Investigation of Community Factors Associated with Child and Adolescent Depression

by

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Submitted in fulfilment of the requirements for the degree of

Doctor of Psychology (Clinical)

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DEAKIN UNIVERSITY
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FOREWARD

The thesis presented in this document has been set out according to the guidelines for research by publication, stipulated by Deakin University for a Doctorate of Psychology thesis by publication. These guidelines require that the candidate be first author on three publications that have been submitted to peer-reviewed journals at the time of the thesis submission. They also specify that the thesis include; a critical introduction that demonstrates understanding of relevant literature and serves as a basis for understanding the work that follows; and a discussion and conclusion which provides a synthesis of all the material and the application of findings to the professional field. In accordance with the guidelines, methods for each study are included in the articles describing each study of the thesis. The thesis author and primary supervisor believe that the following thesis accords with the requirements described by the Deakin guidelines.
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LIST OF ABBREVIATIONS

AACAP = American Academy of Child and Adolescent Psychiatry

AIC = Akaike Information Criteria

APA = American Psychiatric Association

$\beta$ = standardised coefficient

B = unstandardised coefficient

BIC = Bayesian Information Criteria

BDI = Beck Depression Inventory

BHIF = Baltimore How I Feel

BPI = Behaviour Problems Index

BSI = Brief Symptom Inventory

C = Children’s reports

CAPA = Child and Adolescent Psychiatric Assessment

CBCL = Child Behaviour Checklist

CDI = Child Depression Inventory

CES-D = Center for Epidemiologic Studies Depression Scale

CI = Confidence Interval

DAWBA = Development and Well-being Assessment

DD = Dysthymic Disorder

DISC = Diagnostic Interview Schedule for Children

DMDD = Disruptive Mood Dysregulation Disorder

DSM = Diagnostic and Statistical Manual of Mental Disorders

DSRS = Depression Self Rating Scale

$k$ = number of studies
K-SADS = Schedule for Affective Disorders and Schizophrenia for school-aged children

LL = Log Likelihood

M = Mean

MA = Meta-analysis

MDD = Major Depressive Disorder

MDE = Major Depressive Episode

MLM = Multi Level Model

MLM-G = Multi Level Growth Model

MRA = Multiple Regression Analyses

N = sample size

NSA = National Survey of Adolescents

OR = Odds Ratio

P = Parents’ reports

p = p-value (significance)

PDD = Persistent Depressive Disorder

R = Retention

RR = Response Rate

SAS = Social Adjustment Scale

SDQ = Strengths and Difficulties Questionnaire

SEM = Structural Equation Modelling

SES = Socio-Economic Status

SLA = Statistical Local Area

SMFQ = Short Moods and Feelings Questionnaire

SR = Systematic Review
T = Teachers reports
USA = United States of America
WHO = World Health Organisation
YSR = Youth Self-Report
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ABSTRACT

Depression is the leading cause of burden of disease due to: disability, decreased work productivity, and increased use of health services. Depression emerges during childhood and adolescence in the context of risk factors (unique predictors) and protective factors (risk modifiers) at the individual/peer, family, school, and community-level. The focus of the current thesis is to establish whether community-level variables account for an important share of the potentially modifiable factors that predict adolescent depression. This will be achieved across three studies described in later sections.

The negative consequences associated with child and adolescent depression can have cumulative detrimental health and social impacts and increase exposure to negative life events. The cumulative adversity associated with child and adolescent depression can contribute to severe psychopathology in adulthood. Furthermore, longitudinal studies suggest that once an adolescent has experienced a depressive episode there is a high probability of recurrence. Sub-clinical and clinical depression in childhood and adolescence are stronger indicators of later depression relative to other risk factors.

In order to identify opportunities for preventative interventions, risk and protective factors associated with child and adolescent sub-clinical and clinical symptoms must be identified. Current evidence supports predictors of depression at the individual, family, and school-level. However, research examining community-level predictors and interventions is less organised. Given their potential to affect large aggregate populations, identifying community-level factors that influence depression in school-aged children may offer cost-effective opportunities for population-level intervention.
Study 1 presented the first systematic review and meta-analyses of studies that have investigated community factors associated with depression in school-aged children (4-18 years). Study 1 identified 21 studies that met the inclusion criteria relevant to analyses that examined the effect of one or more community-level variables on child depression. The meta-analyses revealed significant effects in the observational studies for community safety and community minority ethnicity and discrimination. Effects failed to reach significance for the observational studies of community connectedness and community disadvantage. The combined effect of the 3 intervention studies examining the effects of alleviating disadvantage also failed to reach significance. However, the systematic review suggested that the effect of community connectedness and community disadvantage may be moderated by other predictors of depression.

The international findings from Study 1 identified that community-level factors are directly and indirectly associated with depression in school-aged children. However, none of the included studies examined depressive symptoms at sub-clinical levels and only one study used an Australian sample. High levels of depressive symptoms during early adolescence predict the subsequent development of psychopathology and health and social problems, while being potentially preventable through community-based interventions. To advance Australian policy and prevention approaches it is necessary to identify the prevalence and predictors of adolescent sub-clinical and clinical depressive symptoms using Australian community samples.

Study 2 aimed to quantify the prevalence of elevated depression symptoms (sub-clinical and clinical) and to identify the prevalence variation between statistical local areas (SLAs) using a large nationally representative sample of
Australian adolescents (10-14 years). Study 2 also aimed to identify whether community-level disadvantage was associated with depression in Australian adolescents. The Short Mood and Feelings Questionnaire was used to assess sub-clinical and clinical depressive symptoms. Multivariate analyses accounted for community and school clustering effects and assessed gender interaction effects.

Study 2 identified that 33.1% (95% CI: 32.1, 34.2) of Australian adolescents (average age 12) reported elevated depressive symptoms. Students (N = 8, 256) were sampled across 30 Australian Statistical Local Areas (SLAs) and multi-level modelling indicated that 6.9% of the variation in depressive symptoms was associated with the community SLA variable. After controlling for a range of covariates including family-level covariates, community-level disadvantage and State were significantly associated with depressive symptoms. Significantly more females (35.7% [95% CI: 34.3, 37.2]) reported depressive symptoms than males (30.3% [95% CI: 28.8, 31.8]), with females 27% (OR = 1.27; P =0.000) more likely to report depressive symptoms. Gender interaction effects were identified for school grade and father’s employment.

Study 2 identified high rates of elevated depressive symptoms, with these problems evident in a third of Australian children in 2006. Study 2 indicated that the prevalence of adolescent depression varied between localities. Together the findings of Study 1 and Study 2 suggested that other predictors of depressive symptoms may moderate the influence of locality, community-level disadvantage and State. It was suggested that the relative importance of community-level factors should be further investigated by using multi-level strategies to examine risk factors at the individual, family, and school levels.

To determine ecologically valid intervention models it is important to
understand how much of the locality variation in adolescent depression can be explained by factors at the community, school, family, and individual/peer level. Identifying these differences by SLA provides practical information for designing interventions, as local governments and regional organisations play an important role coordinating local funding and services in Australia.

As such, Study 3 aimed to identify whether community-level variables explained a large component of locality variation in adolescent depression relative to individual, family, and school factors. Study 3 also aimed to identify whether community-level factors such as community disadvantage and State were explained by other predictors of depressive symptomology. Study 3 used the same representative sample of Australian adolescents. Multi-level modelling was used to identify risk and protective factors associated with elevated depressive symptoms in Australian adolescents (10-14 years, N = 7,070). Using fit statistics different models were compared to identify how much of the variance in depression between SLAs could be explained by factors at the community, school, family, and individual-level.

Study 3 identified that community-level factors (community opportunities for pro-social involvement, community attachment, community safety, community laws and norms favorable to drug use, perceived availability of drugs, transitions and mobility, community disadvantage, and State) accounted for 88% of the variation in depression between SLAs. The final model identified 18 factors that explained 91% of the locality variation in adolescent depression. Findings suggested that the association between community-level disadvantage and depression was explained by community and school-level predictors of depressive symptoms. Community safety and State of residence were significant predictors
of depression; Queensland and Western Australian adolescents were more likely to report depression. Gender interaction effects were identified for community attachment, father living at home, and father’s employment. Thus findings confirmed that community-level factors may be important influences on adolescent depressive symptoms in Australia, suggesting the importance of designing and evaluating community place-based interventions.

Overall findings from the current thesis suggest that community-level variables account for a measurable component of the potentially modifiable factors that predict adolescent depression. Designing community interventions to reduce modifiable risk factors and to increase protective factors may reduce the currently high prevalence of elevated depressive symptoms in Australian adolescents and subsequently prevent the development of depression. Findings support the use of place-based approaches that target multi-level (community, school, family, individual/peer) risk and protective factors to reduce the prevalence of adolescent depression.

Investment in the development and evaluation of interventions targeting risk and protective factors in adolescents, is likely to be a cost-effective prevention strategy to reduce the incidence of depression. However, governments tend to have limited interest in funding prevention research in this area as the cost offset of such approaches will take many years to eventuate. Strengthening the evidence base to support the cost-effectiveness of such intervention is essential to advancing prevention efforts. Further, preventative policy is needed to coordinate approaches as many of the factors influencing the onset of depression lie outside the domain of mental health services.
CHAPTER 1: INTRODUCTION

Introduction

Depression has been identified as an international priority disorder (World Health Organisation [WHO], 2003). Internationally approximately 1 in 20 adults report experiencing depression (WHO, 2012). The 12-month prevalence of depressive disorders for Australian adults is approximately 5.4% (Slade, Johnston, Oakley Browne, Andrews, & Whiteford, 2009). Youth appear to have a higher prevalence. In a large epidemiological study, approximately 18.4% of adolescents in the United States of America (USA) met the criteria for a depressive disorder and 25.9% experienced sub-clinical depression (Lewinsohn, Shankman, Gau, & Klein, 2004). In Australia, approximately 14-16% of adolescents reported depression (Bond, Toumbourou, Thomas, Catalano, & Patton, 2005; Boyd, Gullone, Kostanski, Ollendick, & Shek, 2000).

The relatively high prevalence of depression has significant social and economic consequences, such as disability, functional impairment, reduced work productivity, and costs associated with the increased use of health services (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen 2012; Simon, 2003). Internationally depression is identified as the leading cause of disability (WHO, 2008). In Australia, annual health-related expenditure on depression alone costs approximately 1,107 million dollars, with depression-associated disability estimated at $14.9 billion (Australian Institute of Health and Welfare, 2007a; Beyond Blue, 2005).

Organised efforts to reduce the burden of mental illness have traditionally focussed on the treatment of individuals who are unwell (Herrman, Saxena, Moodie, & Walker, 2005). This has been through services such as the Medicare
Better Access scheme that have increased access to psychological therapies in Australia (Pirkis, Harris, Hall, & Ftanou, 2011). As well as increased use of psychopharmacological treatments such as selective serotonin reuptake inhibitors (McManus et al., 2000).

However, the increasing national and international prevalence of depression in developed and developing countries suggests that a focus on treatment will not be enough to reduce the social and economic costs of this growing phenomenon (WHO, 2001a). If the prevalence of depression is to be curbed and reduced, identifying preventative and more efficient ways of managing this problem needs to be a national and international priority.

To reduce the burden of depression, it is essential to decrease the incidence (first occurrence) of depression during early adolescence when psychopathology often emerges (Jorm, 2014; Kessler et al., 2012). An important aspect of reducing the incidence of mental health disorders is targeting risk and protective factors to reduce the likelihood of mental health disorders occurring in the first instance. Risk factors are defined as, “any influences that increase the chances for harm” (Fraser, 2004, p. 14). Protective factors are defined as “both internal and external resources that modify risk” (Fraser, 2004, p. 28). Identifying the predictors of adolescent depressive symptoms and targeting these factors through population-level approaches offers the best opportunity to reduce the incidence of depression (Commonwealth Department of Health and Aged Care, 2000; Lewinsohn et al., 2004; WHO, 2001b).

The purpose of the current thesis was to establish whether community-level variables account for an important share of the potentially modifiable factors that predict child and adolescent depression. This was achieved across 3 studies
that are described in later sections. A brief summary of the thesis structure follows.

Chapter 1 introduces the key concepts relevant to the area of child and adolescent depression. Prevalence rates, the onset and course, and co-morbidities associated with child and adolescent depression are then outlined before considering the impacts of child and adolescent depression. Chapter 2 provides a rationale for reducing the burden of depression by adopting a preventative approach to target the predictors of depression that emerge during early adolescence. It explores relevant theoretical frameworks including: (a) mental health promotion and prevention theory that support preventative approaches, (b) the developmental psychopathology perspective that explains the emergence of psychopathology during adolescence, and (c) ecological systems theory that provides a framework for organising the risk and protective factors that present across multiple settings. Chapter 2 then considers the current evidence base for predictors of child and adolescent depression. Individual, family, school, and community-level factors associated with child and adolescent depression are presented before a brief summary of the gaps in the current evidence base is provided. Chapter 2 goes on to consider preventative interventions for depression in school-aged children and concludes with a summary of the current evidence base; rationale for the current studies; and aims of the current studies.

Chapters 3, 4, and 5 present the 3 studies submitted to peer-reviewed journals. Chapter 3 presents Study 1 a systematic review and meta-analyses of community-level factors influencing depression in school-aged children (4-18 years). Chapter 4 presents Study 2 that examines the prevalence and variation in elevated depression symptoms between statistical local areas (SLAs) using a large
nationwide representative sample of Australian adolescents (10-14 years). Using the same representative sample chapter 5 presents Study 3 that examines whether community-level variables explain a large component of variation in adolescent depression symptoms relative to individual, family and school factors.

Chapter 6 presents a discussion of the overall findings. Limitations of the current thesis and opportunities for future research are then outlined. The discussion goes on to consider the implications for policy and practice. Finally the current thesis presents a brief conclusion.

**Childhood and Adolescent Depression**

Before examining community-level factors associated with child and adolescent depression, it is necessary to understand the key terms and developmental issues associated with child and adolescent depression. This section will introduce the key concepts relevant to child and adolescent depression. The prevalence rates, onset and course, and co-morbidities associated with child and adolescent depression are then outlined before considering the impacts of adolescent depression.

**The Adolescent Period**

Generally speaking the adolescent period of development refers to the transition between childhood and adulthood (WHO, 2014). It can be conceptualised as beginning with the onset of puberty and ending when the individual successfully individuates from their parents (Casey & Caudle, 2013). Whilst puberty can be specifically defined in neuro-endochronological terms, adolescence is usually not defined by one developmental change, rather as a period of transitions that includes puberty (Rosenblum, 1990; Spear 2000a).
Erickson described the adolescent stage of psychosocial development as a time where an emerging sense of self develops by synthesising earlier experiences to form an identity that includes expectations of future experiences (Gross, 1987). The formation of self-identity is critical for future intimacy and the development of stable long-term relationships (Rosenthal, Gurney, & Moore, 1981).

To successfully navigate the developmental transition from childhood into adulthood, adolescents are challenged to negotiate this period whilst attaining the necessary skills for independence (Blakemore & Mills, 2014; Spear, 2000a). Effects of puberty and brain development result in new behaviours and capacities that support transitions in peer, academic, family, and physical health domains (Viner et al., 2012). Particular behavioural changes are evident during adolescence, including age relevant increases in novelty-seeking, risk taking behaviours, and peer social interactions (Blakemore & Mills, 2014). These behaviours likely assist sexual procreation and independence during this period of transition, as well as supporting the development of a range of skills necessary to survive away from parent caregivers (Blakemore & Mills, 2014; Spear, 2000a; Viner et al., 2012).

Adolescence is considered a period of high-risk for the onset of psychopathology (Spear, 2000b). In the years following the onset of puberty, the incidence of depression noticeably increases and the majority of individuals later diagnosed with a mental disorder show a significant increase in problems (Walker, 2002). Adolescence is characterised by significant neurodevelopment including changes in brain structure and function (Blakemore & Mills, 2014). It has been proposed that surges in hormones following puberty may trigger gene
expression of brain abnormalities that give rise to mental disorders (Spear, 2000b).

Adolescent mental health is also influenced by factors external to the individual such as social and economic factors in the broader community (Viner et al., 2012). As such these determinants of health impact on an adolescent’s developmental trajectory and have broader impacts for population-level health (Viner et al., 2012).

**The Transition from Childhood to Adolescence in Australian Policy**

The periods of childhood and adolescence are defined differently in policy, research, and real-life settings. In Australia, young people legally enter adulthood at age 18, as signified by their right to vote, drink alcohol, attend nightclubs, and drive a motor vehicle (Pitman, O’Neill, Herbert, & Land, 2004; Queensland Commission for Children and Young People, 2004). This is also the time when most young people in Australia finish high school and in some cases transition into independent living. Consequently numerous Australian researchers identify age 18 as the cut-off point between adolescence and adulthood (Fraser & Fraser, 2003; Pitman et al., 2004; Queensland Commission for Children and Young People, 2004).

In Australian policy the term “young people” is frequently used to describe individuals ranging from 12-24 years old (Australian Institute of Health and Welfare, 2011). Whilst children are defined as being aged 0-14 years. The overlap is deliberate, recognising that the transition from childhood to youth is a gradual process that begins and ends at different times for different individuals (Moon, Meyer, & Grau, 1999). However, in practicality Australian policy and programs target two distinct critical periods of development: (a) early childhood (0-8 years)
usually referred to as the “early years” and (b) the period of adolescence (12-18 years) and young adulthood (18-24 years) that is usually grouped under the category of “youth” or “young people”.

Traditionally, Australian federal and state government policies defined the early years as the period from birth to 5 years (Australian Government, 2005). Projects such as the Early Years Learning Framework for Australia (Australian Government, 2009) highlighted the importance of ensuring smooth transitions from kindergarten to school. As a result more recent policy such as the Early Years Strategic Plan, Improving Outcomes for all Victorian Children 2014-2020 (Department of Education and Early Childhood Development, 2014) has extended the early years definition to include children 0 to 8 years.

However, there still remains a distinct gap in current policy and direction for children aged 8-12 years. Some local governments have recognised this absence of policy and practices for children in the “middle years” and have broadened their current policies to include these children under the classification of “middle youth” (e.g., City of Port Phillip, 2014).

**Defining and Assessing Depression**

**Diagnosis and assessment.** Terms such as depression, depressive symptoms, internalising symptoms, internalising disorders, emotional disorders, mood disorders, depressive disorders, and affective disorders are often used interchangeably in the literature to describe depression. However, there are important distinctions in these terms and in the approaches to their assessment. Figure 1 identifies common groupings of mental disorders that are applied in the literature. Figure 2 presents common groupings of mental disorders used when
assessing children and adolescents.

Figure 1. Common groupings of mental disorders. Based on diagnostic criteria from “Diagnostic and Statistical Manual of Mental Disorders: DSM-IV-TR” by American Psychiatric Association, 2000.

There are two approaches commonly used to assess and determine the presence or absence of depression in an individual. The first approach, usually adopted by clinical psychologists and other mental health professionals, considers depression as a clinical disorder that is diagnosed according to quantitative (e.g., number of symptoms) and qualitative (e.g., degree of impairment, change in functioning) dimensions (Rudolph & Lambert, 2007). Diagnoses of depression are assessed by structured clinical interviews (e.g., Diagnostic Interview Schedule for Children (DISC IV: American Psychiatric Association [APA], 1994) based on clinical features identified in the Diagnostic and Statistical Manual of Mental Disorders (DSM-V: APA, 2013) and International Classification of Diseases (ICD-10: WHO, 1993).

In Australia and internationally, psychologists commonly use the DSM to diagnose depression. In 2013 the DSM-IV-TR (APA, 2000) was updated to the DSM-V version (APA, 2013). Under the earlier DSM-IV-TR depressive disorders were grouped together under the category of mood disorders and included Bipolar Disorder and related disorders (APA, 2000). However, in the updated DSM-V depressive disorders are now categorised under a separate chapter from Bipolar and Related Disorders (APA, 2013).

In DSM-V depressive disorders include: Disruptive Mood Dysregulation Disorder (DMDD); Major Depressive Disorder (MDD) including Major Depressive Episode (MDE); Persistent Depressive Disorder (PDD); Premenstrual Dysphoric Disorder; Substance/medication-induced Depressive Disorder; Depressive Disorder due to another Medical Condition; other Specified Depressive Disorder; and Unspecified Depressive Disorder (APA, 2013). Depressive disorders are usually differentiated by the absence of a manic or...
hypomanic episode (APA, 2000, 2013). Manic and hypomanic episodes are characterised by abnormally and persistently elevated, expansive, or irritable mood and increased activity or energy (APA, 2013).

An exception to this rule is DMDD, a new diagnosis under DSM-V (APA, 2013). DMDD was introduced in response to concerns regarding the over diagnosis and associated treatment of Bipolar Disorder in children (APA, 2013). DMDD is diagnosed in children up to 18 years of age, but symptoms must present before 10 years of age (APA, 2013). DMDD is characterised by persistent irritability and frequent episodes of extreme behavioural dyscontrol (APA, 2013). Given, the relatively new introduction of this diagnosis there is little research considering DMDD, and as such it is not considered further in the current thesis.

The most commonly assessed and diagnosed depressive disorders in children and adolescents are MDD and Dysthymic Disorder (DD; Kessler et al., 2012; Merikangas et al., 2010). MDD is characterised by recurrent MDEs. Diagnostic criteria for MDD and MDE in DSM-V are largely unchanged from the DSM-IV-TR, though in DSM-V greater consideration is given to differentiating appropriate sadness associated with grief (APA, 2013). However in DSM-V, DD has been replaced by PDD. PDD is a consolidation of DSM-IV-defined Chronic MDD and DD (APA, 2013).

Adolescents who have a MDE are diagnosed on having at least five symptoms such as depressed mood or irritability, decreased interest or pleasure, excessive guilt, diminished concentration, fatigue, and suicidal ideation (APA, 2013). Depressive symptoms must be experienced nearly every day during the same two week period and must constitute a change in functioning and
impairment in one or more major areas of life (American Academy of Child and Adolescent Psychiatry [AACAP], 2007; APA, 2013).

Adolescents with PDD experience a depressed or irritable mood most of the day for more days than not for at least one year (APA, 2013). During this period adolescents experience two or more symptoms such as change in appetite or sleep, low energy, low self-esteem, poor concentration, and feelings of helplessness (APA, 2013). The diagnostic criteria for DD and PDD is similar (APA, 2000, 2013). Given, the relatively new introduction of the diagnosis PDD the majority of current research discussed in the current thesis refers to DD rather than PDD.

It is important to note that adolescent depression may also present with psychotic symptoms whereby adolescents experience hallucinations or delusions. In such cases an individual may be diagnosed with a depressive disorder such as MDD with a specifier (e.g., with psychotic features; APA, 2013). Depression may also evolve in the context of a medical condition or substance use, such cases have separate classifications such as Substance/medication-induced Depressive Disorder or Depressive Disorder due to another Medical Condition. Adolescents may also move between diagnostic categories during the course of their depression (McDermott et al., 2010).

The second approach to assess depression utilises self-report measures to identify depressive symptoms (Rudolph & Lambert, 2007). In this approach, commonly adopted by researchers, clinical cut-off scores are frequently used to determine the presence or absence of depression based on the number of items endorsed by an individual. For example using the Short Moods and Feelings Questionnaire (SMFQ) a cut off score of 12 out of a possible 26 is used to
determine clinical significance and identify clinical depression (Angold, Costello, Messer. & Pickles, 1995a).

This approach considers depression in terms of a set of empirically determined behavioural and emotional symptoms that cluster together in multivariate analysis, with symptoms fluctuating on a continuum (Rudolph & Lambert, 2007). The most common application of this approach suggests that depressive and anxiety symptoms cluster together to form internalising behaviours (Child Behaviour Checklist: Achenbach, 1991) or emotional disorders (Strengths and Difficulties Questionnaire: Goodman, 1997) in children as measured by parent, child, or teacher reports on behaviour.

Whilst some researchers adopt the first (diagnostic) approach to identifying depression in participants (e.g., Ford, Goodman, & Meltzer, 2004; Meltzer, Vostanis, Goodman, and Ford, 2007), the second (self-report) approach is more commonly applied in research (e.g., Bond et al., 2005; Boyd et al., 2000). This approach has frequently been used in epidemiology research given the power difficulties associated with small proportions within populations achieving clinical diagnosis, and the costs and difficulties associated with conducting clinical interviews.

**Clinical and sub-clinical depression.** Depression ranges from sub-syndromal or sub-clinical (not achieving the criteria for a diagnosable depressive disorder) to syndromal or clinical (meeting the criteria for a depressive disorder: AACAP, 2007). In addition to the diagnosable forms of depression many adolescents experience sub-clinical depression that may benefit from monitoring and possibly treatment (McDermott et al., 2010). This has sparked debate amongst researchers as to whether depression is best conceptualised as a discrete
category or a continuum of symptoms (Hankin, Fraley, Lahey, & Waldman, 2005).

One side of the argument considers that clinical and sub-clinical depression represent qualitatively different phenomena (Rudolph & Lambert, 2007). Conversely, recent taxometric analysis suggests that adolescent depression is best conceptualised on a continuum rather than as a discrete entity because depression is continuously not categorically distributed (Hankin et al., 2005). Further, sub-clinical depression in adolescence is associated with significant impairment and risk for MDD (Angst, Sellaro, & Merikangas, 2000; Pine, Cohen, Cohen, & Brook, 1999). As such identifying the prevalence and predictors of depressive symptoms applying sub-clinical cut offs is essential to reducing the incidence of depression.

National and International Prevalence of Depression and Sub-Clinical Depression

International prevalence of depression. Approximately 350 million people worldwide suffer from depression (WHO, 2012). Depression combined with other disorders affects approximately 5-10% of adolescents (WHO, 2012). Lifetime prevalence rates for most countries fall between 8 to 12 % (WHO, 2012). However rates vary greatly between countries, from approximately 3% in Japan to 16.9% in the USA (WHO, 2012).

There is evidence that the prevalence of depressive disorders is increasing in adolescents (Kessler, Avenevoli, &Merikangas, 2001; Kessler et al., 2003). Reasons attributed to this phenomenon include increased individualism, materialism and community mobility, academic and economic pressure in free-market societies, a rise in family conflict and breakdown, sedentary behaviour,
and changes in other underlying risk and protective factors (Twenge et al., 2010). It has been suggested that the increased rates of depression reflect greater detection and awareness (Costello, Erkanli, & Angold, 2006), while large meta-analyses suggest a real trend in adults (Twenge et al., 2010). A meta-analysis of 26 studies carried out between 1965 and 1996, suggests that there have been unchanging levels of child and adolescent depression over the last 30 years, with many young people being undiagnosed in earlier studies (Costello et al., 2006).

It is estimated that 40% (12 month prevalence) of young people (13-18 years) in the USA suffer from a mental disorder (Kessler et al., 2012). A nationally representative sample (N = 10,123) of American adolescents (13-18 years) were assessed through structured clinical interviews to determine clinical diagnoses according to the DSM-IV criteria for mental disorders (Merikangas et al., 2010). The most common adolescent mental health disorders were: anxiety (31.9%); behavioural (19.1%); mood (14.3%); and substance-use (11.4%) disorders (Merikangas et al., 2010). Twenty-two percent of adolescents with mental disorders (11.2% mood disorders, 9.6% behaviour disorders, 8.3% anxiety disorders) experienced severe impairment and/or distress (Merikangas et al., 2010). The median age of onset was 6 years for anxiety disorders, 11 years for behavioural disorders, 13 years for mood disorders, and 15 years for substance use disorders (Merikangas et al., 2010).

Of those affected by mood disorders, 11.7% met the criteria for MDD or DD and 2.9% met the criteria for Bipolar Disorder (Merikangas et al., 2010). Severe mood disorders, identified as severe by the presence of both significant impairment and distress, impacted 74.4% of all adolescents with a MDD or DD (Merikangas et al., 2010). Prevalence rates of all mood disorders increased with
age with an almost two fold-increase from adolescents 13-14 years old to adolescents 17-18 years old (Merikangas et al., 2010).

**Australian prevalence of depression.** In Australia, there is limited data available to assess the prevalence of mental disorders in children and adolescents (McDermott et al., 2010). A representative Australian study (N = 4083) of school-aged children (4-17 years), using parent reports on the Child Behaviour Checklist, suggested that approximately 13% (N = 523) experienced internalising problems and approximately 4% (N = 143) suffered from a depressive or anxiety disorder (Sawyer et al., 2001). A more recent national study (N = 6310) of children and adolescents (4-17 years) indicated 2.8% of children and adolescents met the criteria for Major Depressive Disorder as assessed by parent reports using the DISC-IV (Lawrence et al., 2015). A higher proportion of adolescents (12-17 years: male 12 month prevalence = 4.3%, female 12 month prevalence = 5.8%) met the criteria for Major Depressive Disorders compared to children (4-11 years: male = 1.1, female = 1.2%, Lawrence et al., 2015).

Due to the internalising nature of symptoms, parent reports may underrepresent true emotional difficulties, and as such adolescent reports may provide a more accurate estimate (Cantwell, Lewinsohn, Rohde, & Seeley, 1997; van de Looij-Jansen, Jansen, de Wilde, Donker, & Verhulst, 2011). Indeed, Lawrence’s et al.’s (2015) findings suggested that female’s (11-17 years) and males (15-17 years) were more likely to meet the criteria for Major Depressive Disorder based on adolescent reports than parent reports.

Two large Victorian studies quantified prevalence rates of depression using adolescent self-reports (Bond et al., 2005; Boyd, et al., 2000). Using the Reynold’s Adolescent Depression Scale to determine clinical cut-off scores, Boyd
et al. (2000) identified that 14.2% of adolescents (11-18 years) were depressed and 13.2% of adolescents were anxious. Boyd et al. (2000) argued that rates of depression and anxiety in Australia were similar to those identified in other western countries (e.g., Britain, USA, and Canada). Bond et al. (2005) identified similar rates. In a representative sample of Victorian secondary school students, using the Short Moods and Feelings Questionnaire (SMFQ), Bond et al. (2005) identified that 16.6% of secondary school students experienced depression.

**Prevalence variation.** Prevalence rates of depression have been found to vary by key demographic factors. A nationally representative study of American adolescents (13-18 years) indicated that compared to male adolescents, female adolescents were twice as likely to report depression (Merikangas et al., 2010). These findings were replicated in the studies of Australian adolescents. Boyd et al. (2000) found that girls reported significantly higher rates of depression than boys (18.8% versus 9.3%). Bond et al. (2005) identified that 10.5% of males and 21.7% of females experienced depression.

However, this 2:1 ratio of prevalence does not emerge until adolescence (Bond et al., 2005; Boyd et al., 2000; Garber & Flynn, 2001). Indeed, gender is not a risk factor for developing depression in childhood (Garber & Flynn, 2001). Gender differences in depression emerge by age 14 (Wade, Cairney, & Pevalin, 2002). Meta-analytic findings suggest that depression rates in boys appear to be relatively stable with a peak at age 12, for girls the prevalence of depression slightly increases after age 11 with another significant increase at age 13, peaking at age 15 (Twenge & Nolen-Hoeksema, 2002).

Findings regarding the role of ethnicity are mixed. Meta-analysis results identified that whilst Hispanic children (8-16 years) reported significantly more
depressive symptoms than White and African American children, no differences between White and African American children were found (Twenge & Nolen-Hoeksema, 2002). Prevalence rates of depression have also been reported to vary by geographical location (Boyle & Lipman, 2002). Some studies have suggested an association with socio-economic status ([SES] Wight, Aneshensel, Botticello, & Sepulveda, 2005; Xue, Leventhal, Brooks-Gunn, & Earls, 2005) whilst others argue that socio-economic status is not associated with children’s depressive symptoms (Twenge & Nolen-Hoeksema, 2002).

**Prevalence of sub-clinical depression.** The above prevalence rates of clinical depression may be an underestimate of the true degree of emotional difficulties experienced by adolescents, with many adolescents exhibiting elevated but sub-clinical levels of depression (Bayer & Sanson, 2003; Gonzalez-Tejera et al., 2005). Despite the negative consequences associated with sub-clinical depression (Fergusson, Horwood, Ridder, & Beautrais, 2005; Gonzalez-Tejera et al., 2005; Lewinsohn et al., 2004), few studies have examined the prevalence of sub-clinical depression.

An American study (N =10,200) used the Schedule for Affective Disorders and Schizophrenia for school-aged children (K-SADS) to assess sub-clinical versus clinical symptoms, for a range of psychiatric disorders in high school students (Lewinsohn et al., 2004). The study identified 25.9% of adolescents (average age 16.6 years) experienced sub-clinical depression, and 18.4% of adolescents met the criteria for MDD (Lewinsohn et al., 2004).

The high prevalence of sub-clinical depression, the continuity of depression into adulthood, and the high use of health services by adolescents with sub-clinical depression has resulted in calls for greater research in this area (Bayer
& Sanson, 2003; Fergusson et al., 2005; Gonzalez-Tejera et al., 2005; Lewinsohn et al., 2004). However, to date no Australian studies have quantified the prevalence of elevated depressive symptoms (sub-clinical and clinical) using a representative sample. Quantifying the prevalence of depressive symptoms including both sub-clinical and clinical depression is important in advancing research and public health outcomes in this area.

**Onset and Course of Child and Adolescent Depression**

Depression and sub-clinical depression frequently present in early adolescence and often have a recurrent course with significant associated impairment (Rudolph & Lambert, 2007). Substantial evidence supports the view that depression in adults first emerges during childhood and adolescence. A large representative study (N = 9,282) in the USA suggested that approximately 75% of mental disorders present before the age of 24 and 50% begin before the age of 14 (Kessler et al., 2005).

Early adolescence, usually classified as between 10-14 years, represents a period of vulnerability to depression. Retrospective studies of depressed adults and prospective studies of adolescents suggest that depression likely emerges during the early-mid adolescent period (Hankin et al., 1998; Lewinsohn & Essau, 2002). Some research suggests that depression rates increase as early as 12 years of age, as children enter puberty (DeRose, Wright, & Brooks-Gunn, 2006). Longitudinal data indicates a dramatic increase in the prevalence of MDD after age 11 and again after age 15, with rates levelling in young adulthood (Kim-Cohen et al., 2003).

In contrast to adults, depression in children and adolescents may be characterised by greater irritability than sadness; a less easily recognised onset;
and greater co morbidity with other disorders including anxiety, conduct disorder, hyperkinesia, and learning problems (AACAP, 2007; Birmaher et al., 1996). Anxiety is often a precursor to depression in children and adolescents (McDermott et al., 2010). Adolescent depression is frequently associated with somatic complaints, social withdrawal, and body image dissatisfaction, particularly in teenage girls (Wichstrom, 1999). In comparison to adults, it is imperative to consider the developmental (cognitive, social, and emotional) and environmental (e.g., family, school) context in which children and adolescents exist (McDermott et al., 2010).

Depression often takes a chronic, recurrent, and episodic course (Merry et al., 2011). In clinical samples the median duration of a Major Depressive Episode (MDE) is 8 months and approximately 1 to 2 months for community samples (AACAP, 2007). Whilst the majority of adolescents recover from their first depressive episode, longitudinal studies suggest that the probability of recurrence reaches 20 to 60% 1 to 2 years after remission, and increases to 70% after 5 years (Birmaher, Arbelaez, & Brent, 2002; Costello et al., 2002). Sub-clinical depression in childhood (Mazza et al., 2009) and adolescence (Lewinsohn et al., 1994) are the strongest indicators of later depression. A substantial proportion of adolescents with MDD will continue to experience MDD in adulthood. Large-scale studies of community (e.g., Lewinsohn, Rohde, Klein, & Seeley, 1999; Pine, Cohen, Gurley, Brook, & Ma, 1998) and clinic populations (e.g., Birmaher et al., 2002; Weissman et al., 1999) indicate significant continuity of depression from adolescence through to adulthood.

The consequences of experiencing emotional difficulties during the critical period of adolescent development can have cumulative impacts further increasing
exposure to negative life events and reducing access to work and social resources that can otherwise reduce the recurrence of depression (Kessler et al., 2001). These reciprocal effects result in cumulative adversity that contributes to adolescent depression, increasing the risk of severe psychopathology in adulthood (Kessler, Foster, Saunders, & Stang, 1995).

**Co-morbidities of Child and Adolescent Depression**

MDD and DD frequently present with other mental disorders and medical conditions, and often co-occur (AACAP, 2007). Contingent on the setting and referral source, it has been estimated that 40 to 90% of adolescents with a depressive disorder have co-morbid psychiatric disorders, with approximately 50% presenting with 2 or more psychiatric disorders (AACAP, 2007).

A representative community sample of American adolescents identified that approximately 40% that met criteria for one disorder also met criteria for another lifetime disorder (Merikangas et al., 2010). In another American representative community study of adolescents who met the criteria for MDD, 24.4% had a co-morbid psychiatric disorder and 17.1% had more than 2 psychiatric disorders (Lewinsohn et al., 2004). Similar comorbidity rates have been found in community samples of adolescents presenting with sub-clinical symptoms. Lewinsohn et al. (2004) found that of the high school students who experienced sub-clinical depression, 33.6% also experienced another sub-clinical mental health condition and 14.3% had more than 2 sub-clinical mental health conditions (Lewinsohn et al., 2004).

In depressed adolescents anxiety disorders are the most frequent co-morbid condition, followed by conduct/oppositional defiant disorders, and attention-deficit hyperactivity disorder (AACAP, 2007; Angold, Costello, &
Erkanli, 1999). The presence of early depressive symptoms is also associated with and predicts substance use by adolescents (Diego, Field, & Sanders, 2003; Sihvola et al., 2008). A high proportion of adolescents who experience depression will experience other chronic medical conditions (Beyond Blue, 2005; Bhatia & Bhatia, 2007).

