

**With a Little Help From my Friends<sup>1</sup>: The Role of Personality in the Relationship between Social  
Support and Adolescent Depression**

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A thesis submitted for the degree of Doctor of Clinical Psychology at The Australian National  
University

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<sup>1</sup> Beatles. "With a Little Help from My Friends." Sgt. Pepper's Lonely Hearts Club Band. Capitol Records, 1967.

## Declaration

I declare that this thesis is the product of my own work carried out under the supervision of Professor Don Byrne. I affirm that this thesis is in accordance with the Australian National University Guidelines for higher degree research.

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21st August 2015

## Abstract

Relationships provide young people with a sense of integration and personal worth. The ability of adolescents to acquire and maintain support from their social networks is of clinical interest as inadequate social support is posited to be a primary causal factor in the onset of adolescent depression. The literature analysing adolescent depression has been extensive and has suggested that depressed young people are less rewarding for social contact, which results in a reduction of their social networks and support systems. The current thesis argues that traditional interpersonal theories of adolescent depression have failed to take into account the intersection between normative and atypical development, the continuous transition between young people and their environments, as well as the long term effect of an episode of depression on personality formation.

The current work suggests that an integrated interpersonal theory of adolescent depression needs to investigate potential mechanisms for the onset, maintenance and consequences of youth depression. Two studies explored the relationship between the main variables using the three interacting pathways presented by the cognitive vulnerability transactional stress model (CVTSM). The model posits firstly that cognitive vulnerability and stressors are predictors of depressive symptoms (vulnerability model), secondly, that depressive symptoms and cognitive vulnerabilities are predictors of stressors (stress generation model). Finally, that depressive symptoms and stressors as predictors of future vulnerabilities (consequence model).

Study one provides insight into the relationship among the main variables and their relationship to adolescent depression. The study demonstrated that the relationship between neuroticism and stress was the most robust risk factor for depression. The small protective effect of social support is also discussed as well as additional pathways to depression. Study two demonstrated that initial depression resulted in heightened levels of stress two years later. This relationship was apparent even when depression was in remission, and independent of personality,

suggesting that stress generation which stems from depression may be potential mechanism for relapse. The results were consistent with pathway one and two of the CVTSM, however no support was found for the third pathway.

The manner in which the results support the CVTSM and interpersonal theories of depression is explored as well as the theoretical and practical implications of the research. It is concluded that personality vulnerabilities, stress and depression have a reciprocal relationship which transacts to provide insight into the onset and maintenance of youth depression.

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*“The desire to reach for the  
stars is ambitious. The desire  
to reach hearts is wise.”*

*– Dr Maya Angelou*

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## CHAPTER ONE: Literature Review

**With a Little Help From my Friends<sup>1</sup>: The Role of Personality in the Relationship between Social Support and Adolescent Depression**

Humans are social beings born with a fundamental drive to belong. The psychological value of personal relationships is significant, providing people with a sense of integration and personal worth (Hames et al., 2013; Paykel, 1994). The desire for social belonging is thought to peak during adolescence, when young people strive to define themselves as autonomous individuals (Compass, Connor-Smith, Saltzman, Thomsen & Wasworth, 2001). For young people, social relationships, especially those with their peers, provide a vehicle to practice new coping mechanisms, a sense of normalisation and community (Hames et al., 2013). However, both anecdotal and empirical evidence suggests a distinct segment fail to form social bonds, and research has shown that the absence of such support renders this proportion vulnerable to higher levels of distress, hopelessness and depressive pathology (Heinrich & Gullone, 2006). In part, this may be because such individuals demonstrate social behaviours, thought to be influenced by their personality, which renders them less rewarding for social contact (Leskela et al., 2009). The role that personality plays in a young person's psychosocial world, and their ability to acquire and maintain support from their networks, is of clinical interest as the ability to navigate stress inherent to a life stage is a primary causal factor, precipitating either positive social functioning, or the onset of a depressive episode (Elias, Gara, Schuyler, Branden-Muller, & Sayette, 1991; Nicolai et al., 2013).

Research into the psychosocial variables associated with adolescent depression has predominately focused on depressive symptomology, social support and personality as discrete variables. Interpersonal theories of depression point to social-behavioural deficits of depressed young people, as a causal factor in social networks dissipating. These deficits are thought to arise due to the individual's state (i.e. depression) or traits they possess (i.e. neuroticism; Leskela et al., 2009; Paykel, 1994; Rudolph, Flynn & Abaided, 2008). However, research indicates that personality

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may be a stable risk factor which is apparent well before the first episode of depression. Neuroticism has been shown to have a strong causal relationship to depression, explaining up to 55 per cent of the genetic risk of the disorder expression (Kercher, Rapee, & Schiering, 2009; Weissman et al., 2005). Furthermore, even in the absence of depression, neuroticism has been found to further exert a disruptive influence on the interpersonal sphere, and generate stress (Kercher et al., 2009).

As a result, transactional frameworks have attempted to delineate the variables, and explore the developmental conditions that may see personality and inadequate social support interact to result in depression (Hames et al., 2013; Lonigan, Phillips & Hooe, 2003). The cognitive vulnerability transactional stress model (CVTSM) suggests that neuroticism may interact with the social sphere to increase the likelihood of depression via three interacting pathways (Costello, Erkanli, Angold, 2006; Hankin, 2012). Firstly the vulnerability model whereby intrinsic individual vulnerabilities such as personality are apparent early in life and interact with interpersonal stressors to predict depression. Secondly, via the stress generation model, which suggests that personality traits such as neuroticism influence behaviour to generate additional stressors, leading to a relapse in depression. Thirdly, the consequence model which suggests that depressive symptoms and stress, may have an adverse impact on a young person's developing personality, resulting in vulnerabilities that continue to generate stress across the lifespan (Calvete, Orue & Hankin, 2012).

Given the pernicious nature of depression and young people's focus on the interpersonal world, it is crucial to understand how antecedents of depression may interact within the interpersonal context to influence the onset, maintenance and chance of relapse (Hames et al., 2013). Elucidating factors associated with early onset of depression is of high importance as depression which emerges in adolescence has been linked to a higher lifetime risk for relapse, and tends to follow a more severe trajectory than depression which first occurs during adulthood (Merikangas et al., 2010; Newman et al., 1996; Feng, Shaw & Silk, 2008). Finally, as social bonds are amenable to deliberate change, this may signal a treatment inlet suitable for early intervention to

prevent depression becoming a lifelong disorder (Allen, Hetrick, Simmons & Hickie, 2007; Dutton, 2014; Henderson, Bryne, & Duncan-Jones, 1980).

### **Epidemiology of Adolescent Depression**

Depression is an important area of inquisition as it is the most common psychiatric condition faced by adolescents. Young people describe depression as debilitating and difficult to cope with, and unsurprisingly it is associated with a range of negative life outcomes, such as substance abuse and suicide (Murray & Lopez, 1997; Wilkinson, Kelvin, Roberts, Dubicka & Goodyer, 2011).

Depression during adolescence is thought to have an adverse effect on neurobiological, cognitive, social and emotional functioning at a key time of skill acquisition (Monroe & Harkness, 2005; Lewinson, Rohde, Seeley, Klein & Gotlib, 1998; Vythilingam, Heim, & Newport, 2002). One episode of depression during adolescence greatly increases the chances a young person will become depressed again and also that they will be dissatisfied with their relationships, and general life (Birmaher, 2014). Depression not only impacts the way young people think and feel, but also the way in which they interact with others in their environment and their ability to set in motion the patterns which make for a fulfilling life (Hames et al., 2013).

### **Diagnosis**

Depression is a familiar human condition with the word being used interchangeably with feeling sad, blue or despondent. The normal experience of sadness or being 'down in the dumps' is differentiated from a mental illness based on the chronicity of low mood and accompanying symptoms, but also the degree to which the symptoms impact on an individual's ability to function. The Diagnostic and Statistical Manual of Mental Disorders (DSM V, 2013) specifies the diagnostic threshold for a Major Depressive Episode as the experience of either low mood or adhedonia (loss of pleasure), and at least four additional symptoms, occurring in excess of two weeks which must be accompanied by distress or functional impairment (including social and occupational). In children and adolescents functional impairment may be noted by parents or teachers and may take the form of not meeting milestones or a notable change in functioning, such as suddenly failing school

assignments. Additional symptoms which may manifest in a major depressive episode include a variety of emotional, cognitive and behavioural aspects, such as change in appetite, insomnia, psychomotor retardation or agitation, low energy, difficulty concentrating or making minor decisions, feelings of worthlessness or guilt, and suicidal ideation. Many of the symptoms are self-reported, and require the individual to describe to some degree their inner experiences. While other symptoms are observable, such as tearfulness, psychomotor retardation and withdrawal. A summary of the DSM V criteria for a major depressive episode is presented in Table 1 on the next page.

The expression of depression varies greatly within individuals and as such the DSM-V provides additional criteria to define the severity, the course and additional diagnostic features (APA, 2013). The disorder can present as mild, moderate or severe, and the classification of the severity is based on the number of symptoms the individual experiences. A severe depressive episode contains more symptoms, has a profound effect on functioning, and is more likely to contain suicidal ideation or attempts (APA, 2013). The course of the disorder is based on whether the episode is single, as in the first time the individual has been depressed or recurrent. A recurrent major depressive disorder notes that the individual has experienced one or more episodes in the past, but that there had been at least two consecutive months symptom free in between depressive episodes. The course of the disorder is important to define as some individuals never experience remission, whereas others have years in between episodes. In addition, the presence of multiple depressive episodes suggests the likelihood of a comorbid disorder and reduces the chance that treatment will result in full symptom resolution (APA, 2013). In addition, if psychotic or melancholic features were apparent it would change the treatment recommendations, as these differ based on the severity, course and also additional features of the disorder (APA, 2013; NICE, 2005). For example current best practice recommends that a severe depressive disorder with psychotic features is treated with psychopharmacological means, whereas a mild depression which is a single episode is treated with psychological therapy (NICE, 2005).

Table 1.

*Major Depressive Episode Diagnostic Criteria DSM-V (APA, 2013).*

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- A. Five (or more) of the following symptoms have been present during the same 2 week period and represent change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.
    - 1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g. feels sad, empty, hopeless) or observation by others (e.g. tearful). Note in children and adolescence can be irritable mood.
    - 2. Marked diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation)
    - 3. Significant weight loss when not dieting or weight gain (change of more than 5% of body weight within a month), or decreased, or increased appetite nearly every day (Note: in children, consider failure to make expected weight gain)
    - 4. Insomnia or hypersomnia nearly every day
    - 5. Psychomotor agitation or retardation nearly every day (observable by others, not merely feelings of being restless or slowed down).
    - 6. Fatigue or loss of energy nearly every day.
    - 7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)
    - 8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or observation by others)
    - 9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide
  - B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
  - C. The episode is not attributed to the physiological effects of a substance or to another medical condition.
  - D. The occurrence of the disorder is not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorders.
  - E. There has never been a manic or hypomanic episode.
-



## Trends

In Western cultures, the prevalence of adolescent depression is rising, while the age of onset is decreasing (Abela & Hankin, 2008). The onset of the first depressive episode is usually in early to mid-adolescence, with epidemiological studies demonstrating a two-fold increase from childhood to adolescence, such that by 18 years up to 20 per cent of young people will have experienced an episode (Lewinson et al., 1998; Merikangas et al., 2010). The most significant increase in depressive disorders occurs at age 13 years, which is significantly lower than ten years ago (ABS, 2009). Rates continue to rise throughout adolescence until approximately 17 years, when they taper off (ABS, 2009; Lewinson, Clarke, Seeley & Rohde, 1994).

In Australia, it is estimated that up to 7 per cent of young people experience a depressive episode in a year (ABS, 2009; Kitchener & Jorm, 2009). In 2006, depression represented the highest number of hospital stays in Australian adolescents aged 13-19 years (AIHW, 2003). This is a contrast to 1998, which saw schizophrenia account for the highest number of adolescent mental health inpatient admissions (AIHW, 2013). Australian rates of depression are comparable with other Western countries such as America, where approximately 8.5 per cent of youth in the United States experience a depressive episode in a year (SAMSHA, 2008). In addition, The National Comorbidity Survey- Adolescent Supplement (NCS-A) found about 11 per cent of American adolescents had experienced a depressive episode by the age of 18 years (Merikangas et al., 2010; SAMSHA, 2008). The overall lifetime prevalence of depression is estimated at to be 15-20% (Lewinson et al., 1986).

Within the current literature depression rates vary significantly by of gender. By age 13 years females double male rates of diagnosis (ABS, 2013). In 2007, 8.4 per cent of Australian adolescent females were clinically depressed compared to 4.3 per cent of males (ABS, 2007). Females have been also been found to endure longer episodes of depression and more severe symptom profiles (Klein et al., 2013).

**Course**

One episode of depression in youth greatly increases the chances of a recurrent episode, and many individuals report ongoing residual symptoms even in the event the disorder remits (Costello et al., 2002). It is estimated that one third of young people relapse into another major depressive episode within four years of treatment (Birmaher, 2014; Lewinson et al., 1994). On average most individuals experience one episode of depression every five years, and approximately 20 per cent endure a chronic unremitting course (Fava & Kendler, 2000). Youth who relapse tend to have residual symptoms that persist even after the disorder remitted (Birmaher, 2014). Residual symptoms refer to lingering symptoms of depression or depressed behaviour, such as withdrawal or anhedonia that do not alone meet diagnostic threshold. For example a young person who presents for treatment with low mood and four additional depressive symptoms, who by the end of treatment endorse one symptom will be at higher chance of relapse than a young person with no ongoing symptoms. The chronic, recurrent course of depression is a major clinical issue.

**Burden**

Depression is also considered a global health burden, currently ranked the third highest burden of disease in Australia, and predicted to be the number one health concern in the developed world by 2030 (ABS, 2009; WHO, 2008). The current trends are concerning as depression in the formative years is associated with a range of negative outcomes such as academic and interpersonal difficulties, increases in stressful life events, substance abuse, high rates of comorbidity and suicide (ABS, 2009; O'Neill, Connor & Kendal, 2011; Seeley et al., 2011; SAMSHA, 2008; Thompson, Connelly, Thomas-Jones, & Eggert, 20

13). Depression results in a large number of days absent from school or work and has a profound effect on the maintenance of normal social roles. The disorder often co-occurs with other mental illnesses such as anxiety, substance abuse and personality disorders, leading to further distress, disability and more complex treatment requirements (ABS, 2009; Rohde et al., 2013). Young people with a history of depression are not only at an increased risk for the re-emergence of a

depressive episode, but also have a substantial increase in the chance of developing bipolar affective disorder and persistent depressive disorder (previously classified as dysthymia; Costello et al., 2002; Kovacs & Gatsonis, 1989; Lewinson et al., 2000).

### **Consequences**

The onset of a depression in early adolescence is likely to have long term consequences as it corresponds with a key point in maturation where a number of social, physical, and emotional changes are taking place. Early adolescence corresponds with the transition to high school, increased significance of the peer group, increases in autonomy from parents, and puberty. Depression often results in a failure to complete milestones, consolidate skills, grapple with their own moral concepts, and navigate difficult interpersonal situations (i.e. peer pressure, intimate relationships) which are useful skills to take into adult life.

Classic childhood theories of development explain the impact of this disruption by way of suggesting that depression impacts on the final operation stage of development, which occurs from age 11-15 (Inhelder & Piaget, 1958; Hames et al., 2013). This stage consists of skills such as problem solving and integrating abstract ideas, as well as the ability to grapple with moral concepts.

Depression may disrupt the achievement of this stage as the symptoms have an adverse effect on cognitive and biological functioning needed to implement such higher order reasoning. Specifically symptoms such as reduced concentration, psychomotor retardation and poor motivation have been shown to have an reduce problem solving and academic achievement, especially in timed tests which require effortful processing (Wilkinson et al., 2001). Academic failure may contribute to negative learning experiences and can undermine self-belief in the ability to tackle new novel concepts at school.

In addition, symptoms such as social withdrawal, anhedonia and irritability can negatively influence social skills and peer interactions, leading to a reduction in the opportunity to grapple with interpersonal conundrums. Depressed young people have been found to be less socially competent, demonstrate poor eye contact and fail to reciprocate in interpersonal situations (Hames et al., 2013).

Such behaviours increase social isolation, and reduce the availability of others to practice interpersonal skills. Lengthy periods of depression during development disrupt normal progression and transactions with the environment which assist in the learning and development of new skills which will be used later in life (Kovacs & Goldston, 1991; Lewinson et al., 2000). The long term social and academic consequences of depression are likely to be more severe the earlier the disorder begins as it may disrupt the processes prior to achieving any milestones, resulting in a young person trying to catch up with peers who have more extensive skills (Costello et al., 2002).

The most immediate and devastating burden resulting from depression is the relationship to suicide. Depression is the psychiatric disorder most commonly associated with suicide and depressed mood may increase the risk for self-injurious behaviour due to significantly impairing judgement capacity (APA, 2013). In 1996 suicide was the second leading cause of mortality in youth, behind car accidents, however by 2010 suicide had moved into the leading cause of mortality (ABS, 2012; Harrison, Moller, & Bordeauz, 1997). The rates of suicide have shown steady incline from 1960 onwards, and these trends were especially strong in Aboriginal youth, who are at greater risk of completed suicide. A troubling trend has emerged in suicide rates among young men, with an increase of 71% between 1979 and 1997 (Harrison et al., 1997; Moon, Meyer, & Grau, 2000).

### **Treatment**

The Australian Government has recognised the burden of depressed and the requirement for early intervention, pledging a significant portion of the budget to early intervention. The Minister for Health announced in December 2014 that Headspace, an initiative for the early intervention and timely treatment of psychological disorders in 12-25 year olds, would open 15 new centres across the nation (Dutton, 2014). The goal of additional centres would be to increase the availability of treatment for depression and other psychological disorders, in the hope that it may translate to more young people receiving timely first onset interventions that reduce their long term risk of relapse. This is timely as the figures and trends suggest the rates of depression are rising in Australian adolescents, the disorder is appearing more debilitating and requiring higher levels of

community care. Treating depression has become an imperative in preventing youth suicide and other negative developmental outcomes.

Two areas of intervention gaining attention in the field of youth depression are prevention and early intervention. Prevention programs aim to provide young children with skills that may prevent depression, such as social functioning, thinking styles and effective communication. Prevention programs usually take place in pre and primary schools. An Australian designed and implement program, the Pals Preschool Program, teaches children as young as four years social skills for interacting with peers (Cooper, Paske, deHaa, Zuzic, 2003). The program is designed to prevent behaviours linked to later depression, such as poor social skills, which have been shown to predict conduct issues (Cooper et al., 2003). In addition, the Penn Prevention Program teaches cognitive strategies to all young people from age 10 with the goal of protecting against the first onset of a depressive episode (Jaycox, Reivich, Gillham, & Seligman, 1994).

In comparison, early intervention refers to the optimal treatment of the first presentation of depression in a young person. Optimal early intervention encompasses ameliorating the symptoms of the presenting episode, but also preventing the development or exacerbation of risk factors linked to relapse (Allen et al., 2007). Early intervention is complimentary with prevention as it seeks to provide targeted intervention and treatment for those young people who become depressed despite prevention. This is important as current rates of adolescent depression suggest avoiding the onset of depression altogether is not always possible. Evidence exists suggesting that intervention in the first episode may significantly reduce the chance of developing vulnerabilities linked to relapse, for example, Harrington and Clarke (1998) stated that assertive intervention during the first childhood episode would reduce the chance of relapse by 10 per cent. As such, intervening in the first episode of depression may halt the development of vulnerabilities, be they cognitive or social functioning, that are linked to relapse.

Early intervention in practice refers to the provision of treatments for depression currently endorsed by psychiatry, clinical psychologists and medicine. Currently two types of treatment exist for depression, psychopharmacological, such as antidepressants and mood stabilisers, and psychological therapies, such as cognitive behavioural therapy (CBT) and interpersonal therapy (IPT). Psychological therapies are the first choice of treatment in young people, as very limited evidence exists to support the use of antidepressants for youth. In fact, some research suggests the use of Selective Serotonin Reuptake Inhibitors (SSRIs) may be linked to increased suicidal ideation in young people (Bridge et al., 2007). CBT and IPT are best practice therapy, with rigorous clinical research to support their use as the first line of treatment (NICE, 2005). Currently, Australian young people can access a private psychologist who can provide these therapies at a subsidised rate through Medicare (Allen et al., 2007). However, these therapies render success rates of at best half and at many young people who complete a course of treatment have been found to relapse (Kennard et al., 2006). Current treatment guidelines call for further specificity of the type of treatment clinicians provide as presently the type of therapy is chosen based on the client presentation, their skills/ experience and the young person's preference (NICE, 2005). As the rates of depressive disorder rise within Australia, research objectives are to address poor rates of recovery using current best practice theory, and investigate the most cost effective use of public health such as Medicare resources (Littlefield & Giese, 2008; Martin, Swannell, Harrison, Hazell & Taylor, 2010).

### **Developmental Trajectory of Adolescent Depression and Individual Factors**

Appropriate and timely treatment of the first episode of depression is a priority as depression during adolescence is not merely a passing difficulty. For a proportion of young people, the early onset of depression is the beginning of a chronic disorder which disrupts social functioning across the life span. It has been suggested that adult depression is continuous with adolescent depression, and such assertions are supported by figures indicating that up to 84 per cent of individuals who were depressed during adolescence experience a further episode in adulthood (Rutter, Kim-Cohen, & Maughan, 2006). Additionally, depressed adolescents have two to seven

times the increased odds of recurrence in adulthood, compared to young people without an episode (Harrington et al., 1996; Rutter et al., 2006).

Research indicates that for a large proportion of individuals the risk and markers for the expression of depression occurs well before the age of 18 years. Specifically, it is possible that symptoms consistent with earlier onset are expressed very early in life and apparent to others. Mesman and Koot (2001) noted that parental ratings of their child's internalising behaviours in preschool were predictive of depressive disorders at age 10. In addition, Rutter and colleagues (2006) found that even when intelligence and childhood adversity were controlled, a direct risk was present from depression in childhood to adulthood of approximately 2-7 times. Indeed, it has been suggested that the onset in adulthood is much rarer and individuals who only present later in life may be the result of delayed later treatment seeking as the problem becomes more long term, pervasive and impossible to attribute to a developmental phase (Newman et al., 1996). Rates of relapse across the life span also suggest there is likely a subset of individuals who first become depressed early in life, and continue to experience episodic reoccurrences of depression (Birmaher, 2014; Kovacs & Delvin, 1998; Lewinsohn et al., 1986; Newman et al., 1992).

### **Biology**

An explanation for the early onset of depression in youth, has been subject to much empirical attention, with many theorists posing a biological explanation. Familial, twin and adoption studies have supported such a notion, with studies demonstrating that up to 40 per cent of the variance in depression may be heritable (Gold, Goodwin & Cheousos, 1988; Sullivan, Neale & Kendler, 2000). Family studies reveal a threefold increased risk for depression in the first-degree relatives of individuals with depression, when compared to the general population (Sullivan et al., 2000). Twin studies have revealed that much of the inherited tendency toward a mood disorder is likely through genetic factors as opposed to shared environmental influences (Sullivan et al., 2000). Inherited vulnerability is linked to earlier age of onset, more chronic course and a greater degree of symptom severity (Kendler et al., 1999).

Researchers propose that inherited genetic and neural vulnerabilities underlie the neurochemical dysfunction in the mood regulatory serotonin, norepinephrine and dopamine circuits, implicated in depression (Hasler, 2010; Mayberg, 2006). The genes of neurotransmitter systems, such as 5-HTT gene (Caspi et al., 2003), have been linked to depression and neurological imaging has revealed structural abnormalities in limbic-paralimbic (e.g. hippocampus) and subcortical (e.g. basal ganglia) regions among individuals with depression (Mayberg, 2006).

Early onset depression has also been associated with smaller in size genu (Lyo et al., 2002). The genu is an area at the bottom of the corpus callosum, which develops in parallel to cognitive processes in adolescence and deficits are thought to affect both working memory and emotion regulation (MacMaster, Carrey & Langevin, 2013). Symptoms of depression such as disrupted sleep and appetite which are controlled by bodily systems such as the limbic system and the hypothalamus further point to a biological underpinning. Biological or hormonal changes are also implicated in the high proportion of onset in girls at age 13, as this corresponds with the onset of puberty (Goodyer, Herbert, Tamplin, & Altham, 2000). It is suggested that the biological changes which take place in puberty may expose dormant inherited biological vulnerabilities relevant to early onset of depression. Current research indicates that an early onset of depression may be partially attributed to a genetic vulnerability intrinsic to the individual and that inherited biology may serve as a vulnerability (Abela & Hankin, 2008; Garriock et al., 2006).

### **Temperament and Emotion Regulation**

It is generally accepted that the biological vulnerability may shape or be exhibited through individual aspects, such as temperament (Lonigan et al., 2003 & Hankin, 2012). Temperament is defined as an emotional and behavioural style which is apparent early in a child's life and remains relatively consistent over time and situation (Compas et al., 2004). A central feature of temperament linked to early onset depression is emotion regulation, which may be a biologically rooted process (Silk, Steinberg & Morris, 2003). According to Thomson (1994), emotion regulation is an intrinsic process responsible for monitoring, evaluating and modifying emotional reactions,



especially their temporal features to accomplish one's goals. Emotion regulation is required in times when strong emotions, such as sadness, fear and anger are enacted, be it the result of profound life stress, or chronic daily hassles. The human desire to regulate emotions and establish equilibrium is apparent very early in life, such that when a baby cries it may need comfort from the primary caregiver to help it sooth. The development of mood regulation strategies thus begins very early in life, but continues to be modified throughout the course. During adolescence, young people are learning to regulate their own emotions and self-sooth during stressful periods; however these processes aren't yet fully formed and many of the hormonal, neural and cognitive systems thought to underlie emotion regulation are still maturing (Silk et al., 2003). As such, young people often require emotional support form peers and family in order to effectively regulate their emotional experiences (Longian et al., 2003).

Poor emotion regulation is a specific risk factor for depression, as the inability to regulate ones emotions results in sustained experiences of negative affect or low mood, consisting of feelings of sadness, anger or stress (Cole, Michel & Teti, 1994; Silk et al., 2003). Females and certain subgroups such as young people with medical disorders have been found to have higher levels of negative affect which corresponds a higher prevalence of depression in these groups (van Noorden, Giltay, den Hollander-Gijsman, van der Wee, van Veen & Zitman, 2010). Watson & Walker (1996) exemplified with a sample of young adults that trait measures of negative affect remain relatively stable across time spans of 6-7 years and that negative affect demonstrated the power to predict future levels of depression across this time span. Similarly, Silk & Morris (2003) found that adolescents who reported more lability in their emotions, had higher levels of depression.

### **Personality**

Sustained periods of negative affect are thought to have an adverse effect on a young person's developing personality and the way in which they relate to others (Ordonana et al., 2013). Personality, which is theorised to be an emotional, cognitive and behavioural style that remains relatively consistent over time and situation, is thought to give a distinct tone to a young person's

interaction style, social conduct and the manner in which they are perceived by others (Compass et al., 2004; Lonigan et al., 2003). Personality traits are heritable, and have demonstrated a strong causal link to depressive disorders over lifetime studies (Kendler, Kuhn & Prescott, 2004).

It is postulated that negative affect most clearly impinges upon and shapes the development of the personality trait neuroticism. Neuroticism categorises a higher order personality factor comprised of elevated scores on traits such as dysphoria, tension, hostility, emotional reactivity, low self-esteem, and sadness (Eysenck & Eysenck, 1967). Neuroticism is characterised by a tendency to experience negative emotion states and to respond to stress in manners that are disproportionate to the situation (Yoon, Maltby, & Joormann, 2012; Wienstock & Whisman, 2006). Neuroticism is thought to affect core beliefs, cognitions and the way in which people feel about themselves. Importantly, neuroticism has established biological foundations, being found to mediate the association between 5HTT-LPR genotype and lifetime major depression, accounting for 55 per cent of the genetic effect on depression (Kercher et al., 2009; Munafo, Clarke, Roberts & Johnstone, 2006; Weissman et al., 2005). It is also associated with the use of maladaptive coping styles such as rumination which prolong the experience of negative emotion states, and higher levels have been found in girls which coincides with higher levels of depression in current literature (Yoon et al., 2012). Neuroticism has been found to predict lower levels of life and relationship satisfaction, and higher rates of relapse in depression across the life span (Suldo, Minch, & Hearon, 2014). Research suggests neuroticism may be the expression of a genetic vulnerability closely associated with the early onset of adolescent depression.

### **Interpersonal Perspectives of Depression**

Adolescent depression is a disorder with firm interpersonal antecedents and consequences. Positive supportive relationships are thought to assist young people to regulate their emotions and assist with coping and self-esteem management, in a time of biological, social and emotional changes that inevitably bring about stress (Stice et al., 2004). However, depressed youth often encounter social rejection and as such do not receive adequate social support and assistance in

managing their emotions. This is concerning as cross section studies have demonstrated that low levels of perceived social support predict greater depressive symptoms in adolescence (Bogard, 2005; Field et al., 2001).

Interpersonal theories of depression begin from the premise that the characteristics and behaviours of depressed young people disrupt their social relationships by evoking negative responses from others (Coyne, 1976; Stice, Rohde, Gau & Ochner, 2011). Depressed young people themselves, and those around them, evaluate their social skills as inferior and report they are less rewarding for social contact (Rudolph, Flynn, & Abaided, 2008). They demonstrate less social competency in that they have a reduced range of cognitive and behavioural skills, as well as difficulty understanding others' emotions (Ordonana et al., 2013). Due to such social-behavioural deficits, they have a heightened chance of social rejection.

Such trends are worrisome as relationships are very important to young people, especially those with their peers, who are similarly trying to navigate the inherently stressful life stage. It is during adolescence that the network of significant others is restructured and friends replace parents in the central position (Helsen, Vollebergh & Meeus, 2000). At this time friends become the primary source of intimacy and support, seeing young people turn to their peers for assistance with normative stressors such as transition to high school, peer problems and romantic relationships. This change may be due to an objective match between the developmental strivings of the young person toward differential from the family and the need to maintain support while learning new coping mechanisms.

### **Social- Behavioural Deficits of Depressed Youth**

Depressed youth may have difficulty maintaining positive interpersonal functioning due to the social- behavioural deficits they display across peer, romantic and family relationships (Rudolph et al., 2008). Depressed young people are thought to become displeasing to others due to symptoms of the illness. Symptoms such as negativity, irritability, withdrawal and low energy are displeasing to others but also may diminish the opportunities for social interactions (Flynn & Rudolph, 2011;

Kercher et al., 2009). Feelings of worthlessness or excessive guilt may precipitate excessive reassurance seeking, feedback seeking, dependency and premature disclosure (Hammen, Shih & Brennan, 2004). In particular, girls who are depressed, tend to engage others in their depressive processes, such as co-rumination, which involves extensively discussing problems without coming to a solution (Rose & Rudolph, 2006). Characteristics such as, speaking in a slower voice, with less modulation, and the tendency toward more negative conversation topics are also aversive to others (Sergrin & Abramson, 1994).

Studies show that romantic partners of young depressed women tend to perceive them as less competent in interpersonal interactions, and teachers report depressed youth as being more helpless than their peers, and more likely to spend time alone (Rudolph & Clarke, 2001). During parent child interactions depressed young people demonstrate less autonomous assertion and confidence and are less able to compromise (Rudolph et al., 2008).

#### **Relationship Disturbances of Depressed Youth**

Such deficits inevitably disturb youth's relationships. Depressed youth tend to find rejection from all sources of support, including peers and parents. Parents of depressed children reported lower levels of intimacy and satisfaction in their relationship with their children, and children report higher levels of maternal criticism (Frye & Garber, 2005; Rudolph et al., 2002). Within the peer group, the disorder is linked to low popularity, isolation, bullying and poorer quality friendships (Borelli & Prinstein, 2006; Nolan et al., 2003). Depressed youth describe themselves as less effective in conflict resolution, less skilled at providing emotional support to friends, and are more likely to respond in a hostile manner to disputes (Hammen et al., 2004). Depressed women report receiving less support from their romantic partners than their counterparts, and that their relationships tend to involve higher levels of criticism (Daley & Hammen, 2002).

