperament (all $p >0.05$), stressful life events (all $p >0.05$), or neighbourhood disadvantage (all $p >0.05$). There were systematic differences in terms of depressive symptoms at T1 ($p >0.05$) and four of the twelve maternal affective behaviours ($p >0.05$). Systematic drop out of participants is expected to happen occasionally in long-term studies. For instance, adolescents who are more depressed or who have more aggressive mothers are likely to be more dysfunctional, and thus less willing or able to take part in ongoing assessments. Chapter 9.2.6 outlines the way these differences were handled using the Expectation Maximization technique. The following sections outline the various assessments undertaken during the three relevant data collection periods.
8.2.2 Baseline assessment

The baseline assessments included two relevant projects: a home assessment and family assessment.

The home assessment included the collection of data relating to affective temperament and clinical symptoms. 245 Participants (including one parent or guardian and child) were explained the procedure for the assessment, and written consent was obtained from the parent and the child (See Appendix B1 for an example of these forms). They were then administered a number
of questionnaires including the EATQ – R, CESD and the BAI. They were asked to return these questionnaires by mail. When questionnaires were completed, participants were reimbursed with $50 cash and a $30 voucher. Families completed the home assessment an average of 9.91 months (SD = 3.10) after the adolescents had taken part in the school screening.

The family assessment involved the collection of observational data regarding family interaction style. Families of adolescents who comprised the ADS sample were contacted as soon as they completed the home assessment, with a mean number of 1.97 months (SD = 1.44) between home and family assessments. 197 families (82% of the ADS sample) agreed to take part in the family assessment phase, which involved one parent or guardian (80% mothers and .01% guardians) and the target child.

Family assessments were conducted at ORYGEN Youth Health, a joint clinical and research facility. The procedure for the assessment was explained to the participants, and written consent was obtained from the parent and the child (See Appendix B2 for an example of these forms). A number of interview measures were administered and audio-taped for coding purposes, including the Issues Checklist (IC) and Pleasant Events Checklist (PEC), which were administered to both parents and their children. Participants were taken to an interaction room with two mounted video cameras, set up to be remote-controllable from a separate room where the interviewer was out of sight of participants. In order to ensure the recordings were of high enough quality for the observational coding, the vision from these video cameras was monitored by the interviewer throughout the tasks.

There were two tasks that participants engaged in: an Event-Planning Interaction (EPI), and a Problem-Solving Interaction (PSI). The EPI is designed to encourage positive behaviours between participants, while the PSI is designed to encourage conflictual behaviour. Participants engaged in the EPI followed by the PSI. This order was chosen as it was thought that participants would be more easily able to switch from a positive affective state (elicited by the EPI) to the conflictual affective state (elicited by the PSI), given that positive affective states have a faster decay than conflictual states (Gilboa, Revelle, Van Goozen, Van de Poll, & Sergeant, 1994). In the PEC conducted earlier, children and parents were asked to indicate several activities that they found very pleasant, while the IC interview asked them to note issues that they found most conflictual. The interviewer selected up to five of the activities found very pleasant by both child and parent, and up to five issues that were reported as most conflictual.
Directions were given to the participants in turn, just prior to the start of each task, and the selected activities or issues were verified with the participants as relevant and/or suitable before the task. Participants were told to start with the most desirable event (for the EPI) or the most conflictual issue (for the PSI) and only move on the next topic if and when the previous topic was exhausted or resolved. They were encouraged to remain engaged in the interaction task for the full twenty minutes, after which the interviewer would enter the room, indicate the end of the task and stop the recording.

After completing both the interaction tasks, participants were debriefed and encouraged to voice any concerns they had regarding the assessment session. Concerns were addressed by the interviewer, who referred participants to relevant individuals or services as required. If required, the interviewer conducted individual debriefing sessions with participants, after the general debriefing. Participants were also given a debriefing letter, including suggestions of people to contact if needed, including the contact details of the project investigators. Participants were provided with a summary of the aims of the project and information regarding the use of data. They were then given a $50 cash and $30 gift voucher, and thanked for their participation.

8.2.3 Follow-up assessments

The following information relates to both follow-up assessments, while information relevant to specific assessments is provided in the following two paragraphs. Participants were contacted regularly between baseline and follow-up assessments with regular newsletters and season’s greetings and birthday cards. This was done partly to reduce attrition during the period between baseline and the follow-up assessment. Prior to each assessment, families were informed of the forthcoming wave of assessment via telephone. They were invited to participate, and families that consented were then telephoned by a researcher in order to organise an assessment time. The follow-up assessments were similar in structure to the home assessment conducted at baseline. Several assessments were conducted by telephone, and questionnaires were sent by post to families if they had moved interstate or overseas. After consent procedures were complete (See Appendix B3 for an example of forms given to participants), where confidentiality was assured, participants were thanked, debriefed and reimbursed with $50 cash and a $50 voucher. Information and referrals to relevant contacts were provided where the interviewer believed that the adolescent was at risk of harm.
8.2.4.1 Assessment of stressful life events (T2)

The first follow-up assessment took place approximately 2.5 years after baseline home assessment, when the average age of participants was 15.04 years (SD = 0.45 years). Of the 245 initial participants, 214 families (87%) agreed to participate in the T2 assessment. The Stressful Life Events questionnaire and interview were completed during a home assessment in accord with the procedure outlined in section 8.3.4.

8.2.4.2 Assessment of symptoms of depression and anxiety during later adolescence (T3)

The second follow-up assessment took place approximately 4 years after the baseline home assessment, when the average age of participants was 16.64 years (SD = 0.60 years). Of the initial 245 participants, 181 families (74%) agreed to participate. During the T3 home assessment, adolescents were given the CESD-R and the BAI questionnaires to assess current emotional functioning.

8.3 Measures

8.3.1 Neighbourhood Disadvantage

The level of relative disadvantage of participants’ neighbourhoods was assessed by combining Postal Area data collected during the first wave of assessment with a measure of disadvantage called the Socio-Economic Indexes For Areas (SEIFA) developed by the Australian Bureau of Statistics (Pink, 2006).

The specific SEIFA measure used in the current study was the Index of Relative Socio-Economic Advantage and Disadvantage, which was developed with the aim of encompassing the entire socio-economic spectrum of each neighbourhood area (postcode). The Index of Relative Socio-economic Advantage and Disadvantage was chosen from several indexes created by the ABS. This index was selected as it is the most comprehensive and can be used to measure socio-economic wellbeing in a continuum, from the most disadvantaged areas to the most advantaged areas. In addition this index is more appropriate for research aiming to compare the entire range of areas, rather than focusing on relatively disadvantaged areas only (Pink, 2006). SEIFA indexes are assigned to areas, not to individuals, and indicate the collective socio-economic status of the people living in an area. These indexes are derived from a weighted composite of 31 different factors...
variables, including those related to income, education, employment, occupation, housing and other indicators of relative advantage and disadvantage (see Table 3 for a full list of the 21 variables included in the Index of Relative Advantage and Disadvantage). Variables were chosen from a larger set of variables assessed during census periods, and their inclusion was guided by international research, which indicates that these chosen variables are important indicators of socio-economic disadvantage and advantage (e.g., Bailey et al., 2003; Walker & Hiller, 2007).

Table 3
*Included Variables in the Index of Relative Advantage and Disadvantage (Pink, 2006)*

**Percentage in each postcode:**

<table>
<thead>
<tr>
<th>%</th>
<th>People aged 15 years and over with no post-school qualifications</th>
</tr>
</thead>
<tbody>
<tr>
<td>%</td>
<td>Occupied private dwellings with no internet connection</td>
</tr>
<tr>
<td>%</td>
<td>People with stated annual household equivalised income between $13,000 and $20,799 (approx. 2nd and 3rd deciles)</td>
</tr>
<tr>
<td>%</td>
<td>Employed people classified as Labourers</td>
</tr>
<tr>
<td>%</td>
<td>Households paying rent less than $120 per week (excluding $0 per week)</td>
</tr>
<tr>
<td>%</td>
<td>People aged under 70 who have a long-term health condition or disability and need assistance with core activities</td>
</tr>
<tr>
<td>%</td>
<td>Employed people classified as Machinery Operators and Drivers</td>
</tr>
<tr>
<td>%</td>
<td>People (in the labour force) unemployed</td>
</tr>
<tr>
<td>%</td>
<td>One parent families with dependent offspring only</td>
</tr>
<tr>
<td>%</td>
<td>Households renting from Government or Community organisation</td>
</tr>
<tr>
<td>%</td>
<td>Employed people classified as Low Skill Community and Personal Service Workers</td>
</tr>
<tr>
<td>%</td>
<td>Occupied private dwellings requiring one or more extra bedrooms (based on Canadian National Occupancy Standard)</td>
</tr>
<tr>
<td>%</td>
<td>Occupied private dwellings with no car</td>
</tr>
<tr>
<td>%</td>
<td>Occupied private dwellings with four or more bedrooms</td>
</tr>
<tr>
<td>%</td>
<td>People aged 15 years and over at university or other tertiary institution</td>
</tr>
</tbody>
</table>
- Households paying mortgage greater than $2,120 per month
- Households paying rent greater than $290 per week
- People aged 15 years and over with an advanced diploma or diploma qualification
- Employed people classified as Professionals
- Occupied private dwellings with a broadband internet connection
- People with stated annual household equivalised income greater than $52,000 (approx 9th and 10th deciles)

The Index of Relative Advantage and Disadvantage distribution can be seen below in figure 8. The scores range from around 500 to around 1300. The SEIFA indexes were calculated using Principal Components Analysis (a data reduction technique). This allows a large number of correlated variables to be summarised into a smaller set of transformed variables, where each component is a weighted linear combination of the original variables. Further explanation of this analysis and a more detailed explanation of the development of SEIFA can be found in the Socio-Economic Indexes for Areas (SEIFA) - Technical Paper (Pink, 2006). The SEIFA has been assessed and approved by an external group of experts who reviewed the methodology and variables used in its creation (Pink, 2006).

![Index of Relative Advantage and Disadvantage distribution](image)

Figure 10: Index of Relative Advantage and Disadvantage distribution (adapted from (Pink, 2006)).
Postcode data were collected in 2005. These data were used in combination with the SEIFA score for each postcode to assign an individual SEIFA score to each participant. Below in figure 9 is a representation of the Index of Relative Advantage and Disadvantage quintiles by Postal Code for the greater Melbourne metropolitan area, the city studied in the current thesis.

Figure 11: Index of Relative Advantage and Disadvantage quintiles by Postal Code, Melbourne

It is worth noting that moving or length of residence in a postcode area was not included in the analysis as it was determined that it was likely that participants remained in a neighbourhood of similar disadvantage between initial intake and follow up. A preliminary analysis determined that 45 participants (18.4%) had moved to a different postcode between baseline and follow-up four years later. When participants did move, only 11 (4.5%) moved to neighbourhoods with a SEIFA score more than one standard deviation lower or higher than their previous neighbourhood. This
indicates that participants generally remained in a neighbourhood of similar disadvantage throughout their adolescence, irrespective of whether or not they have moved.

8.3.2 Adolescent Depressive Symptomatology

The *Center for Epidemiological Studies Depression Scale, Revised* (CES-D-R; Radloff, 1977) is a self-report questionnaire designed to measure depressive symptoms in the general population (see Appendix A1). It consists of 20 statements that describe depressive symptoms (e.g., “I was bothered by things that don’t usually bother me”, “I felt sad”) that were chosen from previously validated studies. Symptoms measured by the questionnaire include depressed mood, feelings of guilt and worthlessness, feelings of helplessness and hopelessness, loss of appetite, sleep disturbance, and psychomotor retardation. Participants were instructed to indicate how often they had felt or behaved as described in each statement over the past week. They indicated this on a four-point scale ranging from 1 (rarely or not at all [less than 1 day]) to 4 (most or all of the time [5–7 days]). Higher scores indicate greater frequency and number of depressive symptoms. It is worth noting that the traditional version of the CES-D-R used for determining cut-offs uses the 0 to 3 scale, so scores obtained in this study were adjusted accordingly to match the standard, with a score of 26 suggesting a high level of depressive symptoms (traditionally 16), while a score of 24 or above has been shown to have a sensitivity of 84% and a specificity of 75% in predicting major depression in adolescence (Roberts, Lewinsohn, & Seeley, 1991). The CES-D has been used in many studies to measure adolescent depressive symptomatology, and a validation study using junior and senior high school students found that it had good internal consistency and validity (Radloff, 1991).

8.3.3 Adolescent symptoms of Anxiety

The *Beck Anxiety Inventory* (BAI; Beck, Epstein, Brown, & Steer, 1988) is a 21 item self-report questionnaire used to measure symptoms of anxiety in adults and adolescents (a copy is provided in Appendix A2). It is specifically designed to measure symptoms of anxiety that are distinct from symptoms of depression (Steer, Ranieri, Beck, & Clark, 1993). It instructs participants to report the presence and severity of various symptoms over the past week, on a scale ranging from 1 (not at all) to 4 (severely). A score below 15 indicates minimal or mild anxiety, while scores of 26 or above indicate severe anxiety (Steer, Willman, Kay, & Beck, 1994). The BAI has been
shown to have good convergent and discriminant validity in terms of anxiety and depression (Steer et al., 1993).

8.3.4 Stressful life events

Stressful life events were assessed using a life events questionnaire and an accompanying stress interview (Lewinsohn, Joiner Jr, & Rohde, 2001). The life events questionnaire used content from several well-established stressful life events questionnaires including the Schedule of Recent Experiences (Holmes & Rahe, 1967) and the Life Events Schedule (Sandler & Block, 1979). The questionnaire was developed with the aim of assessing highly valent (positive or negative) events that occurred relatively frequently in an adult population. These include both normative (e.g., starting at new school) and non-normative (e.g., death of a family member) experiences representative of the types of events empirically determined to be stress-inducing for most young people (Holmes & Masuda, 1973; Lewinsohn, Rohde, & Jeffrey, 2003). A checklist of potential events, which comprised 31 specific events (both positive and negative stressful events), with a 32\textsuperscript{nd} event designated for unspecified major stressful events, was administered to participants and they indicated whether these had occurred to them at any point in their lives.

Each life event that was endorsed from the chart was then explored individually with the researcher in the stress interview. The stress interview was adapted by Lewinsohn et al. (2003) from the Life Events and Difficulties Schedule (Brown & Harris, 1989). Participants were asked to describe each event, during which the interviewer would write a brief record and date of the event. Participants were also asked to rate the valence (positive or negative) and subjective impact of the event from 1 (not at all) to 4 (a lot). Interviewers would verify objective impact (how much they believed the event would have affected the life of an average person on a scale from 1 (not at all) to 4 (a lot)) and also whether the participants’ description of the event was consistent with the item as intended in an associated scoring manual. The interview has been shown to have good psychometric properties, with excellent intraclass correlation coefficient values for the interview endorsement of events to self and to others ($r = .97$ and $.96$, respectively), and good to excellent intrarater reliability for objective effects: Self $kappa = .84$; Others $kappa = .65$ (Lewinsohn et al., 2003), where kappa coefficients represent a stringent estimate of reliability based on point-by-point agreement, corrected for chance. Authors also note that the questionnaire and interview allows for assessment of ‘false positives’ but not ‘false negatives’, thus a significant portion of life events may be missed (Duggal et al., 2000). While it would be ideal to measure stressful life
events using an initial interview, this was thought to be too much of a burden on participants, with the current methodology representing a balance between participant burden and validity and depth of assessment (Lewinsohn et al., 2003). A copy of the Stressful Life Events questionnaire and Interview measure can be found in Appendix A3.

Despite measuring the valence and impact of each event, the current thesis only used total number of events endorsed by participants as an index of stressful life events. Although not all events assessed in the SLE-R would be considered aversive, they are all usually associated with some form of coping behaviour due to a change in life circumstances on the part of the affected individual or significant others. Many researchers have argued that, regardless of whether it is positively or negatively valenced, it is the adaptation in the face of life change which is the core defining feature of a “stressful life event” (Holmes & Masuda, 1973).

8.3.5 Socioeconomic Status

Socioeconomic status was assessed using data on parental occupation at baseline. Occupations were coded in accord with the ANU_4 scale (Jones & McMillan, 2001), which is a socioeconomic index developed for the Australian context. It classifies occupation based on social and economic characteristics. When occupational data on parents was missing, a measure of parental education was substituted (total years in school, scaled to reflect ANU_4 codes). While the ANU_4 scale does not use measures of SES other than occupation, it is a long-established measure, which is based on linkages between occupation, education and market income. Thus it brings these three factors together (Hehir, 2012).

8.3.6 Adolescent Temperament

Adolescent temperament was measured twice using self-report - first at a school screening sample of 2479, second at a home assessment in a sample of 245 (with a mean interval of 9.91 months (SD = 3.10)). For the purpose of hypothesis testing, the mean of the two self-report measurements was used as an indicator of adolescent temperament. The Early Adolescent Temperament Questionnaire—revised version (EATQ-R)—self-report, repeated measures (Ellis & Rothbart, 2001) (found in Appendix A4) is a revision of Capaldi and Rothbart’s (1992) EATQ. It was designed to assess aspects of adolescent temperament related to self-regulation, and has 10
subscales that load onto four higher order factors including Negative Affectivity, Surgency, Affiliation, and Effortful Control. These scales are Activation Control, Affiliation, Attention, Fear, Frustration, Surgency, Inhibitory Control, Shyness, Pleasure Sensitivity, and Perceptual Sensitivity. Scales measuring aggression and depressive mood were also included in this revision to examine possible relationship between temperament and social-emotional functioning. However these scales do not form part of the temperament factor structure and are treated as distinct scales. Participants respond to items of the EATQ-R by indicating how true each statement is for them on a scale of 1 (almost always untrue) to 5 (almost always true). The higher a score on a given trait, the higher a participant is considered to be similar to the particular trait measured by the scale. Only the higher order factors were examined in the current thesis.

8.3.7 Family Interaction Tasks

8.3.7.1 Family Event-Planning Interactions (EPI)

Families engaged in a family planning task, where the parent and adolescent planned between one to five enjoyable activities to do together as a family. Participants were told to discuss each activity and move to the next activity only after fully exhausting each activity being discussed. This was done until the full twenty-minute duration was complete. These activities were established prior to the interaction session using a checklist of likely activities, comprising an adolescent-appropriate modification of the Pleasant Events Checklist (see Appendix A5) (MacPhillamy & Lewinsohn, 1976), in addition to a subset of items that could be engaged in with family members. The checklist comprises 50 activities that people may enjoy participating in, such as “skiing” or “taking a trip or vacation”. Participants were instructed to rate how pleasant they would find each activity if they did it with their fellow participant (parent or child) on a 3-point scale, from 1 (Not Pleasant) to 3 (Very Pleasant). If the participant had not engaged in any of the activities, they were asked to rate how pleasant they thought it would be to engage in the activity together. There were several reasons for using this task. The first reason was that targeting fun and pleasant activities encourages positive affect (Melby, Ge, Conger, & Warner, 1995). This was important to examine as it allowed measurement of parental expression of warm/positive behaviour, which, as discussed in Chapter 5, is associated with decreased risk for anxiety and depression in adolescents (Steinberg, Lamborn, Darling, Mounts, & Dornbusch, 1994). Secondly, it facilitates a non-confictual interaction between parents and adolescent. This allows for derivation of indices of parental expression of positive emotion independently of
conflictual behaviour, the latter being more likely to be elicited in the other interaction task (the Problem-Solving Interaction task).

8.3.7.2 Family Problem-Solving Interactions (PSI)

The problem-solving interaction task (PSI) was used to encourage both conflictual and problem solving behaviour during family interactions. During the task, families were instructed to discuss and try to resolve between one and five conflictual issues, which were chosen from the responses to the Issues Checklist (Appendix A6; Prinz, Foster, Kent, & O'Leary, 1979). As in the previous task, participants were asked to resolve each issue before moving onto a new one. This was done until the full twenty-minute duration was complete. The Issues Checklist is a checklist of 44 topics about which adolescents and parents may disagree, such as “[adolescent] talking back to parents”. Participants noted which topics they had discussed in the last two weeks, and provided an estimate of how frequently the topic arose during that time. They also rated how angry they felt during the discussion of these topics on a 5-point scale, from 1 (Calm) to 5 (Very Angry). Items with the greatest conflict ratings (Frequency x Intensity) on the adolescents’ and parents’ Issues Checklist were chosen for the PSI.

The PSI was used for several reasons. The first is that it allowed for assessment of parent-child conflict, which increases in adolescence (Collins, 1990), with the ability to resolve differences being a developmentally salient skill for families (Kobak & Ferenz-Gilles, 1995) and has been shown to be predictive of adolescent functioning (Hammen, Burge, & Stansbury, 1990). As discussed in Chapter 6.4, high levels of family conflict are associated with increased risk for internalising (e.g., Kaslow, Deering, & Racusin, 1994). The second reason the PSI was used is that it is a more efficient procedure for eliciting information than a completely unstructured, naturalistic observation procedure (Robin & Foster, 2002). In particular, between-group differences in aggressive and dysphoric behaviour tend to be more apparent during conflict-resolution tasks than more neutral interactions (Schmaling & Jacobson, 1990). Thus this task provides a context from which to derive indices of parental affective behaviour. Generally it is thought that the behaviour of participants during these tasks is reflective of their usual interactions with each other in the naturalistic context. It is worth noting that, while indices of positive affect are more likely to be salient during the EPI task, and aggressive and dysphoric behaviours more salient in the PSI task, analyses will examine all expressions of emotion of both tasks.
8.3.7.3 Observational Assessment

The *Living in Family Environments Coding System* (LIFE; Hops, Davis, & Longoria, 1995) is an observational coding system that facilitates a detailed analysis of family members’ behaviours and sequential patterns of behaviour between two or more family members. In the current thesis it was used to code behaviour in two family interaction tasks (i.e., the EPI and the PSI, discussed previously). While the LIFE was originally created to assess the impact of depressive behaviour within families, allowing for the differentiation of aggressive and depressive behaviours, it also includes codes to record positive, prosocial and neutral behaviours. Thus it has been used to examine many forms of interaction amongst family members of many different ages and clinical presentations (Hops et al., 1995).

Coding of behaviour and affect is done in real time, using an event-based protocol whereby new codes are entered each time the affect or content of one of the participants changes. The LIFE system allows the coding of 10 types of nonverbal affect (contempt, anger, anxious, dysphoric, pleasant, neutral, happy, caring, whine, and belligerence) and 27 types of verbal affect (e.g., validation, complaint, provoke). The full list of codes can be found in Appendix A7. Each code is made up of five digits, which represent (in order), the identity of the subject who is speaking or behaving (one digit), the subject’s content code (two digits), the identity of the person interacting with the subject (one digit), and the subject’s affect code (one digit). Behavioural codes that do not occur during an interaction were recorded as missing data. The 10 affect codes and 27 verbal content codes were used to develop composite behaviour constructs, which in this study included Aversive, Dysphoric, and Positive behaviour (See Appendix A7 for further details on the composition of the behaviour constructs).

Aversive behaviour included all contemptuous, angry, or belligerent affect codes, and codes relating to cruel, provocative, annoying/disruptive, or argumentative verbal statements made with neutral affect. Dysphoric behaviour consisted of all dysphoric, anxious, or whining affect codes, and codes relating to complaints and self-derogatory verbal comments made with neutral affect. A new Positive behavioural construct was developed for the ADS study, which included all happy, pleasant, and caring affect codes as well as codes related to approving, validating, affectionate or humorous comments made with neutral affect. This construct was adapted from a previously developed facilitative behavioural construct, which contains similar codes (Hops et
al., 1995). It is not unusual for the LIFE system to be altered, with many studies adapting it to suit specific needs.

The individual codes and the established constructs have demonstrated adequate reliability and validity, with kappa coefficients for the behavioural constructs ranging between .60 and .80 (Hops et al., 1995). All video recordings of the family interaction tasks were coded at the Oregon Research Institute. The coders were well trained and blind to the clinical and demographic characteristics of the participants. A second observer coded approximately 20% of the interactions in order to provide an estimate of observer agreement. Average kappa coefficients were found to be .66 and .78 for the content and affect codes respectively, and had values of .77, .68, and .89 for the composite constructs of Aversive, Dysphoric, and Positive behaviour respectively. Random pairs of observers were assigned to the interactions to ensure that all observers met minimal criteria for acceptable observations and to minimize drift between any two observers. The base rate for coding an affect or content code was one. This means that when it fails to occur at least once, it arises as a missing value for that code for that particular participant. This results in variations in $N$ for different constructs in different tasks.

The Aversive, Dysphoric, and Positive constructs were used to calculate several variables designed to represent measures of parental affective behaviour, which were a focus of the current thesis. These variables will be described in the following sections.

While both fathers and mothers took part in the family interaction task, the current thesis only used data from mothers, given they formed the majority of participating parents (163 mothers, compared to 35 fathers). While fathers are likely to play a significant role in adolescent development, there are likely to be differing processes involved in parenting by mothers versus fathers. Thus these processes should be studied separately. However, the small sample of fathers in the current study lacked the power to allow for examination of processes involved in parenting by fathers.

8.3.7.4 Rate per minute (RPM)

The RPM variable represents the average number of times a particular behaviour construct occurred per minute of interaction between participants. This variable was calculated by dividing the total number of times the construct occurred during the interaction by the total duration (in
minutes) of the observed interaction (Hops et al., 1995). The higher the RPM, the more frequently the parent expressed the behaviour construct of interest. RPM was taken as a measure of affective expression.

8.3.7.5 Duration per episode (DPE)

The DPE variable represents the average length of time that a behaviour was maintained each time it was displayed. It was calculated by dividing the overall duration of the behaviour by the frequency with which it occurred in the interaction (Sheeber et al., 2009). This variable provided a measure of affect regulation, as it was considered to gauge the parent’s ability to move out of negative affective states. Longer duration of Aggressive and Dysphoric behaviours and shorter duration of positive behaviours were taken as indices of greater affective dysregulation.

8.4 Summary of measures and assessment periods

A summary of measures used in the current thesis, along with the assessment time points during which they were collected is represented in Table 4 below.
<table>
<thead>
<tr>
<th>Time point</th>
<th>Construct</th>
<th>Method</th>
<th>Measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Screening</td>
<td>Temperament</td>
<td>Adolescent self-report questionnaire</td>
<td>EATQ-R</td>
</tr>
<tr>
<td></td>
<td>Depressive symptoms</td>
<td>Adolescent self-report questionnaire</td>
<td>CES-DR</td>
</tr>
<tr>
<td></td>
<td>Anxious symptoms</td>
<td>Adolescent self-report questionnaire</td>
<td>BAI</td>
</tr>
<tr>
<td>Time 1</td>
<td>Parental affective behaviour</td>
<td>Observational assessment</td>
<td>LIFE coding system (parent RPM and DPE)</td>
</tr>
<tr>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>Socio-demographic data</td>
<td>SEIFA (Index of Relative Socio-economic Advantage and Disadvantage)</td>
</tr>
<tr>
<td></td>
<td>SES</td>
<td>Parent questionnaire</td>
<td>ANU-4 scale of SES</td>
</tr>
<tr>
<td>Time 2</td>
<td>Stressful Life Events</td>
<td>Questionnaire and interview</td>
<td>Lewinsohn Stressful Life Events Questionnaire and Interview</td>
</tr>
<tr>
<td>Time 3</td>
<td>Depressive symptoms</td>
<td>Adolescent self-report questionnaire</td>
<td>CES-DR</td>
</tr>
<tr>
<td></td>
<td>Anxious symptoms</td>
<td>Adolescent self-report questionnaire</td>
<td>BAI</td>
</tr>
</tbody>
</table>
CHAPTER 9
DESIGN AND DATA ANALYSIS

This chapter will provide a description of data analysis for the current thesis.

9.1 Statistical analysis

The following sections detail the analyses performed in the current thesis to explore each of the hypotheses. Section 9.1.1 will outline procedures relevant to all analyses, whilst section 9.1.2 details the analysis of direct relationships. Section 9.1.3 outlines the analysis of mediating relationships, while 9.1.4 discusses the inclusion of gender as a moderator in all analyses.

9.1.1 Details pertaining to all analyses

9.1.1.1 Statistical package

All regression analyses, along with mediational analyses, t-tests, and missing data imputation, were conducted using the Statistical Package for the Social Sciences (SPSS), Version 20.0.0 (SPSS Inc., 2011).