Co-morbidity is generally associated with increased impairment, increased severity of symptoms, higher service use, poorer treatment outcomes, and greater economic and psychosocial burden (Andrews, Slade, & Issakidis, 2002; Canavera, Ollendick, May, & Pincus, 2010; Rao, 2006). The co-morbidity of substance use disorders in depressed adolescents is associated with: earlier onset and increased severity of drug and alcohol related problems (Rohde, Lewinsohn, & Seeley, 1996); increased duration and recurrence of depressive episodes (Rao, Daley, & Hammen, 2000); more severe impairment in academic and interpersonal capabilities (Rao et al., 2000); greater frequency of behavioural problems (Rao et al., 2000); elevated risk for suicidal behaviour (Lewinsohn et al., 1994); and increased use of mental health services (Kessler et al., 1996; Rohde et al., 1996).

Research utilising symptom scales suggests significant covariance between adolescent depression and other internalising (e.g., anxiety, and somatic complaints) as well as externalising (e.g., aggression, oppositional) presentations (Rudolph & Lambert, 2007). Some researchers argue that co-morbidity rates may be inflated and reflect problems with specificity of assessment tools (Rudolph & Lambert, 2007). However, the majority of researchers suggest that high co-morbidity rates are likely influenced by the presence of shared risk factors (e.g., genetic predisposition); co-occurring risk factors (e.g., parental depression and family discord increasing risk for depression and conduct disorder); or a causal
relationship between disorders (e.g., decreased social and academic functioning associated with a behavioural disorder may increase the risk for depression: Rudolph & Lambert, 2007).

**Life-Course Impacts of Child and Adolescent Depression**

Child and adolescent depression is associated with a number of negative outcomes including: academic difficulties, impaired social relationships, high-risk sexual behaviour, and a thirty-fold increased risk of completed suicide (Horowitz & Garber, 2006). Depression in adolescence interferes with individual and social functioning and predicts subsequent difficulties in psychological, academic, and social domains (Cole, Martin, Peeke, Seroczynski, & Hoffman, 1998; Pomerantz & Rudolph, 2003). Following the remission of a depressive episode, psychosocial difficulties frequently persist, underpinning the need for continued treatment of depression as well as treatment to address accompanying psychosocial and contextual issues (Birmaher et al., 1996; Fergusson & Woodward, 2002; Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2003). Adolescents with depressive disorders are at high risk for legal problems and exposure to negative life events (AACAP, 2007). Adolescent depression is also associated with adolescent pregnancy and early parenthood (AACAP, 2007; Jaycox et al., 2009).

School aged-children with depression experience decreased classroom performance (Favre et al., 2009; Günther, Holtkamp, Jolles, Herpertz-Dahlmann, & Konrad, 2004; Riglin, Petrides, Fredriskson, & Rice, 2014). Meta-analytic findings (number of included studies \(k = 17\)) indicate that children and adolescents with MDD had significantly lower full scale, verbal, and performance IQs in comparison to healthy children (Wagner, Müller, Helmreich, Huss, & Tadić, 2014). In terms of executive functioning children and adolescents with
MDD had reduced capacity across the following domains: planning ability, inhibition capacity, working memory, shifting ability, phonemic verbal fluency, and the semantic verbal fluency performance (Wagner et al., 2014).

There is evidence that the impact of cognitive dysfunction results in poor classroom performance, low self-esteem, and academic and psychosocial failure (Brooks, Iverson, Sherman, & Roberge, 2010; Lundy, Silva, Kaemingk, Goodwin, Quan, 2010). Academic difficulties can have significant long-term impacts. Indeed depression has been associated with school and college dropout (Kessler, Foster, Saunders, & Stang, 1995). Educational achievement can be considered a predictor of future earning capacity. If school attendance and performance is impacted by depression this is likely to result in an impact on future career and earning potential (Glied & Pine, 2002).

The impacts of depression extend beyond the individual impacting adolescents’ families and communities. Adolescents with depression report significantly greater impairment in family functioning compared to adolescents without depression (Jaycox et al., 2009). Furthermore these impacts may extend to their family members, with parents of depressed adolescents reporting lower parent mood and increased strain (Jaycox et al., 2009). Depression in adolescents also places an economic burden on family and societal systems (Lynch & Clarke, 2006).

The WHO (2008) identifies depression as the leading cause of disability. In Australia, annual health-related expenditure on depression alone costs approximately 1,107 million dollars, with depression-associated disability estimated at $14.9 billion (Australian Institute of Health and Welfare, 2007a; Beyond Blue, 2005). Depression and anxiety are the leading contributors to the
burden of disease and injury for Australian children and adolescents (Australian Institute of Health and Welfare, 2007b). Some studies suggest that medical expenditure on children with depression is significantly higher in comparison to children with other mental health conditions (Glied & Neufeld, 2001; Mandell, Guevara, Rostain, & Hadley, 2003).

These difficulties are also evident in adolescents with sub-clinical levels of depression (Gotlib, Lewinsohn, & Seeley, 1995). Adolescents with sub-clinical depression experience significant psychosocial impairment (Gonzalez-Tejera et al., 2005); increased suicide risk (Fergusson et al., 2005); and increased likelihood of experiencing other sub-clinical and clinical mental health conditions (Lewinsohn et al., 2004). Sub-clinical depression also contributes significant unique variance in the prediction of lifetime MDD (Klein et al., 2013).

In summary, the material presented above in Chapter 1 provided an introduction to the thesis by outlining the key concepts associated with child and adolescent depression. Prevalence rates, the onset and course, and co-morbidities associated with child and adolescent depression were then presented before considering the impacts of child and adolescent depression. Chapter 1 highlighted the immediate negative impacts and long-term consequences associated with depressive symptoms during early adolescence, indicating the need for research in this area. Preventing depression in adolescence offers an opportunity to potentially reduce subsequent health and social burden. However, in order to advance prevention efforts it is necessary to identify the risk and protective factors associated with the development of adolescent depression.

A theoretical framework is required in order to identify appropriate methods and approaches for advancing prevention research. Prevention models
and research pertaining to child and adolescent depression will now be considered in Chapter 2.
CHAPTER 2: PREVENTION MODELS AND THEORIES FOR ORGANISING RESEARCH IN CHILD AND ADOLESCENT DEPRESSION

The previous chapter provided an overview of key terms and factors associated with child and adolescent depression, highlighting the burden of depression and the importance of examining sub-clinical and clinical depression during early adolescence. This chapter will provide a rationale for reducing the burden of depression by adopting a preventative approach to target the predictors of depression that emerge during early adolescence. This chapter highlights the importance of adopting a determinants approach to mental health and presents a framework for organising risk and protective factors including those more distal to the individual at the community-level. A brief review of the literature pertaining to predictors associated with child and adolescent depression is then considered. Finally, the chapter concludes with a summary of the current evidence base, rationale, and aims for the current thesis.

Preventative Approach to Depression

This section begins by considering theory relevant to mental health promotion and the importance of prevention in reducing the burden of mental illness. It goes on to consider the role of preventative interventions and the importance of adopting a determinants approach, before outlining the cost-effectiveness of such an approach.

Mental Health Promotion

*Mental health promotion framework.* The WHO defines *mental health* as “a state of well-being in which the individual realises his or her own abilities, can cope with the normal stresses of life, can work productively and fruitfully, and is able to make a contribution to his or her community” (WHO, 2001b, p.1).
Mental health is not merely the absence of illness it is the foundation of well-being and effective functioning for an individual and for a community (WHO, 2001b). Mental health promotion is any action taken to influence the determinants of mental health in order to maximise the mental health and wellbeing of populations, communities, or individuals (Commonwealth Department of Health and Aged Care, 2000; Keleher & Armstrong, 2005). This is generally achieved not simply through work at an individual-level but through the implementation of effective multi-level interventions that operate across a number of sectors, policies, programs, settings, and environments (Keleher & Armstrong, 2005).

Figure 3. Mental health promotion framework. Based on “Promotion, Prevention and Early Intervention for Mental Health: A Monograph” by the Commonwealth Department of Health and Aged Care, 2000.

Frameworks for conceptualising mental health promotion are presented in later sections (Institute of Medicine, 1994; 2009). As presented in Figure 3, mental health promotion in influential Australian policy frameworks is organised into three categories: (a) prevention, (b) early intervention, and (c) treatment. Prevention refers to interventions that are delivered before the onset of a disorder in order to prevent the disorder from occurring (Commonwealth Department of
Early intervention refers to interventions that specifically target people with early signs and symptoms of mental health problems and people experiencing or developing their first episode of mental illness (Commonwealth Department of Health and Aged Care, 2000). Whereas treatment refers to interventions that are delivered after the onset of a disorder in order to minimise the duration and impact of the clinical episode (Commonwealth Department of Health and Aged Care, 2000). The three approaches can be conceptualised as enhancing protective factors and reducing risk factors to prevent mental health problems; early intervention to minimise the symptoms and impact of mental health problems and disorders; and the timely treatment and rehabilitation of those affected by mental health disorders (Commonwealth Department of Health and Aged Care, 2000; Herrman et al., 2005). These elements are distinct, although have some overlap (Herrman et al., 2005).

Mental health promotion in Australia. In 1997, the first National Survey of Mental Health and Wellbeing in Australia indicated that mental disorders were relatively common (Andrews, Henderson, & Hall, 2001). Based on DSM-IV criteria, approximately 20% of Australian adults reported experiencing a mental disorder in the previous 12 months (Andrews et al., 2001). However, 65% of those identified as having a mental disorder had not consulted a health professional in the past year (Andrew et al., 2001). Of those with an affective disorder, 44% reported that they had not received any treatment in the 12 months prior (Australian Bureau of Statistics [ABS], 1997).

In the late 1990s high prevalence rates and significant treatment gaps were not only an issue for Australia, but rather were concerns experienced globally. The 2001 World Health Report called for governments to implement mental
health policy and coordinate service responses to reduce the growing burden of mental illness (WHO, 2001a). Whilst the WHO called for preventative and public health approaches to curb the impact of mental disorders, the report largely focused on addressing the *treatment gap* (WHO, 2001a).

**Reducing the treatment gap.** In Australia, initial attempts to reduce the burden of mental disorders largely targeted the *treatment gap* (Jorm, 2014). Mental health promotion was aimed at reducing the duration, severity, and recurrence of mental disorders through improved access to treatment. Indeed, this recovery focus is still a key component of Australia’s approach to reducing prevalence rates (Victorian Mental Health Act, 2014).

In the last decade significant progress has been made in reducing the treatment gap in Australia. From 1992/1993 to 2010/11 government spending on mental health increased by 178% (Department of Health and Ageing, 2013). The use of both psychological and psychopharmacological treatments increased dramatically over this period. The use of antidepressants increased from 45 defined daily doses per 1000 population in 2000 to 89 in 2011 (Organisation for Economic Cooperation and Development, 2013). The introduction of the Medicare Better Access program also saw a dramatic increase in the use of psychological services (Pirkis et al., 2011). Psychological treatment rates for mental disorders increased from 37% of those treated in 2006-07 to 46% in 2009-10 (Whiteford et al., 2014). In 2004, modelling estimated that treatment services in Australia averted approximately 15% of the burden related to affective disorders (Andrews, Issakidis, Sanderson, Corry, & Lapsley, 2004).

Despite significant advances in reducing the treatment gap, evidence suggests that prevalence rates of depression in Australia remain relatively
unchanged (Jorm & Reavley, 2012; Reavley, Jorm, Cvetkovski, & Mackinnon, 2011). Similarly, no changes have been found internationally (Kessler et al., 2005; Mojtabai, 2011). There are multiple possible explanations for why prevalence rates have not decreased (Jorm, 2014). One possibility is that the advances made have been small and studies may lack the necessary statistical power to identify them (Jorm, 2014). It is estimated that current methods may be unable to detect a difference of less than 2% (Jorm, 2014). Another potential explanation is that improvements have been made but broader societal/environmental factors may have impacted prevalence, such as the increased use of social media (Jorm, 2014).

Another plausible suggestion is that the number of people receiving treatment has increased, however the quality of services may not have improved (Meadows & Bobevski, 2011). An alternative explanation is that current strategies have increased access for those with more severe impairments, although there may still be a treatment gap concerning milder cases (Burgess et al., 2009). This suggestion is somewhat supported by findings of the 2011 National Survey of Mental Health that indicated 63.5% of people with severe disorders; 40.2% of people with moderate disorders; and only 17.7% of people with mild disorders had used treatment services (Jorm, 2014).

All of these possibilities warrant further exploration and each may somewhat contribute to stable prevalence rates. However, a likely explanation for the unchanging rates of depressive disorders is that approaches to reduce prevalence in Australia have largely focused on treatment and to a lesser extent early intervention, and have so far ignored prevention (Jorm, 2014). Modelling studies suggest that less than 35% of the burden of affective disorders can be averted through treatment, even if we were to achieve optimal treatment and
access to services (Andrews et al., 2004). Whilst treatment is an important element of mental health promotion it is not in itself a complete answer. Population-level prevention approaches that target the predictors of depression may be a valuable strategy in reducing future prevalence.

The change in prevalence of mental health disorders is the product of the incidence (the rate of new disorders) and stability in disorders which is influenced by the rate of recovery or death of those with disorders (Jorm, 2014). To date Australian policy and practice has largely focused on improving the rate of recovery; however less has been done to address the incidence of depression (Jacka & Reavley, 2014; Jorm, 2014). Depression emerges during adolescence and thus the adolescent period of development provides a unique opportunity to reduce the incidence of depression. Reducing the incidence of depression will require prevention approaches.

**Prevention Approaches**

**Preventative interventions.** The Institute of Medicine (1994; 2009) conceptualises preventative interventions as: (a) universal interventions that target an entire population (e.g., everyone in a particular school); (b) selective preventions that target high-risk groups (e.g., children of parents with mental illness); and (c) indicated preventions that target those with sub-clinical symptoms. Some researchers consider selective and indicated interventions in combination calling them selective interventions whilst other differentiate between the two (e.g., Christensen, Pallister, Smale, Hickie, & Calear, 2010).

Preventative interventions should not be considered one-off events but rather need to be delivered in an ongoing way across the lifespan because depression is highly recurrent and environmental factors change over time.
COMMUNITY FACTORS AND ADOLESCENT DEPRESSION

(Birmaher et al., 2002; Costello et al., 2002; Jorm, 2014). An important aspect of reducing the incidence of mental health disorders is targeting risk and protective factors over the life-course to reduce the likelihood of mental health disorders occurring in the first instance. Risk and protective factors can be targeted through preventative interventions aimed at groups of individuals, however targeting predictors at the community-level is likely to offer a more cost effective means of preventing depression due to the large aggregate populations affected by community factors.

**Determinants of health.** There is considerable evidence that enhancing mental health in populations and reducing health inequalities requires addressing the underlying structural determinants of social and economic deprivation (Beaglehole & Bonita, 2009). Marmot and Wilkinson (1999) suggest it is firstly necessary to identify these determinants and study their impact on health, and it is then possible to address these determinants and subsequently improve mental health. It is important to distinguish between individual and environmental risk and protective factors, as the causes of differences in individual health may not be the same as the determinants of differences between populations (Rose, 1992; Syme, 1996).

A determinants approach integrates an understanding of how behaviour impacts on social processes and mental health risks, as well as how social and structural conditions act to enhance or diminish the opportunities for communities and populations to be healthy (Keleher & Armstrong, 2005). A determinants approach emphasises the importance of multi-level, community-wide interventions that target one or more influences on health rather than focussing directly on the disease (Keleher & Armstrong, 2005). Such an approach is
particularly pertinent to children and adolescents as the risk and protective factors associated with depression will likely influence other mental and physical disorders, as well as broader functioning and performance across a number of domains. The growing body of literature on health determinants suggests complex interactions operate among determinants and across social, economic, environmental, and biological dimensions (Keleher & Armstrong, 2005; WHO, 2003).

Whilst research examining preventative interventions has advanced rapidly over the last decade there is limited research examining how to best reduce the determinants of mental illness. The determinants of mental illness largely exist outside of the mental health sector making research and intervention in this area somewhat challenging (Jacka & Reavley, 2014). Addressing the determinants of mental health requires a national prevention strategy that coordinates preventative interventions across multiple settings and policy decisions that address the underlying determinants of mental health (Jorm, 2014).

**Cost-effectiveness of prevention.** Emerging evidence suggests that preventative approaches offer a cost-effective solution to reducing the economic burden of mental illness (Muñoz, Cuijpers, Smit, Barrera, & Leykin, 2010; Mihalopoulos & Chatterton, 2014). Munoz et al. (2010) review of 33 universal, selective, and indicated interventions suggested that current preventative interventions are effective at reducing the incidence of depression. A meta-analysis including 32 randomised control trials (RCTs) of universal, selective, and indicated interventions of both adolescent and adult populations found a 21% reduction in the incidence of depressive disorders (Van Zoonen et al., 2014). Munoz et al. (2010) suggested that a minimum of 22% of cases (Cuijpers, van...
Straten, Smit, Mihalopoulos, & Beckman, 2008) could be prevented. With multi-modal approaches (e.g., Cognitive Behavioural Therapy [CBT], Problem Solving Therapy, and nurse home visits) it has been estimated that up to 50% of cases could be prevented each year (van’t Veer-Tazelaar et al., 2009). It was estimated that in order to prevent one new case of depression it is necessary to deliver preventative interventions to 20 people (Van Zoonen et al., 2014).

To assess cost-effectiveness, the cost of intervening is considered in the context of the ongoing cost associated with an individual living with a mental health disability. In Australia the disability-adjusted life-years (DALYs) threshold of $50,000 is widely applied as an estimate of the ongoing per capita costs of mental illness, meaning that effective prevention interventions with per capita costs less than $50,000 are deemed cost-effective (Mihalopoulos & Chatterton, 2014). A review examining the cost effectiveness of preventative interventions of both adult and adolescent populations indicated that all of the identified effective preventative interventions cost less than the disability-adjusted life-years (DALYs) threshold of $50,000 (Mihalopoulos, & Chatterton, 2014). These findings suggest that a range of preventative approaches are cost-effective in offsetting the costs associated with depression.

Preventative interventions specifically targeting children and adolescents are also cost-effective at reducing the incidence of depression (Mihalopoulos, Vos, Pirkis, & Carter, 2012). A meta-analysis of 8 studies suggested that school-based CBT interventions for children with sub-clinical depression cost approximately $5400 (Mihalopoulos et al., 2012). Thus, the costs associated with intervening to prevent the onset of depression are significantly lower than the ongoing expenses associated with depression (Mihalopoulos et al., 2012).
Intervening during adolescence is likely to have significant long-term economic benefits for society. The next section considers opportunities for prevention approaches during the adolescent period of development.

**Frameworks for Organising Predictors of Depression in Children and Adolescents**

In order to apply prevention frameworks to the development of depression it is necessary to consider relevant developmental science. This section provides frameworks for identifying and organising the predictors of child and adolescent depression. It begins by presenting a developmental psychopathology perspective relevant to the onset of psychopathology. It goes on to consider sensitive periods of development and highlights adolescence as a critical period for prevention. It then presents Bronfenbrenner’s framework for organising risk and protective factors associated with depression.

**Developmental Psychopathology**

*Developmental psychopathology.* Developmental psychopathology provides a framework for understanding the aetiology of psychopathological and normative behaviours by emphasising the developmental context and its impact (Cicchetti & Toth, 1998; Cicchetti & Rogosch, 2002). Developmental psychology is grounded in an understanding of how normal developmental pathways whereby individuals do not develop a particular disorder differ from atypical pathways of development that predict psychopathology. The goal of developmental psychologists is to understand how individuals develop the capacities necessary to navigate a particular stage of development, and the factors that result in the failure to develop these capacities (Sroufe, 2013).
Developmental psychopathology aims to inform prevention and intervention by identifying the precursors and pathways that predict psychopathology, and the factors that cause individuals to move along such pathways or return to normal developmental pathways (Sroufe, 2013). The principles of equifinality and multifinality are used to explain patterns of developmental pathways. Equifinality posits that there can be multiple different pathways to the same outcome (e.g., multiple factors predict depression), whereas multifinality argues that one predictor or outcome can have several different outcomes (e.g., family conflict is a risk factor for depression and Oppositional Defiance Disorder; Cicchetti & Rogosch, 1996).

Another important principle of developmental psychopathology is the study of continuity and change. Homotypic and heterotypic continuity describes different types of pathways or continuities over time. Homotypic continuity refers to the stability of the same type of problem over time (e.g., depressive symptoms in adolescence preceding depression in adulthood), supporting the concept that a particular problem presents across an individual’s development (Costello, Copeland, & Angold, 2011).

Heterotopic continuity refers to the development of one type of problem leading to another type of problem (e.g., anxiety preceding depression in adolescents), supporting the idea that the nature of a presentation may shift over time (Costello et al., 2011). Both forms of continuity may stem from underlying vulnerability factors, either genetic or environmental, and/or the cumulative impact of the first problem that increases the risk of developing the same problem again or developing a different type of problem (Costello et al., 2011). Heterotypic continuity may also reflect the idea that different phenotypic
expressions of an underlying problem manifest in diverse ways at different developmental stages (Costello et al., 2011).

**Sensitive stages of development.** Developmental psychopathology argues that similar types of input experience may manifest differently during different developmental stages (Pickles & Hill, 2006; Cicchetti, 2006). Theories of early developmental vulnerability suggest there are sensitive stages of development where risk and protective factors may have a stronger impact than during other developmental stages (O’Connor, 2006). Expanding knowledge relevant to the life-course development and aetiology of mental illness points to childhood and adolescence as critical periods (Letcher, Smart, Sanson, & Toumbourou, 2009).

Numerous studies support the notion of sensitive periods with a large body of research suggesting that early childhood is a particularly important period with early childhood risk factors leading to later symptom development (Karevold, Roysamb, Ystrom, & Mathiesen, 2009; Leckman-Westin, Cohen, & Stueve, 2009). Exposure to stressors during early childhood may impact the development of important neurological, cognitive, and behavioural processes and capabilities; underlying attachment; interpersonal skills; emotion regulation; and stress responses that subsequently increase risk for developing depression (Goodman & Brand, 2009).

Whilst much research has been dedicated to understanding the impact of stressors in early childhood, fewer studies have examined middle childhood (6-10 years) as a vulnerable period (Huston & Ripke, 2006). Middle childhood is characterized by the development of specific physical, cognitive, emotional and social abilities. During this stage, most children master academic (e.g., reading, writing, and numeracy), emotional and social (e.g., developing meaningful
friendships, peer conflicts and emotional-social competencies) and cognitive (e.g., increased self-awareness) skills (Huston & Ripke, 2006). The transition to school introduces increased demands and expectations on children in terms of academic and social performance. As such social competence and positive peer interactions during this stage may be critical for the prevention of depression (Huston & Ripke, 2006).

Adolescence is widely accepted as a critical period of development. Indeed developmental theorists argue that it is one of the most crucial phases of psychological and biological development, second only to early childhood (Viner et al., 2012). During adolescence biological, social, and psychological systems undergo significant developmental changes (Feldman & Elliott, 1990; Spear, 2000a, 2000b). Developmental effects of puberty and brain development during adolescence result in new capacities and behaviours that influence a young person’s trajectory in social, educational, and health domains (Viner et al., 2012). Whilst the transitions to high school is widely recognised as a significant period of stress and vulnerability, there is limited research examining this period of early adolescence (10-14 years).

**Adolescent development and psychopathology.** Evidence indicates that the risk for numerous mental health disorders emerges during adolescence, including depression, schizophrenia and substance-use disorders (Paus, Keshavan, & Giedd, 2008; Walker, 2002). However, each adolescent is not at equal risk for developing psychopathology, either because they do not exhibit problem behaviours or because the demonstration of such behaviours does not result in long-term negative consequences (Luciana, 2013). It is important to dissect the
context and broader systems in which problematic behaviours develop in order to understand the multifinality of outcomes (Cicchetti & Rogosch, 1996).

Whilst the majority of individuals thrive during the adolescent period, for some navigating the tasks of adolescent development presents unique challenges. An important element of the adolescent period of development is navigating choices relating to potentially health compromising behaviours (Spear, 2000a). Both individual and environmental factors likely determine the pathways different adolescents take as they negotiate self-management decisions. Some adolescents experience serious negative consequences of poor decisions (e.g., death and disability; incarceration; school failure); some develop psychiatric disorders (e.g., substance use disorder; adolescent onset mental disorders) in relation to decision-making and stressors in this period; while for the majority trial and error learning in this period leads to positive outcomes (Cicchetti & Rogosh, 2002). Influential factors may involve individual differences (e.g., neurobiology; gender; and physical health) and environmental factors such as differences in family and peer contexts, and community factors such as disadvantage (Casey & Caudle, 2013; Farah, Noble, & Hurt, 2007; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001). The next section presents a framework for organising these many contexts.

**Ecological Systems Theory**

One of the most widely applied theories of children’s development is Bronfenbrenner’s (1979) ecological systems theory. Bronfenbrenner’s ecological theory provides a framework for organising the biological and psychological aspects of the child’s development in terms of the interactive influence of the peer, family, school, neighbourhood, community, and society level social systems.
Urie Bronfenbrenner criticised traditional psychological approaches to research for focussing on narrow understandings of individual influences on behaviour and neglecting the broader system environment influences (Brendtro, 2006).

Bronfenbrenner argued that children’s development must be considered in the context of the child’s environment (Vaughn & Jacquez, 2011). Children and adolescents develop in the context of multiple environmental influences. Adolescents’ mental health is modified by factors external to the individual such as: social and economic factors in the broader community (Viner et al., 2012); family context (McLeod, Weisz, & Wood, 2007; Yap, Pilkington, Ryan, & Jorm, 2014); and the peer and school environments (Blakemore & Mills, 2014; Kidger, Araya, Donovan, & Gunnell, 2012). The present thesis focuses on emerging research supporting the role of community factors in adolescent mental health and wellbeing (Fowler, Tompsett, Braciszewski, Jacques-Tura, & Baltes, 2009).

Bronfenbrenner’s model conceptualises how community influences interact with other developmental influences. At the core of Bronfenbrenner’s model is the individual who brings personal experiences and characteristics (e.g., gender, race, sexual orientation) to every setting and context in which they interact (Bronfenbrenner, 1993). The innermost layer is the microsystem, the individual’s immediate context of development (Bronfenbrenner, 1979). The microsystem represents the activities, roles, and interpersonal relationships experienced by the individual in their immediate surroundings such as peers, family, and school (Bronfenbrenner, 1979). All relationships are reciprocal and bidirectional such that the peers, parents, and teachers influence the individual, but the individual also impacts on the behaviour of others (Bronfenbrenner, 1995).

From the microsystem the layers move outward to factors more distal to the individual. The next layer is the mesosystem, which represents the interaction between two immediate environments (or microsystems) that impact the individual (Bronfenbrenner, 1995). For example a parents’ involvement in neighbourhood activities may strengthen support networks for the adolescent. The next layer, the Exosystem, refers to settings that may not directly include the individual but indirectly affect the individual’s experience in immediate settings (Bronfenbrenner, 1979). The exosystem may include things such as school policies on bullying that impact on peer relationships in the school settings or work policies that impact on a parents’ employment. The final layer, the macrosystem, includes broader societal and cultural influences such as values, laws, and customs (Bronfenbrenner, 1979). The macrosystem includes factors
such as community disadvantage that influence neighbourhood attitudes and culture. The macrosystem may also include health promotion strategies that have broad impacts on the society in which an individual lives.

**Predictors associated with Child and Adolescent Depression**

This section identifies predictors of adolescent depression and current preventative interventions are reviewed. This chapter concludes by presenting a rationale for the current thesis in the context of current gaps in the evidence base.

**Predictors of Child and Adolescent Depression**

The literature, research, and theories discussed in the earlier sections above suggest that depression emerges during the early adolescent period of development. Adolescents are particularly vulnerable given the biological and social development processes they experience during this transition as well as the developmental tasks required of them during this critical period (Blakemore & Mills, 2014). The first step towards preventing depression is identifying risk and protective factors present in early adolescence that are associated with depression (Fraser, 2004).

The section that follows summarises the findings of current research to identify what is known of the predictors of child and adolescent depression. Table 1 presents risk and protective factors at the individual, family, school, and community-level that have been identified through meta-analytic findings or systematic reviews. Systematic reviews (SR) and meta-analyses (MA) are considered the highest hierarchy of evidence in synthesizing the findings of health research (Evans, 2003; Green, 2005; National Health and Medical Research Council, 1999). In the current thesis strong evidence was determined by significant meta-analytic findings or the presence of significant findings in three
or more studies included in systematic reviews with little contradictory evidence (Cairns, Yap, Pilkington, & Jorm, 2014).

Table 1 presents risk and protective factors and citations to the reviews that identified their effect. As presented in Table 1, strong evidence indicated that a large number of risk and protective factors were associated with child and adolescent depression at the individual-level with these effects identified from a large range of systematic reviews (Cairns et al., 2014 [MA; k = 113]; Davis, Matthews, & Twamley, 1999 [MA; k = 119]; Gladstone & Kaslow, 1995 [MA; k = 28]; Grant et al., 2003 [MA; k = 46]; Hosang, Shiles, Tansey, McGuffin, & Uher, 2014 [MA; k = 22]; Joiner & Wagner, 1995 [MA; k = 27]; Kaufman, Martin, King, & Charney, 2001 [MA; k = 89]; Lopez-Duran, Kovacs, & George, 2009 [MA; k = 34]; Marshal et al., 2011 [MA; k = 12]; Nelson & Harwood, 2011 [MA; k = 31]; Twenge & Nolen-Hoeksema, 2002 [MA; k = 310]). As shown in Table 1, emerging evidence also supported an association between depression and a number of individual-level factors (Cairns et al., 2014 [MA; k = 113]). Further as presented in Table 2, a number of studies indicated strong evidence that predictors in childhood were associated with depression in adulthood (systematic reviews that identified these findings included: Lindert et al., 2014 [MA; k = 14]; Loth, Drabick, Leinbenluft, & Hulvershorn, 2014 [MA; k = 10]).

As presented in Table 1, strong evidence indicated that a large number of risk and protective factors were associated with child and adolescent depression at the family-level (Goodman et al., 2011 [MA; k = 121]; Grant et al., 2003 [MA; k = 46]; Kane & Garber, 2004 [MA; k = 23]; McLeod et al., 2007 [MA; k = 45]; Mendes et al., 2012 [SR; k = 30]; Yap et al., 2014 [MA; k = 140]). As shown in Table 1 relatively few studies examined school and community factors. Strong
evidence was associated with only two factors at the school-level (Kidger et al., 2012 [SR; $k = 28$]). Further as presented in Table 2, strong evidence indicated only one school-level predictor in childhood associated with depression in adulthood (Ttofi, Farrington, Losel, & Loeber, 2011 [SR; $k = 8$]).

As presented in Table 1, there was strong evidence for only one predictor associated with child and adolescent depression at the community-level (Fowler et al., 2009 [MA; $k = 114$]. Whilst two meta-analyses examined the association between poverty and adolescent depression, in one study it was not clear whether disadvantage (SES) was determined at the individual, family, or community-level (Twenge & Nolen-Hoeksema, 2002 [MA; $k = 310$]), and the other only included studies that examined disadvantage across all levels (Grant et al., 2003 [MA; $k = 46$]). One study suggested that poverty was associated with immediate but not later depression (Grant et al., 2003), whilst the other did not identify any significant association between depression and poverty (Twenge & Nolen-Hoeksema, 2002).
Table 1  
Summary of Risk and Protective Factors Identified from Systematic Reviews to have Strong Evidence as Predictors of Depression in School-Aged Children (4-18 years)

<table>
<thead>
<tr>
<th>Individual-level factors</th>
<th>Family-level factors</th>
<th>School-level factors</th>
<th>Community-level factors</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Risk factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sexual orientation</td>
<td>Maternal depression</td>
<td>School connectedness</td>
<td>Witnessing &amp; victim of</td>
</tr>
<tr>
<td>(Marsh et al., 2011)</td>
<td>(Goodman et al., 2011; Mendes et al., 2012)</td>
<td>(Kidger et al., 2012)</td>
<td>community violence (Fowler et al., 2009)</td>
</tr>
<tr>
<td>Hispanic background</td>
<td>Paternal depression</td>
<td>Teacher support</td>
<td></td>
</tr>
<tr>
<td>(Twenge &amp; Nolen-Hoeksema, 2002)</td>
<td>(Kane and Garber, 2004)</td>
<td>(Kidger et al., 2012)</td>
<td></td>
</tr>
<tr>
<td>Alcohol, cannabis, and other drug use</td>
<td>Parental over involvement (McLeod et al., 2007; Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Cairns et al., 2014)</td>
<td>Parental aversiveness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tobacco use (Cairns et al., 2014)</td>
<td>Parental aversiveness (McLeod et al., 2007; Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daily stress and life events (Davis et al., 1999)</td>
<td>Interparental conflict (Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Media use (Cairns et al., 2014)</td>
<td>Parental aversiveness (McLeod et al., 2007; Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dating during adolescence (Cairns et al., 2014)</td>
<td>Interparental conflict (Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HPA axis dysregulation indicated by increased cortisol production (Lopez-Duran et al., 2009)</td>
<td>Parental aversiveness (McLeod et al., 2007; Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Learning disabilities (Nelson &amp; Harwood, 2011)</td>
<td>Interparental conflict (Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child and adolescent externalising symptoms (Grant et al., 2003)</td>
<td>Inconsistent discipline (Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative emotion regulation strategies* (Cairns et al., 2014)</td>
<td>Inconsistent discipline (Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dieting* (Cairns et al., 2014)</td>
<td>Negative parenting (Grant et al., 2003; Mendes et al., 2012)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative coping strategies (Cairns et al., 2014)</td>
<td></td>
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</tbody>
</table>

**Protective factors**

<table>
<thead>
<tr>
<th>School connectedness</th>
<th>Teacher support</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Kidger et al., 2012)</td>
<td>(Kidger et al., 2012)</td>
</tr>
</tbody>
</table>

* = Emerging Evidence
### Table 2

**Summary of Risk Factors Associated with Adult Depression (18 years +) that Emerge During Childhood (before 18 years)**

<table>
<thead>
<tr>
<th>Individual-level factors</th>
<th>Family-level factors</th>
<th>School-level factors</th>
<th>Community-level factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical activity and sport (Cairns et al., 2014)</td>
<td>Parental withdrawal* (McLeod et al., 2007; Yap et al., 2014)</td>
<td>Victim of school bullying (Tufti et al., 2011)</td>
<td></td>
</tr>
<tr>
<td>Relationship with positive peers (Cairns et al., 2014)</td>
<td>Parental warmth (McLeod et al., 2007; Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-disclosure to parents* (Cairns et al., 2014)</td>
<td>Parental monitoring (Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Healthy diet and weight (Cairns et al., 2014)</td>
<td>Parental autonomy granting (Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep (Cairns et al., 2014; Kaufman et al., 2001)</td>
<td>Parental encouragement of sociability* (Yap et al., 2014)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brain-derived neurotrophic factor gene mediates the impact of stressful life events (Hosang et al., 2014)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Positive coping strategies (Cairns et al., 2014)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Attributional style (Gladstone &amp; Kaslow, 1995; Joiner &amp; Wagner, 1995)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* – Emerging Evidence
**Gaps in the evidence base.** The current evidence base indicates a number of factors are associated with child and adolescent depression. However, there are a number of gaps in the current literature including:

1. As outlined above and in Table 1 and 2, evidence is well established at the individual, family, and school-level with a large number of meta-analyses and systematic review carried out in this area. However, the literature regarding community-level predictors is less organised with only one meta-analysis examining factors associated with child and adolescent depression at the community-level (Fowler et al., 2009).

2. All of the meta-analyses and systematic reviews outlined in Table 1 and 2 assess clinical depression despite sub-clinical depression being associated with negative outcomes (Klein et al., 2013; Lewinsohn et al., 2004; Fergusson et al., 2005).

3. Evidence indicates gender differences in risk and protective factors for depression (Fernandez Castelao & Kroner-Herwig, 2013; Saraceno, Heron, Munafo, Craddock, & van de Bree, 2012; Sweeting, Young, West, & Der, 2006). Gender difference have been identified at the individual (e.g., Saraceno et al., 2012; Twenge & Nolen-Hoeksema, 2002), family (e.g., Leve, Kim, & Pears, 2005; Yap et al., 2014), and school (e.g., Kidger et al., 2012) levels. However, research in this area has been criticized for failing to explore the effect of predictors by gender (Shanahan, Copeland, Costello, & Angold, 2008).

4. Whilst current research has established associations between individual, peer, family, school and community-level factors and child and adolescent depression research in this area has been criticized for
failing to include a wide range of risk and protective factors at all levels (Shanahan et al., 2008).

5. The above review of the literature did not identify any research examining how much of the variance in depressive symptoms could be attributed to factors at each tier (individual/peer, family, school, and community).

6. There is limited research using Australian samples (Cairns et al., 2014; Yap et al., 2014). Indeed, Australian policy in this area largely relies on international research (McDermott et al., 2010).

**Preventative Interventions for Depression in School-aged Children**

The previous sections established the importance of preventative approaches in reducing the prevalence of depression. Intervening at the community-level likely provides a cost-effective means for reducing the burden of depression. The next section will consider reviews of current preventative interventions.

Numerous reviews support the effectiveness of preventative interventions in adolescent samples (Christensen et al., 2010; Horowitz & Garber, 2006; Merry et al., 2011). Initial findings suggested that selective and indicated interventions were more effective than universal programs in the prevention of child and adolescent depression (Horowitz & Garber, 2006). Some researchers suggested that due to the limited evidence supporting the effectiveness of universal interventions offered in school settings, it would be premature to disseminate these interventions more widely (Spence & Shortt, 2007). Indeed, the 2010 Clinical Practice Guidelines recommended:
Psychosocial interventions of the types investigated to date are not
currently recommended for universal prevention of depressive symptoms
or major depressive disorder in the adolescent population. More research
is needed to identify effective approaches. (McDermott et al., 2010, p. 19).
However, a Cochrane Review later suggested that universal approaches
are effective in reducing depression (Merry et al., 2011). These findings were
replicated in a review of school-based RCTs (Corrieri et al., 2013). Further, a
meta-analysis of 32 RCTs, including both adult and adolescent populations,
indicated no difference in the effectiveness of interventions based on the type of
approach (universal, selective, or indicated) implemented (Van Zoonen et al.,
2014).