#### **Social Support Disturbances of Depressed Youth**

Unsurprisingly, depressed youngsters have difficulty sourcing support from their networks. The role of social support on psychological wellbeing is derived from an attachment perspective,

suggesting that individuals experience higher levels of wellbeing when a match is achieved between their objective requirements and the levels of support they receive. There is an established theoretical and empirical link between depression and the perception of low peer social support, with cross sectional and longitudinal studies demonstrated that a lack of social support significantly correlates with and predicts depression over time (Auerbach, Bigda-Peyton, Eberhart, Webb, & Ho,, 2010; Kendler et al., 2005; Stice et al., 2011; Windle, 1992).

According to the stress and coping perspective on social support (Haber, Cohen, & Lucus, 2007) the benefits of social support stem from the objective match between the needs of the support recipient and the level and type of support provided. While all social support is thought to be beneficial, the stress buffering hypothesis suggests it may have a key role in assisting with wellbeing in times of stress, namely to assist in emotion regulation and to protect against the negative consequences of stress (Dwyer, et al., 2014; Wheaton, 1985). Life stress has been systematically tested as a predictor of adolescent depression and found to be one of the most reliable variables associated with the onset of an episode (Goodyer et al., 2000). Life stress includes both major isolated events such as parental divorce, and multiple negative life events such as serious health problems or a succession of relationship breakdowns (Burton, Stice & Seeley, 2004). The theory posits that those with greater support from friends, family and other significant people are less likely to become depressed in the face of stress, as social support is thought to exert a stress buffering affect, assisting individuals to feel accepted and valued in their social environment. Social support is also thought to foster a greater sense of confidence and efficacy to regards to coping abilities. For young people support from their peers may normalise experiences, provide alternate means of coping and assistance with ego support (Sarason, Levine, Basham & Sarason, 1983).

Social support consists of two basic elements; whether an individual perceives they have a satisfactory pool of available (actual social support) others to turn to in times of adversity, and whether they believe that the support is of an appropriate standard (perceived social support; Sarason et al., 1983). Measures of actual social support pertain to concrete supportive behaviours

that are provided to recipients by support networks, whereas perceived social support is a measure of the recipient's perception of the quality and availability of the support (Haber et al., 2007). While received social support is a nonbiased measure, meta analytical studies have found that it only represents 15 per cent of the total variance of social support; as such, perceived social support may reflect a judgement of the quality and function of the support which is inherent to the individual (Haber et al., 2007). In adolescent research it has been suggested that it is most important to young people, how supported they feel as opposed to what supports actually exist (Lieberman, 1982; Haber et al., 2007). Ellonen et al (2008) found that the subjective fit between the adequacy of social support directly impacts on the severity of the depressive symptoms and inadequate social support may be most noxious for certain subgroups such as young adolescents and females. The perception that support is not adequate is likely to result in feelings of rejection and abandonment which serves to increase feelings of stress and emotional unease (Katainen et al., 1999). Multiple stressors and inadequate social support are a concern as they overburden coping resources, and thus put the individual at risk for developing depression (Burton et al., 2004; Paykel, 1994).

### **Role of Neuroticism**

Similar to the role of depressed mood in reducing social support, interpersonal theories of depression have investigated the role of personality traits, such as neuroticism, in undermining social support (Henderson et al., 1980). Neuroticism is characterised by a stable tendency to experience negative emotion states and to respond to stress in manners that are disproportionate to the situation (Yoon et al., 2012; Wienstock & Whisman, 2006). One implication of such instability is that social interactions are greatly impacted, and indeed, those high in neuroticism reporting significant difficulties forming and maintaining meaningful connections with their parents and peers, and less satisfaction in their relationships in general (Henderson, 1984; Hendrick & Hendrick, 2004; Swickert & Owens, 2010).

### **Neuroticism and Interpersonal Functioning**

Researchers have suggested neuroticism may exert influence by way of adolescent's mannerisms, expressions and social conduct in a manner that their peers may find aversive, resulting in difficulties developing and maintaining meaningful connections (Leskela et al., 2009; Paykel, 1994). Neuroticism has been found to reduce self-confidence and the degree to which individuals believe they have the skills to function in a socially appropriate manner. Gilbert (1991) suggested that poor social competency in neurotic individuals was related to their lack of friendly, pleasant or appropriate social behaviours, and studies suggest they are viewed as more needy and volatile. Santor & Rosebluth (2005) found that elevated scores heightened adolescents' interpersonal difficulties, but also precipitated the onset of specific peer problems such as friendship breakdowns. Individuals with high levels of neuroticism endorse conflict within their interpersonal domains, high levels of interpersonal stress and report that their social support is inadequate (Auerbach et al., 2010; Swickert & Owens, 2010).

### **Function of Social Support for Neurotic Individuals**

Inadequate social support may be particularly detrimental for neurotic individuals as it is a preferred method of coping and emotion regulation. Social support in particular, is theorised to have a protective role and offset the impact of the inherent heightened stress response and negative emotion states, those high in neuroticism experience (Suls & Martin, 2005). Dwyer and Colleagues (2014) found that social support had a positive buffering effect on severely depressed and highly neurotic young people, whereas no relationship was found for non-neurotic individuals. Such findings led the authors to conclude that social support may have a role in helping neurotic young people regulate their emotional experience, compensating for emotional instability. This stress buffering role may protect against depression in neurotic young people. Similarly, supportive relationships have been found to protect against the intrinsic vulnerabilities associated with neuroticism and there is a causal relationship between relationship support satisfaction and neurotic symptoms (Henderson et al., 1984).

Within the interpersonal literature neuroticism has very much been viewed as a vulnerability factor which may further exacerbate the interpersonal difficulties depressed adolescents face. Research suggests that neurotic individuals find benefit from social relationships and they may be particularly important in helping avoid or manage depression; however, the individual may not have the skill set to maintain them. Such a lack of interpersonal support may be particularly distressing for neurotic individuals who seem to require the most external assistance re-establishing emotional equilibrium.

### **New Directions: Moving from Unidirectional Theories to Transactional Perspectives**

Despite a large body of empirical literature demonstrating that personality and social support play an important role in adolescent depression, limited research has focused on their manner of interaction to explain the precipitating and maintaining mechanisms (Compas et al., 2004; Henderson, 1984). Traditional interpersonal theories have focused on unidirectional explanations, suggesting that depressed individuals are less rewarding for social contact either due to their state (depression) or trait (neuroticism) and this cascades into social rejection (Flynn & Rudolph, 2011; Kercher et al., 2009). Social rejection is then thought to adversely affect young people's ability to source support from their networks in order to manage high stress times, and this may be particularly devastating for neurotic young people who appear to derive the greatest benefit from social contact (Burton et al., 2004).

However, none of the variables in isolation have been able to account for the social disruption apparent in depressed youth. Specifically, limited evidence has supported social rejection in depressed youth when other variables such as personality are controlled (Joiner & Metalsky, 1995). Nor have comprehensive studies of the stress buffering hypothesis been able to support the role of social support in preventing depression when looked at in isolation (Burton et al., 2004). Furthermore, in a recent longitudinal study, Leskela and colleagues (2009) demonstrated that even in the event depression is treated neurotic individuals maintain low levels of social support and report ongoing interpersonal stress across the lifespan. Interestingly, support seeking results in



increases in negative emotionality, stress and emotional unease for individuals high in neuroticism, which suggests there are additional processes at play in for the stress buffering effect of social support for neurotic individuals (Katainen et al., 1999). The current criticisms may shed some light onto the findings that Interpersonal therapy (IPT) which aims to address social skills deficits, interpersonal sensitivities and role transitions in depressed youth, has a success rate of at best half, suggesting there is a subset of young people with social pitfalls who aren't responding to best practice depression treatments (Mufson, Dorta, Moreau & Weissman, 2004).

The current review of the literature suggests that it might be prudent to acknowledge that unidirectional interpersonal models of adolescent depression have limited prospective abilities to explain the etiology and course of the disorder, nor are treatments derived from them adequate. Current theoretical shortcomings may stem from the origins of interpersonal theories of depression, which originated from adult research. As such, the theories are noted to fail to incorporate the developmental context of adolescent onset depression. Specifically, a detailed account of the intersection between normative and atypical development, the continuous transition between young people and their environments, as well as the long term effect of an episode of depression on personality formation are not adequately addressed (Hankin, 2012; Rudolph et al., 2008).

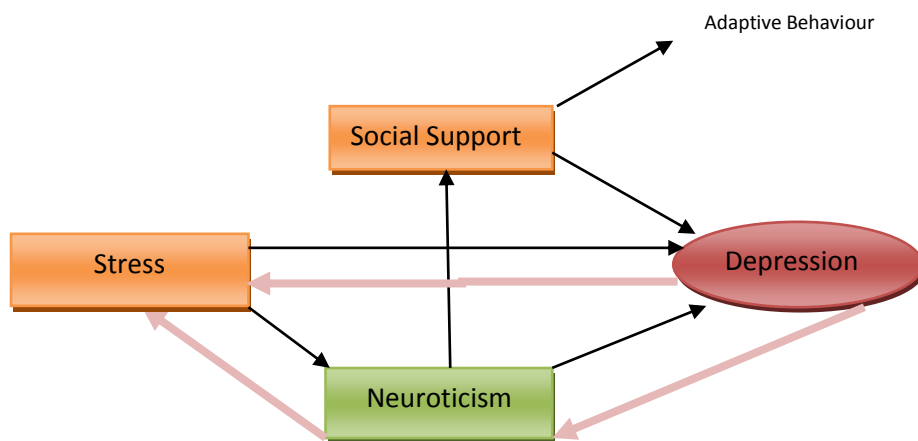
As such, influential theorists have suggested research efforts should be focused on how risk factors may work in unison to foster depressive pathology (Auerbach et al., 2011; Hankin, 2012; Kercher & Rapee, 2009). Specifically, Hankin (2012) suggests that there are likely additional aspects of interpersonal processes of depression than those presented, and as such, the variables are confounded and actually play an intersecting role in adolescent depression. Any contemporary model of adolescent depression needs to delineate the interpersonal antecedents, correlates and consequences of depression during one's youth (Rudolph et al., 2008). Specifically in order to move from prevalence to look at process, and to address current best practice treatment shortfalls, a detailed account of the antecedents, mediating, maintaining and consequential mechanisms is required to further the current field.

### **Cognitive Vulnerability Transactional Stress Model**

The Cognitive Vulnerability Transactional Stress Model (CVTSM) attempted to consolidate current depression theory and address criticisms to provide an integrated transactional framework with mechanisms for onset, maintenance and consequences of depression (Hankin et al., 2001). Such a theory can be applied to the relationship between neuroticism, social support and adolescent depression and may assist in explaining the chronic life course that early onset depression usually follows. The core concept is that the early onset of depression may reflect an underlying vulnerability, which is largely genetic in nature and may predispose the young person to an internalising disorder, but also to future transactions with the environment that is an antecedent to relapse (Costello, Erkanli, Angold, 2006; Hankin, 2012; Burcusa & Iacono, 2007; Shih et al., 2007).

The CVTSM theory begins from the premise that significant stress may cause negative affect in all individuals but negative emotion states will persist in those with pre-existing biological or temperament vulnerabilities, such as neuroticism. The pre-existing vulnerabilities may be a moderating factor between the stress and coping, but they may also serve to influence behaviour in a manner that creates further interpersonal stress that exacerbates the condition. As such, the theory accounts for both individual differences, and the role of an individual's behaviour plays in the process (Coyne, 1976; Hankin & Abramson, 2001). The model has been supported via a four wave prospective study, showing that adolescents with high neuroticism experienced greater levels of depression symptoms, and encountered more negative interpersonal life events over time (Hankin, 2006). Such interpersonal patterns are thought to have a profound effect on the young person's developing personality and social skills, which may partially explain why some individuals experience repeated episodes of the disorder across the lifespan. (Hames et al., 2013). In allowing for complex bidirectional transactions the theory builds on existing models in an attempt to understand the complex role of personality and social support in adolescent depression.

The three pathways are represented in Figure 1 below. The model begins from the premise that individuals process stressors through the lens of their personality, with personality traits such as neuroticism heightening the stress response and subsequent negative affect. In attempting to cope with stress, individuals may seek social support. Social support may be judged as adequate to meet the demands of the stressor, in which case adaptive coping will be the outcome. However in the event it is viewed as inadequate, depression may ensue. The pink arrows represent the stress generation and consequence model which suggest that depression may heighten stress, and that a sustained episode of depression during adolescent may lead to personality changes which further incite the stress response and generate additional interpersonal stress. These relationships will be discussed at length in the next section.



*Figure 1. Basic Cognitive Vulnerable Transactional Stress Model of the Proposed Relationship Between Depression, Social support, Stress and Neuroticism.*

**Pathway One: Intrapersonal Vulnerability**

According to vulnerability models, neuroticism is an individual specific vulnerability which precipitates the first episode of depression (Hankin & Abramson, 2001). Neuroticism is seen as an intrinsic vulnerability due to its influence on stress sensitivity, susceptibility to negative mood states,

inadequate coping and mood congruent cognitions (Kendler et al., 2004). Importantly, stressful events are seen as insufficient to contribute to depression without an underlying vulnerability such as neuroticism (Hankin & Abela, 2005). When faced with a stressor, those with high levels of neuroticism are more likely to experience a major depressive episode, than those low in the trait (Ormel, Rosmalen, & Farmer, 2004). Neurotic individuals overreact to stress and find it very difficult to regain emotional equilibrium. The combination of an inherent stress reaction coupled with lacking support is theorised to limit coping and result in sustained experiences of low mood (Auerbach et al., 2001).

### **Stress Response**

Neuroticism is posited to result in quick arousal in the response to stressful stimuli, and also such arousal then falls slowly following stimulation (Barnhofer & Chittka, 2010). Adolescence is inherently a very stressful life period, with Byrne & Mazanov (2002) finding that adolescents report a large number of stressors linked to depressed mood, such as peer problems, opposite sex interactions, future life concerns, academic worries and parental conflict. In addition, hormonal changes due to puberty, such as the activation of the hypothalamic-pituitary-adrenal axis (HPA) which regulates the body's response to stress, and the ongoing development of the prefrontal cortex, which relates to emotional control and decision making process further increases the likelihood that stressful events will be viewed as disproportionately harmful (Wolfe et al., 2006). Larson & Ketelaar (1991) found that during a mood induction, compared to normal controls neurotic individuals' demonstrated heightened emotional reactivity and an increase in negative cognitions when faced with the same stressor.

Heightened stress responses are problematic as they have a negative impact on an individual's appraisal of their ability to meet environmental demands. In the event a stressor is appraised to exceed an individual's intrinsic abilities, another evaluation of coping resources to manage the strain and cognitive discomfort is applied. Neuroticism has been found to result in

heightened appraisals of the threat of a stressor and the belief that the demands of are in excess of their coping resources (Gunthert et al., 1999). Furthermore, neurotic individuals have been found to worry about their abilities to meet the demands of stressors and also to engage in post event processing (Widiger, Hurt & Frances, 1984). As such, it is possible that during adolescence, which is a stressful life period encompassing rapid physical and social changes, the vulnerability that exists between stress and neuroticism becomes pronounced.

### **Cognitive Vulnerability**

The intrinsic stress reactivity found in neurotic individuals may also enhance cognitive reactivity. Cognitive reactivity relates to the ease at which patterns of negative thinking are activated in response to low mood and stress (Barnhofer & Chittka, 2010). Cognitive reactivity increases the accessibility of the failure beliefs, negative automatic thoughts and schemas about the self (Christensen, Carney, & Segal, 2006). Kercher et al (2009) found with a sample of adolescent girls that failure beliefs mediated the relationship between neuroticism and depression. In addition, neuroticism is thought to predispose individuals to respond to stress and low mood, with rumination, which prolongs the experience of negative cognitions and has been linked to depression (Barnhofer & Chittka, 2010; Roelofs, Huibers, Peeters & Arntz, 2008). Negative cognitions often lead to preferential attention toward confirming stimuli, which serve to reinforce negative beliefs, and impede the processing of disconfirming evidence. Cognitive reactivity has been found to be stable, and exist for neurotic individuals even when in remission from depression (Seeds & Dozois, 2010). Once a tendency toward cognitive reactivity is established, it has been suggested that negative cognitions can be easily reactivated by relatively benign stressors or subtle changes in mood (Scher, Ingram & Segal, 2005). Specifically, Barnhofer & Chittka (2010) found that the higher the level of neuroticism a previously depressed individual had, the more likely they would be to respond to negative affect with the reactivation of negative cognitions, which was linked to current levels of depression. As such, cognitive reactivity appears linked to neuroticism and may be an innate risk factor for the early onset of depression.

### **Coping Strategy Choice**

A specific consequence of heightened emotion reactivity and negative coping appraisals is the utilisation of ineffective coping strategies, which may lead to a further decrease in mood, more prominent failure beliefs, and chronic stress (Hankin, 2008; Hankin & Abramson, 2001). Coping strategies include attempts to manage or resolve conflicts, to reduce emotional distress or both (Caltabiano, Byrne, Martin & Sarafino, 2002; Folkman, Lazarus, Dunkel-Schetter, DeLongis & Gruen, 1986). Coping has been described as a personality process under stress, with research demonstrating that personality and coping have a shared genetic basis (Kato & Pedersen, 2005). Personality can either facilitate or constrain coping by affecting coping strategy selection, for example individuals high in neuroticism, favour emotion based coping comprised of strategies to minimise negative emotions such as withdrawal and wishful thinking as opposed to problem focused action strategies that attempt to influence the source of the stress (Connor-Smith & Flachsbart, 2007; Lazarus & Folkman, 1984). Murberg (2009) studied a sample of Norwegian youth and discovered that neurotic subjects were most likely to use strategies, such as hostility, venting, co-rumination (excessively discussing problems with others) and passivity. Whereas, high extraversion, characterised by traits such as enthusiasm, talkativeness and assertiveness, was related to problem solving strategies and cognitive restructuring, which reduced the experience of negative affect (Murberg, 2009). Whilst there is no consensus on the universal utility of problem focused coping, it generally predicts better physical and mental health, while emotion focused predicts poorer overall outcomes (Compas et al., 2001). Personality may affect coping strategy selection directly by constraining or facilitating use of specific strategies or indirectly by influencing the nature and severity of stressors experiencing or the effectiveness of coping strategies (Bolger & Zuckerman, 1995). As a result of poor coping, chronic stress may ensue, and the result of such is the strengthening of existing cognitive vulnerabilities.

### **Pathway Two: Interpersonal Vulnerabilities**

The second pathway in the CVTSM model suggests that, in part due to the intrinsic susceptibilities discussed above, individuals with personality vulnerabilities are more inclined to generate interpersonal stress (Hankin & Abramson, 2001). Specifically, vulnerable individuals have been shown to experience the same rates of stressors occurring by chance as others in the general population (i.e. death of a family member), but they have higher rates of dependent stressful events (i.e. relationship break down, job loss), particularly within the interpersonal domains (Hammen, 2006). As such, neuroticism may have a role in the maintenance and chance of relapse in depression due to influencing behaviour in a manner that has been linked to increased stress generation (Weinstock & Whisman, 2006). Munafo et al (2006) suggested that an individual's personality plays a substantial role in influencing exposure to environmental adversity and this may be the mechanism through which neuroticism mediates the association between stress, social support and depression. Such transactional frameworks posit that individuals actively contribute to the occurrence of stressors they experience through stress generation (Hammen, 1991). Interestingly, stress generation appears to be unique to depression, with studies investigating the phenomenon in bipolar, anxiety disorders and schizophrenia, not replicating the finding (Auerbach, Laskin, Frantsve, & Orr, 2001).

### **Stress Generation**

Comprehensive studies of stress generation have attributed dependent stress to high trait neuroticism (Leskela et al., 2009; Paykel, 1994). Specifically it has been demonstrated that neurotic individuals are more likely to experience negative events that are dependent on their own behaviour, above and beyond the variance explained by depressed mood (Hammen, 1991). Specifically, Uliaszek and colleagues (2012) investigated the role of personality and stress generation in a large longitudinal study of adolescents and found that those with a moderate to severe depression had more dependent life stress, and that neuroticism partially accounted for this

relationship. In addition, individuals high in neuroticism have the greatest chance of developing depression from stress in the interpersonal domain, and they also place high importance on relationships while at the same time endorsing conflict within them (Auerbach, Bigda-Peyton, Eberhart, Webb & Ho, 2010; Lara, Leader & Klein, 1997). Hankin and Colleagues (2009) found that initial levels of neuroticism in an adolescent sample predicted the occurrence of additional life stressors and these stressors explained the association between neuroticism and depression over time. Similarly, Gunthert et al. (1999) found that differences in neuroticism did not result in significant variation in reports of academic, health or work stressors, however they reported significantly more interpersonal stress. Individuals who are both depressed and neurotic have been found to generate the highest number of dependent stressors in the interpersonal domain, and this relationship remained even when depression was treated (Daley et al., 1997). Research suggests that the stress generation vulnerability is intrinsic to neurotic individuals, occurs in the interpersonal domain, and remains apparent when individuals are in remission from depression (Hammen 1991; Hammen & Brennan, 2002).

### **Mechanisms**

Although the link between neuroticism, stress and depression is unequivocal, mechanisms underlying this link remain unclear (Yoon et al., 2012). It has been suggested that one of the ways through which stress generation occurs is that neurotic individuals engage in interpersonal stress generating behaviours such as excessive reassurance seeking (Gunthert et al., 1999). Excessive reassurance seeking is a relatively stable tendency to excessively and persistently seek reassurance from others that one is loveable and worthy, despite reassurance already being provided (Swann, Martin, & De La Ronde, 1992). A pattern has been suggested whereby support networks originally offer encouragement and reassurance to the individual, however, over time there is an inconsistency between other's reassurance and support, and the way in which the individual feels about themselves. In order to manage their affective state the person increases the expression of



the symptoms, with the goal of gaining further reassurance. Behavioural changes result from this dissidence and result in attempts to gain additional support by way of excessive calling, texting, reassurance seeking, co-rumination, and emotional displays. Over time, such behaviours have a negative effect on relationships that were previously protective (Leskela et al., 2009). Alternatively, individuals with limited social support may rely too heavily on a small number of specific relationships which may burn out supportive individuals. In line with Coyne's interaction model (1976) such behaviour elicits rejection from others and in adolescent samples excessive support seeking has a demonstrated link to support group burn out, and results in the individual being viewed as less competent and friends withdrawing (Ruldolph et al., 2008). In a meta-analysis, such excessive reassurance seeking was associated with negative reactions from others, increases in symptom expression and social rejection (Starr & Davila, 2008). Over time, such behaviours have the opposite effect than was desired, and precede rejection.

Overall, the behaviour of neurotic individuals is theorised to create a troubled interpersonal context which reduces their ability to gain and maintain supportive individuals, but also burns out existing supports (Hames et al., 2013). Further attempts to gain support and establish emotional equilibrium only serve to heighten interpersonal failures (Gunthert et al., 1999; Windle & Windle, 1997). As such, the individual often remains in the uncomfortable affective state, whereby they perceive their social support to be inadequate and begin to experience chronic stress. The experience of chronic stress would then contribute to higher levels of depressive symptoms (Hammen, 2006). Auerbach and colleagues (2011) found just this, using a sample of adolescents, they demonstrated that those with low social support experienced a greater occurrence of dependent interpersonal stress, and this stress partially mediated the relationship between social support and depression. From the research, it is possible to conclude that depression relapse may partially be explained by stress generation, which mediates the relationship between personality and stress, to partially explain the link between social support and depression (Haber et al., 2007).

### **Pathway Three: Consequences and Maintenance Factors**

It is hypothesised that experiencing a sustained episode of depression during one's youth may lead to the development of maladaptive social cognitive processes and negative self-evaluation, that persist after the depression remits. Such vulnerabilities become a risk factor for relapse, and this may explain the high rates of lifetime relapse found in childhood onset depression (Jylha, Melartin, Rytsala, & Isometsa, 2009).

### **Scar hypothesis**

The Scar Hypothesis postulates that individuals are permanently changed by an episode of depression, in much in the same way a cut leaves a visible scar when it heals (Rohde et al., 1994). These changes may permanently worsen an individual's depression vulnerabilities including cognitive and personality risks and these are thought to persist after the depression remits (Lewinsohn et al., 1981). The majority of the research into the scar theory was conducted with adults and failed to support medium-long term changes in personality traits such as neuroticism after recovery (Jylha et al., 2009). However, research focusing on young people has found a range of personality and psychosocial changes, suggesting that during the formative years a depressive episode has a long-lasting impact on personality. Puig-Antich and Colleagues(1985) found that measures of school behaviour and academic achievement returned to baseline upon recovery, whereas relationships with peers, mothers and siblings continued to be problematic. In addition, Rohde and colleagues (1994) found psychosocial scars following remission such as increased support seeking in their adolescent sample. Nolen-Hoeksema et al (1992) suggested the scar theory might be especially relevant to young people as they are developing their view of the world in conjunction with their achievements and contrasting this against their peers. Should they find their interpersonal skills lacking it may interact with their thinking style and core beliefs to foster negative beliefs which are easily accessed in the future. Thus, the young person becomes more vulnerable due to

having had an episode of depression when their personalities are still forming which heightens their chances of a re-emergence of the disorder.

### **Life Stress Theory and the Kindling Effect**

The scar theory is complimentary with the Life Stress Theory and the Kindling Effect, which both attempt to explain the manner through which an episode of depression during one's youth may continue to have a profound effect on psychosocial functioning across the lifespan. The Life Stress Theory begins from the premise that the first episode of depression is usually reactive in that it is triggered by an important psychosocial stressor, while subsequent episodes become increasingly endogenous (Monroe & Harkness, 2005; Post, 1992). Life stress refers to major negative events such as death of a loved one, serious illness or the loss of employment. External life stressors play a greater role in the onset of the first episode of depression than on subsequent episodes, implying a reduced need for external triggering (Allen et al., 2007). Kendler and Colleagues (2000) confirmed these findings, demonstrated that the impact of stressful life events on the onset of depression decreased with every depressive episode, and importantly that the more robust predictors of subsequent episodes were individual characteristics.

The Kindling Effect suggests that initial depressive episodes and the stress which occurs as a result may be traumatic enough to cause neurobiological changes, which render the individual more sensitive to stress (Abela & Hankin, 2008; Monroe & Harkness, 2005; Post, 1992; Seeds & Dozis, 2010). Specifically, the initial stress is thought to sensitise the brain and results in negative affect becoming activated for increasingly minor stressors. Findings cited in support of the Life Stress Theory and Kindling Effect have remarked that individuals who are depressed and also experienced trauma as children exhibited six times more adrenocorticotrophic hormone response to mild stress than depressed controls with no abuse history (Heim, Newport, Heit, Graham, Wilcox & Bonsall, 2000). The theory suggests that an episode of depression during adolescence can be conceptualised as an initial stressor, and that this may worsen existing vulnerabilities or result in biological changes

for certain individuals which then renders the person susceptible to become depressed in response to minor stressors in the future (Monrie & Harkness, 2005; Post & Weiss, 1999).

### **Cognitive Changes: Negative Self Views**

In addition to biological changes, the scar theory also suggests that cognitive changes might arise from depression during adolescence. Specially, youth who consistently experience interpersonal dysfunction as reflected from their own social behavioural deficits, may develop negative beliefs about their own interpersonal competency. If interpersonal rejection occurs as a one-off the young person can make global attributes (i.e. the person was in a bad mood today), if it occurs continuously they begin to make depressogenic stable inferences about the self (i.e. no one wants to spend time with me because I'm boring).

Repeated social failures may cause the individual to internalise the blame and feel helpless regarding their ability to change the current situation (Rudolph, Abaied, Flynn, Sugimura, & Agoston, 2011). A perceived loss of control over their social world, may precipitate negative emotions, feelings of worthlessness, negative cognitive attributions (i.e. this is all because of me) and feelings of hopelessness (Hankin & Abramson, 2002). Such negative cognitive styles can become fixed and difficult to change, without therapeutic intervention. Together, these cognitive characteristics of depression not only constitute aetiological factors of depression, but also maintain and potentially exacerbate depressive symptoms via vicious cycles of perseverance of negative affect.

### **Behavioural Changes: Social Skills Deficit**

In an extension of the scar theory, it is hypothesised that the personality changes which arise from one episode of depression might be sufficient to cause poor social skills as the early onset of transactions with the environment that result in depressed mood and strain social networks may impede the mastery of developmentally important social skills. Conceptual definitions of social skills include 'the ability to maximise the rate of positive reinforcement and minimise the strength of

punishment from others' (Libet & Lewinsohn, 1973). As such, it reflects a specific set of interpersonal abilities which result in interactions with other people in a way that is both appropriate and effective for getting one's needs met (Segrin, 2000). There is evidence to suggest social skills are impaired by high levels of emotional reactivity and core skills of social competence such as empathy are impacted by strong emotions (Davies, Stankov, & Roberts, 1998). In a one-year study by Herzberg and colleagues (1998) individuals who reported long term chronic interpersonal stress demonstrated poorer social skills. In a study of marital couples whereby one partner had been depressed the interaction between the two was described as more verbally aggressive, less constructive and reduced ability to problem solve (Kahn, Cohen, & Margolin, 1985). Furthermore, Rohde and Colleagues (1990) found that social skills ratings remained lower in once depressed individuals, than never previously depressed individuals up to two years after remission from the episode.

The inability to master social situations may be a maintenance factor for further episodes of depression as social abilities remain low event in the event depression remits. Poor social functioning is comprehensively linked to relapse in depression and this core individual, and also behavioural vulnerability, may help explain high rates of relapse in childhood onset depressive disorders. This is especially pertinent as failure to establish rewarding social relationships may be especially devastating for younger people who are just beginning to expand their social networks, and have less developed coping strategies.

These maintenance factors then are thought to result in additional vulnerability factors which given another instance of stress begin the chain reaction again and possibly cause further damage. As such, the consequence model accounts for the impact of a sustained period of depression in the developmental years, on the development of a young person's personality (Flynn & Rudolph, 2011). The current literature suggests that neurobiological, cognitive and personality changes from one episode of depression prior to maturation could render an individual more

susceptible to depression, serving as a latent vulnerability factor but also a mechanism for relapse (Garriock et al., 2006).

### **Additional Exploratory Variables**

#### **Sex and Age Differences**

It is important to incorporate age and sex variables in any exploratory model of adolescent depression, as differences have been well documented. Current literature highlights a high preponderance of female onset of depression at age thirteen. In addition, females report higher levels of overall stress, generate more stress that is interpersonal and use less effect coping strategies which tax their personal relationships (Prinstein & Aikins, 2004). Females have been found to be more likely to seek out social support than males, and socialisation differences are thought to influence the importance of social support for males and females (Auebach et al., 2011). Females and those lower in neuroticism have been found to have more robust social ties and their satisfaction with their social support tends to be higher.

In addition, Shih (2007) found that being female and placing high importance on interpersonal relationships resulted in a greater number of dependent stressors and predicted depression. Females have been found to favour emotion based coping such as rumination and peer support which corresponds to higher rates of stress and depression within this group (Jose & Ratcliffe, 2004). The interpersonal area appears to be is a more salient area of vulnerability in females compared to males, with research demonstrating mixed findings on the role of peer social support in depression for teenage boys (Pettit et al., 2011). Research has called for further exploration of sex differences in personality vulnerabilities and the relationship to depression (Coyne & Wiffen, 1995).

### **Personality Traits**

Although neuroticism is the most comprehensively studied personality vulnerability, prominent personality researchers such as Costa & McCrae (1995) found evidence for a five-factor model of personality. The five factors are extraversion, neuroticism, openness, agreeableness and conscientiousness. The factors are demonstrated to be relatively stable across age groups and culture (Hendriks, Hofstee, & Raad, 1999). Extraversion, agreeableness and openness have been linked to social support and depression and as such are important to include in any exploratory study. Extraversion includes aspects such as positive emotionality, sociability, assertiveness and sensitivity to reward. Such traits are linked to having an outgoing nature, positive social functioning and persistence in coping. In addition, higher levels of extraversion are thought to be protective against depressive episodes (Connor-Smith & Flachsbart, 2007). Extraversion may influence a young person's ability to create and maintain social support networks, and thus source support in times of needed (Kendler, Gardner, & Prescott, 2003). The absence of extraversion have been found to result in young people experiencing increased depressive symptoms and feelings of loneliness (Joiner, 1997).