9.1.1.2 Control variables

All analyses controlled for participant gender. Age was not controlled as the sample was an age cohort and therefore there was relatively little variance in age. All analyses were also performed and reported both with and without family SES as an additional control variable. The reasoning behind this was that there are arguments for and against controlling for SES (discussed in Chapter 2.8), with the addition of family SES providing a more stringent, but perhaps overly conservative test of the effect of neighbourhood disadvantage (given the expected significant correlation between these two variables). Presentation of both analyses will give a more comprehensive and detailed picture of the influence of both neighbourhood disadvantage and
individual SES on the various dependent variables examined. Further analyses controlling for family SES will only be conducted for relationships found to be significant in initial analyses. Each results chapter will first report analyses without controlling for SES, followed by a section in which family SES is controlled. Initial symptoms of anxiety and depression were controlled for when examining anxious and depressive symptoms at T3 (follow up). This was done to remove the variance in depression/anxiety attributable to baseline symptoms, so that the independent variable would effectively be predicting the change in depression/anxiety from T1 to T3. This provided a more rigorous method for investigating the prospective prediction of depressive and anxious symptoms by the independent variables, after controlling for initial symptoms.

9.1.1.3 Centring data

All non-categorical predictor variables were centred to reduce problems with multicollinearity (where the cross-product interaction terms may be highly correlated) and to allow for easier and more meaningful interpretations of regression coefficients. This approach is recommended by most statisticians (e.g., Garson, 2012).

9.1.1.4 Correcting for multiple comparison

As I performed 15 linear regressions and 40 mediational regressions, it was necessary to consider correcting for multiple comparisons to control for type 1 error. A Bonferroni correction was considered, however, the use of corrective procedures like these is now commonly considered overly conservative and greatly increases the chance of type II error (Perneger, 1998). One recommended approach to multiple comparisons is to simply describe what tests have been performed and why (Perneger, 1998), which enables the reader to reach a reasonable conclusion regarding the possibility of Type I error without the restrictions associated with Bonferroni adjustments (Savitz & Olshan, 1995). This approach was taken with the current thesis and was considered particularly appropriate given that I reported results of all analyses that were performed, and also that all these analyses were hypothesised and theory driven.
9.1.1.5 Assumptions

Hierarchical regression (including mediational analyses) requires several assumptions to be met. These include linearity of relationships, low multicollinearity (intercorrelation of independent variables), homoscedasticity (the same level of relationship throughout the range of the IV), that the distribution of the dependent variables are normally distributed at each value of the independent variable, interval or near-interval measurement level, absence of outliers, and data whose range is not truncated. All variables were checked to ensure that they met the assumptions of linearity and univariate and multivariate normality. Standardised residuals were examined to ensure normally distributed residual error. Outliers were examined using dfbeta residuals. No assumptions were violated, unless otherwise noted.

9.1.2 Statistical analysis of direct relationships

To address hypotheses 1, 2, 4, 5, 6 and 8 (i.e., those excluding mediation testing), linear regressions were utilised to test the associations between neighbourhood disadvantage (the predictor variable) and the dependent variables, including internalising symptoms, temperament, maternal affective behaviour, and stressful life events (see figure 12 for a diagrammatic representation of the relationships to be examined). Significance testing for all direct effects using regression analyses was determined on the basis of a p-value of less than .05. Unless noted otherwise, a 1-tailed t-test was used for all analyses, given the direction of deviation from the null value was clearly specified in each hypothesis.
Linear regression was performed with adolescent gender entered in the first step alongside neighbourhood disadvantage. Other control variables were added when relevant as described in the section 9.1.1.2. Results of analyses of direct effects between neighbourhood disadvantage and dependent variables are reported in Chapter 10.

9.1.3 Statistical analyses of mediational analyses

To address hypotheses 3, 7 and 9 relating to mediational relationships, regression analyses were used as represented in figure 13. The online interactive mediation analytic tool by Preacher and Hayes (2008) was utilized for all mediation tests. In particular, the test statistics of the recommended INDIRECT macro was used, which estimates the path coefficients in a multiple mediator model and generates bootstrap confidence intervals (percentile, bias-corrected, and bias-corrected and accelerated) for total and specific indirect effects of X on Y through the mediator variable. Bootstrapping was also used, which involves the generation of an empirical sampling distribution of $a \times b$, taking the sample size of $N$ and from it drawing replacement $N$ values ($X, M, Y$) to create a new sample. Baron and Kenny’s equations 1 and 3 are estimated for each bootstrap sample, which allows for the estimation of $a, b$ and $a \times b$. 5000 bootstrap samples were drawn for each analysis, and $a \times b$ estimated for each, after which SPSS was able to estimate the indirect effect as the mean of these estimates. The INDIRECT test (including bootstrapping) is considered superior to the SOBEL test, as it adjusts all paths for the potential influence of covariates not proposed to be mediators in the model. In addition, as discussed in Chapter 3.2, it
has more statistical power and more accurate confidence intervals when testing indirect effects (MacKinnon et al., 2004; Pituch et al., 2006). The significance of indirect effects in the mediational analysis was determined on the basis of the 95% confidence intervals (CI). That is, if the 95% CI of a parameter did not pass through zero, then the effect was considered statistically significant. Bootstrapping relies on the 95% confidence intervals from the empirical distribution of the $a \times b$ estimates. The lower bound of the 95% confidence interval is at the 2.5% point on this cumulative distribution, and the upper bound of the 95% confidence interval is at the 97.5% point.

Figure 13: Representation of potential mediating relationships analysed in the current thesis.

Separate analyses were conducted for each mediator in question. Putative mediators were selected for these analyses if they were found to be significantly related to neighbourhood disadvantage (thus indicating a significant $a$ path) in the previous analyses of direct relationships described in section 9.1.2). All mediational analyses examined whether these potential mediators statistically mediated the relationship between neighbourhood disadvantage and (1) depressive symptoms at T1; (2) anxious symptoms at T1; (3) change in depressive symptoms from T1 to T3; and (4) change in anxious symptoms from T1 to T3.

As discussed in chapter 3.2, even if a significant relationship was not detected between neighbourhood disadvantage and internalising symptoms (the $c$ path), mediational analyses were conducted to determine whether there were any indirect, mediating relationships between these variables. This approach is encouraged by many researchers and statisticians, who note that, particularly for longitudinal research, an examination of indirect pathways is justified even when
a significant $c$ path is not detected (e.g., Collins, Graham, & Flaherty, 1998; MacKinnon et al., 2002; Shrout & Bolger, 2002). They argue that the most important test in a mediational analysis is of the indirect path, and that other unexplored *suppressor* variables can suppress other mediator variables, leading to a non-significant $c$ path. Collins and associates (1998) argue that a mediational relationship is an intra-individual process that unfolds over time. Whilst the independent variable (neighbourhood disadvantage) may provide the initial influence, a variety of proximal causes specific to the individual (e.g., mediating variables such as parent quality or stress), are the more direct influencer of the dependent variable. Thus an overall direct effect may be suppressed through a complex interplay of mediating variables, which exert different effects over time.

Shrout and Bolger (2002) go so far as to recommend that a direct effect should not even be tested when there is temporal distance between the variables being assessed. This is justified on the ground that the when causal variables become more temporally distal from one another, the direct effect most often weakens. Thus a non-significant direct effect is often found. The greater the time between measurement of each variable, the more chance the effect will be influenced by additional links in a causal chain, which will likely include competing and random factors. Thus, according to Preacher and Hayes (2004), the most important indicator of a mediational relationship is a significant indirect effect.

9.1.4 Gender Differences

Gender was tested as a potential moderator in all analyses. For main effect linear regression, a gender by neighbourhood disadvantage interaction term was added. For mediation analyses, moderated-mediation was tested using the MOD-MED program by Preacher and colleagues (2007). Moderated mediation refers to the situation where an indirect relationship varies systematically as a function of another variable (e.g., a potential moderator such as gender). Specifically, it is when the effect of an independent variable on the mediator depends on the moderator, or when the partial effect of the mediator on the dependent variable depends on the moderator, or both (Muller, Judd, & Yzerbyt, 2005). Details of the procedure for this analysis are discussed in Preacher, Rucker and Hayes (2007).
9.2 Missing data imputation

Missing data is an almost unavoidable occurrence in longitudinal studies. In the current project, missing data resulted from incomplete questionnaires and family’s non-participation in assessments. The following sections summarise missing data for all measures as well as outlining how this was managed.

9.2.1 Temperament

All participants completed the EATQ at the initial screening. Seven participants (2.85% of the total sample) did not complete the EATQ at the baseline assessment. At the item level there was 1.03% missing data at the in-school screening, and 2.76% at the baseline assessment. Scores were averaged across the two assessments once the temperament scales had been constructed from the item-level data. When participants were missing data at one assessment, their score at the other assessment was substituted. Once data was combined from the school screening and the baseline assessments using this process, the final amount of missing data on the 10 temperament scales was 0.12%

9.2.2 Internalising symptoms

With regard to non-completion of baseline symptom questionnaires, 8 participants (3.27% of the total sample of 245) did not complete the BAI, and 18 participants (7.35%) did not complete the CES-DR. In terms of T3 questionnaires, 70 (28.6% of the full sample) did not complete the CES-DR and 69 (28.2% of the full sample) did not complete the BAI.

Aside from participants who did not complete the questionnaires at all, the percentage of missing data at the item level for each questionnaire was: 5.09% of the CES-DR at T1, .15% of the CES-DR at T3, 1.23% for the BAI at T1 and 0.22% at T3.

9.2.3 Family assessment
48 of the ADS families (19.6% of the full ADS sample) did not complete the family assessment, while one family only completed the EPI and not the PSI.

9.2.4 Neighbourhood disadvantage

There were no missing postcode data, and thus no missing neighbourhood disadvantage data.

9.2.5 Stressful life events

80 adolescents (32.7% of the full sample) did not complete the Stressful Life Events questionnaire and interview.

9.2.6 Data imputation technique

For all missing data outlined in the previous sections (9.2.1, 9.2.2, 9.2.3, 9.2.4 9.2.5), data imputation was conducted using the expectation-maximisation (EM) algorithm to impute all missing data, including diagnostic, observational and questionnaire data, using SPSS. This method is an iterative process, which estimates the means, covariances, and correlations of quantitative variables. There are two steps for each iteration. The first step computes expected values conditional on the observed data and the current estimates of the parameters. The second step calculates maximum likelihood estimates of the parameters based on values that are computed in the first step (SPSS Inc., 2011).

This process continues until there is convergence in the parameter estimates, thus indicating that further iterations will not result in significant changes in the estimated values (SPSS Inc., 2011; Schafer & Olsen, 1998). As discussed in Chapter 8.3.1, there was evidence of some bias in the pattern of missingness in the data, indicating that the data were missing at random (MAR). This suggests that what caused the data to be missing does not depend upon the missing data itself, in comparison to data that is missing completely at random (MCAR), which suggests that the events that lead to any particular data-item being missing are independent both of observable variables and of unobservable parameters of interest. Whilst data MAR is not ideal, The EM procedure is fairly robust to MAR, particularly when there are several predictors that can predict
the missingness (as exist for the current thesis) (Bennett, 2001). All data used in the current thesis was imputed data.

9.3 Descriptive statistics

The distribution of each variable was calculated and the mean, median, standard deviation, range of scores, skewness, and kurtosis are displayed in relevant tables.

9.3.1 Depressive and anxious symptoms

Descriptive statistics for the two clinical scales are summarised in Table 5. Depressive symptoms were found to be relatively low at both time points in the ADS sample, with the majority of participants (135; 55% at T1 and 141; 58% at T3) reporting total scores between 0-10 at baseline. Scores were relatively high for 25 participants (10.20%) at T1 and 16 (6.5%) at T3, who had total scores of 24 or higher, which suggests that they were at risk of meeting criteria for major depressive disorder. Most participants (199; 81.22% at T1 and 207; 84.45% at T3) had a total of 14 or less on the BAI, indicating that they experienced only minimal or mild anxiety. Fourteen participants (5.71%) at T1 and ten (4.08%) at T3 had scores of 26 or above, indicating severe anxiety. There was a relative decline in self-reported depressive and anxious symptoms between T1 and T3. This is commonly found in longitudinal studies of internalising symptoms, and there is evidence that suggests this is not due to a genuine decline in symptoms, but instead is a product of measurement effects (Twenge & Nolen-Hoeksema, 2002).

Table 5

<table>
<thead>
<tr>
<th>Scale</th>
<th>Mean (median)</th>
<th>SD</th>
<th>Range</th>
<th>Skewness (SE skewness)</th>
<th>Kurtosis (SE Kurtosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CES-D (T1)</td>
<td>11.46 (9.00)</td>
<td>9.00</td>
<td>0-55</td>
<td>1.79 (0.16)*</td>
<td>4.41 (0.31)</td>
</tr>
<tr>
<td>BAI (T1)</td>
<td>8.47 (6.00)</td>
<td>8.95</td>
<td>0-44</td>
<td>1.79 (0.16)*</td>
<td>3.38 (0.31)</td>
</tr>
<tr>
<td>CES-D (T3)</td>
<td>10.62 (9.00)</td>
<td>7.61</td>
<td>0-48</td>
<td>1.52 (.16)*</td>
<td>3.37 (.31)</td>
</tr>
<tr>
<td>BAI (T3)</td>
<td>8.58 (7.00)</td>
<td>7.65</td>
<td>0-47</td>
<td>2.00 (.16)*</td>
<td>5.64 (.31)</td>
</tr>
</tbody>
</table>

* $p = <.001$ ($z$-score)
Both clinical scales at both time points showed high positive skewness and kurtosis. This indicates that adolescents were reporting relatively low levels of depressive and anxious symptoms. This was not unexpected given it was a non-clinical, community based sample. The high kurtosis or skewness was not considered to be a problem, as linear regression is robust against small to moderate departures from normality, particularly when they exist in the dependent variable and the sample size is large (as is the ADS sample) (Tabachnick, Fidell, & Osterlind, 2001).

In order to assess whether anxiety and depression questionnaires measured separate constructs (symptoms of anxiety and symptoms of depression), the correlation between these measures was assessed and found to be .69 for symptoms at T1 and .68 for symptoms at T3. This suggests that, while these constructs do share significant overlap, there is not such a high correlation as to make them redundant. Supporting this assertion are previous studies using these measures, which have not always found the same results for measures of anxiety and depression. For instance, Schwartz and colleagues (2011) used the same measures of depressive and anxious symptoms as the current thesis and found that higher levels of positive parenting predicted greater depressive symptoms, but not greater symptoms of anxiety.

9.3.2 Temperament

Descriptive statistics for the four higher order factors of the EATQ-R are presented in Table 6. All temperament data were normally distributed.
Table 6
*EATQ-R Descriptive Statistics (n = 245)*

<table>
<thead>
<tr>
<th>Scale</th>
<th>Mean (median)</th>
<th>SD</th>
<th>Range</th>
<th>Skewness (SE skewness)</th>
<th>Kurtosis (SE Kurtosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative Affectivity</td>
<td>3.20 (3.20)</td>
<td>0.77</td>
<td>1.21-</td>
<td>-0.16 (0.16)</td>
<td>-0.28 (0.31)</td>
</tr>
<tr>
<td>Effortful Control</td>
<td>3.46 (3.45)</td>
<td>0.60</td>
<td>1.74-</td>
<td>0.03 (0.16)</td>
<td>-0.43 (0.31)</td>
</tr>
<tr>
<td>Surgency</td>
<td>3.35 (3.36)</td>
<td>0.63</td>
<td>1.74-</td>
<td>-0.10 (0.16)</td>
<td>-0.64 (0.31)</td>
</tr>
<tr>
<td>Affiliativeness</td>
<td>3.44 (3.45)</td>
<td>0.52</td>
<td>1.97-</td>
<td>-0.18 (0.16)</td>
<td>-0.08 (0.31)</td>
</tr>
</tbody>
</table>

9.3.3 Stress

Descriptive statistics for the stressful life events data are summarised in Table 7. The Stressful Life Events scale showed high positive skewness and kurtosis. This indicates that adolescents were reporting a relatively low number of stressful life events, with a mode of 1 stressful life event. As above, this was not unexpected given it was a non-clinical, community based sample.

Table 7
*Stressful Life Events – Questionnaire Descriptive Statistics (n = 245)*

<table>
<thead>
<tr>
<th>Scale</th>
<th>Mean (median)</th>
<th>SD</th>
<th>Range</th>
<th>Skewness (SE skewness)</th>
<th>Kurtosis (SE Kurtosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stressful Life Events</td>
<td>8.64 (8)</td>
<td>6.12</td>
<td>1.0-27.0</td>
<td>0.671 (0.193)</td>
<td>-0.085 (0.384)</td>
</tr>
</tbody>
</table>

9.3.4 Neighbourhood disadvantage

Descriptive statistics for neighbourhood disadvantage scores are summarised in Table 8. Note that a higher score on the scale of neighbourhood disadvantage actually indicates a lower level of neighbourhood disadvantage (e.g., a postcode with a score of 800 would be more disadvantaged than a postcode with a score of 1100). In comparison to the SEIFA score distribution from the ABS sample (represented in figure 14), the score distribution from the ADS sample (represented
in figure 15) shows a slight overrepresentation of adolescents from neighbourhoods with above average scores on neighbourhood disadvantage. This is to be expected from a cohort of families who are willing to participate in research, as studies generally find an overrepresentation of research participants from a higher SES background (Heinrichs, Bertram, Kuschel, & Hahlweg, 2005). Also note that, unlike the SIEFA sample, there is no elongated left tail, indicating that the current sample may be under representative of the most disadvantaged neighbourhoods.

Table 8
Neighbourhood Disadvantage Descriptive Statistics (n = 245)

<table>
<thead>
<tr>
<th>Scale</th>
<th>Mean (median)</th>
<th>S.D.</th>
<th>Range</th>
<th>Skewness (SE skewness)</th>
<th>Kurtosis (SE kurtosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neighbourhood disadvantage</td>
<td>1032.16 (1037)</td>
<td>66.27</td>
<td>828.00 – 1174.0</td>
<td>-.312 (.156)*</td>
<td>-.003 (.310)</td>
</tr>
</tbody>
</table>

* p = <.001 (z-score)
Figure 14: Histogram showing distribution of neighbourhood disadvantage scores (Index of Relative Advantage and Disadvantage) in the ABS sample (from Pink (2006)).

Figure 15: Histogram showing distribution of neighbourhood disadvantage scores (SEIFA) in the ADS sample.
9.3.5 Family SES

Descriptive statistics for family SES scores are summarised in Table 9. The SES scale showed high positive skewness, with a slight overrepresentation of higher SES families. As with neighbourhood disadvantage, this is to be expected from a cohort of families who are willing to participate in research (Heinrichs et al., 2005).

Table 9

<table>
<thead>
<tr>
<th>Scale</th>
<th>Mean (median)</th>
<th>S.D.</th>
<th>Range</th>
<th>Skewness (SE skewness)</th>
<th>Kurtosis (SE kurtosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family SES</td>
<td>56.50 (55.60)</td>
<td>20.026</td>
<td>7.20 – 100.00</td>
<td>.041 (.156)*</td>
<td>-.755 (.310)</td>
</tr>
</tbody>
</table>

*p = <.001 (z-score)

9.3.6 Maternal affective behaviour (RPM and DPE) data

Descriptive statistics for the three styles of maternal affective behaviour are displayed in Table 10 (RPM) and Table 11 (DPE). It can be seen that more frequent and persistent negative expressions of emotion are displayed during the conflictual task (PSI). Mothers are also more likely to display more aggressive behaviour more often and for longer periods during the PSI compared to the EPI. In terms of the rate per minute of expressed emotion, it can be seen that parents displayed positive behaviour almost four times per minute more than aversive or dysphoric behaviours on the EPI.

In terms of RPM variables, the rates of Aversive and Dysphoric behaviour on both tasks, and Positive behaviour on the PSI were slightly positively skewed, indicating that parents displayed these behaviours relatively infrequently. Positive behaviour was normally distributed on the EPI task, presumably because the task was designed to elicit this style of interaction.
### Table 10

**Parent Behaviour RPM Descriptives (N = 245)**

<table>
<thead>
<tr>
<th>Scale</th>
<th>EPI Mean</th>
<th>EPI SD</th>
<th>PSI Mean</th>
<th>PSI SD</th>
<th>EPI Skewness (SE skewness)</th>
<th>EPI Kurtosis (SE kurtosis)</th>
<th>PSI Skewness (SE skewness)</th>
<th>PSI Kurtosis (SE kurtosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aversive</td>
<td>0.59</td>
<td>0.4</td>
<td>1.30</td>
<td>.58</td>
<td>1.809 (0.156)</td>
<td>4.235 (0.310)</td>
<td>0.281 (0.156)</td>
<td>0.505 (0.310)</td>
</tr>
<tr>
<td>Dysphoric</td>
<td>0.56</td>
<td>0.38</td>
<td>0.53</td>
<td>.31</td>
<td>0.796 (0.156)</td>
<td>1.457 (0.310)</td>
<td>1.760 (0.156)</td>
<td>4.052 (0.310)</td>
</tr>
<tr>
<td>Positive</td>
<td>2.30</td>
<td>0.52</td>
<td>1.66</td>
<td>.70</td>
<td>-0.072 (0.156)</td>
<td>1.396 (0.310)</td>
<td>0.326 (0.156)</td>
<td>0.718 (0.310)</td>
</tr>
</tbody>
</table>

### Table 11

**Parent Behaviour DPE Descriptives (N = 194)**

<table>
<thead>
<tr>
<th>Scale</th>
<th>EPI Mean</th>
<th>EPI SD</th>
<th>PSI Mean</th>
<th>PSI SD</th>
<th>EPI Skewness (SE skewness)</th>
<th>EPI Kurtosis (SE kurtosis)</th>
<th>PSI Skewness (SE skewness)</th>
<th>PSI Kurtosis (SE kurtosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aversive</td>
<td>3.98</td>
<td>2.76</td>
<td>10.74</td>
<td>8.20</td>
<td>1.808 (0.156)</td>
<td>6.339 (0.310)</td>
<td>2.806 (0.156)</td>
<td>14.549 (0.310)</td>
</tr>
<tr>
<td>Dysphoric</td>
<td>5.13</td>
<td>2.51</td>
<td>5.73</td>
<td>2.49</td>
<td>1.780 (0.156)</td>
<td>8.184 (0.310)</td>
<td>1.696 (1.56)</td>
<td>7.320 (0.310)</td>
</tr>
<tr>
<td>Positive</td>
<td>7.95</td>
<td>3.11</td>
<td>7.34</td>
<td>2.54</td>
<td>1.792 (0.156)</td>
<td>5.778 (0.310)</td>
<td>1.538 (0.156)</td>
<td>3.980 (0.310)</td>
</tr>
</tbody>
</table>
CHAPTER 10

RESULTS OF LINEAR REGRESSION ANALYSES

This chapter discusses the results of analyses that aim to answer the broad question – how do families in disadvantaged neighbourhoods differ from those in less disadvantaged neighbourhoods? That is, are disadvantaged neighbourhoods associated with:

- Increased rates of depressive and anxious symptoms? (Section 10.1)
- Increased rates of stressful life events? (Section 10.2)
- Higher Negative Affectivity, and lower Surgency, Effortful Control and Affiliation in adolescents? (Section 10.3)
- Increased frequency of maternal aversive and dysphoric behaviour and decreased frequency of positive behaviour? (Section 10.4)
- Increased duration of maternal aversive and dysphoric behaviour and decreased duration of positive behaviour? (Section 10.5)

Each of these sections will report analyses without family SES as a control variable, while section 10.6 will provide a summary of analyses that include SES as a control variable. A detailed discussion of the findings in each section will be provided in relevant sections in Chapter 14.

10.1 Neighbourhood disadvantage and depressive and anxious symptoms

A series of linear regression analyses were conducted to test the relationship between neighbourhood disadvantage and adolescent self-report depressive and anxious symptoms at baseline and follow-up, controlling for any adolescent gender effects. Four specific hypotheses were tested:

- 1a. Neighbourhood disadvantage will be associated with increased symptoms of depression measured at baseline.
- 1b. Neighbourhood disadvantage will be associated with increased symptoms of depression measured at follow-up controlling for depressive Symptoms at baseline.
- 1c. Neighbourhood disadvantage will be associated with increased symptoms of anxiety
measured at baseline.

1d. Neighbourhood disadvantage will be associated with increased symptoms of anxiety measured at follow-up, controlling for anxious symptoms at baseline.

10.1.1 Neighbourhood disadvantage and depressive symptoms (T1)

A linear regression was run using neighbourhood disadvantage as a predictor of depressive Symptoms at T1 (controlling for gender). Neighbourhood disadvantage and gender (female) predicted higher levels of depressive symptomatology at baseline. Regression coefficients and standard errors can be found in Table 12 (below). To avoid confusion, it is worth noting again that a higher score on the scale of neighbourhood disadvantage actually indicates lower levels of neighbourhood disadvantage (e.g., a neighbourhood with a disadvantage score of 800 would be more disadvantaged than a neighbourhood with a disadvantage score of 1000). A gender X neighbourhood disadvantage interaction term was also entered into the model, but was not found to be significant. Thus it was removed from the analysis. In fact, a gender X neighbourhood disadvantage did not moderate any of the relationships tested in this Chapter, thus it was removed from all analyses.

Table 12

Summary of Linear Regression (Controlling for Gender), with Neighbourhood Disadvantage Predicting Depressive Symptoms at Baseline

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Significance and model fit for final model</th>
<th>Predictors in model</th>
<th>Standardised beta (B)</th>
<th>t</th>
<th>p (1-tail)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressive Symptoms (T1)</td>
<td>F(2, 242) = 4.16, p &lt; .05, AR2 = .033</td>
<td>Gender</td>
<td>-.13</td>
<td>-1.97</td>
<td>.025</td>
</tr>
<tr>
<td>Neighbourhood disadvantage</td>
<td>-.13</td>
<td>-2.05</td>
<td>.02</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Bolding indicates p < .05

10.1.2 Neighbourhood disadvantage and change in depressive symptoms between T1 and T3

A linear regression was run using neighbourhood disadvantage as a predictor of depressive
symptoms at T3 (controlling for gender and depressive symptoms at baseline). Neighbourhood disadvantage did not predict greater increase or less reduction in depressive symptomatology. It is worth noting that there was a trend towards significance for neighbourhood disadvantage, in that higher levels of disadvantage were associated with greater increase in depressive symptoms. Regression coefficients and standard errors can be found in Table 13 (below).

Table 13
Summary of Linear Regression (controlling for gender and T1 symptoms of anxiety) with Neighbourhood disadvantage Predicting Depressive Symptoms at follow-up

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Significance and model fit for final model</th>
<th>Predictors in model</th>
<th>Standardised beta (B)</th>
<th>t</th>
<th>p (1-tail)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressive Symptoms (T3)</td>
<td>$F(3, 241) = 23.9, \ p &lt; .05$, $\ AR^2 = .16$</td>
<td>Gender</td>
<td>.074</td>
<td>28.86</td>
<td>.11</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Depressive Symptoms (T1)</td>
<td>.40</td>
<td>6.64</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>-.084</td>
<td>-1.42</td>
<td>.078</td>
</tr>
</tbody>
</table>

Bolding indicates $p < .05$

10.1.3 Neighbourhood disadvantage and Anxious Symptoms (T1)

A linear regression was run using neighbourhood disadvantage as a predictor of anxious symptoms at T1 (controlling for gender). Three multivariate outliers were detected (4 standard deviations above the mean) and removed. It is common practice to remove outliers that are 3.5 standard deviations above the mean if they are shown to over-influence the results of the regression analysis and if their removal can be justified on theoretical grounds (Garson, 2012). However, removing all outliers 3.5 standard deviations above the mean led to the removal of an excessive number of cases. Thus, only outliers 4 standard deviations above the mean were removed. Outliers were removed on the grounds that they had an undue influence on the results of the regression analysis. Their removal was also justified on theoretical grounds – these outliers were cases from less disadvantaged neighbourhoods with particularly high levels of depressive symptomatology. It was theorised that people with very high levels of depressive symptoms may be influenced by factors that override neighbourhood influences (such as extreme trauma). Thus findings from these
individuals may not be generalizable to a community sample.

After removing these outliers, a new regression analysis was conducted, which contained no new multivariate outliers (above 4 standard deviations above the mean). Neighbourhood disadvantage and gender (female) significantly predicted higher symptoms of anxiety at T1. Regression coefficients and standard errors can be found in Table 14 (below).