The failure to achieve significant effects or only produce small effect sizes
in earlier studies may reflect the content of interventions rather than the overall
approach (Horowitz and Garber, 2006). The majority of preventative interventions
are Cognitive Behavioural Therapy (CBT) based and are offered in school settings
(Christensen et al., 2010; Merry et al., 2011; Mychailyszyn, Brodman, Read, &
Kendall, 2012). Across reviews CBT approaches have been shown to be
efficacious (Christensen et al., 2010; Mychailyszyn et al., 2012).

Reviews have also assessed the effectiveness of different therapeutic
approaches. A meta-analysis of 32 RCTs, including both adult and adolescent
populations, indicated no difference in the effectiveness of interventions based on
the treatment (CBT, Interpersonal Psychotherapy, or other) provided (Van Zoonen
et al., 2014). Further, a review of Australian interventions indicated that CBT,
Interpersonal Psychotherapy, and psycho-education based-interventions
implemented in school settings all indicate effectiveness (Neil & Christensen,
2007). Similarly, a review that assessed treatment content (e.g., problem solving skills, reducing negative cognitions) indicated no differences in effect sizes (Stice, Shaw, Bohon, Marti, & Rohde, 2009).

The failure to identify differences in effect sizes based on the type of treatment or intervention approach has led researchers to consider other possible moderators. A meta-analysis examining moderators of treatment effects identified that programs with homework tasks; programs of shorter duration; and interventions delivered by mental health professionals as opposed to teaching staff produced larger effect sizes (Stice et al., 2009). Programs were more effective with females; older adolescents; and high-risk samples (Stice et al., 2009).

Whilst prevention interventions appear to have some efficacy in reducing the prevalence of depression, effect sizes indicate small-scale effectiveness (Corrieri et al., 2013). Current preventative intervention have been criticised for being more treatment than preventative focused, usually only targeting individual-level factors despite calls for the use of multimodal approaches (AACAP, 2007; Horowitz & Garber, 2006; Spence & Shortt, 2007). Brief interventions that only target individual-level factors such as improving problem solving skills will likely be insufficient to produce lasting effects in reducing the prevalence of depression (Spence & Shortt, 2007).

Further, to achieve population-level reductions in depression it is necessary to address the underlying determinants of mental health through coordinated responses across a range of services and settings (Jacka & Reavley, 2014; Jorm, 2014). Population-level approaches are needed to reduce community prevalence rates. Such approaches have proven to be effective in Australia in minimising the impact of other health outcomes such as suicide and smoking.
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(Jorm, 2014). However, there is limited understanding of community-level predictors of adolescent depression that could be targeted through such approaches. Further, there are no known reviews of community interventions targeting risk and protective factors for adolescent depression.

Summary of Chapter 2 and Rationale for the Current Studies

Australia has made advances in closing the treatment gap for mental health disorders (Andrews et al., 2004). However, even in a perfect health care system, modelling suggests that only 35% of the burden of affective disorders could be averted through optimal treatment (Andrews et al., 2004). The unchanging pattern of prevalence rate trends in recent years suggest that the current approach of increasing treatment investment has had little success at reducing the significant burden of mental disorders (Jorm and Reavley, 2012; Reavley et al., 2011). In order to reduce the prevalence of depression, prevention approaches are needed.

Evidence indicates that preventative interventions offer a cost-effective means of reducing the incidence of depression (Merry et al., 2011; Mihalopoulous & Chatterton, 2014; Munoz et al., 2010; Van Zoonen et al., 2014). Some researchers suggest that the limited effectiveness of current universal preventative approaches may reflect the content of these interventions (Horowitz & Garber, 2006; Spence & Shortt, 2007). Current preventative interventions have been criticised for being more focussed on treatment and early intervention than prevention, usually only targeting individual-level factors despite calls for the use of multimodal approaches (AACAP, 2007). To achieve population-level reductions in depression it is necessary to address the underlying determinants of mental health through coordinated responses across a range of services and settings (Jacka & Reavley, 2014; Jorm, 2014).
Identifying predictors of adolescent depression more distal to the individual such as community-level factors offers a potentially cost-effective means for intervening at the population-level. Evidence is well established for predictors that can be targeted at the individual, family, and school-level (e.g., Carins et al., 2014; Yap et al., 2014). However, the literature regarding community-level predictors and interventions is less organised.

The majority of research in this area has focused on clinical depression. However, in order to reduce the burden of depression it is necessary to reduce the incidence of depression by preventing its onset. Targeting elevated depressive symptoms (sub-clinical and clinical) during adolescence may offer an opportunity to prevent the development of clinical depression (Lewinsohn et al., 1994; Mazza et al., 2009). Quantifying the prevalence of elevated depressive symptoms and whether this varies by locality in Australian adolescents is important in advancing research and public health knowledge in this area (Christensen, Batterham, Griffiths, Gosling, & Hehir, 2013).

Identifying and targeting the predictors of sub-clinical and clinical depression through indicated interventions and population-level approaches may offer important new opportunities to reduce the incidence of depression. It is important to understand how much of the variance in adolescent depression between localities is explained by factors at the community, school, family, and individual/peer level in order to determine the most cost-effective opportunities for intervention. Identifying these differences by local areas (SLAs) provides practical opportunities to intervene as local funding and services in Australia are often coordinated by local governments and regional organisations (e.g., Primary Health Networks).
Meta-analytic findings indicate important gender differences in prevalence rates of depression (Twenge & Nolen-Hoeksema, 2002). Emerging evidence suggests these gender differences may be influenced by risk and protective factor differences during adolescence (Fernandez Castelao & Kroner-Herwig, 2013; Saraceno et al., 2012; Sweeting et al., 2006). As such research needs to consider gender specific prevalence and predictors of adolescent depression.

**Study Aims**

The purpose of the current thesis was to establish whether community-level variables accounted for an important share of the potentially modifiable factors that predict child and adolescent depression. This was achieved by three studies:

1. Study 1 aimed to complete a systematic review of community risk and protective factors and interventions targeting community-level factors for depression in school-aged children (4-18 years).

2. Study 2 aimed to quantify the prevalence of elevated depression symptoms and to identify the variation in adolescent depression between statistical local areas (SLAs) using a large nationally representative sample of Australian adolescents (10-14 years). Study 2 also aimed to identify whether community-level disadvantage was associated with depressive symptoms in Australian adolescents.

3. Study 3 aimed to identify whether community-level variables explained a large component of locality variation in depression symptoms relative to individual, family, and school factors. Study 3 also aimed to identify whether community-level factors such as community disadvantage and State were explained by other predictors of depressive symptomology.
The next chapter (Chapter 3) introduces Study 1 a systematic review of community level factors influencing depression in school-aged children.
CHAPTER 3: STUDY 1

Context and Rationale

Current Australian mental health policies have not emphasised community factors as influences on child and adolescent depression. Whilst systematic reviews and meta-analyses have identified individual, peer, family and school-level risk and protective factors as predictors of depression in school-aged children, there is limited understanding of the role community-level factors play in child and adolescent depression.

Improving understanding of the role of community-level variables may be of particular importance in preventative science, as community factors affect large aggregate populations and hence may provide the opportunity for cost effective interventions. Further community-level variables such as community disadvantage have been associated with a range of child outcomes such as health and education, as well as influencing outcomes for a broad section of community members. Thus addressing these underlying determinants of health is likely to have wide and far-reaching impacts for society.

The next chapter presents Study 1 that aimed to complete a systematic review of community risk and protective factors and interventions targeting community-level factors for depression in school-aged children (4-18 years). Given there has been no prior review in this area, broad search terms were adopted to identify both peer-reviewed and non-peer reviewed studies that identified community-level observational and intervention studies associated with depression in school-aged children.
## Authorship Statement for Article 1

**Submitted to:** Australian and New Zealand Journal of Psychiatry  
**Reference Style:** SAGE Harvard

### 1. Details of publication and executive author

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<th>Publication details</th>
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<td>Community Factors Influencing Child and Adolescent Depression: A Systematic Review and Meta-Analysis</td>
<td>Published in the Australian and New Zealand Journal of Psychiatry</td>
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<th>Email or phone</th>
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<tbody>
<tr>
<td>Katie Stirling</td>
<td>School of Psychology</td>
<td><a href="mailto:kstirlin@deakin.edu.au">kstirlin@deakin.edu.au</a></td>
</tr>
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### 2. Inclusion of publication in a thesis

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### 3. HDR thesis author’s declaration

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<td>School of Psychology</td>
<td>Investigation of Community Factors Associated with Child and Adolescent Depression</td>
</tr>
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</table>

If there are multiple authors, give a full description of HDR thesis author’s contribution to the publication (for example, how much did you contribute to the conception of the project, the design of methodology or experimental protocol, data collection, analysis, drafting the manuscript, revising it critically for important intellectual content, etc.)

Conception, writing and compilation of manuscript, established methodology, completed literature search and appraisal, data analyses, preparation of tables and figure.

*I declare that the above is an accurate description of my contribution to this paper,*

Signature and date: Katie Stirling
4. Description of all author contributions

<table>
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<th>Name and affiliation of author</th>
<th>Contribution(s) (for example, conception of the project, design of methodology or experimental protocol, data collection, analysis, drafting the manuscript, revising it critically for important intellectual content, etc.)</th>
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<tr>
<td>Katie Stirling, Deakin University</td>
<td>Conception, writing and compilation of manuscript, established methodology, completed literature search and appraisal, data analyses, preparation of tables and figure.</td>
</tr>
<tr>
<td>Professor John Toumbourou, Deakin University</td>
<td>Supervised and assisted with manuscript compilation, editing of manuscript, and contributed to methodology.</td>
</tr>
<tr>
<td>Dr Bosco Rowland, Deakin University</td>
<td>Editing of manuscript, provided feedback on the methodology, and completed independent review of study quality of included studies.</td>
</tr>
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</table>

5. Author Declarations

I agree to be named as one of the authors of this work, and confirm:

i. that I have met the authorship criteria set out in the Deakin University Research Conduct Policy,

ii. that there are no other authors according to these criteria,

iii. that the description in Section 4 of my contribution(s) to this publication is accurate,

iv. that the data on which these findings are based are stored as set out in Section 7 below.

If this work is to form part of an HDR thesis as described in Sections 2 and 3, I further consent to the incorporation of the publication into the candidate’s HDR thesis submitted to Deakin University and, if the higher degree is awarded, the subsequent publication of the thesis by the university (subject to relevant Copyright provisions).

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<td>20.12.15</td>
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<td>Prof John Toumbourou</td>
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<tr>
<td>Dr Bosco Rowland</td>
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<td>20.12.15</td>
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7. Data storage

The original data for this project are stored in the following locations. (The locations must be within an appropriate institutional setting. If the executive author is a Deakin staff member and...
data are stored outside Deakin University, permission for this must be given by the Head of Academic Unit within which the executive author is based.)

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<td>December, 2015</td>
<td>John Toumbourou</td>
</tr>
</tbody>
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This form must be retained by the executive author, within the school or institute in which they are based.

If the publication is to be included as part of an HDR thesis, a copy of this form must be included in the thesis with the publication.
Community Factors Influencing Child and Adolescent Depression: A Systematic Review and Meta-analysis

Abstract

Objective: Depression has been identified as a priority disorder among children and adolescents. While numerous reviews have examined the individual and family factors that contribute to child and adolescent depressive symptoms, less is known about community-level risk and protective factors. The aim of this study was to complete a systematic review to identify community risk and protective factors for depression in school-aged children (4–18 years).

Method: The review adopted the procedures recommended by the Cochrane Non-Randomised Studies Methods Working Group and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. A comprehensive literature search was conducted to identify both observational and intervention study designs in both peer-reviewed and non-peer reviewed publications.

Results: A total of 21 studies met the inclusion criteria. Seventeen of the 18 community association studies and 2 of the 3 intervention studies reported one or more significant effects. Results indicated that community safety and community minority ethnicity and discrimination act as risk factors for depressive symptoms in school-aged children. Community disadvantage failed to achieve significance in meta-analytic results but findings suggest that the role of disadvantage may be influenced by other factors. Community connectedness was also not directly associated with depressive symptoms.

Conclusion: There is evidence that a number of potentially modifiable community-level risk and protective factors influence child and adolescent
depressive symptoms suggesting the importance of continuing research and intervention efforts at the community-level.
Introduction

Worldwide, approximately 5% of adolescents suffer from depression (Costello et al., 2006; World Health Organisation [WHO], 2012). Adolescence is considered a period of high risk for the onset of psychopathology (Spear, 2000). In the United States, approximately 75% of mental disorders present before the age of 24, and 50% begin before the age of 14 (Kessler et al., 2005). Adolescent depression has also been identified as a predictor for depression in adulthood (Lewinsohn and Essau, 2002). Retrospective studies of depressed adults and prospective studies of adolescents indicate that depression often emerges during the mid-adolescent (13–15 years) period of development (Hankin et al., 1998; Lewinsohn and Essau, 2002). Longitudinal evidence suggests more pronounced increases in the incidence of major depressive disorder after age 11 and again after age 15, with rates levelling in young adulthood (Kim-Cohen et al., 2003). Expanding knowledge relevant to the life-course development and aetiology of internalising problems in childhood and adolescence suggest these periods in development as opportune times for intervention (Letcher et al., 2009).

Depressive disorders are identified as the leading contributor to the burden of disease and injury for children and adolescents (Gore et al., 2011; WHO, 2012). Some studies suggest that medical expenditure on children with depression is significantly higher in comparison to children with other mental health conditions (Glied and Neufeld, 2001; Mandell et al., 2003). For this age group, depression is associated with a number of negative outcomes including academic difficulties, impaired social relationships, high-risk sexual behaviour and a 30-fold increased risk of completed suicide (Horowitz and Garber, 2006). Adolescents with depressive disorders are also at high risk for legal problems,
exposure to negative life events (American Academy of Child and Adolescent Psychiatry [AACAP], 2007), as well as adolescent pregnancy and early parenthood (Jaycox et al., 2009). A high proportion of adolescents who experience depression will also experience another mental disorder (commonly a substance-use, anxiety or behavioural disorder), as well as an increased likelihood of developing other chronic illnesses (Bhatia and Bhatia, 2007). The effects of adolescent depression extend beyond the individual. Parents of depressed adolescents report lower parent mood and increased strain (Jaycox et al., 2009).

Due to its prevalence and association with impairment and long-term consequences, depression has been identified as an international priority (WHO, 2012). Historically, efforts to reduce the impact of mental illness have focussed on the treatment of intra-psychic elements of individuals (Herrman et al., 2005). However, even in a perfect health care system, modelling suggests that only 35% of the burden of affective disorders could be averted through optimal treatment (Andrews et al., 2004). The unchanging pattern of prevalence rate trends in recent years suggests the current approach of increasing treatment investment has had little success at reducing the significant burden of mental disorders (Jorm and Reavley, 2012; Reavley et al., 2011). In order to reduce the prevalence of depression, prevention approaches are needed.

Emerging evidence suggests that preventative approaches offer a cost-effective solution to reducing the economic burden of mental illness (Mihalopoulos and Chatterton, 2015; Mihalopoulos et al., 2012; Muñoz et al., 2010). Furthermore, evidence indicates that high quality, comprehensive programmes delivered in collaboration with schools and communities can not only improve child mental health but also lead to improved general health, social
functioning and academic and work performance (Barry et al., 2009). Intervening at the community-level holds substantial promise as it affords an opportunity to affect whole populations (Rose, 1992, 2001).

Mental health promotion aims to modify determinants of mental health in order to improve the mental health and well-being of populations, communities or individuals (Commonwealth Department of Health and Aged Care [CDHAC], Mental Health and Special Programs Branch, 2000; Keleher and Armstrong, 2005). It is generally achieved through the implementation of multiple evidence-based interventions across a number of programmes, settings, sectors and policies (Keleher and Armstrong, 2005). These interventions aim to reduce risk factors and increase protective factors, in all areas of influence – individual, family, school and community (Beaglehole and Bonita, 2009).

The impact of individual-, peer- and family-level factors on child and adolescent depression is frequently reported in the literature (Cairns et al., 2014; McLeod et al., 2007; Twenge and Nolen-Hoeksema, 2002; Yap et al., 2014). Meta-analytic findings indicate sound evidence for a number of individual-/peer-level factors including relationship with positive peers, alcohol and other drug use, adaptive stress coping, sleep, weight and dieting (Cairns et al., 2014). Meta-analytic findings also suggest family conflict, parental over involvement, parental warmth, parental hostility and a number of other parenting factors are associated with adolescent depression (McLeod et al., 2007; Yap et al., 2014). Similarly, meta-analytic evidence suggests that the school environment also exerts a significant influence on depression in school-aged children. Adolescents’ perceptions of school connectedness (Kidger et al., 2012), teacher support (Kidger et al., 2012) and school attainment (Riglin et al., 2014) are associated with
adolescent depression. School bullying has also been associated with later depression (Ttofi et al., 2011).

The research examining community-level risk and protective factors is less organised. There has only been one meta-analysis or review in this area. A meta-analysis that examined the impact of community violence exposure on mental health outcomes (Fowler et al., 2009) identified that community violence was associated with increased internalising symptoms.

Little is known about what other community-level factors may influence the development of adolescent depression. While socio-economic status is frequently implicated as a risk factor for child and adolescent depression (Herrman et al., 2005; McDermott et al., 2010), there appears to be limited evidence to support these claims. Indeed, a meta-analysis of individual-level variables did not find a significant association between socio-economic status and childhood depression (Twenge and Nolen-Hoeksema, 2002). To date, the existing literature relevant to community risk and protective factor impacts on child and adolescent depression is disorganised, providing an overall limited evidence base to advance researchers and policy makers. This study aims to identify what community risk and protective factors influence the development of depression in school-aged children (4–18 years).

Numerous reviews support the effectiveness of preventative interventions in adolescent samples (Calear and Christensen, 2010; Horowitz and Garber, 2006; Merry et al., 2011). Early findings suggested that selective and indicated interventions were more effective than universal programmes in the prevention of child and adolescent depression (Horowitz and Garber, 2006). Some researchers argued that due to the limited evidence supporting the effectiveness of universal
interventions offered in school settings, it would be premature to disseminate these interventions more widely (Spence and Shortt, 2007). Policy makers suggested that further research is required to identify effective universal approaches (McDermott et al., 2010).

However, a Cochrane Review later suggested that universal approaches are effective in reducing depression (Merry et al., 2011). These findings were replicated in a review of school-based randomised controlled trials (RCTs) (Corrieri et al., 2013). Furthermore, a meta-analysis of 32 RCTs, including both adult and adolescent populations, indicated no difference in the effectiveness of interventions based on the type of approach (universal, selective or indicated) implemented (Van Zoonen et al., 2014). Overall evidence suggests that universal approaches offer cost-effective means of reducing the prevalence of depression.

Current preventative interventions have been criticised for being more focussed on treatment and early intervention than prevention, usually only targeting individual-level factors despite calls for the use of multimodal approaches (AACAP, 2007; Corrieri et al., 2013; Horowitz and Garber, 2006; Spence and Shortt, 2007). The majority of current preventative interventions are Cognitive Behavioural Therapy (CBT), Interpersonal Therapy (IPT) or psycho-education based and are offered in school settings (Calear and Christensen, 2010; Merry et al., 2011; Neil and Christensen, 2007; Van Zoonen et al., 2014). Brief interventions that only target individual-level factors such as improving problem solving skills will likely be insufficient to produce lasting effects in reducing the prevalence of depression (Spence and Shortt, 2007).

Furthermore, to achieve population-level reductions in depression, it is necessary to address the underlying determinants of mental health through
coordinated responses across a range of services and settings (Jacka and Reavley, 2014; Jorm, 2014). Population-level approaches are needed to reduce community prevalence rates. Such approaches have proven to be effective in Australia in minimising the impact of other health outcomes such as suicide and smoking (Jorm, 2014). However, there is limited understanding of community-level predictors of adolescent depression that could be targeted through such approaches. Furthermore, there are no known reviews of community interventions targeting risk and protective factors for adolescent depression.

The purpose of this study was to complete a systematic review of community risk and protective factors for depressive symptoms in school-aged children (4–18 years). The study adopted a broad scope examining both observational and intervention study designs in both peer-reviewed and non-reviewed publications and used the procedures recommended by the Cochrane Non-Randomised Studies Methods Working Group (Reeves et al., 2011).

**Method**

**Search strategy and bias.** Due to the nature of health promotion, terminology is often imprecise and studies may be found in a variety of electronic databases (Beahler et al., 2000). As such, a broad range of search terms (see Appendix A) were developed based on current meta-analyses and reviews in the topic area (e.g. Merry et al., 2011; Thomson et al., 2006). Using these search terms, an extensive search was conducted through the EbscoHost MegaFILE Premier Facility across the following databases: Education Research Complete, E-Journals, Humanities International Complete, PsycARTICLES, PsycEXTRA, Psychology and Behavioural Sciences Collection, PsycINFO, Social Work Abstracts and Urban Studies Abstracts. After reviewing articles identified in the
initial search, a more specific list of search terms was generated and five further searches were carried out (see Appendix B-F). Database searches included unpublished theses.

Armstrong et al. (2011) and Beahler et al. (2000) suggest that additional search strategies, other than database searching, should be employed to locate non-peer reviewed studies (grey literature). A time-limited grey literature search was conducted, including a search of a number of government and community websites (see Appendix G). In addition, current meta-analyses and reviews in the areas of child and adolescent depression, health promotion and community interventions were located and reference lists reviewed (e.g. Barrera et al., 2007; Cuijpers et al., 2006; Horowitz and Garber, 2006; Keleher and Armstrong, 2005; Perry et al., 2010; Sellström and Bremberg, 2006; Stice et al., 2009; Suter and Bruns, 2009; Thomson et al., 2006; Twenge and Nolen-Hoeksema, 2002).

Furthermore, the reference lists of included studies were reviewed to identify potential studies. The main search was carried out over a three-month period from July to October, 2012. The extensive search conducted minimised any potential problems of fugitive literature; the issue of failing to include hard-to-locate articles (Rosenthal, 1995).

Eligibility criteria. Studies in languages other than English, unless appropriately translated, were excluded from this analysis. Given that prevention research in this area is relatively new and prior reviews in this area have not identified studies before this time (Fowler et al., 2009; Centre for Allied Health Evidence, 2009), all searches were restricted to studies after 1992. In order to minimise potential bias in study inclusion, the following pre-determined inclusion and exclusion criteria were established.
School age participants. Participants had to be between the ages of 4–18 years at the time the outcome variable was assessed.

Depressive symptom variable. The study had to include a valid measure of childhood depressive symptoms (e.g. self-report of depressive symptoms) or participants had to be diagnosed through clinical interview with a depressive disorder (e.g. Major Depressive Episode). For the purpose of this study, depressive symptoms included: dysthymia, major depressive disorder, mood disorders, depressive symptoms, internalising symptoms or disorders and emotional symptoms or disorders. Studies that did not disaggregate internalising but only provided a combined score of internalising and externalising behaviours (e.g. Child Behaviour Checklist [CBCL] total internalising and externalising problem behaviours) were excluded from the study. Retrospective accounts of depressive symptoms (i.e. adults reporting on their depressive symptoms as a child) were also excluded.

Community-level factors. The study had to examine either community-level risk or protective factors, or an intervention targeting community risk and protective factors. The study had to include a community measure (e.g. Index of Relative Disadvantage) or individual report (e.g. Children’s Report of Exposure to Violence) of a community-level variable or compare depressive symptoms across two distinct communities (e.g. community affected by political violence and a community not affected by political violence). Alternatively, studies examining the effectiveness of a community-level intervention needed to assess an intervention aimed at addressing a community risk or protective factor (e.g. intervention increasing socio-economic status). In this review, the term
community refers to the environment external to the individual, family and school settings.

Studies included in previous meta-analyses. The decision was made to exclude studies that exclusively examined school-level variables or the effectiveness of school-level interventions, given the extensive research conducted in this area (e.g. Kidger et al., 2012; Merry et al., 2011). At the time, the literature search was conducted, the only review/meta-analysis examining community-level factors associated with children’s internalising symptoms was Fowler et al.’s (2009) meta-analysis. Fowler et al. (2009) conducted a comprehensive meta-analysis on the effect of community violence on internalising symptoms. Fowler et al.’s (2009) study included studies up to 2007. As such, the decision was made to search for community violence studies 2007 and after, to identify research that may add to existing findings.

Relevant statistical comparison. The relationship between the predictor variable and the outcome variable had to be tested statistically. Studies that only reported unadjusted prevalence rates (e.g. 10% children from low-socio-economic communities had depression and 11% of children from high socio-economic communities had depression) were excluded from this analysis.

Study quality and bias. The quality of studies was assessed using the validated Newcastle-Ottawa Scale (NOS: Wells et al., 2008) in accordance with the Cochrane Non-Randomised Studies Methods Working Group (Reeves et al., 2011). The NOS evaluates the quality of non-randomised studies, including cohort and case-control studies using a ‘star’ rating system to judge the quality of a study and potential for within study bias. Ratings are applied over three areas: the selection of study groups, the comparability of the groups, and the ascertainment
of either the exposure or outcome of interest for case-control or cohort studies respectively. The maximum numbers of stars a study may receive over these categories are four, two and three respectively to produce a combined maximum score of nine. The raters met prior to assessing studies to discuss any discrepancies in their interpretation of the assessment tool. Question 4 of the NOS scale states that controls with previous occurrences of the outcome of interest should be excluded. Given the participants are children and adolescents, the decision was made to not remove a star if studies did not assess and exclude previous occurrences of depression.

Data extraction, analysis and reporting bias. This study employed systematic strategies to identify and appraise relevant studies to ensure quality and limit potential bias, in accordance with the Preferred Reporting Items for Systematic Reviews and Meta- Analyses (PRISMA) guidelines (Liberati et al., 2009) and the Cochrane Non-Randomised Studies Methods Working Group (Reeves et al., 2011). Each individual study was independently reviewed by a minimum of two authors. There were some minor disagreements between raters, but these were clarified by consensus. After discussions, the inter-rater agreement was 100%.

Given the heterogeneity of study designs, measures and analyses in the included studies, it was not appropriate to pursue traditional methods of using effect sizes to calculate meta-analytic results. Following previous reviews of adolescent mental health that examined similar heterogeneous literature (Ryan et al., 2010; Yap et al., 2014), we adopted Stouffer’s method. Stouffer’s method was used to combine $p$-values and determine whether the synthesised results were significant (Becker, 1994). Stouffer’s $z$ was calculated by dividing the sum of the
COMMUNITY FACTORS AND ADOLESCENT DEPRESSION

\( z(Pi) \) values by the square root of \( k \), where \( k \) is the number of included studies.

Where possible, included studies were grouped into themes and a meta-analysis was completed reporting sample effect size and \( p \)-values. Where no exact \( p \)-value was provided, conservative estimates were adopted. For example, if a study stated an effect was \( p < 0.05 \), it was estimated at \( p = 0.049 \). Effect sizes were conservatively estimated based on the \( p \)-value and analytic sample size.

**Results**

**Study selection and bias.** The literature search (see Figure 1) identified 21 studies that met the inclusion criteria (see Tables 1 and 2). For a full reference list of the studies that were considered for inclusion but ultimately excluded from this study, see Appendix H. Thirty-eight percent of the included studies were retrieved through the grey literature search, thus reducing the likelihood of publication bias in this study (Armstrong et al., 2011). The NOS scale was used to identify potential within study bias (see Tables 3 and 4). Overall, included studies were of high quality and received the following star ratings: 7 stars \((k = 1)\), 8 stars \((k = 15)\) and 9 stars \((k = 2)\).

**Participants.** All studies included in the review used independent data sets. The total number of participants included in this review was 55,655 (range = 100 to 18,473). Studies were conducted in the following countries: Australia \((k = 1)\), Canada \((k = 1)\), Israel \((k = 1)\), Nepal \((k = 1)\), Netherlands \((k = 1)\), Uganda \((k = 1)\), United Kingdom \((k = 2)\) and the United States of America \((k = 13)\). The majority of studies examining community risk and protective factors included samples of participants that were representative of the general population \((k = 9)\).
Figure 1. Flow diagram for article selection

5944 abstracts identified through database searching

4925 abstracts reviewed to determine relevance after

189 full-text articles identified through database search

55 Full-text articles identified through grey literature search

222 full-text articles assessed for eligibility after duplicates

201 Full-text articles excluded, with reasons

21 Studies included in qualitative synthesis (database, k = 13 grey N = 8)

Reasons for Exclusion
- Study sample outside specified age range (k = 40)
- No appropriate community-level measure or intervention (k = 33)
- No appropriate measure of depressive symptoms (k = 89)
- No appropriate statistical comparison (k = 37)
- Reproduced study (k = 2)
Table 1. Findings from observational studies examining community-level risk and protective factors

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<tr>
<th>Study</th>
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<th>Study Design</th>
<th>Outcome Measure</th>
<th>Community-level Factors</th>
<th>Summary of Findings</th>
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<td>Al-Krenawi and Graham, 2012</td>
<td>Israel</td>
<td>MRA</td>
<td>C: BSI</td>
<td>Exposition to political violence, Nationality</td>
<td>Adjusting for individual and family variables, political violence (β = -0.25, p &lt; .001) and nationality (β = -0.07, p &lt; .05) were associated with depressive symptoms.</td>
</tr>
<tr>
<td>Bayer et al., 2011</td>
<td>Australia</td>
<td>MRA</td>
<td>P: SDQ</td>
<td>Neighbourhood disadvantage, Time in centre-based care</td>
<td>In unadjusted analyses, neighbourhood disadvantage (p &lt; .001) &amp; centre-based care (p = .02) were associated with internalising problems. Adjusting for individual, family, and community variables, neighbourhood disadvantage (β = 0.18, CI = 0.14 to 0.28) &amp; centre-based care (β = 0.01, CI = -0.18 to 0.19, p = .95) were not associated with internalising problems.</td>
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<td>Behnke et al., 2010</td>
<td>USA</td>
<td>SEM</td>
<td>C: CES-D</td>
<td>Neighbourhood risk (disadvantage), Perceived discrimination</td>
<td>Neighbourhood disadvantage had a significant direct effect (r = 0.15, p &lt; .05) and an indirect, via self-esteem, effect (r = 0.10, p &lt; .05) on boys’ depressive symptoms. Neighbourhood disadvantage had a total effect on boys’ (r = 0.25, p &lt; .05) and girls (r = 0.14, p &lt; .05) depressive symptoms. Perceived discrimination had a direct and total effect on boys’ (r = 0.10; r = 0.10, p &lt; .05) and girls’ (r = 0.20; r = 0.24, p &lt; .05) depressive symptoms.</td>
</tr>
<tr>
<td>Boyle and Lipman, 2002</td>
<td>Canada</td>
<td>MLM</td>
<td>P &amp; T: CBCL</td>
<td>Neighbourhood disadvantage, % of lone parents, Small rural vs larger urban communities</td>
<td>P: Living in a small rural community (β = 0.25, p &lt; .05) and percentage of lone parents (β = 0.16, p &lt; .05) were associated with internalising problems. Quadratic association between Neighbourhood disadvantage (β = -0.11, p &lt; .05) and internalising problems. T: Not significant.</td>
</tr>
<tr>
<td>Study</td>
<td>Sample</td>
<td>Study Design</td>
<td>Outcome Measure</td>
<td>Community-level Factors</td>
<td>Summary of Findings</td>
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<tr>
<td>Caughy et al., 2008</td>
<td>USA 5-6yrs N = 405</td>
<td>MLM</td>
<td>P: CBCL</td>
<td>Neighbourhood economic disadvantage Neighbourhood negative social climate Neighbourhood involvement with children</td>
<td>Adjusting for neighbourhood and parenting factors, high negative social climate was associated with high internalising problems ($\beta = 2.70, p &lt; .10$). High neighbourhood involvement with children was also associated with high internalising problems but only in neighbourhoods with high economic disadvantage ($\beta = -0.79, p &lt; .05$).</td>
</tr>
<tr>
<td>Ford et al., 2004</td>
<td>England 5-15yrs N = 6,685</td>
<td>LRA</td>
<td>P, C, &amp; T: DAWBA</td>
<td>Neighbourhood deprivation</td>
<td>Adjusting for a number individual and family factors, and co-morbid disorders neighbourhood deprivation (OR = 0.86, CI = 0.70, 1.07, $p &gt; .05$) was not associated with Depression. Findings impacted by low power as only 79 cases of depression.</td>
</tr>
<tr>
<td>Kennedy et al., 2010</td>
<td>USA 8yrs + (M = 9.9yrs) N = 100</td>
<td>MLM</td>
<td>C: CDI</td>
<td>Community and school violence exposure</td>
<td>Multivariate growth model: exposure to CSVE at start of study predictive of depressive symptoms at end of two years ($\beta = 5.19, p &lt; .001$). Increases in CSVE over time was associated with increased depression scores ($\beta = 2.77, p &lt; .001$).</td>
</tr>
<tr>
<td>Kohrt et al., 2010</td>
<td>Nepal 11-17yrs N = 142</td>
<td>MRA</td>
<td>C: DSRS</td>
<td>Conflict mortality Community proportion of female literacy High caste proportion</td>
<td>Conflict mortality was significantly associated with depressive symptoms ($\beta = 1.82, CI = 0.05, 3.59, p = .04$). Whilst caste background was significant at the family level, high caste proportion at the community-level was not associated with depressive symptoms ($\beta = 1.12; CI = -1.5, 3.73; p = .40$). Female literacy was not significantly associated with depressive symptoms ($\beta = -0.49; CI = -1.92, 0.94; p = .50$).</td>
</tr>
<tr>
<td>Lambert et al., 2010</td>
<td>USA 10-13yrs N = 500</td>
<td>LTA</td>
<td>C: BHIF</td>
<td>Community violence exposure</td>
<td>Year 6 students in the high exposure class had more depressive symptoms ($\beta = 0.84, p = .03$), controlling for intervention effects and accounting for individual and parenting factors. The effect of community violence was not significant for year 7 or 8 students.</td>
</tr>
<tr>
<td>Study</td>
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<td>Summary of Findings</td>
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<tr>
<td>Lindsey et al., 2008</td>
<td>USA 8yrs N = 298</td>
<td>SEM</td>
<td>P: CBCL</td>
<td>Neighbourhood satisfaction</td>
<td>Controlling for gender and caregiver support neighbourhood satisfaction was significantly associated with child’s internalising problems ($\beta = -0.24, p &lt; .0005$). Controlling for gender, caregiver support, parent mental health, and parent alcohol use, neighbourhood satisfaction was not associated with children’s internalising problems ($\beta = -0.08, p = .163$).</td>
</tr>
<tr>
<td>Meltzer et al., 2007</td>
<td>England, Wales &amp; Scotland 11-16yrs N = 3340</td>
<td>LRA</td>
<td>C &amp; P: DAWBA</td>
<td>Neighbourhood trustworthiness and safety Neighbourhood socio-economic status</td>
<td>Adjusting for individual factors, family factors, community factors, and other disorders, neighbourhood trustworthiness and safety was associated ($p &lt; .05$) with emotional disorders.</td>
</tr>
<tr>
<td>Rosario et al., 2008</td>
<td>USA 11-14yrs N = 667</td>
<td>MRA</td>
<td>C: BDI</td>
<td>Witnessed community violence Victim of community violence</td>
<td>Adjusting for prior witnessing of community violence, individual factors, peer factors, and family factors, recently witnessed community violence was significantly associated with higher depressive symptoms at T3 for boys ($\beta = 0.18, p &lt; .01$) and girls ($\beta = 0.25 p &lt; .001$). Being a victim of community violence was associated with boys ($\beta = 0.20 p &lt; .01$) and girls ($\beta = 0.20 p &lt; .001$) depressive symptoms at T2 and girls ($\beta = 0.15, p &lt; .001$) depressive symptoms at T3.</td>
</tr>
<tr>
<td>Schneiders et al., 2003</td>
<td>Netherlands 12-14yrs N = CBCL: 2,496; YSR: 232</td>
<td>MLM</td>
<td>P: CBCL &amp; C: YSR</td>
<td>Neighbourhood disadvantage</td>
<td>Adjusting for individual and family factors, neighbourhood disadvantage was associated with CBCL ($\beta = 0.39, p &lt; .01$) &amp; YSR ($\beta = 0.58, p &lt; .01$) internalising problems in analyses combining T1 and T2 responses.</td>
</tr>
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</table>
## Community Factors and Adolescent Depression

<table>
<thead>
<tr>
<th>Study</th>
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<th>Community-level Factors</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Simons et al., 2002</td>
<td>USA 10-12yrs N = 810</td>
<td>MLM RR = 93%</td>
<td>C: DISC</td>
<td>Community poverty, Community cohesion, Community ethnic identification, Prevalence of discrimination, Incidence of violent community crime</td>
<td>In unadjusted analyses, all community-level variables were associated (p &lt; .05) with depressive symptoms. MLM: Controlling for individual and family factors: Community-level variables, ethnic identification (β = -0.39, p = .042) and prevalence of discrimination (β = 0.31, p = .039) were associated with depressive symptoms. Community poverty (β = -0.04, p = .563), community cohesion (β = -0.08, p = .660), and community crime (β = 0.13, p = .248) were non-significant. Community crime was also associated with depressive symptoms but only in high poverty (β = -0.24, p = .022) &amp; low ethnic identification (β = -0.83, p = .025) communities.</td>
</tr>
<tr>
<td>Wight et al., 2005</td>
<td>USA 11-18yrs N = 18,473</td>
<td>MLM RR = 79%</td>
<td>C: CES-D</td>
<td>Ethnic minority status, Median household income of the community</td>
<td>Adjusting for individual and family factors, ethnic minority status (β = 0.73, p &lt; .01) and low median household income (β = 0.42, p &lt; .05) were associated with higher depressive symptoms.</td>
</tr>
<tr>
<td>Witherspoon et al., 2009</td>
<td>USA 10-12yrs N = 437</td>
<td>MLM RR = 72%</td>
<td>C: SAS</td>
<td>Connectedness, Neighbourhood fear, Residential mobility, Neighbourhood problems</td>
<td>Profiles characterized by at least one or more connections to neighbourhood contexts were (B = -0.19, p &lt; .001) associated with fewer depressive symptoms. Adjusting for individual, family, and school factors, neighbourhood fear (B = 0.07, p &lt; .001) was associated with higher symptoms. Perceived neighbourhood problems (B = -0.01) and community mobility (B = -0.02) were not significant (p &gt; .10).</td>
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</table>
## Community Factors and Adolescent Depression