Agreeableness and openness are also positive predictors of interpersonal functioning with traits such as trust, compliance, altruism, flexibility and openness to experience thought to be positive interpersonal traits. In addition, agreeableness is comprehensively linked to high levels of perceived and actual social support (Tong et al., 2004). While the final personality trait, conscientiousness, has limited theoretical relevance in interpersonal behaviours, and is linked to functioning in other domains such as academic performance (Derryberry et al., 2003).

### **Summary**

There is no doubt that the relationship between personality, social support and adolescent onset depression is very complex. Relationships are very important to young people. The sheer

amount of time spent in pursuit of friendships and the significant distress that alienation from the peer group results in, highlights that being socially connected is an important developmental striving. As such, it is no surprise that one of the most robust predictors of adolescent depression is inadequate social support (Mufson et al., 2004). However, this relationship is likely confounded with personality, which has been found to predispose the individual to poor social functioning due to intrinsic stress reactions, but also indirectly, through stress generation and straining interpersonal relationships. The indirect effects of these patterns is thought to result in a vicious cycle of stressful conditions which may permanently change a young person's developing personality, resulting in enduring psychosocial deficits that may, in part, explain the high rates of relapse in adolescent onset depression. As such, there exists a need to investigate the manner in which the variables may intersect and influence one another. The CVTSM provides a framework for understanding the reciprocal relationship between personality, social support and depression via three interacting pathways. Understanding the multiple dimensions and variables involved in adolescent depression is important, as neuroticism is a temporal order stable variable which is thought to be relatively resistant to treatment over time. As such, a focus on factors which mediate the relationship between neuroticism and depression, such as social support, would provide treatment inlets (Barnhofer & Chittka, 2010). Such a focus on mechanisms and treatment inlets is timely as current rates of depression suggest that the disorder is not a passing difficulty, but instead that the early onset leads to a more severe developmental trajectory which is becoming an increasing burden on the Australian Health System.

### **The Studies: Context Statement**

From the literature reviewed, there exists a significant gap in the understanding of adolescent depression and psychosocial functioning. Current research has begun to acknowledge the multiple transactions which take place between young people and their social environments, and the role that these have not only on the development of personality, but also that personality also plays a role in shaping one's social world. Personality and social support are two important



concepts established as factors in the onset, maintenance and relapse of adolescent depression. Social support has a marked importance for young people, but it may also represent an area suitable for early intervention. The current study sought to expand the extant research base and is the first to the author's knowledge to investigate neuroticism, social support and depression in Australian adolescents, using a transactional perspective via a two year longitudinal design. Such an approach allows for the careful examination of vulnerability factors, stress, and depressive symptoms and thus clearly delineates the role of social support and personality on the emergence, maintenance and chance of relapse in adolescent depression.

The thesis is exploratory in nature, and aims to explore the relationship between the main variables using the three interacting pathways presented by the cognitive vulnerability transactional stress model (CVTSM). The model posits that 1) That cognitive vulnerability and stressors are predictors of depressive symptoms (vulnerability model), 2) depressive symptoms and cognitive vulnerabilities as predictors of stressors (stress generation model), 3) depressive symptoms and stressors as predictors of vulnerabilities (consequence model). Additional variables with a theoretical link to the main variables were also explored. These include gender and age differences, coping styles, and additional personality traits such as extraversion.

The current thesis is comprised of two studies. The first being a cross sectional exploration of the data, and the second being a longitudinal follow up, which surveyed a proportion of the initial respondents, two years later. A follow up study was required, as adequate time is needed to test personality and depression changes. The aim of first study was to replicate the first part of the CVTSM- the vulnerability model, which would investigate the well-established relationship between neuroticism and depression and also social support and depression. The aim of the second study was to test parts two and three of the CVTSM, the stress generation and the consequence model.

## Research Questions

### Vulnerability Model

1. How much symptom variance in depression is explained by neuroticism?
2. How much symptom variance in depression do the other variables explain?
3. Do interpersonal stressors exhibit a stronger relationship to depression than overall stress?
4. Does neuroticism mediate the relationship between stress and depression? Does coping style influence this relationship?
5. Does social support mediate the relationship between neuroticism and depression?
6. Do negative life events have a direct relationship to depression that is not impacted by personality?
7. What is the best model fit to explain the interaction between stress, personality and the social support variables to predicting cross sectional depression scores?

### Stress Generation and Consequence Model

1. Does time one neuroticism predict higher levels of stress at time two? Does this occur across the spectrum, or only in the interpersonal domain above and beyond that explained by depression?
2. Does time one depression predict time two stress?
3. Does neuroticism partially mediate the risk for depression through the generation of stress?
4. Does time one depression predict time two neuroticism when initial levels of depression are controlled?
5. Does time one depression result in reduced social support at time two? Does this relationship exist if neuroticism is controlled?
6. Did depressed and non depressed youth differ at time two when neither group was in a depressive episode. Did these differences exist at time one or did they come into existence as the result of the depressive episode.

7. Do negative life events or high levels of stress at time one result in increases in neuroticism at time two?

**Overall**

1. What is the final model for the variables which best explains the relationship between personality, social support and depression?
2. Does gender or age make a significant contribution to the model?
3. Does social support mediate the relationship between stress and depression (stress buffering hypothesis)?
4. Is there a significant difference between non depressed, mild/moderate and severely depressed youth in any of the relationships?

## CHAPTER TWO: Study One

**Overview**

The current study aims to explore the relationship between neuroticism, social support and depression in Australian adolescents. A second aim was to explore additional variables that have been linked to the relationship. In order to do this, information was collected on individual differences in personality traits, depressive symptoms, coping strategies, social support and stress. It is expected that neuroticism will exhibit a strong relationship to depression and that social support and stress will significantly influence this relationship.

**Method**

The aim of the present study was to investigate the relationship between neuroticism and social support on depression levels in Australian adolescents. Neuroticism and inadequate social support are understood to be predictors of adolescent depression which may interact to trigger and prolong the disorder. Stress will be examined as an addition measure due to research which suggests that personality vulnerabilities only become apparent in high stress times. In addition, negative life events will also be measured by way of control as no relationship is expected between personality and independent negative life events. Gender differences will be explored as will a young person's coping style. A cross sectional design was used for the first study to explore the variables.

**Participants**

The present study was cross sectional and sourced Australian adolescents via the convenience of their educational institutions. The study surveyed 360 adolescents in grades 7-10 across Australia. The gender and grade breakdown of the participants is presented in Table 2 and Table 3 below. The *M* age was 14.4 (*SD* = 1.2), with a non-normal distribution; skewness of -0.31 (*SE* = 0.13) and kurtosis of -0.6 (*SE* = 0.26). Five schools from Australia participated, two in Canberra, one in South-Western NSW, one in Southern NSW and one in Western Australia. Two of the schools were

Catholic, one was a private Anglican School and two were Government Schools. Four were co-educational and one was a single sex (girls) school. Participants were recruited based on class availability in each school and while their representation of a typical student for that school cannot be ascertained, the sample parameters suggest the ability to generalise the results to Australian youth is high.

Table 2

*Gender breakdown for the sample*

Gender	Total	Percent Surveyed	Final Sample	Percent of N
		(%)		(%)
Male	151	41.9	147	42.7
Female	197	12.5	1	57.3
Missing	12	3.3	0	0
Total (N)	360	100	344	100

Table 3

*Grade breakdown for the sample*

Grade	Total	Percent Surveyed	Final Sample	Percent of N
		(%)		(%)
7	64	17.8	64	18.6
8	45	12.5	44	12.6
9	135	37.5	134	39
10	104	28.9	102	29.7
Total (N)	360	100	344	100

## Measures

The study utilized a battery of online questionnaires. All participants completed demographic questions (age, school year and gender), the generation of a code unique to each individual, the nomination of their three closest friends, and six self-report measures (Appendix C).

Participants generated a unique code as per the guidelines developed by Yurek, Vasey & Havens (2008) to allow the data to be anonymously linked over two time points for a follow up study. The unique code consisted of person specific but not readily identifiable information such as their mother's maiden name and the number of brothers they have. Participants also nominated their three closest friends to allow for a cluster analysis of friendship groups to be undertaken.

The self-report measures were : The Junior Eysenck Personality Questionnaire (JEPQ ; Eysenck & Eysenck, 1975; Francis & Pearson, 1988); the NEO-Five Factor Inventory (NEO-FFI; Costa & McCrae, 1985); The Centre for Epidemiological Studies Depression Scale (CES-D; Eaton, Muntaner, Smith, Tien & Ybarra, 2004); The Short Form Social Support Questionnaire (SSQ; Sarason, Levine, Basham & Sarason, 1983), The Adolescent Stress Questionnaire (ASQ; Byrne, Davenport & Mazanov, 2007), The Life Events Questionnaire (LEQ; Newcomb et al., 1981), and The Adolescent Orientation For Problem Experiences Dispositional Inventory (A-COPE; Patterson & McCubbin, 1987). Each of these is described in detail below.

### Measures of Personality

*JEPQ.* Aspects of personality were measured with the JEPQ, a scale designed to measure three orthogonal dimensions of personality: extraversion (E), neuroticism (N) and psychoticism (P). It also contains a lie scale to evaluate threats to validity such as overly positive self-presentations. The measure consists of 81 items, including, "Are you an irritable person?" and "Are you the life of the party?" that are answered on a dichotomous yes/no basis. Extraversion refers to an individual who is thought to be under stimulated with the tendency to be outgoing, energetic, sociable and to

experience positive emotions (Eysenck, 1991). Whereas neuroticism measures an individual's tendency to be nervous, sensitive and to readily experience unpleasant emotions such as sadness (Eysenck, 1991; Williams, 1990).

Over twenty three studies have demonstrated that the JEPQ is a reliable and consistent measure of personality when analysed with self-report across different cultures (Caruso & Edwards, 2001; Eysenck, 1991; Ferrando, Chico & Lorenzo, 1997; Maltby & Talley; 1998; Ormel & Rijdsdijk, 2000). Internal consistency alpha reliabilities range from .78 to .87, and concurrent validity ranged from .59 to .95 (Aluja, Garcia, & Garcia, 2002; Katz & Francis, 2000). The JEPQ is not freely available in the public domain and was purchased for use in the study by The Research School of Psychology, ANU.

*Modifications to the JEPQ.* Psychoticism and overly positive self-view were omitted from the current study as they do not have a sound theoretical link to the main variables (Alexopoulos & Kalaitzidis, 2004). Namely, studies suggest high scores on psychoticism may reflect an individual at greater risk for a psychotic or Cluster A personality disorder as opposed to a unipolar mood disorder (Gillespie et al., 2008). In addition, the median result of over twenty three studies demonstrated that the P scale has the lowest test-retest reliability coefficient .68, significantly less than the N scale ( $r=0.8$ ) and the E scale ( $r=0.73$ ; Caruso & Edwards, 2001). Furthermore, as the questionnaire is anonymous the risk of secondary gain from socially desirable responding was deemed low (Holtgraves, 2004). The JEPQ utilized in the current study consisted of 41 items, measuring neuroticism and extraversion.

*NEO-FFI.* While research into personality disorders has primarily focused on E & N aspects to explain vulnerable structures influential researchers such as Costa & McCrae (1985) focused on a five factor model to measure personality. Two additional structures are Openness (O) and Agreeableness (A), the first of which refers to an appreciation of emotion, art and adventures and a tendency toward curiosity and exploration whereas agreeableness is a person's tendency to be

compassionate, cooperative and ever tempered (Costa & McCrae, 1985). Some current evidence suggests that Openness (O) and Agreeableness (A) may be linked to affective disorders and social skills (Bienvenu et al., 2004). Therefore, O and A were included in the current study to measure their association with depression, neuroticism and social support. Participants indicate on a scale of 0=very inaccurate to 5=very accurate how accurately a sentence such as "I am interested in other people" describes them. Both O and A have been shown to have concurrent and face validity, and reliability coefficients of .71 and .69 respectively (Viswesvaran & Ones, 2000).

### **Depression**

*CES-D*. Depressive symptoms were measured using the CES-D which is a commonly used self-report scale with normative data for adolescents in community and clinical samples (Hogue & Steinberg, 1995). The scale has high internal consistency of .85 in nonclinical and clinical samples, moderate test-retest coefficients ranging from .51 to .32 for time intervals varying between 2 weeks and 12 months and moderate correlations with convergent measures of depression (Caracciolo & Giaquinto, 2002; Radloff, 1991). Items on the scale reflect the four dimensions of depression outlined in the DSM-V (APA, 2013), including depressed affect, lack of positive affect, somatic retarded activity, and interpersonal relations (Radloff, 1991).

The scale consists of 20 questions and respondents indicate how often they have experienced symptoms of depression over the past week, on a 4-point scale ranging from 0 (rarely or none of the time) to 3 (most or all of the time) for questions such as 'I was bothered by things that usually don't bother me' and 'I did not feel like eating; my appetite was poor'.

A score of 16 or higher is indicative of mild depressed mood, however some evidence suggests that young adolescents tend to inflate self-report questionnaires due the emotional instability brought on by hormonal changes in this developmental period (Radloff, 1991). To ensure pathological levels of mood disorder were analysed a conservative cut off of 20 was utilized for the



current study to indicate mild/ moderate depressed mood (Radloff, 1991). Scores greater than 40 indicate depressed mood consisted with a severe major depressive episode (Radloff, 1991).

### **Social Support**

(SSQ). Social support was measured using the short form of the SSQ which is widely used and validated in research settings cross culturally (Martinez & Lau, 2001; Sarason et al., 1983). It has been shown to have adequate internal consistence =.71 and good reliability ( $r = 0.8$ ; Kafetsios & Nezlek, & Vassilakou, 2012; Martinez & Lau, 2001). The short form consists of 12 items, each requiring subjects to list the people to whom they could turn and on whom they could rely in a specific hypothetical situation. They are then asked to indicate on a five point scale their level of satisfaction with the social support they would receive in each situation. Thus allowing the derivative of two scores; the average number of support persons listed for each item and the average satisfaction with support for each item which allows researchers to comment on the actual and also the individual's perception of their support.

### **Stress and Coping**

ASQ. The ASQ is a 58 item self-report inventory designed to measure stress in domains relevant to adolescence. Participants self-report on a stressor by appraising it from 1 (not stressful or relevant to me) to 5 (very stressful) and total scores are summed to yield a range between 58-290 with higher scores indicative of higher levels of perceived stress. The ASQ can also measure domain specific stress in 10 areas, such as school performance, home life, school attendance, romantic relationships, peer pressure, future uncertainty, school/ leisure conflict, financial pressure, teacher interaction and emerging adult responsibilities.

The scale is a reliable and valid measure of stress in adolescence, with 8 of the 10 scales demonstrating a Cronbach Alpha above .8 and up to .96 in recent studies, and concurrent validity with scales measuring anxiety and depression (Byrne et al., 2007; Murray, Byrne & Reiger, 2011).

One week test-retest correlation coefficients range between .68 and .88, with seven having exceeded .8 in recent studies (Byrne et al., 2007).

*LEQ.* Exposure to adversity was quantified using a modified version of the LEQ developed by Newcomb et al. (1981) for use with adolescents. The measure consists of 22 of the original 37 events that respondents indicate if they have experienced same during the previous year, over one year ago, or never. The emotional impact of those events is rated from very negative (-2) to very positive (+ 2). Stressful events include items such as school change and parental divorce. Items omitted by the current researcher were considered inappropriate and posing an emotional risk in the age of the current sample (for example questions such as 'I became pregnant' were removed). The number of stressful life events demonstrate concurrent validity with measures of depression and face validity (Compas, Wagner, Slavin, & Vannatta, 1986; Newcomb, 1981). Studies have reported low internal consistency from .38 to .59 as the events presented are not likely to co-occur (Compas et al., 1986).

*A-COPE.* Adolescent coping styles will be assessed by the A-COPE scale which asks respondents to indicate on a 5-point scale (ranging from 1 = never to 5 = most of the time) how often they used each coping style when feeling tense or facing a problem or difficulty. A set of 16 items covering active- and avoidance-oriented coping styles are presented and respondents are instructed to indicate how often they used a given response when they experienced problems. Items include "let off steam by complaining to your family or friends", or "buy things you like". Studies have confirmed the validity and reliability of the scale with eight of the twelve subscales demonstrating Cronbach's  $\alpha$  above 0.7 (Copeland & Hess, 1995; Patterson & McCubbin, 1987).

### **Scales**

All scales in the present study were found to be reliable for analysis. The Cronbach  $\alpha$  for each scale is presented in Table 4.

Table 4

*Reliability of Each Scale*

Scale	Number of Items	Cronbach's $\alpha$
JEPQ	44	.77
NEO-FFI	20	.78
CES-D	20	.84
SSQ-S	6	.9
ASQ	58	.98
LEQ	28	.88
A-COPE	52	.88

**Procedure**

Prior to commencing the study written approval was gained from the ANU Ethics Committee, the area specific Catholic Education Offices and the ACT and NSW Departments of Education. Once approval was obtained schools across the nation were chosen at random via a Google search. School principals were contacted via email to ascertain the likelihood of their school participating in the study and initially twenty schools agreed to participate. Eleven of the schools dropped out during correspondence and during the course of the study a further four schools withdrew due to internal time pressures.

Prior to data collection teachers distributed and collected parent consent forms. Data collection took place in whole class groups in computer labs with teacher supervision. Prior to students being provided the URL for the questionnaire they read and signed consent forms and were made aware that they could withdraw without consequence at any time. The online platform for the questionnaire was designed with Qualtrics Software, owned by the ANU Research School of Psychology. The questionnaire took approximately 40 minutes to complete. At the conclusion

participants were provided with a debrief form, containing information about the study and relevant numbers for help in the event they became distressed. All statistical analysis were undertaken with Microsoft Excel, SPSS software including AMOS.

## **Results**

Prior to analysis, data were inspected following guidelines from Tabachnick & Fidell (2007) to ensure their suitability for statistical analysis. A missing values analysis (MVA) was run and fourteen cases that had more than 10% of data missing were deleted. The remaining missing data was distributed at random and Hot Deck was used to input the mean, based on the average score of individuals of the same sex, age and school year (Myers, 2011). Twelve cases with extreme z scores were found to be univariate outliers, and two other cases were identified through Mahalanobis distance as multivariate outliers,  $p < .001$ . Univariate outliers were transformed to the mean while multivariate outliers were deleted, leaving 344 cases for analysis.

Linearity and homoscedasticity were assessed using bivariate and pair wise scatterplots, while the assumption of normality was assessed statistically and graphically using histograms (Appendix E). No variables were transformed to improve pairwise linearity or skewness and kurtosis as all skew was in the expected direction, in addition, as the final sample contained more than 200 cases, the underestimation of variance due to a non-normal distribution was negated (Tabachnick & Fidell, 2007).

### **Preliminary Analysis**

#### **Depression Analysis**

The mean score for depression was 15.74 (SD= 11.75). Twenty per cent of the sample demonstrated symptoms of mild depression (cut off score on the CES-D of 20>) and within this, 5 per cent of the sample were classified as severely depressed (cut off score on the CES-D of 40>). Females were found to be significantly more depressed than males, ( $F [1, 343] = 11.03, p < .001 \eta^2 = .031$ ), with nearly 23 per cent demonstrating low mood and over 7 per cent of females having scores

indicative of severe depression. In contrast, only one per cent of males were classified as severely depressed and 17 per cent demonstrated depressive symptoms. These findings are presented in Table 5 which illustrates the prevalence in depression scores overall and then broken down by each gender and severity.

Table 5

*Levels of Depressed Mood in Current Sample as Scored by the CES-D*

	M	Depressed Mood		
		No Symptoms (%) of sample	Mild/ moderate (%) of sample	Severe (%) of sample
Overall	15.74	75	20.3	4.7
Boys	13.36	82.3	17	0.7
Girls	17.53	70	22.9	7.1

**Gender**

A gender effect in depression literature is one of the most consistently reported findings (Hankin et al., 1998). Gender differences have also shown on measures of personality, stress and social support (Compas et al., 1998; Flynn et al., 2011). The current sample contained 150 males and 197 females. As the sample violated Levene’s test for homogeneity in ANOVA, a preliminary exploration of the impact on gender on the IVs and the DV was further explored using the Mann-Whitney U Test. Significant gender effects were found on measures of, depression (U = 11802, p = .003), neuroticism (U = 9233, p = .000), agreeableness (U = 11746, p = .003), stress (U = 13361, p = 0.005), actual social support ( U = 12190, p = .012), and social support satisfaction (U = 12361, p = .018). . As such gender was retained for further analysis. Extraversion, openness, coping and bad life events had no gender effect, p>.05.

## Age

A one way ANOVA was conducted to examine whether significant differences were apparent in depression scores in the school years. Sixty four students were in year 7, forty five in year 8, 134 in year 9 and 104 in year 10. The majority of students were aged 15 years (34% of the sample). Year 9 students demonstrated the highest rates of depression  $M= 18$  ( $SD= 12.81$ ), and year 8 students the lowest,  $M=12.14$  ( $SD= 10.38$ ). Overall differences in depression scores in the different year groups did not reach significance ( $F [3, 341] = 4.511, p>.05 \eta^2= .038$ ). However post-hoc Games Howell tests did reveal a small, but statistical difference,  $p<0.05$ , between year eight and nine, suggesting that in the current study year nine was the highest risk group for depression. Due to the mixed findings, age group was excluded from further analysis.

## Correlations

The relationship between all of the variables was initially investigated using scatterplots and bivariate correlations (Appendix D). Table 6, displayed on the next page, has the correlation coefficients for the total sample and also broken down by gender. Analysis of the initial correlations reveals that the main variables correlated in the expected directions.

With the exception of coping, agreeableness and openness, depression was related to all of the main variables. The strongest relationship was apparent between neuroticism and depression,  $r=.643, n=344, p<.001$ , with a medium effect size as per Cohen's D (Cohen, 1988). The social support variables exhibited a small, negative relationships to depression,  $p<.001$ . Stress and depression had a medium, positive relationship, while extraversion and bad life events had a small, negative relationship to depression,  $p<.005$ .

The two main IVs, social support and neuroticism demonstrate small, negative relationships to one another,  $p<.001$ . Finally, stress had small relationships to both social support variables and neuroticism. Variables that were not related to depression were excluded from further analysis.



Table 6.  
Correlations of primary measures at Time 1

	Sample	Depression	Life Good	Life Bad	SSS	SSA	Stress	Coping	Neuroticism	Extraversion	Agreeableness	Openness
Depression	Male	-										
	Female	-										
	Total	-										
Life Good	Male	-.121	-									
	Female	.159*	-									
	Total	0	-									
Life Bad	Male	.254**	-.24	-								
	Female	.412**	.083	-								
	Total	<b>.330**</b>	<b>.026</b>	-								
Social Support Satisfaction (SSS)	Male	-.218**	.26	-.056	-							
	Female	-.312**	-.138	-.056	-							
	Total	<b>-.237**</b>	<b>-.063</b>	<b>-.059</b>	-							
Social Support Actual (SSA)	Male	-.155	-.016	-.016	.356**	-						
	Female	-.294**	-.020	-.143*	.414**	-						
	Total	<b>-.207**</b>	<b>-.039</b>	<b>-.090</b>	<b>.396**</b>	-						
Stress	Male	.317**	.099	.095	-.157	-.080	-					
	Female	.577**	.009	.381**	-.226**	-.213**	-					
	Total	<b>.500**</b>	<b>.019</b>	<b>.242**</b>	<b>-.168**</b>	<b>-.130*</b>	-					
Coping	Male	.020	-.024	-.067	.073	.126	.023	-				
	Female	-.108	-.040	.038	.213**	.296	-.031	-				
	Total	<b>-.045</b>	<b>-.037</b>	<b>-.019</b>	<b>.143**</b>	<b>.213**</b>	<b>.001</b>	-				
Neuroticism	Male	.500**	-.089	.211*	-.193*	-.235**	-.118	-.118	-			
	Female	.714**	.009	.209**	-.257**	-.296**	.599**	-.117	-			
	Total	<b>.643**</b>	<b>-.089</b>	<b>.190**</b>	<b>-.171**</b>	<b>-.209**</b>	<b>.570**</b>	<b>-.097</b>	-			
Extraversion	Male	-.221**	.060	.007	-.026	.010	.235**	.235**	-.027	-		
	Female	-.288**	.075	-.216**	.027	.222**	-.077	.317**	-.147*	-		
	Total	<b>-.257**</b>	<b>.070</b>	<b>-.101</b>	<b>-.004</b>	<b>.112*</b>	<b>-.040</b>	<b>.271**</b>	<b>-.096</b>	-		
Agreeableness	Male	-.042	-.003	-.071	.231**	.079	.127	.210*	-.014	.050	-	
	Female	-.038	.007	-.086	-.008	.080	.072	.342**	.030	.312**	-	
	Total	<b>-.1</b>	<b>-.23</b>	<b>-.082</b>	<b>.117*</b>	<b>.099</b>	<b>.116*</b>	<b>.280**</b>	<b>.061</b>	<b>.186**</b>	-	
Openness	Male	-.051	.007	-.124	.206**	.091	.103	.258**	-.047	.150	.654**	-
	Female	-.081	.124	-.127	.045	.130	.043	.313**	-.084	.228**	.537**	-
	Total	<b>-.062</b>	<b>.054</b>	<b>-.126*</b>	<b>.125*</b>	<b>.116*</b>	<b>.073</b>	<b>.285**</b>	<b>-.053</b>	<b>.189**</b>	<b>.582**</b>	-

Note. \*\* Correlation is significant at  $p < .01$

\*Correlation is significant at  $p < .05$



**Regression Analysis**

Linear regression was used to test the effect of the significant variables on the variance on depression as the dependent variable (DV). Gender was included as a separate variable in the regression. The  $\beta$  coefficients, t-values,  $R^2$ , F values, confidence intervals and significant for each independent variable (IV) on the DV is presented in Table 4.

Table 7 shows that each single IV model was significant at,  $p < .001$ . As all the variables were significant a combined model was tested which found that the IVs together explained 51% of the variance,  $F(6, 337) = 61.36$ , CI [0.43, 0.58], in depression scores in the current sample.

Table 7

*Output for individual regression analysis with depression as the dependent variable (DV)*

IV	R <sup>2</sup>	F (df1, df2)	$\beta$	t-value	Confidence Interval 95%
Life Bad	.109	41.907 (1343)**	.33	6.474**	[0.047; 0.171]
Social Support Satisfaction	.056	20.399 (1343)**	-.237	-4.517**	[0.009, 0.103]
Social Support Actual	.043	15.290 (1343)**	-.207	-.390**	[0.001, 0.085]
Stress	.25	113.713 (1343)**	.5	10.664**	[0.17, 0.329]
Neuroticism	.414	241.16 (1343)**	.643	15.53**	[0.335, 0.493]
Extraversion	.066	24.091 (1343)**	-.257	-.489**	[0.016, 0.116]
Gender	.028	11.030 (1343)**	.177	3.321**	[0.213, 0.347]

\*Denotes significant at  $p < .05$ , \*\* Denotes significant at  $p < .001$ .

To investigate the relationship among the variables, a more complex model testing their combined effect was performed using hierarchical multiple regression, which is illustrated in Table 8. Variables were entered into the regression based on established theoretical understandings and order of influence in the CVTSM of depression (Hankin & Abramson, 2001). Firstly the established variables such as gender and personality traits were entered. Stress and negative life events were

entered second, followed by the social support variables. Studies have suggested that personality and gender can impact on the reporting of social support and negative life events, as such hierarchical regression will control for these and identify the unique variance offered by these variables (Swickert & Owens, 2010).

As shown in Table 8, the first block demonstrating a significant model relationship that explained 45%,  $F(2, 340) = 130.896$ ,  $CI [0.376, 0.530]$ , of the variance in depression. After controlling for the fixed personality variables, the entry of stress and negative life events at Step 2, saw the model explain 51% of the variance,  $F(2, 388) = 19.178$ ,  $CI [0.439, 0.577]$ . The social support variables were added step 3, explaining a further 1% of the variance,  $F(2, 388) = 4.891$ ,  $CI [0.454, 0.59]$ .

In the final model neuroticism, extraversion, stress, bad life events and social support satisfaction contribute significantly to the variance of score on depression in the current study, explaining up to 59% of the variance in depression. Neuroticism made the most significant unique contribution to the overall model, explaining 47% of the variance, extraversion 19%, stress explained 15%, bad life events 18 % and social support satisfaction, 12%.

Unexpectedly, gender became non-significant when included with neuroticism,  $p > .05$ . Partial correlation was used to identify the interactions between the variables. Neuroticism was found to mediate the relationship between gender and depression, as no relationship was found when controlling for neuroticism,  $r = -.034$ ,  $n = 341$ ,  $p > .05$ . Similarly, in the third model there was a non-significant negative partial correlation between actual social support and depression when controlling for neuroticism,  $r = -.097$ ,  $n = 342$ ,  $p > .005$ . An inspection of the zero order correlation ( $r = -.207$ ) suggests that controlling for neuroticism renders the relationship between the variables non-significant.

Neuroticism lost a proportion of variance from model 1 to 3, as it originally explained 63% of the variance, suggesting that stress, negative life events and social support may play a role in offsetting the relationship between neuroticism and depression. In addition, stress lost a proportion

of variance with the inclusion of the social support variables, while negative life events remained the same, suggesting that social support has an important role in reducing overall stress. As gender and actual social support were not found to be significant independent predictors of depression they were excluded from further analysis. The final model was investigated further using structural equations modelling.

Table 8.

*Output for the total sample hierarchical multiple regression analysis*

Model	R <sup>2</sup>	F (df1, df2)	IVs	β	t-value
1	.453	130.896 (2,340)**	Gender	-.29	-.679
			Neuroticism	.633	14.925**
			Extraversion	-.197	-4.887**
2	.508	19.178 (2,388)**	Gender	-.008	-.201
			Neuroticism	.500	10.288**
			Extraversion	-.185	-4.796**
			Stress	.166	3.521**
			Bad Life	.176	4.439**
3	.522	4.891 (2, 388)*	Gender	.018	.430
			Neuroticism	.477	9.699**
			Extraversion	-.186	-4.866**
			Life bad	.176	4.49**
			SSS	-.121	-2.884*
			SSA	-.005	-.126
			Stress	.154	3.294*

Note. Model 1: Gender, Neuroticism, Extraversion  
 Model 2: Gender, Neuroticism, Extraversion, Stress, Bad Life Events  
 Model 3: Gender, Neuroticism, Extraversion, Stress, Bad Life Events, Social Support Satisfaction, Social Support Actual  
 \*Denotes significant at p<.05, \*\* Denotes significant at p<.001.

### Structural Equations Modelling

#### The Hypothesised Model

An exploratory factor analysis (EFA) was performed on the five IVs and one DV. The analysis confirmed the suitability of the data to be summarised into six factors, Kaiser-Meyer-Olkin Measurement = .755. A confirmatory factor analysis (CFA) based on the EFA and results from the regression analysis was performed using SPSS AMOS. The hypothesised model from this analysis is

presented in Figure 2 below, where rectangles represent measured variables. A solid line connecting two variables implies a hypothesised direct effect while a dashed line pertains to mediation. The absence of a line connecting the variables implies no hypothesised direct effect. A model comprised of direct and indirect effects is hypothesised and these relationships are shown in the figure.