Table 14
Summary of Linear Regression (Controlling for Gender), with Neighbourhood disadvantage Predicting Symptoms of Anxiety at baseline

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Significance and model fit for final model</th>
<th>Predictors in model</th>
<th>Standardised beta ((B))</th>
<th>(t)</th>
<th>(p) (1-tail)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxious Symptoms (T1)</td>
<td>(F(2, 242) = 3.89, p &lt; .05, AR^2 = .027)</td>
<td>Gender</td>
<td>-.13</td>
<td>-2.04</td>
<td>.022</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>-.13</td>
<td>-2.08</td>
<td>.020</td>
</tr>
</tbody>
</table>

Bolding indicates \(p < .05\)

10.1.4 Neighbourhood disadvantage and change in Anxious Symptoms between T1 and T3

The results of a linear regression analysis did not find neighbourhood disadvantage to be a significant predictor of anxious symptoms at T3 while controlling for gender and anxious symptoms at T1. Regression coefficients and standard errors can be found in Table 15 (below). Note that this analysis removed the same outliers as the analysis described in section 10.1.3.
Table 15

Summary of Linear Regression (Controlling for Gender and Anxious Symptoms at baseline), with Neighbourhood disadvantage Predicting Depressive Symptoms at T3

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Significance and model fit for final model</th>
<th>Predictors in model</th>
<th>Standardised beta (B)</th>
<th>t</th>
<th>p (1-tail)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxious Symptoms (T3)</td>
<td>$F(3, 241) = .21, p &gt; .05, AR^2 = .002$</td>
<td>Gender</td>
<td>.025</td>
<td>.41</td>
<td>.34</td>
</tr>
</tbody>
</table>

Bolding indicates $p < .05$

10.2 Neighbourhood disadvantage and Adolescent Stressful life events

A linear regression analysis was conducted to test the relationship between neighbourhood disadvantage and adolescent stressful life events. It was predicted that adolescents from disadvantaged neighbourhoods would experience more stressful life events. Results of this linear regression analysis found neighbourhood disadvantage was a significant predictor of increased stressful life events, while controlling for gender. Regression coefficients and standard errors can be found in Table 16 (below).

Table 16

Summary of Linear Regression (Controlling for Gender), with Neighbourhood disadvantage Predicting Stressful Life Events

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Significance and model fit for final model</th>
<th>Predictors in model</th>
<th>Standardised beta (B)</th>
<th>t</th>
<th>p (1-tail)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stressful Life Events</td>
<td>$F(2, 242) = 4.26, p = .015, AR^2 = .026$</td>
<td>Gender</td>
<td>.086</td>
<td>1.37</td>
<td>.085</td>
</tr>
<tr>
<td>Neighbourhood disadvantage</td>
<td>-.17</td>
<td>-2.61</td>
<td>.005</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Bolding indicates $p < .05$
10.3 Neighbourhood disadvantage and adolescent temperament

A series of linear regression analyses were conducted to test the relationship between neighbourhood disadvantage and adolescent self-report of temperament. It was predicted that adolescents from disadvantaged neighbourhoods would score lower on measures of Affiliation, Effortful Control and Surgency, and higher on measures of Negative Affectivity.

As hypothesised, neighbourhood disadvantage was associated with increased Negative Affectivity and decreased Effortful Control, controlling for gender. Gender was also found to significantly predict Effortful Control, with males scoring significantly higher on Effortful Control. Regression coefficients and standard errors can be found in Table 17 (below). Neighbourhood disadvantage was not found to be a significant predictor of Affiliation.

Neighbourhood disadvantage was found to be a significant predictor of Surgency, however the overall model was not significant. This was not considered a problem given the focus of the current thesis on individual predictors, rather than the predictive power of the overall model. However, as a further test, the linear regression was run again without gender as a predictor (Regression coefficients and standard errors can be found in Table 18). This is an approach recommended by Garson (2012), who suggests removing the most non-significant predictor (in this case gender) from the analysis when the overall model is not significant. When gender was taken out of the analysis, the overall model was found to be significant, and neighbourhood disadvantage remained a significant predictor of Surgency.
Table 17

Summary of Linear Regression (Controlling for Gender), with Neighbourhood Disadvantage Predicting EATQ-R Higher-order Temperament Factors

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Significance and model fit for final model</th>
<th>Predictors in model</th>
<th>Standardised beta (β)</th>
<th>t</th>
<th>p (1-tail)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Negative Affectivity</strong></td>
<td>F(2, 242) = 3.27, p &lt; .05, R² = .026, AR² = .018</td>
<td>Gender</td>
<td>-.036</td>
<td>-.57</td>
<td>.29</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>-.16</td>
<td>-2.49</td>
<td>.007</td>
</tr>
<tr>
<td><strong>Surgency</strong></td>
<td>F(2, 242) = 3.021, p = .057, R² = .024, AR² = .016</td>
<td>Gender</td>
<td>-.052</td>
<td>-.81</td>
<td>.29</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>.15</td>
<td>2.33</td>
<td>.012</td>
</tr>
<tr>
<td><strong>Effortful Control</strong></td>
<td>F(2, 242) = 7.44, p &lt; .01, R² = .058, AR² = .050</td>
<td>Gender</td>
<td>.14</td>
<td>2.28</td>
<td>.012</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>.19</td>
<td>3.088</td>
<td>.001</td>
</tr>
<tr>
<td><strong>Affiliation</strong></td>
<td>F(2, 242) = 1.45, p = .24, R² = .012, AR² = .004</td>
<td>Gender</td>
<td>.068</td>
<td>1.064</td>
<td>.095</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>.084</td>
<td>1.32</td>
<td>.14</td>
</tr>
</tbody>
</table>

Bolding indicates p < .05

Table 18

Summary of Linear Regression (Controlling for Gender), with Neighbourhood Disadvantage Predicting Surgency

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Significance and model fit for Final Model</th>
<th>Predictors in model</th>
<th>Standardised beta (β)</th>
<th>t</th>
<th>p (1-tail)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Surgency</strong></td>
<td>F(1, 243) = 5.39, p &lt; .05, R² = .15, AR² = .022</td>
<td>Neighbourhood Disadvantage</td>
<td>.15</td>
<td>2.32</td>
<td>.010</td>
</tr>
</tbody>
</table>


Bolding indicates $p < .05$

10.4 Neighbourhood disadvantage and frequency of maternal affective behaviours (rate per minute)

A series of linear regression analyses were conducted to test the relationship between neighbourhood disadvantage and the frequency of maternal affective behaviours (RPM). It was predicted that mothers from disadvantaged neighbourhoods would more frequently express aggression and dysphoria, and less frequently express positive emotion.

Contrary to all hypotheses, neighbourhood disadvantage was not associated with any significant differences in maternal interaction style (RPM) on either interaction task (PSI or EPI) (see Table 19). A detailed discussion of these results can be found in Chapter 14.2.4.
### Table 19

**Summary of Linear Regression (Controlling for Gender), with Neighbourhood disadvantage Predicting Maternal Interaction Style (RPM)**

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Significance and model fit for Final Model</th>
<th>Predictors model</th>
<th>Standardised beta (B)</th>
<th>t</th>
<th>p (1-tail)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysphoric DPE (EPI)</td>
<td>F(2, 242) = .251, p = .778, AR² = .006</td>
<td>Gender</td>
<td>-.038</td>
<td>-60</td>
<td>.55</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>.025</td>
<td>.39</td>
<td>.70</td>
</tr>
<tr>
<td>Aggressive DPE (EPI)</td>
<td>F(2, 242) = 1.023, p = .36, AR² = .00</td>
<td>Gender</td>
<td>-.053</td>
<td>-83</td>
<td>.41</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>.075</td>
<td>1.18</td>
<td>.241</td>
</tr>
<tr>
<td>Positive DPE (EPI)</td>
<td>F(2, 242) = .98, p = .38, AR² = .00</td>
<td>Gender</td>
<td>-.081</td>
<td>-1.26</td>
<td>.21</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>.04</td>
<td>.625</td>
<td>.53</td>
</tr>
<tr>
<td>Dysphoric DPE (PSI)</td>
<td>F(2, 242) = 1.02, p = .36, AR² = .00</td>
<td>Gender</td>
<td>-.055</td>
<td>-85</td>
<td>.40</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>.074</td>
<td>1.15</td>
<td>.25</td>
</tr>
<tr>
<td>Aggressive DPE (PSI)</td>
<td>F(2, 242) = 1.7, p = .185, AR² = .006</td>
<td>Gender</td>
<td>-.046</td>
<td>-72</td>
<td>.47</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>-.108</td>
<td>-1.69</td>
<td>.092</td>
</tr>
<tr>
<td>Positive DPE (PSI)</td>
<td>F(2, 242) = 2.39, p = .093, AR² = .011</td>
<td>Gender</td>
<td>-.074</td>
<td>-1.17</td>
<td>.24</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood</td>
<td>.12</td>
<td>1.86</td>
<td>.064</td>
</tr>
</tbody>
</table>
10.5 Neighbourhood disadvantage and duration of maternal affective behaviours (duration per episode)

A series of linear regression analyses were conducted to test the relationship between neighbourhood disadvantage and the duration of maternal affective behaviours (DPE). It was predicted that mothers from disadvantaged neighbourhoods would express aggression and dysphoria for a longer duration, and express positive emotion for a shorter duration.

Analyses indicated that neighbourhood disadvantage was a significant predictor of higher duration per episode of aggressive behaviour on both the PSI and EPI (See Table 20). It was also found to be a significant predictor of increased duration of dysphoric behaviour, and decreased duration of positive behaviour on the EPI. Neighbourhood disadvantage was not found to be a significant predictor of dysphoric or positive behaviour on the PSI.
Table 20

Summary of Linear Regression (Controlling for Gender), with Neighbourhood disadvantage Predicting Maternal Interaction Style (DPE)

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Significance and model fit for Final Model</th>
<th>Predictors in model</th>
<th>Standardised beta (B)</th>
<th>t</th>
<th>p (1-tail)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysphoric DPE (EPI)</td>
<td>F(2, 242) = 7.74, p = .001, AR² = .052</td>
<td>Gender</td>
<td>.005</td>
<td>.088</td>
<td>.47</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood</td>
<td>-.25</td>
<td>-3.93</td>
<td>.00</td>
</tr>
<tr>
<td>Aggressive DPE (EPI)</td>
<td>F(2, 242) = 3.83, p = .023, AR² = .023</td>
<td>Gender</td>
<td>.096</td>
<td>1.52</td>
<td>.065</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood</td>
<td>-.15</td>
<td>-2.35</td>
<td>.0095</td>
</tr>
<tr>
<td>Positive DPE (EPI)</td>
<td>F(2, 242) = 5.43, p = .005, AR² = .035</td>
<td>Gender</td>
<td>.093</td>
<td>1.48</td>
<td>.070</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood</td>
<td>-.19</td>
<td>-2.98</td>
<td>.0015</td>
</tr>
<tr>
<td>Dysphoric DPE (PSI)</td>
<td>F(2, 242) = 2.29, p = .104, AR² = .016</td>
<td>Gender</td>
<td>.16</td>
<td>2.083</td>
<td>.019</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood</td>
<td>-.040</td>
<td>-.51</td>
<td>.31</td>
</tr>
<tr>
<td>Aggressive DPE (PSI)</td>
<td>F(2, 242) = 8.97, p = .000, AR² = .061</td>
<td>Gender</td>
<td>.037</td>
<td>.59</td>
<td>.28</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood</td>
<td>-.26</td>
<td>-4.21</td>
<td>.00</td>
</tr>
<tr>
<td>Positive DPE (PSI)</td>
<td>F(2, 242) = 9.97, p = .000, AR² = .068</td>
<td>Gender</td>
<td>.27</td>
<td>4.38</td>
<td>.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood</td>
<td>-.059</td>
<td>-.96</td>
<td>.16</td>
</tr>
</tbody>
</table>
10.6 Analyses controlling for family SES

In order to provide a more stringent test of the effect of neighbourhood disadvantage independent of family SES, the previous analyses in which neighbourhood was found to be a significant predictor were reanalysed with the addition of family SES as a control variable. These analyses found that neighbourhood disadvantage remained a significant predictor in nine of the ten analyses (duration per episode of aggressive behaviour on the EPI was no longer significant) (See Table 21 for a summary of these analyses). This indicates that there is a significant effect of neighbourhood disadvantage on these outcome variables over and above the influence of family SES (which includes the shared influence of neighbourhood disadvantage and family SES). Surprisingly, compared to neighbourhood disadvantage, family SES was found to be a less consistent predictor of the outcome variables examined in these analyses (four out of ten). A detailed discussion of these results is provided in Chapter 14.2.6.

Table 21
Summary of Linear Regression (Full Sample, Controlling for Gender and individual SES), with Neighbourhood Disadvantage Predicting Internalising Symptoms, Temperament, Maternal Affective Behaviour and Stressful Life Events

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Significance and model fit for Final Model</th>
<th>Predictors in model</th>
<th>Standardised beta (B)</th>
<th>t</th>
<th>p (1-tail)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressive Symptoms (T1)</td>
<td>$F(3,242) = 3.691, p &lt; .05$, AR² = .053</td>
<td>Gender</td>
<td>-.16</td>
<td>-2.60</td>
<td>.005</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Family SES</td>
<td>-.020</td>
<td>-.29</td>
<td>.39</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>-.16</td>
<td>-2.35</td>
<td>.010</td>
</tr>
<tr>
<td>Anxious Symptoms (T1)</td>
<td>$F(3, 240) = 3.737, p &lt; .05$, AR² = .033</td>
<td>Gender</td>
<td>-.13</td>
<td>-2.08</td>
<td>.019</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Family SES</td>
<td>-.065</td>
<td>-.95</td>
<td>.17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neighbourhood disadvantage</td>
<td>-.13</td>
<td>-1.88</td>
<td>.030</td>
</tr>
<tr>
<td>Variable</td>
<td>Source of Variance</td>
<td>$F(3, 241)$</td>
<td>$p$</td>
<td>$AR^2$</td>
<td>$\beta$</td>
</tr>
<tr>
<td>-----------------------</td>
<td>--------------------</td>
<td>-------------</td>
<td>------</td>
<td>--------</td>
<td>-----------</td>
</tr>
<tr>
<td>Negative Affectivity</td>
<td>$\beta$</td>
<td>2.39</td>
<td>.069</td>
<td>.017</td>
<td>- .036</td>
</tr>
<tr>
<td></td>
<td>Family SES</td>
<td></td>
<td></td>
<td></td>
<td>-.080</td>
</tr>
<tr>
<td></td>
<td>Neighbourhood</td>
<td></td>
<td></td>
<td></td>
<td>-.12</td>
</tr>
<tr>
<td></td>
<td>disadvantage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgency</td>
<td>$\beta$</td>
<td>2.17</td>
<td>.092</td>
<td>.026</td>
<td>- .052</td>
</tr>
<tr>
<td></td>
<td>Family SES</td>
<td></td>
<td></td>
<td></td>
<td>.058</td>
</tr>
<tr>
<td></td>
<td>Neighbourhood</td>
<td></td>
<td></td>
<td></td>
<td>.12</td>
</tr>
<tr>
<td></td>
<td>disadvantage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Effortful Control</td>
<td>$\beta$</td>
<td>7.06</td>
<td>.001</td>
<td>.081</td>
<td>1.14</td>
</tr>
<tr>
<td></td>
<td>Family SES</td>
<td></td>
<td></td>
<td></td>
<td>.18</td>
</tr>
<tr>
<td></td>
<td>Neighbourhood</td>
<td></td>
<td></td>
<td></td>
<td>.11</td>
</tr>
<tr>
<td></td>
<td>disadvantage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stressful life events</td>
<td>$\beta$</td>
<td>3.68</td>
<td>.05</td>
<td>.028</td>
<td>.083</td>
</tr>
<tr>
<td></td>
<td>Family SES</td>
<td></td>
<td></td>
<td></td>
<td>-.079</td>
</tr>
<tr>
<td></td>
<td>Neighbourhood</td>
<td></td>
<td></td>
<td></td>
<td>-.15</td>
</tr>
<tr>
<td></td>
<td>disadvantage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dysphoric DPE – EP</td>
<td>$\beta$</td>
<td>7.966</td>
<td>&lt; .001</td>
<td>.09</td>
<td>-.001</td>
</tr>
<tr>
<td></td>
<td>Family SES</td>
<td></td>
<td></td>
<td></td>
<td>-.20</td>
</tr>
<tr>
<td></td>
<td>Neighbourhood</td>
<td></td>
<td></td>
<td></td>
<td>-.16</td>
</tr>
<tr>
<td></td>
<td>disadvantage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aggressive DPE – EP</td>
<td>$\beta$</td>
<td>3.768</td>
<td>&lt; .05</td>
<td></td>
<td>.092</td>
</tr>
<tr>
<td>Variable</td>
<td>coeff</td>
<td>SE</td>
<td>p-value</td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------</td>
<td>-------</td>
<td>----</td>
<td>---------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family SES</td>
<td>-0.13</td>
<td>1.91</td>
<td>0.029</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neighbourhood disadvantage</td>
<td>-0.098</td>
<td>1.43</td>
<td>0.077</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Positive DPE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>0.093</td>
<td>1.50</td>
<td>0.14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family SES</td>
<td>0.13</td>
<td>1.88</td>
<td>0.061</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neighbourhood disadvantage</td>
<td>-0.26</td>
<td>-3.92</td>
<td>0.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aggressive DPE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>0.034</td>
<td>0.56</td>
<td>0.58</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family SES</td>
<td>0.073</td>
<td>1.091</td>
<td>0.27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neighbourhood disadvantage</td>
<td>-0.29</td>
<td>-4.27</td>
<td>0.00</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
10.7 Summary: Factors associated with Neighbourhood disadvantage.

Analyses revealed that families from disadvantaged neighbourhoods differ from those from less disadvantaged neighbourhoods in several key ways (see figure 16 for a representation of these differences). They were more likely to report a greater number of stressful life events, and depressive and anxious symptoms. They were also more likely to score higher on measures of Negative Affectivity, and lower on measures of Surgency and Effortful control. Interestingly, although mothers from disadvantaged neighbourhoods did not display various affective behaviours more or less frequently. However, they were more likely to display aggressive and dysphoric behaviour for longer periods, and positive behaviour for shorter periods.

It is also worth noting that gender did not moderate any of the relationships examined. Reasons for this will be discussed in Chapter 14.2.5.

<table>
<thead>
<tr>
<th>Neighbourhood disadvantage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adolescent:</strong></td>
</tr>
<tr>
<td>+ Depressive symptoms (baseline)</td>
</tr>
<tr>
<td>+ Anxious symptoms (baseline)</td>
</tr>
<tr>
<td>+ Negative Affectivity</td>
</tr>
<tr>
<td>- Effortful Control</td>
</tr>
<tr>
<td>- Surgency</td>
</tr>
<tr>
<td>+ Stressful Life events</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Maternal:</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>+ aggressive behaviour (DPE) on the PSI and EPI</td>
</tr>
<tr>
<td>+ dysphoric behaviour (DPE) on the EPI</td>
</tr>
<tr>
<td>- positive behaviour (DPE) on the EPI</td>
</tr>
</tbody>
</table>
Figure 16: Summary of characteristics that are significantly associated with neighbourhood disadvantage. ‘+’ indicates higher levels, ‘-’ indicates lower levels

It is possible that these some of these differences may mediate the relationship between Neighbourhood disadvantage and internalising symptoms in adolescents. I investigated this possibility in the following three chapters, which outline analyses of the potential mediating role of stressful life events (Chapter 11), temperament (Chapter 12), and maternal affective behaviour (Chapter 13) between neighbourhood disadvantage and adolescent internalising symptoms. Note that, despite no significant direct relationship found between neighbourhood disadvantage and change in internalising symptoms between T1 and T3, analyses will still be conducted to explore whether any factors mediate the relationship between these two variables (for reasons outlined in Chapter 9.1.3).
CHAPTER 11

STRESSFUL LIFE EVENTS AS A MEDIATOR BETWEEN NEIGHBOURHOOD DISADVANTAGE AND SYMPTOMS OF ANXIETY AND DEPRESSION

Previous analyses indicate that neighbourhood disadvantage is associated with significantly higher rates of stressful life events in adolescents. The following chapter outlines the mediational analyses undertaken to explore whether stressful life events mediate the relationship between neighbourhood disadvantage and symptoms of anxiety and depression. Section 11.1 outlines the analyses concerning neighbourhood disadvantage, stressful life events and depressive and anxious symptoms at T1. Section 11.2 outlines the analyses concerning neighbourhood disadvantage stressful life events and change in depressive and anxious symptoms from T1 to T3.

11.1 Stressful life events as a mediator between neighbourhood disadvantage and symptoms of depression and anxiety at T1

A series of hierarchical and multiple regression analyses (controlling for adolescent gender) and INDIRECT (an interactive mediation analytic tool utilized for all mediation tests) were conducted to test whether stressful life events mediated the relationship between neighbourhood disadvantage and depressive and anxious symptoms. No evidence for gender related moderated mediation was detected, thus the moderation term was removed from the final analyses reported. Analysis revealed that stressful life events mediated the relationship between neighbourhood disadvantage and depressive symptoms at T1 (figure 17). Higher levels of neighbourhood disadvantage predicted higher levels of stressful life events, which in turn predicted greater depressive symptoms.
Stressful life events significantly mediated the relationship between neighbourhood disadvantage and depressive symptoms. CI for indirect path (-.0090 to -.00090) $F(4, 240) = 4.88, p < .05, \Delta R^2 = .046$

Figure 17: Diagram showing significant mediation of stressful life events in the relationship between neighbourhood disadvantage and depressive symptoms at T1 (controlling for gender). The standardised regression coefficient between neighbourhood disadvantage and depressive symptoms controlling for Stress is in parentheses. *p < .05, 2-tail.

Analysis revealed that stressful life events mediated the relationship between neighbourhood disadvantage and anxiety (T1) (figure 18). Higher levels of neighbourhood disadvantage predicted a greater number of stressful life events, which in turn predicted greater symptoms of anxiety.
Stressful life events significantly mediated the relationship between neighbourhood disadvantage and symptoms of anxiety. CI for indirect path (-.010 to -.0020) \( F(4, 238) = 6.19, p < .05, \) AR2 = .06

Figure 18: Diagram showing significant mediation of stressful life events in the relationship between neighbourhood disadvantage and anxiety at T1 (controlling for gender). The standardised regression coefficient between neighbourhood disadvantage and anxiety controlling for Stressful Life Events is in parentheses. *p < .05, 2-tail.

11.2 Stressful life events and change in depressive and anxious symptoms

A series of hierarchical and multiple regression analyses (controlling for adolescent gender) and INDIRECT were conducted to test whether the relationship between neighbourhood disadvantage and change in depressive symptoms or anxiety was partially mediated by stressful life events.

Stressful life events were found to significantly mediate the relationship between neighbourhood disadvantage and change in depressive symptoms (figure 19). Higher levels of neighbourhood disadvantage predicted higher stressful life events, which in turn predicted a lower reduction in symptoms of depression.
Stressful life events significantly mediated the relationship between neighbourhood disadvantage and change in depressive symptoms. CI for indirect path (-.010 to -.00050). F(4, 240) = 9.98, p < .05, AR2 = .11.

Figure 19: Diagram showing significant mediation of stressful life events in the relationship between neighbourhood disadvantage and depressive symptoms at T3 (controlling for gender and symptoms of depression at T1). The standardised regression coefficient between neighbourhood disadvantage and depressive symptoms controlling for stressful life events is in parentheses. *p < .05, 2-tail.

Stressful life events were found to significantly mediate the relationship between neighbourhood disadvantage and change in anxious symptoms (figure 20). Higher levels of neighbourhood disadvantage predicted higher levels of stressful life events, which in turn predicted a greater increase in symptoms of anxiety.
Stressful life events significantly mediated the relationship between neighbourhood disadvantage and change in symptoms of anxiety. CI for indirect path (-.01 to -.001) $F(4, 238) = 15.28, p < .05$, AR2 = .19

Figure 20: Diagram showing significant mediation of stressful life events in the relationship between neighbourhood disadvantage and anxiety at T3 (controlling for gender and symptoms of anxiety at T1). The standardised regression coefficient between neighbourhood disadvantage controlling for stressful life events is in parentheses. *p < .05, 2-tail.

11.3 Mediating analyses of significant parent effects controlling for family SES

A series of additional mediational analyses were conducted to determine whether the significant results described in the current chapter still remained after controlling for family SES. After controlling for SES and gender, Stress was still found to be a significant mediator for the relationship between neighbourhood disadvantage and depression and anxiety at T1 (see Table 22). However, stress was no longer found to significantly mediate the relationship between neighbourhood disadvantage and change in symptoms of depression or anxiety, although it should be noted that there was still a strong trend towards significance for these relationships.
Table 22

Regression Parameter Estimates (B) for Mediation Analyses with Neighbourhood as the Independent Variable, Stress as the Mediator, and Symptoms of Internalising Disorders as the Dependent Variable, Controlling for Family SES and Gender

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Significance and model fit</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total effect (c)</th>
<th>Direct effect (c')</th>
<th>95% Confidence interval for indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression (T1)</td>
<td>$F(4, 240) = 3.68, p &lt; .05$, $\text{AR}^2 = .04$</td>
<td>-.012*</td>
<td>.25*</td>
<td>-.018</td>
<td>-.015</td>
<td>(.0080 to .0000)</td>
</tr>
<tr>
<td>Depression (change)</td>
<td>$F(5, 239) = 14.16, p &lt; .05$, $\text{AR}^2 = .21$</td>
<td>-.010</td>
<td>.35*</td>
<td>-.0077</td>
<td>-.0041</td>
<td>(-.010 to .00)</td>
</tr>
<tr>
<td>Anxiety (T1)</td>
<td>$F(4, 238) = 5.06, p &lt; .05$, $\text{AR}^2 = .06$</td>
<td>-.012*</td>
<td>.30*</td>
<td>-.016</td>
<td>-.01</td>
<td>(-.0080 to -.0007)</td>
</tr>
<tr>
<td>Anxiety (change)</td>
<td>$F(5, 237) = 13.03, p &lt; .05$, $\text{AR}^2 = .20$</td>
<td>-.010</td>
<td>.50*</td>
<td>-.0025</td>
<td>-.0077</td>
<td>(-.0014 to .00)</td>
</tr>
</tbody>
</table>

* = p < .05, 2-tail

11.4 Summary of the mediating role of stressful life events in the relationship of neighbourhood disadvantage and internalising symptoms

The aim of these analyses was to determine whether stressful life events mediated the relationship between neighbourhood disadvantage and internalising symptoms. Most hypotheses were supported: stressful life events mediated the relationship between neighbourhood disadvantage and (1) depressive symptoms at T1 (2) change in depressive symptoms, (3) anxious symptoms at T1 and (4) change in anxious symptoms. Mediating effects involving symptoms at T1 held up even when controlling for family SES, indicating a unique effect of neighbourhood disadvantage. These results indicate a possible persistent mediating role of stressful life events during both early and later adolescence.
CHAPTER 12
ADOLESCENT TEMPERAMENT AS A MEDIATOR BETWEEN NEIGHBOURHOOD DISADVANTAGE AND SYMPTOMS OF ANXIETY AND DEPRESSION

Previous analyses indicate that neighbourhood disadvantage is associated with significant differences in the temperament dimensions of Negative Affectivity, Surgency, and Effortful Control. The following chapter outlines the mediational analyses undertaken to explore whether these variables mediated the relationship between neighbourhood disadvantage and symptoms of anxiety and depression. Section 12.1 outlines the analyses concerning neighbourhood disadvantage, temperament and depressive symptoms at T1. Section 12.2 outlines the analyses concerning neighbourhood disadvantage, temperament and anxious symptoms at baseline. Section 12.3 outlines the analyses concerning neighbourhood disadvantage, temperament and change in depressive and anxious symptoms. Section 12.4 summarises mediating analyses of significant temperament effects controlling for family SES.

12.1 Adolescent temperament as a mediator between neighbourhood disadvantage and symptoms of depression at baseline

A series of hierarchical and multiple regression analyses (controlling for adolescent gender) and INDIRECT were conducted to test whether the temperament dimensions Negative Affectivity, Surgency, and Effortful Control mediated the relationship between neighbourhood disadvantage and depressive symptoms. A summary of each of these analyses will be provided in turn. Note that all mediational analyses initially tested for the possibility that gender moderated each potential mediating pathway. No evidence for gender related moderated mediation was detected, thus the moderation term was removed from the final analyses reported.

12.1.1 Negative Affectivity
Analyses revealed that Negative Affectivity mediated the relationship between neighbourhood disadvantage and depressive symptoms at T1 (figure 21). Higher levels of neighbourhood disadvantage predicted higher levels of Negative Affectivity, which in turn predicted higher levels of depressive symptoms.

Figure 21: Diagram showing significant mediation of Negative Affectivity in the relationship between neighbourhood disadvantage and depressive symptoms at T1 (controlling for gender). The standardised regression coefficient between neighbourhood disadvantage and depressive symptoms, controlling for Negative Affectivity is in parentheses. *p < .05, 2-tail.