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</tr>
</thead>
<tbody>
<tr>
<td>Xue et al., 2005</td>
<td>USA 5-11yrs</td>
<td>MLM RR = 78%</td>
<td>P: CBCL</td>
<td>Concentrated disadvantage; Immigrant concentration; Residential stability; Collective efficacy; Organisational participation</td>
<td>MLM-G: controlling for baseline depression and accounting for child and family factors, concentrated disadvantage was associated with clinical cut off CBCL scores ($B = 0.19, p &lt; .05$). When social processes were added collective efficacy was ($B = -0.07, p &lt; .05$) associated with higher CBCL. Concentrated disadvantage was no longer significant. In the final model, organisational participation was associated with clinical cut off CBCL scores ($B = -0.19, p &lt; .05$) and higher CBCL ($B = -0.06, p &lt; .05$). Collective efficacy was no longer significant.</td>
</tr>
<tr>
<td>Zinzow et al., 2009</td>
<td>USA 12-17yrs</td>
<td>MVA RR = 54%</td>
<td>C: Depression Module of NSA survey</td>
<td>Witnessed community violence</td>
<td>Adjusting for individual and family factors, adolescents who witnessed sexual assault (OR = 2.11, $p &lt; .05$); mugging (OR = 1.69, $p &lt; .05$); threat with a weapon (OR = 1.66, $p &lt; .05$); and beating (OR = 2.01, $p &lt; .001$) were more likely to have Depression. Adolescents who witnessed multiple incidents of violence (OR = 2.18, $p &lt; .001$) or incidents where they were afraid they might be injured or killed (OR = 1.90, $p &lt; .05$) were more likely to have Depression.</td>
</tr>
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</table>

$\beta$ = standardised coefficient; $B$ = unstandardised coefficient; BDI = Beck Depression Inventory (Beck, 1988); BHIF = Baltimore How I Feel (Ialongo et al., 1999); BPI = Behavior Problems Index (Zill, 1983); BSI = Brief Symptom Inventory (Derogatis, 1983); C = Children’s reports; CAPA = Child and Adolescent Psychiatric Assessment (Angold et al., 1995); CBCL = Child Behaviour Checklist (Achenbach, 1991); CDI = Child Depression Inventory (Kovacs, 1984); CES-D = Center for Epidemiologic Studies Depression Scale (Radloff, 1977); CI = Confidence Interval; DAWBA = Development and Well-being Assessment (Goodman and Ford, 2000); DSRS = Depression Self Rating Scale (Birleson, 1987); DISC = Diagnostic Interview Schedule for Children (American Psychiatric Association, 1994); M = Mean; MLM = Multi Level Model; MLM-G = Multi Level Growth Model; MVA = Multivariate Analyses; N = sample size; NSA = National Survey of Adolescents; OR = Odds Ratio; P = Parents’ reports; $p$ = p-value (significance); R = Retention; RR = Response Rate; SAS = Social Adjustment Scale (Birmaher, 1990); SDQ = Strengths and Difficulties Questionnaire (Goodman 1997); SEM = Structural Equation Modelling; T = Teachers reports; USA = United States of America; YSR = Youth Self-Report (Achenbach, 1991)
### Table 2. Findings from studies assessing the effectiveness of community interventions

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample</th>
<th>Study Design</th>
<th>Outcome Variable</th>
<th>Intervention</th>
<th>Summary of Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Costello et al., 2003</td>
<td>USA 9-13yrs N = 1,420</td>
<td>GEM, R = 83% Longitudinal, Quasi experiment, included a program logic analysis.</td>
<td>C. &amp; P. CAPA</td>
<td>Tribal members received a percentage of profits from a gambling casino every 6 months. Children’s earnings were paid into a trust fund until 18 years. Opening of the casino also provided increased employment opportunities to community members.</td>
<td>American Indian children whose families moved out of poverty after the casino opened showed a significant decrease in emotional symptoms (OR = 1.43; 95% CI = 1.02, 2.03; p = .04). However, in comparing ex-poor families to consistently poor and never poor families no significant differences were found.</td>
</tr>
<tr>
<td>Fauth et al., 2007</td>
<td>USA 8-18yrs N = 221 (In = 128, Co = 93)</td>
<td>MRA, Longitudinal, Quasi experiment, included a program logic analysis</td>
<td>C. BPI</td>
<td>Yonkers Family and Community Project was a court-ordered neighbourhood desegregation project in 1985. Low income black and Latino families residing in low-socio economic communities were randomly selected to move to publicly funded townhouses in middle-class communities.</td>
<td>Movers reported significantly more anxiety/depression symptoms (B = 0.27, SE = 0.16, p &gt; .10). Neighbourhood social climate partly mediated the effect of moving on anxiety/depression symptoms (B = -0.17, SE = 0.07, p &gt; .05). The lack of infrequent interactions with neighbours during the first several years of relocation was associated with an increase in anxious/depressive symptoms for children who moved.</td>
</tr>
<tr>
<td>Ssewamala et al., 2012</td>
<td>Uganda M = 13.71yrs N = 262 (Co = 148, In = 138)</td>
<td>MLM, RCT, baseline differences, no program logic analysis</td>
<td>C. CDI</td>
<td>Participants were children who had lost at least one parent to AIDS. All participants received treatment as usual including counselling and educational supplies. Participants in the treatment group also received the microfinance intervention. This included (a) matched savings accounts, the program provided $2 for every $1 contributed by the adolescent or their family; (b) financial management and small business workshops to generate sources of income for families; and (c) mentorship program for adolescents.</td>
<td>In: significant reduction in depressive symptoms over time (B = -0.34; 95% CI = -0.61,-0.06; t(256) = -2.41; p = .02). Co: Not significant. However, the two groups were not significantly different (B = -2.1, t(256) = -2.18, p = .38; 95% CI = -.67, -.26).</td>
</tr>
</tbody>
</table>

**Legend:** P = standardised coefficient; B = unstandardised coefficient; BPI = Behaviour Problems Index (Zill, 1985); C = Children’s reports; CAPA = Child and Adolescent Psychiatric Assessment (Angold et al., 1995); CDI = Child Depression Inventory (Kovacs, 1984); CI = Confidence Interval; Co = Control group; In = Intervention group; GEE = Generalised Estimated Equations; M = Mean; MLM = Multi Level Model; MRA = Multiple Regression Analyses; N = sample size; OR = Odds Ratio; P = Parents’ reports; p = p-value (significance); R = Retention; USA = United States of America.
Table 3. Cohort studies assessment of study quality

<table>
<thead>
<tr>
<th>Longitudinal Observational Studies</th>
<th>Newcastle-Ottawa Scale Cohort</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Selection</td>
</tr>
<tr>
<td>Kennedy et al. (2010)</td>
<td>3</td>
</tr>
<tr>
<td>Lambert (2010)</td>
<td>3</td>
</tr>
<tr>
<td>Rosario et al. (2008)</td>
<td>3</td>
</tr>
<tr>
<td>Schneiders et al. (2003)</td>
<td>4</td>
</tr>
<tr>
<td>Xue et al. (2005)</td>
<td>4</td>
</tr>
</tbody>
</table>

Table 4. Cross-sectional studies assessment of study quality

<table>
<thead>
<tr>
<th>Cross-sectional Observational Studies</th>
<th>Newcastle-Ottawa Scale Case Control</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Selection</td>
</tr>
<tr>
<td>Al-Krenawi and Graham (2012)</td>
<td>3</td>
</tr>
<tr>
<td>Bayer et al. (2011)</td>
<td>3</td>
</tr>
<tr>
<td>Behnke et al. (2010)</td>
<td>3</td>
</tr>
<tr>
<td>Boyle and Lipman (2002)</td>
<td>3</td>
</tr>
<tr>
<td>Caughy et al. (2008)</td>
<td>3</td>
</tr>
<tr>
<td>Ford et al. (2004)</td>
<td>3</td>
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<tr>
<td>Kohrt et al. (2010)</td>
<td>3</td>
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<tr>
<td>Lindsey et al. (2008)</td>
<td>3</td>
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<tr>
<td>Meltzer et al. (2007)</td>
<td>3</td>
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<tr>
<td>Simons et al. (2002)</td>
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<tr>
<td>Wight et al. (2005)</td>
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<tr>
<td>Witherspoon et al. (2009)</td>
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<tr>
<td>Zinzow et al. (2009)</td>
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</table>

Observational studies that examined sub-sets of the population \((k = 9)\) included the following: children from politically violent areas in the Gaza Strip and West Bank (Al-Krenawi and Graham, 2012), children of Latin American background (Behnke et al., 2010), children of mothers exposed to partner violence.
(Kennedy et al., 2010), child soldiers of war (Kohrt et al., 2010); children of African American background (Lambert et al., 2010; Simons et al., 2002), children of African American background who are at risk of or have been abused (Lindsey et al., 2008), children from Hispanic and African American backgrounds (Rosario et al., 2008) and children from low-socio-economic backgrounds (Witherspoon et al., 2009). Details of participants in studies examining community interventions are provided in Table 2.

**Depression outcome variables.** The majority of studies used self-report measures. Only four studies used clinical interviews to assess child and adolescent depression. Depressive symptom measures were rated by or based on the reports of children ($k = 12$), parents ($k = 4$), both child and parent ($k = 3$), both parent and teachers ($k = 1$) and parent, child and teachers ($k = 1$).

Studies examined the following outcome variables: depressive symptoms ($k = 10$), depressive and anxiety symptoms ($k = 1$), internalising symptoms ($k = 6$), emotional disorder symptoms ($k = 1$) and depressive and emotional disorders ($k = 3$). Fourteen different measures were used to measure depression; the most common was the CBCL (Achenbach, 1991; $[k = 5]$).

**Community risk and protective factors.** Studies included in the review identified a number of community risk and protective factors that were grouped into four categories. The sections that follow consider the three risk factors (community disadvantage, community safety and community minority ethnicity and discrimination) and one protective factor (community connectedness). In this review, community disadvantage refers to communities of lower socio-economic status. The majority of studies assessing the impact of disadvantage used measures that assessed multiple domains (e.g. rate of unemployment, mean
household income). Community safety refers to safety within the community. The majority of studies assessed an individual’s perception of community safety on domains such as witnessing violence, whether individuals felt safe in their neighbourhood and perceptions of crime. Community minority ethnicity and discrimination included studies that examined communities with high levels of individuals from culturally and linguistically diverse backgrounds and communities with high levels of perceived discrimination. Community connectedness refers to an individual’s perceived connection to their community and includes measures of social cohesion, community involvement and attachment to community.

Community disadvantage. Nine observational studies (Bayer et al., 2011; Behnke et al., 2010; Boyle and Lipman, 2002; Caughy et al., 2008; Ford et al., 2004; Schneiders et al., 2003; Simons et al., 2002; Wight et al., 2005; Xue et al., 2005) included a community measure of disadvantage. Using the Stouffer method, the combined effect on child depressive symptoms across all studies was not significant (N = 46,171, p = 0.073, Effect Size [ES] = 0.025). The differences and distinctions of the individual studies are discussed in more detail below.

Four studies reported direct significant effects. Three studies, using representative samples, identified a significant association between community disadvantage and increased levels of depressive symptoms (4–11 years: Boyle and Lipman, 2002; 12–14 years: Schneiders et al., 2003; 11–18 years: Wight et al., 2005). Similarly, a study of Latin American adolescents (14–16 years) identified that neighbourhood disadvantage had a significant total effect on boys and girls (Behnke et al., 2010).
The findings of a number of studies suggested that the effects of community disadvantage may be mediated through other factors. Bayer et al. (2011) identified a significant association between internalising problems and neighbourhood disadvantage in unadjusted analyses; however, in accounting for a large number of risk factors using multivariate regression, no significant relationship was maintained. Some studies suggested that the impact of community disadvantage may be influenced by other community-level variables. Simons et al.’s (2002) study of African American children (10–12 years) suggested that community safety was only significantly associated with increased depressive symptoms in poor communities. Caughy et al. (2008) identified that neighbourhood involvement with children was significantly associated with children’s (5–6 years) internalising problems, but only in disadvantaged communities. Another study, that used multi-level modelling in a representative sample, identified that community disadvantage was significant until community social processes were added, and then community disadvantage was no longer significant (Xue et al., 2005).

Community safety. A total of nine observational studies (Al-Krenawi and Graham, 2012; Caughy et al., 2008; Kennedy et al., 2010; Kohrt et al., 2010; Lambert et al., 2010; Meltzer et al., 2007; Rosario et al., 2008; Witherspoon et al., 2009; Zinzow et al., 2009) included individual reports of community safety. Using the Stouffer method, the combined effect on child depressive symptoms was significant \( (N = 7951, p = 0.027, ES = 0.069) \). The differences and distinctions of the individual studies are discussed in more detail below.

Eight studies identified that low community safety was directly associated with increased depressive symptoms. Some studies suggested that the impact of
community safety on child depressive symptoms may also vary by other community-level variables such as community disadvantage and ethnic identification. In a study of African American children (10–13 years), the majority of whom resided in urban settings with violent crime rates that exceeded national averages, high exposure to community violence was associated with significantly more depressive symptoms (Lambert et al., 2010).

Community minority ethnicity and discrimination. Six observational studies (Al-Krenawi and Graham, 2012; Behnke et al., 2010; Kohrt et al., 2010; Simons et al., 2002; Wight et al., 2005; Xue et al., 2005) utilised both self-report and community measures of minority ethnicity or discrimination. Using the Stouffer method, the combined effect on child depressive symptoms was significant (N = 22,872, p = 0.028, ES = 0.041). The differences and distinctions of the individual studies are discussed in more detail below.

Four identified that high community minority ethnicity and discrimination were directly associated with increased depressive symptoms. The findings of two multivariate analyses suggest that the effects of community minority ethnicity may be mediated through other factors. A representative sample that used multi-level modelling to control for a large number of individual and family variables did not identify a significant association between immigrant concentration and younger children’s (5–11 years) internalising problems (Xue et al., 2005). Similarly, applying multivariate regression models to control for village clusters and accounting for a range of individual and family variables, no significant association was identified between high caste proportion and depressive symptoms in adolescent (11–17 years) soldiers of war (Kohrt et al., 2010).
Community connectedness. Four observational studies (Caughey et al., 2008; Simons et al., 2002; Witherspoon et al., 2009; Xue et al., 2005) used individual reports of community connectedness. Using the Stouffer method, the combined effect on child depressive symptoms was not significant ($N = 3800$, $p = 0.241$, ES = 0.051). The differences and distinctions of the individual studies are discussed in more detail below.

One study, of children (10–12 years) from disadvantaged communities reported that high neighbourhood connectedness was directly associated with significantly fewer depressive symptoms (Witherspoon et al., 2009). The findings of this study and the other three studies suggest that the effects of community connectedness may be mediated through other factors, such as other community-level variables. In a representative sample of children (5–11 years), informal social control and social cohesion were significantly associated with higher internalising symptoms, but after adding organisational participation to the model, social control and cohesion were no longer significant (Xue et al., 2005). A study of African American middle-aged children (10–12 years: Simons et al., 2002) identified significant associations between community connectedness and depressive symptoms in an unadjusted analysis, but relationships were not significant in the adjusted analysis. Caughey et al. (2008) identified that high neighbourhood potential for community involvement with children was significantly associated with children’s (5–6 years) internalising problems, but only in disadvantaged communities.

Other community factors. There is some evidence that children may fare less well in remote and poorly organised communities. A study of Canadian children utilising a representative sample, indicated that children (4–11 years)
from small remote communities had higher levels of internalising symptoms than children from large urban communities (Boyle and Lipman, 2002). In a representative sample of children (5–11 years), residents’ involvement in local organisations was significantly associated with lower CBCL scores (Xue et al., 2005). Six separate studies examining time in centre-based care, residential stability, residential mobility, neighbourhood problems, neighbourhood satisfaction and female literacy did not identify any significant associations with depressive symptoms.

**Community intervention studies addressing disadvantage.** Three intervention studies examined effects of poverty alleviation on child emotional disorder symptoms or depression. One reported significant effects; however, using the Stouffer method, the combined effect on child depressive symptoms was not significant (N = 1903, p = 0.055, ES = 0.127).

However, the interventions were not homogenous in content. One intervention relocated families from disadvantaged communities into middle-class communities, another was a microfinance intervention for children who had lost a parent from AIDS. The third intervention provided financial gains and employment opportunities to parents after a casino opened in a native Indian reserve.

**Discussion**

**Summary of evidence.** This report presents the first systematic review and meta-analyses of studies that have investigated associations between community factors and depressive symptoms in school-aged children (4–18 years). The meta-analyses revealed significant effects in the observational studies for community safety and community minority ethnicity and
discrimination. Effects failed to reach significance for the observational studies of community connectedness, community disadvantage and for the three intervention studies examining effects of alleviating disadvantage. Seventeen of the 18 observation studies and two of the three intervention studies reported one or more significant effects.

Similar to the findings of a meta-analysis assessing the association between exposure to violence outside the home and mental health outcomes (0–25 years: Fowler et al., 2009), this study confirmed a significant association between community safety and depressive symptoms in school-aged children (4–18 years). Fowler et al. (2009) identified that community violence exposure had a moderate significant overall effect on young people’s internalising symptoms (d = 0.45, 95% CI = [0.44, 0.47], k = 82, N = 25,960). The effect size was substantially higher than that of this study (ES = 0.069, p = 0.027, k = 9, N = 7951). There are a number of potential reasons for this. First, Fowler et al. (2009) utilised unadjusted correlations from studies to calculate an overall effect size, whereas this study reported adjusted effect sizes that accounted for a large number of covariates. Second, the Stouffer method adopted in this study likely produced a more conservative estimate of the overall effect size.

Third, Fowler and colleagues’ inclusion criteria differed to that of this review in: including studies with an older age group (participants up to 25 years of age), and also adopting a broader definition of community violence including any violence that occurred outside the home including school violence and shootings.

While minority ethnicity has been identified as an individual-level risk factor in policy documents (McDermott et al., 2010), to date, there has been no systematic review on the impact of minority ethnicity and discrimination at the
community-level. This study identified a significant association between community minority ethnicity and discrimination and depressive symptoms in school-aged children.

A growing body of research supports the association between community disadvantage and a range of developmental, physical and mental health outcomes for children and adolescents (Brooks-Gunn and Duncan, 1997). While meta-analytic results did not achieve significance, given the conservative estimates adopted using Stouffer’s method, these results should be interpreted with caution. Effect sizes were estimated at 0.025 for the observational studies and at 0.127 for the intervention studies suggesting that findings would likely be significant in larger samples. Findings from a number of the included studies suggested that community disadvantage may influence the role of other community-level factors (e.g. community connectedness and community minority ethnicity and discrimination).

There has also been limited prior research systematically reviewing the association between community connectedness and depressive symptoms in school-aged children. Meta-analytic findings in this study did not reach significance. However, as there were only four studies included in the analysis, caution is advised in interpreting these results. The effect size was estimated at 0.049, suggesting effects would be significant with a larger sample size. Furthermore, some of the included studies indicated that the impact of community connectedness on depressive symptoms may be mediated by other factors such as community disadvantage. Indeed, one community intervention indicated that children relocated from disadvantaged communities into middle-class communities who had little social interaction with their neighbours experienced
greater levels of depressive symptoms (Fauth et al., 2007). Such findings highlight that while it is evident that community-level variables act as risk and protective factors for depression in school-aged children, greater understanding of the process of how community-level factors impact depressive symptoms is required.

**Limitations.** A potential limitation of this review was that only one of the included studies was an RCT (Ssewamala et al., 2012). A second limitation of the current evidence base was the heterogeneous range of community-level outcomes and measures, depressive symptom outcomes and measures and analysis used across studies. This heterogeneity led to difficulties comparing findings and limits the conclusions that can be drawn, particularly in terms of generalisability. The exclusion of non-validated measures of community-level and depression outcomes also limited this review, as 61% of studies were excluded for these reasons. This particularly restricted the number of intervention studies included in the current analysis. A third limitation of this review was the limited range of potential community influences that have been investigated in the included studies. A fourth limitation is that relatively few studies have been well integrated with literature and theories relevant to the development of child and adolescent depression. A fifth limitation of this study was the use of Stouffer’s method to determine effect sizes. In this study, conservative estimates were adopted for studies that did not provide exact significance values, thus producing the lower bounds of effect sizes because of this. It is likely that some non-significant findings would be significant in larger samples. A final limitation of this study was time and resource constraints. Given that 38% of the included studies were retrieved through the grey literature search, the availability of further search time may have revealed additional studies.
Future research. While stakeholders identify the areas of prevention, children and adolescents and affective disorders as priority areas for research, there is little funding supporting preventative research for children and adolescents (Christensen et al., 2013). Considering the increasing prevalence, high costs and poor outcomes associated with childhood depression, further observational studies are recommended to address the limitations of the current evidence base. Given the difficulties and ethical constraints associated with randomising individuals to communities and communities to interventions, population and longitudinal studies offer the next highest level of evidence to evaluate the effect of community-level variables on childhood depressive symptoms. Future research should give careful consideration to study design to minimise the heterogeneous nature of research in this area, and support the development of a robust evidence base.

The use of psychometrically validated mental health and community-level measures is advised. The use of sub-clinical outcomes in epidemiology research is recommended due to difficulties with power when using clinical diagnoses (Ford et al., 2004). Furthermore, the significant negative outcomes associated with sub-clinical symptoms (González-Tejera et al., 2005; Lewinsohn et al., 2004) and the importance of early intervention calls for further research examining sub-clinical presentations. Depressive symptoms in children and adolescents are frequently comorbid with anxiety symptoms (AACAP, 2007; Angold et al., 1999) and indeed in school-aged children, anxiety is frequently a precursor to depression (McDermott et al., 2010). Given the similarities in presentation and treatment of internalising symptoms in school-aged children, the use of measures that assess overall internalising symptoms (e.g. CBCL and Strengths and Difficulties
 COMMUNITY FACTORS AND ADOLESCENT DEPRESSION

Questionnaire (SDQ) is suggested for future research. Future research should also utilise widely used psychometrically valid measures to assess community-level variables.

Studies included in this review also used a wide variety of analytic methods. Not all studies controlled for individual, family and other community-level variables. It is important that analyses account for variation between communities and adjust for multiple risk and protective factors in the one analysis to identify true effects. It is suggested that future research utilise appropriate statistical analyses relevant to examining variables at the community-level such as multi-level modelling.

This study considered a limited number of community factors that may influence child and adolescent depression. For example, there were no studies examining effects of community opportunities for physical activity or for improved nutrition. Community variables to consider in future research identified through the literature search include: availability of local services; community-levels of child abuse and neglect, community attitudes to child discipline, built environment characteristics, the impact of natural disasters, and availability of open space and opportunities for physical activity. Again, based on excluded studies, community interventions that could be evaluated in future research include: neighbourhood investment and regeneration programmes, coordination between child welfare and mental health services, policy changes such as the 4-year-old kindergarten universal access programme and early developmental interventions that may subsequently impact depression in school-aged children such as facilitated playgroups, early attachment initiatives and infant mental health programmes in at-risk populations such as disadvantaged communities.
Relatively few studies in this review were well integrated with literature and theories relevant to the development of child and adolescent depression. As one example, efforts to reduce the impact of economic disadvantage on child depressive symptoms may benefit by being better integrated with developmental theories. Developmental theory and some evidence in the present review would imply that community disadvantage and safety may act indirectly, for example, by disrupting mother–child bonding. Thus, relevant child mental health promotion efforts may be better directed at protecting mother–child bonding in disadvantaged families (Letcher et al., 2009).

Indeed, the findings of the current meta-analysis indicate that community variables act as risk and protective factors for depression in school-aged children. Future research would benefit from understanding the process by which this occurs. For example, one of the studies in this review suggested that small remote communities may act as a risk factor for depression in school-aged children. Given the geographic spread of communities in countries such as Australia, Canada, the United States and China, further research in this area is relevant. It may be useful to compare small remote indigenous and non-indigenous communities as well as examine how access to services may impact depressive symptoms in rural areas. While not as relevant to developed nations, international research could assess the impact female literacy, an important indicator of community gender equality, has on depressive symptoms. In examining the impact of community-levels of female literacy, it may be important to assess how this impacts self-esteem and in turn depressive symptoms.

**Clinical and policy implications.** Given the high prevalence and significant long-term impacts and costs associated with child and adolescent
depression, there has been a significant shift in recent years towards prevention and early intervention by researchers, governments, mental health services and policy makers. The lack of systematic research on community-level factors influencing child and adolescent depression has been noted in policy documents (McDermott et al., 2010). This study provides a significant contribution to the field of child and adolescent depression by identifying and quantifying a number of potentially modifiable community risk and protective factors.

Identified community-level risk and protective factors for depressive symptoms in school-aged children can be targeted in health promotion through: (a) universal programmes preventing depression by targeting community-level variables associated with depression across the whole population, (b) early intervention by identifying at-risk individuals and sub-populations for targeted interventions and (c) treatment by considering identified risk and protective factors in individual assessment and treatment planning.

A determinants approach argues that addressing the underlying community influences for mental health (e.g. community safety, discrimination) is likely to have a greater and more sustainable impact across large populations. Addressing many of the community-level risk and protective factors for depression will likely impact positively on other child and adolescent outcomes and indeed on a range of outcomes for other community members. For example, as risk factors such as community safety and community minority ethnicity and discrimination act on large population aggregates, they not only impact child depression, but also other costly outcomes such as injury and poor physical health.

Findings from this study echo support for the demands for a national mental health prevention strategy (Jorm, 2014). To achieve population-level
reductions in depression, it is necessary to address the underlying determinants of mental health (e.g. community safety, community minority ethnicity and discrimination) through coordinated responses across a range of services and settings (Jacka and Reavley, 2014; Jorm, 2014). The findings of this study indicate that risk factors for child and adolescent depression operate outside the mental health sector (Jacka and Reavley, 2014). Comprehensive government policies are required to support action across sectors and disciplines in order to address these determinants and reduce the prevalence of depression (Jorm, 2014).

**Conclusion.** In conclusion, the current systematic review reveals significant associations between community-level risk and protective factors and child and adolescent mental health. It is argued that with the increasing rise in prevalence and associated cost of mental health problems, community health promotion and early intervention are necessary to minimise the impact of depressive symptoms. As such, further observational studies are required to investigate how community-level risk and protective factors influence depressive symptoms and how effective community interventions can be designed.
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Yap MBH, Pilkington PD, Ryan SM, et al. (2014) Parental factors associated with depression and anxiety in young people: A systematic review and meta-
analysis. *Journal of Affective Disorders* 156: 8-23.


CHAPTER 4: STUDY 2

Context and Rationale

Findings from the systematic review/meta-analyses identified that community-level factors are directly and indirectly associated with child depression. However, none of the included studies examined adolescent depressive symptoms at sub-clinical levels despite a growing body of evidence supporting the association between elevated depression symptoms during adolescence and a range of negative outcomes including later depression. Further, only one study used an Australian sample. Australian guidelines largely rely on international research and suggest that there is currently not enough evidence to support population-level approaches (McDermott et al., 2010). To advance prevention efforts in Australia, research examining depressive symptoms at sub-clinical and/or clinical levels using Australian samples is needed.

An important first step in identifying the extent of the problem is quantifying the prevalence of elevated depression symptoms (sub-clinical and clinical) during early adolescence when psychopathology first emerges. Research suggests that the early adolescence (10-14 years) offers a critical period for preventative intervention. Findings from Study 1 suggested that the prevalence of adolescent depression may vary between communities. Identifying locality differences in the prevalence of rates of depressive symptoms in Australian samples is important in establishing the feasibility and need for place-based interventions.

Research suggests that variation in prevalence rates between communities may be attributed to differences in community-level disadvantage (Schneiders et al., 2003; Wight et al., 2005; Xue et al., 2005). Study 1 indicated mix findings
regarding the impact of community disadvantage on depression symptoms. To assess the impact of community disadvantage on elevated depression symptoms in Australian adolescents it is important to identify if community-level disadvantage is significantly associated with depression after controlling for family level disadvantage (household composition and parent employment; two commonly applied indicators) and a range of demographic variables.

Few of the studies included in the systematic review/meta-analyses quantified the association between depression and community-level factors by gender. Gender differences in the prevalence rates of clinical depression have been identified in Australian adolescents (Boyd et al., 2000; Bond et al., 2005). As such it is important to examine gender differences in prevalence rates. Further, despite the limited reporting of gender-specific statistics, emerging evidence suggests gender differences in risk and protective factors for adolescent depression (Cairns et al., 2014; Yap et al., 2014). As such gender interactions effects need to be examined in univariate and multivariate analysis.

Study 2 is presented in the next chapter and aims to quantify the prevalence of elevated adolescent depression symptoms and to identify the variation between SLAs using a representative sample of Australian adolescents (10-14 years). Study 2 also aimed to identify whether community-level disadvantage was associated with depression symptoms in Australian adolescents after controlling for a number of covariates including family-level disadvantage.
**Authorship Statement for Article 2**

**Submitted to:**  *Australian Psychologist*

**Reference Style:**  APA 6th

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<td>Investigation of Community Factors Associated with Child and Adolescent Depression</td>
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*I declare that the above is an accurate description of my contribution to this paper,*

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<tr>
<td>Katie Stirling</td>
<td>Conception, writing and compilation of manuscript, established methodology, data analyses, preparation of tables and figure.</td>
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<tr>
<td>Dr Bosco Rowland</td>
<td>Supervised and assisted with manuscript compilation, editing of manuscript, and provided feedback on the methodology.</td>
</tr>
<tr>
<td>Professor John Toumbourou</td>
<td>Supervised and assisted with manuscript compilation, and provided the data set used for analysis</td>
</tr>
<tr>
<td>Associate Professor Joanne Williams</td>
<td>Designed and managed the study and data collection. Assisted with manuscript finalisation.</td>
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Community Variation in Depression Symptoms amongst Australian Adolescents

Abstract

Objective: Elevated depressive symptoms during early adolescence predict the subsequent development of psychopathology and health and social problems, while being potentially preventable through community-based interventions. The present study aimed to estimate the prevalence of adolescents experiencing elevated depressive symptoms in Australia and the extent of variation due to community characteristics such as disadvantage.

Method: The prevalence of depressive symptoms was estimated within a representative Australian sample (N = 7,988) of grade 6 and 8 students selected from 30 communities across 3 states (Victoria, Queensland, Western Australia). The study was designed to represent the range of socio-economic disadvantage and urban/ regional variation in Australia. A cut-off of 8+ on the Short Moods and Feelings Questionnaire was used to identify elevated adolescent depressive symptoms.

Results: Of Australian adolescents, 33.1% (95% CI: 32.1-34.2) reported elevated (sub-clinical and clinical) depressive symptoms at average age 12, with significant community variation. Across the 30 Australian communities prevalence estimates varied from 28.0% to 39.5%, with this variation only partially explained by community disadvantage (socioeconomic status). Multi-level modelling indicated community variation in depressive symptoms was associated with: household composition; parents’ employment; community disadvantage; and state location. Gender interaction effects were identified for school grade and father’s employment.
Conclusions: The findings provide a baseline that can be used to evaluate Australia’s future progress in preventing and reducing adolescent depressive symptoms. Given that depression symptoms varied across communities, place-based community interventions should be included in national strategies to prevent the development of adolescent depressive symptoms in Australia.

KEY POINTS

What is already known about this topic?

1. Adolescents with elevated depressive symptoms are known to be at risk of future depressive disorders and related health and social problems.
2. As there is limited data on the Australian national prevalence of elevated (sub-clinical and clinical) depressive symptoms in adolescence it is difficult to set national targets to prevent and reduce these problems.
3. Place-based community strategies are increasingly being used to improve child and adolescent mental health outcomes, however there is little understanding of whether adolescent depressive symptoms vary between communities and what factors may contribute to these differences.

What this topic adds?

1. In the current study, 33.1% of Australian adolescents (10-14 years) were identified as experiencing depressive symptoms sufficiently elevated to pose risks for future adjustment. The findings provide a baseline that can be used to evaluate Australia’s future progress in preventing and reducing adolescent depressive symptoms.
2. Rates of elevated depressive symptoms varied significantly between communities and this variation was not completely explained by differences in community-level disadvantage.
3. The findings support the view that Australian national strategies to prevent and reduce adolescent depressive symptoms should include support for place-based community interventions that target malleable risk and protective factors.
Introduction

Recent papers have noted that despite increasing investment in treatment of mental health disorders, the prevalence of common anxiety and depressive disorders has been either unchanged or rising in Australia in recent decades (Jorm, 2014). These findings have led to calls to increase the strategic investigation of options to prevent the development of common mental disorders such as anxiety and depression by targeting risk and protective factors that emerge during childhood and adolescence (Jacka et al., 2013; McDermott et al., 2010). Increasingly prevention and early intervention attempts have utilised place-based (community-level) approaches to improve child and adolescent well-being (Moore, 2014; Moore & Fry, 2011; Rushton, 2014). The present paper sought to quantify depressive symptoms (sub-clinical and clinical) in a representative sample of Australian adolescents that can be used to assess population level changes in prevention approaches. Additionally, the present paper sought to investigate an important assumption that underpins place-based prevention and early intervention strategies, namely whether there is variation in depressive symptoms across Australian communities. The transition from childhood to adolescence is a high-risk period for the onset of common mental disorders (Kessler et al., 2005; Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Second only to early childhood, it is a crucial phase for psychological, social, and biological development in areas that underlie mental health (Viner et al., 2012). The majority of incident mental disorders arise during early or late adolescence; approximately 74% before the age of 24, and 50% before the age of 14 (Kessler et al., 2005). Further, retrospective studies of depressed adults and prospective studies of adolescents suggest that depression likely emerges during
the early-adolescent (13-15 years) period (Hankin et al., 1998; Lewinsohn & Essau, 2002). Given its prevalence and long-term consequences, depression has been identified as a disorder that should be prioritised for prevention during the early adolescent period (American Academy of Child and Adolescent Psychiatry, 2007; World Health Organisation, 2003).

**Impacts of sub-clinical depression**

Identifying the extent to which adolescents experience depressive symptoms may have benefits for preventive intervention (McDermott et al., 2010). Depressive symptoms in adolescents range from sub-syndromal or sub-clinical (not achieving the criteria for a diagnosable depressive disorder) to syndromal or clinical levels (meeting the criteria for a depressive disorder; American Academy of Child and Adolescent Psychiatry, 2007). Similar to adolescents with clinical depression, adolescents with sub-clinical depression experience significant psychosocial impairment (González-Tejera et al., 2005); increased suicide risk (Fergusson, Horwood, Ridder, & Beautrais, 2005); and increased likelihood of experiencing other sub-clinical and clinical mental health conditions (Lewinsohn, Shankman, Gau, & Klein, 2004). Sub-clinical depression in childhood (Mazza et al., 2009) and adolescence (Lewinsohn & Roberts, 1994; McKenzie et al., 2011) are stronger indicators of later depression relative to other risk factors. Sub-clinical depression also predicts a higher risk of lifetime Major Depressive Disorder (Klein et al., 2013).

The cumulative adversity of experiencing depressive symptoms during adolescence contributes to severe psychopathology in adulthood (Kessler et al., 1995). Furthermore, longitudinal studies suggest that once an adolescent has experienced a depressive episode there is a high probability of recurrence
Birmaher, Arbelaez, & Brent, 2002). A major premise guiding the analyses in the present paper is that preventing elevated depression symptoms (sub-clinical and clinical) in adolescents can have direct benefits (Fergusson et al., 2005; González-Tejera et al., 2005; Lewinsohn et al., 2004) and in addition can also contribute to preventing the incidence and negative trajectory of depressive disorders (Lewinsohn & Roberts, 1994; McKenzie et al., 2011). Thus, from a prevention perspective, identifying the national prevalence of sub-clinical and clinical depressive symptoms during the early adolescent period (12-14 years) when these symptoms first emerge is important in establishing national targets to reduce and prevent these problems.

Assessing depressive symptoms

Australia’s treatment system focuses on a diagnostic approach to identifying depression in participants and this approach predominates particularly in studies examining the effectiveness of treatment interventions (e.g., Ford, Goodman, & Meltzer, 2004; Meltzer, Vostanis, Goodman, & Ford, 2007). However, child and adolescent self-reports of depressive symptoms have been commonly used in epidemiology studies, particularly in those assessing population-wide prevalence rates and the effects of community interventions (e.g., Buttigieg et al., 2015; Bond, Toumbourou, Thomas, Catalano, & Patton, 2005; Boyd, Gullone, Kostanski, Ollendick, & Shek, 2000). Population wide-prevalence rates that include both sub-clinical and clinical depression are needed to assess the effectiveness of population-level preventative attempts that aim to reduce risk and protective factors that influence both sub-clinical and clinical presentations.
It is possible to identify early adolescent internalising symptoms and other risk factors that predict the onset of adolescent depression from parent and child observations (Toumbourou, Williams, Letcher, Sanson, & Smart, 2011) or from the observations of teachers and health professionals (Letcher, Smart, Sanson, & Toumbourou, 2009). In order to fully utilise the prognostic value of adolescent observations it is important to identify early indicators that forecast the development of depression.