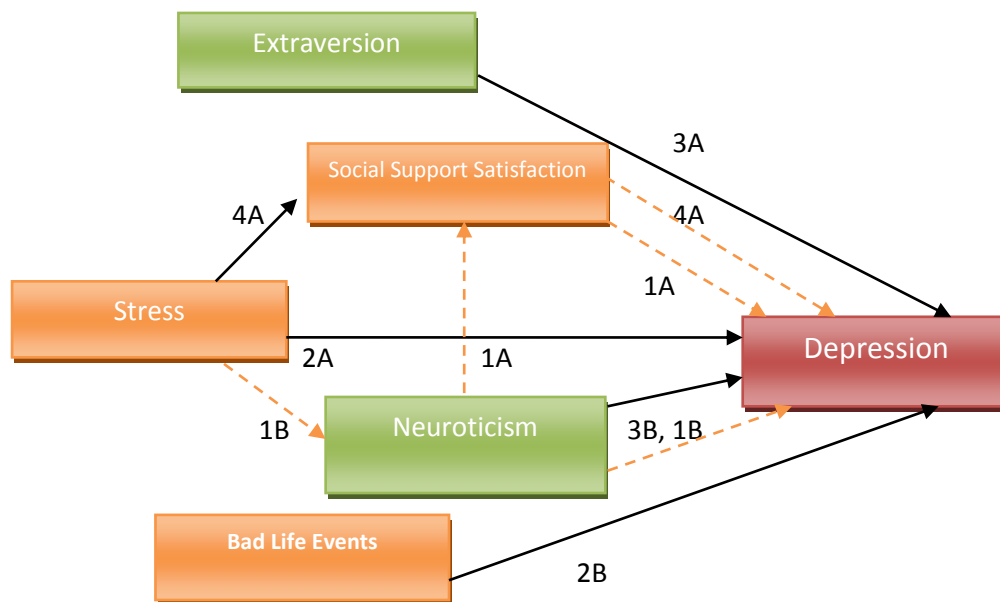


Figure 2. Hypothesised Model between IVs and the DV- Depression.

Direct effects are shown with black arrows whereas indirect effects are illustrated with dashed arrows.

Path 1A- Social support satisfaction mediates the relationship between neuroticism and depression

Path 1B- Neuroticism mediates the relationship between stress and depression

Path 2A Stress has a unique relationship to depression

Path 2B- Bad life events have a unique relationship to depression

Path 3A- Extraversion has a unique path to depression

Path 3B Neuroticism has a unique relationship to depression

Path 4A- Social support satisfaction mediates the relationship between stress and depression

### Model Estimation

Given the presence of non-normal variables (i.e. variables which exceed the cut off values for skew and kurtosis; West et al., 1995), all SEM analysis were performed using the maximum likelihood robust estimator (MLR) which takes into account non-normal distributions. Maximum

likelihood estimation was employed to estimate all models. The independence model assuming all tests are uncorrelated was rejectable,  $\chi^2 (15, N=344) = 429.077$ ,  $p > .05$ . The hypothesised model was tested next and was found to be a more suitable fit for the data,  $\chi^2 (6, 344) = 7.67$ ,  $p > .05$ , comparative fit index (CFI; values of 0.95 or greater indicate a good fit) = .999, p of close fit (PCLOSE; figure greater than 0.05 indicates a good model) = .696, standardised root mean square residual (RMSEA; values of 0.08 or less indicate the model adequately fits the data) = .028. A chi square difference test indicated a significant improvement in fit between the independent model and the hypothesised model. Post hoc modifications were performed in an attempt to improve the model fit. The path between stress and social support satisfaction was not significant and removed (unstandardized coefficient =  $-.003$ ,  $p > .05$ ). This had no impact on the theoretical model as this path was testing the stress buffering effect and not a central part of the CVTSM. The final model fit was,  $\chi^2 (7, 344) = 10.255$ ,  $p > .05$ , comparative fit index (CFI) = .992, PCLOSE = .628, RMSEA = .037. The final model with standardised and unstandardized coefficients is shown in Figure 3 below.

### **Direct Effects**

Depression was predicted by greater levels of stress (unstandardised coefficient =  $.16$ ,  $p < .001$ ), higher levels of trait neuroticism (unstandardised coefficient =  $.5$ ,  $p < .001$ ), and lower social support satisfaction (unstandardised coefficient =  $-.12$ ,  $p < .01$ ). A higher number of bad life events (unstandardised coefficient =  $.18$ ,  $p < .001$ ), had a direct relationship to depression, and this relationship was not influenced by other variables. Similarly, extraversion had a direct relationship to depression (unstandardised coefficient =  $-.19$ ,  $p < .001$ ), which did not interact with social support satisfaction or negative life events,  $p > .05$ .

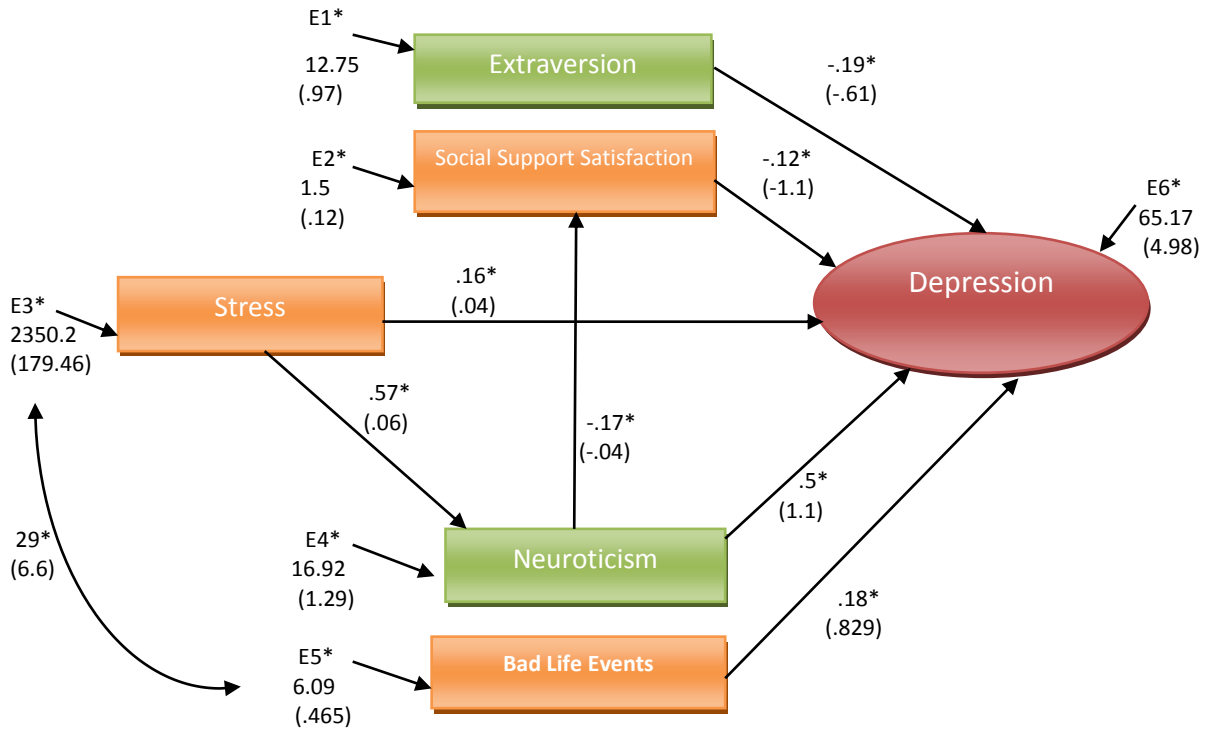


Figure 3. Final Structural Equations Model

**Indirect Effects**

The significance of the intervening variables was evaluated using tests of indirect effects and interaction analysis. The results of the mediation are shown below in Table 9 below (Baron & Kenny, 1986; Sobel, 1988). Using both the Sobel and the Barron & Kenny method, neuroticism was found to mediate the relationship between stress and depression. Similarly, social support satisfaction mediated the relationship between neuroticism and depression.

Table 9

*Mediation Analysis*

Relationship Tested	Direct Effect Without Mediation	Direct Effect With Mediator	Bootstrapped Indirect Effect
Neuroticism mediates stress and depression	.446**	.159**	Sig 0.05
Social support satisfaction mediates neuroticism and depression	.61**	-.129*	Sig 0.05

The nature of the moderation relationships on depression was tested using interaction terms. Graph 1 below demonstrates that neuroticism strengthens the positive relationship between stress and depression (unstandardised coefficient= 2.909,  $p < .001$ ). This finding is confirmatory of the vulnerability model hypothesis, in that, in the event of stress; those with high levels of neuroticism are at a significantly increased chance of becoming depressed. In the absence of stress, the relationship between high and low neuroticism and depression is not significantly different,  $p > .05$ . However, when neuroticism is low there is a negative effect between stress and depression. Specifically individuals low in neuroticism appear to have lower levels of depression when under stress.

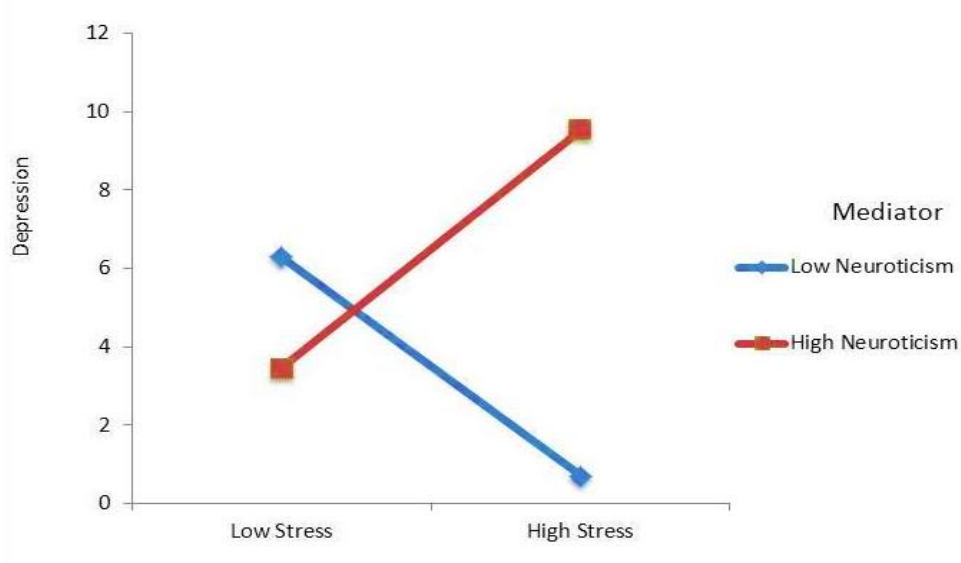


Figure 4. Graph of the Relationship between Stress and Depression, Mediated by Neuroticism

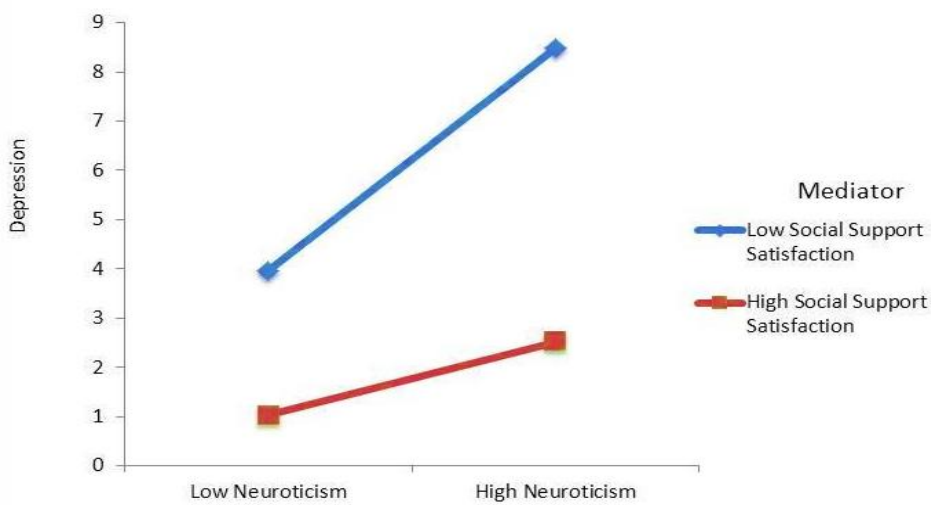


Figure 5. Graph of the Relationship between Neuroticism and Depression, Mediated by Social Support Satisfaction

Using the same technique, social support satisfaction dampened the relationship between neuroticism and depression, (unstandardized coefficient =  $-.760$ ,  $p > .05$ ). Graph 2 suggested that when social support satisfaction is low there is a positive effect between neuroticism and depression. However the interaction relationship did not reach statistical significance. These results



suggest that social support satisfaction may have a small positive effect on reducing depression, however this may be for young people in general. Specifically, social support does appear to offset the inherent vulnerabilities to depression that neuroticism brings with it. A three way interaction was explored between neuroticism, social support satisfaction and stress on depression using the general linear model and this was found to be non-significant,  $F(1, 344) = .123, p > .05$ .

### **Additional Analyses**

#### **Analysis of Stressor Domains**

Interpersonal stress has a strong theoretical link to the main variables in the current study (Eberhart & Hammen, 2009; Kendler et al., 2004). In order to investigate the unique role of interpersonal stress the 10 subscales of the ASQ were investigated (Byrne et al., 2007; Moksnes et al., 2010). Peer pressure, romantic relationships, home life and teacher interaction were expected to be significantly related to depression. A standard multiple regression on all of the ASQ scales, as IVs, was performed with depression as the DVs. Interpersonal domains were expected to have a stronger relationship to depression than the overall stress scale.

The suitability of the current data for use with the subscales of the ASQ was investigated using a principle components analysis (PCA). Inspection of the correlation matrix found many coefficients above .3 and the Kaiser-Meyer-Olkin value was .953, exceeding the recommendation of .6 (Kaiser, 1974). In addition, Bartlett's Test of Sphericity reached statistical significance, supporting the factorability of the scale (Bartlett, 1954). Principle components analysis confirmed the presence of 10 components exceeding 1, explaining 25%, 3%, 3%, 2%, 2% and the remaining five explained 1% respectively. As such the sample was found to be appropriate for separation into the subscales.

The model was significant at  $p > .001$ , explaining 29% of the variance in depression,  $F(9, 334) = 15.488, 95\% \text{ CI} [ ]$ . However B coefficients indicated that only peer pressure ( $\beta = .192, p < .05$ ), home life stress ( $\beta = .249, p < .05$ ), and school attendance ( $\beta = .314, p < .001$ ) contributed

significant unique variance to the model. Romantic relationship stress and teacher interactions did not reach significance,  $p > .05$ .

### **Analysis of Depressive Symptoms**

Given the sample contained a small number of severely depressed young people ( $N = 17$ ), the mild/moderate and severe group were collapsed into a depressive symptoms group to allow for exploratory analysis based on symptoms of clinical depression. The amalgamated depressive symptom group contained,  $N = 93$ , and this group was compared to the subclinical group ( $N = 251$ ; CES D scores of  $< 20$ ). Both standard and hierarchical regression based on the sequence for the full sample was repeated with the depressive symptoms group. Both models were significant,  $p < .05$  and the hierarchical model 3 with all the main variables explained approximately 49 per cent of the variance,  $F(2, 85) = 3.745$ ,  $CI [0.31, 0.63]$ . Interestingly, only neuroticism made a significant contribution to the model in both examples,  $p < .05$ . In the hierarchical regression, neuroticism alone explained approximately 44 per cent of the variance ( $\beta = .441$ ). Given the findings no further analysis based on severity of depressive symptoms was undertaken.

### **Discussion**

Relationships are very important to young people and the absence of such supportive ties is comprehensively linked to the early onset of depression. Traditional interpersonal theories have inherently assumed the role of depression symptoms and personality in reducing social functioning and support, and exacerbating the disorder. The current study was a cross sectional exploration of a sample of Australian adolescents aged 12-16 years, and was the first step in examining the prospective relationship between neuroticism, social support and depression. The study also looked at the role of stress, independent negative life events, and additional personality variables. The findings of the current study are confirmatory of the vulnerability model of depression (Hankin & Abramson, 2001), suggesting that, while a number of variables are significantly related to depression, it is the combination of interpersonal stress and neuroticism which contributes the most significant pathway to adolescent depression. Furthermore, adequate perceived social support was

found to reduce depression and in fact buffer some of the effects of the noxious relationship between neuroticism and stress, however this relationship was not as robust as the vulnerability model. The findings are discussed in line with the cognitive vulnerability transactional stress model and the relationship between inherent vulnerabilities, and the environment Australian adolescents are growing up in.

The prevalence of depression in the current study was comparable to recent Australian rates, suggesting the findings are a true representation of Australia adolescents' experience (Black, Roberts & Li-Leng T, 2012). The study found approximately a quarter of the sample had symptoms of depression, and 5 per cent fell into the category of severe depression as classified by the Centre for Epidemiological Studies Depression Scale (CES-D). In line with current literature, females were found to be significantly more depressed than males, and had higher rates of severe depression (Alison et al., 2001). While, males and females were similar in rates of mild/ moderate depression, overall, more males were symptom free. Such findings are comparable to Boyd and colleagues (2000), who reported 15 per cent of adolescents in their Australian sample to be depressed (utilising a more stringent inclusion criteria), and similarly found a gender effect.

Bivariate correlations revealed that higher levels of neuroticism and lower levels of actual and perceived social support were associated with depression. Higher stress scores and negative life events resulted in higher rates of depression in the current study. While the inverse pattern was apparent for extraversion, with higher rates resulting in lower depression levels. Furthermore, young people with high levels of neuroticism were found to experience more stress and have lower actual, as well as perceived levels of social support. These findings are consistent with previous studies, such as Swickert & Owens (2011), but the study also confirms the findings for Australian youth.

Hierarchical regression analysis indicated that, in order of contribution, neuroticism, extraversion, stress, negative life events and social support satisfaction were related to depression. The combined model of these variables explained between 45% -59% of the variance in scores. This

model accounted for a greater degree of variance than any of the signal predictors, lending to the suggestion of a very complex model of adolescent depression, with structural equations models illustrating both direct and interacting relationships to depression risk.

Neuroticism accounted for the largest unique direct variance, explaining approximately 48% of the variance in depression. Extraversion exerted the second highest unique relationship, explaining 19% of the variance. Neuroticism and extraversion had no relationship to one another, confirming that each of the higher order personality variables had a unique contribution to depression. None of the other personality variables were found to be related to depression. As such, the current study supports a two factor model of personality (Eysenck & Eysenck, 1975).

However, each of the two personality factors each related differently to depression. Specifically, high levels of extraversion resulted in lower levels of depression, whereas high neuroticism results in higher. Extraversion exhibited a straight forward protective effect, suggesting it is not part of a vulnerability model. The positive effects of extraversion on a reduction in the risk for psychopathology are well documented and the current findings support this (Farmer et al., 2002; Saklofske et al., 1995).

One way in which extraversion is thought to have a protective effect is via improving social relations, thus resulting in a larger pool of supportive individuals available in times of need. Contradictory to a number of studies, the current research found no relationship between extraversion and the social support variables (Farmer et al., 2002; Leskela et al., 2009). While it is possible that the relationship is not apparent due to the cross sectional design of the study, the results may also suggest that extraversion interacts with the social sphere in a complex manner. For example, research suggests that high levels of extraversion are associated with an energetic approach to the world such as seeking out activities and social outings (Barlow, Wright, Sheasby, Turner & Hainsworth, 2002). As such, it is possible that these behaviours could lead to an increase chance of having positive events, and more transactions with their environment that would allow for positive emotions, and thus reduced depression (Spinhoven et al., 2011; Sarubin et al., 2015). Such a

relationship would be unlikely to show up statistically in the current study and may suggest extraversion has a complex protective effect.

High levels of neuroticism had a strong independent and well as indirect link to depression. Neuroticism was found to act in a manner which strengthened the relationship between stress and depression, and the interaction of the two accounted for the strongest relationship to depression in the current study. The findings support the concept of a neurotic individuals having an exaggerated emotional response to stress, which likely results in feelings of an ability to cope, increased low mood, and perceptions of a lack of control over their environment (Barlow et al., 2014). This is in line with the vulnerability model, suggesting that neuroticism is an inherent vulnerability to depression, which is triggered by stress (Costello et al., 2006; Hankin, 2012; Burcusa & Iacono, 2007; Shih, 2007). Or put differently, it is in the event of stress that neurotic adolescents are at a significantly increased risk for becoming depressed. In the absence of stress the relationship between high and low neuroticism and depression was not significantly different. These findings suggest neuroticism may be an inherent risk factor that lays dormant until acted upon.

Interestingly, a negative relationship was found between low neuroticism and stress, in that individuals who were low on neuroticism were found to have lower levels of depression when under stress. This finding was unexpected, and may suggest that for non-neurotic young people, stress may in fact be activating. Studies have shown that when under stress, psychologically healthy subjects, can show increased initiative and proactive problem solving (Fay & Sonnentag, 2002; Gerber et al., 2013). Specifically, setting and meeting goals can result in feelings of mastery which is linked to lower levels of depression in adolescent samples (Colman et al., 2014). As such, stress in and of itself may not be dangerous (Crum, Salovey & Achor, 2013), but it is the unique combination of an inherent vulnerability and stress which appears to be a risk for adolescent depression.

A preliminary analysis of the domains of stress was undertaken to explore whether interpersonal stress was particularly salient in the current vulnerability model. Given neuroticism is comprehensively linked to interpersonal stress, it was expected that, romantic relationship, peer

pressure, and home life stress would exhibit the strongest relationship to depression. With the exception of romantic relationship stress, the current study supported this.

The findings that home life stress was strongly implicated was not surprising, as neuroticism has been shown to be a highly heritable trait, and as such, it is likely that neurotic young people also have family members high in the trait (Kercher et al., 2009; Munafo et al., 2006; Weissman et al., 2005). This would increase the chances of home life consisting of high expressed emotion and conflict, which are linked to greater emotional unease and less supportive family relationships (Lue, Wu & Yen, 2010). Such attributes are reflected in the scale with items such as, disagreements with your parents, lack of understanding by your parents, and parents hassling you about the way you look. Pettit and Colleagues (2011) also suggested that family stress is more influential than the peer group stress in the onset of adolescent depression, given young people are unable to find reprieve from a dysfunctional home life.

Quite similarly, school attendance is a compulsory, and as such this form of stress is enduring (Hampel & Petermann, 2006). The social demands of school are great, especially for those young people who may be interpersonally sensitive and have difficulties with relationships. It is unclear from the cross sectional study if this finding is the result of stress or if it reflects the findings of already depressed young people, as getting up early and concentrating for the entire day is demanding on a depressed individual. Finally, given the mean age of the current study was fourteen years, it may have been too early to discover romantic stress trends. The stress of romantic relationship scale is made up of items such as getting along with your partner and making the relationship work. In the current sample the young people may be at the stage of attempting to forge relationships with romantic partners and may not have experienced the stressors with maintaining them. Overall, the analysis of the stressor domains suggested that the home life stress, stress due to school attendance and peer stress exhibited the strongest relationship to depression in the current sample. This is not surprising, given individuals with high levels of neuroticism are particularly vulnerable to the presence of interpersonal stress or threats. As such, the combination

of interpersonal stress and neuroticism has the strongest relationship to depression in the current study.

As expected, the vulnerability model did not interact with independent negative life events. Profound negative life events, such as, death of a parent, parent divorce, change in school, can result in depression, over and above any risk which stems from personality (Kessler, 1997; Tennent, 2002). This is in accordance with the original conceptualisation of neuroticism by Eysenck & Eysenck (1975), who suggested that a proportion of individuals who become depressed have been subjected to extreme life stress, quite dissimilar to neurotic individuals who require relatively little comparative stress to become depressed. Furthermore, these findings are also supported by modern research (Kercher & Rapee, 2009) and in line with the stress generation theory, suggests that neurotic individuals did not experience any higher rates of independent life stress than their counterparts.

Regarding the role of social support in buffering stress from either independent or dependent life stress, and therefore reducing the chance of depression, no support was found. A protective effect of social support satisfaction on depression was apparent, and social support satisfaction was found to dampen the relationship between neuroticism and depression but this did not reach significance. This is consistent with results from Burton and Colleagues (2004) who suggest that social support is a complex variable which often has indirect effects on depression as opposed to a straight forward buffering effect. Furthermore, no relationship was apparent from negative life events to social support, suggesting that the emotional experience from these stressors may be too profound to be offset by any form of protective effect social support may offer.

The study contained a number of unexpected results which warrant further discussion. In isolation, gender had a significant relationship to depression, however with the inclusion of neuroticism it became non-significant. Suggesting, that during times of stress, high levels of neuroticism may result in a heightened experience of negative affect which overrides other variables. Similarly, Swickert & Owens (2011) found a gender effect ceased to exist at high levels of neuroticism, with males and females reporting the same levels of depression and social support.

Furthermore, no relationship was found between coping and depression. While the link between coping and depression is well established, recent work by Horwitz, Hill & King (2011) suggests it is best measured categorically, using specific behavioural manifestations of coping such as self-blame or avoidance, as these have strong relations to depression, whereas problem focused coping is inconsistent in predicting depression. Such assertions are in accordance with experimental research by Brinker, Pidcock & Boyer (2013), which suggests that behavioural manifestations such as rumination may be the most salient in depression. As such, it is likely that the use of a broad coping measure in the current study was not sensitive enough to discover variance in the coping scores.

The present study had several limitations. As it is cross sectional, any inferences regarding the directional effects of the main variables and causality are premature, and some caution should be taken in interpreting a number of the main findings. Firstly, there is a body of literature which posits that depressed mood can inflate or invalidate self-report measures of neuroticism and social support (Enns, Larsen & Cox, 2000; Karsten et al., 2012). A longitudinal design is needed to explore the temporal stability of personality in the context of depression, in order to adequately control for this limitation. In addition, the gender differences were unexpected and warrant replication with a more even distribution of males and females. Finally, differentiating between peer and family social support may be of benefit in future studies, specifically as research suggests there may be a hierarchy of social support, with maternal and family support being the most critical in early adolescence (Auerbach et al., 2011; Malecki & Demaray, 2003; Raffaelli et al., 2013). In replication, future studies should endeavour to differentiate between types of support, such as overall family, maternal, peer, teacher, and other important figures such as pastoral care advisors. Differentiating support may increase the chances of finding an effect from social support.

A number of clinical and research recommendations can be drawn from the current study. As neuroticism was found to be the most profound vulnerability, researchers and therapists should aim to intervene in this area. Originally neuroticism was thought to be a fixed variable, not amenable to therapeutic intervention (Amelang & Ullwer, 1991; Eysenck & Eysenck, 1975). However, recent



studies have suggested that aspects of this trait may be malleable, providing inlets for treatment, and reducing the public health burden from mental illness (Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, 2014; Lahey, 2009). Specifically, altering the behavioural manifestations which stem from such traits may be possible, and over time lead to a reduction in the trait (Barlow et al., 2014). Future research should aim to elucidate the behavioural mechanisms of neuroticism, as potential treatment inlets.

In addition, any treatment of adolescent depression should include stress management programs. While stress reduction will likely be most important to neurotic young people, gaining information on each young person's neuroticism scores is unrealistic. A program which aims to teach stress management may be protective for all young people, as well as limiting the opportunity for neurotic young people to experience stress which overwhelms their ability to manage. Stress reduction techniques which focus on home life, the demands of school and peer pressure may be the most beneficial starting point.

The current findings also suggest that professionals in the provision of services to young people such as teachers, school counsellors and chaplains, as well as parents should be vigilant to signs of depression in all young people who experience a significant life event. As such, all young people who experience a significant event should be offered counselling regardless of if they exhibit any symptoms for depression. This is in line with current best practice guidelines for adolescent depression, such as NICE (2005) recommendations, which suggest early referral for counselling for any young person who experiences a severe negative event.

Furthermore, as extraversion had an independent protective effect on depression, future studies should investigate if mechanisms associated with extraversion can be cultivated and included in prevention programs. For example if certain aspects of extraversion are protective, such as utilising problem focused coping or increasing positive events scheduling, this may be a skill which can be taught in prevention programs.

## CHAPTER THREE: Longitudinal Study

**Method**

The aim of the second study was to explore the second and third pathways of the cognitive vulnerability stress model. A longitudinal design was needed to explore the stress generation model, which assumes that inherent vulnerabilities from personality will over time predict future stress and thus reduce social support. Similarly, the consequence model requires a longitudinal design as it posits that depressive symptoms and stress during the formative years may influence a young person's developing personality and lead to further vulnerabilities. This study surveyed a proportion of the original sample two and a half years later, repeating the measures by using the same modified battery of questionnaires.

**Participants**

The present study was a longitudinal design which sourced participants from one of the Canberra based co-educational schools, which participated in the original study. The participants were sourced via an email, which invited the young people who originally participated to complete the follow up study. The participants completed the follow up study on the internet in their own time. An incentive to participate, in the form of the chance to win an iPad via a lotto was offered. In order to complete the follow up, participants needed to have access to the internet at home and a computer.

The follow up surveyed 112 adolescents in grades 9-12. The gender and grade breakdown of the participants is presented in Table 10 and 11 below. The *M* age was 17 (*SD* = 1.5), with a non-normal distribution; skewness of -1.68 (*SE* = .277) and kurtosis of -.839 (*SE* = .548), and the majority of participants were in year 11.

Table 10

*Gender profile of the sample From study two*

Gender	Total	Percent Surveyed (%)	Final Sample	Percent of N (%)
Male	33	39	27	36
Female	51	61	48	64
Missing	28	25	0	0
Total (N)	112	100	75	0

Table 11

*Grade profile of the sample from study two*

Grade	Total	Percent Surveyed (%)	Final Sample	Percent of N (%)
9	21	18.8	16	21.3
10	27	24.1	19	25.3
11	35	31.3	22	29.3
12	29	38.6	18	24
Total (N)	112	100	75	100

**Measures**

The follow up study utilised the same battery of online questionnaires as study one. All participants completed demographic questions such as age, school year and gender. The self-report measures were : The Junior Eysenck Personality Questionnaire (JEPQ ; Eysenck & Eysenck, 1975); the NEO-Five Factor Inventory (NEO-FFI; Costa & McCrae, 1985); The Centre for Epidemiological Studies Depression Scale (CES-D; Eaton, Muntaner, Smith, Tien & Ybarra, 2004); The Short Form Social Support Questionnaire (SSQ; Sarason, Levine, Basham & Sarason, 1983), The Adolescent Stress Questionnaire (ASQ; Byrne, Davenport & Mazanov, 2007), The Life Events Questionnaire (LEQ; Newcomb et al., 1981), and The Adolescent Orientation For Problem Experiences Dispositional Inventory (A-COPE; Patterson & McCubbin, 1987). A detailed description of these measures is

available in the method of study one. All the measures in the present study were found to be reliable for analysis. The Cronbach  $\alpha$  for each scale is presented in Table 12 below.

Table 12

*Reliability of each scale in study two*

Scale	Number of Items	Cronbach's $\alpha$
JEPQ	44	.77
NEO-FFI	20	.76
CES-D	20	.88
SSQ-S	6	.85
ASQ	58	.96
LEQ	28	.97
A-COPE	52	.89

## Procedure

Written approval was gained by the ANU Ethics office and the Catholic Education Offices to complete the follow up study. The school principals from each school were contacted by email to ascertain the likelihood that their students might participate in the follow up study. One coeducational Catholic school in Canberra agreed. This wave took place 2.5 years following the first study.

A senior teacher was appointed as the contact by the Principal and they emailed a generic email to all of the young people inviting them to participate in the follow up. The email provided the young people with the URL to complete the study in their own time. The email also contained information on an incentive to complete the study which was the chance to win an iPad via a lotto

system. The ACT Gambling and Racing Commissioner, Gaming Regulation office was contacted in writing to gain approval to run a lotto in Canberra prior to lotto being offered.

The online platform for completing the survey was open for participants for four months, from November to February. The lotto was drawn in March with a neutral individual present to draw it. The winner was notified via email and also by a school teacher. The iPad was delivered to the school office for the young person to pick up.

The online platform for the questionnaire was designed by Qualtrics software, owned by the ANU. At the conclusion the young people were provided with a debrief page on their computer screen which contained information about the study, relevant numbers for help in the event they found themselves distressed and also discussed the way to enter the lotto. All statistical analysis were undertaken using Microsoft Excel, SPSS and AMOS software.