12.1.2 Surgency

Analysis revealed that Surgency also mediated the relationship between neighbourhood disadvantage and depressive symptoms at T1 (figure 22). Higher levels of neighbourhood disadvantage predicted lower levels of Surgency, which in turn predicted higher levels of depressive symptoms.
12.1.3 Effortful Control

Analysis revealed that Effortful Control mediated the relationship between neighbourhood disadvantage and depressive symptoms at T1 (figure 23). Higher levels of neighbourhood disadvantage predicted lower levels of Effortful Control, which in turn predicted higher levels of depressive symptoms.
Effortful Control significantly mediated the relationship between neighbourhood disadvantage and depressive symptoms. CI for indirect path (-.025 to -.0041) $F(3, 241) = 31.70, p < .05$, AR2 = .27

12.2 Adolescent temperament as a mediator between neighbourhood disadvantage and symptoms of anxiety at baseline

A series of hierarchical and multiple regression analyses (controlling for adolescent gender) and INDIRECT were conducted to test whether the temperament dimensions Negative Affectivity, Surgency, and Effortful Control mediated the relationship between neighbourhood disadvantage and anxiety at T1.

12.2.1 Negative Affectivity

Analysis revealed that Negative Affectivity mediated the relationship between neighbourhood disadvantage and anxiety at T1 (figure 24). Higher levels of neighbourhood disadvantage predicted higher levels of Negative Affectivity, which in turn predicted higher symptoms of anxiety.
12.2.2 Surgency

Analysis revealed that Surgency mediated the relationship between neighbourhood disadvantage and anxiety at T1 (figure 25). Higher levels of neighbourhood disadvantage predicted lower levels of Surgency, which in turn predicted higher symptoms of anxiety.
Surgency significantly mediated the relationship between neighbourhood disadvantage and symptoms of anxiety. CI for indirect path (-.010 to -.0011) \(F(3, 241) = 8.20, p < .05, \text{AR2} = .082\)

12.2.3 Effortful Control

Analysis revealed that Effortful Control mediated the relationship between Neighbourhood disadvantage and Anxiety at T1 (figure 26). Higher levels of neighbourhood disadvantage predicted lower levels of Effortful Control, which in turn predicted higher symptoms of anxiety.
Effortful Control significantly mediated the relationship between neighbourhood disadvantage and symptoms of anxiety. CI for indirect path (-.016 to -.0025) $F(3, 241) = 13.60, p < .05$, $AR^2 = .14$

Figure 26: Diagram showing significant mediation of Effortful Control in the relationship between neighbourhood disadvantage and anxious symptoms at T1 (controlling for gender). The standardised regression coefficient between neighbourhood disadvantage and anxiety controlling for Effortful Control is in parentheses. *p < .05, 2-tail.

12.3 Temperament and change in depressive and anxious symptoms

While all temperament dimensions examined were found to mediate the relationship between neighbourhood disadvantage and symptoms of depression and anxiety at T1, no temperament dimensions mediated the change in depressive or anxious symptoms from T1 to T3. Table 23 summarises mediational analyses pertaining to neighbourhood disadvantage, temperament and change in depressive symptoms. Table 24 summarises mediational analyses pertaining to neighbourhood disadvantage, temperament and change in anxiety.
Table 23

Regression Parameter Estimates (B) for Mediational Analyses with Neighbourhood Disadvantage as the Independent Variable, Temperament as the Mediator, and Depressive Symptoms (T3) as the Dependent Variable, Controlling for Depressive Symptoms at T1 and Gender

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Significance and model fit</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total effect (c)</th>
<th>Direct effect (c')</th>
<th>95% Confidence interval for indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative Affectivity</td>
<td>$F(3, 241) = 15.30, p &lt; .05, \text{AR}^2 = .19$</td>
<td>-.0010</td>
<td>2.030*</td>
<td>-.0076</td>
<td>-.0058</td>
<td>(-.0058 to .0040)</td>
</tr>
<tr>
<td>Surgency</td>
<td>$F(3, 241) = 17.10, p &lt; .05, \text{AR}^2 = .21$</td>
<td>.0010</td>
<td>-2.85*</td>
<td>-.0097</td>
<td>-.0068</td>
<td>(-.0070 to .0010)</td>
</tr>
<tr>
<td>Effortful Control</td>
<td>$F(3, 241) = 12.71, p &lt; .05, \text{AR}^2 = .16$</td>
<td>.0011*</td>
<td>-.87</td>
<td>-.0097</td>
<td>-.0088</td>
<td>(-.0042 to .0005)</td>
</tr>
</tbody>
</table>

* = p < .05, 2-tail

Table 24

Regression Parameter Estimates (B) for Mediational Analyses with Neighbourhood Disadvantage as the Independent Variable, Temperament as the Mediator, and Anxiety (T3) as the Dependent Variable, Controlling for Depressive Symptoms at T1 and Gender

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Significance and model fit</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total effect (c)</th>
<th>Direct effect (c')</th>
<th>95% Confidence interval for indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative Affectivity</td>
<td>$F(3, 236) = 6.38, p &lt; .05, \text{AR}^2 = .082$</td>
<td>-.0012</td>
<td>.36*</td>
<td>-.0019</td>
<td>-.0015</td>
<td>(-.0028 to .00060)</td>
</tr>
<tr>
<td>Surgency</td>
<td>$F(3, 236) = 6.74, p &lt; .05, \text{AR}^2 = .087$</td>
<td>.0012*</td>
<td>-.85</td>
<td>-.0019</td>
<td>-.0010</td>
<td>(-.0039 to .00020)</td>
</tr>
<tr>
<td>Effortful Control</td>
<td>$F(3, 236) = 6.30, p &lt; .05, \text{AR}^2 = .080$</td>
<td>.0012*</td>
<td>.22</td>
<td>-.0019</td>
<td>-.57</td>
<td>(-.0018 to .0027)</td>
</tr>
</tbody>
</table>
12.4 Mediating analyses of significant temperament effects controlling for family SES

A series of additional rigorous mediational analyses were conducted to determine whether the significant mediating pathways described in the current chapter still remained after controlling for family SES.

After controlling for SES and gender, no temperament dimensions were found to significantly mediate the relationship between neighbourhood disadvantage and depressive symptoms at T1 (see Table 25). Surgency was still found to significantly mediate the relationship between neighbourhood disadvantage and anxiety at T1, but Negative Affectivity and Effortful Control were no longer found to significantly mediate this relationship (see Table 26) (although there was a strong trend towards significance for Effortful Control).

Table 25
Regression Parameter Estimates (B) for Mediation Analyses with Neighbourhood as the Independent Variable, Temperament as the Mediator, and Depressive Symptoms (T1) as the Dependent Variable, Controlling for Individual SES and Gender

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Significance and model fit</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total effect (c)</th>
<th>Direct effect (c')</th>
<th>95% Confidence interval for indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative Affectivity</td>
<td>$F(4, 240) = 21.55, p &lt; .05$, $R^2 = .25$</td>
<td>-.0015</td>
<td>5.62*</td>
<td>-.018</td>
<td>-.009</td>
<td>(.019 to .00040)</td>
</tr>
<tr>
<td>Surgency</td>
<td>$F(4, 240) = 7.60, p &lt; .05$, $R^2 = .20$</td>
<td>.0012</td>
<td>-3.94*</td>
<td>-.018</td>
<td>-.013</td>
<td>(.011 to .0040)</td>
</tr>
<tr>
<td>Effortful Control</td>
<td>$F(4, 240) = 23.90, p &lt; .05$, $R^2 = .27$</td>
<td>.0011</td>
<td>-8.077*</td>
<td>-.018</td>
<td>-.0086</td>
<td>(.019 to .00040)</td>
</tr>
</tbody>
</table>

* = $p < .05$, 2-tail
Table 26

Regression Parameter Estimates (B) for MediationAnalyses with Neighbourhood as the Independent Variable, Temperament as the Mediator, and Anxiety (T1) as the Dependent Variable, Controlling for Individual SES and Gender

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Significance and model fit</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total effect (c)</th>
<th>Direct effect (c')</th>
<th>95% Confidence interval for indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative Affectivity</td>
<td>$F(4, 238) = 15.60, p &lt; .05, AR^2 = .20$</td>
<td>-.0016*</td>
<td>4.48*</td>
<td>-.016</td>
<td>-.0093</td>
<td>(-.016 to .00050)</td>
</tr>
<tr>
<td>Surgency</td>
<td>$F(4, 238) = 6.30, p &lt; .05, AR^2 = .080$</td>
<td>.0012</td>
<td>-3.04*</td>
<td>-.016</td>
<td>-.013</td>
<td>(-.0090 to -.00030)*</td>
</tr>
<tr>
<td>Effortful Control</td>
<td>$F(4, 238) = 12.71, p &lt; .05, AR^2 = .16$</td>
<td>.0011</td>
<td>-4.86*</td>
<td>-.016</td>
<td>-.011</td>
<td>(-.013 to .00000)</td>
</tr>
</tbody>
</table>

* = $p < .05$, 2-tail

12.4 Summary of findings regarding temperament as a mediator between neighbourhood disadvantage and internalising symptoms

The aim of these analyses was to determine whether temperament was a mediator of the relationship between neighbourhood disadvantage and internalising symptoms. Hypotheses were supported regarding the mediating role of all three temperament dimensions examined (Negative Affectivity, Surgency and Effortful Control) and internalising symptoms at T1. Most of these mediating relationships did not remain significant with addition of family SES as a control variable. Analysis also failed to find a significant mediating role of temperament in the relationship between neighbourhood disadvantage and change in depressive symptoms or anxiety from T1 to T3. In summary, temperament appears to mediate internalising symptoms during early adolescence, but not the change in symptoms from early to later adolescence.
CHAPTER 13
MATERNAL AFFECTIVE BEHAVIOUR AS A MEDIATOR BETWEEN NEIGHBOURHOOD AND INTERNALISING SYMPTOMS

Previous analyses indicated that neighbourhood disadvantage was associated with significant differences in the duration per episode (but not frequency) of mother’s affective behaviour. The following chapter outlines the mediational analyses undertaken to explore whether maternal effective behaviour (DPE) mediated the relationship between neighbourhood disadvantage and symptoms of anxiety and depression. Section 13.1 outlines the analyses concerning neighbourhood disadvantage, parent interaction and depression and anxiety at T1. Section 13.2 outlines the analyses concerning neighbourhood disadvantage, parent interaction style and change in depressive and anxious symptoms.

13.1 Parent interaction style as a mediator between neighbourhood disadvantage and symptoms of depression and anxiety at T1

A series of hierarchical and multiple regression analyses (controlling for adolescent gender) and INDIRECT were conducted to test whether the relationship between neighbourhood disadvantage and depression or anxiety at T1 was partially mediated by DPE of parent affective behaviours (aggressive, dysphoric and positive) on either interaction task (PSI and EPI). Contrary to hypotheses, no affective behaviour styles were found to mediate these relationships. For results of these mediation analyses, see Table 27 (depressive symptoms) and Table 28 (symptoms of anxiety).
Table 27
Regression Parameter Estimates (β) for Mediation Analyses with Neighbourhood as the Independent Variable, Parent Interaction Style as the Mediator, and Depressive symptoms (T1) as the Dependent Variable, Controlling for Gender

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Significance and model fit</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total effect (c)</th>
<th>Direct effect (c’)</th>
<th>95% Confidence interval for indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal Aversive (DPE) during EPI</td>
<td>$F(3, 240) = 3.60, p &lt; .05, AR^2 = .031$</td>
<td>-.0046*</td>
<td>.34</td>
<td>-.020*</td>
<td>-.018*</td>
<td>(-.0060 to .0014)</td>
</tr>
<tr>
<td>Maternal Aversive (DPE) during PSI</td>
<td>$F(3, 240) = 4.065, p &lt; .05, AR^2 = .036$</td>
<td>-.029*</td>
<td>.013</td>
<td>-.020*</td>
<td>-.016</td>
<td>(-.0096 to .0030)</td>
</tr>
<tr>
<td>Maternal Dysphoria (DPE) during EPI</td>
<td>$F(3, 240) = 3.17, p &lt; .05, AR^2 = .026$</td>
<td>-.0078*</td>
<td>-.13</td>
<td>-.020*</td>
<td>-.021*</td>
<td>(-.0029 to .0058)</td>
</tr>
<tr>
<td>Maternal Dysphoria (DPE) during PSI</td>
<td>$F(3, 240) = 3.52, p &lt; .05, AR^2 = .022$</td>
<td>-.0067*</td>
<td>.049</td>
<td>-.020*</td>
<td>-.023*</td>
<td>(-.0031 to .0060)</td>
</tr>
<tr>
<td>Maternal Positive (DPE) during EPI</td>
<td>$F(3, 240) = 3.13, p &lt; .05, AR^2 = .026$</td>
<td>-.0085*</td>
<td>.083</td>
<td>-.020*</td>
<td>-.019*</td>
<td>(-.0060 to .0041)</td>
</tr>
<tr>
<td>Maternal Positive (DPE) during PSI</td>
<td>$F(3, 240) = 3.2, p &lt; .05, AR^2 = .026$</td>
<td>-.0024</td>
<td>.16</td>
<td>-.020*</td>
<td>-.019*</td>
<td>(-.0041 to .0024)</td>
</tr>
</tbody>
</table>

* = p < .05, 2-tail
Table 28

Regression Parameter Estimates (B) for Mediation Analyses with Neighbourhood as the independent Variable, Parent Interaction Style as the Mediator, and Symptoms of Anxiety (T1) as the Dependent Variable, Controlling for Gender

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Significance model fit</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total effect (c)</th>
<th>Direct effect (c')</th>
<th>95% Confidence interval for indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal Aversive (DPE) during EPI</td>
<td>$F(3, 240) = 2.55, p &gt; .05, AR^2 = .019$</td>
<td>-.0046*</td>
<td>.22</td>
<td>-.015</td>
<td>-.014</td>
<td>(-.0044 to .00090)</td>
</tr>
<tr>
<td>Maternal Aversive (DPE) during PSI</td>
<td>$F(3, 240) = 2.35, p &gt; .05, AR^2 = .016$</td>
<td>-.029*</td>
<td>.017</td>
<td>-.015</td>
<td>-.015</td>
<td>(-.0060 to .0046)</td>
</tr>
<tr>
<td>Maternal Dysphoria (DPE) during EPI</td>
<td>$F(3, 240) = 2.65, p &lt; .05, AR^2 = .020$</td>
<td>-.0078*</td>
<td>-.26</td>
<td>-.015</td>
<td>-.017</td>
<td>(-.0015 to .0068)</td>
</tr>
<tr>
<td>Maternal Dysphoria (DPE) during PSI</td>
<td>$F(3, 240) = 3.52, p &lt; .05, AR^2 = .21$</td>
<td>-.0061*</td>
<td>-.054</td>
<td>-.015</td>
<td>-.015</td>
<td>(.0011 to .0021)</td>
</tr>
<tr>
<td>Maternal Positive (DPE) during EPI</td>
<td>$F(3, 240) = 2.65, p &lt; .05, AR^2 = .020$</td>
<td>-.0078*</td>
<td>-.26*</td>
<td>-.015</td>
<td>-.017</td>
<td>(.0014 to .0068)</td>
</tr>
<tr>
<td>Maternal Positive (DPE) during PSI</td>
<td>$F(3, 240) = 2.81, p &lt; .05, AR^2 = .022$</td>
<td>-.0024</td>
<td>.32</td>
<td>-.015</td>
<td>-.014</td>
<td>(.0054 to .00050)</td>
</tr>
</tbody>
</table>

* = p < .05, 2-tail

14.2 Maternal interaction style and change in depressive and anxious symptoms

A series of hierarchical and multiple regression analyses (controlling for adolescent gender and baseline symptomatology) and INDIRECT were conducted to test whether the relationship between neighbourhood disadvantage and change in depressive or anxious symptoms was partially mediated by maternal affective behaviour on either interaction task.
DPE of Maternal Aggression during the PSI was found to significantly mediate the relationship between neighbourhood disadvantage and change in depressive symptoms (figure 27). Higher levels of neighbourhood disadvantage predicted longer duration of maternal aggression during the PSI, which in turn predicted less reduction in symptoms of depression.

Figure 27: Diagram showing significant mediation of Aggression (DPE) on the PSI task significantly mediated the relationship between neighbourhood disadvantage and depressive symptoms. CI for indirect path (-.0084 to -.0010) $F(4, 240) = 13.99$, $p < .05$, $\text{AR}^2 = .18$

DPE of Maternal Aggression during the EPI was also found to significantly mediate the relationship between neighbourhood disadvantage and change in depressive symptoms (figure 28). Higher levels of neighbourhood disadvantage predicted longer duration of maternal aggression during the EPI, which in turn predicted a lower reduction in symptoms of depression.
Aggression (DPE) on the EPI significantly mediated the relationship between neighbourhood disadvantage and change in depressive symptoms. CI for indirect path (-.0090 to -.00020) $F(4, 240) = 17.50, p < .05$, $AR^2 = .21$

![Diagram](image)

Figure 28: Diagram showing significant mediation of Aggression (DPE) on the EPI in the relationship between neighbourhood disadvantage and depressive symptoms at T3 (controlling for gender and baseline symptoms of depression). The standardised regression coefficient between neighbourhood disadvantage and depressive symptoms controlling for maternal aggression on the EPI (DPE) is in parentheses. *p < .05, 2-tail.

DPE of Maternal Aggression during the PSI was found to significantly mediate the relationship between neighbourhood disadvantage and change in anxious symptoms (figure 29). Higher levels of neighbourhood disadvantage predicted longer duration of maternal aggression during the PSI, which in turn predicted less reduction in symptoms of anxiety.
Figure 29: Diagram showing significant mediation of maternal aggression (DPE) on the PSI in the relationship between neighbourhood disadvantage and change in symptoms of anxiety. CI for indirect path (-.010 to -.00050) $F(4, 240) = 7.10$, $p < .05$, $AR^2 = .090$

Neighbourhood Disadvantage  \[ \rightarrow \] Aggression (DPE) during PSI  \[ \rightarrow \] Anxiety (change)  \[ \rightarrow \] -0.029*  \[ \rightarrow \] -0.0028 (-0.0011)  \[ \rightarrow \] .14*  

Aggression (DPE) during the EPI was found to significantly mediate the relationship between neighbourhood disadvantage and change in symptoms of anxiety (figure 30). Higher levels of neighbourhood disadvantage predicted longer duration of maternal aggression during the EPI, which in turn predicted a lower reduction in symptoms of anxiety.
Aggression (DPE) on the EPI significantly mediated the relationship between neighbourhood disadvantage and change in symptoms of anxiety. CI for indirect path (-.0089 to -.00040) $F(4, 238) = 8.64$, $p < .05$, $AR^2 = .11$

No other maternal affective behaviours (DPE) were found to mediate the relationship between neighbourhood disadvantage and change in depression or anxiety. Table 29 (change in depression) and Table 30 (change in anxiety) summarises these analyses.

Figure 30: Diagram showing significant mediation of maternal aggression (DPE) on the EPI in the relationship between neighbourhood disadvantage and anxious symptoms at T3 (controlling for gender and baseline symptoms of anxiety). The standardised regression coefficient between neighbourhood disadvantage and anxiety controlling for maternal aggression (DPE) on the EPI is in parentheses. *$p < .05$, 2-tail.*
Table 29

Regression Parameter Estimates (B) for Mediation Analyses with Neighbourhood as the Independent Variable, Maternal Affective Behaviour as the Mediator, and Depression (T3) as the Dependent Variable, Controlling for Depression at T1 and Gender

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Significance and model fit</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total effect (c)</th>
<th>Direct effect (c')</th>
<th>95% Confidence interval for indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal Dysphoria (DPE) during EPI</td>
<td>F(4, 240) = 12.80, p &lt; .05, AR² = .16</td>
<td>-.0011</td>
<td>.042</td>
<td>-.0097</td>
<td>-.0087</td>
<td>(-.00040 to .0015)</td>
</tr>
<tr>
<td>Maternal Dysphoria (DPE) during PSI</td>
<td>F(3, 240) = 10.30, p &lt; .05, AR² = .19</td>
<td>.0017</td>
<td>-.096</td>
<td>-.0097</td>
<td>-.0065</td>
<td>(-.00060 to .0026)</td>
</tr>
<tr>
<td>Maternal Positive (DPE) during EPI</td>
<td>F(3, 240) = 8.80, p &lt; .05, AR² = .11</td>
<td>-.0022</td>
<td>-.45*</td>
<td>-.0097</td>
<td>-.0014</td>
<td>(-.00040 to .0044)</td>
</tr>
<tr>
<td>Maternal Positive (DPE) during PSI</td>
<td>F(3, 240) = 13.68, p &lt; .05, AR² = .17</td>
<td>.0081*</td>
<td>-.35*</td>
<td>-.0097</td>
<td>-.013</td>
<td>(-.00010 to .0068)</td>
</tr>
</tbody>
</table>

* = p < .05, 2-tail
Table 30
Regression Parameter Estimates (B) for Mediational Analyses with Neighbourhood as the Independent Variable, Maternal Affective Behaviour as the Mediator, and Anxiety (T3) as the Dependent Variable, Controlling for Depression at T1 and Gender

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Significance and model fit</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total effect (c)</th>
<th>Direct effect (c')</th>
<th>95% Confidence interval for indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal Dysphoria (DPE) during Event Planning task</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>F(4, 238) = 5.78, p &lt; .05, AR² = .070</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal Dysphoria (DPE) during Problem Solving task</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>F(4, 238) = 17.10, p &lt; .05, AR² = .21</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal Positive (DPE) during EPI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>F(3, 240) = 8.80, p &lt; .05, AR² = .11</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal Positive (DPE) during PSI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>F(3, 240) = 8.00, p &lt; .05, AR² = .10</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = p < .05, 2-tail

14.3 Mediating analyses of significant parent effects controlling for family SES

A series of additional rigorous mediational analyses were conducted to determine whether the significant results described in the current chapter still remained after controlling for family SES. After controlling for SES and gender, maternal aggression (DPE) during the PSI was still found to be a significant mediator for the relationship between neighbourhood disadvantage and change in depressive and anxious symptoms (see Table 31 and Table 32). However, Maternal aggression (DPE) during the EPI was no longer found to significantly mediate the relationship
between neighbourhood disadvantage and change in anxious and depressive symptoms. It should be noted that there was a strong trend towards significance for this relationship.

Table 31
Regression Parameter Estimates (B) for Mediation Analyses with Neighbourhood as the Independent Variable, Maternal Affective Behaviour as the Mediator, and Depressive Symptoms (Change) as the Dependent Variable, Controlling for Family SES, Depressive Symptoms at T1 and Gender

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Significance and model fit</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total effect (c)</th>
<th>Direct effect (c’)</th>
<th>95% Confidence interval for indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal aggression (DPE) PSI</td>
<td>$F(5, 239) = 11.35$, p &lt; .05, $AR^2 = .18$</td>
<td>-.031</td>
<td>.14*</td>
<td>-.0077</td>
<td>-.0033</td>
<td>(-.0087 to -.0080)*</td>
</tr>
<tr>
<td>Maternal aggression (DPE) EPI</td>
<td>$F(5, 239) = 13.94$, p &lt; .05, $AR^2 = .21$</td>
<td>-.0028</td>
<td>.86*</td>
<td>-.0077</td>
<td>-.0053</td>
<td>(-.0072 to .0010)</td>
</tr>
</tbody>
</table>

* = $p < .05$, 2-tail

Table 32
Regression Parameter Estimates (B) for Mediation Analyses with Neighbourhood as the Independent Variable, Maternal Affective Behaviour as the Mediator, and Anxious Symptoms (Change) as the Dependent Variable, Controlling for Family SES, Anxious Symptoms at T1 and Gender

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Significance and model fit</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total effect (c)</th>
<th>Direct effect (c’)</th>
<th>95% Confidence interval for indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal aggression (DPE) PSI</td>
<td>$F(5, 237) = 6.60$, p &lt; .05, $AR^2 = .10$</td>
<td>-.032*</td>
<td>.15*</td>
<td>.0025</td>
<td>.0071</td>
<td>(-.0111 to -.0008)*</td>
</tr>
<tr>
<td>Maternal aggression (DPE) EPI</td>
<td>$F(5, 237) = 7.46$, p &lt; .05, $AR^2 = .12$</td>
<td>-.0026</td>
<td>.67*</td>
<td>.0025</td>
<td>-.0043</td>
<td>(-.0058 to .0012)</td>
</tr>
</tbody>
</table>

* = $p < .05$
10.4 Summary of findings regarding maternal interaction style as a mediator between neighbourhood disadvantage and internalising symptoms

The aim of these analyses was to determine whether maternal affective behaviour was a mediator of the relationship between neighbourhood disadvantage and internalising symptoms. While neighbourhood disadvantage was a significant predictor of dysphoric and positive behaviour in mothers, these behaviours were not associated with altered risk for depression and anxiety in adolescents. Thus the hypothesis was not supported regarding the possibility of positive behaviour mediating the relationship between neighbourhood disadvantage and internalising symptoms.

Hypotheses were supported regarding the mediating role of maternal aggression and change in internalising symptoms. Mothers from disadvantaged neighbourhoods were found to display longer episodes of aggressive behaviour on both tasks and this, in turn, was found to predict lower reductions in symptoms of depression and anxiety throughout adolescence. These findings even held true for the PSI when the effect of family SES was accounted for, indicating a unique effect of neighbourhood over and above the effect of family SES. Interestingly, the effect of maternal aggression did not mediate the relationship between neighbourhood disadvantage and internalising symptoms at T1. These findings suggest that parental aggression may represent a pathway from neighbourhood disadvantage to the emergence of internalising symptoms in adolescence, and suggests that this effect may be more important in adolescence than during childhood.
CHAPTER 14
DISCUSSION

This chapter will review the key findings, theoretical implications and conclusions of this thesis. Section 14.1 will provide a broad overview of key findings. In section 14.2 the results will be related to the nine hypotheses outlined in Chapter 7 with key findings in regard to depressive and anxious symptoms (section 14.2.1), stress (section 14.2.2), temperament (section 14.2.3) and maternal affective behaviour (section 14.2.4). The theoretical implications of the findings of this thesis will be discussed in section 14.3. Section 14.4 reviews limitations of the methodology used, and provides suggestions for future research. Section 14.5 discusses methodological strengths, while section 14.6 outlines potential clinical implications of key findings. The conclusions based on the findings of the thesis will be discussed in section 14.7.

14.1 Overview of key findings

The research reported in this thesis provided evidence that adolescents who live in disadvantaged neighbourhoods differ from those in less disadvantaged neighbourhoods in several key ways, all of which indicate that disadvantaged neighbourhoods are less conducive to healthy and positive development. There was evidence that adolescents from these neighbourhoods were more likely to suffer from depressive and anxious symptoms, and to experience stressful life events. Adolescents from disadvantaged neighbourhoods also differed in their temperaments: they tended to score higher on measures of Negative Affectivity, and lower on measures of Surgency and Effortful Control. Parents from disadvantaged neighbourhoods also differed from parents from less disadvantaged neighbourhoods. They were more likely to express aggression for longer periods (as measured on both interaction tasks). They also expressed positive affect for shorter periods, and dysphoric affect for longer periods during the Event Planning task specifically. Thus, it appears that families and adolescents in disadvantaged neighbourhoods differ in a range of ways from those in less disadvantaged neighbourhoods. Most of these findings remained significant even when the influence of family SES was taken into account. This indicates that neighbourhood disadvantage may have a unique influence on these characteristics, independent of family SES.
Several of these differences between disadvantaged and less disadvantaged neighbourhoods were found to mediate the relationship between neighbourhood disadvantage and internalising symptoms. All three temperament dimensions (Surgency, Negative Affectivity and Effortful Control) mediated the relationship between neighbourhood disadvantage and internalising symptoms at T1, but not the change in symptoms from T1 to T3. Conversely, maternal aggression (as measured on both the PSI and the EPI) mediated the relationship between neighbourhood disadvantage and change in symptoms from T1 to T3, but not T1 symptomatology. Stress mediated the relationship between disadvantage and internalising symptoms at T1, and the change in these symptoms through adolescence. These mediating factors point to several key pathways through which neighbourhood disadvantage may increase risk for depression and anxiety in adolescents.

Interestingly, all of these factors mediated the relationship between neighbourhood disadvantage and both anxiety and depression, suggesting that anxiety and depression share similar risk factors with respect to neighbourhood effects and associated mechanisms. This also suggests that, with respect to these questions at least, depression and anxiety may be more accurately conceptualised through an overarching construct of internalising (Lahey et al., 2008). This is despite the fact that the anxiety measure used in the current thesis (the BAI) is specifically designed to measure symptoms of anxiety that are distinct from symptoms of depression (Steer et al., 1993). A single ‘internalising’ construct has been proposed in a large number of research papers, and has substantial empirical support (Lahey et al., 2008). Given the similarity in results regarding anxiety and depression, findings will be discussed in regard to a general construct of internalising symptoms, rather than separately in terms of anxiety and depression. A more detailed discussion of the overlap between anxiety and depression is provided in section 14.3.4.