The Short Moods and Feelings Questionnaire (SMFQ) was developed to provide a psychometrically sound instrument for assessing child and adolescent self-reports of depressive symptoms at a population-level (Angold et al., 1995; Bond et al., 2005). Good criterion validity (Angold et al., 1995; Kent, Vostanis, & Feehan, 1997; Thapar & McGuffin, 1998) and construct validity (Sharp, Goodyer, & Croudace, 2006) have been established for the SMFQ. The SMFQ has sound construct validity in Australian samples, being longitudinally predicted by prior exposure to family conflict (Habib et al., 2014), parent reports of child internalising and child reports of emotional control and family attachment (Toumbourou et al., 2011). Heerde et al. (2015) found it predicted future self-harm. Kremer et al. (2014) found it was associated with lack of physical activity.

Prior Australian longitudinal studies have found that a cut-off of 8 or more SMFQ symptoms strongly predicts the future development of depressive disorders amongst Australian adolescents (McKenzie et al., 2011). As such, the current study used this cut-off to examine rates of elevated (sub-clinical and clinical) depressive symptoms in a national sample of Australian adolescents.

International and national prevalence rates
There is currently very little information to monitor rates of depressive symptoms amongst adolescents. A representative study of American high school students from Oregon (N= 10,200) utilised the Schedule for Affective Disorders and Schizophrenia for school-aged children (K-SADS) to assess sub-clinical and clinical psychiatric disorders (Lewinsohn et al., 2004). The study identified 25.9% of adolescents experienced sub-clinical and 18.4% of adolescents experienced clinical Major Depressive Disorder (Lewinsohn et al., 2004). A more recent nationally representative sample (N = 10,123) of American adolescents (13-18 years) assessed through structured clinical interviews indicated that 14.3% met the criteria the DSM-IV criteria for mood disorders (Merikangas et al., 2010).

A national study (N = 6310) of Australian children and adolescents (4-17years) indicated lower prevalence rates than those identified in US studies (Lawrence et al., 2015). The study identified 2.8% of children and adolescents met the criteria for Major Depressive Disorder as assessed by parent reports using the DISC-IV (Lawrence et al., 2015). A higher proportion of adolescents (12-17 years: male 12 month prevalence = 4.3%, female 12 month prevalence = 5.8%) met the criteria for Major Depressive Disorders compared to children (4-11 years: male = 1.1, female = 1.2%, Lawrence et al., 2015). Due to the internalising nature of symptoms, parent reports may underrepresent true emotional difficulties, and as such adolescent reports may provide a more accurate estimate (Cantwell, Lewinsohn, Rohde, & Seeley, 1997; van de Looij-Jansen, Jansen, de Wilde, Donker, & Verhulst, 2011). Indeed, Lawrence’s et al.’s (2015) findings suggested that female’s (11-17 years) and males (15-17 years) were more likely to meet the criteria for Major Depressive Disorder based on adolescent reports than parent reports.
In Australia, there is limited evidence identifying the prevalence of adolescent depressive symptoms. A study (N = 1,299; age: 11-18 years) of school children from the state of Victoria has been undertaken (Boyd et al., 2000). Using the Reynolds’ Adolescent Depression Scale, this study identified that 14.2% of adolescents were depressed (Boyd et al., 2000). These rates are similar to those identified in other western countries (e.g., Britain, USA, and Canada: Boyd et al., 2000).

A larger Australian study (N = 8,984) of Victorian secondary school students found similar rates using the SMFQ to assess clinical cut-off depressive symptoms (Bond et al., 2005). This study identified that 16.6% of adolescents experienced depression (Bond et al., 2005). However, the prevalence rates from these two Australian studies may be an underestimate of the true degree of emotional difficulties experienced by Australian children, as many children exhibit elevated sub-clinical levels of depression (Bayer & Sanson, 2003; González-Tejera et al., 2005). Thus identifying the prevalence of depressive symptoms (that apply sub-clinical cut offs) is an important first step in the consideration of preventative interventions.

Prevention and community variation

It is important to understand to what extent adolescent depression prevention efforts can be universal across the nation versus tailored to specific community conditions. Preventative interventions have indicated small scale effectiveness at reducing the prevalence of depression in school-aged children (Corrieri et al., 2014; Merry et al., 2011). Current preventative interventions have been criticized for being more treatment than preventative focused, usually only targeting individual-level factors despite calls for the use of multi-level
Increasingly place-based (community-level) approaches have been utilised in prevention attempts (Moore, 2014; Moore & Fry, 2011; Rushton, 2014). Whilst prevalence rates of depression have been reported to vary by urban or regional geographical location (Boyle & Lipman, 2002) there is limited evidence to assess the extent of variation between communities. To determine the appropriateness of utilising place-based approaches it is important to understand whether the prevalence of adolescent depression varies between geographic areas.

Whilst community disadvantage is one of the most frequently implicated place-based factors posited as a risk factor for child and adolescent well-being (Herrman, Saxena, Moodie, & Walker, 2005; McDermott et al., 2010), there appears to be limited evidence to support these claims. Indeed, a meta-analysis of individual level variables did not support socio-economic status as a moderator of childhood depression (Twenge & Nolen-Hoeksema, 2002). Similarly a more recent meta-analysis did not identify a significant association between child depression and community disadvantage, however this relationship was found to be influenced by other community-level factors (Stirling, Toumbourou, & Rowland, 2015). To determine the appropriateness of utilising place-based approaches in Australia it is important to understand the variation in adolescent depression by community-level disadvantage (socioeconomic status [SES]).

**Demographic factors**

In assessing the prevalence and community variation of adolescent depression it is important to consider the influence of demographic characteristics including individual and family factors. Family demographic characteristics (e.g., household composition and parent employment; Strohschein, 2005) are related to
community disadvantage and have been found in Australian studies to be associated with child depression (Sawyer et al., 2001).

Prevalence rates of adolescent depression vary by gender (Merikangas et al., 2010). For example Australian girls reported significantly higher rates of depression than boys (18.8% versus 9.3; Boyd et al., 2000). These findings were replicated in another Australia study that also identified differences by gender with 21.7% of females and 10.5% of males experiencing depression (Bond et al., 2005). Heterogeneity in meta-analytic findings suggest that these differences may be at least partly explained by differences in factors such as stress responsivity (Twenge & Nolen-Hoeksema, 2002).

Mixed findings are associated with child age. Meta-analytic results suggested that age was not significantly associated with adolescent depression (Twenge & Nolen-Hoeksema, 2002). These findings have been replicated in Australian samples (Bond et al., 2005; Boyd et al., 2000). However, prevalence rates of depression appear to peak at different stages for males and females. Findings suggest that, for adolescent boys, depression appears to be relatively stable with a peak at age 12, for girls the prevalence of depression increases after age 11 with another significant increase at age 13, peaking at age 15 (Twenge & Nolen-Hoeksema, 2002). In this light, some researchers argue that puberty may be a better predictor of depression than age (DeRose, Wright, & Brooks-Gunn, 2006; Hayward, Gotlib, Schraedley, & Litt, 1999). Attempts to reduce adolescent depression during this period have also targeted the transition to secondary school suggesting prevalence rates may rise for females through this transition (Woods & Pooley, 2015). Given, the important puberty and school-related transitions that
occur during early adolescence (12-14 years) this is an important age sample to examine.

Mixed findings are also associated with community variation in ethnicity. Meta-analysis results identified that whilst Hispanic children (8-16 years) reported significantly more depressive symptoms than White and African American children, no differences between White and African American children were found (Twenge & Nolen-Hoeksema, 2002). There has been limited examination of the association between ethnicity and depression in Australian adolescents.

**Study aims**

Using a representative sample of Australian adolescents, the current study aimed to estimate the prevalence of adolescent depressive symptoms (sub-clinical and clinical) in a community sample specifically designed to represent national diversity in community disadvantage (SES). The present study also explored whether prevalence varied across 30 Australian communities. Based on prior studies the study hypotheses were firstly that elevated depression symptoms (sub-clinical and clinical) would be evident for more than 16.6% of adolescents (Bond et al., 2005) and secondly that variation would be evident across communities with higher rates evident for higher disadvantaged communities after controlling for age, gender, ethnicity and other covariates.

**Method**

**Study design and participants**

Data was collected in 2006 as part of the Healthy Neighbourhoods Study; a large cross-sectional study, that assessed adolescents’ health and wellbeing in 30 communities across 3 Australian states (Victoria, community N =14; Queensland, N = 8; Western Australia, N = 8). The study was designed to represent community
### Table 1

**Sample Demographics**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sample %</th>
<th>Prevalence of Sub-clinical and Clinical Depression % (CI)</th>
<th>Prevalence of Clinical Depression % (CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>48.0</td>
<td>30.3 (28.8, 31.8)</td>
<td>15.8 (14.6, 16.9)</td>
</tr>
<tr>
<td>Female</td>
<td>52.0</td>
<td>35.7 (34.3, 37.2)</td>
<td>21.1 (19.9, 22.4)</td>
</tr>
<tr>
<td><strong>School Grade</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 6</td>
<td>54.7</td>
<td>32.1 (30.7, 33.5)</td>
<td>17.3 (16.2, 18.4)</td>
</tr>
<tr>
<td>Grade 8</td>
<td>45.3</td>
<td>34.4 (32.8, 35.9)</td>
<td>20.0 (18.7, 21.3)</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11 and under</td>
<td>46.5</td>
<td>33.7 (32.2, 35.2)</td>
<td>18.6 (17.4, 19.9)</td>
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<tr>
<td>12 and over</td>
<td>53.5</td>
<td>32.6 (31.2, 34.0)</td>
<td>18.5 (17.3, 19.6)</td>
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<tr>
<td><strong>Indigenous</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Indigenous Australian</td>
<td>96.8</td>
<td>33.0 (32.0, 34.1)</td>
<td>18.4 (17.6, 19.3)</td>
</tr>
<tr>
<td>Indigenous Australian</td>
<td>3.2</td>
<td>33.1 (27.2, 38.9)</td>
<td>20.7 (15.7, 25.7)</td>
</tr>
<tr>
<td><strong>Child’s Country of Birth</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Australia</td>
<td>90.7</td>
<td>33.3 (32.3, 34.4)</td>
<td>18.8 (17.9, 19.7)</td>
</tr>
<tr>
<td>Outside Australia</td>
<td>9.3</td>
<td>31.0 (27.7, 34.4)</td>
<td>16.3 (13.7, 19.0)</td>
</tr>
<tr>
<td><strong>Parent’s Country of Birth</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother Born in Australia</td>
<td>73.3</td>
<td>33.3 (32.1, 34.5)</td>
<td>18.7 (17.7, 19.7)</td>
</tr>
<tr>
<td>Mother Born Outside Australia</td>
<td>26.7</td>
<td>32.7 (30.7, 34.7)</td>
<td>18.0 (16.4, 19.7)</td>
</tr>
<tr>
<td>Father Born in Australia</td>
<td>71.9</td>
<td>33.1 (31.9, 34.3)</td>
<td>18.7 (17.7, 19.7)</td>
</tr>
<tr>
<td>Father Born Outside Australia</td>
<td>28.1</td>
<td>32.8 (30.8, 34.7)</td>
<td>17.8 (16.2, 19.4)</td>
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<tr>
<td><strong>Language Spoken at Home</strong></td>
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<td></td>
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<tr>
<td>English only</td>
<td>88.4</td>
<td>32.9 (31.8, 34.0)</td>
<td>18.7 (17.7, 19.6)</td>
</tr>
<tr>
<td>Other language only</td>
<td>1.5</td>
<td>37.7 (29.0, 46.3)</td>
<td>18.0 (11.2, 24.9)</td>
</tr>
<tr>
<td>English and other language</td>
<td>10.1</td>
<td>34.5 (31.2, 37.8)</td>
<td>17.6 (15.0, 20.2)</td>
</tr>
<tr>
<td><strong>Household Composition</strong></td>
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<td></td>
<td></td>
</tr>
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<td>Mother not at home</td>
<td>9.2</td>
<td>42.8 (39.2, 46.4)</td>
<td>26.0 (22.8, 29.2)</td>
</tr>
<tr>
<td>Mother at home</td>
<td>90.8</td>
<td>32.2 (31.1, 33.2)</td>
<td>17.8 (16.9, 18.7)</td>
</tr>
<tr>
<td>Father not at home</td>
<td>25.7</td>
<td>39.4 (37.3, 41.5)</td>
<td>23.5 (21.7, 25.4)</td>
</tr>
<tr>
<td>Father at home</td>
<td>74.3</td>
<td>31.0 (29.8, 32.1)</td>
<td>16.9 (15.9, 17.8)</td>
</tr>
<tr>
<td><strong>Mother’s Employment</strong></td>
<td></td>
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<td></td>
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<tr>
<td>Full-time</td>
<td>36.6</td>
<td>34.2 (32.5, 35.9)</td>
<td>19.7 (18.2, 21.1)</td>
</tr>
<tr>
<td>Unemployed</td>
<td>24.0</td>
<td>34.4 (32.2, 36.6)</td>
<td>19.0 (17.2, 20.8)</td>
</tr>
<tr>
<td>Retired</td>
<td>1.5</td>
<td>36.8 (27.9, 45.7)</td>
<td>21.1 (13.5, 28.6)</td>
</tr>
<tr>
<td>Part-time</td>
<td>36.8</td>
<td>30.0 (28.3, 31.7)</td>
<td>15.9 (15.5, 17.2)</td>
</tr>
<tr>
<td><strong>Father’s Employment</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Full-time</td>
<td>73.9</td>
<td>31.1 (29.9, 32.3)</td>
<td>17.1 (16.1, 18.1)</td>
</tr>
<tr>
<td>Unemployed</td>
<td>5.8</td>
<td>38.9 (34.4, 43.4)</td>
<td>24.1 (20.2, 28.1)</td>
</tr>
<tr>
<td>Retired</td>
<td>1.6</td>
<td>45.5 (36.5, 54.4)</td>
<td>23.1 (15.6, 30.7)</td>
</tr>
<tr>
<td>Part-time</td>
<td>13.8</td>
<td>35.6 (32.7, 38.5)</td>
<td>19.0 (16.6, 21.3)</td>
</tr>
<tr>
<td><strong>State</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Victoria</td>
<td>46.5</td>
<td>29.5 (28.1, 31.0)</td>
<td>15.8 (14.6, 16.9)</td>
</tr>
<tr>
<td>Queensland</td>
<td>30.3</td>
<td>38.5 (36.6, 40.5)</td>
<td>22.4 (20.7, 24.1)</td>
</tr>
<tr>
<td>Western Australia</td>
<td>23.3</td>
<td>33.5 (31.3, 35.6)</td>
<td>19.2 (17.5, 21.0)</td>
</tr>
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<td><strong>Locality</strong></td>
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<tr>
<td>Urban</td>
<td>50.1</td>
<td>34.1 (32.6, 35.5)</td>
<td>18.9 (17.7, 20.1)</td>
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<tr>
<td>Regional</td>
<td>49.9</td>
<td>32.2 (30.7, 33.6)</td>
<td>18.2 (17.0, 19.4)</td>
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<td><strong>Community Disadvantage</strong></td>
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<td></td>
<td></td>
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<tr>
<td>Quartile 1 (high)</td>
<td>28.4</td>
<td>34.9 (32.9, 36.8)</td>
<td>19.1 (17.4, 20.7)</td>
</tr>
<tr>
<td>Quartile 2</td>
<td>24.3</td>
<td>34.7 (32.6, 36.9)</td>
<td>20.8 (19.0, 22.6)</td>
</tr>
<tr>
<td>Quartile 3</td>
<td>23.2</td>
<td>35.0 (32.8, 37.1)</td>
<td>20.3 (18.5, 22.1)</td>
</tr>
<tr>
<td>Quartile 4 (low)</td>
<td>24.2</td>
<td>27.7 (25.7, 29.7)</td>
<td>14.1 (12.5, 15.6)</td>
</tr>
</tbody>
</table>

CI = 95% Confidence Interval
disadvantage (SES) variation across Australia and valid epidemiological estimation within each of the 30 localities within the national sample. Localities were based on Statistical Local Areas (SLAs: based on Australian Bureau of Statistics divisions) stratified by community disadvantage and location in urban and non-urban areas, and were randomly selected from within these strata (Habib et al., 2014; Smith et al., 2014). Half of the communities were from regional areas and the other half from urban areas.

All schools (Government, Independent, and Catholic) in the randomly selected areas were invited to participate with 53% of schools choosing to participate in the study. All students within the schools were invited to participate, of the 15,666 consent forms distributed, 9,830 (63%) were returned and of these 92% of grade 6 and 89% of grade 8 students’ parents consented to their child’s participation. Participants were assessed in school utilising an online self-report survey, or where not available a paper copy. At this stage of data collection no interventions had been implemented. Table 1 presents information on their demographic characteristics. Ethical approval for the study was provided by both the Murdoch Children’s Research Institute Human Research Ethics Committee, and relevant school and institutional ethics committees in each state.

**Measures**

Participants were asked a range of demographic questions including: gender, school grade, age, indigenous status, country of birth, mother and father’s country of birth, language/s spoken at home, whether their mother and father lived in their home, and mother’s and father’s employment status. State of residence, SLA, and urban or regional area were determined at the study design stage. Communities were divided by SLA and the Australian Bureau of Statistics (ABS)
Socio-economic Index for Areas (SEIFA) was used to determine community-level disadvantage. The Index applied was the Index of Relative Socio-economic Advantage/Disadvantage (IRSAD) which uses factors such as the proportion of households with low/high income; proportion of people with or without qualifications; and the proportion of people in a community in low skilled occupations to determine community-level disadvantage (ABS, 2001). A low score represents the most disadvantaged communities (quartile 1) and a high score represents the less disadvantaged communities (quartile 4).

Depression was measured using the Short Mood and Feelings Questionnaire (SMFQ). The SMFQ is a 13 item self-report questionnaire, developed to assess depressive symptoms in epidemiological studies, based on the DSM-III criteria for depression (Angold, Costello, Messer, & Pickles, 1995). The SMFQ has good internal reliability (Cronbach’s Alpha = 0.98) and has moderate correlations with the Children’s Depression Inventory \( r = 0.65 \) and the Diagnostic Interview Schedule for Children \( r = 0.65 \) measures of depression (Angold et al., 1995).

The SMFQ asked participants to respond to questions about their mood, feelings, and behaviours over the past 2 weeks. Participants were asked to respond on a three point Likert scale (i.e., “true” [2], “sometimes” [1], or “not true” [0]) to questions such as “I didn’t enjoy anything at all” and “I found it hard to think properly or concentrate”. Scores from the 13 questions (Cronbach’s Alpha = 0.94) were summed to provide a total symptom count, with a possible maximum of 26 and minimum of 0. As recommended by Angold et al. (1995) a cut-off score of 8 and above was used to determine sub-clinical symptomology. A cut-off of 11 or higher on the SMFQ has been shown to represent the 94th percentile in a
community-based sample, which reflects the 1-year prevalence of clinical depression in the US population of children and adolescents and thus was used to determine clinical symptomology in the current study (Angold, Erkanli, Silberg, Daves, & Costello, 2002). In the current study, as outlined above elevated depressive symptoms include the presence of sub-clinical or clinical symptomology.

Analyses

All statistical analyses were performed using STATA, Version 12.1. Proportions with 95% confidence intervals (CIs) were calculated for demographic variables to determine depression prevalence rates. Multi-level logistic regression was used to calculate univariate and multivariate odds ratios (OR) with 95% CIs. The intercept was allowed to vary by community (i.e., random effect). Univariate analyses compared a referent category with other levels on each predictor. Consistent with previous literature (Kelly et al., 2012), gender interaction effects were assessed for all variables.

The overall prevalence rate was calculated based on 7988 participants (96.7%) who responded to all questions on the SMFQ. For all included variables missing data was less than 5% with the exception of mother’s (5.22%) and father’s employment (5.81%). The multivariate sample included 7,720 participants who responded to all questions related to the variables included in the analyses.

The multivariate model included all significant predictors and significant gender interaction effects identified in univariate analyses. Non-significant effects were removed until only variables that were significant were maintained in the final multivariate model. All significance levels were set at $p < .05$. 

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Results

Overall 33.1% (95 % CI [32.1, 34.2]) of Australian adolescents reported depressive symptomatology with 14.6% (95 % CI [13.8, 15.3]) endorsing sub-clinical and 18.6% (95 % CI [17.7, 19.4]). Multi-level modelling (MLM) analyses were completed to establish the variation in adolescent depression associated with the community SLA variable and this was found to explain 6.9% of the variance.

Figure 1 shows the variation in the prevalence of depressive symptoms across all 30 SLA communities broken down by the independent variable of community disadvantage (1 = high disadvantage [low SES] to 4 = low disadvantage [high SES]). The community estimates presented in Figure 1 are weighted to represent averages at mean age 12 and with a 50% gender proportion and confidence intervals adjusted for sample clustering within school classrooms. The prevalence of children with elevated depressive symptoms ranged across communities from 28.0 to 39.5 %, with variation not completely explained by community-level disadvantage. Figure 1 below shows that high disadvantaged communities consistently had prevalence rates above 30%, while this was less common for low disadvantage communities (i.e., quartile 3 & 4).
Figure 1. Prevalence of Depressive Symptoms as a Function of Community (a, b, c…) and Community Disadvantage (SES) Level (1 to 4)

Demographic details of participants are presented in Table 1. Table 1 also presents the prevalence rates for elevated depressive symptoms (sub-clinical and clinical) and clinical depressive symptoms in sub-samples. In the current sample 48% of children were male and 52% female. More females (35.7%, 95% CI [34.3, 37.2]) reported experiencing depressive symptoms than males (30.3%, 95% CI [28.8, 31.8]).

Table 2 reports prevalence rates of depressive symptomology by school grade broken down by gender and State. Results indicated gender differences in the prevalence rates within Victoria and Queensland. Victorian females were more likely to report depressive symptoms than their male counterparts (OR = 1.40, 95% CI [1.22, 1.62], p < 0.001). Similarly in Queensland female adolescents (OR = 1.23, 95% CI [1.04, 1.46], p = 0.14) were more likely to report depressive symptoms than their male counterparts. No gender differences were identified in WA Adolescents.

Table 2
Prevalence Rates of Adolescent Depressive Symptoms (Combined Sub-clinical and Clinical) by State, Grade, and Gender

<table>
<thead>
<tr>
<th></th>
<th>Victoria Prevalence % (CI %)</th>
<th>Queensland Prevalence % (CI %)</th>
<th>Western Australia Prevalence % (CI %)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 6</td>
<td>26.7 (24.7, 28.6)</td>
<td>41.5 (38.6, 44.4)</td>
<td>32.3 (29.7, 35.0)</td>
</tr>
<tr>
<td>Grade 8</td>
<td>32.9 (30.7, 35.1)</td>
<td>35.8 (33.1, 38.5)</td>
<td>35.5 (31.9, 39.2)</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 6</td>
<td>27.3 (24.6, 30.0)</td>
<td>42.5 (38.5, 46.5)</td>
<td>31.7 (28.0, 35.3)</td>
</tr>
<tr>
<td>Grade 8</td>
<td>39.5 (36.3, 42.7)</td>
<td>39.9 (36.1, 43.7)</td>
<td>38.7 (33.7, 43.7)</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 6</td>
<td>26.0 (23.3, 28.7)</td>
<td>40.4 (36.2, 44.5)</td>
<td>33.1 (29.2, 37.0)</td>
</tr>
<tr>
<td>Grade 8</td>
<td>25.7 (22.7, 28.7)</td>
<td>31.4 (27.6, 35.1)</td>
<td>31.7 (26.4, 36.9)</td>
</tr>
</tbody>
</table>

CI = 95% Confidence Interval
Grade differences for the total population were identified in Victoria and Queensland. In Victoria grade 8 students were more likely to experience depressive symptoms than their grade 6 counterparts (OR = 1.34, 95% CI [1.16, 1.55], p < 0.001) where as in Queensland grade 8 students (OR = 0.81, 95% CI [0.68, 0.96], p = 0.014) were less likely to experience depressive symptoms than their grade 6 counterparts, no differences were found in the WA adolescents.

Table 3 presents univariate (unadjusted) Odds Ratios (ORs) predicting depressive symptoms and univariate gender interaction effects. Univariate analysis indicated that females had a significantly increased odds (OR: 1.27, 95% CI [1.15-1.39]) of experiencing depressive symptoms, compared to males. Univariate analyses also revealed that depression was significantly higher among: students in grade 8; adolescents whose fathers were unemployed, working part-time, or retired; adolescents who lived in Queensland; and adolescents who lived in high disadvantage communities. Univariate analyses indicated that depression was lower for adolescents whose mothers worked part-time; whose mothers lived with them; and whose father lived with them. Univariate gender interaction effects were identified for school grade; age; household composition; father’s employment; and State.

Table 4 presents multivariate ORs (including gender interaction effects) for the prediction of depression in the total sample. Consistent with the findings presented in Figure 1, multivariate analysis (Table 4) indicated higher rates of depressive symptoms for adolescents from more disadvantaged communities after controlling for family demographics and other covariates. Adolescent father’s employment status (unemployed, retired, and part-time employment); and living in Queensland or Western Australia also increased the likelihood of depressive
### Table 3

Univariate Odds Ratios and Gender Interaction Effects of Adolescent Depressive Symptoms (Combined Sub-clinical and Clinical)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariate OR (CI)</th>
<th>P-value</th>
<th>Gender Interaction OR (CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>1.27 (1.15, 1.39)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>School Grade</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 8</td>
<td>1.10 (1.00, 1.22)</td>
<td>0.043</td>
<td>1.55 (1.29, 1.88)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12 years and over</td>
<td>1.05 (0.95, 1.16)</td>
<td>0.338</td>
<td>1.33 (1.10, 1.61)</td>
<td>0.003</td>
</tr>
<tr>
<td>Indigenous</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indigenous Australian</td>
<td>0.91 (0.69, 1.19)</td>
<td>0.485</td>
<td>1.07 (0.62, 1.84)</td>
<td>0.803</td>
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<tr>
<td>Child’s Country of Birth</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outside Australia</td>
<td>0.86 (0.73, 1.02)</td>
<td>0.081</td>
<td>1.04 (0.75, 1.46)</td>
<td>0.799</td>
</tr>
<tr>
<td>Mother’s Country of Birth</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outside Australia</td>
<td>0.94 (0.84, 1.06)</td>
<td>0.327</td>
<td>1.09 (0.88, 1.35)</td>
<td>0.428</td>
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<tr>
<td>Father’s Country of Birth</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outside Australia</td>
<td>0.96 (0.85, 1.07)</td>
<td>0.432</td>
<td>1.20 (0.97, 1.48)</td>
<td>0.099</td>
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<tr>
<td>Language Spoken at Home</td>
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<td></td>
</tr>
<tr>
<td>Other language only</td>
<td>1.22 (0.84, 1.78)</td>
<td>0.299</td>
<td>1.24 (0.57, 2.71)</td>
<td>0.581</td>
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<tr>
<td>English and other language</td>
<td>1.08 (0.91, 1.28)</td>
<td>0.356</td>
<td>1.20 (0.88, 1.64)</td>
<td>0.258</td>
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<tr>
<td>Household Composition</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Mother at home</td>
<td>0.65 (0.56, 0.76)</td>
<td>&lt; 0.001</td>
<td>0.99 (0.72, 1.35)</td>
<td>0.936</td>
</tr>
<tr>
<td>Father at home</td>
<td>0.71 (0.64, 0.79)</td>
<td>&lt; 0.001</td>
<td>1.31 (1.06, 1.61)</td>
<td>0.014</td>
</tr>
<tr>
<td>Mother’s Employment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unemployed</td>
<td>0.97 (0.86, 1.10)</td>
<td>0.650</td>
<td>0.91 (0.71, 1.16)</td>
<td>0.446</td>
</tr>
<tr>
<td>Retired</td>
<td>1.11 (0.75, 1.64)</td>
<td>0.604</td>
<td>0.54 (0.25, 1.21)</td>
<td>0.134</td>
</tr>
<tr>
<td>Part-time</td>
<td>0.82 (0.74, 0.92)</td>
<td>0.001</td>
<td>0.96 (0.77, 1.21)</td>
<td>0.745</td>
</tr>
<tr>
<td>Father’s Employment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unemployed</td>
<td>1.35 (1.10, 1.65)</td>
<td>0.003</td>
<td>0.68 (0.45, 1.01)</td>
<td>0.055</td>
</tr>
<tr>
<td>Retired</td>
<td>1.77 (1.23, 2.55)</td>
<td>0.002</td>
<td>0.46 (0.22, 0.96)</td>
<td>0.039</td>
</tr>
<tr>
<td>Part-time</td>
<td>1.20 (1.04, 1.37)</td>
<td>0.012</td>
<td>0.63 (0.48, 0.83)</td>
<td>0.001</td>
</tr>
<tr>
<td>State</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Queensland</td>
<td>1.55 (1.27, 1.89)</td>
<td>&lt; 0.001</td>
<td>0.88 (0.71, 1.10)</td>
<td>0.262</td>
</tr>
<tr>
<td>Western Australia</td>
<td>1.22 (0.99, 1.49)</td>
<td>0.058</td>
<td>0.77 (0.60, 0.98)</td>
<td>0.031</td>
</tr>
<tr>
<td>Locality</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Regional</td>
<td>0.91 (0.74, 1.12)</td>
<td>0.392</td>
<td>0.90 (0.75, 1.09)</td>
<td>0.299</td>
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<tr>
<td>Community Disadvantage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quartile 1 (highest)</td>
<td>1.42 (1.10, 1.83)</td>
<td>0.007</td>
<td>1.12 (0.85, 1.46)</td>
<td>0.423</td>
</tr>
<tr>
<td>Quartile 2</td>
<td>1.39 (1.07, 1.81)</td>
<td>0.014</td>
<td>0.89 (0.68, 1.18)</td>
<td>0.418</td>
</tr>
<tr>
<td>Quartile 3</td>
<td>1.40 (1.08, 1.81)</td>
<td>0.012</td>
<td>1.06 (0.80, 1.41)</td>
<td>0.661</td>
</tr>
</tbody>
</table>

OR = Odd Ratio, CI = 95% Confidence Interval †Univariate analyses are adjusted only for community clustering, ‡Gender interactions: Females compared to males.

Symptoms. Adolescents who lived with their mother; lived with their father; or whose mother worked part-time were significantly less likely to report depressive symptomology.
Table 4

Multivariate Odds Ratios and Gender Interaction Effects of Adolescent Depressive Symptoms (Combined Sub-clinical and Clinical)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Multivariate OR (CI)</th>
<th>P-value</th>
<th>Gender interaction</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>School Grade</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 8</td>
<td></td>
<td></td>
<td>1.46 (1.20, 1.77)</td>
<td>&lt; .0001</td>
</tr>
<tr>
<td><strong>Household Composition</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother at home</td>
<td>0.78 (0.64, 0.94)</td>
<td>0.01</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Father at home</td>
<td>0.80 (0.70, 0.90)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mother’s Employment</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unemployed</td>
<td></td>
<td></td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Retired</td>
<td></td>
<td></td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Part-time</td>
<td>0.82 (0.73, 0.92)</td>
<td>0.001</td>
<td></td>
<td>N/A</td>
</tr>
<tr>
<td><strong>Father’s Employment</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unemployed</td>
<td>1.58 (1.18, 2.13)</td>
<td>0.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Retired</td>
<td>2.48 (1.48, 4.15)</td>
<td>0.001</td>
<td>0.43 (0.21, 0.91)</td>
<td>0.027</td>
</tr>
<tr>
<td>Part-time</td>
<td>1.51 (1.23, 1.85)</td>
<td>&lt; .0001</td>
<td>0.68 (0.51, 0.89)</td>
<td>0.006</td>
</tr>
<tr>
<td><strong>State</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Queensland</td>
<td>1.52 (1.31, 1.78)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Western Australia</td>
<td>1.20 (1.02, 1.42)</td>
<td>0.026</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Community Disadvantage</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quartile 1 (highest)</td>
<td>1.32 (1.10, 1.59)</td>
<td>0.003</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Quartile 2</td>
<td>1.31 (1.09, 1.59)</td>
<td>0.005</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Quartile 3</td>
<td>1.37 (1.14, 1.66)</td>
<td>0.001</td>
<td>N/A</td>
<td></td>
</tr>
</tbody>
</table>

OR = Odd Ratio, CI = 95% Confidence Interval, †N = 7,720, ‡Multivariate analyses are adjusted for community clustering. †Gender interactions: Females compared to males.

In the multivariate model significant gender interaction effects were identified for the variables school grade and father’s employment. Females in grade eight were more likely (OR = 1.46, 95 % CI [1.20-1.77], p < .001) to report depressive symptomology than males in grade 8. Males were significantly more likely to experience depressive symptomology based on their father’s employment status than females. Females, relative to males were less likely to report depressive symptoms if their fathers were retired (OR = 0.43 95 % CI [0.21-0.91], p = .027) or worked part-time (OR = 0.68, 95 % CI [0.51-0.89], p = .006)

Discussion

Findings supported both of the study hypotheses. First, elevated depressive symptomology was evident for one third of the sample; around double the rate identified by Bond et al. (2005). Second, prevalence varied significantly across
Australian communities with higher rates evident for communities with higher disadvantage, after controlling for covariates.

The current study identified that 33.1% (CI: 32.1-34.2) of Australian adolescents (10-14 years) reported depressive symptoms (sub-clinical and clinical) that were sufficiently elevated to pose risks for developing future health and social problems. As would be expected, this was much higher than the prevalence of clinical-level depression symptoms with a higher cut off score identified in the current study (18.6% [CI: 17.7, 19.4]) and previous Australian samples (14.2% [Boyd et al., 2000]; 16.6% [Bond et al., 2005]) of older adolescents (11-18 years). However, the prevalence rates estimated in the current study were also somewhat lower than findings from a USA study that identified 44.3% of American adolescents (Mean age = 16.6 years) experienced sub-clinical or clinical depression (Lewinsohn et al., 2004). Adolescents in the American sample were comparatively older than adolescents in the Australian sample. Further cross-nationally matched research is warranted in age matched samples to inform accurate comparisons.

In line with the hypotheses, adolescents from communities of higher disadvantage were more likely to report depressive symptoms than children from less disadvantaged communities. A recent systematic review identified that community disadvantage was associated with adolescent depressive symptoms, but effects were not significant in meta-analytic results suggesting that the effect of disadvantage may be influenced by other factors (Stirling et al., 2015). Figure 1 showed that although an effect of community disadvantage was evident, there was considerable variation between communities within each disadvantage (SES) strata. Further research should investigate malleable factors that underlie...
community variation in adolescent depressive symptoms (Stirling et al., 2015). For example, developmental theory and some evidence suggests that community disadvantage may act indirectly, for example, by disrupting mother–child bonding (Letcher et al., 2009; Stirling et al., 2015). Thus, relevant child mental health promotion efforts may be better directed at protecting mother-child bonding in disadvantaged families (Letcher et al., 2009).

In accordance with previous Australian research examining clinical depression (Boyd et al., 2000; Bond et al., 2005), significantly more females reported depressive symptoms than males. Gender interaction effects were identified for father’s employment and school grade. Future research should examine gender differences in modifiable risk and protective factors that may influence depressive symptoms.

Consistent with prior Australian research examining clinical depression, age was not significantly associated with depressive symptoms (Boyd et al., 2000), after adjusting for grade and other factors. However, it is important to note these findings may be impacted by the truncated age sample of the current study and further research with other age groups is warranted to inform accurate comparisons.

Indigenous adolescents; adolescents from culturally and linguistically diverse backgrounds; and adolescent who spoke other languages at home did not report higher levels of depressive symptoms. Meta-analytic findings suggest that community-level minority ethnicity and discrimination are associated with child and adolescent depression (Stirling et al., 2015). A possible explanation for the current findings is that stressful experiences of acculturation and discrimination rather than ethnicity may influence depressive symptoms. Another plausible
explanation is that findings were affected by the small proportion of minority
groups in the current sample. Further research examining the experience of
purposive samples of Australian adolescents from indigenous and culturally and
linguistically diverse backgrounds may be needed to more fully understand
variation within these groups.

Consistent with prior research family-level variables were associated with
depressive symptoms (Yap, Pilkington, Ryan, & Jorm, 2014). As identified in
previous research (Sawyer et al., 2001; Strohschein, 2005) family demographic
factors (household composition and parent employment) were significantly
associated with depressive symptoms.

The current study is the first Australian study to examine the national
prevalence of elevated depressive symptoms in adolescents by State. Accounting
for family demographics and other covariates, adolescents in Queensland and
Western Australia were more likely to report depressive symptoms than Victorian
adolescents; suggesting that State policy and other contextual factors may play an
important role. In Victoria grade 8 students were more likely to report depressive
symptoms than grade 6 students whereas in Queensland the opposite was evident.
Interestingly in Queensland and Western Australia students in 2006 did not
transition to high school until grade 8, whereas in Victoria the transition occurred
in grade 7.

In the context of calls to increase Australia’s strategic investigation of
options to prevent the development of common mental disorders such as
depression (Jacka et al., 2013), the present study reveals one third of Australian
adolescents reported levels of depressive symptoms that place them at risk of
future depression and related health and social problems. By demonstrating that
adolescent depressive symptom levels vary across communities, the current study provided evidence supporting the view that community-level interventions could form a useful component of Australian national strategies to prevent and reduce adolescent depressive symptoms. The present study suggests community context factors that may influence strategic prevention efforts include: high school gender transitions, household composition, parent labour force participation, State policies, and community levels of disadvantage.