## **Results**

A missing values analysis (MVA) was run and thirty seven cases that had more than 15% of data missing were deleted. The remaining missing data was distributed at random and Hot Deck was used to input the mean based on averages of individuals of the same sex, age and school year (Myers, 2011). One case with an extreme z score on one measure was transformed to the mean. Linearity and homoscedasticity were assessed using bivariate and pair wise scatterplots, while the assumption of normality was assessed statistically and graphically using histograms (Appendix E). No variables were transformed to improve pairwise linearity or skewness and kurtosis as all skew was in the expected direction. After screening and cleaning, a significant amount of cases were removed due to the participants failing to complete more than 15 per cent of the battery of questionnaires. After preliminary data cleaning seventy five cases were left for longitudinal analysis.

### **Time Two Data**

Twenty seven males and forty eight females participated in the follow up study. The relationship between the variables at T2 was initially investigated using scatterplots and bivariate correlations (Appendix D). In agreement with T1, agreeableness, openness, positive life events and

actual social support were not significantly related to depression,  $p < .05$ , at T2. Table 13, displays the correlation coefficients for the main variables in the total sample.

An inspection of the correlations showed that depression and neuroticism demonstrated the strongest relationship,  $r = .586$ ,  $p < .05$ , with a medium effect size as per Cohen’s D (Cohen, 1988). Depression was also related to higher stress levels, and lower levels of social support satisfaction and extraversion,  $p < .05$ . At T2 lower social support satisfaction demonstrated a medium relationship to negative life events,  $r = -.441$ , and also to stress,  $r = -.257$ ,  $p < .05$ .

There were a number of unexpected results in the T2 data. In contrast to the T1 findings, negative life events did not have a significant relationship to depression,  $p > .05$ , nor was the interaction between neuroticism and stress significant,  $p > .05$ . In addition, neuroticism was not related to social support satisfaction in the current sample,  $p > .05$ . It is possible that these findings reflected sampling difficulties, due to the large number of cases which were excluded and this will be further explored later in the results section.

Table 13  
*Correlations of primary measures at time 2*

	Depression	Life Bad	SSS	Stress	Neuroticism	Extraversion
Depression	-	-	-	-	-	-
Life Bad	.213	-	-	-	-	-
Social Support Satisfaction (SSS)	-.270*	-.441**	-	-	-	-
Stress	.289*	.529**	-.257*	-	-	-
Neuroticism	.586**	-.02	-.062	.147	-	-
Extraversion	-.294*	.287*	.065	.178	-.252*	-

Note. \*\* Correlation is significant at  $p < .01$   
\*Correlation is significant at  $p < .05$

**Structural Equations Modelling**

Prior to analysing the changes between T1 and T2, the relationships apparent in the T2 data were explored using structural equations modelling (SEM). SEM was used to ensure that the impact of multiple variables on the DV could be analysed simultaneously, given testing each relationship separately in regression would not account for the variance explained by the relationships among variables. The theoretical model used to test the T2 data was based on the findings of T1. Maximum likelihood estimation was employed to estimate all models. The independence model, assuming all tests are uncorrelated was rejectable,  $\chi^2 (15, N=75) = 109.92, p > .05$ . The hypothesised model, based on the T1 findings, was not a good fit for the T2 data, and significant post hoc modifications were performed. The final model was a more suitable fit for the data  $\chi^2 (1, 75) = 1.623, p > .05$ , comparative fit index (CFI; values of 0.95 or greater indicate a good fit) = .979, p of close fit (PCLOSE; figure greater than 0.05 indicates a good model) = .315, standardised root mean square residual (RMSEA; values of 0.08 or less indicate the model adequately fits the data) = .074. The coefficients in the final model, with standardised and unstandardized coefficients are presented in Table 16 below.

As shown in Table 14, at T2, the strongest predictor of depression was high levels of neuroticism, explaining up to 54 per cent of the variance in depression scores. Neuroticism demonstrated a straightforward path to depression, which did not significantly interact with any other variables,  $p < .5$ . In addition, higher levels of stress and lower levels of extraversion and social support satisfaction also independently predicted depression. However, there was some evidence of mediation between stress, social support and depression. Specifically, social support satisfaction was found to partially mediate the effect of stress on depression, using the Baron & Kenny (1986) test for indirect effects, with a reduction from .259,  $P < .05$  to .181,  $p < .05$ , indicating that the relationship between stress and depression reduced when social support satisfaction was included. Such a result suggests that social support satisfaction dampens the positive relationship between stress and depression. However this finding was not supported when bootstrapping via Sobel's (1988) criteria for mediation was employed,  $p < .05$ , and thus should be interpreted with caution.

Table 14

*Unstandardised, standardised and significance levels for SEM of time 2 data (standard errors in parentheses, N = 75)*

Parameter Estimate	Unstandardized	Standardised	p
Neuroticism → Depression	1.102 (.182)	.535	.000**
Extraversion → Depression	-.389 (.196)	-.180	.047*
Stress → Depression	.045 (.022)	.181	.044*
SSS → Depression	-2.74 (.963)	-.249	.004**
Stress → SSS	-.006 (.003)	-.257	.022*
Covariance of error Neuroticism And Extraversion	-6.666 (2.8)	-.282	
Covariance of error Extraversion And Stress	42.776 (22.5)	.217	

Note. \*\* Path is significant at p<.01  
\*Path is significant at p<.05

### Longitudinal Analysis

The mean score (M) for each of the main variables and the statistical significance between T1 and T2 is presented below in Table 15. Using a 90<sup>th</sup> percentile cut off, depression, neuroticism and extraversion were found to be significantly different at T1 and T2, p<.001. The change between stress, social support satisfaction and bad life events was not significantly different in the two years, p>.05. The T2 sample were found to have higher mean levels of neuroticism, depression and lower levels of extraversion, p<.1. While not significantly different between the two time points, at T2 the sample had higher levels of stress, and were in fact more satisfied with their social support.



Table 15

Mean scores for independent variables and dependent variable at T1 and T2 and the statistical difference using T Tests. Standard deviations are in parentheses.

	Time One	Time Two	Significance
	M	M	
Depression	15.75 (11.7)	19.85 (10.53)	.007**
Neuroticism	10.83 (5)	12.64 (5.03)	.008*
Extraversion	19 (3.58)	16.96 (4.73)	.007**
Stress	152.53 (48.56)	157.39 (41.77)	.417
Social Support Satisfaction	4.98 (1.25)	5 (.94)	.218
Life Bad	2.41 (2.47)	2.43 (2.06)	.595

Note. \*\*significant at p<.01  
\*significant at p<.05

At T2 nearly half of the sample had symptoms of depression (cut off score CES-D 20>) and within this 1 per cent of the sample were classified as severely depressed (cut off score on the CES-D 40>). These findings are presented in Table 16 which illustrates the prevalence in depression scores for each gender and severity. Interestingly, at T2 no females were severely depressed, but they demonstrated more symptoms of mild/moderate depression and more young males were symptom free. The mean score for depression for females was M = 20.93, while males was M = 18.08. The difference between the male and female rates of depressed mood was not statistically significant, (F [1, 73] = 1.207, p>.05 η<sup>2</sup>= .016). The prevalence of severe depression was lower than T1, with only 1 per cent of the sample falling into this category. If the T2 data had of contained more depressed young people a thorough examination of the subjects who had depression at T1 compared to those

without depression would have been undertaken, however the small sample precludes such detailed exploration.

Table 16

*Levels of depressed mood in current sample as scored by the CES-D*

	M	Depressed Mood		
		No Symptoms (%) of sample	Mild/ moderate (%) of sample	Severe (%) of sample
Time Two	19.64	52	46.7	1.3
Boys	18.07	59.3	37	3.7
Girls	20.85	47.9	52.1	0

The relationship between the T1 and T2 variables was explored via bivariate correlations. Table 17, shown on the next page, displays the correlation coefficients for the main measures at the two times. Variables not included such as actual social support, agreeableness, openness and positive life events, demonstrated no significant correlation to any of the main measures between T1 and T2,  $p < .05$ . No significant relationship was found between time one and time two depression, nor time one and two neuroticism,  $p < .05$ . Unexpectedly no relationship was found between neuroticism or extraversion and stress over time,  $p < .05$ . Interestingly, T1 and T2 bad life events correlated  $r = .290$ ,  $p < .05$ . As this scale is comprised of isolated negative events, the results suggest that the same young people who experienced a negative event at T1 went on to experience further independent negative events at T2. It is unclear if a larger sample would have yielded a relationship between T2 negative life events and depression.

The only variable at T2 which related to initial depression was stress, demonstrating a small effect size,  $r = .237$ ,  $p > .05$ . The stress generation effect was explored via further investigation of the relationship between T1 depression and T2 stress using regression. Linear regression found a

significant relationship between T1 depression and T2 stress,  $F(1, 74) = 4.331$ ,  $CI[-0.042, 0.154]$ ,  $p > .05$ . Suggesting that depression at T1 may have accounted for up to 15 per cent of the stress participants reported at T2. Importantly, this relationship occurred independent of the effect of personality and T2 depression. However, when controlling for the variance from T2 stress, this relationship became non-significant,  $F(1, 75) = 2.642$ ,  $CI[-0.038, 0.176]$ ,  $P < .05$ . This suggests the result should be interpreted with caution and further analysis of power in the current sample is required.

Table 17  
*Correlations of primary measures at time 1 and time 2*

Time Two	Time One					
	Depression	Life Bad	SSS	Stress	Neuroticism	Extraversion
Depression_2	.220	.2	.011	.131	.064	.089
Life Bad_2	.023	.290*	.069	.07	.02	-.132
Social Support Satisfaction (SSS)_2	.032	-.192	.041	-.037	-.059	-.079
Stress_2	.237*	.18	-.011	.219	.065	-.034
Neuroticism_2	.026	.094	-.039	.120	-.166	.122
Extraversion_2	-.111	-.061	.006	-.206	-.110	-.099

Note. \*Correlation is significant at  $p < .05$

**Post Hoc Power Analysis**

To investigate whether the non-significant results in the longitudinal study were due to a lack of statistical power, post hoc power analyses using GPower were undertaken (Erdfelder, Faul, & Buchner, 1996). Initially, power ( $1 - \beta$ ) was set at 0.80 and  $\alpha = .05$ , two-tailed. This showed that the T2 sample size would have to increase to  $N = 128$  to detect a small effect size of,  $R = .25$ , or as a more conservative measure with power ( $1 - \beta$ ) set at 0.95,  $N$  would of needed to increase to 225 participants to reach statistical significance at the .05 level. Thus it is very likely that the longitudinal results under report the relationship between the main variables. These findings were further

confirmed by repeated measures tests of the power between the T1 and T2 variables. The findings are illustrated in table 18 below. While the personality variables and depression may have had adequate statistical power to detect large effects, the life stress and social support variables did not. The power analysis suggests that if the original sample from T1 had of been retained the study may have yielded results. Given the apparent limited power in the T2 data, no further analysis of the data were undertaken.

Table 18.

*Post Hoc observed power measurement between variables at T1 and T2.*

Measure	Observed Power
Depression	.79
Neuroticism	.77
Extraversion	.79
Stress	.13
Social Support Satisfaction	.23
Life Bad	.082

### Discussion

The emergence of a depressive disorder during the formative years can be the precursor to a series of relapses throughout an individual’s life, and protracted episodes can profoundly disrupt a young person’s long term interpersonal functioning (Burcusa & Lacono, 2007). The current study aimed to explore the second and third pathways of the cognitive vulnerability transactional stress model (CVTSM), which posit that the transactions that a previously depressed young person has with their social environment, and long term changes resulting from depression, may partially explain their increased risk for relapse (Kochel, Ladd & Rudolph, 2012). The current study resurveyed 124 young people who took part in the time one study, two years after the initial wave. The objective of

the longitudinal analysis to elucidate mechanisms occurring in the transactional pathways, with the goal of informing targeted intervention programs. Partial support was obtained for the second pathway, the stress generation model, in which initial depression was found to predict subsequent stress two years later. However, no support was found for the consequence or the 'scar' model, which suggests that depressive symptoms create longer term social problems due to personality changes which emerge from the first episode. These findings and the sampling difficulties encountered at time two will be discussed.

The prevalence of depression at time two was significantly higher than time one. Nearly half of the sample reported depressive symptoms, as classified by the Center for Epidemiological Studies Depression Scale (CES-D; Caracciolo & Giaquinto, 2002). However, only one per cent of the sample fell into the severe clinical range. A small positive relationship was apparent between time one and two depression, however it was not significant,  $p < .05$ , suggesting the majority of young people recovered from the initial episode.

While depression has been found to increase with age, especially in females (i.e. Zahn-Waxler, Shirtcliff & Marceau, 2008), the trend in depressive symptoms at follow up was not comparable to time one, or the current Australian rates for older adolescents, which range from 5 -15 per cent of young people (Boyd et al., 2000). As such, it is possible that more youth with depressive symptoms were sampled at the follow up. This may be due to the nature of the sampling, in that young people to complete the questionnaire in his or her own time. Moreno and colleagues (2012) found that depressed older adolescents tend to spend more of their free time on the internet, as opposed to engaged in activities, and the follow up sample may be inflated by the availability of such youth to complete the study. This concept may also explain the high rate of incompleteness, whereby a third of the sample failed to complete 15 per cent of the questionnaire or more. It is possible that these participants began the study, and were not sufficiently motivated to complete it. Furthermore, the absence of severely depressed young people may be explained by symptoms stemming from

depression such as anhedonia and fatigue, which would render completing a battery of questionnaires in one's own time difficult.

The time two sample differed on many of the independent variables. They were significantly higher in neuroticism and extraversion and they were found to report higher levels of stress, but reported being more satisfied with their social support. Given such differences, bivariate correlations and structural equations modelling was used to examine the time two data in isolation prior to analysing the longitudinal results. The independent variables in the second study demonstrated similar results to the first, and again supported the vulnerability model, tested at time one. Specifically, neuroticism demonstrated the most influential relationship to depression, explaining over half of the variance in scores. Similarly, extraversion and social support satisfaction demonstrated a protective effect, and higher stress levels were linked to depression. Social support satisfaction was found to dampen the relationship between stress and depression, however it did not completely mediate the relationship. Finally, in contrast to study one, negative life events were not found to predict depression. However, as this scale is comprised of profound negative life events, this findings was likely due to the sample size, as only a small number of young people sampled experienced such occurrences.

The longitudinal data was then used to investigate pathways two and three of the CVTSM. The findings demonstrated partial support for the second pathway of the CVTSM, the stress generation hypothesis (Hammen, 2006). This pathway posits that due to stable characteristics many depressed young people have, they are more inclined to generate future stress (Hankin & Abramson, 2001). The generation of stress is posited to occur even in the absence of depression and may be the precursor to a future depressive episode. The stress generation theory suggests that stable individual traits, such as high neuroticism may lead to maladaptive relationship appraisals, heightened stress responses, and social behavioural deficits, such as excessive reassurance seeking, which generate interpersonal stress, leading to a relapse (Hammen & Brennan, 2002). Within the

theory, young people are seen as active agents in shaping their environments and social functioning, and as such may be responsible for the greater stress they encounter (Lui et al., 2012).

The longitudinal study found evidence in support of the stress generation model, in that, depression at time, resulted in higher levels of stress at time two. Specifically, up to 15 per cent of the variance in stress, two years later, was attributable to initial depression. The relationship between time one depression and time two stress existed independent of the influence of current depression, suggesting that even in the event depression remits, a subset of individuals who were previously depressed continue to report heightened stress. Importantly, no relationship was found between time one depression and independent negative life events at time two, suggesting the stressors encountered by the sample were not independent in nature, and may have been generated by the young person's own behaviour. Overall, the findings suggest that key individual aspects or symptoms stemming from a depressive episode likely remain salient, and continue to generate dependent stress.

The current findings are in line with a robust subset of psychological literature which found the stress generation effect to be a unique phenomenon in previously depressed individuals (Hammen, 1991; Leskela et al., 2009; Paykel, 1994; Uliaszek et al., 2012). The current results highlight the stability of the stress response over time and suggest that the stress response may be a stable characteristic of depression prone young people that emerges early in development and reflects their unique temperament (Compas et al., 2004). This is known as the stress continuation theory, whereby the prospective relationship between stress and depression is attributed to the continuity or stability of the stress response over time (Uliaszek et al., 2012).

While the stress response may be a stable characteristic, the current study did not find it to be the result of personality traits such as neuroticism or interpersonal functioning. As such, it is unclear in the current study what the mechanism may be through which stress continues after depression remits. It is possible that the effect measured may be due to chronic long term stress which is born from attempting to meet milestones which are missed during a depressive episode.

For example, it is possible that the heightened stress at follow up stemmed from failing to meet milestones, such as falling behind in academic milestones at time one, which is a common struggle for depressed young people. As such, they would likely have additional stress in catching up with their peers. Given home life stress was linked to depression, it is also possible that the experience of chronic high stress may reflect the young person's day to day social world, and that depressive episodes occur on the background of the difficult or chronically stressful home life.

The third pathway of the CVTSM, the consequence model, posits that a sustained episode of depression during the formative years can lead to personality changes, such as heightened neuroticism, that result in increased stress susceptibility and drive maladaptive social functioning (Monroe & Harkness, 2005). Specifically, young people with a history of depression are often characterised by high levels of neuroticism, they lack social confidence, seek reassurance and are interpersonally dependent (Shea, Leon, Mueller, & Solomon, 1996). The depressive episode during adolescence is posited to cause a lasting change in personality, such as elevating levels of neuroticism, hence leaving a long term scar (Jylha et al., 2009; Rohde et al., 1994). The scar is thought to lead to poor social functioning over the long term and elevate the long term risk of relapse.

The current study did not find evidence to support the consequence model. While neuroticism increased between time one and two, initial depression was not linked to this increase. Nor did time one negative life events result in increases in neuroticism as suggested by the kindling theory, whereby a trauma during adolescence is hypothesised to lead to long term neurobiological changes (Monroe & Harkness, 2005). There was also no relationship between time one depression and reduced social support. This is in contrast to studies completed in adolescent samples supporting the scar and life stress theory (i.e. Abena & Hankin, 2008; Kendler et al., 2000; Monroe & Harkness, 2005; Nolen-Hoeksema et al., 1992; Rohde et al., 1994).

It is possible that the results for pathways two and three, found in the current study are



actually the intermediate in a three part model. Lui (2012) criticised the current stress generation literature, suggesting that the current published works only provide a partial assessment of the etiological change underlying the chronicity of depression, and noting that only one study to date has looked at stress generation over three waves ( i.e. Bos, Bouhuys, Geerts, Van Os & Ormel, 2007). In this study, Bos and Colleagues (2007) found that interpersonal stress predicted depression recurrence, and at time two dependent stress mediated this effect. Specifically, it is possible that had a third wave have taken place, the effect found at time two (depression predicts stress) may have resulted in depression and social support changes at time three. The generation of stress is theorised to exist as a sequelae to interpersonal problems and depression contagion, and as such, a third wave may detected this aspect of the theory.

Such criticism could also be applied to the consequence model findings. It is possible that scars from depression take longer to become apparent. The current study did find that time one depression resulted in increased stress at time two, suggesting there may have been an increase in stress reactivity which emerged from the first depressive episode, and it had not yet resulted in sustained personality changes. As such, there may have been scars present in the previously depressed young people, however they are in the form of stress susceptibility, not personality changes.

Future research could improve the current study in two key ways. Firstly a larger sample is required. Depression is a growing phenomenon which is a burden of disease, however within the total population it remains relatively rare, suggesting the vast majority of young people do not become depressed. As such, in order to adequately study such phenomena with a myriad of small, inter and intra personal changes a very large population sample is needed. Power analysis recommended over 200 young people would need to be sampled at follow up. The number of incomplete surveys returned in the current study would suggest that if young people are asked to complete the survey in their own time, over 300 are included in the follow up. The sample should be drawn from Australia wide as only using Canberran young people in the follow up may have bias the

results. Specifically, Canberra is a unique economic, social and political climate, whereby young people have the highest rates of education in the country (ABS, 2009). As such, they may not be representative of Australia young people. In addition, study could be improved with a three wave design, following up the sample again in two years, as discussed throughout this section.

The current longitudinal study had some considerable strengths, and treatment recommendations which can be born from it. The study was able to follow up a large proportion of the original respondents two years following the initial wave, providing a longitudinal analysis of the main variables. All the scales were found to be reliable, valid, and able to be applied to real world psychological phenomena and inform treatment. The results suggest that depression may lead to future stress, and initial treatments for depression should target potential stress generation mechanisms to reduce the risk of relapse. Given depressed young people tend to experience dependent stress that is at least partially due to their own behaviour, a clinical analysis of the areas whereby the young person may generate stress and treatment of these mechanisms (i.e. excessive reassurance seeking) may assist in reducing the chances of relapse. CBT has been found to assist in coping skills and adaptive cognitive responses to stress, and behavioural activation may have the side effect of increasing social support. Therapy which incorporates skills to cope with stress should and also addresses the physiological experience of heightened stress may assist depressed individuals to develop the necessary skills to cope with the higher rates of dependent stress that they encounter throughout their lives (Lui, 2012).

## CHAPTER FOUR: General Discussion

**Discussion**

The aim of the current body of work was to examine the predictive associations between depression, social functioning and personality within the three pathways of the cognitive vulnerability transactional stress model (CVTSM). The current thesis explored the CVTSM in an adolescent sample using a longitudinal design, with established measures for adolescent populations. The current study is the first to the author's knowledge to examine the transactional role of personality, social support, and depression in Australian youth. The three pathways provide a framework for analysing the simultaneous processes which take place between a young person and their social world and may elucidate the mechanisms underlying high rates of relapse in adolescent onset depression. Understanding the interplay between young people's stable traits, their social functioning and depression is critical in understanding the processes involved in the etiology of adolescent onset depression, which is an increasing burden of disease (Mathers, Fat, & Boerma, 2008). The results of the current study demonstrated support for the vulnerability model and the stress generation model (first and second pathways of the CVTSM), while no support was found for the consequence model (third pathway). The current study found inherent personality traits such as neuroticism to be the strongest risk factor for adolescent depression. In addition, stress and depression were demonstrated to share a reciprocal relationship, with neuroticism and interpersonal stress increasing susceptibility to depression, and depression, in turn, predicting a greater likelihood of subsequent stressors. Social support was found to partially mediate the effect of neuroticism on depression, suggesting it offers a small protective effect for neurotic individuals, while profound negative life events were found to be a risk factor for depression regardless of personality or social support. These findings, treatment recommendations and future research implications will be discussed.

**Pathway One: The Vulnerability Model**

Consistent with the vulnerability model, the current study found the personality trait neuroticism to be the strongest risk factor for adolescent depression. The vulnerability model is based on a diathesis-stress formula, suggesting that personality traits such as neuroticism are a genetically predisposed vulnerability which increase stress sensitivity and susceptibility to negative mood states (Hankin & Abela, 2005). These vulnerabilities are thought to lay dormant until they are triggered by stress, and then result in the individual having difficulty re-establishing emotional equilibrium (Kendler et al., 2004). As a result, the susceptibility to the depressogenic effects of initial stress is heightened, as is the probability of depression ensuing. The current study supported this concept, noting that in and of itself neuroticism was a robust predictor of depression, however when combined with stress the risk increased significantly. The current findings that neuroticism and stress were the strongest predictor of depression is in line with a preponderance of classical and current literature suggesting innate personality traits such as neuroticism are one of the strongest predictors of childhood onset depression (Auerbach et al., 2001; Beck, 1967; Hankin & Abela, 2005; Ormel, Rijksdijk, Sullivan, van Sonderen, & Kempen, 2002).

Stress which was predominately born from the interpersonal domain, such as home life stress and peer pressure was found to be most noxious for neurotic young people. This is in line with a number of studies that have suggested individuals high in neuroticism are particularly sensitive to stress in the interpersonal domain (Rubin & Bukowski, 2011; Chango, Boykin McElhaney, & Allen, 2009). The results suggest that personality may be a lens through which the world is interpreted, and that stable aspects of the individual influence the way they interpret, manage and cope with the interpersonal stress.

**Pathway Two and Three: Stress Generation and Consequence Model**

The current study also found that depression increases the susceptibility to stress. Specifically, even in the absence of current depressed mood, stress remained elevated two years later in previously depressed young people. This finding supported the stress generation pathway,

which suggests that young people who are depressed continue to experience heightened stress, often of an interpersonal nature, even when they are in remission from depression (Hammen, 1991). As the young person is no longer depressed, the elevated stress cannot be attributed to their current mood state or the behaviours which result from depression (i.e., withdrawal, irritability). Consequently, the stress generation effect was thought to have an underlying mechanism that was specific to the individual but also a common trait which depressed youth shared, such as neuroticism (Hammen, 1991; Lui & Alloy, 2010). Neuroticism was thought to be a possible mechanism given its temporal stability, sound empiric links to adolescent depression and behavioural manifestations which cause interpersonal stress (i.e. excessive reassurance seeking).

However, the current study did not support the notion that the stress generation effect may be partially due to personality. Nor did depression result in sustained changes in personality or social functioning over time. The only variable which time one depression predicted two years later was stress. The results suggest that stress is chronically elevated in young people who have ever experienced depression. Stress is a well-established risk factor for adolescent depression, and chronic stress which begins early in adolescence is often linked to impoverished upbringings and enduring stress due to the environmental factors such young people are exposed to (Hammen, 2005; Kendler et al., 2004). It is possible that stress is a relatively stable risk factor, and that depressive episodes are superimposed over the stress. From the current study there is no evidence that depression changes inherent aspects of the young person, nor does it appear to increase stress susceptibility as the relationship between stress and depression was already apparent in such young people at time one.

#### **Role of Social Support within the CVTSM**

Social support satisfaction was found to dampen the relationship found in the vulnerability model, and as such may have a small protective effect regarding the relationship between neuroticism and depression. In disparity with the stress buffering theory of social support, satisfaction with support and actual support was not found to buffer the effects of stress in non-

neurotic young people. Specifically, it is possible that individuals high in neuroticism have a preference toward social support to maintain emotional equilibrium, whereas non-neurotic individuals may be able to use independent strategies. This suggests that social support may offer a small benefit in increasing adaptive coping and protecting against depression in neurotic young people. A similar finding was demonstrated by Dwyer and Colleagues (2014) who found that social support was particularly beneficial for those high in neuroticism in preventing depression. However, the current study found that the protective effect was small in comparison to the risk demonstrated by the vulnerability model and would be unlikely to offset a large proportion of the inherent liability neuroticism brings. While social support did have a protective role within the vulnerability model, no changes in social support were noted over time, and social support was not found to be a significant variable in the second or third pathways of the CVTSM.

#### **Additional Pathways to Depression and Protective Factors**

The current study found that in addition to the vulnerability model and the stress generation effect, there was another prominent risk pathway to depression in the study. The second pathway was through significant psychosocial adversity, whereby profound negative events lead directly to depression. The findings that a significant life stress can result in depression are well established in the literature (Johnson, Whisman, Corley, Hewitt & Rhee, 2012; Hammen, 2005). However, the current study extended the findings by differentiating the role of profound negative life events in depressive illness, and the role of personality and stress on depression. Specifically the current study showed profound events can result in depression and that this does not interplay with personality, as such, the death of a parent or other traumatic event, may in and of itself cause depression regardless of the individual's susceptibility to negative mood states.

In addition, a protective pathway was also found, in that high levels of trait extraversion protected against depression. The current study supported the two factor model of personality (Eysneck & Eysneck, 1975) as only neuroticism and extraversion were found to interact with depression. Extraversion was not implicated in any of the CVTSM pathways, nor did it demonstrate

a relationship to social support or profound negative life events. As such, extraversion exhibited a straightforward protective effect against depression. The study suggests that extraversion offers an inherent protective effect against depressive illnesses and this may be due to the stable aspects of extraverted individuals. Specifically those high in extraversion often seek new experiences, utilise problem focused coping and have a positive disposition (Murberg, 2009). Such factors may assist them to establish emotional equilibrium during difficult times.

### **Overview of Pathways to Depression**

Overall, the current findings suggest that there may be a number of different direct and indirect pathways risk pathways to depression. Firstly via the vulnerability model, whereby stress acts on an inherent vulnerability, and secondly due to adverse events which result in depression independent of personality vulnerabilities. The current study suggests stress can interact with personality but that profound stress does not require any vulnerability to cause depression. Within the two models social support was only found to play a role in offsetting some of the risk associated with the vulnerability model.

From the results it is difficult to ascertain the temporal patterning of associations between the main variables. It would appear that stress plays an important role in the initial susceptibility to neurotic young people becoming depressed, but depressive symptoms may also increase stress over time. As such, it is possible that stress is the ignition point for each depressive episode. In line with the recommendations of Hankin and Colleagues (2010) the current findings highlight the importance of prevention as once this complex and dynamic process among stress, personality and depressive symptoms initiates the effects accumulate and magnify over time (Hankin, Stone, Ann Wright, 2010). Specifically, if stress never returns to a baseline after each depressive episode it is effectively a residual symptom of depression that remains untreated and may be a risk factor for relapse.

### **Future Directions**

The present study emphasises the importance of appropriately timed interventions aimed at multiple facets of risk, including the complex dynamics among stress, interpersonal functioning and

depression, over time. In terms of prevention, the current study suggests that programs which aim to modify trait neuroticism would likely have the most impact in reducing the risk to depression (Barlow et al., 2014; Clarke & Currie, 2009). Such concepts are in their infancy as traditionally neuroticism was thought to be treatment resistant and stable. However, future research into treatment programs would clarify if this were a suitable area, amendable to change. In addition, the findings confirm the NICE (2005) guidelines recommendation that young people who experience a significant negative life event should be offered counselling as they are at an increased risk of becoming depressed. While the interpersonal sphere was not a robust variable within the current study, there was evidence that improving a young person's perception of their social support would be beneficial, and as such, IPT therapy may be of particular benefit to neurotic young people.

The current study suggests that in addition to traditional therapeutic approaches such as CBT and IPT, which target cognitive vulnerabilities and interpersonal deficits, intervention aimed at decreasing stress generation may be promising (Calvete et al., 2012). Stress generation is a complex phenomenon which likely incorporates appraisals of threat, cognitive beliefs about the stressor, physiological responses and coping choice. Targeting areas that neurotic young people are likely to have deficits in, would be beneficial in teaching stress management.

While the mechanism between stress, neuroticism and depression is unclear at present, preliminary works have investigated the presence of depressive schemas that are activated by stress in neurotic young people (Daffern, Gilbert, Lee & Chu, 2015). Schemas are complex internal structures of stimuli, ideas, cognitions, and experiences that are used as a heuristic for the filtering and processing of new information (Ingram & Luxton, 2005). Evans and colleagues (2005) demonstrated that holding a negative self-schema was a risk factor for depression, and these tend to be present for neurotic young people (Calvete, 2014). The content of these schemas tends to be dysfunctional and this colours the individuals appraisal of themselves, the world, their coping abilities and the future (Abela & Hankin, 2008).



Schemas may serve to reinforce already heightened emotional responses occurring in neurotic young people when they are under stress and lead to feelings of being unable to cope. Feelings of inability to cope may lead to external locus of control, in that neurotic young people tend to rely on external sources, such as social support, to re-establish emotional equilibrium. There is no doubt that neurotic young people have more interpersonal difficulties and these are one of the key reasons which brings them into therapy (Mandelli et al., 2015). In addition, schemas have been found to be stable and maintained even in the event depression remits, suggesting they may be partially responsible for stress generation.

In terms of additional therapeutic interventions, techniques such as biofeedback may assist young people with high levels of neuroticism to understand their heightened emotional responses to stress and could be used in vivo during therapy as a form of exposure (Gustainiene, Perminas, Peculiene & Jarsiunaite, 2015). Once an individual is able to link their emotional experience to their cognitive world they may find exploring the behavioural manifestations of their emotions easier. Specifically, treatment which incorporates understanding the internal physiological, emotional, cognitive and behavioural manifestations of the vulnerability model could present as a comprehensive treatment program.