The following sections will discuss findings in relation to the hypotheses proposed in Chapter 7. Aside from the section discussing the direct relationship between anxiety and depression, each section will be structured to first discuss the ‘a’ path of the mediation pathway (i.e., neighbourhood disadvantage to the mediator in question), followed by the overall mediation pathway. This was done in order to provide a clearer discussion of each pathway between neighbourhood disadvantage and internalising. Following a discussion of each specific mediation pathway, section 14.2.5 will explain the findings with regard to gender effects, while section 14.2.6 explains findings with regard to socioeconomic status.
14.2 Evaluation of hypotheses

14.2.1 Evaluation of hypothesis 1 – neighbourhood and internalising symptoms

Hypothesis 1 (internalising symptoms) stated that there would be a positive relationship between neighbourhood disadvantage and the number of symptoms of depression/anxiety in adolescents at T1, and change in symptoms between T1 and T3.

The results partially support this hypothesis. In particular, it was found that neighbourhood disadvantage was associated with increased symptoms of anxiety and depression at T1. These cross-sectional findings regarding depressive symptoms are consistent with a large number of studies indicating that disadvantaged neighbourhoods are associated with increased risk for depression in adolescents (summarised by Kim, 2008). They also add important weight to the small amount of literature examining the neighbourhood environment and its associations with anxiety, with very few studies having examined neighbourhood environment and anxiety in adult populations, let alone in adolescents (e.g., Schneiders et al., 2003; Ezpeleta et al., 2001). The findings are further strengthened with the analysis indicating that neighbourhood disadvantage was still a predictor of depressive and anxious symptoms when controlling for the influence of family SES. This suggests that there may be a unique effect of neighbourhood disadvantage on internalising symptoms in adolescents, over and above the shared influence of neighbourhood and SES. It is somewhat surprising that these differences exist despite higher investment in welfare and infrastructure in Australian neighbourhoods compared to some disadvantaged neighbourhoods in the USA and even neighbourhoods in many European countries (Gough et al., 1997; Whiteford, 2005). It is also surprising given that participants from the most disadvantaged neighbourhoods were more likely to be missing from these analyses, and that the sample size was small relative to many other studies of neighbourhood effects (Mair et al., 2008).

While these findings may point to an important influence of neighbourhood on risk on internalising symptoms, they do not, as such, provide any information to elucidate the mechanisms through which this effect occurs.

Support was not found for the prediction that adolescents from disadvantaged neighbourhoods would experience less reduction in their symptoms of anxiety and depression between T1 and T3. A lack of a significant relationship between neighbourhood disadvantage and change in symptoms was not unexpected given that an analysis controlling for initial levels of
symptomatology is a particularly rigorous test of the influence of the neighbourhood environment (given the high correlation between symptom levels across assessments). The findings from the current thesis are contrary to those of Schneiders and colleagues (2003), who found that neighbourhood disadvantage prospectively predicted change in anxiety and depression in adolescents over a two year period, however it is worth noting that the sample size of the study by Schneiders and colleagues was much greater (2587) than the current study, and, given there was a trend towards significance for the relationship between neighbourhood disadvantage and change in depressive symptoms, a larger sample size may have allowed for detection of a significant relationship between neighbourhood disadvantage and change in internalising, at least for depressive symptoms.

It is worthwhile considering various factors that may have influenced the strength of association observed between neighbourhood disadvantage and internalising symptoms. Several factors are likely to have led to an underestimation of the relationship between neighbourhood disadvantage and internalising symptoms. First, measures of distal factors such as neighbourhood disadvantage are likely to contain more ‘data noise’ than more proximal measures. In other words, there are likely to be many unmeasured factors (such as intra-neighbourhood variation) that weaken perceived effects. Second, as previously mentioned, the relatively small sample size of the current study is also likely to reduce the power of the study to detect effects, with many other studies of this nature using samples in excess of 10,000 (Mair et al., 2008). The current study also lacked full representation from participants from the most disadvantaged neighbourhoods. Melbourne is commonly rated as one of the most liveable cities in the world (Holden & Scerri, 2012) (presumably making it one of the least disadvantaged in the world), and the current study also did not include participants from rural areas (where neighbourhood disadvantage is lowest (Pink, 2006). Furthermore, families would have had to be functional enough to take part in the battery of assessments involved in the ADS, which is likely to have decreased the number of low SES families who took part. These factors are likely to have created somewhat truncated data, which is also likely to have weakened any observed associations. Finally, the constructs used to measure neighbourhood disadvantage and internalising were completely independent: internalising was measured using self-report, while neighbourhood disadvantage was measured independently by the Australian Bureau of Statistics. This is in contrast to studies that use subjective measures of neighbourhood disadvantage, which more often find significant associations between neighbourhood disadvantage and internalising, possibly partly due to reporting bias and shared method variance (Mair et al., 2008). All these...
factors could have acted to obscure or reduce effects, and therefore point to the robustness of the association between neighbourhood disadvantage and internalising symptoms in the current study.

14.2.2 Evaluation of hypothesis 2 and 3 – neighbourhood and stressful life events

Hypothesis 2 (stressful life events) stated that adolescents from disadvantaged neighbourhoods would experience a greater number of stressful life events.

Hypothesis 3 (stressful life events) stated that stressful life events would partially mediate the relationship between neighbourhood disadvantage and internalising symptoms in adolescents. Specifically, neighbourhood disadvantage was hypothesised to be associated with increased stressful life events, which would in turn be associated with increased symptoms of anxiety and depression in adolescents.

The results supported these hypotheses. It was determined that adolescents from disadvantaged neighbourhoods were more likely to experience a greater number of stressful life events. Stressful life events were then found to mediate the relationship between neighbourhood disadvantage and internalising symptoms.

14.2.2.1 Pathways from neighbourhood disadvantage to stressful life events

The findings of the current thesis support previous studies that have found that people living in disadvantaged neighbourhoods are much more likely to experience stressful life events, fear of victimization and chronic stress in comparison to those from less disadvantaged neighbourhoods (e.g. Warr et al., 2009; Hill et al., 2005). While the current analysis does not indicate the nature of these stressful life events, it is clear that adolescents from disadvantaged neighbourhoods are more likely to report a greater number of stressful life events. This effect was found independently of family SES, indicating that disadvantaged neighbourhoods have a unique influence on risk for stressful life events. The increased risk for stressful life events is likely to be due to a number of factors associated with disadvantaged neighbourhoods. These stressors may result directly from the neighbourhood environment, through poorer physical characteristics of the neighbourhood, which create an unsafe environment, and from people in the neighbourhood, who pose threats to emotional or physical safety (Kim, 2008). They are also
likely to be a result of more proximal factors associated with disadvantaged neighbourhoods such as family abuse resulting from poor parenting for example.

14.2.2.2 Pathways from neighbourhood disadvantage to stressful life events to internalising symptoms

The current thesis also provides evidence for the theory that stress mediates the relationship between neighbourhood disadvantage and internalising symptoms. These findings add to the small body of literature that directly examines the mediating role of stressful life events between neighbourhood disadvantage and internalising disorders. Despite strong evidence for a link between neighbourhood disadvantage and stress, and a link between stress and internalising disorders, existing research examining a mediating pathway has been mixed. One study failed to find a significant mediating role of stressful life events (Attar, Guerra, & Tolan, 1994), while another found an inconsistent mediating effect of stressful life events, whereby stressful life events mediated the relationship between neighbourhood disadvantage and internalising symptoms in fourth generation Mexican children, but not in first generation children (Roosa et al., 2010). However, these studies either lacked comprehensive measures of neighbourhood disadvantage or used poor assessment of internalising symptoms (e.g., provided by teachers who are have found to be much less able to accurately assess internalising symptoms than self-reporting of internalising symptoms (DiBartolo & Grills, 2006). In contrast to these studies, and in line with the current thesis, Wheaton and Clarke (2003) used a longitudinal design to demonstrate a significant mediating role of stressful life events between neighbourhood disadvantage and internalising disorders. These researchers found that life-course stress and ambient neighbourhood stress mediated the relationship between neighbourhood and change in internalising symptoms over time. This was found even when individual- and family-level variables were taken into account. Researchers concluded that most of the effect of neighbourhood on mental health over time is a combined function of the accumulation of first discrete, and then chronic stressors at the individual level.

The current thesis, along with Wheaton and Clarke’s paper, supports the ‘stress-adversity’ model of psychopathology. This theory, formulated by Bruce Dohrenwend (2000), posits that the degree to which environments present danger and hardship to individuals will be positively associated with risk for psychopathology, including internalising disorders. These findings run counter to the theory proposed by Attar and colleagues (1994), who argue that internalising
behaviour may be less common in harsher environments, where it may be more effective to engage in aggressive behaviour.

As discussed, mediational analyses do not necessarily delineate the direction of causation. However, there is enough existing literature to suggest that a causal chain exists whereby the neighbourhood environment influences stressful life events, which then influence risk for internalising symptoms. For instance, a number of studies have found that disadvantaged neighbourhoods are associated with a greater number of stressors in the environment (e.g., Braveman & Egerter, 2008; Matheson et al., 2006) and that those who move to less disadvantaged neighbourhoods experience decreased danger and fewer stressors in their new neighbourhood environment (Katz et al., 2001; Rubinowitz & Rosenbaum, 2000). In terms of stress and internalising disorders, there is an enormous number of studies demonstrating that both stressful life events and the experience of stress precede the development of internalising disorders (e.g., Goodyer, 1996; Hillegers et al., 2004; Kendler et al., 1999), and that stressful life events may be particularly impactful on risk for internalising disorders in adolescents (Kim et al., 2003). In addition, the timing of measurement is in line with the mediating pathway proposed in the current thesis. Neighbourhood was measured initially at T1, followed by measurement of stressful life events at T2, followed by the final measurement of internalising symptoms at T3. While measurement of stressful life events is inclusive of events that occurred before T1, there is likely to be an overemphasis on more recent stressful life events (which occurred after T1).

14.2.2.3 Evaluation of hypothesis 4 – neighbourhood, stressful life events and gender

Hypothesis 3 (stressful life events x gender) stated that boys from disadvantaged neighbourhoods would experience a greater number of stressful life events than girls. That is, there would be an interaction between neighbourhood and gender, whereby boys would be more sensitive to the level of disadvantage in their neighbourhood in terms of stressful life events.

Hypotheses regarding moderating effects of gender were not supported. The association between neighbourhood disadvantage and stressful life events were no greater in males than in females, and gender did not moderate the mediating influence of stress in the relationship between neighbourhood disadvantage and internalising symptoms. This indicates that both male and females are affected similarly by neighbourhood disadvantage, and that these stressful life events have a similar effect in terms of risk for depression and anxiety. This is contrary to some
research suggesting that males are more likely to be influenced by the neighbourhood environment (Sigfusdottir & Silver, 2009) and also research suggesting that girls’ risk for anxiety and depression may be more influenced by stressful life events (Coleman, 2007; Frydenberg, 2008). However, the sample size may not have been large enough to detect any significant moderating effect of gender, thus it is difficult to draw any strong conclusions regarding this issue.

14.2.3 Evaluation of hypothesis 5 and 6 – neighbourhood and temperament

Hypothesis 5 (temperament) stated that adolescents from disadvantaged neighbourhoods would score lower on measures of Affiliation, Effortful Control and Surgency, and higher on measures of Negative Affectivity.

Hypothesis 6 (temperament) stated that adolescent temperament (Surgency, Negative Affectivity, Affiliation and Effortful Control) would mediate the relationship between neighbourhood disadvantage and internalising symptoms in adolescents. Specifically, disadvantaged neighbourhoods were predicted to be associated with decreased Affiliation, Effortful Control and Surgency, and increased Negative Affectivity, which would in turn be associated with increased depressive and anxious symptoms.

Most of these hypotheses were supported, with analyses indicating that adolescents living in more disadvantaged areas are more likely to score higher on measures of Negative Affectivity, and lower on measures of Surgency and Effortful Control. Affiliation was not found to differ as a function of neighbourhood disadvantage. It was also found that Surgency, Negative Affectivity and Effortful Control mediated the relationship between neighbourhood disadvantage and internalising symptoms at T1, but not the change in internalising symptoms from T1 to T3.

These findings will be discussed in turn according to each specific temperament dimension. It is important to note that given the cross-sectional nature of this research, it is difficult to say whether disadvantaged neighbourhoods and associated factors actually effect change in temperament. However there are several reasons to believe that this could be the case. These will be discussed in each relevant section. Section 14.2.3.5 will discuss findings in relation to the lack
of a mediating role of temperament in the relationship between neighbourhood disadvantage and change in internalising symptoms between T1 and T3.

14.2.3.1 Neighbourhood and Effortful Control

The hypothesis of a mediating role of Effortful Control between neighbourhood disadvantage and internalising symptoms at T1 was supported. Neighbourhood disadvantage was associated with decreased Effortful Control in adolescents (suggesting they are less able to regulate their emotions and behaviour), which in turn was associated with increased risk for symptoms of depression and anxiety at T1.

There are several reasons to believe that neighbourhood disadvantage may cause decreased Effortful Control. First, there is evidence that Effortful Control is malleable - it matures throughout childhood and adolescence and is commonly thought to be influenced by environmental factors (Hart et al., 2008). Stress – including stress resulting from the neighbourhood environment – may cause decreased ability to regulate emotions and behaviour. There is evidence to suggest that stress can tax or exceed the cognitive resources of a person, leading to poorer affective and behavioural regulation (Lazarus & Folkman, 1984; Halverson & Deal, 2001). For example, a longitudinal study supports this assertion, with findings demonstrating that stressful and disorganised environments are associated with decreased persistence (a component of Effortful Control) (Halverson & Deal, 2001). There is even evidence to suggest that stress actually inhibits the development (and reduces the size) of the frontal lobes - a key brain region involved in regulation of emotions and behaviour (Lupien et al., 2009). Thus disadvantaged neighbourhoods may provide a maladaptive and stressful environment, which gradually erodes the ability to regulate emotions and behaviour, thus decreasing Effortful Control.

Another reason that disadvantaged neighbourhood may lead to decreased Effortful Control is the higher number of poor role models in the environment who may fail to teach effective behavioural control strategies. There is evidence that parents from disadvantaged neighbourhoods are less likely to demonstrate effective self-control (Guterman et al., 2009), with studies indicating that this is likely to have implications for adolescents’ emotional development, including poorer emotion regulation (Eisenberg et al., 1998; Shipman & Zeman, 2001). For instance, studies have found that children with less responsive mothers are more likely to be impulsive (Olson et al., 1990) and are more likely to have lower Effortful Control (Kochanska et
al., 2000). One longitudinal study found that positive parenting attributes predicted increases in persistence in children from ages 4 to 7 years (Halverson & Deal, 2001). Thus the people who make up disadvantaged neighbourhoods may adversely influence the development of Effortful Control in children and adolescents.

A further reason for decreased Effortful Control in disadvantaged neighbourhoods could be related to the helplessness and hopelessness reported by some people from these neighbourhoods (Guterman et al., 2009). When adolescents feel hopeless or helpless, they may lose the ability or motivation to regulate their emotions and behaviour in effective or helpful ways. Supporting this is Seligman’s learned helplessness theory of depression (1997), which suggests that people become unmotivated, emotionally dysregulated and depressed when they perceive their environment as uncontrollable and believe they cannot influence what happens around them (Kazdin, Esveldt-Dawson, Sherick, & Colbus, 1985).

Of course, a competing hypothesis is that parents with poorer effortful control may ‘drift down’ to more disadvantaged neighbourhoods, due to dysfunction associated with poor effortful control. However, the findings by Hart and colleagues (2008), of a prospective link between stressful environments and poorer persistence, suggest that disadvantaged neighbourhoods may play a causal role in lowered Effortful Control.

The link found between decreased Effortful Control and internalising symptoms was not surprising, with many studies finding strong links between these constructs (e.g., Lengua, 2006; Muris, Van Der Pennen, Sigmond, & Mayer, 2008; Oldehinkel et al., 2007). It is thought that those low on Effortful control are less able to regulate anxiety and depression, leading to exacerbation of depressive and anxious symptoms.

14.2.3.2 Neighbourhood and Surgency

The hypothesis that Surgency would have a mediating role in the relationship between neighbourhood disadvantage and internalising symptoms at T1 was supported. Neighbourhood disadvantage was associated with decreased Surgency in adolescents (suggesting they may be less likely to engage in approach related behaviour and are higher on fearfulness and shyness), which in turn was associated with increased risk for symptoms of depression and anxiety.
As discussed in chapter 8.3.6, Surgency is composed of several subfactors including fearfulness, shyness, and sensation seeking. There are several reasons why neighbourhood disadvantage may be associated with these factors.

It is possible that adolescents may respond to stressful and dangerous neighbourhoods by engaging in less approach and sensation seeking behaviours. These behaviours may be a more adaptive response to dangerous environments than high-Surgency behaviours. Aware of the dangers of their neighbourhood, parents may also discourage riskier, approach-style behaviours. There is qualitative and quantitative evidence of this from studies indicating that parents from disadvantaged neighbourhoods may use more restrictive, controlling and harsher parenting to protect their children from dangers in the neighbourhood (Furstenberg, 1993; Baumrind, 1972; Dearing, 2004). Again, it is possible that this could discourage high-Surgency behaviour and consequentially alter the development of this temperament dimension.

Further support for the argument that disadvantaged neighbourhoods could cause lower rates of approach style behaviour comes from a prospective study by Hart and colleagues (2008), who compared children from disadvantaged neighbourhoods to children from affluent neighbourhoods. Those living in economically disadvantaged neighbourhoods were more likely to demonstrate a decrease in self-confident behaviour (which shares some overlap with Surgency) over a two-year period, indicating a possible causal effect of these disadvantaged environments. Finally, given that adolescents in disadvantaged neighbourhoods experience more stressful life events (Braveman & Egerter, 2008; Matheson et al., 2006), it is plausible that they may also experience less positive events, which Vaidya and colleagues (2002) found in their study was associated with lower Extraversion (similar to low Surgency).

It is also likely that, just as lower Surgency may lead to increased risk for internalising disorders, internalising disorders may in turn lead to withdrawal behaviours, associated with low Surgency. Thus the perceived associations may be due in part to the increased internalising disorders experienced by adolescents from disadvantaged neighbourhoods.

The findings of the current thesis run somewhat counter to many studies that have found neighbourhood disadvantage to be associated with increased levels of externalizing behaviour, (e.g., Leventhal & Brooks-Gunn, 2000), which is an approach style behaviour (and thus may be positively associated with aspects of Surgency). Thus, it may be that particular adolescents either
respond to disadvantaged environments with a more externalizing and approach style of behaviour, or alternatively, more withdrawal style behaviour. These contrasting reactions to neighbourhood disadvantage are likely to dampen associations observed between neighbourhood disadvantage and approach related behaviour. Thus it is possible that the lower Surgency observed in adolescents from disadvantaged neighbourhoods in the current study is primarily associated with increased fearfulness and shyness.

Perhaps the most important way that disadvantaged neighbourhoods may increase fearfulness and shyness is through chronic stress resulting from neighbourhood danger and disorder (Braveman & Egerter, 2008; Matheson et al., 2006). Chronic stress is likely to lead to greater fearfulness and increased shyness (Kessler et al., 1985; Mirowsky & Ross, 1986). Persistent experience of fearfulness and shyness may encourage the development of low Surgency. Research supporting this hypothesis comes from a study by Vaidya and colleagues, who observed changes in neuroticism over 2.5 years in response to stressful life events, even after controlling for neuroticism at Time 1 (Vaidya, Gray, Haig, & Watson, 2002). Similarly, Hart and colleagues (2008) found that, compared to children from affluent neighbourhoods, those living in economically disadvantaged neighbourhoods were more likely to demonstrate increases in shy and anxious temperaments over a two year period. The development of low Surgency (specifically the high fearfulness and shyness aspects) in response to stress may even occur through changes to the development of a part of the brain involved in negative emotion – the amygdala. Research indicates that the amygdala increases in size in response to stress, and may encourage behaviours and emotions associated with fearfulness (Lupien et al., 2009).

In summary, it was hypothesised that the increased neighbourhood danger and stress found in disadvantaged neighbourhoods may primarily lead to increased fearfulness and shyness, but also may lead to decreased approach related behaviour.

It was not surprising to find that this decreased Surgency was associated with increased internalising symptoms, as previous research has indicated that low Surgency prospectively predicts later internalising disorders (Forbes & Dahl, 2005). It is thought that adolescents who are lower on Surgency are less likely to form social connections, and less likely to demonstrate positive approach behaviours (Forbes & Dahl, 2005). This means they are more likely to withdraw (a hallmark of depression), and are less likely to have social supports.
14.2.3.3 Neighbourhood and Negative Affectivity

The hypothesis of a mediating role of Negative Affectivity between neighbourhood disadvantage and internalising symptoms at baseline was supported. Neighbourhood disadvantage was associated with increased Negative Affectivity in adolescents (suggesting they experience greater levels of frustration), which in turn was associated with increased risk for symptoms of depression and anxiety.

Similar arguments outlined above for the association between increased fearfulness and shyness and neighbourhood disadvantage could also be made for Negative Affectivity. Disadvantaged neighbourhoods may increase Negative Affectivity through neighbourhood stress (Braveman & Egerter, 2008; Matheson et al., 2006). Chronic stress and stressful life events are likely to lead to lower frustration tolerance (Keenan & Newton, 2007). Persistent experience of stress and frustration and associated behaviours may encourage the development of higher Negative Affectivity. As noted above, research supporting this hypothesis comes from a study by Vaidya and colleagues (2002), who observed changes in neuroticism over 2.5 years in response to stressful life events, even after controlling for neuroticism at Time 1 (Vaidya et al., 2002).

Poor parenting, which is found more commonly in disadvantaged neighbourhoods, may also encourage the development of Negative Affectivity. Several studies have linked certain parenting styles with negative emotionality. One study found evidence that maternal inconsistent discipline prospectively predicted sub-factors of Negative Affectivity, including irritability, even after controlling for prior levels of temperament and parenting (Lengua & Kovacs, 2005). Other studies have found that maternal involvement and contingency responding (actively responding to a baby’s attempts at communication) predicted increases in positive emotionality in infants (Belsky et al., 1991; Malatesta & Haviland, 1982), while maternal negative emotionality predicts increases in negativity in infants over time (Malatesta & Haviland, 1982). These studies, demonstrating a shift in negative emotionality over time, point to the possibility that these disadvantaged neighbourhoods and associated poor parenting may play a causal role in Negative Affectivity in children. Modelling of the behaviour of residents in disadvantaged neighbourhoods is also likely to encourage higher Negative Affectivity. Rentfrow and colleagues (2008) proposed that communities higher on Neuroticism, where people are more likely to be moody and anxious, may influence those who are lower on Neuroticism to be more frustrated and anxious, which in turn is likely to encourage the development of neurotic traits.
The link between Negative Affectivity and internalising symptoms was not surprising, with many studies finding that Negative Affectivity is predictive of later internalising disorders (e.g., Ormel et al., 2005; Hirshfeld et al., 1992; Schwartz et al., 1999; Caspi et al., 1996).

14.2.3.4 Neighbourhood and Affiliation

No hypotheses were supported regarding Affiliation. This temperament dimension did not differ as a function of neighbourhood disadvantage (and thus was not tested as a mediating factor in the relationship between neighbourhood disadvantage and internalising symptoms). One explanation for this is that the measure of neighbourhood disadvantage used in the current thesis might not have been sensitive to levels of social connectedness, given that it was not developed primarily with this factor in mind. Alternatively, it is possible that individual differences in Affiliation may not be influenced by neighbourhood disadvantage. In other words, there may be a universal need for connectedness found in people, irrespective of the neighbourhood in which they reside. In less disadvantaged and more socially cohesive neighbourhoods, residents may be encouraged to socialise through membership of clubs and societies. These neighbourhoods are likely to be safer, thus further encouraging social connections between neighbours (Young, Russell, & Powers, 2004). Alternatively, in disadvantaged neighbourhoods, residents may seek mutual support to help deal with the difficult circumstances and stressful life events that occur more frequently in disadvantaged neighbourhoods (Attar et al., 1994). Thus, although driven by different motivations, individuals in both disadvantaged and less disadvantaged neighbourhoods may show similar levels of Affiliation.

14.2.3.5 Temperament and change in internalising symptoms

Temperament was not found to mediate any of the associations between neighbourhood disadvantage and change in internalising symptoms from T1 to T3. There are several possible reasons for this. The first is that the sample size was too small to detect a change in symptoms, which is a more rigorous measure of internalising symptoms. Another possibility is that temperament does continue to change through adolescence, and that it may be the change in temperament (not measured in the current thesis) that is related to change in internalising symptoms throughout adolescence. A final, and perhaps most plausible hypothesis relates to the general finding that temperament is more malleable during childhood (Roberts & DelVecchio,
Temperamental traits formed during childhood may predispose young adolescents to a ‘baseline’ vulnerability to internalising symptoms, measured in this thesis at T1. However, given temperament remains relatively stable during adolescence, it is possible that the neighbourhood’s influence on temperament decreases during adolescence, and that temperament diminishes as a causal factor in further changes in internalising symptoms, as other external factors such as stress or parenting become more influential throughout adolescence.

14.2.4 Evaluation of hypothesis 7 and 8 – neighbourhood and maternal affective behaviour.

Hypothesis 7 (frequency of expressed emotion) stated that mothers from disadvantaged neighbourhoods would express a higher frequency of aggressive and dysphoric emotion, and a lower frequency of positive emotion.

No hypotheses were supported regarding frequency of maternal expressed emotion. Mothers from disadvantaged neighbourhoods did not express more frequent dysphoric or aggressive behaviour, or less frequent positive behaviour. This is somewhat surprising given that previous studies have found links between neighbourhood disadvantage and less warm and supportive parenting (Simons et al., 1996; Downey & Coyne, 1990), and more harsh and hostile parenting (Pinderhughes, Nix, Foster, & Jones, 2004; Downey & Coyne, 1990). Rather than discussing reasons for this in isolation, these findings will be discussed in conjunction with a discussion of findings regarding duration of affective behaviour, in section 14.2.4.1.1. Given the lack of significant findings regarding frequency of affective behaviour and neighbourhood disadvantage, mediation analyses were not examined regarding frequency of affective behaviour.

Hypothesis 8 (duration of expressed emotion) stated that parents from disadvantaged neighbourhoods would display a longer duration of aggressive and dysphoric behaviour and a shorter duration of positive behaviour.

Hypothesis 9 stated that maternal aggressive and positive behaviour (DPE) would mediate the relationship between neighbourhood disadvantage and internalising symptoms in adolescents. It was also predicted that parent dysphoria (DPE) would not mediate the relationship between neighbourhood disadvantage and internalising symptoms.
The results partly supported this hypothesis. Parents from disadvantaged neighbourhoods were more likely to display longer periods of aggression on both the EPI and PSI. They were also more likely to display longer periods of dysphoric behaviour and shorter periods of positive behaviour on the EPI. Higher duration of aggressive behaviours on both tasks (but not dysphoric or positive behaviours on either task) was then found to mediate the relationship between neighbourhood and the change in internalising symptoms from T1 to T3 (but not baseline symptoms at T1).

14.2.4.1 Pathway from neighbourhood disadvantage to parenting

The following section will discuss possible explanations for the relationship between neighbourhood disadvantage and duration of affective behaviour, with reference to the null findings regarding frequency of affective behaviour.

14.2.4.1.1 Why might there be differences in the duration but not the frequency of affective behaviours?

Previous research indicates that parents from disadvantaged neighbourhoods are more likely to demonstrate poorer parenting practices, such as less warm, consistent and supportive parenting (Simons et al., 1996; Downey & Coyne, 1990), and are more likely to demonstrate harsh, hostile and coercive parenting (Pinderhughes et al., 2004; Simons et al., 1996; Downey & Coyne, 1990). These studies, however, have not generally directly observed parents, and do not provide comparisons between frequency and duration of parenting styles. The current thesis is the first to provide an opportunity to assess how each of these aspects of parent behaviour is associated with neighbourhood disadvantage and internalising symptoms. The following section will discuss reasons for why mothers from disadvantaged neighbourhoods were more likely to display differing durations, but not frequencies, of affective behaviour.