**Limitations and future research**

In generalising the current findings it is important to consider a number of limitations. First, the study is limited in being non experimental and cross-sectional and hence not designed to make causal claims. Intervention and longitudinal research is needed to investigate modifiable risk and protective factors that can be targeted in place-based approaches. Second, whilst the current sample is largely considered a representative Australian sample, it only includes adolescents where parents consented to the survey. Third, the use of self-report measures may be influenced by recall bias and social desirability. However, adolescent self-report measures of depression (Angold et al., 1995; Bond et al., 2005; Boyd et al., 2000; F. N. Jacka et al., 2010) and individual and family-level variables are widely used and shown to be valid in epidemiological studies (Habib et al., 2014; Jacka et al., 2010). Finally, the current study only considers basic demographic variables and does not adjust for a comprehensive range of individual (e.g., adaptive stress coping), and social environmental (e.g., family conflict; parent attachment) risk and protective factors. Future research should examine a wider range of potential risk and protective factors in order to identify modifiable factors that can be targeted through interventions.
Implications

Given the high and unchanging prevalence and significant long-term impacts and costs associated with depression, there has been a significant shift in recent years towards prevention and early intervention by researchers, governments, mental health services, and policy makers. Increasingly governments and policy makers are investing in prevention and early intervention programs that target risk and protective factors that emerge during childhood and adolescence. For example in Australia the Department of Social Services funds the national Family Mental Health Support Service that aims to prevent and reduce the impact of child mental health by working with children and families who present with multiple risk factors.

This study has important implications in the prevention of child and adolescent depression. Firstly it quantifies the prevalence of elevated depression symptoms (sub-clinical and clinical) using a large representative sample of Australian adolescent, that be used to assess population level prevention efforts. Further, the current study supports an important assumption that underpins place-based prevention and early intervention approaches with findings suggesting significant variation in adolescent depression symptoms across Australian communities. Given the high prevalence of depression symptoms in Australian adolescents, this is an important area for prevention research and investment. Further research using Australian samples is needed to identify the malleable risk and protective factors that contribute to these community differences.

Whilst the findings of the current study provide important first steps in establishing an evidence base for preventative interventions, further research is needed. Findings from this study echo support for the demands for a national
mental health prevention strategy (Jorm, 2014). Comprehensive government policies are required to support action across sectors and disciplines in order to identify and assess malleable risk and protective factors to reduce the prevalence of depression (Jorm, 2014). For example, funding contracts should support government funded programs and services to work with researchers in a collaborative approach to develop evidence informed practice approaches (e.g., that target identified risk and protective factors) and utilise robust measures to assess outcomes.

Conclusion

Overall, the current study identified a high prevalence rate of depressive symptoms evident in a third of Australian children in 2006. After controlling for family-level disadvantage and other covariates, community-level disadvantage was significantly associated with depressive symptoms. There was considerable variation between communities within each disadvantage (SES) strata. The findings are important in demonstrating some of the basic conditions that would warrant efforts to design and evaluate place-based early intervention and prevention strategies to reduce adolescent depressive symptoms as a means of preventing the development of depression. Future research should further investigate malleable factors underlying community-level variation including differences at the neighbourhood, individual- peer, family, and school levels and gender-interactions.
References


Hayward, C., Gotlib, I. H., Schraedley, P. K., & Litt, I. F. (1999). Ethnic differences in the association between pubertal status and symptoms of


CHAPTER 5: STUDY 3

Context and Rationale

Overall findings from Study 1 and Study 2 suggested that community factors that are relatively distal to the individual including community disadvantage and Australian State location (Study 2) may influence the prevalence of depression symptoms. In addition, some researchers and policy advocates summarised in earlier chapters (AACAP, 2007; Horowitz & Garber, 2006; Spence & Shortt, 2007) have argued that integrating multi-level interventions targeting predictors more distal to the individual will likely improve the effectiveness of current preventative interventions. Further, intervening at the community-level likely offers a cost-effective opportunity for reducing the burden of depression (Mihalopoulous & Chatterton, 2014; Munoz et al., 2010; Jorm, 2014; Van Zoonen et al., 2014).

Study 1 identified that community safety, community ethnicity and discrimination, community connectedness, and community disadvantage were directly or indirectly associated with child depression. Study 2 identified important community variation in the rates of depression symptoms in early adolescence. The next chapter presents Study 3 that aimed to identify whether community-level variables explained a large component of locality variation in depression symptoms relative to individual, family and school factors, using the same representative sample of Australian adolescents (10-14 years). Further, Study 3 aimed to identify whether the effect of community-level factors such as community disadvantage and State were explained by other predictors of adolescent depression. In accordance with research suggesting gender differences in the developmental trajectory of adolescent depression (Cairns et al., 2014;
Dekker et al., 2007; Fernandez-Castelao & Kröner-Herwig, 2013, Yap et al., 2014), and gender interaction effects identified for demographic variables in Study 2, gender interaction effects were also examined in Study 3.
Authorship Statement for Article 3

Submitted to: Journal of Adolescence
Reference Style: APA 6th

1. Details of publication and executive author

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<th>Publication details</th>
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<td>Multi-level Factors Associated with Community Variation in Adolescent Depressive Symptoms</td>
<td>Submitted to the Journal of Adolescence</td>
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</table>

<table>
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<th>School/Institute/Division if based at Deakin; Organisation and address if non-Deakin</th>
<th>Email or phone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Katie Stirling</td>
<td>School of Psychology</td>
<td><a href="mailto:kstirin@deakin.edu.au">kstirin@deakin.edu.au</a></td>
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2. Inclusion of publication in a thesis

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<td>If Yes, please complete Section 3 If No, go straight to Section 4.</td>
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3. HDR thesis author’s declaration

<table>
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<th>School/Institute/Division if based at Deakin</th>
<th>Thesis title</th>
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</thead>
<tbody>
<tr>
<td>As above</td>
<td>School of Psychology</td>
<td>Investigation of Community Factors Associated with Child and Adolescent Depression</td>
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</table>

If there are multiple authors, give a full description of HDR thesis author’s contribution to the publication (for example, how much did you contribute to the conception of the project, the design of methodology or experimental protocol, data collection, analysis, drafting the manuscript, revising it critically for important intellectual content, etc.)

Conception, writing and compilation of manuscript, established methodology, completed literature search and appraisal, data analyses, preparation of tables and figure.

I declare that the above is an accurate description of my contribution to this paper, and the contributions of other authors are as described below.

<table>
<thead>
<tr>
<th>Signature and date</th>
<th>20.12.15</th>
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4. Description of all author contributions

<table>
<thead>
<tr>
<th>Name and affiliation of author</th>
<th>Contribution(s) (for example, conception of the project, design of methodology or experimental protocol, data collection, analysis, drafting the manuscript, revising it critically for important intellectual content, etc.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Katie Stirling</td>
<td>Conception, writing and compilation of manuscript, established methodology, data analyses, preparation of tables and figure.</td>
</tr>
<tr>
<td>Dr Bosco Rowland</td>
<td>Supervised and assisted with manuscript compilation, editing of manuscript, and provided feedback on the methodology.</td>
</tr>
<tr>
<td>Professor John Toumbourou</td>
<td>Supervised and assisted with manuscript compilation, and provided the data set used for analysis.</td>
</tr>
<tr>
<td>Professor Joanne Williams,</td>
<td>Developed the study and managed the data collection. Contributed to the manuscript.</td>
</tr>
<tr>
<td>Deakin University</td>
<td></td>
</tr>
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5. Author Declarations

I agree to be named as one of the authors of this work, and confirm:

xi. that I have met the authorship criteria set out in the Deakin University Research Conduct Policy,

xii. that there are no other authors according to these criteria,

xiii. that the description in Section 4 of my contribution(s) to this publication is accurate,

xiv. that the data on which these findings are based are stored as set out in Section 7 below.

If this work is to form part of an HDR thesis as described in Sections 2 and 3, I further

xv. consent to the incorporation of the publication into the candidate’s HDR thesis submitted to Deakin University and, if the higher degree is awarded, the subsequent publication of the thesis by the university (subject to relevant Copyright provisions).

<table>
<thead>
<tr>
<th>Name of author</th>
<th>Signature*</th>
<th>Date</th>
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</thead>
<tbody>
<tr>
<td>Katie Stirling</td>
<td></td>
<td>20.12.15</td>
</tr>
<tr>
<td>Dr Bosco Rowland</td>
<td></td>
<td>20.12.15</td>
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<tr>
<td>Prof John Toumbourou</td>
<td></td>
<td>20.12.15</td>
</tr>
<tr>
<td>Associate Professor Joanne Williams</td>
<td></td>
<td>20.12.15</td>
</tr>
</tbody>
</table>

7. Data storage
The original data for this project are stored in the following locations. (The locations must be within an appropriate institutional setting. If the executive author is a Deakin staff member and data are stored outside Deakin University, permission for this must be given by the Head of Academic Unit within which the executive author is based.)

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<th>Storage Location</th>
<th>Date lodged</th>
<th>Name of custodian if other than the executive author</th>
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</thead>
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<tr>
<td>Electronic</td>
<td>Deakin University</td>
<td>December, 2015</td>
<td>John Toumbourou</td>
</tr>
</tbody>
</table>

This form must be retained by the executive author, within the school or institute in which they are based.

If the publication is to be included as part of an HDR thesis, a copy of this form must be included in the thesis with the publication.
Multi-level Factors Associated with Community Variation in Adolescent Depressive Symptoms

Abstract
Depression during adolescence is a predictor of later psychopathology. Few studies have examined modifiable influences targetable in community interventions. A large representative sample (N = 8,256) of Australian grade 6 and 8 (aged 10-14 years) students were sampled from 30 geographic localities across 3 states. A cut-off of 8+ on the Short Mood and Feelings Questionnaire identified depressive symptoms. Multilevel modelling estimated locality variation associated with predictors of depression at the community, school, family, and individual/peer levels. Gender interaction effects were also examined. 33% of Australian adolescents reported depressive symptoms, with 7% of variation explained by locality. Community opportunities for pro-social involvement, community attachment, community safety, community laws and norms favorable to drug use, perceived availability of drugs, transitions and mobility, disadvantage, and State accounted for 88% of the locality variation in depression. Findings support the potential importance of place-based strategies addressing community-level factors in the prevention of depression.
Introduction

Internationally, 5% of the population report suffering from depressive disorders (World Health Organization, 2012) with rates projected to increase in coming decades (Bor, Dean, Najman, & Hayatbakhsh, 2014). Depressive disorders have significant health, social, and economic consequences (Simon, 2003). While treating individuals for depression is important, a preventative approach is required if the rising prevalence of depression is to be curbed and reduced (Jorm, 2014; World Health Organization, 2012).

Evidence indicates that depressive disorders emerge during adolescence, often in the form of sub-clinical symptom elevations (Hankin et al., 1998; Kessler, Avenevoli, & Ries Merikangas, 2001; Kessler et al., 2005; Lewinsohn & Essau, 2002). The early adolescent period of development offers an important window to identify modifiable risk and protective factors that predict an increased or decreased likelihood that an individual will develop depression during adolescence or later in life. In order to inform community-based prevention efforts, the present paper aimed to investigate community-level variation in adolescent depressive symptoms and modifiable factors that may contribute to this variation.

Sub-clinical and clinical depressive symptoms during adolescence have been associated with a range of negative outcomes. Similar to adolescents with clinical depression, adolescents with sub-clinical depression experience significant psychosocial impairment (González-Tejera et al., 2005); increased suicide risk (Fergusson, Horwood, Ridder, & Beautrais, 2005); and increased likelihood of experiencing other sub-clinical and clinical mental health conditions (Lewinsohn, Shankman, Gau, & Klein, 2004). Sub-clinical and clinical depression
in adolescence is the strongest predictor of later depression compared to other risk factors (Lewinsohn & Roberts, 1994; McKenzie et al., 2011).

The negative consequences associated with adolescent-onset depression can have cumulative impacts (Kessler et al., 2001). Adolescents with depressive disorders are at higher risk for: legal problems (American Academy of Child and Adolescent Psychiatry, 2007), increased exposure to negative life events (American Academy of Child and Adolescent Psychiatry, 2007; Jaycox et al., 2009), decreased educational attainment (Kessler, Foster, Saunders, & Stang, 1995; Wagner, Muller, Helmreich, Huss, & Tadic, 2014), and early parenthood (Jaycox et al., 2009). Depressed adolescents frequently experience co-morbid physical and mental health disorders including substance-use disorders (Merikangas et al., 2010).

The cumulative adversity of experiencing depressive symptoms during adolescence contributes to severe psychopathology in adulthood (Kessler et al., 1995). Furthermore, longitudinal studies suggest that once an adolescent has experienced a depressive episode there is a high probability of recurrence (Birmaher, Arbelaez, & Brent, 2002). A major premise guiding the analyses in the present paper is that preventing elevated depression symptoms (sub-clinical and clinical) in adolescents can have direct benefits (Fergusson et al., 2005; González-Tejera et al., 2005; Lewinsohn et al., 2004) and in addition can also contribute to preventing the incidence of depressive disorders (Lewinsohn & Roberts, 1994; McKenzie et al., 2011).

If effective prevention strategies are to be developed, it is important to understand the multiple influences affecting adolescent development (Cicchetti & Rogosch, 1996). Bronfenbrenner’s (1979) ecological perspective provides a
framework for organizing understanding as to how community-level systems influence adolescent development (Andrews, Slade, & Issakidis, 2002).

At the core of Bronfenbrenner’s model is the individual who brings personal experiences and characteristics (e.g., gender, ethnicity) that interact with other systems (Bronfenbrenner, 1995). The microsystem represents the individual’s immediate context of development including peers, family, and school (Bronfenbrenner, 1979). Bronfenbrenner defined the mesosystem to represent the interaction between the environmental systems that most immediately impact the adolescent (Bronfenbrenner, 1995). For example a parent's involvement in neighbourhood activities may strengthen support networks for the adolescent. The next layer, the exosystem, referred to settings that may not directly include the individual but indirectly affect the individual’s experience in immediate settings (Bronfenbrenner, 1979). For example a parent's employment may influence family conflict. The final layer, the macrosystem, included broader societal and cultural influences such as values, laws, customs, and government policies (Bronfenbrenner, 1979). These systems and their influences can be organized on the basis of those more proximal to the individual and moving to those more distal to the individual: individual/peer, family, school, and community.

In order to guide prevention strategies, it is important to identify factors that may explain variation across geographic communities (localities) in adolescent depressive symptoms. There are a range of factors that have previously been found to influence adolescent symptoms. At the individual/peer-level meta-analytic results indicate a number of modifiable factors associated with adolescent depression including: pro-social relationships with peers (Cairns, Yap, Pilkington,
& Jorm, 2014); alcohol and other drug use (Cairns et al., 2014); adaptive coping strategies (Cairns et al., 2014); dieting (Cairns et al., 2014); and externalizing behaviours (Grant et al., 2003). Emerging evidence suggests that physical activity may also be associated with depression (Cairns et al., 2014).

A range of family-level variables have also been indicated in the development of adolescent depression. Meta-analytic results indicate parental over involvement (Yap, Pilkington, Ryan, & Jorm, 2014); parental over control (McLeod, Weisz, & Wood, 2007); parental hostility (Yap et al., 2014); parental rejection (McLeod et al., 2007); and family conflict (Yap et al., 2014) may act as risk factors for depression in children and adolescents. Whereas involving adolescents in decision-making processes (Yap et al., 2014); parental monitoring (Yap et al., 2014); and parental warmth (McLeod et al., 2007; Yap et al., 2014) have been indicated as protective factors.

Factors more distal to the individual have also been implicated in adolescent depression. At the school-level meta-analyses suggest adolescents’ perceptions of school connectedness (Kidger, Araya, Donovan, & Gunnell, 2012); teacher support (Kidger et al., 2012); and academic failure (Riglin, Petrides, Frederickson, & Rice, 2014) are associated with adolescent emotional symptoms. Being a victim of school bullying has also been indicated as a predictor for adult depression (Ttofi, Farrington, Lösel, & Loeber, 2011).

Although less studied, there is also evidence that locality variation in adolescent depressive symptoms arise from community-level factors. Meta-analytic findings indicate that community safety (Fowler, Tompsett, Braciszewski, Jacques-Tiura, & Baltes, 2009; Stirling, Toumbourou, & Rowland, 2015) and community ethnicity and discrimination (Stirling et al., 2015) are risk
It is important to consider gender differences in risk and protective factors for adolescent depressive symptoms. Prevalence rates of sub-clinical (Stirling et al., under submission) and clinical depression (Bond, Toumbourou, Thomas, Catalano, & Patton, 2005; Boyd, Gullone, Kostanski, Ollendick, & Shek, 2000) differ by gender, with females more likely to report depression. Heterogeneity in meta-analytic findings suggest that moderator variables contribute to these differences (Twenge & Nolen-Hoeksema, 2002). Emerging evidence suggests different developmental trajectories of adolescent depression for males and females (Dekker et al., 2007; Fernandez Castelao & Kröner-Herwig, 2013). Findings from the Healthy Neighbourhood Study suggest some gender interactions in demographic variables associated with adolescent depressive symptoms (Stirling et al., under submission). The present paper sought to further investigate gender interactions in the prediction of adolescent depressive symptoms.

Whilst research has established associations between individual, peer, family, school, and community-level factors and adolescent clinical depression, less is known about predictors of sub-clinical depression. To reduce the population burden of depression it is necessary to not only treat existing cases but also to prevent its onset. Sub-clinical depressive symptoms in childhood (Mazza et al., 2009) and adolescence (Peter M. Lewinsohn & Roberts, 1994; McKenzie et al., 2011) are stronger predictors of the onset of depressive disorders relative to other risk factors. Approximately 33% of Australian adolescents experience
elevated (sub-clinical and clinical) depressive symptoms (Stirling et al., under submission). Thus, from a prevention perspective, identifying the predictors of elevated adolescent depressive symptoms is likely to provide important directions for preventive interventions.

Preventative interventions have indicated some effectiveness in reducing the prevalence of depression in school-aged children (Merry et al., 2011), however the effects are small (Corrieri et al., 2013). Current preventative interventions have been criticized for being more treatment than preventative focused, usually only targeting individual-level factors despite calls for the use of multi-level approaches (American Academy of Child and Adolescent Psychiatry, 2007). Further, to achieve population-level reductions in depression it is essential to address the underlying determinants of mental health through coordinated responses across a range of services and settings (Jorm, 2014). Population approaches that target factors more distal to the individual offer opportunities for cost-effective intervention.

To determine the most cost-effective means of intervening, it is important to understand how locality variation in prevalence is explained by modifiable factors at the community, school, family, and individual/peer level. Identifying these differences provides practical opportunities to intervene as at least a component of local funding and services are coordinated at the locality level (county, municipality or region) in most nations (Jacka et al., 2013). As such, the current study aimed to identify locality-level variation in adolescent depressive symptoms and ecological contributors to this variation at the, individual/peer, family, school and community-levels. Gender interaction effects were also examined in the present study.
Method

Study design and participants. The study used data from the 2006 Healthy Neighborhoods Study; a large cross-sectional study that assessed adolescents’ health and wellbeing in 30 localities (with populations of at least 17,000 people) across 3 Australian states (Victoria, community N =14; Queensland, N = 8, Western Australia, N = 8). The Healthy Neighbourhoods Study was designed to support valid epidemiological estimation of each of the 30 localities within the national sample. Localities were Statistical Local Areas (SLAs: based on Australian Bureau of Statistics divisions) stratified by community disadvantage (SES) and location in urban and non-urban areas, and were randomly selected from within these strata (Habib et al., 2014; Smith et al., 2014). Half were from regional areas and the other half from urban areas. The study was designed to represent the community disadvantage (SES) variation in Australian localities. Table 1 presents information on the demographic characteristics of participants. Grade 6 and 8 (N = 8,256) students were assessed in school classrooms utilising an online self-report survey, or where not available a paper copy.

All schools (Government, Independent, and Catholic) in the randomly selected areas were invited to participate with 53% of schools choosing to enter the study. All students within the schools were invited to participate, of the 15,666 consent forms distributed, 9,830 (63 %) were returned and of these 92% of grade 6 and 89% of grade 8 students’ parents actively consented to their child’s participation. Ethical approval for the study was provided by both the Murdoch Children’s Research Institute Human Research Ethics Committee, and relevant school and institutional ethics committees in each state.
Table 1

Sample Demographics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>48.0</td>
</tr>
<tr>
<td>Female</td>
<td>52.0</td>
</tr>
<tr>
<td><strong>Grade</strong></td>
<td></td>
</tr>
<tr>
<td>Grade 6</td>
<td>54.7</td>
</tr>
<tr>
<td>Grade 8</td>
<td>45.3</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>7.6</td>
</tr>
<tr>
<td>11</td>
<td>38.8</td>
</tr>
<tr>
<td>12</td>
<td>40.9</td>
</tr>
<tr>
<td>13</td>
<td>12.3</td>
</tr>
<tr>
<td>14</td>
<td>0.3</td>
</tr>
<tr>
<td><strong>Indigenous</strong></td>
<td></td>
</tr>
<tr>
<td>Indigenous Australian</td>
<td>3.2</td>
</tr>
<tr>
<td>Non-Indigenous Australian</td>
<td>96.8</td>
</tr>
<tr>
<td><strong>Child’s Country of Birth</strong></td>
<td></td>
</tr>
<tr>
<td>Australia</td>
<td>90.7</td>
</tr>
<tr>
<td>Outside Australia</td>
<td>9.3</td>
</tr>
<tr>
<td><strong>Mother’s Country of Birth</strong></td>
<td></td>
</tr>
<tr>
<td>Australia</td>
<td>73.3</td>
</tr>
<tr>
<td>Outside Australia</td>
<td>26.7</td>
</tr>
<tr>
<td><strong>Father’s Country of Birth</strong></td>
<td></td>
</tr>
<tr>
<td>Australia</td>
<td>71.9</td>
</tr>
<tr>
<td>Outside Australia</td>
<td>28.1</td>
</tr>
<tr>
<td><strong>Speak English at Home</strong></td>
<td></td>
</tr>
<tr>
<td>English only</td>
<td>88.4</td>
</tr>
<tr>
<td>Other language only</td>
<td>1.5</td>
</tr>
<tr>
<td>English and other language</td>
<td>10.1</td>
</tr>
<tr>
<td><strong>State</strong></td>
<td></td>
</tr>
<tr>
<td>Victoria</td>
<td>46.5</td>
</tr>
<tr>
<td>Queensland</td>
<td>30.3</td>
</tr>
<tr>
<td>Western Australia</td>
<td>23.3</td>
</tr>
<tr>
<td><strong>Locality</strong></td>
<td></td>
</tr>
<tr>
<td>Urban</td>
<td>50.1</td>
</tr>
<tr>
<td>Regional</td>
<td>49.9</td>
</tr>
<tr>
<td><strong>Community Disadvantage</strong></td>
<td></td>
</tr>
<tr>
<td>Quartile 1 (high)</td>
<td>28.4</td>
</tr>
<tr>
<td>Quartile 2</td>
<td>24.3</td>
</tr>
<tr>
<td>Quartile 3</td>
<td>23.2</td>
</tr>
<tr>
<td>Quartile 4 (low)</td>
<td>24.2</td>
</tr>
</tbody>
</table>

Measures. Depression was measured using the Short Mood and Feelings Questionnaire (SMFQ) - Child Version. The SMFQ has good internal reliability (Cronbach’s Alpha = 0.98) and has moderate correlations with the Children’s Depression Inventory ($r = 0.65$) and the Diagnostic Interview Schedule for Children ($r = 0.65$) measures of depression (Angold, Costello, Messer, & Pickles,
1995). Criterion validity (Angold et al., 1995; Kent, Vostanis, & Feehan, 1997; Thapar & McGuffin, 1998) and construct validity (Sharp, Goodyer, & Croudace, 2006) have been established for the SMFQ-Child Version. The SMFQ has been longitudinally predicted by prior exposure to family conflict (Habib et al., 2014), parent reports of child internalising and child reports of emotional control and family attachment (Toumbourou et al., 2014). Heerde et al. (2015) found it predicted future self-harm. Kremer et al. (2014) found it was associated with lack of physical activity.

The SMFQ is a 13 item self-report questionnaire developed to assess depressive symptoms in epidemiological studies based on the DSM-III criteria for depression (Angold et al., 1995). Participants responded to questions about their mood, feelings, and behaviours over the past 2 weeks. Participants were asked to respond on a three point Likert scale (i.e., “true” [2], “sometimes” [1], or “not true” [0]) to questions such as “I didn’t enjoy anything at all” and “I found it hard to think properly or concentrate”. Scores from the 13 questions were summed to provide a total symptom count, with a possible maximum of 26 and minimum of 0. As suggested by Angold et al. (1995) a cut-off score of 8 or more on the SMFQ was used to define the presence of elevated depressive symptoms.

Respondents completed the Communities that Care (CTC) youth survey, an instrument for assessing a wide range of risk and protective factors (Arthur et al., 2007). This included a demographics section that determined: gender, school grade, age, indigenous status, country of birth, mother and father’s country of birth, language/s spoken at home, whether their mother and father lived in their home, and mother’s and father’s employment status.
State of residence, SLA, and urban or regional area were determined at the study design stage. Communities were divided by SLA and the Australian Bureau of Statistics (ABS) Socio-economic Index for Areas (SEIFA) was used to determine community-level disadvantage (ABS, 2001). The Index applied was the Index of Relative Socio-economic Advantage/Disadvantage (IRSAD) which uses factors such as the proportion of households with low/high income; proportion of people with or without qualifications; and the proportion of people in a community in low skilled occupations to determine community-level disadvantage (ABS, 2001). A low score represents the most disadvantaged communities (quartile 1) and a high score represents the most advantaged communities (quartile 4).

The Communities that Care survey has been widely used in research examining risk and protective factors associated with a variety of internalizing and externalizing behaviors. These include drug, alcohol use, antisocial behavior, and depression (Bond, Thomas, Toumbourou, Patton, & Catalano, 2000; Hemphill et al., 2010; Jacka et al., 2010). The survey assessed risk and protective factors across four domains: individual/peer, family, school, and community. There were 38 scales used to measure items across these domains. Table 2 presents information on each of the scales used in the present study including number of items, example questions, and Cronbach’s alpha for the current sample.

Respondents’ scores on each item of a particular risk or protective factor were totaled. Respondents were determined to be ‘elevated” on a specific risk or protective factor if their total score was in the upper third of the distribution of scores for the total sample on that particular risk or protective factor (Arthur et al., 2007).
Table 2

Risk and Protective Factor Measures Included in Analyses

<table>
<thead>
<tr>
<th>Variable</th>
<th>Example question</th>
<th>No. of items</th>
<th>α</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Individual/peer-level factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional control (P)</td>
<td>“I know how to calm down when I am nervous.”</td>
<td>4</td>
<td>0.92</td>
</tr>
<tr>
<td>Belief in moral order (P)</td>
<td>“I think it’s OK to take something without asking if you can get away with it.”</td>
<td>4</td>
<td>0.91</td>
</tr>
<tr>
<td>Interaction with prosocial peers (P)</td>
<td>“In the past 12 months, how many of your four best friends have tried to do well in school.”</td>
<td>2</td>
<td>0.53</td>
</tr>
<tr>
<td>Religiosity (P)</td>
<td>“How often do you attend religious services or activities?”</td>
<td>1</td>
<td>N/A</td>
</tr>
<tr>
<td>Social skills (P)</td>
<td>“How are you at helping someone feel better when they are upset?”</td>
<td>6</td>
<td>0.90</td>
</tr>
<tr>
<td>Adaptive stress coping (P)</td>
<td>“When I have a problem I blame myself?”</td>
<td>4</td>
<td>0.90</td>
</tr>
<tr>
<td>Rewards for antisocial behaviour (R)</td>
<td>“What are the chances you would be seen as cool if you smoked cigarettes?”</td>
<td>4</td>
<td>0.96</td>
</tr>
<tr>
<td>Sensation seeking (R)</td>
<td>“How many times have you done crazy things, even if they are a little dangerous?”</td>
<td>3</td>
<td>0.76</td>
</tr>
<tr>
<td>Favourable attitude towards antisocial behaviour (R)</td>
<td>“How wrong do you think it is for someone your age to pick a fight with someone?”</td>
<td>5</td>
<td>0.94</td>
</tr>
<tr>
<td>Interaction with antisocial peers (R)</td>
<td>“In the past year, how many of your 4 best friends have been suspended from school?”</td>
<td>8</td>
<td>0.89</td>
</tr>
<tr>
<td>Favourable attitude towards drug use (R)</td>
<td>“How wrong do you think it is for someone your age to drink beer or wine regularly?”</td>
<td>5</td>
<td>0.93</td>
</tr>
<tr>
<td>Friends use of drugs (R)</td>
<td>“In the past year, how many of your 4 best friends have used marijuana?”</td>
<td>4</td>
<td>0.84</td>
</tr>
<tr>
<td>Rebelliousness (R)</td>
<td>“I like to see how much I can get away with.”</td>
<td>3</td>
<td>0.90</td>
</tr>
<tr>
<td>Perceived risk of drug use (R)</td>
<td>“How much do you think people risk harming themselves (physical or in other ways) if they use marijuana regularly?”</td>
<td>4</td>
<td>0.92</td>
</tr>
<tr>
<td>Health</td>
<td>“In general how would you describe your health?”</td>
<td>1</td>
<td>N/A</td>
</tr>
<tr>
<td>Dieting</td>
<td>“Do you try to avoid ‘fattening’ foods or foods with sugar in them?”</td>
<td>8</td>
<td>0.90</td>
</tr>
<tr>
<td>Physical Activity</td>
<td>“During the past 12 months on how many sports teams did you play at school?”</td>
<td>11</td>
<td>0.89</td>
</tr>
<tr>
<td><strong>Family-level factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parent attachment (P)</td>
<td>“Do you feel close to your father?”</td>
<td>4</td>
<td>0.95</td>
</tr>
<tr>
<td>Opportunities for prosocial involvement (P)</td>
<td>“If I had a personal problem I could ask my mum or dad for help.”</td>
<td>3</td>
<td>0.93</td>
</tr>
<tr>
<td>Rewards for prosocial involvement (P)</td>
<td>“My parents notice when I am doing a good job and let me know about it.”</td>
<td>4</td>
<td>0.86</td>
</tr>
<tr>
<td>Poor family management (R)</td>
<td>“The rules in my house are clear.”</td>
<td>9</td>
<td>0.97</td>
</tr>
<tr>
<td>Family conflict (R)</td>
<td>“People in my family have serious arguments.”</td>
<td>3</td>
<td>0.93</td>
</tr>
<tr>
<td>Parental over control (R)</td>
<td>“My parents try to control everything I do.”</td>
<td>2</td>
<td>0.95</td>
</tr>
<tr>
<td>Family history of antisocial behaviour (R)</td>
<td>“Have your brothers or sisters ever used marijuana?”</td>
<td>10</td>
<td>0.95</td>
</tr>
<tr>
<td>Parental attitudes favourable to antisocial behaviour (R)</td>
<td>“How wrong do your parents feel it would be for you to steal something worth more than $10?”</td>
<td>3</td>
<td>0.96</td>
</tr>
</tbody>
</table>
**Variables**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Example question</th>
<th>No. of items</th>
<th>( \alpha )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parental attitudes favourable to drug use (R)</td>
<td>“How wrong do your parents feel it would be for you to drink beer or wine regularly?”</td>
<td>4</td>
<td>0.98</td>
</tr>
</tbody>
</table>

**School-level factors**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Example question</th>
<th>No. of items</th>
<th>( \alpha )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opportunities for prosocial involvement (P)</td>
<td>“In my school students have lots of chances to help decide things like school activities and rules.”</td>
<td>5</td>
<td>0.78</td>
</tr>
<tr>
<td>Rewards for prosocial involvement (P)</td>
<td>“My teachers notice when I am doing a good job and let me know about it.”</td>
<td>4</td>
<td>0.64</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Example question</th>
<th>No. of items</th>
<th>( \alpha )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low commitment (R)</td>
<td>“How interesting are most of your school subjects to you?”</td>
<td>7</td>
<td>0.89</td>
</tr>
<tr>
<td>Academic failure (R)</td>
<td>“Putting them all together, what were your marks like last year?”</td>
<td>1</td>
<td>N/A</td>
</tr>
<tr>
<td>Victim of bullying (R)</td>
<td>“Have you been bullied recently (teased called names, had rumours spread about you)?”</td>
<td>1</td>
<td>N/A</td>
</tr>
</tbody>
</table>

**Community-level factors**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Example question</th>
<th>No. of items</th>
<th>( \alpha )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opportunities for prosocial involvement (P)</td>
<td>“Are the following activities available to people your age in your community?” E.g., Sports teams</td>
<td>5</td>
<td>0.95</td>
</tr>
<tr>
<td>Rewards for prosocial involvement (P)</td>
<td>“There are people in my neighbourhood that encourage me to do my best.”</td>
<td>3</td>
<td>0.97</td>
</tr>
<tr>
<td>Low neighbourhood attachment (R)</td>
<td>“If I had to move I would miss the neighbourhood I now live in.”</td>
<td>3</td>
<td>0.95</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Example question</th>
<th>No. of items</th>
<th>( \alpha )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Community safety (referred to as Community disorganization in HNS) (R)</td>
<td>“I feel safe in my neighbourhood.”</td>
<td>5</td>
<td>0.97</td>
</tr>
<tr>
<td>Laws and norms favourable to substance use (R)</td>
<td>“If a kid drank some alcohol in your neighbourhood, would he or she be caught by police?”</td>
<td>6</td>
<td>0.95</td>
</tr>
<tr>
<td>Perceived availability of drugs (R)</td>
<td>“If you wanted to get a drug like cocaine, heroin, LSD, or amphetamines, how easy would it be for you to get some?”</td>
<td>4</td>
<td>0.98</td>
</tr>
<tr>
<td>Transitions and mobility (R)</td>
<td>“Have you changed homes in the past year?”</td>
<td>4</td>
<td>0.56</td>
</tr>
</tbody>
</table>

\( \alpha = \text{Cronbach’s Alpha}, \text{No. = number}, P = \text{Protective factor}, R = \text{Risk factor}, \text{N/A = Not Applicable} \)

**Analyses.** All statistical analyses were performed using STATA, Version 12.1. All variables in the current study had less than 5% missing data with the exception of: community opportunities for prosocial involvement (7.69%); family conflict (6.53%); father’s employment (5.81%); parental overcontrol (5.37%); mother’s employment (5.22%); and community perceived availability of drugs (5.18%). Students completing all measures were included in the multivariate analyses (N = 7,070).
Multilevel logistic regression modelling was used to predict depressive symptoms with hierarchical models exploring contributions of predictors at different levels. A top-down approach was used to develop a model to best explain the variation in adolescent depression between SLAs. First, a null model (only random intercept, varying by area) was used to estimate the amount of variance between the 30 SLAs associated with depressive symptoms. Subsequent models were then developed by first introducing variables more distal to the individual, then introducing in stages variables more proximal to the individual. Model building was theoretically guided by the objective of establishing which factors explained SLA variations in adolescent depression symptoms.

Model 1 included all potential community-level risk and protective factors, ranging from those assessed at the state-level through to those subject to neighborhood variation. In Model 2 all school-level variables were added. In model 3 all family-level variables were added and finally in Model 4 all individual/peer-level variables were entered. Table 3 outlines the factors included in each model. At each stage of model-building, between and cross-level interactions, and random effects were examined. All non-significant variables were removed from each subsequent model. Gender interaction effects that were significant in univariate analyses were assessed in the multivariate analysis for each model (1-4) and those that maintained significant effects were retained in each of the final models. The log likelihood (LL) and the Akaike Information Criteria (AIC) and Bayesian Information Criteria (BIC) were used to assess model fit.
Results

Table 3 presents univariate odds ratios (ORs) for risk and protective factors associated with adolescent depressive symptoms. The majority of risk and protective factors were associated with adolescent depression. Univariate analysis assessed gender interaction effects and are presented in Table 3. Those predictors observed to show gender differences were entered as gender interaction terms in the multi-level analyses.

Table 4 presents a multi-level model of risk and protective factors, organized at the community, school, family, and individual/peer-level, that were significantly \( (p < .05) \) associated with adolescent depressive symptomology.

Table 5 presents model fit statistics for the models presented in Table 4. In the Null Model 7.1% of the variation in adolescent symptoms was explained by differences between SLAs.

In Model 1 (community-level risk and protective factors), eight factors maintained significant effects with ORs ranging from 1.77 (95% CI [1.50, 2.09]) for community disorganization (safety) to 1.22 (95% CI [1.05, 1.42]) for transitions and mobility. In this model community locality (urban/regional) and community rewards for prosocial involvement were not significant predictors of adolescent depression. In Model 1, significant community-level factors reduced the variance in depressive symptomology between SLAs (in the Null Model) by 89% and significantly improved model fit \( (\chi^2[14] \Delta \text{ LL} = 444, p < 0.001) \).