### **Strengths and Limitations of the Study**

The current findings need to be considered in context of the strengths and weaknesses of the study. The strengths include the large representative sample size and the longitudinal design which allowed changes in three different processes to be analysed simultaneously. The sample was drawn from across Australia allowing it to be related to Australian youth as a whole and represented a wide range of socioeconomic strata.

There are several areas which future research could attempt to improve. Common method variance bias is always a concern in single respondent self-report measures. Future research could strengthen the empirical validity by gathering information from other sources such as parents and teachers. This would be especially useful for the social support and coping variables in the current

study, as it is likely that they lacked specificity into the various aspects of the social sphere and behavioural manifestations of coping. A sound methodological approach for future studies would be to include a measure of coping with an objective measure of self-report such as a daily journal of stress, action and outcome. Within this measure the young person could also write an objective journal of social support instances and behaviours. A larger sample size would also improve the design, as would a three wave follow up, to investigate the presence of the third pathway- the consequence model. Future researchers should also be mindful of the inflated Type I error that can occur when a large number of analysis are conducted in this form of research. As suggested previously, increasing the number of participants may reduce the risk of spurious association and such error.

### **Conclusion**

The findings of the current studies suggest there are both direct and indirect pathways that transact over time to produce a very complex model of adolescent depression. When the two studies are considered together, the findings regarding the vulnerability model and the stress generation effect suggests that stress and depression share a reciprocal relationship, with personality and interpersonal stress increasing susceptibility to depression, and depression in turn being associated with greater likelihood of subsequent stressors. Stable forms of dysfunction such as personality and depression were found to drive and influence upon less stable difficulties such as stress and social support, however over time it appears to be the relationship between stress and depression which requires intervention. Additionally, the study found there are a proportion of young people, without a vulnerability who will become depressed as a result of significant life events.

While the transactions taking place are bound to have an effect on young people's interpersonal functioning, and it is possible that social support can offer a protective effect to neurotic young people, the relationship pales in comparison to the robust effect of the vulnerability model. Future research should aim to further the current study by using experimental designs to

explore the exact mechanisms that explain the relationship between stress, neuroticism and depression. While future clinical interventions should aim to treat stress and teach appropriate coping to neurotic youngsters and also develop interventions to modify trait neuroticism.

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Table 6.  
Correlations of primary measures at Time 1

	Sample	Depression	Life Good	Life Bad	SSS	SSA	Stress	Coping	Neuroticism	Extraversion	Agreeableness	Openness
Depression	Male	-										
	Female	-										
	Total	-										
Life Good	Male	-.121	-									
	Female	.159*	-									
	Total	0	-									
Life Bad	Male	.254**	-.24	-								
	Female	.412**	.083	-								
	Total	<b>.330**</b>	<b>.026</b>	-								
Social Support Satisfaction (SSS)	Male	-.218**	.26	-.056	-							
	Female	-.312**	-.138	-.056	-							
	Total	<b>-.237**</b>	<b>-.063</b>	<b>-.059</b>	-							
Social Support Actual (SSA)	Male	-.155	-.016	-.016	.356**	-						
	Female	-.294**	-.020	-.143*	.414**	-						
	Total	<b>-.207**</b>	<b>-.039</b>	<b>-.090</b>	<b>.396**</b>	-						
Stress	Male	.317**	.099	.095	-.157	-.080	-					
	Female	.577**	.009	.381**	-.226**	-.213**	-					
	Total	<b>.500**</b>	<b>.019</b>	<b>.242**</b>	<b>-.168**</b>	<b>-.130*</b>	-					
Coping	Male	.020	-.024	-.067	.073	.126	.023	-				
	Female	-.108	-.040	.038	.213**	.296	-.031	-				
	Total	<b>-.045</b>	<b>-.037</b>	<b>-.019</b>	<b>.143**</b>	<b>.213**</b>	<b>.001</b>	-				
Neuroticism	Male	.500**	-.089	.211*	-.193*	-.235**	-.118	-.118	-			
	Female	.714**	.009	.209**	-.257**	-.296**	.599**	-.117	-			
	Total	<b>.643**</b>	<b>-.089</b>	<b>.190**</b>	<b>-.171**</b>	<b>-.209**</b>	<b>.570**</b>	<b>-.097</b>	-			
Extraversion	Male	-.221**	.060	.007	-.026	.010	.235**	.235**	-.027	-		
	Female	-.288**	.075	-.216**	.027	.222**	-.077	.317**	-.147*	-		
	Total	<b>-.257**</b>	<b>.070</b>	<b>-.101</b>	<b>-.004</b>	<b>.112*</b>	<b>-.040</b>	<b>.271**</b>	<b>-.096</b>	-		
Agreeableness	Male	-.042	-.003	-.071	.231**	.079	.127	.210*	-.014	.050	-	
	Female	-.038	.007	-.086	-.008	.080	.072	.342**	.030	.312**	-	
	Total	<b>-.1</b>	<b>-.23</b>	<b>-.082</b>	<b>.117*</b>	<b>.099</b>	<b>.116*</b>	<b>.280**</b>	<b>.061</b>	<b>.186**</b>	-	
Openness	Male	-.051	.007	-.124	.206**	.091	.103	.258**	-.047	.150	.654**	-
	Female	-.081	.124	-.127	.045	.130	.043	.313**	-.084	.228**	.537**	-
	Total	<b>-.062</b>	<b>.054</b>	<b>-.126*</b>	<b>.125*</b>	<b>.116*</b>	<b>.073</b>	<b>.285**</b>	<b>-.053</b>	<b>.189**</b>	<b>.582**</b>	-

Note. \*\* Correlation is significant at  $p < .01$

\*Correlation is significant at  $p < .05$

## **APPENDIX A:**

**Participant information sheets, consent forms and debrief**

DEPARTMENT OF PSYCHOLOGY  
Building No. 39  
Canberra ACT 0200 Australia  
Telephone: 61255545  
Facsimile: 61250499  
www.anu.edu.au

Researcher: Kate Fenton (BSc Science (Psychology) Hons. / B Arts)  
Provisional Psychologist  
Doctorate Candidate

Project: Adolescent Depression: The Relationship Between Neuroticism and Available Social Support

Participant Consent Form

Dear Participant,

The current research is being conducted as part of a Clinical Doctorate Project by Kate Fenton from the School of Psychology at the ANU. The current study forms part of the major Doctorate Thesis. The research is supervised by Professor Don Byrne. This research has been approved by the ANU Human Research Ethics Committee, protocol number....

By signing this form I agree to participate in a study investigating various facets of adolescence; including adolescent depression, personality and how I cope with stress. I understand that there are no right or wrong answers and I will try to answer as honestly as possible.

I understand that my participation comprises responding to questions displayed on a computer by selecting the appropriate response with my mouse. I have been made aware that my participation is voluntary that I am not required to participate. I know that I am able to withdraw at any stage of the survey for any reason without any consequences. I understand that my responses are anonymous and that as far as the law allows no effort will be made on the part of the researchers to identify my responses at any part of the data collection or analysis process. The data collected today must remain by law at the ANU under lock and key for 7 years and thereafter will be destroyed.

Name: ... ..

Signature: ... ..

Date: ... ..

DEPARTMENT OF PSYCHOLOGY  
Building No. 39  
Canberra ACT 0200 Australia  
Telephone: 61255545  
Facsimile: 61250499

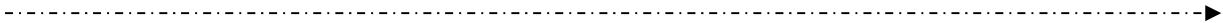
Dear Parent or Guardian,

Your child's school, SCHOOL NAME, has agreed to participate in a study run by the Australian National University School of Psychology and approved by the ANU Human Research Ethics Committee (protocol number ...). The research will be conducted by Kate Fenton, who is a Provisional Psychologist and Doctorate candidate and it is supervised by Professor Don Byrne. It is possible your child may be asked to complete a set of questionnaires during class time during the second semester.

The purpose of this study is to gain a greater understanding of the experience of adolescent depression and various factors, such as coping and personality which may impact on a young person's ability to gain adequate social support during this transition period. In light of the increasing prevalence of depression in the adolescent years, and the multitude of negative impacts that may stem it, the present study aims to investigate possible protective factors. Due to the need for research in this area I hope to provide useful information for health providers, public policy and schools to promote the mental health of today's youth.

Please indicate in the space below if you give permission for your child to participate in the research. If for any reason you object to your child taking part please indicate this by circling the appropriate option. Your child will also have the opportunity to withdraw from participation, but if they do complete the survey their responses will remain anonymous as far as the law allows. All students will be provided with an information sheet upon completion. We will also inform SCHOOL NAME of our results in late 2011. If you have any questions regarding this research please contact Kate on [kate.fenton@anu.edu.au](mailto:kate.fenton@anu.edu.au).

Yours sincerely,  
Kate Fenton



Please circle one option below

**I give permission/ I do not give permission** for my child to participate in the current study by Kate Fenton

Student Name: ... ..      Year/Tutor Group: ... ..  
Parent/Guardian Name: ... ..      Signature: ... ..  
Date: ... ..

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Project: Adolescent Depression: The Relationship Between Neuroticism and Available Social Support

Dear Participant,

The purpose of this study was to explore various aspects of adolescent depression, and the link between depression, personality and available social support. How you cope with stress and changes in your life was also explored.

The researcher is gathering data from year 7 to 11 students around Canberra. Once the data is collected the results will be analysed to determine the relationship between adolescent depression, personality, coping and social support. Thankyou for taking part in the research. Your participation is much appreciated and will contribute to policy makers and health professionals in order to understand the unique experience of adolescence.

If you have any further questions regarding the study please contact Kate Fenton on [kate.fenton@anu.edu.au](mailto:kate.fenton@anu.edu.au). This research was approved by the Australian National University's Human Research Ethics Committee, protocol number...If you have any concerns or feel uncomfortable contacting any of the researchers or the supervisor please contact the Australian National University's Human Ethics Committee on 6125 7945 or [Research.Office@anu.edu.au](mailto:Research.Office@anu.edu.au).

**If you feel you need to speak to a professional regarding any psychological concerns please contact in the first instance your school counsellor. Kid's Helpline are there to listen at all hours on 1800 55 1800 if you need to talk to someone outside of school time.**

**APPENDIX B:**

**MEASURES**

## Adolescent Stress Questionnaire - ASQ

Here are some statements about things or situations which you might find stressful. Please tell us how stressful each of these things or situations has been for you **in the past year**, by circling one number from 1-5 depending on whether you have found this:

- 1 Not at all stressful (or is irrelevant to me)
- 2 A little stressful
- 3 Moderately stressful
- 4 Quite stressful
- 5 Very stressful

Please respond to all items in this section. If you have not experienced something, circle 1 = not at all stressful (or is irrelevant to me).

---

1. Disagreements between you and

your father

1      2      3      4      5

2. Not being taken seriously

1      2      3      4      5

3. Getting up early in the morning

1      2      3      4      5

4. Little or no control over your life

1      2      3      4      5

5. Having to study things you do

not understand

1      2      3      4      5

6. Teachers expecting too much from you

1      2      3      4      5

7. Concern about your future

1      2      3      4      5

8. Being hassled for not fitting in

1      2      3      4      5



9. Keeping up with school work	1	2	3	4	5	
10. Employers expecting too much of you	1	2	3	4	5	
11. Having to take on new family responsibilities as you get older		1	2	3	4	5
12. Difficulty of some subjects	1	2	3	4	5	
13. Abiding by petty rules at home		1	2	3	4	5
14. Having to concentrate for too long during school hours	1	2	3	4	5	
15. Inadequate school resources		1	2	3	4	5
16. Having to study things you are not interested in		1	2	3	4	5
17. Being ignored or rejected by a person you want to go out with		1	2	3	4	5
18. Disagreements between you and your teachers		1	2	3	4	5
19. Not enough time to have fun		1	2	3	4	5
20. Putting pressure on yourself to meet your goals		1	2	3	4	5

21. Disagreements with your brothers and sisters	1	2	3	4	5
22. Pressure to work to make money	1	2	3	4	5
23. Not enough time for leisure activities	1	2	3	4	5
24. Too much homework	1	2	3	4	5
25. Not getting enough feedback on schoolwork in time to be helpful	1	2	3	4	5
26. Not enough time for activities outside school hours	1	2	3	4	5
27. Making the relationship work with your boyfriend/girlfriend	1	2	3	4	5
28. Being judged by your friends	1	2	3	4	5
29. Disagreements between your parents	1	2	3	4	5
30. Changes in your physical appearance with growing up	1	2	3	4	5
31. Arguments at home	1	2	3	4	5
32. Pressure to fit in with peers	1	2	3	4	5
33. Compulsory school attendance	1	2	3	4	5

34. Having to make decisions about future work or education	1	2	3	4	5
35. Living at home	1	2	3	4	5
36. Satisfaction with how you look	1	2	3	4	5
37. Disagreements between you and your mother	1	2	3	4	5
38. Not enough money to buy the things you want	1	2	3	4	5
39. Going to school	1	2	3	4	5
40. Not enough time for your boyfriend/ girlfriend	1	2	3	4	5
41. Teachers hassling you about the way you look	1	2	3	4	5
42. Abiding by petty rules at school	1	2	3	4	5
43. Pressure of study	1	2	3	4	5
44. Lack of trust from adults	1	2	3	4	5
45. Not being listened to by teachers	1	2	3	4	5
46. Parents expecting too much from you	1	2	3	4	5

47. Having to take on new financial responsibilities as you grow older	1	2	3	4	5
48. Lack of understanding by parents	1	2	3	4	5
49. Parents hassling you about the way you look	1	2	3	4	5
50. Work interfering with school and social activities	1	2	3	4	5
51. Not enough money to buy the things you need	1	2	3	4	5
52. Getting along with your boyfriend/ girlfriend	1	2	3	4	5
53. Lack of freedom	1	2	3	4	5
54. Peers hassling you about the way you look	1	2	3	4	5
55. Lack of respect from teachers	1	2	3	4	5
56. Disagreements between you and your peers	1	2	3	4	5
57. Getting along with your teachers	1	2	3	4	5
58. Breaking up with your boyfriend/ girlfriend	1	2	3	4	5

**Center for Epidemiologic Studies Depression Scale (CES-D), NIMH**

Below is a list of the ways you might have felt or behaved. Please tell me how often you have felt this way during the past week.

Week	During the Past			
	Rarely or none of the time (less than 1 day)	Some or a little of the time (1-2 days)	Occasionally or a moderate amount of time (3-4 days)	Most or all of the time (5-7 days)
1. I was bothered by things that usually don't bother me.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
2. I did not feel like eating; my appetite was poor.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
3. I felt that I could not shake off the blues even with help from my family or friends.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
4. I felt I was just as good as other people.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
5. I had trouble keeping my mind on what I was doing.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
6. I felt depressed.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
7. I felt that everything I did was an effort.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
8. I felt hopeful about the future.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
9. I thought my life had been a failure.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
10. I felt fearful.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
11. My sleep was restless.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
12. I was happy.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
13. I talked less than usual.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
14. I felt lonely.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
15. People were unfriendly.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
16. I enjoyed life.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
17. I had crying spells.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
18. I felt sad.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
19. I felt that people dislike me.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
20. I could not get "going."	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

SCORING: zero for answers in the first column, 1 for answers in the second column, 2 for answers in the third column, 3 for answers in the fourth column. The scoring of positive items is reversed. Possible range of scores is zero to 60, with the higher scores indicating the presence of more symptomatology.

Junior Eysenck & Eysenck personality trait measure

Unable to attach a copy as this is copy write protected material which requires purchase

Adolescent Orientation for Problem Experiences dispositional inventory (A-COPE; Patterson & McCubbin, 1987).

**Scale items:**

When you face difficulties or feel tense, how often do you...

1. Go along with parent's requests and rules
2. Read
3. Try to be funny and make light of it all
4. Apologize to people
5. Listen to music-stereo, radio
6. Talk to a teacher or counselor at school about what bothers you
7. Eat food
8. Try to stay away from home as much as possible
9. Use drugs prescribed by doctor
10. Get more involved in activities in school
11. Go shopping, buy things you like
12. Try to reason with parents and talk things out, compromise
13. Try to improve yourself (get body in shape, get better grades, etc.)
14. Cry
15. Try to think of the good things in your life
16. Be with a boyfriend or girlfriend
17. Ride around in the car
18. Say nice things ("warm fuzzies") to others
19. Get angry and yell at people
20. Joke and keep a sense of humor
21. Talk to a minister/priest/rabbi
22. Let off steam by complaining to family members
23. Go to church
24. Use drugs (not necessarily prescribed by a doctor)
25. Organize your life and what you have to do
26. Swear
27. Work hard on school work or school projects
28. Blame others for what's going on

29. Be close with someone you care about
30. Try to help other people solve their problems
31. Talk to your mother about what bothers you
32. Try, on your own, to figure out how to deal with your problems or tension
33. Work on a hobby you have (sewing, biking, etc.)
34. Get professional counseling (not a school teacher or school counselor)
35. Try to keep up friendships or make new friends
36. Tell yourself the problem(s) is not important
37. Go to a movie
38. Daydream about how you would like things to be
39. Talk to a brother or sister about how you feel
40. Get a job or work harder at one
41. Do things with your family
42. Smoke
43. Watch T. V.
44. Pray
45. Try to see the good things in a difficult situation
46. Drink beer, wine, liquor
47. Try to make your own decisions
48. Sleep
49. Say mean things to people, be sarcastic
50. Talk to your father about what bothers you
51. Let off steam by complaining to your friends
52. Talk to a friend about how you feel
53. Play video games (Space Invaders, Pac-Man), pool, pinball, etc.
54. Do a strenuous physical activity (jogging, biking, etc.)

**USE THE FOLLOWING RESPONSE CATEGORIES.**

- 1. Never**
- 2. Hardly**
- 3. Sometimes**
- 4. Often**
- 5. Most of the time**







1. Whom can you really count on to listen to you when you need to talk?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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2. Whom could you really count on to help you if a person whom you thought was a good friend insulted you and told you that he/she didn't want to see you again?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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3. Whose lives do you feel that you are an important part of?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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4. Whom do you feel would help you if you were married and had just separated from your spouse?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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5. Whom could you really count on to help you out in a crisis situation, even though they would have to go out of their way to do so?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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6. Whom can you talk with frankly, without having to watch what you say?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
--------------------	----------------------	------------------------	---------------------------	-------------------------	-----------------------

7. Who helps you feel that you truly have something positive to contribute to others?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
--------------------	----------------------	------------------------	---------------------------	-------------------------	-----------------------

8. Whom can you really count on to distract you from your worries when you feel under stress?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
--------------------	----------------------	------------------------	---------------------------	-------------------------	-----------------------

9. Whom can you really count on to be dependable when you need help?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
--------------------	----------------------	------------------------	---------------------------	-------------------------	-----------------------

10. Whom could you really count on to help you out if you had just been fired from your job or expelled from school?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
--------------------	----------------------	------------------------	---------------------------	-------------------------	-----------------------

11. With whom can you totally be yourself?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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12. Whom do you feel really appreciates you as a person?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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13. Whom can you really count on to give you useful suggestions that help you to avoid making mistakes?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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14. Whom can you count on to listen openly and uncritically to your innermost feelings?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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15. Who will comfort you when you need it by holding you in their arms?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
--------------------	----------------------	------------------------	---------------------------	-------------------------	-----------------------

16. Whom do you feel would help if a good friend of yours had been in a car accident and was hospitalized in serious condition?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
--------------------	----------------------	------------------------	---------------------------	-------------------------	-----------------------

17. Whom can you really count on to help you feel more relaxed when you are under pressure or tense?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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18. Whom do you feel would help if a family member very close to you died?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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19. Who accepts you totally, including both your worst and your best points?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
--------------------	----------------------	------------------------	---------------------------	-------------------------	-----------------------

20. Whom can you really count on to care about you, regardless of what is happening to you?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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21. Whom can you really count on to listen to you when you are very angry at someone else?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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22. Whom can you really count on to tell you, in a thoughtful manner, when you need to improve in some way?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
--------------------	----------------------	------------------------	---------------------------	-------------------------	-----------------------

23. Whom can you really count on to help you feel better when you are feeling generally down-in-the-dumps?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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24. Whom do you feel truly loves you deeply?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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25. Whom can you count on to console you when you are very upset?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
--------------------	----------------------	------------------------	---------------------------	-------------------------	-----------------------

26. Whom can you really count on to support you in major decisions you make?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
--------------------	----------------------	------------------------	---------------------------	-------------------------	-----------------------

27. Whom can you really count on to help you feel better when you are very irritable, ready to get angry at almost anything?

No one	1)	4)	7)
	2)	5)	8)
	3)	6)	9)

How satisfied?

6 - very satisfied	5 - fairly satisfied	4 - a little satisfied	3 - a little dissatisfied	2 - fairly dissatisfied	1 - very dissatisfied
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**TO SCORE SSQ:**

1. Add total number of people for all 27 items. (Max. is 243).  
Divide by 27 for per item score. This gives you SSQ Number Score, or SSQN.
2. Total Satisfaction scores for all 27 items (Max. is 162).  
Divide by 27 for per item score. This gives you SSQ Satisfaction score or SSQS.
3. You can also add up total number of people that are family members and that can give the SSQ Family score.

Reference for reliability and validity of SSQ in addition to 1983 Sarason, Levine, Basham, and Sarason article:

Heitzmann, C.A. and Kaplan, R.M. (1988). Assessment of methods for measuring social support.  
Health Psychology, 1(1), 75-109.

Shortened version of the Life Event Questionnaire (LEQ) modified from 39 items Newcomb, Juba & Bentler, 1981 to for use with Australian adolescents

Event Effect of Event on Your Life	Type of Effect		No effect	Some effect	Moderate effect	Great effect
	Good	Bad				
			0	1	2	3
<b>Family</b>						
1. Parents divorced						
2. Family had money problems						
3. Parents argued or fought						
4. Parents remarried						
5. Parent abused alcohol or other substances						
6. Gain of a new family member (through birth, adoption, relative moving in, etc)						
<b>Accident/ Illness</b>						
7. Family accident or serious illness						
8. Death in the family						
9. Serious accident or illness (self)						
10. Death of a close friend (accidental or purposeful)						
11. Death of acquaintance						
<b>Deviance</b>						
12. Got in trouble with the law						
13. Got in trouble at school						
14. Being robbed or a victim of crime						
<b>Relocation/ change</b>						
15. Changed schools						
16. Family moved						
15. Began part time work						
<b>Distress</b>						
17. Poor grades in school						
18. Ran away from home						
19. Gained a lot of weight						
20. Got bullied						
21. Significant friendship ended						
22. Boyfriend/girlfriend ended relationship						



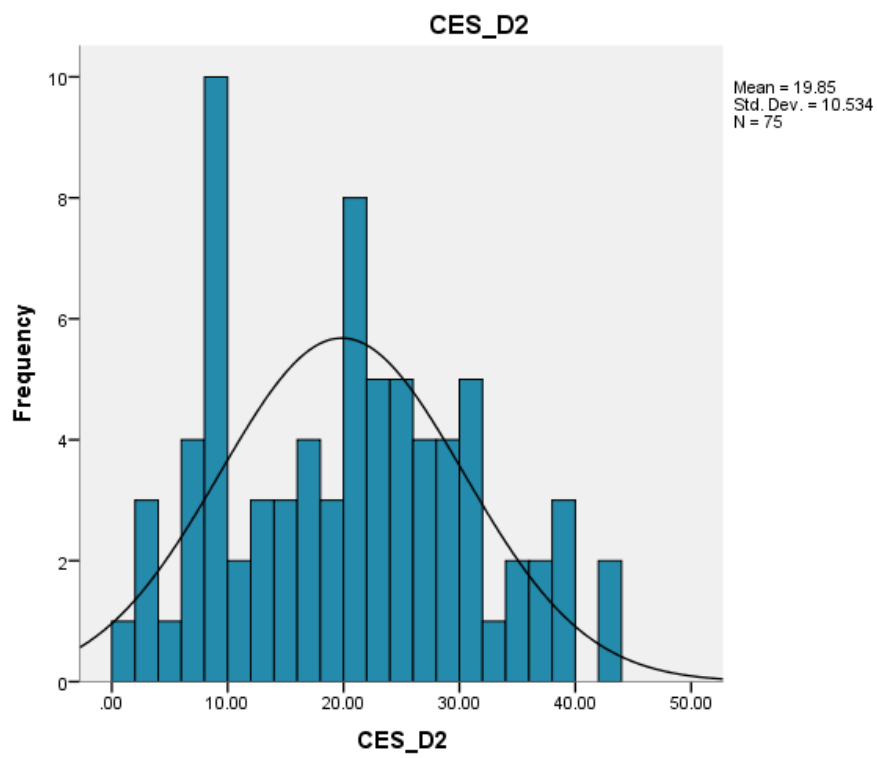
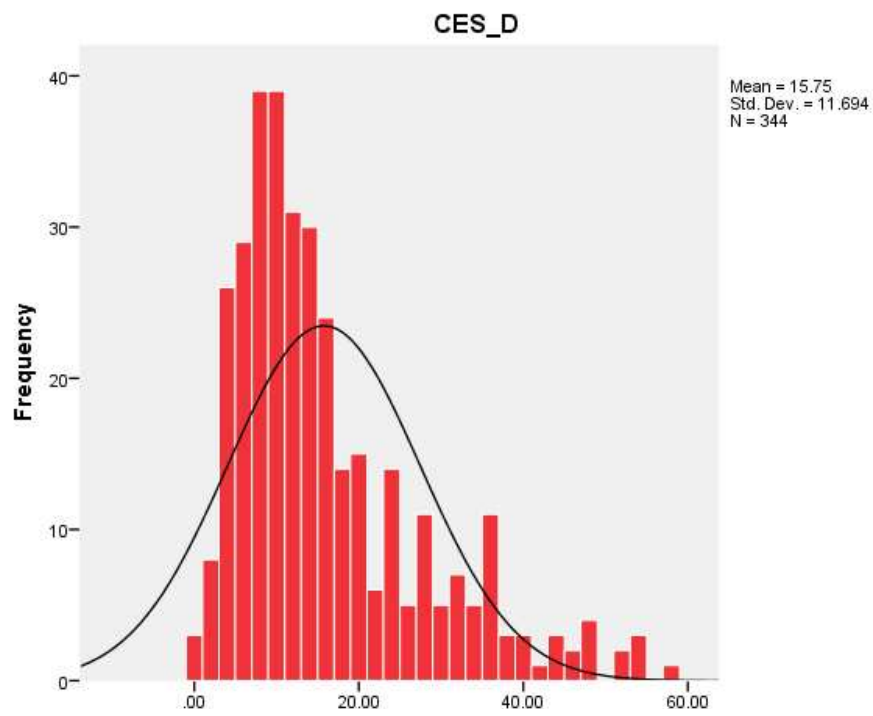
## **APPENDIX C**

### **Bivariate Correlations and Histograms of the Variables Time One and Time Two**

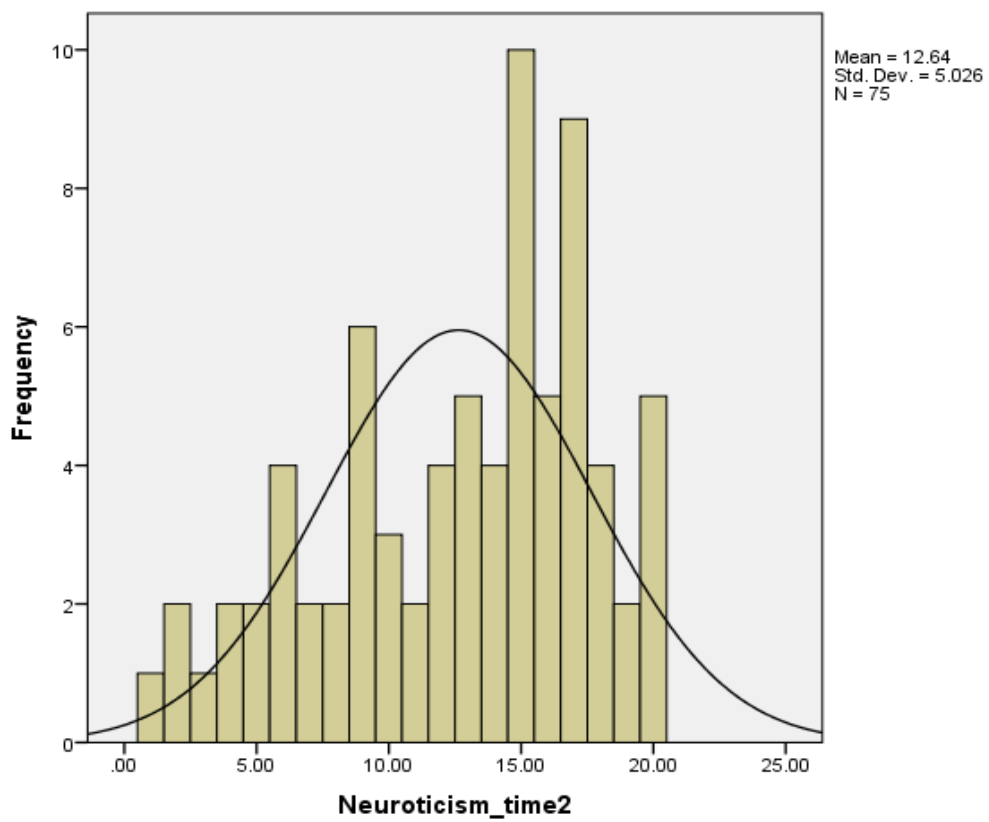
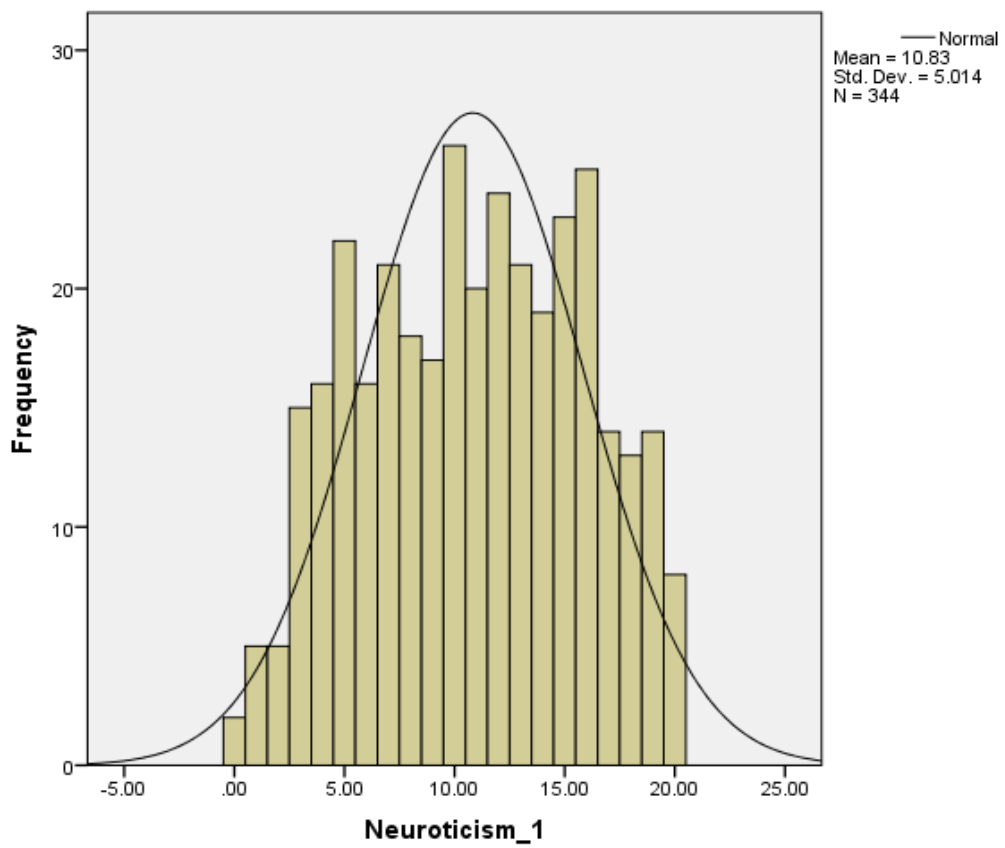