When parents demonstrate affective behaviour towards their children, even dysphoric or aggressive behaviour, expressing negative behaviours for a shorter duration may not necessarily be indicative of maladaptive parenting. For instance, if a child is misbehaving, a brief display of aggression towards children may be an adaptive response (it is worth noting that the LIFE coding system included very subtle displays of affective behaviour – thus even a subtle display of aggression would have been coded as aggression). In comparison, extended displays of aggressive or dysphoric behaviour may reflect inappropriate and maladaptive responding to
situations, where a parent is not responding effectively to the changing needs of their children. These displays of affective behaviour may also reflect poor affect regulation, as a high duration of negative behaviour (such as aggression or dysphoria) is thought to reflect an inability to shift out of undesirable mood states, while a low duration of positive behaviour it thought to reflect an inability to stay in desirable mood states (Bariola et al., 2011).

It is likely that the same factors that are thought to lower Effortful Control in adolescents may also lower Effortful Control in parents, including a sense of lack of control, poorer neighbourhood role models, and factors relating to social drift (all outlined in section 14.2.3.1). In summary, it is possible that although the initiation of various expressions of emotion may not be maladaptive, and may simply reflect a normal or appropriate responding to situations, lengthened periods of negative affective behaviour and shorter periods of positive behaviour may reflect maladaptive parenting and an inability to regulate emotion.

There are some alternative hypotheses to explain these differences in duration of affective behaviour. It is possible that differences in rates of anxiety and depression between parents from disadvantaged and less disadvantaged neighbourhoods explain the differences in duration of affective behaviour. Several studies indicate that depression and anxiety is associated with increased levels of hostile, coercive, and disengaged parenting practices (Lovejoy, Graczyk, O’Hare, & Neuman, 2000). Another possibility is that the differences in duration of affective behaviours are due to more frequent misbehaviour from children from disadvantaged neighbourhoods: externalizing symptoms are more common in these neighbourhoods (Leventhal & Brooks-Gunn, 2000), which may induce submissive/dysphoric, aggressive or less positive behaviour from mothers. However, if this were the case, it might be expected that mothers would also show a greater frequency of aggressive or dysphoric behaviour to reflect more frequent misbehaviour.

A final possibility relates only to aggression - it is possible that mothers from disadvantaged neighbourhoods are more likely to use aggression as a form of punishment when their children misbehave. This may occur for several reasons; mothers from disadvantaged neighbourhoods are more likely to be stressed and thus have a lower frustration tolerance (Guterman et al., 2009), and their children may be more likely to use aggressive and externalizing behaviour (Leventhal & Brooks-Gunn, 2000), which is likely to encourage more aggressive parenting. Mothers from disadvantaged neighbourhoods may also display longer periods of aggression due to the more
highly developed culture of honour present in disadvantaged neighbourhoods. Due to this factor, both mothers and children may be encouraged to behave in more aggressive ways during conflictual interactions. Parents may also feel they need to be stricter in order to prevent their children from being exposed to dangers in their neighbourhood (Furstenberg, 1993; Baumrind, 1972). Supporting these theories is previous research indicating that parents from disadvantaged neighbourhoods are more likely to report using coercive or punishing parenting (Downey & Coyne, 1990), and are more likely to believe that this form of parenting is an effective response to misbehaviour from children (Kohen et al., 2008).

In summary, the increased duration of aggressive and dysphoric behaviour and decreased duration of positive behaviour of mothers from disadvantaged neighbourhoods may be due to affective dysregulation, increased use of coercive and punitive parenting, and higher rates of maternal depression and anxiety.

It is interesting that differences in dysphoric and positive behaviour were only apparent on the EPI task. One possible reason for this is that on the PSI task, high dysphoric and low positive behaviour may be normal for all families, and thus this task may not effectively differentiate between dysfunctional and non-dysfunctional family interactions. However, an inability to maintain positive mood states on the EPI task (which is designed to elicit positive interactions) may signal greater dysfunction, and thus be better at distinguishing between these families. In contrast, differences in aggression appeared on both the EPI and PSI, indicating mothers from disadvantaged neighbourhoods may display increased aggression over many different contexts.

14.2.4.2 Mediating role of parent affective behaviour between neighbourhood and internalising symptoms

Of all three affective behaviours associated with neighbourhood disadvantage, aggression was the only factor found to mediate the relationship between neighbourhood disadvantage and internalising symptoms. This is probably because aggression is a much stronger predictor of internalising symptoms in children (e.g., Gar & Hudson, 2008; Burge & Hammen, 1991) than dysphoric behaviour (Schwartz et al., 2011), or deficits in parents’ expression of positive behaviour (McLeod et al., 2007).

The results of the current thesis are in line with other studies that directly examine the mediating role of parenting. These studies have found aggressive parenting, but not positive parenting, to
play a significant mediating role in the relationship between neighbourhood and internalising symptoms (Deng et al., 2006; White & Roosa, 2012; Lewinsohn et al., 1994). Deng and colleagues (2006) found that mother-reported conflict significantly mediated the relationship between mother’s perceptions of their neighbourhood quality and child internalising disorders. Also in line with the current thesis, Gonzales and colleagues (2010) found that parental warmth did not mediate the relationship between a variety of measures of neighbourhood disadvantage and internalising symptoms in children, while White and colleagues (2012) found paternal warmth did not mediate the association between fathers’ perceptions of neighbourhood danger and adolescent internalising symptoms. The current study extends upon these findings by examining associations prospectively. It also improves upon the study by White and colleagues by providing independent measures of neighbourhood disadvantage.

While maternal aggression mediated the relationship between neighbourhood disadvantage and change in internalising symptoms from T1 to T3, aggression did not mediate the relationship between neighbourhood disadvantage and internalising symptoms at baseline (T1). These findings suggest that the period from 12 to 16 is perhaps a critical period during which parent interaction and affective behaviour impacts on adolescents. This is potentially due to the fact that aggressive interactions may be more prominent during adolescence, as conflict increases and closeness between parents and their children decreases during this time (Steinberg & Morris, 2001). Thus the pathway from neighbourhood environment to parent interaction to internalising symptoms may be most prominent during this time.

In conclusion, the current thesis provides further evidence that parental aggression, but not dysphoric or positive behaviour, mediates the relationship between neighbourhood disadvantage and change in internalising symptoms through adolescence.

14.2.5 Gender differences

The current study found no support for gender as a moderator of the relationship between neighbourhood disadvantage and internalising symptoms. Gender also did not moderate any of the mediating relationships examined (i.e., there was no evidence for moderated mediation). It is possible that these results reflect Type II error and are due to the sample size lacking power to detect gender moderation. However, is also possible that neighbourhoods influence males and
females similarly. Previous research has more consistently found no effect of gender in the relationship between neighbourhood and mental health outcomes, even with larger sample sizes, suggesting that gender may not play a significant role in neighbourhood effects (Mair et al., 2008).

14.2.6 Socio-economic Status

In order to provide a more stringent test of the effect of neighbourhood disadvantage independent of family SES, analyses in which neighbourhood was found to be a significant predictor were reanalysed with the addition of individual family SES as a control variable. In terms of direct effects of neighbourhood disadvantage, analyses controlling for SES found that neighbourhood disadvantage remained a significant predictor in eight of the ten analyses. In terms of mediating relationships, four of fourteen relationships remained significant after controlling for SES. This indicates that there is a significant effect of neighbourhood disadvantage on some of these outcome variables over and above the shared influence of neighbourhood and family SES.

It may be helpful to ask what it means when a relationship remains significant whilst controlling for SES, and what it means when a relationship is no longer significant when controlling for SES. When a finding remains significant despite the addition of SES as a control variable, this indicates that the effect of neighbourhood disadvantage on the dependent variable is at least partially independent of family SES. This rules out the possibility that the observed relationship is merely due to the fact that lower SES individuals are more concentrated in disadvantaged neighbourhoods. Moreover, the actual effect of neighbourhood disadvantage is likely to be greater than the effect estimated in these analyses, as the variance shared by neighbourhood and SES (much of which may be primarily related to neighbourhood effects) has been removed from analyses.

When a finding does not remain significant with the addition of SES as a control variable, it is difficult to say whether or not the neighbourhood environment is a genuine predictor of the dependent variable. Some statisticians have argued that it is impossible to separate the individual-level from neighbourhood-level effects on a statistical level (e.g., Oakes, 2004). Controlling for family SES isolates the variance due to neighbourhood effects alone. Doing so means that the variance shared by neighbourhood and individual SES is also removed, potentially weakening the perceived effect of neighbourhood below its genuine effect (possibly leading to Type 2 error).
People usually have some degree of choice with respect to where they live, thus individual traits and family characteristics may partially be associated with broader neighbourhood characteristics through these selective choice mechanisms (Aaronson, 1998). The neighbourhood context also affects human development and behaviour through a variety of mechanisms (Ellen & Turner, 1997), thus individual traits and family characteristics are likely to be associated with neighbourhood characteristics. For example, it is likely that someone in a poor neighbourhood is going to be poorer partly because their neighbourhood offers them fewer high-paying job opportunities and fails to provide them with the support needed to gain higher paid work. Thus, given that family SES and neighbourhood are such overlapping constructs, it is very difficult, if not impossible, to determine whether the non-significant findings in the current study are actually indicative of there being no genuine neighbourhood effect, or whether the neighbourhood effect still exists, but only in combination with family SES.

One indication that the neighbourhood environment may still play a significant role in non-significant findings (when controlling for SES) is that neighbourhood disadvantage appears to be a more consistent and stronger predictor than SES of various outcomes measured. Family SES predicted only four of ten dependent variables examined in Chapter 10 (in comparison to eight out of ten variables predicted by neighbourhood disadvantage). This suggests that, despite neighbourhood being a distal factor, it may be as important, if not more important a predictor of outcomes than family SES.

14.3 Broad Theoretical implications

The following sections will discuss the results of the current thesis with reference to broader theories and conclusions. Differences between disadvantaged and non-disadvantaged neighbourhoods will be discussed (section 14.3.1), before a model will be proposed that attempts to explain associations between neighbourhood disadvantage and internalising symptoms in adolescents (section 14.3.2). Section 14.3.3 discusses theoretical implications regarding regulation of emotion and behaviour, while section 14.3.4 discusses implications in regard to anxiety and depression.

14.3.1 Differences between disadvantaged and non-disadvantaged neighbourhoods
The first major significant findings reported in this thesis are that disadvantaged neighbourhoods differ in many ways from non-disadvantaged neighbourhoods, and are potentially causative of a range of negative outcomes in neighbourhood residents. Adolescents from disadvantaged neighbourhoods are more likely to have maladaptive temperaments: they are more likely to demonstrate lower Surgency, Effortful Control, and higher Negative Affectivity. They are also at greater risk of stressful life events, and depressive and anxious symptoms. Parents from disadvantaged neighbourhoods also differ from those from less disadvantaged neighbourhoods. They are more likely to demonstrate longer periods of aggressive and dysphoric behaviour and shorter periods of positive behaviour whilst interacting with their children. This provides important evidence to suggest that a range of differences exist in the characteristics and social makeup of neighbourhoods, even in countries with a strong welfare “safety net” and less income inequality than the USA, such as Australia (Gough et al., 1997; Whiteford, 2005). As discussed previously, while it is interesting that these differences exist between disadvantaged and less disadvantaged neighbourhoods, these associations do not indicate the mechanisms through which neighbourhoods influence these various outcomes. Mediational analyses are one way to examine potential mechanisms, and broad findings from these analyses will be discussed in the following section.

14.3.2 A model of adolescent risk factors for anxiety and depression in adolescents

Shortt and Spence, in their 2003 paper, discuss the importance of understanding not only the various risk factors for internalising disorders, but also the interplay of these factors. Their model, which attempts to describe these risk and protective factors and their interrelation, is shown in figure 31. The current thesis used this model to explore neighbourhood influences on adolescents. Disadvantaged neighbourhoods appear to provide a relatively harmful environment and affect residents in a variety of different ways. Understanding the complex interplay between neighbourhood disadvantage and various proximal risk factors was a central aim of the current thesis. Based on mediational analyses, the results in this thesis indicate that neighbourhoods may affect risk for depression and anxiety not only directly, but also through indirect pathways. That is, the neighbourhood environment (a distal factor) may influence more proximal factors (i.e., parent interaction and stressful life events), and also individual factors (i.e., temperament), to then influence risk for symptoms of depression and anxiety in adolescents. While these pathways are often theorised to affect risk for internalising, there are few empirical studies examining the
plausibility of these hypotheses. There are less than five studies examining the mediating role of parenting and stressful life events, and no studies examining the mediating role of temperament between neighbourhood disadvantage and internalising disorders. Thus, the current thesis adds a much needed empirical examination of these pathways. The findings of this thesis are in line with theories that suggest that, in young people, mediating pathways may be more powerful than the direct effect of neighbourhood on risk for internalising symptoms (Ingoldsby & Shaw, 2002), for instance through the impact of disadvantaged neighbourhoods on stress in parents (White & Roosa, 2012).

Figure 31: An ecological–developmental model showing factors associated with depression in youth (Adapted from Shortt & Spence, 2003). Double headed arrows indicate interactions between various factors and levels.

Shortt and Spence state that transactions between different factors are dynamic and reciprocal, and are likely to change over time, in such a way that specific factors may increase or decrease the risk of internalising disorders or have no impact depending upon the child’s developmental stage (figure 32 shows a representation of this model). Perhaps one of the most interesting findings in the current thesis was in regard to the timing of influence of the mediating factors analysed.
We could ask two questions regarding the possible timing of various influences - during which period of a child/adolescent’s life does neighbourhood disadvantage influence the various mediating factors examined in the current thesis, and during which period do these factors influence risk for internalising symptoms? Each mediating factor will be discussed in turn.

Temperament was found to mediate the relationship between neighbourhood disadvantage and internalising symptoms at baseline, but not during the period between early to later adolescence. Potentially, neighbourhoods exert their main influence on temperament during early childhood, as there is strong evidence that temperaments are more malleable during this period (Roberts & DelVecchio, 2000). Specifically, Hofferth and Sandberg (2001) argue that temperament would be most open to change by neighbourhood factors between the ages of 3 to 5 years (Hofferth & Sandberg, 2001). They measured the amount of time spent by children in different environments and found that neighbourhood contact increases 75% between infancy and early childhood (Hofferth & Sandberg, 2001), thus this period may be a time in which neighbourhood disadvantage may also have its greatest effect on temperament. Temperament (formed during childhood) may predispose young adolescents to a ‘baseline’ vulnerability to internalising symptoms, measured in this thesis at T1. However, given temperament remains relatively more
stable during adolescence, it is possible that temperament diminishes as a causal factor of change in internalising symptoms, as other factors become more influential throughout adolescence.

Maternal aggression was found to mediate the relationship between neighbourhood disadvantage and internalising symptoms in the period between early- to later-adolescence, but not the relationship between internalising symptoms and neighbourhood disadvantage at baseline. There are several possible reasons for this. Neighbourhood disadvantage is likely to influence parents’ affective behaviour throughout the life of their child, through various mechanisms discussed in Chapter 6.2. However, it is possible that it has a greater effect when their children are older and interacting more with their neighbourhood (Massey, 1998). This is likely to create greater stress for parents and thus may increase aggressive interactions throughout adolescence. In addition, there is increased conflict and decreased closeness between parents and children during this time (Steinberg & Morris, 2001) that is likely to exacerbate the influence of maternal aggression on internalising symptoms through mid- to later-adolescence.

It is likely that neighbourhood disadvantage influences stress throughout childhood and adolescence. Potentially, neighbourhood may increase risk for stressful life events in younger children through mediating factors (before children are significantly directly exposed to their neighbourhood), whilst in adolescence the rate of these events may also be increased by direct neighbourhood stressors, such as antisocial peer behaviour associated with neighbourhood gangs (for example). Stressful life events appear to increase risk for internalising symptoms during both early adolescence and the period from early to later adolescence. In summary, risk for stressful life events is likely to be influenced by neighbourhood disadvantage throughout childhood and adolescence, and stressful life events are likely to increase risk for internalising symptoms also throughout childhood and adolescence. See figure 33 for a representation of the model of proposed timing of various influences.
14.3.3 Behavioural and emotional regulation

One of the strongest themes to emerge from the current thesis was the importance of emotional and behavioural regulation in linking neighbourhood disadvantage and internalising symptoms. Both adolescents and parents from disadvantaged neighbourhoods demonstrated evidence of poorer regulation of emotions and behaviour, and both these factors were associated with increased risk for internalising symptoms in adolescents. Very little research has examined the links between neighbourhood disadvantage and emotional and behavioural regulation specifically, which is surprising, given there are a number of ways in which disadvantaged neighbourhoods could inhibit effective behavioural and emotion regulation, as outlined in section 14.2.3.1. It is suggested that a possible pathway exists for both parents and adolescents from neighbourhood to stress and helplessness, to low effortful control then to depression and anxiety. While the current study has not empirically examined this full model, future research could aim to directly examine this pathway.
14.3.4 Anxiety and depression

The current thesis found no differences in the pattern of predictors of depressive and anxious symptoms, suggesting that, at least in terms of neighbourhood influences, there is strong aetiological continuity between depression and anxiety. All significant mediators for the relationship between neighbourhood and depressive symptoms were also found for the relationship between neighbourhood and anxiety. These findings are surprising, given that the specific anxiety questionnaire was developed to measure features of anxiety that are distinct from depression. This suggests that neighbourhood disadvantage may primarily explain the shared variance between anxiety and depression. These findings also support research indicating that anxiety and depression share significant overlap and risks, and may reflect similar pathological processes (Wittchen, Kessler, Pfister, Höfler, & Lieb, 2010). Some researchers even posit that anxiety and depression are manifestations of the same disease (e.g., Wetherell, Gatz, & Pedersen, 2001). These researchers point to the high comorbidity rates between anxiety and depression: in a 40-year follow-up of levels of anxiety and depression, the probability of having anxiety for those who already suffered from depression was found to range from 0.54 to 0.98, while, for those who did not suffer from depression, the probability ranged from 0.03 to 0.10 (Murphy et al., 2004). In regard to adolescent comorbidity, there is also strong genetic continuity between depression and anxiety in adolescence (Stein et al., 2001). Several studies have found that all axis I disorders reflect two major dimensions: internalising versus externalising (e.g., Kendler et al., 2011). This two-factor model has been replicated across genders, nations, ages and lifetime and past-year diagnoses (Kruger, 2002). Kruger argues that axis I disorders should be viewed as correlated facets of broad, underlying dimensions of psychopathological variation (internalising and externalizing), with depression and anxiety both part of the internalising construct. Results from the current study lend further support to this argument: that the distinction between anxiety and depression may focus on separate facets of an underlying internalising construct, and it is this general internalising construct that is influenced by neighbourhood disadvantage.

14.4 Limitations and future directions

The following sections will discuss the limitations of the methodology presented in this thesis and also suggest possible future directions for research. Broad theoretical limitations relating to the cross-sectional design will be discussed in section 14.4.1, followed by limitations specific to
each of the factors under examination including neighbourhood disadvantage (section 14.4.2) stressful life events (section 14.4.3), temperament (section 14.4.4), and maternal affective behaviour (section 14.4.5).

14.4.1 Broad limitations

While the inclusion of prospective longitudinal analyses was a strength of this research, a weakness was the cross-sectional assessment of several other factors including stressful life events, temperament, and maternal affective behaviour. Given the cross-sectional assessment of these factors, there is less evidence to suggest that neighbourhood showed temporal precedence with respect to changes in these factors, a factor that weakens the strength of causal inferences that can be drawn from the design. For example, the current analysis does not allow us to distinguish between the effects of selective migration versus genuine neighbourhood influences with respect to these factors. It is likely that both selective migration and neighbourhood factors explain the associations between neighbourhood disadvantage and the various outcome variables examined in the current thesis. Research examining this issue has either concluded that migration effects can be accounted for by controlling for SES (Curtis et al., 2009), or that selective migration only weakly contributes to the associations between neighbourhood and health (van Lenthe et al., 2007), though the study by Lenthe and colleagues did not examine mental health. Thus, while I cannot rule out the possibility of selective migration, it is likely that neighbourhood influences (or other unmeasured factors) account for most of the perceived associations between neighbourhood disadvantage and the various outcome variables. Outside of a study using experimental design, measurement at several time points could have helped to further elucidate whether the neighbourhood environment was having a causal effect on the various factors examined. This would also have provided further understanding of the timing of influence of the various factors, to create a more accurate model of the pathways through which neighbourhood disadvantage influences risk for internalising disorders.

The effect sizes observed in regard to neighbourhood influences were relatively small, with neighbourhood and gender explaining 1.6-6.8% of the variance in the dependent variables. This was expected, as the majority of neighbourhood research has found small effect sizes relative to other environmental factors such as familial factors or peer influences (Pickett and Pearl, 2001). Two review papers determined that neighbourhood effects generally explain up to 10% of the variation in certain child health outcomes including depression, after controlling for a number of
different family characteristics (Sellström & Bremberg, 2006). There are thought to be several reasons for the relatively small effect sizes. One possibility is that the influence of neighbourhoods is actually less important than other, more proximal effects. However, while neighbourhood effects are thought to be generally subtle, they are also pervasive: despite being a more distal influence, people are exposed to them on a daily basis. There are other explanations for the relatively modest neighbourhood effects found in research. One possible reason is that distal effects are generally more difficult to measure, and thus they are likely to contain more data ‘noise’. For instance, the Australian Bureau of Statistics measure of neighbourhood disadvantage may be insensitive to differences in levels of disadvantage of areas within postcodes, such as the housing commission areas that exist in more affluent postcodes in Melbourne. Even if neighbourhood effects are modest, many researchers argue that interventions targeted at whole areas could improve the mental health for the whole population in those neighbourhoods. A small effect on an individual level still means that the net effect on the population would be larger than from interventions that have larger effects for individuals, but that only influence a small proportion of the population (Rose, 1992).

Another general limitation of the current study was the relatively small sample size compared to other neighbourhood studies (Mair et al., 2008), which introduces the possibility of type II error. It could be expected that, had the sample been larger, some of the null findings (e.g., those controlling for family SES) may have been significant. Future studies could aim to increase their sample size in order to more accurately assess the relationships examined in the current study. On the other hand, the current study included a number of detailed observational assessments, especially of family interactions, that would be difficult to implement on a larger scale due to their cost and time-consuming nature. Another limitation related to the sample was the lack of measurement of pubertal development, which research indicates is an important factor in the development of anxiety and depression (e.g. Deardorff et al., 2007). To control for the influence of pubertal development on the development of anxiety and depression, future studies should consider including this factor in analyses.

Another possible limitation in terms of measurement of each variable was the relatively small amount of time (less than a year) between measurement of neighbourhood disadvantage, and measurement of mediating variables. Despite the measurement of neighbourhood disadvantage (IV) being temporally close to the measurement of temperament and maternal affective behavior (potential mediating variables), there are several reasons to believe the measure of
neighbourhood disadvantage is likely to satisfy temporal precedence in regard to the measurement of these variables. The SEIFA technical paper determined that most postcodes did not change significantly during the period from 2001 (when the previous SEIFA was compiled) to 2006. It was found that 80-90% of Postcodes did not change deciles or changed only by one decile. Even then, there have been numerous changes to the methodology of SEIFA in 2006 (compared to 2001) which may have affected compatibility and led to an exacerbation in differences found between 2001 and 2006. Thus participants are likely to have been exposed to similar levels of disadvantage between 2001 and 2006 indicating that neighbourhood disadvantage, as the independent variable significantly predated the measurement of temperament and parental affective behaviour.

It is worth noting that several of the significant findings discussed in this thesis did not include SES as a control variable. While there are arguments for and against inclusion of SES as a control variable, findings that do not involve the controlling of SES are less robust and therefore should be viewed as more exploratory than those which do control for SES. Thus they should perhaps be interpreted with more caution and conclusions reached should be seen as tentative. Future studies should aim to replicate these findings with larger samples and with more solid controls.

Limitations and future directions specific to each measure will now be discussed in terms of neighbourhood disadvantage, stressful life events, temperament and maternal affective behaviour.

14.4.2 Neighbourhood disadvantage

One limitation of the current thesis is that school effects and school quality were not measured and thus not accounted for. As most children in a neighbourhood would have attended the same schools, and given that schools are recognised to influence various outcomes in children (Goodman, Huang, Wade, & Kahn, 2003), it is possible that the perceived associations between neighbourhood disadvantage and internalising symptoms could actually be partly due to school effects. There are two broad ways of conceptualising the relationship between neighbourhoods and schools. The first is that the school environment is seen as independent of the neighbourhood environment and should thus be controlled for. The other approach views the
school environment as one aspect of the neighbourhood environment – most disadvantaged neighbourhoods also have disadvantaged schools, with disadvantaged students (Lupton, 2006). Thus these two factors – neighbourhood and school effects are highly linked, and findings associated with neighbourhoods may also apply to schools (Lupton, 2006). Future research could aim to assess whether school effects partly account for some of the associations between neighbourhood disadvantage and internalising symptoms.

14.4.3 Stressful life events

One limitation of the stress measure used was the self-report checklist (albeit combined with an interview measure). While this approach is generally considered an efficient, cost-effective estimate of overall subjectively experienced stress (e.g., Gibbs & Rude, 2004), reporting bias means that there could be a risk of over-identification of stressful life events in anxious or depressed individuals. However, it should be noted that the separation between internalising symptoms and stressful life events in the current thesis may have reduced the bias from internalising symptoms on reporting of stressful life events (Dalgleish & Watts, 1990). Future tests of stressful life events and neighbourhood influences would benefit from the use of an interview-based measure such as the Bedford Life Events and Difficulties Schedule (Brown & Harris, 1989), rather than a questionnaire measure, which would allow for more effective probing of stressful life events and could thus minimise the effect of reporting bias. Future research could also determine which specific stressful events (e.g., those resulting from neighbourhood danger, or events occurring in the home) are influencing risk for internalising symptoms.

Given the overlap between time periods measured by the stressful life events and internalising symptoms questionnaires, it is difficult to delineate the temporal associations between variables and consequentially the causal direction of relationships. Based on previous research, it is likely that the relationship between stressful life events and internalising symptoms is reciprocal: stressful life events are likely to cause increased internalising symptoms, while internalising symptoms are likely to predispose individuals to increased risk for stressful life events (Kim et al., 2003). Authors suggested that various factors associated with anxiety and depression (such as poor social relationships) could increase risk for stressful life events, such as bullying or failure at academic studies. The current study cannot provide further information regarding this issue.
However, future research should aim to measure stressful life events and internalising symptoms at multiple time points to give further clarity regarding the direction of influence.

14.4.4 Temperament

No significant relationship was detected between neighbourhood and Affiliation. It was hypothesised that neighbourhood social connectedness would explain any links detected between neighbourhood disadvantage and Affiliation. It is possible that the neighbourhood measure used in the current thesis was insensitive to levels of neighbourhood social connectedness. Future research could perhaps use a more specific measure of neighbourhood level social connectedness, such as the Vinson scale of Social Connectedness (Vinson, 2004), to examine its associations with Affiliation.

The current thesis was also unable to provide clear empirical data regarding the timing of neighbourhood influence on temperament. Future research could aim to measure temperament at several time points beginning early in life. Hart and colleagues (2008) argue that early childhood would be an important time to study these influences, when temperament is more open to change (Roberts & DelVecchio, 2000). Measurement at various time points from early childhood through adolescence would allow for an examination of whether temperament continues to change from childhood through adolescence as a function of neighbourhood disadvantage, and whether changes in temperament are related to changes in internalising symptoms throughout adolescence.

In terms of temperament and stress, future studies may wish to directly examine the potential associations between neighbourhood, stress and behavioural and emotional regulation in both parents and adolescents. Very little research has examined the links between these variables, which is surprising, given there are a number of ways in which disadvantaged neighbourhoods could inhibit effective behavioural and emotion regulation, as outlined in section 14.2.3.1. It is suggested that future research directly explores a possible multiple mediating pathway from (1) neighbourhood disadvantage to (2) stress and helplessness/hopelessness, to (3) Effortful control to (4) depression and anxiety. It would be recommended that multiple measurements of all variables be taken, spaced temporally, in order to further delineate the direction of causality.
14.4.5 Maternal affective behaviour

While it was argued that maternal affective behaviour would influence risk for depression and anxiety in adolescents, it was not possible to discount the possibility that that adolescents’ behaviour influenced maternal affective behaviour. This is somewhat problematic given that adolescents from disadvantaged neighbourhoods are more likely to demonstrate anxious, depressive and aggressive behaviour (Brooks-Gunn & Duncan, 1997). Thus perceived associations between neighbourhood disadvantage and maternal affective behaviour could simply be a product of the problematic behaviour of adolescents from these neighbourhoods. There is a general consensus amongst researchers that the relationship between parenting and children’s behaviour is bi-directional (e.g., Brody & Ge, 2001; Chiariello & Orvaschel, 1995), although some studies suggest that the influence of parenting on children may be slightly stronger than the influence of child factors on parenting (Hammen et al., 1990; Sheeber et al., 1997). The lack of a difference in frequency of maternal aggression between mothers from disadvantaged and less disadvantaged neighbourhoods may suggest that there is no difference in the misbehavior of adolescents from disadvantaged and less-disadvantaged neighbourhoods, or at least in their parents’ initial reactions to such behavior. However, the possibility cannot be discounted that differences in adolescents’ behaviour could partly explain the associations between internalising symptoms, neighbourhood effects and maternal affective behaviour. Measures of adolescent affective behaviour could be used in future research to account for this.