After school-level variables were added in Model 2 community transitions and mobility was no longer significant. Model 2 reduced the overall variance in SLAs by 77%, and adding school-level factors increased the variance explained by 87% compared to Model 1. Model 2 significantly improved model fit.
Table 3

Univariate Analyses of Risk and Protective Factors for Sub-clinical Depression

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariate OR (CI)</th>
<th>P-value</th>
<th>Univariate Gender Interaction Effects OR (CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
</tr>
<tr>
<td><strong>Individual/peer-level factors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender (R: referent male)</td>
<td>1.27 (1.15, 1.39)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade (R: referent grade 6)</td>
<td>1.10 (1.00, 1.22)</td>
<td>0.043</td>
<td>1.55 (1.29, 1.88)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Indigenous (P: referent non-Indigenous)</td>
<td>0.91 (0.69, 1.19)</td>
<td>0.485</td>
<td>1.07 (0.62, 1.84)</td>
<td>0.803</td>
</tr>
<tr>
<td>Country of Birth (P: referent born outside of Australia)</td>
<td>0.86 (0.73, 1.02)</td>
<td>0.081</td>
<td>1.04 (0.75, 1.46)</td>
<td>0.799</td>
</tr>
<tr>
<td>Language other than English only spoken at home (R: referent English only)</td>
<td>1.22 (0.84, 1.78)</td>
<td>0.299</td>
<td>1.24 (0.57, 2.71)</td>
<td>0.581</td>
</tr>
<tr>
<td>English and language other than English spoken at home (R: referent English only)</td>
<td>1.08 (0.91, 1.28)</td>
<td>0.356</td>
<td>1.20 (0.88, 1.64)</td>
<td>0.258</td>
</tr>
<tr>
<td>Health (R)</td>
<td>3.07 (2.55, 3.69)</td>
<td>&lt; 0.001</td>
<td>0.94 (0.65, 1.36)</td>
<td>0.737</td>
</tr>
<tr>
<td>Dieting (R)</td>
<td>2.34 (1.99, 2.74)</td>
<td>&lt; 0.001</td>
<td>1.66 (1.20, 2.29)</td>
<td>0.002</td>
</tr>
<tr>
<td>Physical Activity (P)</td>
<td>0.82 (0.71, 0.94)</td>
<td>0.005</td>
<td>0.80 (0.59, 1.07)</td>
<td>0.129</td>
</tr>
<tr>
<td>Emotional control (P)</td>
<td>0.34 (0.30, 0.38)</td>
<td>&lt; 0.001</td>
<td>0.76 (0.61, 0.95)</td>
<td>0.015</td>
</tr>
<tr>
<td>Belief in moral order (P)</td>
<td>0.43 (0.38, 0.49)</td>
<td>&lt; 0.001</td>
<td>0.66 (0.51, 0.86)</td>
<td>0.002</td>
</tr>
<tr>
<td>Interaction with prosocial peers (P)</td>
<td>0.58 (0.52, 0.66)</td>
<td>&lt; 0.001</td>
<td>1.03 (0.81, 1.31)</td>
<td>0.810</td>
</tr>
<tr>
<td>Religiosity (R)</td>
<td>1.04 (0.94, 1.15)</td>
<td>0.463</td>
<td>1.12 (0.91, 1.37)</td>
<td>0.276</td>
</tr>
<tr>
<td>Social skills (P)</td>
<td>0.62 (0.56, 0.69)</td>
<td>&lt; 0.001</td>
<td>0.94 (0.76, 1.17)</td>
<td>0.576</td>
</tr>
<tr>
<td>Adaptive stress coping (P)</td>
<td>0.15 (0.14, 0.18)</td>
<td>&lt; 0.001</td>
<td>0.74 (0.56, 0.96)</td>
<td>0.024</td>
</tr>
<tr>
<td>Rewards for antisocial behavior (R)</td>
<td>2.26 (2.00, 2.55)</td>
<td>&lt; 0.001</td>
<td>0.97 (0.76, 1.24)</td>
<td>0.795</td>
</tr>
<tr>
<td>Sensation seeking (R)</td>
<td>1.95 (1.73, 2.21)</td>
<td>&lt; 0.001</td>
<td>1.40 (1.08, 1.81)</td>
<td>0.011</td>
</tr>
<tr>
<td>Favorable attitude towards antisocial behavior (R)</td>
<td>2.12 (1.91, 2.36)</td>
<td>&lt; 0.001</td>
<td>1.29 (1.03, 1.60)</td>
<td>0.023</td>
</tr>
<tr>
<td>Interaction with antisocial peers (R)</td>
<td>4.44 (2.89, 6.82)</td>
<td>&lt; 0.001</td>
<td>1.28 (0.45, 3.63)</td>
<td>0.638</td>
</tr>
<tr>
<td>Favorable attitude towards drug use (R)</td>
<td>2.18 (1.94, 2.45)</td>
<td>&lt; 0.001</td>
<td>1.38 (1.09, 1.75)</td>
<td>0.008</td>
</tr>
<tr>
<td>Friends use of drugs (R)</td>
<td>2.43 (2.04, 2.89)</td>
<td>&lt; 0.001</td>
<td>1.43 (1.01, 2.02)</td>
<td>0.044</td>
</tr>
<tr>
<td>Rebelliousness (R)</td>
<td>3.62 (3.19, 4.10)</td>
<td>&lt; 0.001</td>
<td>1.17 (0.90, 1.51)</td>
<td>0.238</td>
</tr>
<tr>
<td>Perceived risk of drug use (R)</td>
<td>1.60 (1.43, 1.79)</td>
<td>&lt; 0.001</td>
<td>1.03 (0.82, 1.30)</td>
<td>0.775</td>
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<tr>
<td><strong>Family-level factors</strong></td>
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<td></td>
</tr>
<tr>
<td>Father born in Australia (P)</td>
<td>0.96 (0.85, 1.07)</td>
<td>0.432</td>
<td>1.20 (0.97, 1.48)</td>
<td>0.099</td>
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<tr>
<td>Mother born in Australia (P)</td>
<td>0.94 (0.84, 1.06)</td>
<td>0.327</td>
<td>1.09 (0.88, 1.35)</td>
<td>0.428</td>
</tr>
<tr>
<td>Father at home (P)</td>
<td>0.71 (0.64, 0.79)</td>
<td>&lt; 0.001</td>
<td>1.31 (1.06, 1.61)</td>
<td>0.014</td>
</tr>
</tbody>
</table>

(continued)
<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariate OR (C1)</th>
<th>P-value</th>
<th>Univariate Gender Interaction Effects OR (C1)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother at home (P)</td>
<td>0.65 (0.56, 0.76)</td>
<td>&lt; 0.001</td>
<td>0.99 (0.72, 1.35)</td>
<td>0.936</td>
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<tr>
<td>Father unemployed (R)</td>
<td>1.35 (1.10, 1.65)</td>
<td>0.003</td>
<td>0.68 (0.45, 1.01)</td>
<td>0.055</td>
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<tr>
<td>Father retired (R)</td>
<td>1.77 (1.23, 2.55)</td>
<td>0.002</td>
<td>0.46 (0.22, 0.96)</td>
<td>0.039</td>
</tr>
<tr>
<td>Father part-time employed (R)</td>
<td>1.20 (1.04, 1.37)</td>
<td>0.012</td>
<td>0.63 (0.48, 0.83)</td>
<td>0.001</td>
</tr>
<tr>
<td>Mother unemployed (P)</td>
<td>0.97 (0.86, 1.10)</td>
<td>0.650</td>
<td>0.91 (0.71, 1.16)</td>
<td>0.446</td>
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<tr>
<td>Mother retired (R)</td>
<td>1.11 (0.75, 1.64)</td>
<td>0.604</td>
<td>0.54 (0.25, 1.21)</td>
<td>0.134</td>
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<tr>
<td>Mother part-time employed (P)</td>
<td>0.82 (0.74, 0.92)</td>
<td>0.001</td>
<td>0.96 (0.77, 1.21)</td>
<td>0.745</td>
</tr>
<tr>
<td>Parent attachment (P)</td>
<td>0.32 (0.27, 0.38)</td>
<td>&lt; 0.001</td>
<td>0.52 (0.37, 0.73)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Opportunities for prosocial involvement (P)</td>
<td>0.39 (0.35, 0.44)</td>
<td>&lt; 0.001</td>
<td>0.67 (0.53, 0.83)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Rewards for prosocial involvement (P)</td>
<td>0.39 (0.35, 0.44)</td>
<td>&lt; 0.001</td>
<td>0.67 (0.53, 0.85)</td>
<td>0.001</td>
</tr>
<tr>
<td>Poor family management (R)</td>
<td>2.03 (1.78, 2.32)</td>
<td>&lt; 0.001</td>
<td>1.56 (1.19, 2.04)</td>
<td>0.001</td>
</tr>
<tr>
<td>Family conflict (R)</td>
<td>3.55 (3.20, 3.94)</td>
<td>&lt; 0.001</td>
<td>1.08 (0.88, 1.33)</td>
<td>0.453</td>
</tr>
<tr>
<td>Parental over control (R)</td>
<td>1.96 (1.78, 2.16)</td>
<td>&lt; 0.001</td>
<td>0.91 (0.75, 1.11)</td>
<td>0.347</td>
</tr>
<tr>
<td>Family history of antisocial behavior (R)</td>
<td>2.18 (1.94, 2.46)</td>
<td>&lt; 0.001</td>
<td>1.25 (0.99, 1.60)</td>
<td>0.066</td>
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<tr>
<td>Parental attitudes favorable to antisocial behavior (R)</td>
<td>1.72 (1.54, 1.93)</td>
<td>&lt; 0.001</td>
<td>1.28 (1.02, 1.60)</td>
<td>0.036</td>
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<tr>
<td>Parental attitudes favorable to drug use (R)</td>
<td>1.69 (1.50, 1.90)</td>
<td>&lt; 0.001</td>
<td>1.24 (0.98, 1.56)</td>
<td>0.076</td>
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<tr>
<td><strong>School-level factors</strong></td>
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<tr>
<td>Opportunities for prosocial involvement (P)</td>
<td>0.70 (0.63, 0.77)</td>
<td>&lt; 0.001</td>
<td>0.92 (0.75, 1.13)</td>
<td>0.422</td>
</tr>
<tr>
<td>Rewards for prosocial involvement (P)</td>
<td>0.51 (0.46, 0.57)</td>
<td>&lt; 0.001</td>
<td>0.76 (0.62, 0.93)</td>
<td>0.007</td>
</tr>
<tr>
<td>Low commitment (R)</td>
<td>2.48 (2.25, 2.73)</td>
<td>&lt; 0.001</td>
<td>1.17 (0.96, 1.42)</td>
<td>0.119</td>
</tr>
<tr>
<td>Academic failure (R)</td>
<td>1.93 (1.70, 2.19)</td>
<td>&lt; 0.001</td>
<td>0.96 (0.74, 1.24)</td>
<td>0.745</td>
</tr>
<tr>
<td>Victim of bullying (R)</td>
<td>4.48 (3.94, 5.11)</td>
<td>&lt; 0.001</td>
<td>1.21 (0.93, 1.57)</td>
<td>0.149</td>
</tr>
<tr>
<td><strong>Community-level factors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>State (R: referent Victoria)</td>
<td></td>
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</tr>
<tr>
<td>Queensland</td>
<td>1.55 (1.27, 1.89)</td>
<td>&lt; 0.001</td>
<td>0.88 (0.71, 1.10)</td>
<td>0.262</td>
</tr>
<tr>
<td>Western Australia</td>
<td>1.22 (0.99, 1.49)</td>
<td>0.058</td>
<td>0.77 (0.60, 0.98)</td>
<td>0.031</td>
</tr>
<tr>
<td>Locality</td>
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<tr>
<td>Regional (P: referent urban)</td>
<td>0.91 (0.74, 1.12)</td>
<td>0.392</td>
<td>0.90 (0.75, 1.09)</td>
<td>0.299</td>
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<tr>
<td>Community Disadvantage (R: referent quartile 4 [high])</td>
<td></td>
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</tr>
<tr>
<td>Quartile 1 (low)</td>
<td>1.42 (1.10, 1.83)</td>
<td>0.007</td>
<td>1.12 (0.85, 1.46)</td>
<td>0.423</td>
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<tr>
<td>Quartile 2</td>
<td>1.39 (1.07, 1.81)</td>
<td>0.014</td>
<td>0.89 (0.68, 1.18)</td>
<td>0.418</td>
</tr>
<tr>
<td>Quartile 3</td>
<td>1.40 (1.08, 1.81)</td>
<td>0.012</td>
<td>1.06 (0.80, 1.41)</td>
<td>0.661</td>
</tr>
<tr>
<td>Opportunities for prosocial involvement (P)</td>
<td>0.64 (0.57, 0.72)</td>
<td>&lt; 0.001</td>
<td>0.85 (0.68, 1.08)</td>
<td>0.180</td>
</tr>
<tr>
<td>Variable</td>
<td>Univariate OR (CI)</td>
<td>P-value</td>
<td>Univariate Gender Interaction Effects OR (CI)</td>
<td>P-value</td>
</tr>
<tr>
<td>----------------------------------------------------</td>
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<td>-----------------------------------------------</td>
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</tr>
<tr>
<td>Rewards for prosocial involvement (P)</td>
<td>0.76 (0.69, 0.84)</td>
<td>&lt; 0.001</td>
<td>0.84 (0.70, 1.02)</td>
<td>0.084</td>
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<tr>
<td>Low neighbourhood attachment (R)</td>
<td>2.16 (1.94, 2.40)</td>
<td>&lt; 0.001</td>
<td>1.32 (1.06, 1.63)</td>
<td>0.012</td>
</tr>
<tr>
<td>Community disorganisation (R)</td>
<td>2.52 (2.18, 2.90)</td>
<td>&lt; 0.001</td>
<td>1.15 (0.86, 1.53)</td>
<td>0.345</td>
</tr>
<tr>
<td>Laws and norms favourable to substance use (R)</td>
<td>1.80 (1.62, 2.00)</td>
<td>&lt; 0.001</td>
<td>1.17 (0.95, 1.45)</td>
<td>0.129</td>
</tr>
<tr>
<td>Perceived availability of drugs (R)</td>
<td>2.32 (2.03, 2.64)</td>
<td>&lt; 0.001</td>
<td>1.57 (1.20, 2.05)</td>
<td>0.001</td>
</tr>
<tr>
<td>Transitions and mobility (R)</td>
<td>1.48 (1.29, 1.70)</td>
<td>&lt; 0.001</td>
<td>0.99 (0.75, 1.29)</td>
<td>0.930</td>
</tr>
</tbody>
</table>

P = Protective factor, R = Risk factor, OR = Odds Ratio, CI = Confidence Interval. Univariate Gender Interaction Effects – Referent is Male

compared to the null model ($\chi^2 [16] \Delta LL = 1089, p < 0.001$) and Model 1 ($\chi^2 [2] \Delta LL = 643, p < 0.001$).

After family-level variables were added in Model 3 the community-level risk factors opportunities for pro-social involvement, laws and norms favorable to drug use, perceived availability of drugs, and community disadvantage were no longer significant. Model 3 reduced the variance between SLAs by 88% and reduced a further 49% of variance compared to Model 2. Model 3 significantly improved model fit compared to the Null Model ($\chi^2 [22] \Delta LL = 1456, p < 0.001$) and Model 2 ($\chi^2 [6] \Delta LL = 367, p < 0.001$).

After adding individual/peer-level variables the school-level factor academic failure was also no longer significant. The final model accounted for 91% of the variance between SLAs when compared to the null model and reduced a further 25% of the variance compared to Model 3. Model 4 significantly improved model fit compared to the null model ($\chi^2 [30] \Delta LL = 2086, p < 0.001$) and Model 3 ($\chi^2 [8] \Delta LL = 629, p < 0.001$). In the final model State and community safety were the only community-level predictors that maintained a significant association with adolescent depressive symptomology.
Table 4

Multi-level Model of (N = 7,070) Risk and Protective Factors for Sub-clinical Depression

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1 Community-level factors OR (CI)</th>
<th>P-value</th>
<th>Model 2 School-level factors OR (CI)</th>
<th>P-value</th>
<th>Model 3 Family-level factors OR (CI)</th>
<th>P-value</th>
<th>Model 4 Individual/peer-level factors OR (CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Community-level factors</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Opportunities for prosocial involvement (P)</td>
<td>0.77 (0.68, 0.87)</td>
<td>&lt; 0.001</td>
<td>0.82 (0.72, 0.94)</td>
<td>0.003</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Low neighbourhood attachment (R)</td>
<td>1.38 (1.16, 1.64)</td>
<td>&lt; 0.001</td>
<td>1.21 (1.01, 1.46)</td>
<td>0.039</td>
<td>1.16 (0.96, 1.40)</td>
<td>0.127</td>
<td>1.05 (0.86, 1.28)</td>
<td>0.617</td>
</tr>
<tr>
<td>Low neighbourhood attachment (R) # Gender</td>
<td>1.35 (1.06, 1.70)</td>
<td>0.013</td>
<td>1.41 (1.10, 1.81)</td>
<td>0.006</td>
<td>1.50 (1.16, 1.93)</td>
<td>0.002</td>
<td>1.45 (1.11, 1.89)</td>
<td>0.006</td>
</tr>
<tr>
<td>Community disorganisation (R)</td>
<td>1.77 (1.50, 2.09)</td>
<td>&lt; 0.001</td>
<td>1.65 (1.38, 1.97)</td>
<td>&lt; 0.001</td>
<td>1.43 (1.20, 1.71)</td>
<td>&lt; 0.001</td>
<td>1.26 (1.04, 1.52)</td>
<td>0.018</td>
</tr>
<tr>
<td>Laws and norms favourable to substance use (R)</td>
<td>1.37 (1.21, 1.54)</td>
<td>&lt; 0.001</td>
<td>1.24 (1.09, 1.40)</td>
<td>0.001</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Perceived availability of drugs (R)</td>
<td>1.34 (1.09, 1.66)</td>
<td>0.006</td>
<td>1.20 (0.96, 1.50)</td>
<td>0.104</td>
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<tr>
<td>Perceived availability of drugs (R) # Gender</td>
<td>1.47 (1.10, 1.96)</td>
<td>0.010</td>
<td>1.47 (1.08, 2.00)</td>
<td>0.013</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transitions and mobility (R)</td>
<td>1.22 (1.05, 1.42)</td>
<td>0.011</td>
<td></td>
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<tr>
<td>Community Disadvantage</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quartile 1 (high)</td>
<td>1.22 (1.02, 1.45)</td>
<td>0.026</td>
<td>1.25 (1.03, 1.53)</td>
<td>0.026</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quartile 2</td>
<td>1.16 (0.97, 1.38)</td>
<td>0.115</td>
<td>1.12 (0.91, 1.38)</td>
<td>0.275</td>
<td></td>
<td></td>
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<tr>
<td>Quartile 3</td>
<td>1.23 (1.03, 1.48)</td>
<td>0.023</td>
<td>1.16 (0.94, 1.42)</td>
<td>0.166</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>State</td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Queensland</td>
<td>1.57 (1.35, 1.82)</td>
<td>&lt; 0.001</td>
<td>1.64 (1.39, 1.94)</td>
<td>&lt; 0.001</td>
<td>1.50 (1.28, 1.75)</td>
<td>&lt; 0.001</td>
<td>1.53 (1.31, 1.79)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Western Australia</td>
<td>1.24 (1.06, 1.45)</td>
<td>0.008</td>
<td>1.29 (1.08, 1.54)</td>
<td>0.006</td>
<td>1.22 (1.04, 1.44)</td>
<td>0.017</td>
<td>1.22 (1.03, 1.45)</td>
<td>0.018</td>
</tr>
<tr>
<td>School-level factors</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Low commitment (R)</td>
<td>2.15 (1.92, 2.42)</td>
<td>&lt; 0.001</td>
<td>2.12 (1.89, 2.39)</td>
<td>0.000</td>
<td>1.72 (1.52, 1.96)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(continued)
## Community Factors and Adolescent Depression

### Variable Model 1 Community-level factors OR (CI) P-value Model 2 School-level factors OR (CI) P-value Model 3 Family-level factors OR (CI) P-value Model 4 Individual/peer-level factors OR (CI) P-value

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1 Community-level factors OR (CI)</th>
<th>P-value</th>
<th>Model 2 School-level factors OR (CI)</th>
<th>P-value</th>
<th>Model 3 Family-level factors OR (CI)</th>
<th>P-value</th>
<th>Model 4 Individual/peer-level factors OR (CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Academic failure (R)</td>
<td>1.38 (1.19, 1.61)</td>
<td>&lt; 0.001</td>
<td>1.32 (1.13, 1.54)</td>
<td>&lt; 0.001</td>
<td>1.30 (1.12, 1.51)</td>
<td>&lt; 0.001</td>
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<td></td>
</tr>
<tr>
<td>Victim of bullying (R)</td>
<td>4.47 (3.86, 5.17)</td>
<td>&lt; 0.001</td>
<td>4.12 (3.55, 4.80)</td>
<td>&lt; 0.001</td>
<td>3.65 (3.11, 4.28)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
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<tr>
<td>Family-level factors</td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Father at home</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unemployed</td>
<td>0.75 (0.61, 0.93)</td>
<td>0.007</td>
<td>0.69 (0.55, 0.85)</td>
<td>0.001</td>
<td>0.67 (0.51, 0.89)</td>
<td>0.123</td>
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<td></td>
</tr>
<tr>
<td>Retired</td>
<td>2.05 (1.10, 3.80)</td>
<td>0.023</td>
<td>1.70 (0.85, 3.38)</td>
<td>0.132</td>
<td>1.60 (1.00, 2.56)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Part-time</td>
<td>1.51 (1.19, 1.91)</td>
<td>0.001</td>
<td>1.60 (1.25, 2.05)</td>
<td>&lt; 0.001</td>
<td>1.57 (1.17, 2.07)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Father’s employment # Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unemployed # Gender</td>
<td>0.68 (0.43, 1.10)</td>
<td>0.114</td>
<td>0.67 (0.40, 1.11)</td>
<td>0.123</td>
<td>0.67 (0.41, 1.11)</td>
<td>0.123</td>
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<td></td>
</tr>
<tr>
<td>Retired # Gender</td>
<td>0.47 (0.19, 1.14)</td>
<td>0.096</td>
<td>0.58 (0.22, 1.57)</td>
<td>0.287</td>
<td>0.58 (0.22, 1.57)</td>
<td>0.287</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Part-time # Gender</td>
<td>0.55 (0.40, 0.77)</td>
<td>&lt; 0.001</td>
<td>0.54 (0.38, 0.75)</td>
<td>&lt; 0.001</td>
<td>0.54 (0.38, 0.75)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family conflict (R)</td>
<td>2.52 (2.23, 2.84)</td>
<td>&lt; 0.001</td>
<td>2.18 (1.91, 2.48)</td>
<td>&lt; 0.001</td>
<td>2.18 (1.91, 2.48)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parental over control (R)</td>
<td>1.48 (1.32, 1.67)</td>
<td>&lt; 0.001</td>
<td>1.42 (1.26, 1.62)</td>
<td>&lt; 0.001</td>
<td>1.42 (1.26, 1.62)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family history of antisocial behaviour (R)</td>
<td>1.53 (1.32, 1.77)</td>
<td>&lt; 0.001</td>
<td>1.37 (1.17, 1.60)</td>
<td>&lt; 0.001</td>
<td>1.37 (1.17, 1.60)</td>
<td>&lt; 0.001</td>
<td></td>
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<tr>
<td>Individual/peer-level factors</td>
<td></td>
<td></td>
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<tr>
<td>School Grade</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Indigenous</td>
<td>0.81 (0.71, 0.92)</td>
<td>0.001</td>
<td>0.81 (0.71, 0.92)</td>
<td>0.001</td>
<td>0.81 (0.71, 0.92)</td>
<td>0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional control (P)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional control (P)</td>
<td>0.67 (0.58, 0.77)</td>
<td>&lt; 0.001</td>
<td>0.67 (0.58, 0.77)</td>
<td>&lt; 0.001</td>
<td>0.67 (0.58, 0.77)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interaction with prosocial peers (P)</td>
<td>0.79 (0.68, 0.92)</td>
<td>0.003</td>
<td></td>
<td></td>
<td>0.79 (0.68, 0.92)</td>
<td>0.003</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adaptive stress coping (P)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adaptive stress coping (P)</td>
<td>0.25 (0.21, 0.30)</td>
<td>&lt; 0.001</td>
<td>0.25 (0.21, 0.30)</td>
<td>&lt; 0.001</td>
<td>0.25 (0.21, 0.30)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rewards for antisocial behaviour (R)</td>
<td>1.50 (1.26, 1.76)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
<td>1.50 (1.26, 1.76)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rebelliousness (R)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rebelliousness (R)</td>
<td>1.91 (1.06, 2.26)</td>
<td>&lt; 0.001</td>
<td>1.91 (1.06, 2.26)</td>
<td>&lt; 0.001</td>
<td>1.91 (1.06, 2.26)</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Health</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Health</td>
<td>1.39 (1.09, 1.76)</td>
<td>0.007</td>
<td>1.39 (1.09, 1.76)</td>
<td>0.007</td>
<td>1.39 (1.09, 1.76)</td>
<td>0.007</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

# Gender = Gender Interaction Effect (Referent Male), OR = Odd Ratio, CI = Confidence Interval, *Age OR is measured as a linear trend *In multivariate analysis N = 7,070 *Univariate and Multivariate analyses are adjusted for community and school clustering.
Table 5

Model Fit Statistics for Models Presented in Table 4

<table>
<thead>
<tr>
<th>Model fit</th>
<th>Null</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variance</td>
<td>0.071</td>
<td>0.009</td>
<td>0.016</td>
<td>0.008</td>
<td>0.006</td>
</tr>
<tr>
<td>Difference in variance to null model</td>
<td>88%</td>
<td>77%</td>
<td>88%</td>
<td>91%</td>
<td></td>
</tr>
<tr>
<td>Difference in variance to previous model</td>
<td>-87%</td>
<td>49%</td>
<td>25%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LL</td>
<td>-4461</td>
<td>-4238</td>
<td>-3916</td>
<td>-3418</td>
<td>-3732</td>
</tr>
<tr>
<td>AIC</td>
<td>8925</td>
<td>8509</td>
<td>7868</td>
<td>6900</td>
<td>7513</td>
</tr>
<tr>
<td>BIC</td>
<td>8939</td>
<td>8619</td>
<td>7991</td>
<td>7119</td>
<td>7677</td>
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<tr>
<td>DF</td>
<td>2</td>
<td>16</td>
<td>18</td>
<td>32</td>
<td>24</td>
</tr>
</tbody>
</table>

LL = Log Likelihood, AIC = Akaike Information Criteria, BIC = Bayesian Information Criteria

Gender interaction effects were identified for the community-level factors low community attachment and perceived availability of drugs in Model 1. After adding family-level factors gender interaction effects for the community-level factor perceived availability of drugs was no longer significant. In Model 4 gender interaction effects were maintained for community attachment, father lives at home, and fathers’ employment. Males whose fathers worked part-time were less likely to report depressive symptoms (OR = 0.54, 95% CI [0.38, 0.76]).

Males whose fathers lived with them were more likely to report depression (OR = 1.42, 95% CI [1.06, 1.90]). Whilst females with low community attachment were significantly more likely to report depressive symptoms (OR = 1.45, 95% CI [1.11, 1.89]), community attachment was not significantly associated with depressive symptoms for the total population.

Discussion

Summary of findings. Our prior analyses identified 33.1% (95% CI [32.1, 34.2]) of Australian adolescents reported depressive symptoms (Stirling et al., under submission). The present analyses revealed 7.1% of the variation in adolescent symptoms was explained by differences between SLAs. Our analyses
sought to identify modifiable factors that accounted for SLA (locality) differences. Community-level variables explained a large component (88%) of the locality variation in adolescent depressive symptoms. With the exception of community safety and State, these effects were not maintained in the multivariate models accounting for school, family, and individual/peer influences. These findings suggest that community-level factors may be important to target in locality (place-based) strategies to prevent adolescent depression. Multi-level interventions may be required that account for underlying influences that vary between localities. The findings support the importance of designing and evaluating locality interventions that utilize a range of data through local, state, and national agencies to identify community-level issues and develop appropriate responses (e.g., Hawkins et al., 2009).

An important finding in the current study was that State and specifically residence in Queensland and Western Australia were significantly associated with increased adolescent depressive symptoms even after controlling for a wide range of factors at the community, school, family, and individual/peer level. These findings suggest that State policy domains influence adolescent mental health. Access to services and distance to services may impact on adolescent depressive symptoms particularly in states with a large number of remote communities such as Western Australia and Queensland.

As distinct from Queensland, both Victoria and Western Australia have made large investments in child and adolescent health research institutes in recent decades through the Western Australian Telethon Kids Institute (www.childhealthresearch.org.au) and the Murdoch Children’s Research Institute in Victoria (www.mcri.edu.au). These research institutes have developed strong
partnerships with the health and education sectors, with a range of programs emerging from these centers showing improvements in child and adolescent mental health (Buttigieg et al., 2015; Cross et al., 2011; Toumbourou & Gregg, 2002). Findings suggest that State policy and unmeasured state-level factors in the present study should be further investigated to better understand state differences affecting adolescent depression.

Similar to findings of prior meta-analyses (Fowler et al., 2009; Stirling et al., 2015) community safety was significantly associated with adolescent depressive symptoms even when adjusting for a wide range of covariates. Consistent with the findings of prior meta-analysis, the current study also identified being a victim of bullying (Ttofi et al., 2011) and family conflict (Yap et al., 2014) were significantly associated with increased depressive symptoms. When accounting for a wide range of covariates being a victim of bullying and family conflict were the strongest predictors of adolescent depressive symptoms. Thus violence and safety at the community, family, and school/peer-level are important risk factors to consider in place-based approaches to preventing depression. Further research exploring developmental pathways and trajectories of the impact of violence at the community/school/family/ and individual-peer level is warranted.

The community-level factors community opportunities for pro-social involvement, community attachment, community safety, community laws and norms favorable to drug use, perceived availability of drugs, transitions and mobility, community disadvantage, and State accounted for 88% of the variance in depression between SLAs. The majority of community-level factors (all except State and community safety) were explained by other predictors of adolescent
depressive symptomology. Findings suggested that the effect of community transitions and mobility were explained by factors at the school-level; opportunities for pro-social involvement, community attachment, laws and norms favorable to drug use, perceived availability of drugs, and community disadvantage by family-level factors.

Findings from the present analysis reveal that community disadvantage was associated with adolescent depression even after controlling for other demographic factors (Model 1). However, these effects were not maintained after adjustment for school and family factors (Models 2 to 3). These findings are consistent with those from a recent meta-analysis (Stirling et al., 2015) that suggested the influence of community disadvantage on adolescent depression was likely influenced by other predictors of depression. These findings support Bronfenbrenner’s assertion that the effects of children’s exposure to community-level risk factors arise due to interactions with the school, family, and individual/peer contexts that are embedded within communities.

There is some prior research that supports the present findings. Student transitions and mobility have been previously found to have adverse effects on school-level risk factors (Bradshaw, Sawyer, & O’Brennan, 2009). The finding that laws and norms favorable to drug use are related to family-level factors was observed in a study that found parents were more likely to supply alcohol to underage adolescents if they lived in neighborhoods with high densities of alcohol sales outlets (Rowland, Toumbourou, Satyen, Livingston, & Williams, 2014). Future studies should more directly seek to investigate theoretical mechanisms to provide a firmer basis for developing preventative interventions within ecological community systems.
The final model identified 18 factors (community, school, family, and individual-peer-level factors) that explained 91% of the variance in adolescent depression between communities. Overall, findings from the current study support the view that multi-level place-based prevention approaches targeting predictors across multiple settings will likely improve the effectiveness of current preventative interventions (Jaycox et al., 2009; American Academy of Child and Adolescent Psychiatry, 2007; Spence & Shortt, 2007).

In the final model gender interaction effects were identified for community attachment, father living at home, and father’s employment. For example males with father’s in part-time employment were less likely to report depressive symptoms than females with fathers in part-time employment. There is limited research examining the gender differences in the relationship between father’s employment and adolescent depressive symptoms. Whilst some studies have not identified a direct association between father’s employment and male adolescent depressive symptoms (Sleskova, Tuinstra, et al., 2006), others have suggested that father’s long term unemployment is associated with negative outcomes for male adolescents (Sleskova, Salonna, et al., 2006). There is also limited understanding of the processes that influence the relationship between father’s employment status and adolescent depression. Low perception of self, family cohesion, and perception of future have been identified to account for the relationship between father’s non-employment and adolescent mental health (Bacikova-Sleskova, Jozef, & Olga, 2015). However, gender differences in these processes have not been examined. Future research should explore gender differences.

**Limitations and future research.** In drawing conclusions from the current findings it is important to consider both limitations and strengths. First, the
study is limited in being non experimental and cross-sectional and hence not
designed to make strong assertions regarding causal associations. Second, the
current sample is a community sample of school students in consenting schools
and families and does not include the minority of adolescents who do not attend
school. A range of prior studies have reported on clinical samples. Quartiles of
disadvantage in the current sample match Australia-wide rates, however whilst the
study controls for employment status it does not control for other family-level
SES variables such as parental education and income. Third, whilst the current
study addressed an important criticism of current research by including a large
number of risk and protective factors (Shanahan, Copeland, Costello, & Angold,
2008), there may be important factors that were not included. Future research
should assess other plausible risk factors including family history of mental
illness, and adolescent genetic risks for depression. Fourth, whilst self-report
measures are widely used in epidemiological research (Arthur et al., 2007; Bond
et al., 2000; Hemphill et al., 2010; Jacka et al., 2010) they may be influenced by
recall bias and social desirability. Future research should compare adolescent self-
reports with clinical, school staff behavioral observations, and parent-report
measures. Fifth, it would be worthwhile identifying valid population-level
measures of community-level variables such as crime statistics (community
safety) that can be used to identify high-risk communities. Future research is
needed to compare aggregated self-report measures of known community-level
predictors (e.g., feelings of safety) with community-level measures of the same
construct (e.g., local area police statistics) to establish the validity of using readily
available population-level measures to identify areas for intervention or whether a
combination of both is needed (Raudenbush & Sampson, 1999). Strengths of the
present study include the large and well-designed epidemiological sample and the incorporation of a wider range of risk factors relative to prior studies.

**Conclusion.** Findings reveal that 7% of variation in adolescent depressive symptoms was locality-based, suggesting the importance of nation-wide and state-level responses, but also the feasibility of place-based depression prevention. In line with ecological theories, locality variation was explained by community, school, family, and individual-level factors suggesting the importance of multi-level approaches in place-based interventions. Future studies should investigate theoretical mechanisms that explain risk factors within community systems to provide a firmer basis for developing preventative interventions. Research should continue to explore potential gender differences in these models.
References


*Improving the lives of young Victorians in our community: A survey of risk and protective factors*: Centre for Adolescent Health Melbourne, VIC.


American Psychologist, 34(10), 844. doi:http://dx.doi.org/10.1037/0003-066X.34.10.844


578. doi:10.1037//0021-843X.111.4.578


Chapter 6: Discussion

Discussion of Findings

The chapter that follows presents a discussion of findings. It begins with an integrated overview of the findings from the three studies. Limitations of the current thesis and opportunities for future research are then outlined. The discussion goes on to consider the implications and conclusions for the current thesis.

Summary of the Findings of the Current Thesis

Depression is the leading burden of disease due to disability, decreased work productivity, and increased use of health services (Simon, 2003). Depression emerges during adolescence in the context of risk and protective factors at the individual/peer, family, school, and community-level (Jackson & Nuttall, 2001; Kessler et al., 2012). Current evidence supports predictors of adolescent depression at the individual, family, and school-level. However, research examining community-level predictors and interventions is less organised. Given their potential to influence large aggregate populations, identifying community-level influences associated with depression in school-aged children may provide directions to advance cost-effective opportunities for population-level intervention. The purpose of the current thesis was to establish whether community-level variables account for an important share of the potentially modifiable factors that predict adolescent depression.

The current thesis began with an introduction of the key terms relevant to child and adolescent depression. The prevalence, course, onset, co-morbidities, and impacts associated with child and adolescent depression were then considered. The introduction highlighted the negative long-term trajectory of
depression and identified an opportunity to intervene during early adolescence, when psychopathology first emerges (Klein et al., 2013). Elevated depression symptoms (sub-clinical and clinical) in adolescence is an important predictor of the later development of depression and as such reducing the prevalence of depression symptoms during early adolescence was argued to be of potential value in prevention efforts (Lewinsohn et al., 1994).

Chapter 2 provided a rationale and framework for researching the possibility of preventative approaches focussed on community-level variables during the adolescent period of development. It was argued that efforts to reduce the prevalence of depression must focus on reducing the incidence of depression by preventing its onset. Developmental psychopathology indicated adolescence as a critical period for preventing mental health problems. Identifying and targeting community-level risk and protective factors offers a cost-effective opportunity for reducing adolescent depression (Beaglehole & Bonita, 2009; Keleher & Armstrong, 2005; Marmot & Wilkinson, 1999; Mihalopoulos & Chatterton, 2014; Muñoz et al., 2010).

Adolescents develop in the context of their environments and Bronfenbrenner’s Ecological Systems Theory provided a framework for organising the predictors of depression at the individual/peer, family, school, and community-level. Current evidence indicated a large number of risk and protective factors at the individual, school, and family level (Cairns et al., 2014; Davis et al., 1999; Grant et al., 2003; McLeod et al., 2007; Yap et al., 2014). However, research examining community factors associated with adolescent depression was less organised. Further, reviews examining preventative approaches suggested that current preventative interventions are largely focussed
on individual-level factors and none of the included interventions targeted factors at the community-level (AACAP, 2007; Horowitz & Garber, 2006; Spence & Shortt, 2007). The current thesis aimed to establish whether community-level variables accounted for an important share of the potentially modifiable factors that predict adolescent depression.

Study 1 presented the first systematic review and meta-analyses of prior studies that have examined the role of community risk and protective factors and interventions targeting community-level factors for depression in school-aged children (4-18 years). The systematic review and meta-analyses identified that community safety and community minority/ethnicity and discrimination were significantly associated with child depression. Although significant effects were not identified for studies of community disadvantage and community connectedness or for the 3 intervention studies examining effects of interventions alleviating disadvantage, effect sizes were of a similar magnitude to those that were significant in the meta-analyses.

Findings from the systematic review and meta-analyses identified that internationally a number of community-level factors have been directly and indirectly associated with child depression. However none of the included studies examined depression at sub-clinical levels, despite a growing body of evidence supporting the association between sub-clinical depression and a range of negative outcomes including later depression. Further only one study used an Australian sample. Australian policy advocates (McDermott et al., 2010) have argued that to advance prevention efforts in Australia, research examining adolescent depression symptoms at sub-clinical levels using Australian samples is needed. As such Study 2 and Study 3 aimed to build on the findings from Study 1
by examining prevalence and predictors of elevated depression symptoms (sub-clinical and clinical) using a large representative sample of Australian adolescents.

In Chapter 4, Study 2 aimed to quantify the prevalence of elevated depression symptoms (sub-clinical and clinical) and to identify the prevalence variation between statistical local areas (SLAs) using a large nationally representative sample of Australian adolescents (10-14 years). Study 2 also aimed to identify whether community-level disadvantage was associated with depression in Australian adolescents after controlling for a number of covariates including family-level SES.