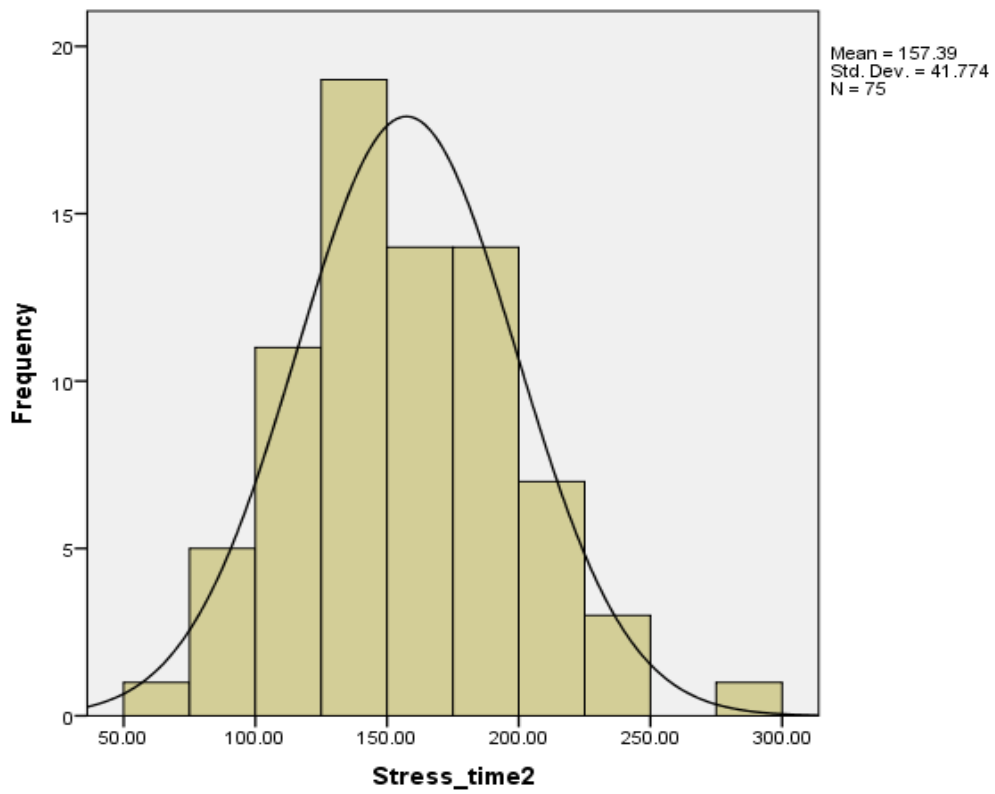
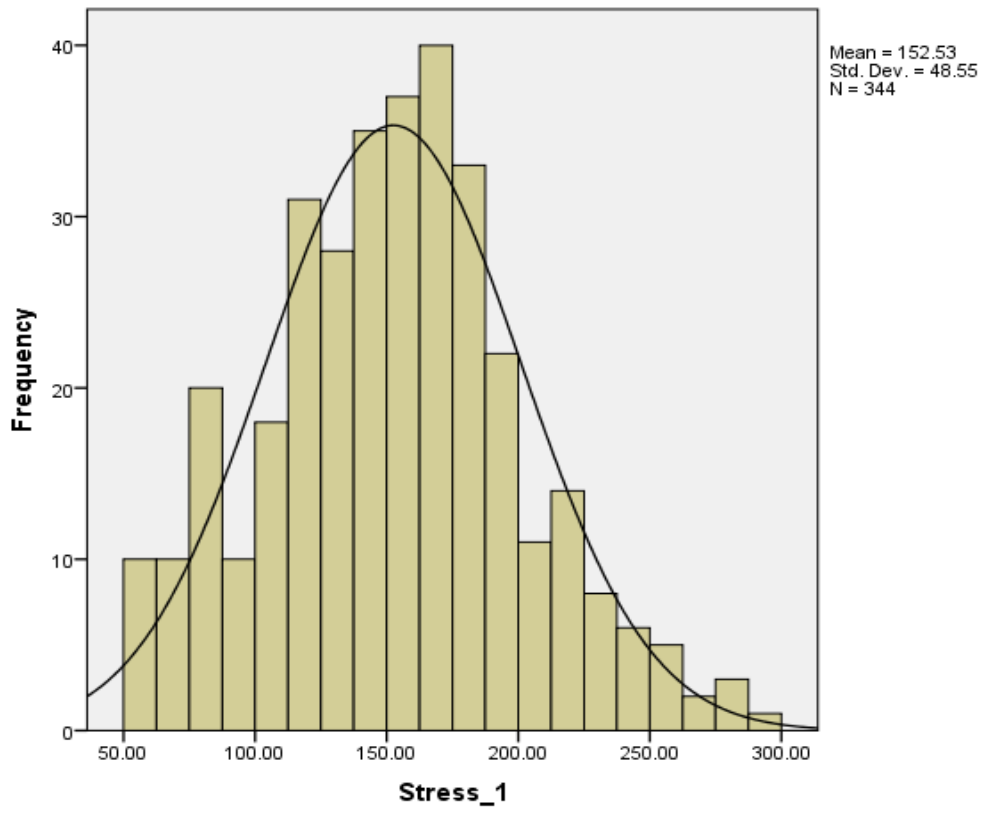
# Histograms of Depression Scores Time 1 and 2



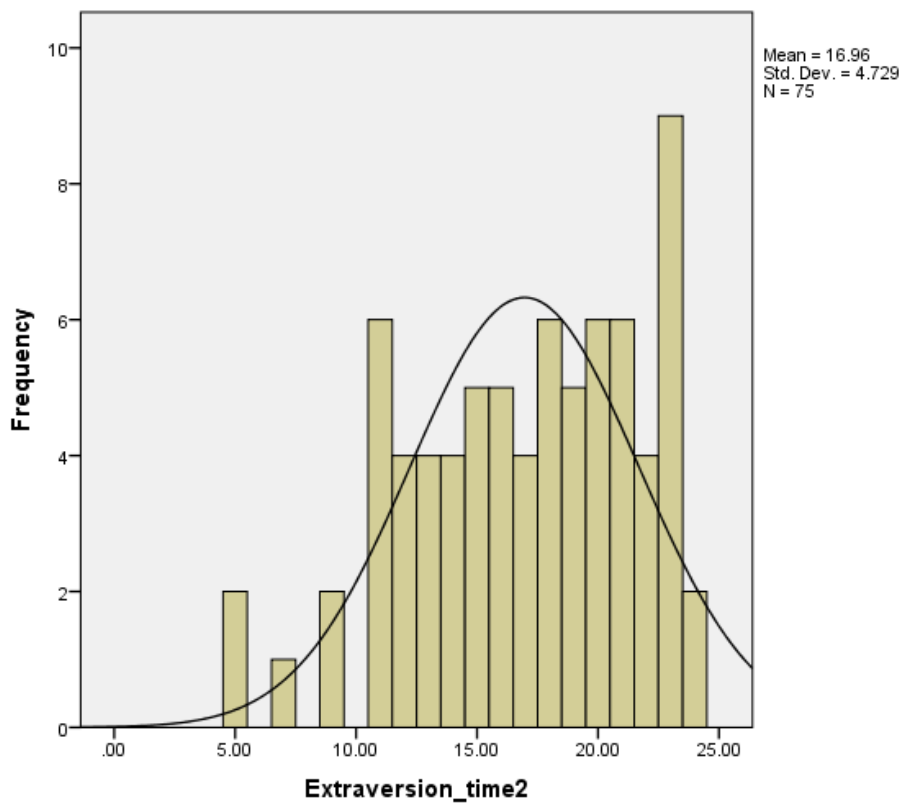
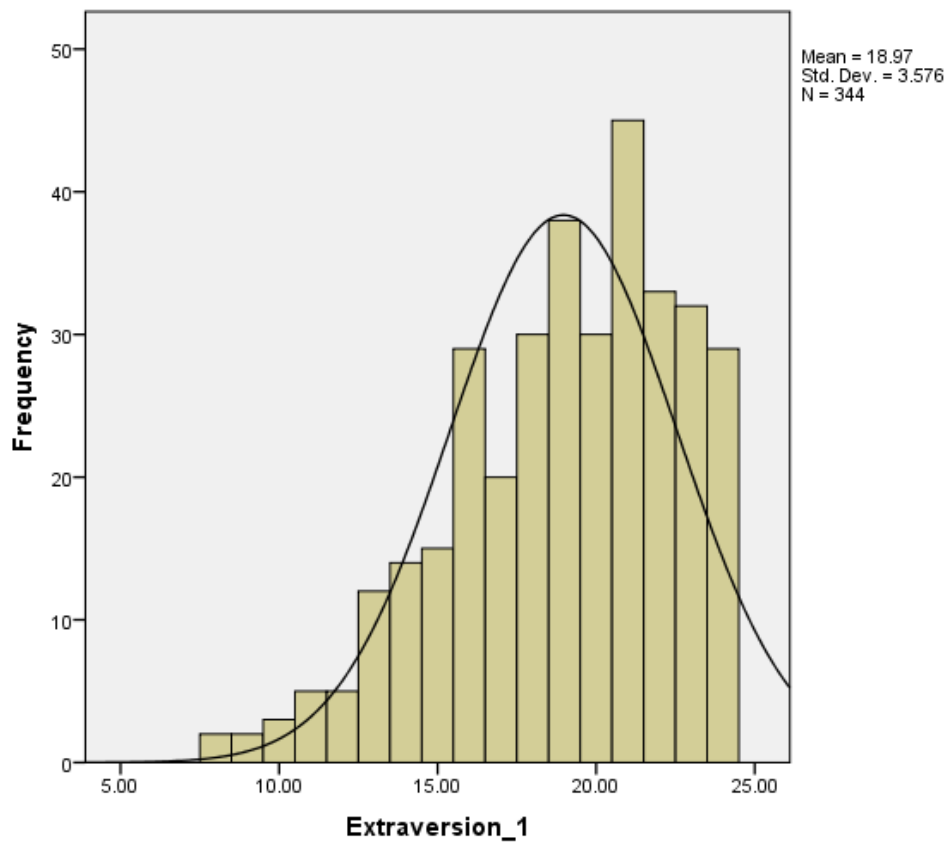
Histogram of Neuroticism Time 1 and 2



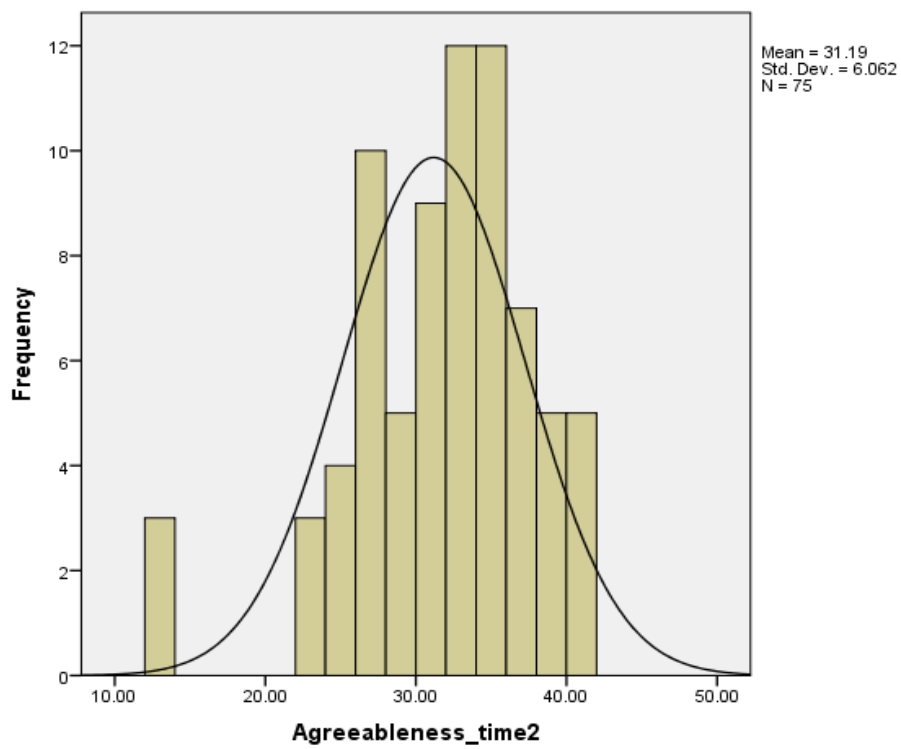
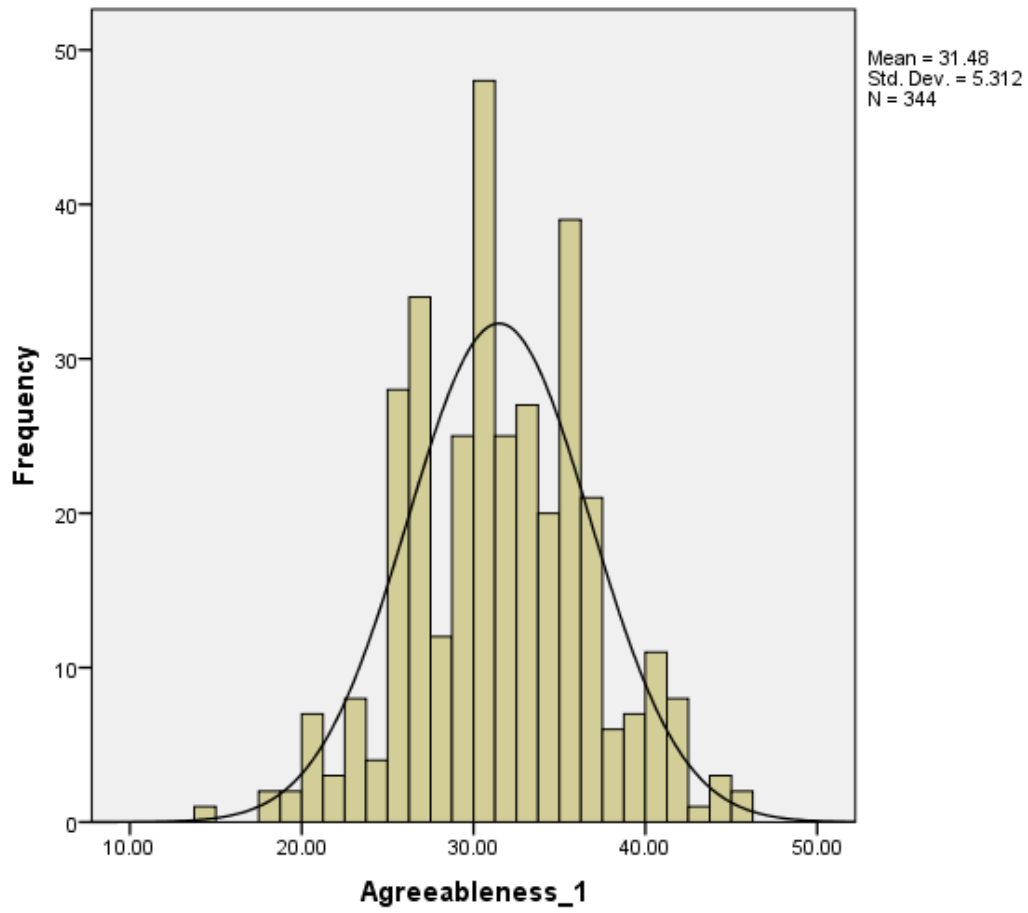
# Histogram of Stress Scores Time 1 and 2



Histogram of Extraversion scores 1 and 2

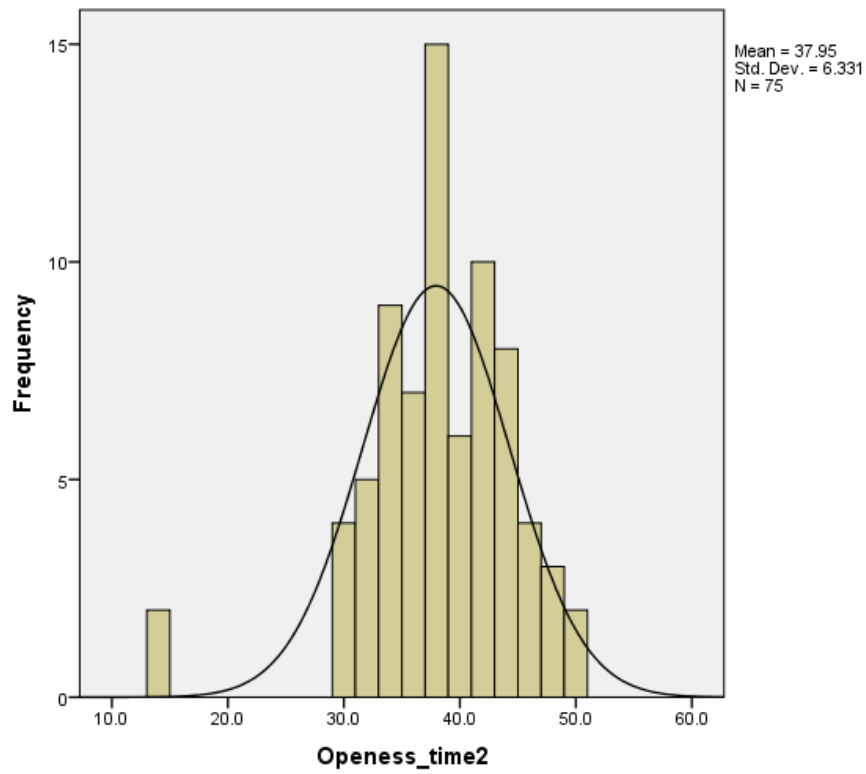
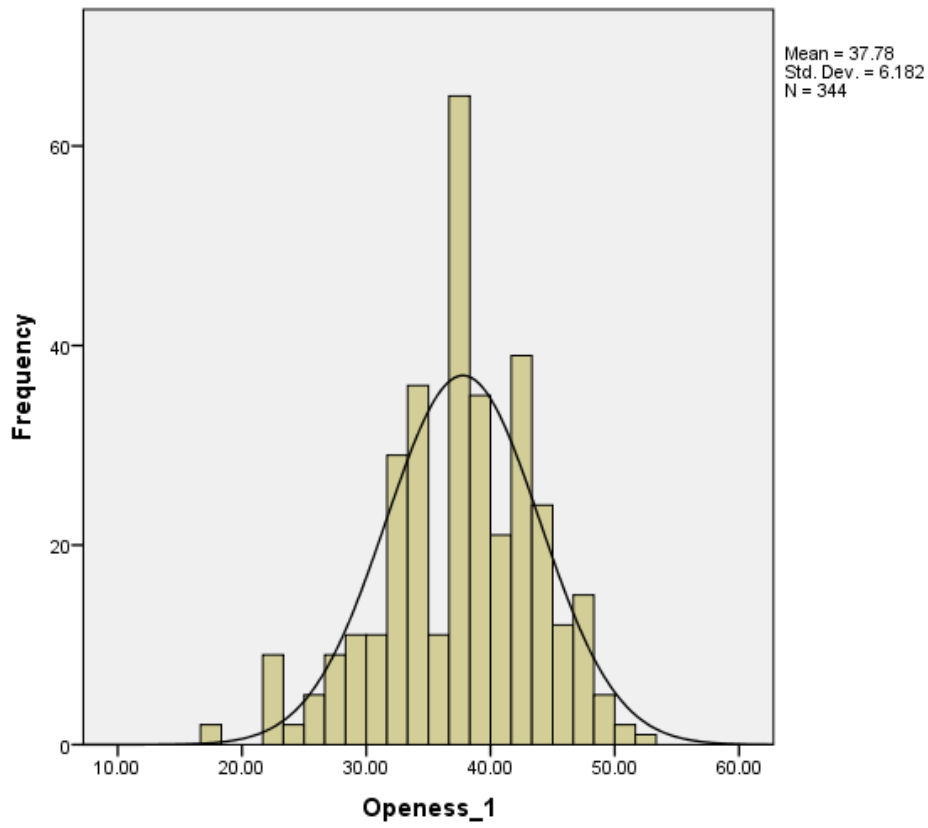


# Histogram of Agreeableness Time 1 and 2

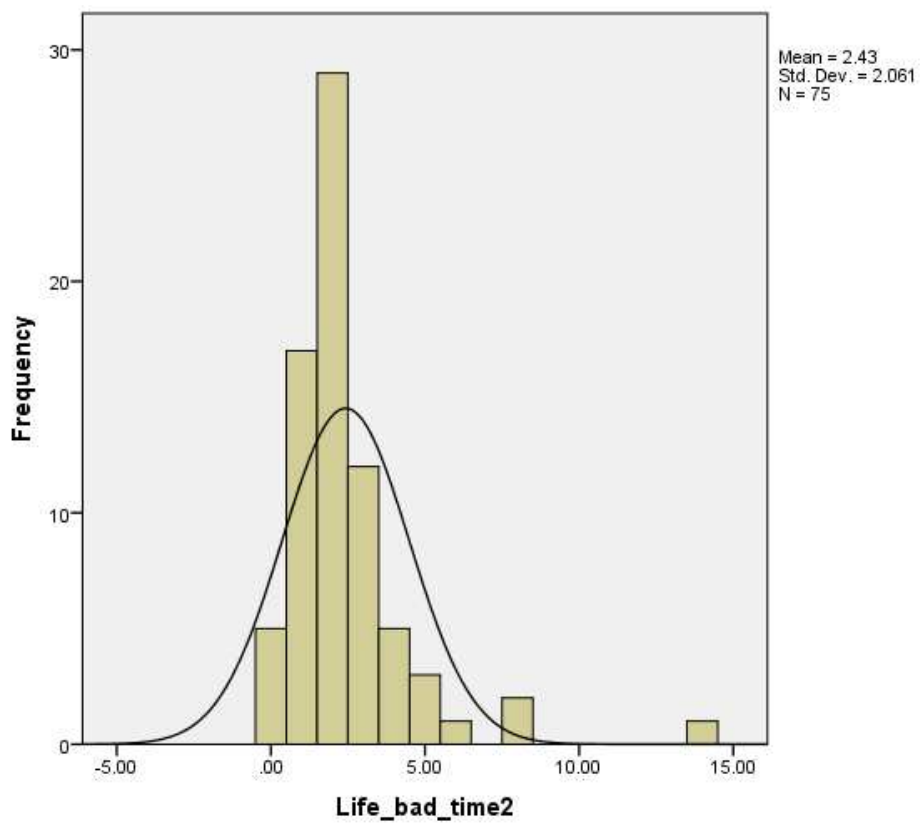
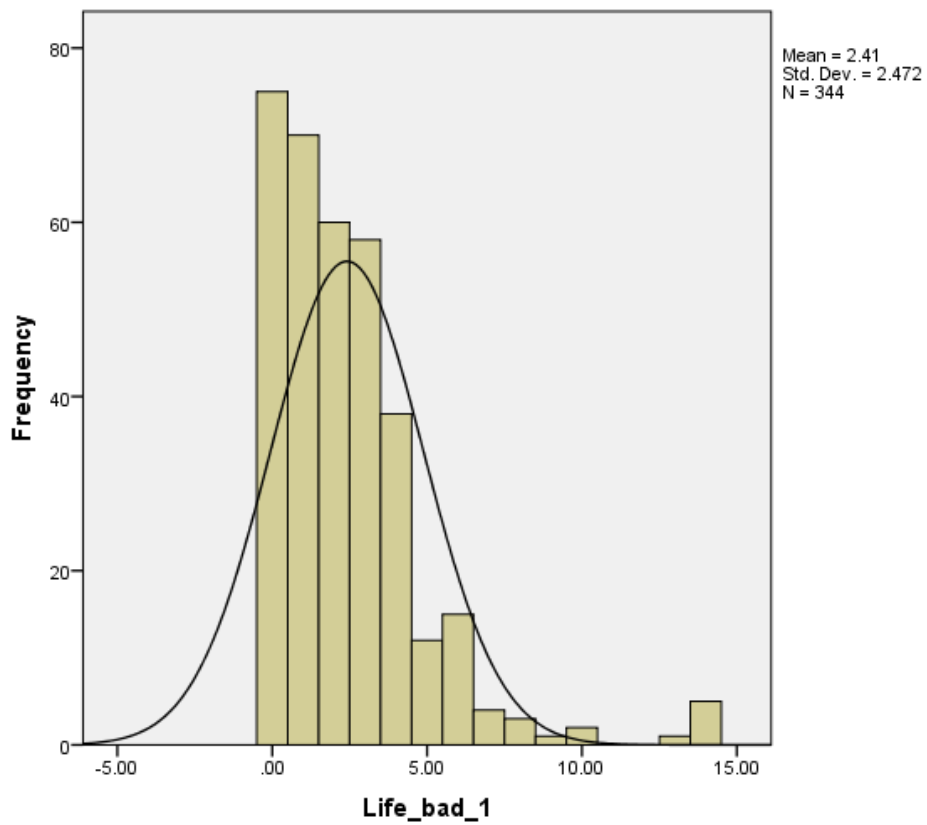




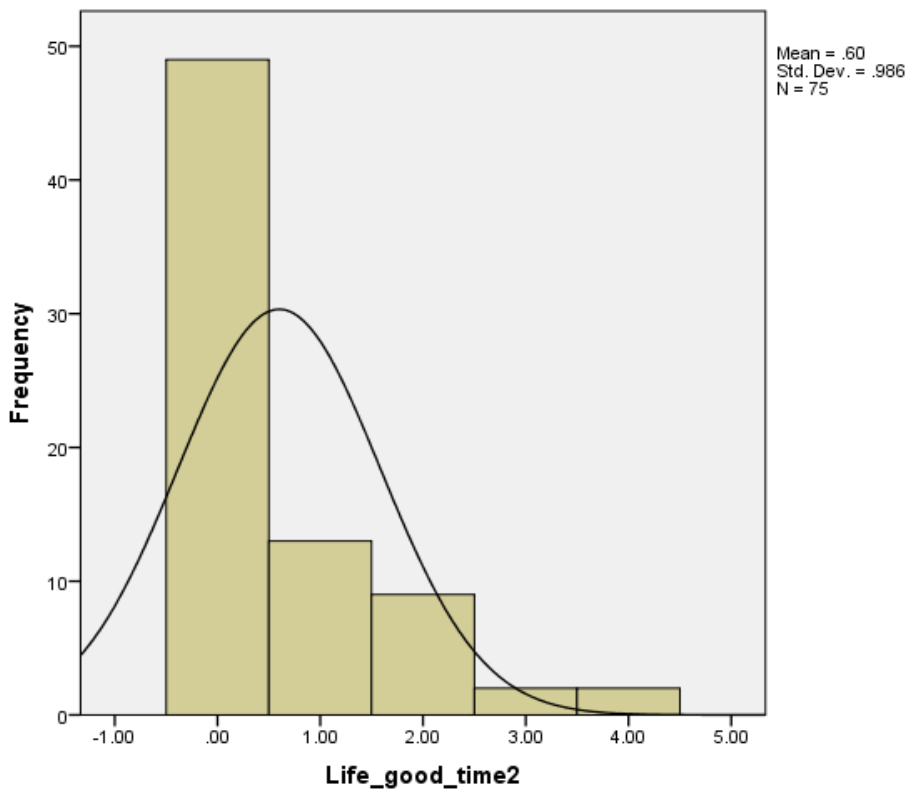
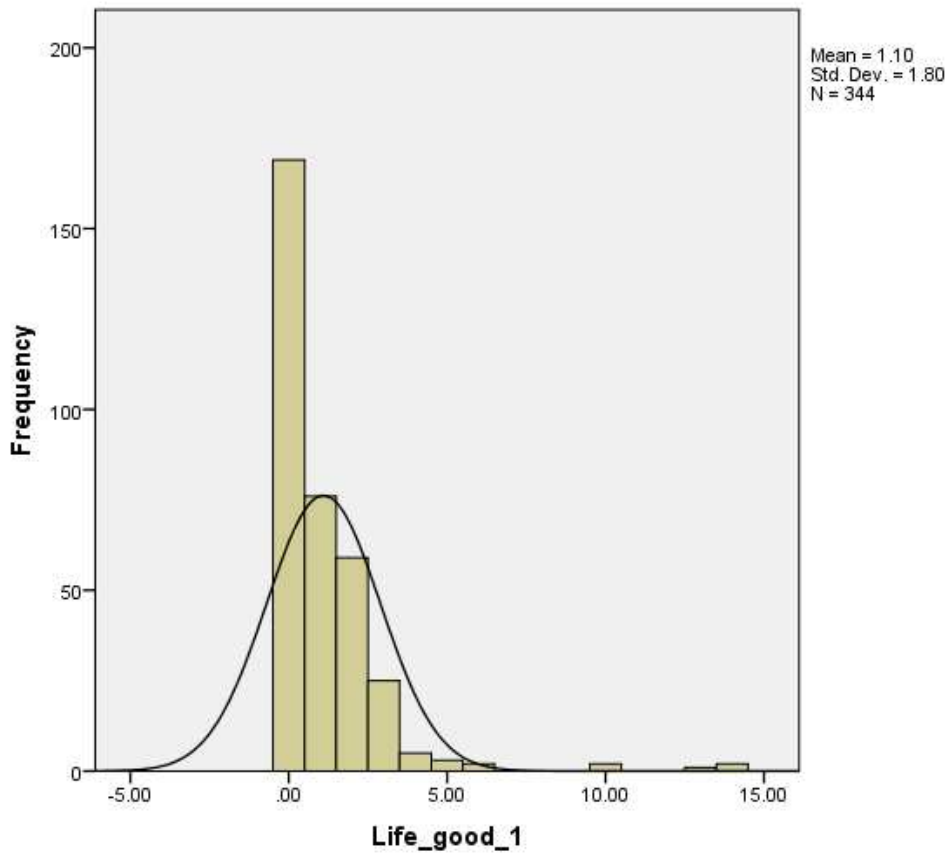
# Histogram of Openness Time 1 and 2