While both fathers and mothers took part in the family interaction task, the current thesis only used data from mothers, given that they formed the majority of participating parents (163, compared to 35 fathers). Under-recruitment of fathers is a common problem in developmental research (Cassano, Adrian, Veits, & Zeman, 2006), and the low sample of fathers in the current thesis lacked the power to compare the affective behaviour of mothers and fathers. Thus it was not possible to comprehensively assess the dynamics of adolescents’ family environments. Future research could aim to actively recruit fathers by explicitly requesting their participation, by seeking direct contact with fathers during the recruitment phase, or by requesting that both parents be present during interaction tasks.

14.5 Strengths
A number of methodological strengths in the research reported in this thesis address significant gaps in the existing knowledge base, and enable these findings to be interpreted with confidence, after due consideration of the limitations described in the previous section.

The current thesis provides new insight into neighbourhood disadvantage in the Australian setting. Mental illness is a significant problem in Australia, with prevalence rates for adolescent anxiety and depression standing at 14% and 13%, respectively (Boyd et al., 2003). While neighbourhoods are considered one important and potentially changeable risk factor for internalising disorders, very little research has examined Australian neighbourhoods and their influence on adolescents. This is a significant problem, as findings cannot always be accurately generalised to all countries. For instance, studies from countries that provide welfare, such as some European countries, less reliably find a significant influence of neighbourhood on depression than studies conducted in the United States (Kim, 2008), and it could be assumed that higher levels of welfare might lead to equality in the neighbourhood context. The current thesis adds to only two studies conducted so far demonstrating that Australian neighbourhoods are associated with significant differences in mental health of children and adolescents (Homel & Burns, 1989; Edwards, 2005). The current study extends these findings by demonstrating differences in stress, parenting and temperament between disadvantaged and less-disadvantaged Australian neighbourhoods. The findings from the current study also indicate that findings from studies conducted in the USA and other countries may be generalizable across different national contexts, including Australia.

The design of this thesis conferred several benefits. Of great advantage was its ability to study neighbourhood effects longitudinally, with the majority of the studies conducted so far being cross-sectional (Mair et al., 2008). Longitudinal research has the benefit of being able to minimise the influence of reverse causation, and allows for greater confidence in the aetiological significance of the findings. Furthermore, the follow-up period of approximately four years allowed a reasonable amount for time for the prospective influence of risk factors assessed at T1 to exert their influence on internalising symptoms. The use of independent measures was another design strength. That is, the various measures did not rely on reporting from the individual, thus lessening the problem of confounding, and associated problems with Type I error. While this does not provide as thorough investigation of subjective neighbourhood effects, it provides a better measure of objective neighbourhood effects, and thus perhaps may be more useful in the development of community level intervention and prevention programs.
The current thesis provided a much-needed examination of the effect of neighbourhood disadvantage on anxiety. A great deal of neighbourhood research has examined the relationship between neighbourhood and depression, while very little has focused on anxiety. Findings of this thesis indicate that many of the patterns observed in studies of neighbourhood and depression may also hold for anxiety, providing further evidence of the impact of neighbourhood on mental health outcomes. The current thesis was relatively unique in that it observed these neighbourhood effects in families of many different levels of SES. Many of the studies conducted so far (e.g., those resulting from the Yonkers study) observed neighbourhood effects in predominantly low-income families, which could bias results, and may prevent these studies from being as generalizable to more affluent families.

The current study is also important in its examination of mediating relationships in regard to neighbourhood influences. These analyses allow for an understanding of the pathways through which neighbourhoods influence risk for internalising in adolescents (Ingoldsby & Shaw, 2002). Examination of mediating pathways at both the neighbourhood and individual levels allows for the establishment of plausibility of associations and the relative importance and timing of multiple pathways. This is likely to contribute to the development of more effective and targeted prevention and intervention programmes. Furthermore, the current thesis used direct mediational analyses, which provide a more rigorous test of mediating relationships. The use of a more contemporary form of mediation analysis (i.e., bootstrapping) also provided more statistical power and more accurate confidence intervals when testing indirect effects (MacKinnon et al., 2004; Pituch et al., 2006).

This thesis was the first to use an interview measure to assess stressful life events, and the first to use observational measures of parenting style, which is likely to provide a more accurate assessment of these factors. Interview measures of stressful life events allow for more confidence in the assertion that stressful life events act as a mediator between neighbourhood and internalising in adolescents. Observational measures of parenting are generally considered the most reliable and valid way to measure parent interaction styles (Zeman et al., 2007) as they are more objective, and provides a more ‘natural’ assessment of behaviour than questionnaire data (Bakeman & Gnisci, 2006; Morris et al., 2006). Observational measures also allow for behaviour to be measured that participants may not be consciously aware of, such as non-verbal behaviour (Bakeman & Gnisci, 2006; Morris et al., 2006). Finally observational measures are
thought to be less biased by social desirability than questionnaire methods, as participants have less control over their behaviour during tasks such as video-taped assessments (Morris et al., 2006). Finally, the examination of both duration and frequency of maternal expressed emotion also allowed for a more detailed examination of parenting processes, providing a more comprehensive understanding of the way in which parent behaviour is influenced by disadvantaged environments and the way in which this behaviour impacts on adolescents.

The current study is also the first to examine temperament as a mediator between neighbourhood disadvantage and internalising symptoms. As described in Chapter 8.3.1, the method by which adolescents were selected to participate in this research over-sampled for adolescents demonstrating more extreme scores on temperament dimensions. This approach overcomes the limitations of research based on both representative community samples (which are likely to be dominated by average levels of risk factors, and may lack the power to detect significant relationships) and clinical samples (which may over-represent extreme levels of risk, and thus findings may not be generalisable to non-clinical populations). The current thesis was representative of a range of temperament dimensions, while still being representative of members in in the general community.

14.6 Clinical implications

The current study provides evidence to suggest which populations to target in intervention and prevention programmes and also what factors to target. Specifically there was some evidence to suggest that adolescents from disadvantaged neighbourhoods should be preferentially targeted, given their increased risk for internalising symptoms (although this conclusion should be seen as speculative, given the small effect sizes observed). There are several ways in which this population could be helped, including improving neighbourhood conditions, decreasing stress and risk for stressful life events, encouraging parents to engage in more helpful parenting practises, and even aiding children to better manage their maladaptive temperaments. Each of these areas will be discussed in turn.

The current thesis provides tentative evidence that disadvantaged neighbourhoods are relatively harmful places in which to reside. Previous research suggests that, of the various neighbourhood factors thought to increase risk for internalising symptoms, perhaps the most important factors are neighbourhood disorder, low community support and cohesion, and poor quality of the built environment. There are many ways that these factors could be addressed. For instance,
governments could aim to provide safer neighbourhood environments, better schools, and increased economic opportunities for residents of disadvantaged neighbourhoods, which may alleviate the negative consequences of growing up in these neighbourhoods (Jarrett, 1999). Other strategies could include further integration of scattered-site public housing in less-disadvantaged neighbourhoods, and encouraging higher SES families to move to disadvantaged neighbourhoods, thereby changing the socio-economic mix of the area (Leventhal & Brooks-Gunn, 2003).

The findings of the current thesis also indicate that adolescents (and perhaps even more so those from disadvantaged neighbourhoods) would benefit from programs aimed at reducing exposure to negative life events and by providing more positive ways of coping with stressful events (White & Roosa, 2012). Interventions targeting aggressive parenting could also be effective. While most prevention and intervention programs place most of the focus on adolescents themselves, some programs have been developed to include parent education sessions aimed to increase the effectiveness of these programs (e.g., Roosa et al., 2010). These interventions often aim to minimize severe family conflict and increase responsive and warm parent-adolescent relationships. The current thesis indicates that interventions of this nature should not only focus on severe family conflict, but also focus on even relatively mild family conflict (as observed during the EPI and PSI). As described below, interventions aimed at behavioural and emotional regulation could also be of benefit for parents (and therefore their children).

The current thesis suggests that various temperament dimensions could be targets for interventions aimed at minimising risk for depression and anxiety. In particular, interventions for both parents and adolescents aimed at behavioural and emotion regulation could be helpful targets. Mindfulness is one intervention that has been shown to increase Effortful Control (Tang & Posner, 2009), and reduce stress and anxiety (Grossman, Niemann, Schmidt, & Walach, 2004). Thus this could be a potentially powerful way of reducing risk for internalising disorders in adolescents low on Effortful Control and even parents with poor emotional and behavioural regulation. Other interventions have targeted specific temperament dimensions such as Negative Affectivity or Impulsiveness and associated behaviours (Conrod, Stewart, Comeau, & Maclean, 2006; Conrod, Castellanos, & Mackie, 2008). These interventions, aimed at minimising alcohol misuse, were found to be more effective than less targeted interventions (e.g., Foxcroft, Ireland, Lister-Sharp, Lowe, & Breen, 2003). Variations on these programs could possibly be used to reduce adolescent depression and anxiety in adolescents with maladaptive temperaments.
It is likely that prevention and intervention programs targeting a range of risk factors, and even tailoring these programs to the specific risk factors of individuals, could be particularly effective at reducing the prevalence of internalising disorders in adolescents. The Harlem Children’s Zone is one intervention that aims to address multiple risk factors in disadvantaged neighbourhoods, such as poor parenting, health, and school quality (Whitehurst & Croft, 2010). It began as a small project and by 2007 had grown to include 100 neighbourhood blocks with millions of dollars in funding. Another US based intervention initiated in 2010 – the Promise Neighbourhoods program - aims to improve educational and developmental outcomes in children from disadvantaged communities through the use of a variety of empirically supported programs and interventions targeting a range of risk factors (Whitehurst & Croft, 2010). The current thesis suggests that perhaps additional interventions should be added to the existing components of both these interventions, including those aimed at reducing stressful life events, aggressive interactions between parents, and the impact of stress on the development of temperament. While these programs are promising, it is perhaps too soon to determine whether they are effective at reducing the impact of poverty and neighbourhood disadvantage on children. However, there is some evidence that the Harlem Children’s Zone has improved various academic outcomes in adolescents (Dobbie & Fryer Jr, 2009).

14.7 Conclusion

In conclusion, the research reported in this thesis provides evidence that disadvantaged neighbourhoods differ from less disadvantaged neighbourhoods in several key ways, all of which indicate that disadvantaged neighbourhoods are less conducive to healthy and positive development in children and adolescents. There was prospective evidence to support a model in which stressful life events, maternal aggressive behaviour, and maladaptive temperament dimensions (low Surgency and Effortful Control, and high Negative Affectivity) mediated the relationship between neighbourhood disadvantage and internalising symptoms. Each of these factors was found to operate at different periods during adolescence. Temperament appears to be important earlier in adolescence, maternal affective behaviour seems to be important during mid- to later-adolescence, while stressful life events appear to act throughout adolescence. These findings suggest that the neighbourhood environment is likely to influence adolescents both directly and indirectly through its effects on more proximal and individual risk factors. The current thesis adds to the growing literature that highlights the importance of the neighbourhood environment in playing a significant role in the development of depression and anxiety in
adolescence. It is recommended that future research continues to investigate this model to provide further understanding of the timing of the effects observed in the current thesis. This could be achieved through measurement of factors at several time points, and an examination of these factors in both childhood and adolescence. It is hoped that the identification of prospective risk factors and their relationship to neighbourhood disadvantage in this thesis contributes to the development of preventive interventions that are effective in reducing the rates of depression and anxiety in adolescence.
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APPENDIX A: MEASURES

Appendix A1: Centre for Epidemiological Studies – Depression Scale

For each item below, please indicate how often you have felt or behaved that way during the past week.

1 = Rarely or not at all (less than 1 day).
2 = Some or a little of the time (1-2 days).
3 = Occasionally or a moderate amount of time (3-4 days).
4 = Most or all of the time (5-7 days).

<table>
<thead>
<tr>
<th></th>
<th>Rarely or Not at All</th>
<th>Sometimes</th>
<th>Occasionally</th>
<th>Most of the Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I was bothered by things that don't usually bother me</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>I did not feel like eating; my appetite was poor</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>I felt that I could not shake off the blues even with help from my family or friends</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>I felt that I was just as good as other people</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td>I had trouble keeping my mind on what I was doing</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6</td>
<td>I felt depressed</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7</td>
<td>I felt that everything I did was an effort</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>I felt hopeful about the future</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9</td>
<td>I thought my life had been a failure</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10</td>
<td>I felt fearful</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11</td>
<td>My sleep was restless</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12</td>
<td>I was happy</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13</td>
<td>I talked less than usual</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>14</td>
<td>I felt lonely</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>15</td>
<td>People were unfriendly</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>16</td>
<td>I enjoyed life</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>17</td>
<td>I had crying spells</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>18</td>
<td>I felt sad</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>19</td>
<td>I felt that people disliked me</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>20</td>
<td>I could not get “going”</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
Appendix A2: Beck Anxiety Inventory

**INSTRUCTIONS:** Below is a list of common symptoms of anxiety. Please read each item in the list carefully. Indicate how much you have been bothered by each symptom during the **PAST WEEK, INCLUDING TODAY** by circling the corresponding number in the column next to each symptom.

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>Mildly It did not bother me much</th>
<th>Moderately It was very unpleasant but I could stand it</th>
<th>Severely I could barely stand it</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Numbness or tingling</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. Feeling hot</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. Wobbliness in legs</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. Unable to relax</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. Fear of the worst happening</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. Dizzy or lightheaded</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. Heart pounding or racing</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8. Unsteady</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9. Terrified</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10. Nervous</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11. Feeling of choking</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12. Hands trembling</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13. Shaky</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>14. Fear of losing control;</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>15. Difficulty breathing</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>16. Fear of dying</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>17. Scared</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>18. Indigestion or discomfort in abdomen</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>19. Faint</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>20. Face flushed</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>21. Sweating (not due to heat)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
Appendix A3: Stressful life events interview and questionnaire

**LIFE EVENTS – Supplement (see LIFE Events Questionnaire):**

v. Event Number (1 – 31): __________ (note: if more than one event, use extra Life Events Supplement provided in the Home Ax kit and insert in appropriate section. If more than four, query the four most significant events, positive and negative).

vi. Happened to: ________ Yourself ________ Mother/Father ________ Other Household Member ________ Other Close Relative ________ Other Close Friend Please describe the event:

vii. When did this happen? (M/Y) ______ / ______

viii. Was that mostly a good or mostly a bad experience at the time it happened? a. Mostly good c. Not applicable b. Mostly bad d. Don’t know

 ix. How much has this event affected your life?

a. Not at all b. A little c. Some d. A lot e. Not applicable f. Don’t know

*(Interviewer completes afterwards):*

6. Does the subject’s description of the event match the item as intended in the questionnaire? ______

   Yes ________ No

7. How much would this event affect the life of an average person?

a. Not at all b. A little c. Some d. A lot e. Not applicable f. Don’t know

8. To what extent was the stressor a result of the subject’s behaviour (when in doubt, rate at least partially determined by subject’s behaviour)?

a. Completely independent of subject’s behaviour (clearly due to external circumstances

b. At least partially determined by the subject’s behaviour

c. Stressor was due to the subject being depressed

*From Lewinsohn, P. M. - Oregon Research Institute*
Life Events Questionnaire

Some events are listed on the left side of the chart below. We would like to know which of them have happened in your life and to whom it has happened. If an event has happened, please put a check (X) in the box under the person(s) it happened to. Our definition of "close relative" is a first-degree relative or other relative that you see or call at least once a month. A "close friend" is someone you see or call at least 3 times a month.

<table>
<thead>
<tr>
<th>Event</th>
<th>Yourself</th>
<th>Mother/Father</th>
<th>Other Household Member(s)</th>
<th>Other Close Relative</th>
<th>Other Close Friend</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Died</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Got seriously sick or injured</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Had illness or accident requiring hospitalization</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Unable to get treatment for illness or injury</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Victim of crime, violence or assault</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Saw a crime or accident in which someone was injured, killed, or assaulted</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>7. Got in serious trouble with the law</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Went to jail</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Appearance in court</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Started working</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Job change (promotion/demotion)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Lost job</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. Started going to new school</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. Academic failure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. Significant change in financial situation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16. Major break-up with girl/boyfriend</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17. Divorced/separated</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yourself</td>
<td>Mother/Father</td>
<td>Other Household Member(s)</td>
<td>Other Close Relative</td>
<td>Other Close Friend</td>
</tr>
<tr>
<td>---</td>
<td>----------</td>
<td>---------------</td>
<td>---------------------------</td>
<td>----------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>18.</td>
<td>Married or started living with someone</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19.</td>
<td>Got pregnant (or got someone pregnant)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20.</td>
<td>Abortion / Miscarriage / Stillbirth</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21.</td>
<td>Birth or adoption of child</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>22.</td>
<td>Lost a close friend</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23.</td>
<td>Major argument or fights</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24.</td>
<td>Had a problem with drugs or alcohol</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25.</td>
<td>Had a mental or emotional problem</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>26.</td>
<td>Moved / changed residence</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>27.</td>
<td>Important possession stolen</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>28.</td>
<td>Got in car, bike, or other accident</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>29.</td>
<td>Home damaged by fire, flood, or other disaster</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30.</td>
<td>Other major stressful event (Describe: )</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>31.</td>
<td>None of the items apply</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix A4: Early Adolescent Temperament Questionnaire - Revised

**Directions**

On the following page you will find a series of statements that people might use to describe themselves. The statements refer to a wide number of activities and attitudes.

For each statement, please circle the answer that best describes how true each statement is for you. There are no best answers. People are very different in how they feel about these statements. Please circle the first answer that comes to you.

You will use the following scale to describe how true or false a statement is about you:

<table>
<thead>
<tr>
<th>Circle number:</th>
<th>If the statement is:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Almost always untrue of you</td>
</tr>
<tr>
<td>2</td>
<td>Usually untrue of you</td>
</tr>
<tr>
<td>3</td>
<td>Sometimes true, sometimes untrue of you</td>
</tr>
<tr>
<td>4</td>
<td>Usually true of you</td>
</tr>
<tr>
<td>5</td>
<td>Almost always true of you</td>
</tr>
</tbody>
</table>

**NOTE.** Please make certain to answer all questions on BOTH SIDES of the page.

Please tell us:

Your date of birth: __________ 

Your gender: M / F 

Family ID code: __________ (please leave blank)
How true is each statement for you?

<table>
<thead>
<tr>
<th>Statement</th>
<th>Almost always untrue</th>
<th>Usually untrue</th>
<th>Sometimes true, sometimes untrue</th>
<th>Usually true</th>
<th>Almost always true</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) It is easy for me to really concentrate on homework problems.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2) I feel pretty happy most of the day.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3) I think it would be exciting to move to a new city.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4) I like to feel a warm breeze blowing on my face.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5) If I’m mad at somebody, I tend to say things that I know will hurt their feelings.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6) I notice even little changes taking place around me, like lights getting brighter in a room.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7) I have a hard time finishing things on time.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8) I feel shy with kids of the opposite sex.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9) When I am angry, I throw or break things.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10) It’s hard for me not to open presents before I’m supposed to.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11) My friends seem to enjoy themselves more than I do.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12) I tend to notice little changes that other people do not notice.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13) If I get really mad at someone, I might hit them.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14) When someone tells me to stop doing something, it is easy for me to stop.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15) I feel shy about meeting new people.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16) I enjoy listening to the birds sing.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17) I want to be able to share my private thoughts with someone else.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18) I do something fun for awhile before starting my homework, even when I’m not supposed to.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19) I wouldn’t like living in a really big city, even if it was safe.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20) It often takes very little to make me feel like crying.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21) I am very aware of noises.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>22) I tend to be rude to people I don’t like.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23) I like to look at the pattern of clouds in the sky.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24) I can tell if another person is angry by their expression.</td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
25) It bothers me when I try to make a phone call
and the line is busy. 1 2 3 4 5
26) The more I try to stop myself from doing
something I shouldn't, the more likely I am to
do it. 1 2 3 4 5
27) I enjoy exchanging hugs with people I like. 1 2 3 4 5
28) Skiing fast down a steep slope sounds scary
to me. 1 2 3 4 5
29) I get sad more than other people realize. 1 2 3 4 5
30) If I have a hard assignment to do, I get started
right away. 1 2 3 4 5
31) I will do most anything to help someone I care
about. 1 2 3 4 5
32) I get frightened riding with a person who likes
to speed. 1 2 3 4 5
33) I like to look at trees and walk amongst them. 1 2 3 4 5
34) I find it hard to shift gears when I go from one
class to another at school. 1 2 3 4 5
35) I worry about my family when I'm not with
them. 1 2 3 4 5
36) I get very upset if I want to do something and
my parents won't let me. 1 2 3 4 5
37) I get sad when a lot of things are going wrong. 1 2 3 4 5
38) When trying to study, I have difficulty tuning
out background noise and concentrating. 1 2 3 4 5
39) I finish my homework before the due date. 1 2 3 4 5
40) I worry about getting into trouble. 1 2 3 4 5
41) I am good at keeping track of several different
things that are happening around me. 1 2 3 4 5
42) I would not be afraid to try a risky sport, like
deep-sea diving. 1 2 3 4 5
43) It's easy for me to keep a secret. 1 2 3 4 5
44) It is important to me to have close
relationships with other people. 1 2 3 4 5
45) I am shy. 1 2 3 4 5
46) I am nervous of some of the kids at school
who push people into lockers and throw your
books around. 1 2 3 4 5
47) I get irritated when I have to stop doing
something that I am enjoying. 1 2 3 4 5
48) I wouldn't be afraid to try something like
mountain climbing. 1 2 3 4 5
49) I put off working on projects until right before
they're due. 1 2 3 4 5
50) When I'm really mad at a friend, I tend to
explode at them. 1 2 3 4 5
51) I worry about my parent(s) dying or leaving 1 2 3 4 5
<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>52) I enjoy going places where there are big crowds and lots of excitement.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>53) I am not shy.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>54) I am quite a warm and friendly person.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>55) I feel sad even when I should be enjoying myself, like at Christmas or on a trip.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>56) It really annoys me to wait in long lines.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>57) I feel scared when I enter a darkened room at home.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>58) I pick on people for no real reason.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>59) I pay close attention when someone tells me how to do something.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>60) I get very frustrated when I make a mistake in my school work.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>61) I tend to get in the middle of one thing, then go off and do something else.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>62) It frustrates me if people interrupt me when I'm talking.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>63) I can stick with my plans and goals.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>64) I get upset if I'm not able to do a task really well.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>65) I like the crunching sound of autumn leaves.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
Appendix A5: Pleasant Events Schedule

**Pleasant Events Schedule (PSE)**

This questionnaire is designed to find out about things you enjoy doing. Go down the list and rate how pleasant each activity is for you to do with your parent/child. *If you haven't done the activity with your parent/child, rate how pleasant you think it would be.* There are many activities on the list, so don't spend too much time thinking about each one. Keep in mind that there are no right or wrong answers—everyone's answers will be different.

<table>
<thead>
<tr>
<th>Activity</th>
<th>Not Pleasant (0)</th>
<th>Somewhat Pleasant (1)</th>
<th>Very Pleasant (2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Being in the country or the mountains</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Playing table games (pool, ping pong, air hockey, etc)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Doing art work (painting, sculpture, drawing, movie-making)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Taking pictures</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Going to a sporting event</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Having a family reunion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Doing volunteer work; working on community service projects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Singing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Waterskiing, surfing, scuba diving</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Taking a walk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Doing craft work (pottery, jewellery, leather, beads, weaving, etc)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Cooking meals</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. Doing heavy outdoor work (cutting or chopping wood, etc)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Rate how pleasant this is, or would be, to do with your parent by putting a tick (✓) in the corresponding box</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>-------------------------------------------------------------------------------------------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Not Pleasant</td>
<td>Somewhat Pleasant</td>
<td>Very Pleasant</td>
</tr>
<tr>
<td>14.</td>
<td>Taking a trip or vacation</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>15.</td>
<td>Bicycling</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>16.</td>
<td>Playing cards</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>17.</td>
<td>Going to the movies</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>18.</td>
<td>Giving a party or get-together</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>19.</td>
<td>Making a major purchase (car, bicycle, stereo, etc)</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>20.</td>
<td>Swimming</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>21.</td>
<td>Acting</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>22.</td>
<td>Going on outings (to the park, a picnic, a barbecue, etc)</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>23.</td>
<td>Restoring or building furniture</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>24.</td>
<td>Going to a restaurant</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>25.</td>
<td>protesting social, political or environmental causes</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>26.</td>
<td>Going to a museum</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>27.</td>
<td>Playing golf, tennis, handball or racquetball</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>28.</td>
<td>Being at the beach</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>29.</td>
<td>Working on machines (cars, bikes, motorcycles, tractors, etc)</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>30.</td>
<td>Going to church functions (socials, revivals, bazaars, etc)</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>31.</td>
<td>Going to a health club, sauna, hot tub, etc</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>32.</td>
<td>Snow skiing</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rate how pleasant this is, or would be, to do with your parent by putting a tick (✓) in the corresponding box</td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Not Pleasant 0</td>
<td>Somewhat Pleasant 1</td>
</tr>
<tr>
<td>33.</td>
<td>Dancing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>34.</td>
<td>Gardening, landscaping, or doing yard work</td>
<td></td>
<td></td>
</tr>
<tr>
<td>35.</td>
<td>Playing computer or video games</td>
<td></td>
<td></td>
</tr>
<tr>
<td>36.</td>
<td>Hiking or camping</td>
<td></td>
<td></td>
</tr>
<tr>
<td>37.</td>
<td>Playing musical instruments</td>
<td></td>
<td></td>
</tr>
<tr>
<td>38.</td>
<td>Bowling</td>
<td></td>
<td></td>
</tr>
<tr>
<td>39.</td>
<td>Going to a fair, carnival, circus, zoo, or amusement park</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40.</td>
<td>Doing &quot;odd jobs&quot; around the house</td>
<td></td>
<td></td>
</tr>
<tr>
<td>41.</td>
<td>Rock climbing or mountaineering</td>
<td></td>
<td></td>
</tr>
<tr>
<td>42.</td>
<td>Playing board games (Monopoly, chess, etc)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>43.</td>
<td>Hunting or fishing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>44.</td>
<td>Going shopping (mall, auctions, garage sales, etc)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>45.</td>
<td>Running, jogging, or doing other fitness activities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>46.</td>
<td>Boating (canoeing, kayaking, motorboating, sailing, etc)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>47.</td>
<td>Attending a concert, ballet, or play</td>
<td></td>
<td></td>
</tr>
<tr>
<td>48.</td>
<td>Playing cricket</td>
<td></td>
<td></td>
</tr>
<tr>
<td>49.</td>
<td>Rearranging or redecorating my room or the house</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50.</td>
<td>Horseback riding</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix A6: Issues Checklist

Below is a list of things that sometimes get talked about at home. We would like you to look carefully at each topic on the left-hand side of the page and decide whether you and your child/parent together have talked about that topic at all during the last 2 weeks.

**STEP 1**
If you and your child/parent together have discussed it during the last 2 weeks, circle the **YES** to the right of the topic. If you and your child/parent together have **not** discussed it during the last 2 weeks, circle the **NO** to the right of the topic.

**STEP 2**
Now, we would like you to go back over the list of topics. For those topics for which you circled **YES**, please answer the two questions on the right-hand side of the page:

How many times during the last 2 weeks did the topic come up?
How "hot" are the discussions?