Findings supported both of the study hypotheses. First, elevated depressive symptomology was evident for one third of the sample; around double the rate identified by Bond et al. (2005). Study 2 identified that the prevalence of sub-clinical and clinical depressive symptoms in Australian adolescents (10-14 years) varied between localities (SLAs) with 6.9% of the variance in depressive symptoms attributed to locality. Second, prevalence varied significantly across Australian communities with higher rates evident for communities with higher disadvantage, after controlling for covariates.

Multivariate analysis suggested that depressive symptomology was associated with a variety of demographic factors: household composition; parents’ employment status; higher community disadvantage; and State. Gender interaction effects were identified for school grade and father’s employment.

Findings from Study 1 and 2 supported the role locality plays in the development of adolescent depressive symptoms. Study 2 showed that although the effect of community disadvantage on adolescent depression was evident, there
was considerable variation between communities within each disadvantage (SES) strata. Thus the combined findings of Study 1 and Study 2 suggested that the impact of community-level factors may be influenced by other risk and protective factors associated with adolescent depression. Analyses that examined a wide range of risk and protective factors was needed to better understand the association between community-level factors and adolescent depression. Further, to determine the most cost-effective opportunities for intervention it was important to understand how much of the variance in adolescent depression could be explained by factors at the community, school, family, and individual/peer level. Identifying these differences by SLAs provides practical opportunities to intervene, as local governments and regional organisations often coordinate local funding and services in Australia.

As such in Chapter 5, Study 3 aimed to identify whether community-level variables explained a large component of locality variation in depression symptoms relative to individual, family, and school factors. Study 3 also aimed to identify whether community-level factors such as community disadvantage and State were explained by other predictors of depressive symptomology. Study 3 identified 7.1% of the variation in adolescent symptoms was explained by differences between SLAs. Analyses sought to identify modifiable factors that accounted for SLA (locality) differences. Community-level variables (community opportunities for pro-social involvement, community attachment, community safety, community laws and norms favorable to drug use, perceived availability of drugs, transitions and mobility, community-level disadvantage, and State) explained a large component (88%) of the locality variation in adolescent depressive symptoms. However, the majority of these effects were explained by
school, family and individual/peer factors within communities. State of residence and community safety were significant predictors of adolescent depression even after controlling for a wide range of covariates. Queensland and Western Australian adolescents were more likely to report depressive symptoms than their Victorian counterparts. Gender interaction effects were identified for community attachment, father living at home, and father’s employment. Thus findings confirmed that community-level factors may be important influences on adolescent depressive symptoms in Australia, suggesting the importance of evaluating community place-based interventions.

Overall findings from the current thesis suggested that community-level variables accounted for an important share of the potentially modifiable factors that predict adolescent depression. Given the high prevalence of elevated depression symptoms in Australian adolescents, reducing the incidence of adolescent depression should be a priority for Australian governments. Overall the findings of the current thesis suggested that developing and evaluating interventions to target community-level risk and protective factors in adolescents, is likely to be a cost-effective prevention strategy to reduce the incidence of depression. Findings support the use of place-based approaches that target multi-level (community, school, family, and individual/peer) risk and protective factors to reduce the prevalence of adolescent depression. However, preventative policy is needed to coordinate approaches as many of the factors influencing the onset of depression lie outside the domain of mental health services.

**Integrated Summary of the Findings from the 3 Studies**

Community-level risk and protective factors associated with depression.
Community safety. In Study 1 using the Stouffer method, the combined effect on child depression was significant. The findings of Study 1 were consistent with findings of a previously reported meta-analysis that identified a significant association between community violence and mental health outcomes (Fowler et al., 2009).

Further to the findings in Study 1, Study 3 identified that community safety was significantly associated with elevated depressive symptoms in Australian adolescents (10-14 years), even when controlling for a wide range of covariates at the community, school, individual/peer level. The final model in Study 3 also indicated that the school-level variable being a victim of bullying and the family-level variable family conflict were the strongest predictors of adolescent depression symptoms. Thus, exposure to violence appears to be an important factor to target in multi-level preventative interventions.

Traditionally research examining the impact of violence on children’s development outcomes has largely focused on direct physical abuse (De Bellis, Spratt, & Hooper, 2011). More recently however evidence has indicated that not only does experiencing direct physical violence have negative impacts on children, but exposure to violence also influences a range of outcomes for children and adolescents (Humphreys, 2008). It is increasingly recognized that the impact of violence exposure extends beyond the family setting to include school and community settings, however there are limited studies that consider violence exposure across multiple settings and the interactions between these settings.

Consistent with ecological systems theory, a growing body of research suggests violence exposure in one setting may influence violence exposure in other settings. Some studies support the influence of violence at the microsystem
level; with family violence exposure identified as a risk factor for school bullying victimization whereby children learn interaction styles characterized by feelings of helplessness and an inability to protect oneself from harm (Bowes et al. 2009; Duncan, 2011; Hong & Espelage, 2012).

Perceptions of school safety (Hong & Eamon, 2012) and school bullying (Espelage, Bosworth, & Simon, 2000) have been associated with community safety perception as an exosystem factor. Social disorganisation theory provides a theoretical explanation for the influence of community level factors whereby neighbourhoods with high transitions and mobility and low socio-economic resources have fewer available resources to manage crime and delinquency and victimisation. (Sampson & Raudenbush, 1997; Sampson, 2012; Laub & Lauritsen, 1998).

Consistent with neurodevelopmental theories, community-level safety is viewed as a chronic stressor that produces psychological distress (Matheson et al., 2006; Steptoe & Feldman, 2001). Neurodevelopment theories suggest that exposure to violence (across settings) leads to stress reactions that damage mood regulation systems. The limbic-hypothalamic-pituitary-adrenal (LHPA) axis is the major neuroendocrine stress response system. Dysregulation of the LHPA axis, elevated corticotrophin releasing hormone, and increased cortisol have been indicated in children with prolonged stress exposure due to factors such as family violence (De Bellis et al., 2011).

The serotonin system is also an important stress response system that activates both anxiogenic and anxiolytic pathways and acts as a master control neurotransmitter of complex neuronal communication (De Bellis et al., 2011; Lesch & Moessner, 1998). Serotonin plays an important roles in the regulation of
emotions (e.g., mood) and chronic stress (Fontenot, Kaplan, Manuck, Arango, & Mann, 1995). Exposure to unpredictable and uncontrollable stress, results in decreased serotonin levels in the amygdala, medial prefrontal cortex, nucleus accumbens, and lateral hypothalamus; depleting serotonin leading to “learned helplessness” (Petty, Kramer, & Wu, 1997). Dysregulation of serotonin has been associated with development of depressive symptoms (De Bellis et al., 2011). Further research exploring developmental processes that lead from violence at the community/school/family/ and individual-peer level to mental health problems is warranted.

*Community minority ethnicity and discrimination.* In Study 1, using the Stouffer method, the combined effect of community minority ethnicity and discrimination on child depressive symptoms was significant. Prior to Study 1, there had been no systematic review of the impact of minority ethnicity and discrimination at the community-level. Current evidence had produced mixed findings with regards to the role of ethnicity at the individual-level. Prior meta-analytic findings in the USA indicated that Hispanic children were at greater risk for depression but there were no significant differences in depression when comparing white and African American adolescents (Twenge & Nolen-Hoeksema, 2002). The findings from Study 1 indicated evidence supporting the association between community-level minority ethnicity and discrimination and child depression.

Study 2 and 3 considered ethnicity at the individual-level. In Study 2 univariate and multivariate analyses (including a number of demographic factors) indicated that indigenous status, country of birth, parents’ country of birth, and language spoken at home were not significantly associated with depression.
symptoms in Australian adolescents. In Study 3, in a model including a number of community, school, family, and individual-peer level factors only being indigenous was significantly associated with adolescent depressive symptoms. Together the findings of the three studies suggest ethnicity, minority status, and cultural factors may act indirectly to influence adolescent depression in Australia. Further research using purposive samples of Australian adolescents from indigenous and culturally and linguistically diverse backgrounds are needed to put these findings in context.

Community disadvantage. Study 1 identified using the Stouffer method the combined effect of community disadvantage on child depressive symptoms was not significant. Effect Sizes were smaller than other factors such as community safety that were found to be significant in the meta-analysis. Studies included in the systematic review suggested that community disadvantage may act indirectly by influencing other community-level variables that have a more direct effect. For example in two studies, community violence (Simons et al., 2002) and community cohesion (Caughy et al., 2008) were only significantly associated with depression in highly disadvantaged communities.

Prior research has supported the impact of community disadvantage on children’s development across a number of domains (Brooks-Gunn & Duncan, 1997). However, consistent with the findings of Study 1, a prior meta-analysis examining individual-level factors did not indicate a significant association between SES and child depression (Twenge & Nolen-Hoeksema, 2002).

Findings from Study 1 suggested that community disadvantage was not directly associated with child depression, but may indirectly effect child depression. In Study 2 adolescents from high disadvantage communities were
more likely to report depression symptoms than children from lower disadvantage communities. Study 2 showed that although this effect was evident, there was considerable variation between communities within each disadvantage strata. The findings from Study 1 and Study 2 suggested that the effect of community-level disadvantage may be explained by other predictors of depression. As such, Study 3 used multi-level models that examined a range of risk and protective factors to examine the effect of community disadvantage on adolescent depression. In Study 3, after adding a number of community, school, and family-level factors, community disadvantage was no longer significant, suggesting that its effect was indeed explained by other factors. Taken together the findings of the current thesis support the view that community disadvantage effects children’s depression, indirectly due to its association with other predictors of depression.

Some researchers suggest that community disadvantage influences adolescent depression by increasing vulnerability to negative events; elevated levels of daily stress; and disrupting social ties (Cutrona, 2006). Developmental theory and some evidence in the present review would imply that community disadvantage may act indirectly, for example, by disrupting child-parent bonding. Child-parent relationships have been found to mediate the impact of community disadvantage on adolescent depression (Wickrama & Bryant, 2003). One study suggested that community disadvantage negatively impacted on community connectedness which affected the child-parent relationship and in turn influenced adolescent depressive symptoms (Wickrama & Bryant, 2003). Thus, relevant child mental health promotion efforts may be better directed at protecting child-parent bonding in disadvantaged families (Letcher et al., 2009).

Community connectedness. In Study 1 using the Stouffer method, the
combined effect of community connectedness on child depressive symptoms was not significant, based on limited studies. There had been no prior systematic review examining the association between community connectedness and childhood depression. Findings from Study 1 indicated that the impact of community connectedness on child depression may vary by other community-level variables such as community disadvantage (Caughy et al., 2008; Witherspoon et al., 2009) and other community-level social processes (Xue et al., 2005).

Study 3 found that after adjusting for other community and school-level risk and protective factors, community connectedness (neighbourhood attachment) was significantly associated with adolescent depressive symptoms. However, after adding family-level factors community connectedness was no longer significant. Gender interaction effects were however significant for community connectedness in all models including the final model that included a wide range of risk and protective factors at the community, school, family, and individual/peer level. Thus community connectedness may influence the development of depression in different ways for males, but have particular benefits for female adolescents.

Given the low number of included studies in the meta-analyses further replication is needed to determine whether the included studies failed to identify true significant effects. Future research should also consider how family level factors explain the effect community connectedness has on the development of adolescent depression. For example one community intervention indicated that children relocated from disadvantaged communities into middle-class communities who had little social interaction with their neighbours had greater
levels of depression symptoms (Fauth, Leventhal, & Brooks-Gunn, 2007). Parents play an important role in facilitating social networks for children as they transition into new neighbourhoods, thus it is important to consider family approaches in preventative approaches targeting community-level factors.

*Other community-level risk and protective factors.* In Study 1, one prior study suggested that children from small remote communities had higher levels of internalising symptoms than children from large urban communities (Boyle & Lipman, 2002). In Study 2 and Study 3 regionality was not significantly associated with depressive symptoms in Australian adolescents. Further research is needed to put these findings in context.

In Study 1 community transitions and mobility was not significantly associated with child depression. Two separate studies failed to find a significant association between residential stability and residential mobility, and child depression. In Study 3 community transitions and mobility was significantly associated with depressive symptoms when examined in a model adjusting for other community-level factors. However, after adjusting for school-level risk and protective factors the association was no longer significant. The combined findings of the current thesis suggest that the effect of community transitions and mobility may be explained by its association with school risk and protective factors that more directly influence adolescent depression.

Some researchers suggest from a social disorganisation and neighbourhood effects perspective, community transitions and mobility influence children’s school bullying victimisation (Sampson & Raudenbush, 1997; Sampson, 2012). Highly transient adolescents are more likely to experience violent victimisation (Haynie & South, 2005). Research suggests increases in
social stress and reduction in social capital (Hagan, MacMillan, & Wheaton, 1996) may increase perpetrators perception of ease of victimisation and decreased fear of resistance or retaliation (Haynie & South, 2005; Hoglund & Leadbeater, 2004). Thus community transitions and mobility may influence the likelihood of experiencing school bullying and in turn increase the likelihood of experiencing depression symptoms.

In Study 3 the community-level risk factors perceived availability of drugs and laws and norms favourable to drug use were both significantly associated with adolescent depressive symptoms in models that adjusted for community-level risk and protective factors. However, these effects were no longer significant after adjusting for school (perceived availability of drugs) and family (laws and norms favourable to drug use) level variables.

The finding that the effect of community laws and norms favorable to drug use influence adolescent mental health indirectly through family-level factors was observed in a prior study that found parents were more likely to supply alcohol to underage adolescents if they lived in neighborhoods with high densities of alcohol sales outlets (Rowland, Toumbourou, Satyen, Livingston, & Williams, 2014). Future studies should more directly seek to investigate theoretical mechanisms to provide a firmer basis for developing preventative interventions within ecological community systems.

In Study 1, one study suggested that residents’ involvement in local organisations was significantly associated with lower CBCL scores (Xue et al., 2005). In Study 1, four separate studies examining time in centre-based care, neighbourhood problems, neighbourhood satisfaction, and female literacy did not identify any significant associations with depressive symptoms.
State. Study 2 and 3 presented the first Australian studies to examine the national prevalence in adolescent depression symptoms by State. The prevalence of depressive symptoms varied by State. An important finding in Study 3 was that State of residence was significantly associated with depression symptoms even after controlling for a wide range of factors at the community, school, family, and individual-level. A possible explanation for these findings is that State policy influences important domains of adolescent development such as health and education. For example in Queensland and Western Australia students in 2006 did not transition to high school until grade 8 whereas in Victoria the transition occurred in grade 7. Such state differences in educational policies may influence adolescent depressive symptoms. Access to services and distance to services (or urban centres) may also impact on adolescent depressive symptoms particularly in states with a large number of remote communities such as Western Australia and Queensland.

Community disadvantage interventions. The systematic review (Study 1) examined the effect of interventions targeting community-level factors on depression in school-aged children (4-18 years). The meta-analysis failed to reach significance for the 3 intervention studies examining the effects of alleviating disadvantage, however the effect size was larger than that for many of the factors found to be significant.

Some of the included studies indicated that the impact of community disadvantage on depressive symptoms may be influenced by other factors such as community connectedness. Indeed one community intervention, indicated that children relocated from disadvantaged communities into middle class communities who had little social interaction with their neighbours experienced
greater levels of depressive symptoms (Fauth et al., 2007). As discussed above, Study 3 identified that community connectedness (neighbourhood attachment) had a protective effect on depressive symptoms in Australian adolescents that was explained by family-level factors. Taken together the findings suggest that interventions aiming to prevent adolescent depression in disadvantaged communities could seek to enhance family strengths (Buttigieg et al., 2015).

Prevalence of sub-clinical and clinical depression in Australian adolescents. Given limited prior data, prevalence rates of elevated depression symptoms (sub-clinical and clinical) in Australian adolescents (10-14 years) were estimated in Study 2. Using a representative sample, Study 2 identified that 33.1% (95% CI: 32.1, 34.2) of Australian adolescents (10-14 years) reported elevated depression symptoms in 2006.

As would be expected, this is much higher than the prevalence of clinical depression, with a higher cut off score, (14.2% [Boyd et al., 2000]; 16.6% [Bond et al., 2005]) identified in previous Australian samples of adolescents (11-18 years). However, the prevalence rates estimated in the current study were also somewhat lower than findings from a USA study that identified 44.3% of American adolescents (Mean age = 16.6 years) experienced sub-clinical or clinical depression (Lewinsohn et al., 2004). Adolescents in the American sample were comparatively older than adolescents in the Australian sample. The present findings highlight an important need to address elevated depression symptoms in Australian adolescents. Further cross-nationally matched research is warranted in age matched samples to inform accurate comparisons.

Gender differences. In accordance with previous Australian research examining clinical depression (Boyd et al., 2000; Bond et al., 2000), significantly
(OR = 1.27; P =0.000) more females (35.7%, 95% CI: 34.3, 37.2) reported depressive symptoms than males (30.3%, 95% CI: 28.8, 31.8). In accordance with research suggesting gender differences in the developmental trajectory of adolescent depression (Cairns et al., 2014; Dekker et al., 2007 Fernandez-Castelao & Kröner-Herwig, 2013, Yap et al., 2014), significant gender interactions were identified for school grade and father’s employment in multivariate analysis in Study 2.

In Study 3 gender interaction effects were identified for, community attachment, father’s employment, and father living at home. Together the findings of the current thesis suggest that whilst only a small number of variables had significant gender interaction effects it is important to consider gender differences otherwise important gender-specific risk and protective factors may fail to be detected. Future research should explore gender differences and potentially different developmental trajectories of psychopathology.

Positive child-father relationships have been associated with a range of positive outcomes for adolescents including low levels of psychological distress (Amato, 2001). Evidence suggests that both male and female children and adolescents experience negative outcomes associated with parental separation (Amato, 2001; Carlson, 2006). However, traditionally there have been mixed findings associated with gender comparisons. Some studies suggested females experienced greater psychological vulnerability associated with father absence (Rudolph, 2002), whereas others indicated worse outcomes for males (Hetherington et al. 1989; Morrison & Cherlin, 1995). Emerging evidence suggests that age may play a role in gender differences. One study found females with absent fathers experienced stronger and more enduring depressive symptoms
at age 14 and 16 years than their male counterparts (Storksen et al., 2006). These findings were replicated in a longitudinal study of parents and children in the UK which identified a significant association between father’s absence in early childhood and depressive symptom at age 14, with the association stronger for girls than boys (Culpin et al., 2013).

**Variance between SLAs at the community, school, family, and individual/peer level.** The community-level factors community opportunities for pro-social involvement, community attachment, community safety, community laws and norms favorable to drug use, perceived availability of drugs, transitions and mobility, community-level disadvantage, and State accounted for 88% of the variance in depression between SLAs. With the exception of community safety and State these effects were not maintained in the multivariate models accounting for school, family, and individual influences. These findings suggest that community-level factors may be important to target in locality (place-based) strategies to prevent adolescent depression, however to achieve this multi-level interventions may be required that account for underlying influences that vary between localities. The findings support the importance of designing and evaluating locality interventions that utilize a range of data through local, state, and national agencies to identify community-level issues and develop appropriate responses (e.g., Hawkins et al., 2009).

**Integrated Thesis Findings.** Overall findings from the current thesis support the position that community-level variables account for an important share of the potentially modifiable factors that predict adolescent depression. The high prevalence (33.1%) of elevated depressive symptoms in Australian adolescents and the opportunities for intervention suggest that targeting the
incidence of depression during early adolescence (10-14 years) should be a priority for Australian governments.

Although State level policies may be independently important, targeting identified community-level factors will likely offer a cost-effective and additional means of reducing sub-clinical rates of depression by impacting large population aggregates. Further, targeting factors at the community, school, family, and individual/peer levels are likely to influence the impact of community factors, supporting the use of multi-level approaches. Multi-level interventions, especially those that include community-level factors, are likely to be the most effective and efficient approach to preventing adolescent depression at a population level.

**Strengths, Limitations, and Future Research**

The discussion section of each study outlines study specific limitations and areas for future research. The next section will focus on limitations of the current thesis as a whole and limitations that require further consideration beyond those already discussed.

**Cross-sectional design.** The majority of studies identified in the systematic review were cross-sectional \((k = 13)\). Study 2 and Study 3 also adopted a cross-sectional design. The cross-sectional nature of these studies limits the capacity to draw firm conclusions about the causal direction of identified associations. Longitudinal research is needed to investigate the direction of identified associations. Intervention research is valuable for testing causal models. Given that adolescent depression is highly co-morbid, future research should also assess multiple mental health disorders to determine the developmental trajectory and influence of co-morbid presentations.

**Heterogeneity of measures.** A limitation of the current evidence base,
and the current thesis, was the heterogeneous range of community-level outcomes and measures, depressive symptom outcomes and measures and analysis, and sample demographics including wide age ranges; varied contexts; and country of origin across studies. This heterogeneity led to difficulties comparing findings and limits the conclusions that can be drawn, particularly in terms of generalisability.

**Self-report measures.** A possible limitation of the current thesis was the use of self-report measures in Study 2 and Study 3. The use of self-report measures with adolescents is subject to situational and cognitive factors that impact on validity (Brener, Billy, & Grady, 2003). Despite their limitations self-report measures are useful for a number of reasons including confidentiality, cost-effectiveness, and accessibility. Further, self-report measures may be useful in examining an individual’s perception rather than a distinct reality. For example in evaluating community safety it may be important to understand an individual’s perceptions of safety in addition to how much crime occurs in an area.

Whilst self-report measures offer a useful means for identifying associations between predictors and depression, establishing the validity of community-level measures will be important to ensure valid research and provide greater cost effectiveness in the long-term. For example, as there is strong evidence to support community safety as a risk factor for depression, it would be worthwhile identifying valid population-level measures of safety such as crime statistics that can be used to identify high-risk communities. Future research is needed to compare self-report measures of known predictors (e.g., feelings of safety) with community-level measures of the same construct (e.g., local area police statistics) to establish the validity of using readily available population-level measures to identify areas for intervention. An important weakness of Study
1 and Study 3 was that official records of community safety and minority status were not included in the analyses.

**Gender.** Another limitation of Study 1 was the failure to examine the meta-analytic effects of community-level factors by gender. This was not possible as the majority of included studies failed to report gender-specific statistics. Research has been criticised for failing to consider risk and protective factors by gender, despite the growing body of evidence that supports gender-specific risk and protective factors during childhood and adolescence. Further Study 2 and Study 3 indicated gender interaction effects for predictors of adolescent depression. Future research should develop gender specific models of psychopathology and report gender-specific statistics.

**Risk and Protective Factors.** Whilst the current thesis addressed an important criticism of current research by including a large number of risk and protective factors (Shanahan et al., 2008), there may be important factors that were not included. Future research should assess other plausible risk factors including family history of mental illness, and adolescent genetic risks for depression. Future research should also consider the impact social media has on adolescent depression. Indeed, Johnson (2010) suggests that given the important role of digital technologies in children's immediate environments Bronfenbrenner’s model should be extended to incorporate “techno-microsystems”.

**Defining communities.** Study 2 and Study 3 examined communities by Australian SLAs. Defining communities by SLA is useful for a number of reasons. Identifying differences by SLAs provides practical opportunities to intervene as local funding and services in Australia are coordinated by local
governments and regional organisations. Also, census and other population-level
data can be readily organised by SLA, providing access to readily available
measures of community-level factors such as SEIFA categories of disadvantage.

However, conceptualising community-level factors may be best achieved
at a more proximal local level such as by suburb or postcode. Important findings
about the impact of community-level factors may be lost when communities are
aggregated to the SLA-level. For example, an SLA may be identified as a high
SEIFA community but may include a number of smaller communities (e.g.,
postcodes) with low SEIFA ratings. People living in low SEIFA postcodes are
likely impacted by the effects of poverty. However, in examining communities at
the SLA-level these impacts may not be identified.

In Australia and internationally, a number of place-based initiatives
support intervening at the postcode level (Ferrie, 2008). For example Community
Renewal projects target urban pockets of disadvantage at the suburb/postcode
level (Ferrie, 2008). Whilst not as publicly available, census and other
community-level statistics are also frequently quantified at the postcode level in
Australia. Future research should explore differences in examining locality
differences at the SLA and postcode-level. Further future research should also
examine interventions at the postcode and SLA levels.

**Determinants of health, mental health, and other outcomes.** The
current thesis identified a number of risk and protective factors associated with
child and adolescent depression, particularly at the community-level. Whilst these
findings indicate important cost-effective opportunities for intervention, it is
necessary to consider these findings in the context of other government priorities.
Consistent with the principles of multifinality, future research is needed to
identify determinants of health that exist across the lifespan and across health disorders. From a scientist-practitioner perspective it is important to adopt a narrow focus in research in order to determine causality. However, advancing prevention requires identifying cost-effective means for governments to intervene. Thus, identifying determinants that impact on a range of outcomes across a large number of individuals and communities is likely to increase government’s interest in providing funding in this area.

For example the current thesis indicated that community safety was associated with child and adolescent depression, future research should examine the impact of community safety on other child and adolescent outcomes such as health and externalising behaviours. It is likely that community safety also impacts on a range of outcomes for adults. Building the evidence base to identify risk and protective factors that impact on a range of outcomes across the developmental lifespan is likely to attract greater government prioritisation and funding to address the underlying determinants of health.

**Government policies.** Study 2 and Study 3 identified state-level differences in prevalence rates. Further Study 1, Study 2, and Study 3 identified predictors of depression that are likely influenced by local and state policies. Future research should identify and review the impact of changes in policies that underpin identified determinants of mental health disorders. For example reviewing State-level health policy or local government safety planning and corresponding mental health outcomes.

**Theoretical frameworks underpinning preventative interventions.** Together the 3 papers supported the need for population-level, place-based preventative interventions that target risk and protective factors to reduce the
social and financial impacts of depression. The current thesis also highlighted important ecological, developmental, and prevention theories and approaches that underpin this work. However, further integration with current literature and theories is needed to develop evidence-based approaches to preventative intervention, which was beyond the scope of the current thesis. As one example, efforts to reduce the impact of community safety on depressive symptoms may benefit by being better integrated with developmental theories. Developmental theory and some evidence in the present thesis would imply that community safety may act indirectly, for example, by disrupting mother–child bonding. Thus, relevant child mental health promotion efforts may be better directed at protecting mother–child bonding in communities with safety concerns (Letcher et al., 2009).

Implications and Conclusions

A growing body of evidence supports the need to intervene during adolescence to prevent the onset of depression and curb the negative trajectory associated with this debilitating illness. However, governments continue to have limited interest in funding prevention research in this area as the cost offset of such approaches will take many years to eventuate (Jacka & Reavley, 2014). Strengthening the evidence base supporting the cost-effectiveness of such intervention is essential to advancing prevention efforts (Jacka & Reavley, 2014; Jorm, 2014). The current thesis adds a novel contribution to the field of adolescent mental health prevention in a number of ways.

Adolescence as a critical period for preventative intervention. Study 2 was the first study to quantify the prevalence of elevated depression symptoms (sub-clinical and clinical) using a large representative sample of Australian adolescents. Study 2 identified that approximately one third of Australian
adolescents experienced elevated depressive symptoms. Findings from Study 2 suggested that given the high prevalence of depression symptoms in Australian adolescents, this is an important area for prevention research and investment.

A significant barrier to prevention research has been the reluctance of government and funding bodies to invest in prevention research, as the benefits of such interventions take many years to eventuate (Jacka & Reavley, 2014). A review of current research funding, volume of publications, and stakeholder priorities support prevention research as a high priority area that receives little funding (Christensen et al., 2013). In a survey of policy professionals, researchers, and representatives of peak bodies, forty-four percent of respondents nominated prevention research in their top 3 priorities (Christensen et al., 2013). However, despite already receiving the lowest amount of funding in 2000, prevention research funding was further halved in 2008 (Christensen et al., 2013). Prevention research accounted for less than 5% of overall publications (Christensen et al., 2013) funded by Australian health research.

Stakeholders rated children and adolescents as their highest priority target group but little funding was dedicated to this area (Christensen et al., 2013). Interestingly, depression was rated as the highest priority in terms of mental disorders (Christensen et al., 2013). However, despite receiving a substantial amount of funding there was relatively few publications in this area (Christensen et al., 2013). The current thesis supports adolescent depression as an important area for investment in prevention research.

Community-level risk and protective factors. The systematic review completed for the current thesis identified strong evidence that in prior studies a number of community-level factors were associated with child depression. Whilst
it is widely acknowledged that population-level approaches are needed to reduce community prevalence rates, researchers and policy makers have argued that there is limited understanding of community-level predictors of adolescent depression that could be targeted through such approaches (Jorm, 2014; McDermott et al., 2010). The current thesis indicated a number of community-level risk and protective factors that are associated with child depression.

To date Australian guidelines for adolescent depression have largely been reliant on international research and have argued that there is not enough evidence to support interventions at the population level (McDermott et al., 2010). As such the findings of the current thesis add a significant and unique contribution by identifying community-level factors that can be targeted to reduce the prevalence of depression symptoms in Australian adolescents. This may have implications for the design of preventative interventions suggesting designs that involve either: targeting high risk communities for selective interventions; targeting community-level risk and protective factors in indicated, selective and targeted interventions; and/or targeting community-level determinants of health through coordinated responses.

**Multi-level interventions.** There is little understanding in the current literature of whether community-level factors are moderated by other predictors of adolescent depression. Study 1 findings suggested that in prior studies community-level factors are likely moderated by other predictors of child depression. Adding to the findings of Study 1, findings from Study 3 suggested that with the exception of community safety and State, all community-level factors were explained by other predictors of depression symptoms in Australian adolescents. These findings are consistent with Bronfenbrenner’s
conceptualisation of the mesosystem, suggesting that the interaction between two immediate environments (or microsystems) impacts the individual (Bronfenbrenner, 1995). Targeting factors at these levels is likely to influence the impact community-level predictors indicating support for the use of multi-level approaches.

**Population-level approaches.** Prior to the current thesis there was limited understanding of processes by which community-level factors may influence adolescent depression in relation to subordinate factors at the school, family, and individual-level. Consistent with Bronfenbrenner’s conceptualisation of the microsystem, findings from study 3 suggested that factors at the individual-peer, family, school, and community-level are all associated with depression symptoms in Australian adolescents. Study 3 identified that community-level factors accounted for 88% of the locality variation in depression symptoms between communities. Whilst adding school, family, and individual/peer factors accounted for a greater amount (91%) of the locality variation in adolescent depressive symptoms, community-level factors account for a large proportion of this variance. Together the findings suggest that targeting community-level factors offers a cost-effective means of reducing the prevalence of sub-clinical and clinical depression in Australian adolescents.

**Gender.** As discussed in previous sections evidence suggests gender differences in prevalence rates and developmental trajectories of adolescent depression. Findings from Study 2 and Study 3 identified gender interaction effects in univariate and multivariate analyses. Ecological system’s theory suggests that the individual brings characteristics such as gender that influences every context in which they interact. Findings from Study 2 and 3 suggest that
gender not only directly influenced the likelihood of experiencing depression symptoms but also impacts on other risk and protective factors. Together the findings of the current thesis suggest that a small number of social relationship variables had significant gender interaction effects. These findings assist in pinpointing how gender differences in adolescent-onset depression might be explained by gender-specific protective factors.

**State differences.** Previously no studies had examined prevalence and predictors of sub-clinical and clinical depression using samples that included multiple Australian states. Together findings from Study 2 and Study 3 indicated that the prevalence of adolescent’ depression symptoms was significantly different by State of residence. An important finding in Study 3 was that State was significantly associated with adolescent depression even after controlling for a wide range of factors at the community, school, family, and individual-level. State of residence maintained a strong independent effect on adolescent depression in the final model.

These findings suggest that consistent with Bronfenbrenner’s theory the exosystem, in this instance the broader environmental and policy context, may play an important role in the development of sub-clinical and clinical depression in Australian adolescents by influencing adolescents’ immediate environments. For example policies that impact on other risk factors for depression such as education vary from State to State. Similarly State employment policy and services that influence parents’ employment likely indirectly impact the likelihood of experiencing depression symptoms.

**Collaboration.** Study 1 identified that a large number of studies assessing community-level risk and protective factors and interventions failed to use
psychometrically sound measures. Progressing community health promotion can be best advanced with the involvement and commitment of a cross section of disciplines, professions, organisations, and government departments. Whilst the need for collaboration is consistently acknowledged and advocated for, too often it is passed over in absence of a practical first step. Policy makers and funding bodies are encouraged to require the use of psychometrically valid measures in outlining the outcomes linked to funding provision. In addition, policy makers and funding bodies should require partnerships between researchers and service providers in the routine collection of data; and the evaluation of service output and interventions. Collaboration between researchers and services provides obvious benefits for both agencies by utilising respective skill sets. Such collaboration will provide a strong base of high quality data that can be used by policy makers and funding bodies to guide future directions.

**Conclusion**

In conclusion, the current thesis indicated that community-level variables accounted for an important share of the potentially modifiable factors associated with adolescent depression. The present study was the first to estimate the prevalence of depression symptoms (sub-clinical and clinical) in Australian adolescents and found very high rates at 33%. Findings from the studies presented in this thesis confirm that although risk and protective factors for depression emerge across multiple settings, those at the community-level may be neglected but important factors to consider in efforts to design and evaluate prevention strategies to reduce the incidence of depression.
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Appendices for Study 1

Appendix A

Database Search 1 using EbscoHost MegaFILE Premier Facility

**Databases:** Education Research Complete, E-Journals, Humanities International Complete, PsycARTICLES, PsycEXTRA, Psychology and Behavioral Sciences Collection, PsycINFO, Social Work Abstracts, and Urban Studies Abstracts

**Restrictions:**
- Scholarly (Peer Reviewed) journals
- Date Range: 1992-2012

**Search Terms:**

1. child* OR adolescen* OR young person* OR young people* OR youth*
   OR student* OR high school* OR primary school* OR secondary school*
   OR girl* OR boy* OR kid* OR juvenil* OR under age OR teen* OR
   minor* OR pubescen* OR pediatric* OR young adult* AND

2. mental health* OR depress* OR mental illness* OR depression* OR
   dysthymi* OR low mood* AND

3. clinical trial* OR randomi?ed OR placebo OR random* OR trial AND

4. communit* OR government area* OR state* OR local area* OR suburb*
   OR LGA* neighbourhood*
Appendix B

Database Search 2 using EbscoHost MegaFILE Premier Facility

**Database:** PsycINFO

**Restrictions:**
- Date Range: 1992-2012

**Search Terms:**

1. KW adolescen* OR teen* OR child* OR minor* OR youth* OR young people AND
2. KW mental health* OR depress* OR mental illness* OR poor outcome* OR depression* OR dysthymic* OR dysthmia* OR low mood* OR mood disturbance* OR CBCL or child behavior OR child behaviour* OR internalising* OR internalizing* AND
3. KW clinical trial* OR randomi?ed OR placebo OR random* OR trial
Appendix C

Database Search 3 using EbscoHost MegaFILE Premier Facility

**Database:** PsycINFO

**Restrictions:**

- Date Range: 1992-2012

**Search Terms:**

1. KW adolescen* OR teen* OR child* OR minor* OR youth* OR young people AND
2. KW mental health* OR depress* OR mental illness* OR poor outcome* OR depression* OR dysthymic* OR dysthmia* OR low mood* OR mood disturbance* OR CBCL or child behavior OR child behaviour* OR internalising* OR internalizing* AND
3. multilevel OR multi-level OR multi level
Appendix D

Database Search 4 using EbscoHost MegaFILE Premier Facility

Database: PsycINFO

Restrictions:

- Date Range: 1992-2012

Search Terms:

1. KW adolescen* OR teen* OR child* OR minor* OR youth* OR young people AND

2. KW mental health* OR depress* OR mental illness* OR poor outcome* OR depression* OR dysthymic* OR dysthmia* OR low mood* OR mood disturbance* OR CBCL or child behavior OR child behaviour* OR internalising* OR internalizing* AND

3. bronfenbrenner* OR ecological systems theory* OR ecological system*
Appendix E

Database Search 5 using EbscoHost MegaFILE Premier Facility

**Database:** Urban Studies Abstracts

**Restrictions:**
- Date Range: 1992-2012

**Search Terms:**

1. **adolescen* OR teen* OR child* OR minor* OR youth* OR young people*AND**

2. **mental health* OR depress* OR mental illness* OR poor outcome* OR depression* OR dysthymic* OR dysthmia* OR low mood* OR mood disturbance* OR CBCL or child behavior OR child behaviour* OR internalising* OR internalizing*AND**

3. **longitudinal* OR hierarchical linear model* OR bronfenbrenner* OR ecological systems theory* OR ecological system* OR multilevel* OR multi-level* OR multi level* OR clinical trial* OR randomi?ed OR placebo OR random* OR trial**
Appendix F

Database Search 6 using EbscoHost MegaFILE Premier Facility

Database: Social Work Abstracts

Restrictions:
- Date Range: 1992-2012

Search Terms:
1. AB longitudinal* OR hierarchial linear model* OR bronfenbrenner* OR ecological systems theory* OR ecological system* OR multilevel* OR multi-level* OR multi level* OR clinical trial* OR randomi?ed OR placebo OR random* OR trial) AND
2. AB adolescen* OR teen* OR child* OR minor* OR youth* OR young people AND
3. AB mental health* OR depress* OR mental illness* OR poor outcome* OR depression* OR dysthymic* OR dysthmia* OR low mood* OR mood disturbance* OR CBCL OR child behavior* OR child behaviour* OR internalising* OR internalizing*

NB: AB= Abstract
Appendix G

List of websites searched in Grey Literature Search

Australian Department of Families, Housing, Community Services, and Indigenous Affairs: http://www.fahcsia.gov.au


Effective Public Health practice Project Canada:

http://www.ephpp.ca/asystrev.html

United Kingdom Department of Education website:

https://www.education.gov.uk/

Appendix H

Full List of References Excluded from the Present Review


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