<table>
<thead>
<tr>
<th>TOPIC</th>
<th>STEP 1</th>
<th>STEP 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Have you discussed this topic</td>
<td>How many times during the last</td>
</tr>
<tr>
<td></td>
<td>together during the last 2</td>
<td>2 weeks did the topic come</td>
</tr>
<tr>
<td></td>
<td>weeks?</td>
<td>up?</td>
</tr>
<tr>
<td>a. Telephone calls</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>b. Time for going to bed</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>c. Cleaning up bedroom</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>d. Doing homework</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>e. Putting away clothes</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>f. Using the television</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>g. Cleanliness (washing,</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>showers, brushing teeth)</td>
<td>NO</td>
<td></td>
</tr>
<tr>
<td>TOPIC</td>
<td>STEP 1</td>
<td>STEP 2</td>
</tr>
<tr>
<td>------------------------------------------------</td>
<td>-------------------------------------------</td>
<td>--------</td>
</tr>
<tr>
<td>h. Which clothes to wear</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>i. How neat clothing looks</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>j. Making too much noise at home</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>k. Table manners</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>l. Fighting with brothers or sisters</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>m. Cursing</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>n. How money is spent</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>o. Picking books or movies</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>p. Allowance</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>q. Smoking marijuana</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>r. Going places without parents (shopping, movies, etc)</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>s. Playing the stereo</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>t. Turning off lights in the house</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>u. Drugs</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>v. Taking of games, records and things</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>w. Drinking beer or other liquor</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>x. Buying records, games and things</td>
<td>YES NO</td>
<td></td>
</tr>
<tr>
<td>TOPIC</td>
<td>STEP 1</td>
<td>STEP 2</td>
</tr>
<tr>
<td>-------</td>
<td>--------</td>
<td>--------</td>
</tr>
<tr>
<td></td>
<td>Have you discussed this topic together during the last 2 weeks?</td>
<td>How many times during the last 2 weeks did the topic come up?</td>
</tr>
<tr>
<td></td>
<td>YES</td>
<td>NO</td>
</tr>
<tr>
<td>y.</td>
<td>Going on dates</td>
<td>YES</td>
</tr>
<tr>
<td>z.</td>
<td>Who should be friends</td>
<td>YES</td>
</tr>
<tr>
<td>aa.</td>
<td>Selecting new clothes</td>
<td>YES</td>
</tr>
<tr>
<td>bb.</td>
<td>Sex</td>
<td>YES</td>
</tr>
<tr>
<td>cc.</td>
<td>Coming home on time</td>
<td>YES</td>
</tr>
<tr>
<td>dd.</td>
<td>Getting to school on time</td>
<td>YES</td>
</tr>
<tr>
<td>ee.</td>
<td>Getting low grades in school</td>
<td>YES</td>
</tr>
<tr>
<td>ff.</td>
<td>Getting in trouble in school</td>
<td>YES</td>
</tr>
<tr>
<td>gg.</td>
<td>Lying</td>
<td>YES</td>
</tr>
<tr>
<td>hh.</td>
<td>Helping out around the house</td>
<td>YES</td>
</tr>
<tr>
<td>ii.</td>
<td>Talking back to parents</td>
<td>YES</td>
</tr>
<tr>
<td>jj.</td>
<td>Getting up in the morning</td>
<td>YES</td>
</tr>
<tr>
<td>kk.</td>
<td>Teenager bothering parents when they want to be left alone</td>
<td>YES</td>
</tr>
<tr>
<td>ll.</td>
<td>Parents bothering teenager when he/she wants to be left alone</td>
<td>YES</td>
</tr>
<tr>
<td>mm.</td>
<td>Putting feet on the furniture</td>
<td>YES</td>
</tr>
<tr>
<td>nn.</td>
<td>Messing up the house</td>
<td>YES</td>
</tr>
<tr>
<td>oo.</td>
<td>What time to have meals</td>
<td>YES</td>
</tr>
<tr>
<td>TOPIC</td>
<td>STEP 1</td>
<td>STEP 2</td>
</tr>
<tr>
<td>-------</td>
<td>--------</td>
<td>--------</td>
</tr>
<tr>
<td>pp.</td>
<td>How to spend free time</td>
<td>YES</td>
</tr>
<tr>
<td></td>
<td>How many times during the last 2 weeks did the topic come up?</td>
<td>1 2 3 4 5</td>
</tr>
<tr>
<td>qq.</td>
<td>Smoking cigarettes</td>
<td>YES</td>
</tr>
<tr>
<td></td>
<td>How many times during the last 2 weeks did the topic come up?</td>
<td>1 2 3 4 5</td>
</tr>
<tr>
<td>rr.</td>
<td>Earning money away from house</td>
<td>YES</td>
</tr>
<tr>
<td></td>
<td>How many times during the last 2 weeks did the topic come up?</td>
<td>1 2 3 4 5</td>
</tr>
<tr>
<td>ss.</td>
<td>What teenager eats</td>
<td>YES</td>
</tr>
<tr>
<td></td>
<td>How many times during the last 2 weeks did the topic come up?</td>
<td>1 2 3 4 5</td>
</tr>
<tr>
<td>tt.</td>
<td>In the blanks below, list any topics that apply to you and your child which have not been listed above:</td>
<td>YES</td>
</tr>
<tr>
<td>uu.</td>
<td></td>
<td>YES</td>
</tr>
<tr>
<td></td>
<td>How &quot;hot&quot; are the discussions?</td>
<td>Calm</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>
## Appendix A7: LIFE Code List

<table>
<thead>
<tr>
<th><strong>Content Codes</strong></th>
<th><strong>Affect Codes</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>10 Validation</td>
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<td>14 Affection</td>
<td>1 Anger</td>
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<td>15 Solicitous</td>
<td>2 Anxious</td>
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<td>16 Humour</td>
<td>3 Dysphoric</td>
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<td>18 Approve</td>
<td>4 Pleasant</td>
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<td>21 Complaint</td>
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<td>24 Negative Substance</td>
<td>7 Caring</td>
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<td>9 Belligerence</td>
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<td>31 Command Unaccountable</td>
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<td>37 Non-Comply</td>
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<td>46 Self Statement</td>
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<td>54 Conversation Tactic</td>
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<td>61 Problem Statement</td>
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<td>64 Propose Solution</td>
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<td>65 Teach</td>
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<td>78 Talk</td>
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<td>81 Inaudible</td>
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<td>98 Dummy</td>
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Aggressive Behaviour Construct

Affect codes 0, 1, or 9 + Content codes 10, 14, 15, 16, 18, 21, 23, 24, 25, 26, 28, 30, 31, 35, 37, 46, 48, 54, 61, 64, 65, 66, 76, 78, 81, or 98

OR

Affect code 5 + Content codes 23, 25, or 26.

Dysphoric Behaviour Construct

Affect codes 2, 3, or 8 + Content codes 10, 14, 15, 16, 18, 21, 23, 24, 25, 26, 28, 30, 31, 35, 37, 46, 47, 48, 54, 61, 64, 65, 66, 76, 78, 81, or 98

OR

Affect code 5 + Content codes 21, or 48.

Positive Behaviour Construct

Affect codes 4, 6, 7 + Content codes 10, 14, 15, 16, 18, 21, 23, 24, 25, 26, 28, 30, 31, 35, 37, 46, 47, 48, 54, 61, 64, 65, 66, 67, 76, 78, 81, or 98

OR

Affect code 5 + Content codes 10, 14, 16, 18, or 47.
Earlier this year, your child participated in the "The ORYGEN Adolescent Emotional Development Study" by completing a short survey about themselves at school. As you will remember, this study is investigating one of the key underlying causes of the onset of mental disorder in adolescence, namely difficulties with regulating moods and emotions. Many of the mental disorders that are highly prevalent in adolescence, namely depression, anxiety and substance use, are all associated with clear problems in regulating moods and emotions.

As we noted when your child participated earlier this year, a key feature of this study is that it is longitudinal, meaning that some of the participants would be asked to participate again over their teenage years. This study design is critical because it will enable us to understand how symptoms of mental disorder have emerged in our sample, and to explore the early signs of these disorders in relation to emotional regulation.

In this phase of the study, we will be visiting a smaller sample of our participating young people (around 400). These young people have been selected to represent the full range of scores measured by the screening phase of the study. That is, these young people demonstrate the different ways in which people regulate their moods and emotions.

During this visit, participating young people will be interviewed by a member of our team and asked a range of detailed questions relating to their mental health. Both they and their parents will be asked to fill out a number of questionnaires relating to mood, coping and relationships.

Details of this phase of the study are provided below.

Whilst there will be no direct benefit to your child in participating in this study, the "The ORYGEN Adolescent Emotional Development Study" provides us with an outstanding opportunity to help us to identify young people at high risk for mental health problems and help us develop preventative programs that enhance young people's awareness and regulation of their moods and promote healthy emotional development. In addition, it will inform early intervention strategies aimed at those most in need.

Before either you or your child participates in this study, you will be asked to provide written consent by signing two consent forms. The first of these indicates that you agree to your own participation in the study, while the second indicates that you agree to your child's participation in the study. By signing the consent forms, you are also indicating that you fully understand the conditions of the study. These are enclosed. Your child will also be asked to confirm their wish to participate prior to the study being conducted. It is important to know that participation in this study is entirely voluntary. Also, if you do not wish to participate, but are happy for your child to do so, your child can still participate.

Moreover, participation in this phase does not mean that you are required to take part in any other phase of the research, you can make your mind up about that at the time.

In order to recompense you for your time and the time offered by your child, you will receive $50 if you agree for your child to participate.

If you agree to your child participating in this phase of the study, two members of our team will visit you and administer a more thorough psychiatric assessment. A psychiatric assessment involves questions relating to your child's experience of symptoms of mental health problems such as anxiety and depression. Your child will be asked about both current and past psychological difficulties. The assessment is organised so that your child will only be asked questions of relevance to them. This means that if your child denies the experience of a particular symptom, they will not be asked any subsequent questions regarding this symptom or related symptoms. Your child will also be asked to fill in a number of short questionnaires to enable us to get a better understanding of his or her psychological profile.

During this visit, you will also be asked to participate by filling in a number of questionnaires which ask about your child's emotions, the way they cope with things, and your interactions with your child. There will also be some questions about yourself, your own moods, and any substances you might use.

Importantly, if we find that your child is currently experiencing any psychological difficulties, we will let you know immediately and can help facilitate support for your child if necessary and if you would like us to do so. Again, however, we would emphasise that if we contact you in the future to invite your child to participate in further research, this does not necessarily mean that your child is at risk for anxiety and/or depression.

You should also know that these procedures have all been well tolerated by young people taking part in similar studies in the past and will assist us in understanding how young people regulate their moods over time and how this links with problems in adolescence.

Altogether, this phase of the study should take about two hours to complete with interviews with yourself (if you consent) and your child being conducted simultaneously. This can be done all at once, or over a series of meetings.

In two years when your child is 14 years old, we will re-administer this assessment after contacting you and making sure that this is still okay.
Any information collected by the researchers is strictly confidential. All of the information provided by you and your child will be stored securely at ORYGEN Youth Health. Within the limits of the law, we will keep all information confidential and will not provide information about any individual participants to anyone. Participants have already been given a numerical identifier and all subsequent data will be stored in locked files and will be identified using these numerical identifiers, not the names of the parents and students participating in the study or any other identifying information. Only investigators and a small number of the research staff working on the study will have access to the raw data. Parents will not have direct access to information provided by their child in the survey. The results of the project will only be reported in ways that do not identify individual participants. Moreover, all data provided by you and your child will be destroyed 5 years after the date of the last publication based on this study. Paper copies will be shredded and computer files will be deleted.

Once again, this is a longitudinal study and it is likely that we will ask your child to participate again over their teenage years. To help us keep in touch with you and your child, we will periodically ask you to confirm your current address or to provide a change of address. We will also ask you to provide us with an alternative contact such as a relative not living with you or a close friend who we could contact if you have moved from your current address and we cannot get in touch with you. This is also important so that you can be kept up to date with any findings from the study.

If you or your child has any concerns about how this study is conducted you can contact the Executive Officer, Human Research Ethics at the University of Melbourne. They may be contacted on 8344 2673 (fax: 9347 6739).

If you have any questions about this study or would like to know more about it, please call us here at ORYGEN Youth Health on 9342 2800 and ask for our project manager, Dr. Melissa O’Shea.
CONSENT FORM FOR PERSONS PARTICIPATING IN RESEARCH PROJECTS

PROJECT TITLE: The ORYGEN Adolescent Emotional Development Study –
Home Assessment Phase. Parent assessment

Name of parent: ____________________________
Address: ____________________________
Phone contact: ____________________________

Name of chief investigator(s): Associate Professor Nicholas Allen
Name of Project Manager: Dr. Melissa O’Shea

3. I (consent/ do not consent) to participating in the above project, the details of which were found in the accompanying information sheet. (Please circle consent status)

4. In my consenting to participating in the above study, I acknowledge that:
   f) the researcher or his assistant will administer the questionnaires to me in my home or I can complete them in my own time and return them to the project team;
   g) the possible effects of the questionnaires have been explained to me to my satisfaction;
   h) I have been informed that I am free to withdraw from the project at any time and in this case, any data I have supplied will be withdrawn and destroyed;
   i) the project is for the purpose of research;
   j) I have been informed that the confidentiality of the information that I provide will be protected.

Signature ____________________________ Date ____________________________
(Parent/Guardian)
Dear Student,

Earlier this year, you were involved in the 'ORYGEN Adolescent Emotional Development Study'. This is a study to learn more about teenagers and find out why some of them find things hard. You will remember that we visited your school and you completed a short survey about yourself. Thanks again for taking part.

You might also remember that we were hoping to ask some of you to take part again. This is what we are approaching you about now.

In this next part of the study, we will be asking you some more questions about yourself and how you are feeling. Someone from our team will be coming to your house or school to ask you these questions face-to-face. You will also be asked to fill in some more surveys about yourself. This will give us lots more information about young people your age. We will even ask your parents to take part too.

Unless we think you might hurt yourself or someone else, or someone is hurting you, all of your answers will be kept private – this means we will not tell anyone what you have said. And if you have any problems with any of the questions, or if you want to talk to someone about your answers, the researcher will be happy to help you out.

Also, if at any time during the interview you would like to finish, you can. And like last time, if you don't want to do it at all, you don't have to.

Your answers to this interview might also mean that we will ask you to take part in other studies when you are a bit older. Of course you can decide then if you want to participate in any of these follow-up studies.

Finally, you will only be able to take part in this study if one of your parents (or the person who cares for you at home) says you can. They will then need to sign a form. You will be asked to sign an agreement form too if you take part.

Thanks for your help!

Associate Professor Nick Allen and Dr. Melissa O'Shea,
ORYGEN Youth Health.

Locked Bag 10 (35 Poplar Road) Parkville Vic Australia 3052
Tel: +61 3 9342 3000 Fax: +61 3 9347 3003 Web: www.orygen.org.au
A program of North Western Mental Health and Melbourne Health
Approval to participate in a research project

**PROJECT TITLE:** The ORYGEN Adolescent Emotional Development Study – Home Assessment Phase

**Name of participant:**

**Name of investigator(s):** Associate Professor Nicholas Allen

**Name of Project Manager:** Dr. Melissa O’Shea

1. After talking about the survey with my parents (or guardian), I **(agree/do not agree)** to take part in this project.

   (Please circle)

2. In taking part, I understand that:

   (a) an adult helping to run the study will visit my home, school or ORYGEN Youth Health, and ask me to answer some questions about myself and how I am feeling, or I can participate over the phone if this suits me better;

   (b) I can stop taking part in the study at any time;

   (c) the study is just for research;

   (d) any answers I give will be kept private except if I talk about hurting myself or someone else, or someone is hurting me.

**Signature**

**Date**
Appendix B2: Consent forms and PLS for Family Assessment

PLAIN LANGUAGE STATEMENT

Principal Investigator: Assoc. Professor Nick Allen
Other Investigator: Ms. Marie Yap

Project Title: A prospective investigation into the impact of family processes, child temperament, and emotion regulation on the development of adolescent depression.

Study Overview
Earlier this year, your child participated in the 'The ORYGEN Adolescent Emotional Development Study' (conducted by ORYGEN Youth Health and the Department of Psychology, University of Melbourne) by completing a short survey about themselves at school. As you will remember, this study is investigating how difficulties with regulating moods and emotions may be related to mental disorder in youths. More recently, members of our research team either visited your home or spoke with you and your child over the phone for the second phase of the study. This involved asking you (if you agreed to take part) and your child questions relating to your emotions, moods, relationships and general coping. As we noted earlier this year, a key feature of this study is that it is longitudinal, meaning that some of the children would be asked to take part again over their teenage years. This study design is critical because it will enable us to understand how symptoms of mental disorder emerge in our sample, and how this relates to their emotional regulation. The young people selected for the next phase of the study represent a full range of scores on the survey they completed at school, and have been found to have no history of mental health problems. Parents can therefore be assured that their child’s selection is not a specific indicator of their level of risk for anxiety and/or depression or other mental health concerns.

In this next phase of the study, we will be inviting both you and your child to come to ORYGEN Youth Health in Parkville for about one hour. We will first ask you and your child (separately) some questions about feelings you have experienced and how you respond when your child experiences certain feelings. You will each fill in two short questionnaires, as well as take part in two family interaction tasks together.
Details of this phase of the study are provided below.

**Purposes and Benefits**

Whilst there will be no direct benefit to you or your child in participating in this study, ‘The ORYGEN Adolescent Emotional Development Study’ provides us with an outstanding opportunity to identify young people at high risk for mental health problems. The results from this study will help us develop preventative programs that enhance young people’s awareness and regulation of their moods, and promote healthy emotional development. In addition, it will inform early intervention strategies aimed at those most in need. Some families may also find the tasks interesting as it involves parents and children taking part together. *In order to thank you for your time and the time offered by your child, you will receive $50 if both you and your child agree to participate. Your child will also receive a Coles Myer gift voucher worth $30 as a direct thank you to him/her.*

**Participation**

Unlike other assessments that you and your child might have taken part in for us, the nature of this particular task requires at least one parent *and* their child participate. This is because we are interested in your relationship and how you get on together. Before you and your child can participate in this study, you will be asked to provide written consent by signing two consent forms. The first of these indicates that you agree to your own participation in the study, while the second indicates that you agree to your child’s participation in the study. Your child will also be asked to sign a consent form if they agree to be involved too. Remember that like other assessments, participation in this assessment is entirely voluntary and *if either you or your child* does not want to participate, this is okay and your family does not have to be involved in this particular assessment for the study. We certainly do not want our participants or parents having to do something they really don’t want to. We also remind you that you and your child are free to withdraw consent at any time, and to withdraw any unprocessed data previously supplied. And as always, participation in this phase does not mean that you are required to take part in any other phase of the research; you can make you mind up each phase separately.

**Interview and questionnaire procedure**

If you and your child agree to participate in this phase of the study, you will be interviewed separately at ORYGEN Research Centre. You will be asked questions about feelings you have experienced, what you think about controlling or expressing feelings, and your attitudes and responses to your child’s behaviour when they are angry or sad. We will ask your child similar questions about his/her experience of these feelings, your response to them when they experience such feelings, and the way your family interacts. These interviews will be audio-taped to assist in later coding. In most cases two interviewers will assist in this process so that you can be interviewed simultaneously with your child. This will take around half an hour. In the case of only one researcher...
conducting the interviews, the total time that we will ask you to provide to us will be one half hour extra.

Both you and your child will also be asked to fill in two questionnaires, one relating to areas of conflict between you and your child, while the other is about activities you can do as a family. This takes around 15 minutes. You will then be left with your child in a room with a wall-mounted video camera, which will record your interactions. You will be given two 20-minute interaction tasks in random order: one of them requires you to discuss two areas of conflict between you (selected from your responses on the first questionnaire) and try to resolve them; whilst the other task will ask you to plan two enjoyable activities, e.g., a vacation, to do as a family (selected from your responses on the second questionnaire). Your interactions will be video-taped because they have to be scientifically coded by researchers in the United States of America, who are trained and experienced in family research. These researchers will have no access to any identifying information relating to you or your child.

We’d like you to know that these interviews and interaction tasks have been used many times previously by us and other family researchers around the world, and have all been well tolerated by young people taking part in similar studies. In particular, this research will help us to understand how young people manage their pleasant and unpleasant feelings, both privately and within the family environment (e.g., during parent-child interactions), and how this links with problems in adolescence. Also, you are able to change your mind about participating at any time, including during the session itself. We would like both you and your child to feel comfortable at all times.

**Altogether, this assessment will take between one and ½ hours and two hours. In most cases, it will be around one hour only.**

**Confidentiality**
Any information collected by the researchers is strictly confidential. Participants have already been given a numerical identifier and all subsequent is identified using these numerical identifiers, not the names of the parents and students participating in the study. Only investigators and a small number of the research staff working on the study will have access to the raw data. All of the information provided by you and your child will be stored securely at ORYGEN Youth Health with the exception of the video material. All video material from this assessment will be sent temporarily to the Oregon Research Institute in the USA for coding. The ORI is a leading research group in the area of family interaction research and provide the most accurate and useful interpretations of family interaction tasks such as this. Parents should note that while coders in the USA will not have names of participants, they will be able to view the footage and any related visual identifying information from you and your child. Saying this, they are bound by the same research ethics protocols as our team in Australia, relating to the ethical use and storage of research data, including confidentiality.

As in our studies within this project, within the limits of the law, we will keep all information confidential and will not provide information about any individual participants to anyone. An important exception to this is in the case that your child revealed that they were going to harm themselves or others or were being
harmed themselves. Parents will not have direct access to information provided by their child in the survey. The results of the project presented in journals, or at conferences, will only be reported in ways that do not identify individual participants. *No video material* will be included in such presentations at all. Moreover, all data provided by you and your child will be destroyed 5 years after the date of the last publication based on this study. Paper copies will be shredded and computer files and video footage will be deleted.

**Contacting us**

If you have any questions about this study or would like to know more about it, please call the project manager, Dr Melissa O’Shea on 9342 2800.

**Concerns**

If you or your child has any concerns about how this study is conducted, you can contact the Executive Officer, Human Research Ethics at the University of Melbourne. They may be contacted on 8344 2073 (fax: 9347 6739).
Consent form for persons participating in research projects

PROJECT TITLE: A prospective investigation into the impact of family processes, child temperament, and emotion regulation on the development of adolescent depression.

Name of parent: ________________________________

Address: ______________________________________

Phone contact: ________________________________

Name of chief investigator(s): Associate Professor Nicholas Allen

Name of other investigator: Ms. Marie Yap

1. I (consent/ do not consent) to participating in the above project, the details of which were found in the accompanying information sheet. (Please circle consent status)

2. In my consenting to participating in the above study, I acknowledge that:

a) the researcher or his assistant will conduct the video-taped family interaction tasks and administer an audio-taped interview and two questionnaires to me in a laboratory at ORYGEN Research Centre;

b) the possible effects of the interaction tasks, interview and questionnaires have been explained to me to my satisfaction;

c) I have been informed that I am free to withdraw from the project at any time and in this case, any data I have supplied will be withdrawn and destroyed;

d) the project is for the purpose of research, and all data provided by me will be destroyed 5 years after the date of the last publication based on this study;

e) footage from the video-taped family interaction task will be forwarded to the Oregon Research Institute (ORI) in the USA for coding and then sent back for stage at ORYGEN Youth Health.

f) I have been informed that the confidentiality of the information that I provide will be protected within the limits of the law.

Signature ________________________________ Date ________________

(Parent/Guardian)
Dear Student,

Earlier this year, you were involved in the ‘ORYGEN Adolescent Emotional Development Study’, a study by researchers at ORYGEN Youth Health and the Department of Psychology at the University of Melbourne. This is a study to learn more about teenagers and find out why some of them find things hard. You will remember that we visited your school and you completed a short survey about yourself. Some time later, someone from our team visited or called you at home and asked you some more questions about yourself and how you were feeling. Thanks again for taking part.

You might also remember that we were hoping to ask some of you to take part again. This is what we are writing to you about now.

In this next part of the study, we will be inviting both you and your parents to come to ORYGEN Research Centre for about one hour. We will ask you to tell us about feelings you may have experienced before, like sadness and anger, and what your parents do when you have these feelings (your parents will not be present when we ask you these questions). We will be asking your parents similar questions as well, but we will do this separately from you. These interviews will be audio-taped.

You and your parents will also be asked to fill in two questionnaires. The first one asks you to choose issues or problems you disagree with each other about. The second one will ask you to choose activities that you can do together as a family. You and your parents will then be left in a room on your own and asked to discuss two issues you disagree about and try to solve the problems together. You will also be asked to plan two fun activities to do together as a family. The room you are in will have a video camera fixed onto the wall, which will record your discussion. Only researchers in the study will watch the recording, which will give us lots more information about how people your age relate to their parents, as well as how parents relate to people your age.

Unless we think you might hurt yourself or someone else or someone is hurting you, all of your answers will be kept private – this means we don’t tell anyone what you have said (or show anyone the video recording of your family discussions). And if you have any problems with any of the questions, or if you want to talk to someone about your answers, the researcher will be happy to help you out.

If at any time during the study you would like to finish, you can. And like last time, if you don’t want to do it at all, you don’t have to.

You will only be able to take part in this study if one of your parents (or the person who cares for you at home) says you can, and if at least one of your parents is willing to take part as well. They will then need to sign a form. You will be asked to sign an agreement form too if you take part.

Thanks for your help!

Associate Professor Nick Allen & Ms. Marie Yap

Locked Bag 10 (35 Poplar Road) Parkville Vic Australia 3052
tel: +61 3 9342 2800 fax: +61 3 9387 3003 web: www.orgyen.org.au
A program of North Western Mental Health and Melbourne Health
Approval to participate in a research project

Project Title: A prospective investigation into the impact of family processes, child temperament, and emotion regulation on the development of adolescent depression.

Name of participant:

Name of investigator(s): Associate Professor Nick Allen
Ms. Marie Yap

1. After talking about the study with my parents (or guardian), I (agree / do not agree) to take part in this project. (Please circle)

2. In taking part, I understand that:
   
   (a) my parent and I will visit the ORYGEN Research Centre where an adult helping to run the study will ask me some questions about feelings I have had in an audio-taped interview, and my parent and I will take part in some discussions between us which will be recorded by a video camera;
   
   (b) the videos of these discussions will be sent to the USA for coding
   
   (c) I can stop taking part in the study at any time;
   
   (d) The study is just for research;
   
   (e) any answers I give will be kept private except if I talk about hurting myself or someone else or someone is hurting me.

Signature ________________________ Date ______________

Witness ________________________ Date ______________
Appendix B3: Consent forms and PLS for Follow-up Home Assessment

Adolescent Information Pages

Hi,

Over the past couple of years, you have very kindly taken part in the ‘ORYGEN Adolescent Development Study’. This is a study to learn more about teenagers and find out why many find things hard sometimes. Thanks again for taking part!

You may remember that we were hoping that you would like to take part again. This is what we are writing to you about now.

In this next part of the study, if you decide that you would like to take part in the follow-up brain imaging study, we would also like you to ask you some more questions about you, as well as your feelings and activities. These questions will be in the form of a phone interview (takes about 30 minutes), and some written questionnaires (take about 45 minutes). The questions will be very similar to those that you answered during the last interview that we did at your home, but there’ll be less of them. Answering these questions will give us lots more information about young people at your age and older, and is an important step as we examine information you have provided us with previously and look at things across the years.

If you do decide to take part again, you may remember that all of the information you provide will be kept private. We will even remove your name from it so we won’t know who said what! The exception to this is if you provide information that suggests to us you may hurt yourself or someone else. We are legally bound to respond to this to make sure you and others are safe. Also, if there is something we are concerned about in your answers, we may talk to you about it and will ask you if it is okay for us to speak to one of your parents about it. We won’t speak to your parents unless you say it is okay.

If you decide to take part, we will need you to sign a consent form (attached) to let us know that you agree. We would like you to know, however, that you may withdraw that consent at any time. It is important that if you take part in this study that you are doing so because you want to. You may also agree to do it now, and then change your mind later. We will talk to you more about this in person if you think you would like to take part, or have any questions.
You will only be able you take part in this phase of the study if one of your parents (or the person who cares of you at home) still thinks it is okay. Please discuss this with them so you can decide together.

Thanks for your help!

The ORYGEN Adolescent Development Team
Assoc. Professor Nick Allen, Dr Sarah Whittle, Dr Paul Dudgeon, Dr Andrew Chanen, Trudi Mackenzie, Meg Dennison, George Youssef, Renee Lichter, Alison Cheetham, Prof Patrick McGorry.
Consent form for persons participating in research projects

PROJECT TITLE: The ORYGEN Adolescent Emotional Development Study – Home Assessment Phase. Child assessment

Name of participant:

Name of chief investigator(s): Associate Professor Nicholas Allen
Name of Project Manager: Julian Simmons

1. After talking to my parents (or guardian) about this study I (agree/ do not agree) to take part. (Please circle)

2. In taking part, I understand that:

k) An adult helping to run the study will ask me some questions about myself and how I am feeling over the telephone, at the Royal Children’s Hospital, and/or in my home;

l) I can stop taking part in the study at any time;

m) The study is just for research;

n) Any answers I give will be kept private, except if I talk about hurting myself or someone else, or if someone is hurting me.

Signature __________________________ Date ____________

(Participant)
Author/s: SPEAR, OWEN

Title: Neighbourhood disadvantage and internalising symptoms in adolescents: the mediating role of stressful life events, temperament, and maternal aggression

Date: 2013


Persistent Link: http://hdl.handle.net/11343/38512

File Description: Neighbourhood disadvantage and internalising symptoms in adolescents: the mediating role of stressful life events, temperament, and maternal aggression

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