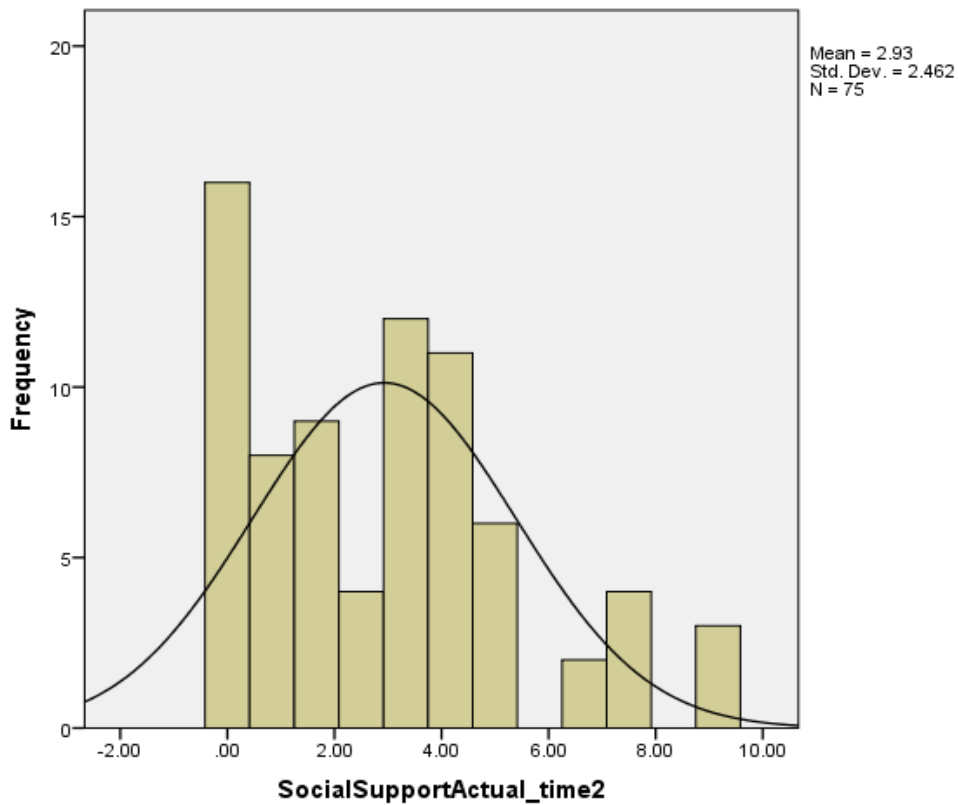
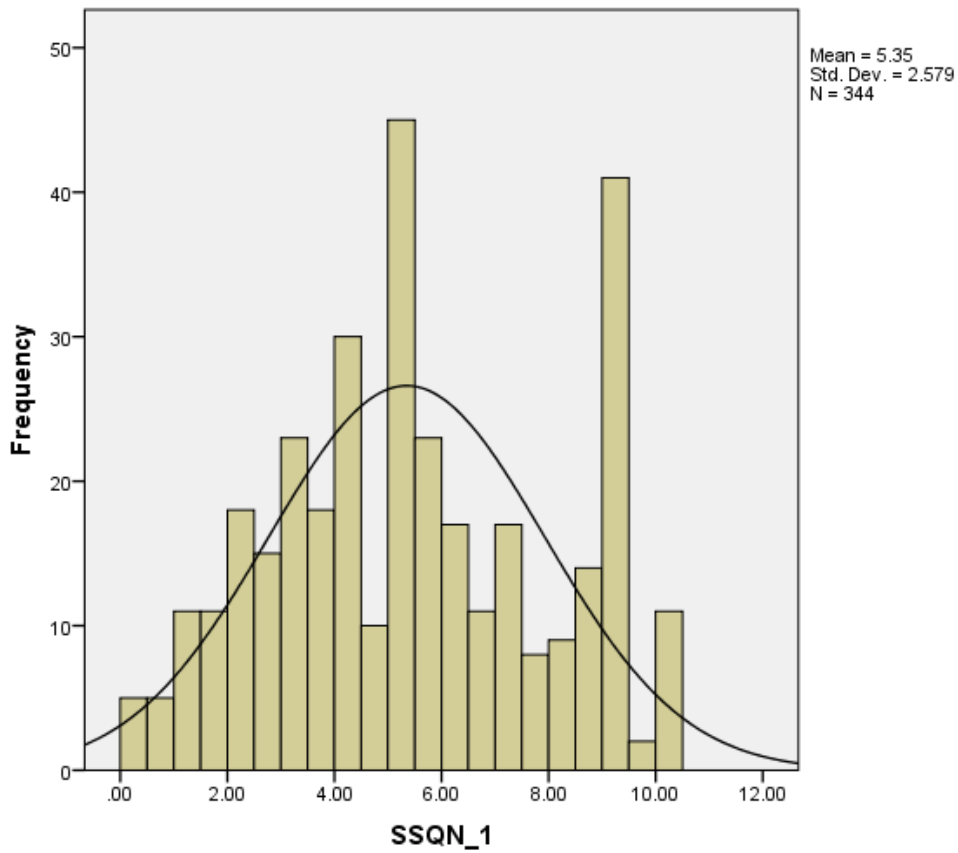
## Histogram of Life Bad Time 1 and 2



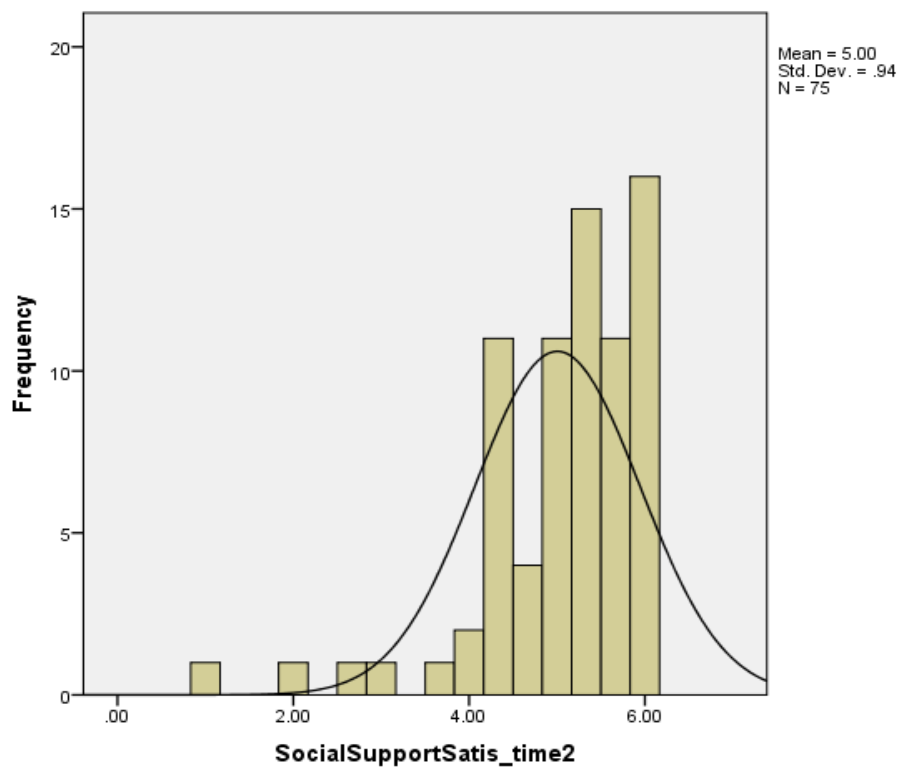
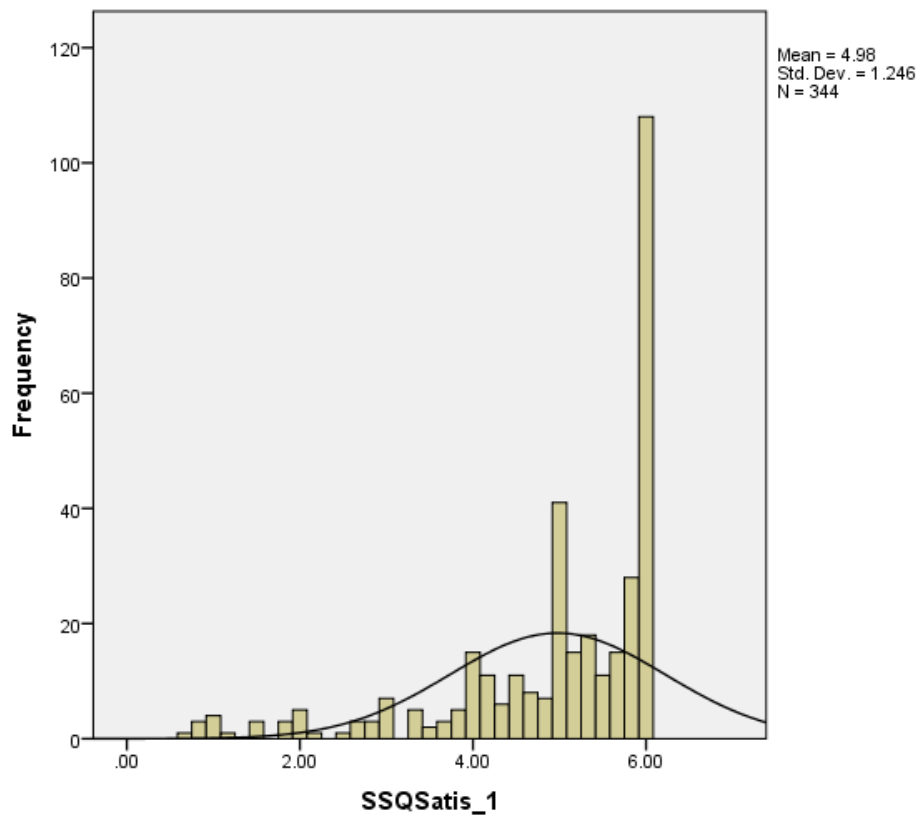
Histogram of Life Good 1 and 2



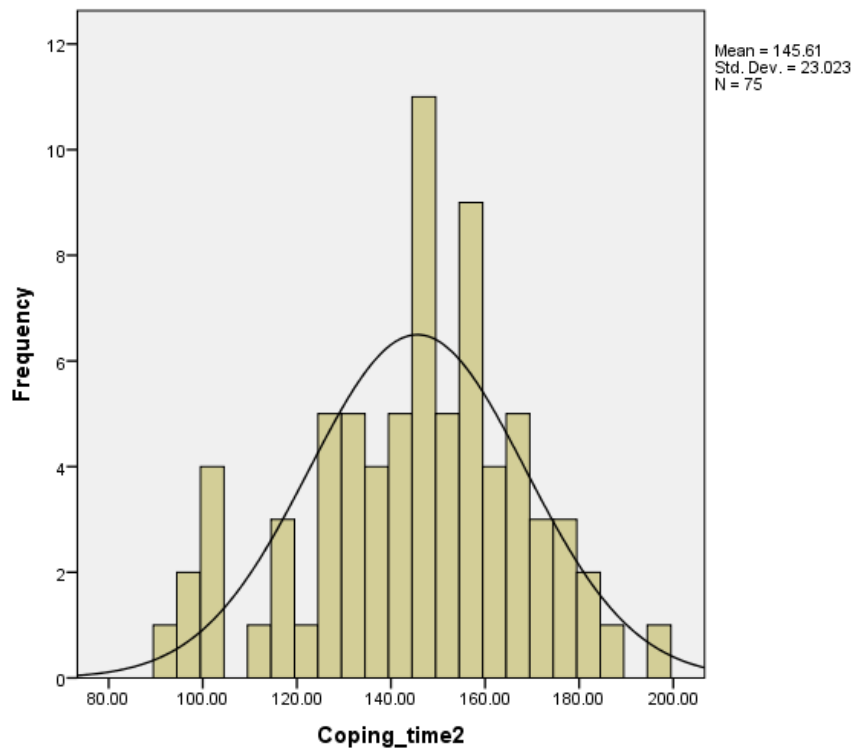
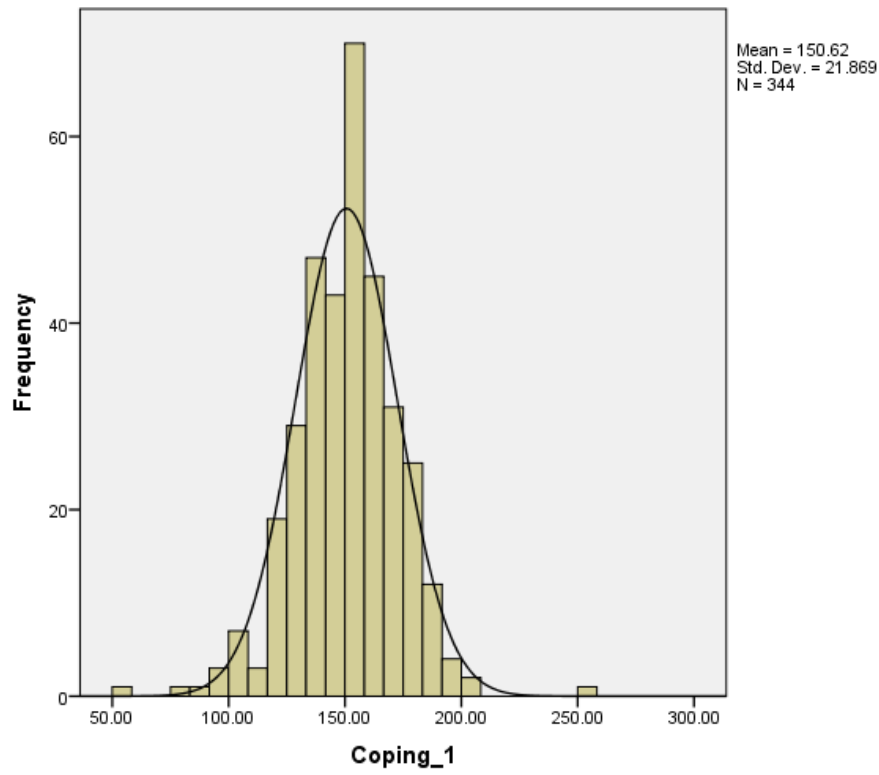
Histogram of Social Support Actual Time 1 and 2



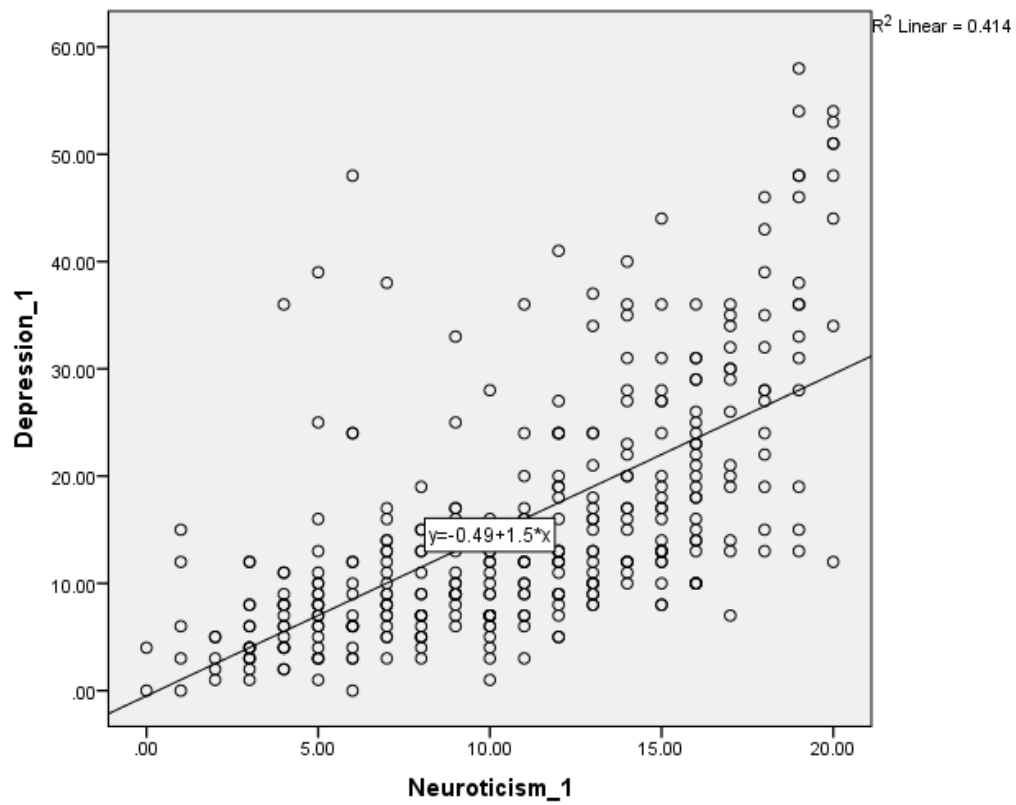
## Histograms of Perceived Social Support Time 1 and 2



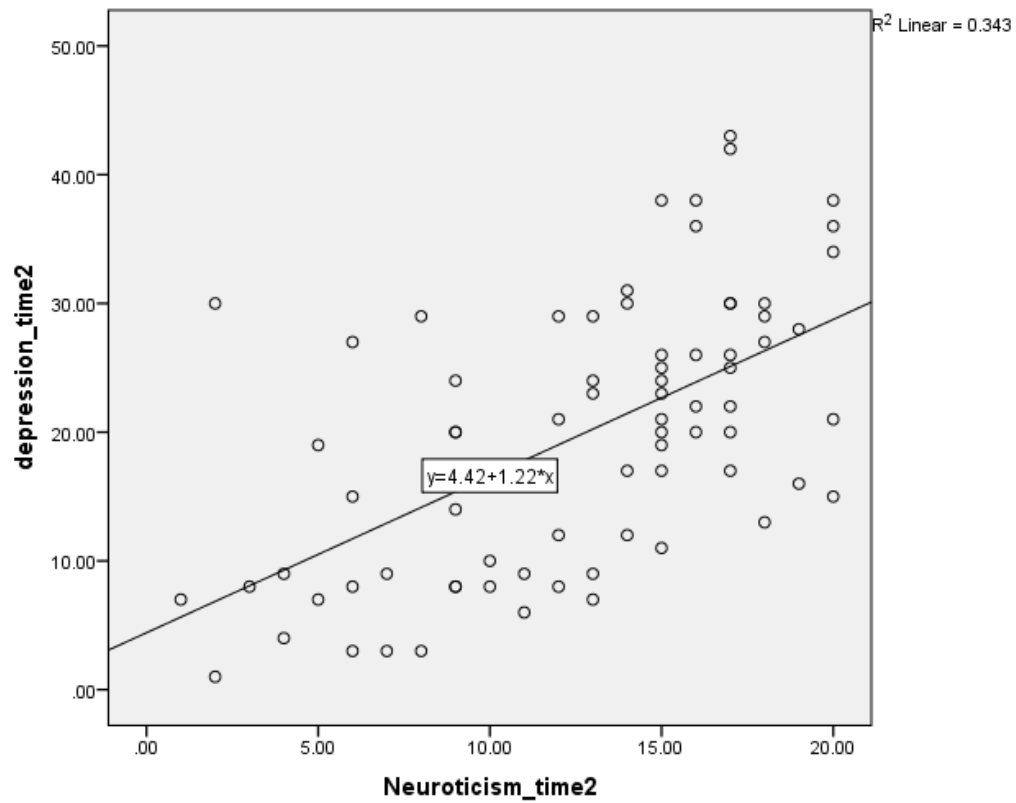
# Histogram of Coping Time 1 and 2



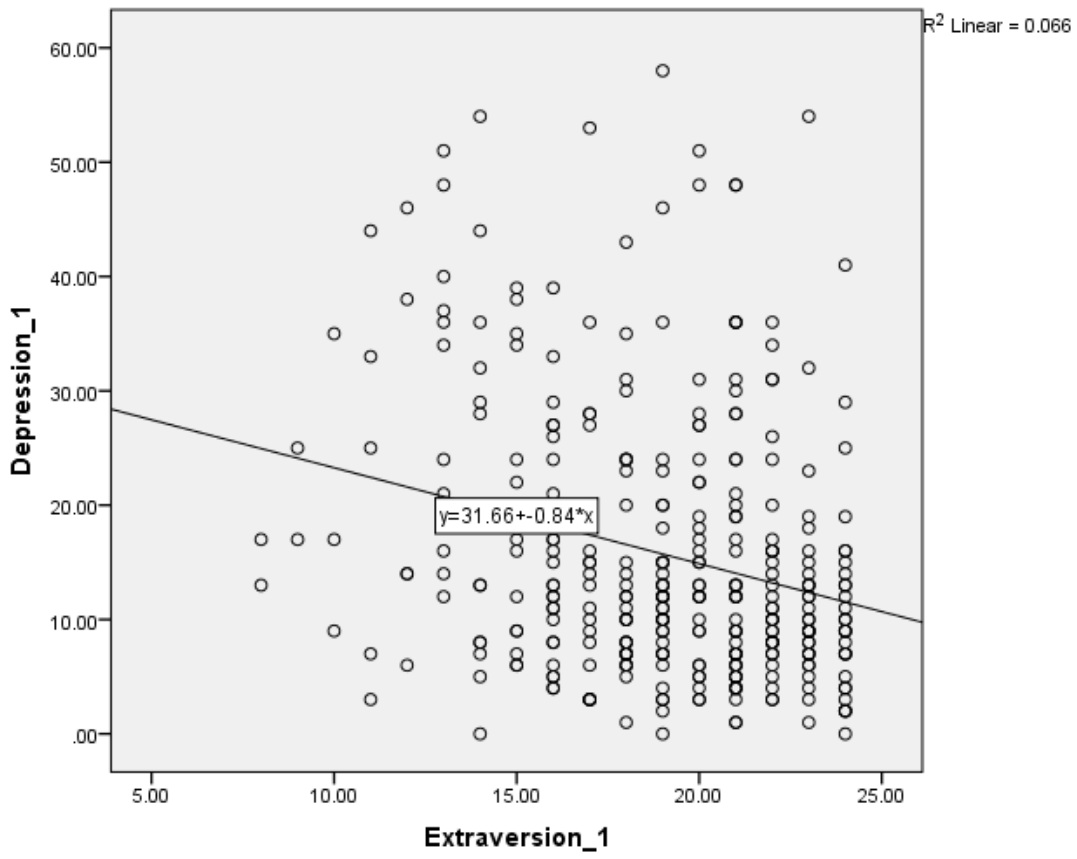
Scatter plot of Neuroticism and Depression Time 1



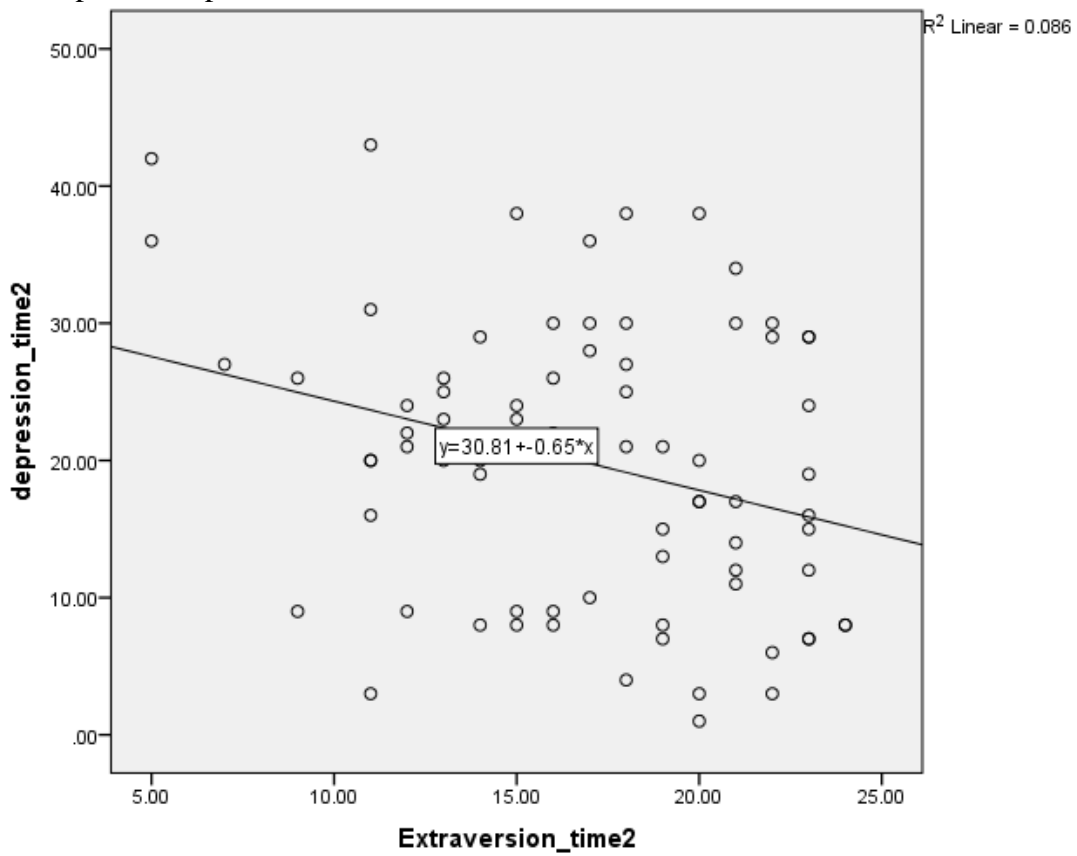
Scatter plot of Neuroticism and Depression Time 2



Scatterplot of Depression and Extraversion Time 1

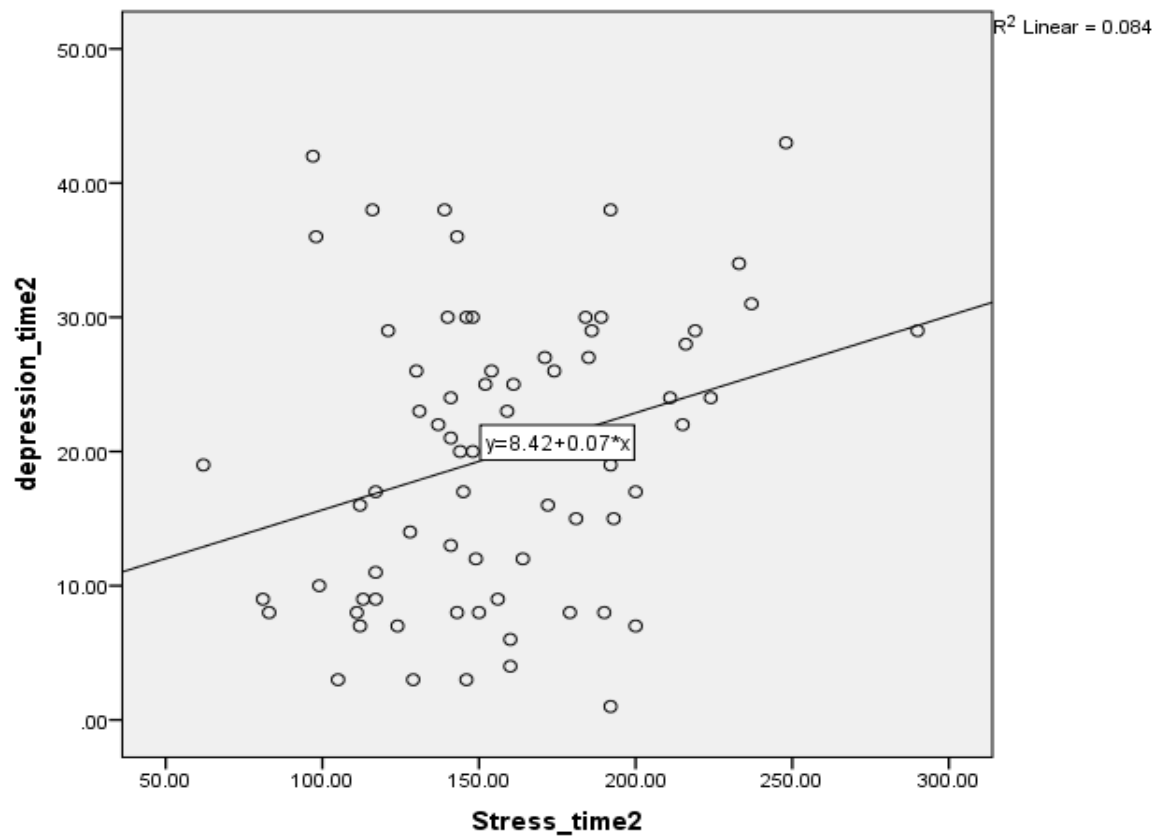
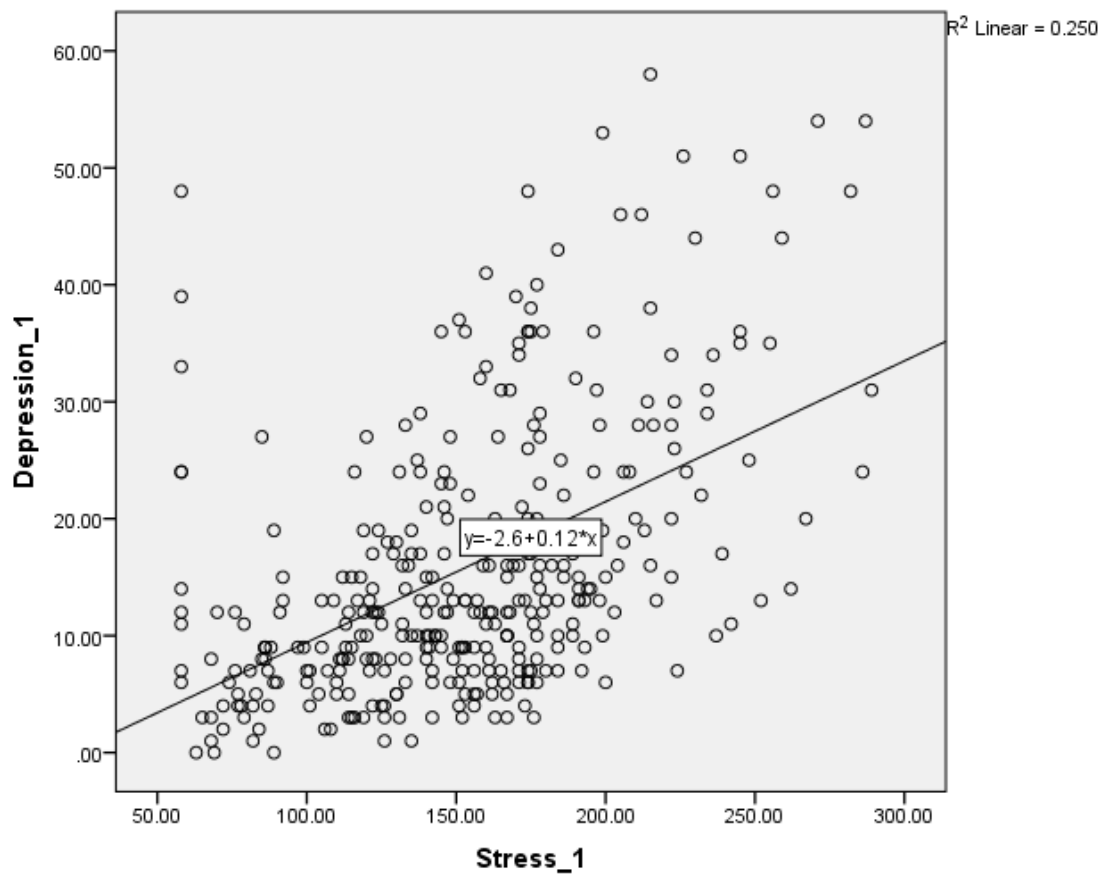


Scatter plot of Depression and Extraversion Time 2

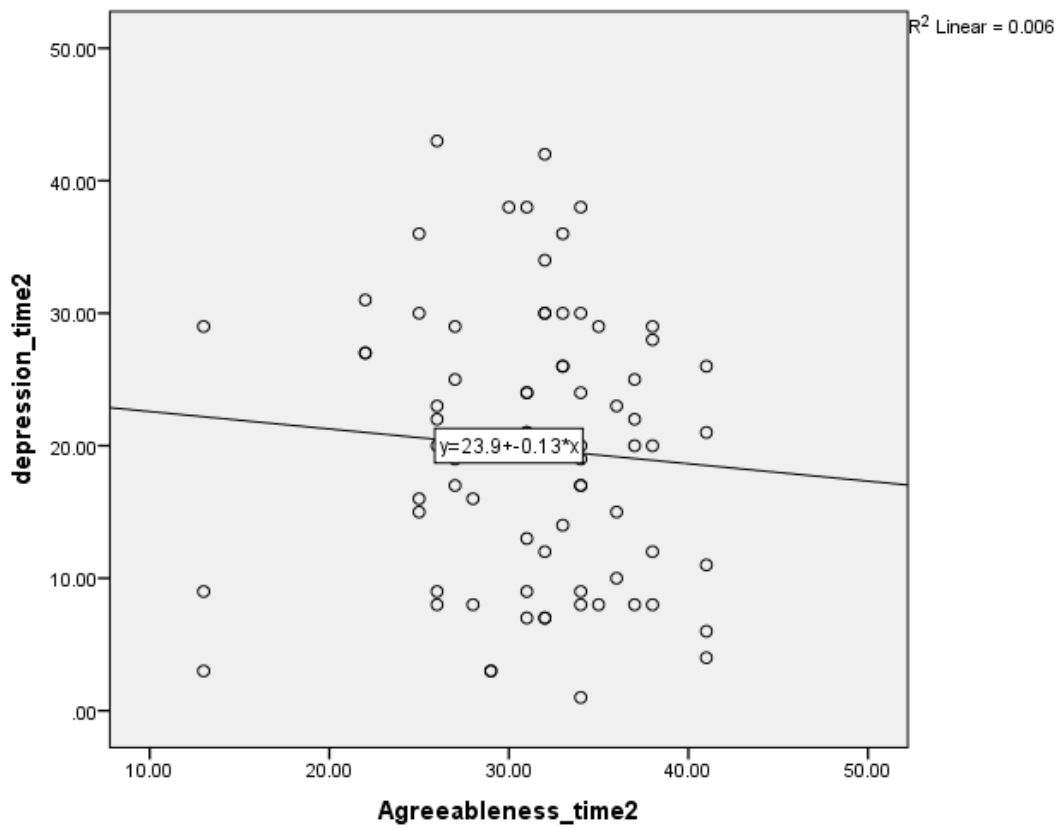
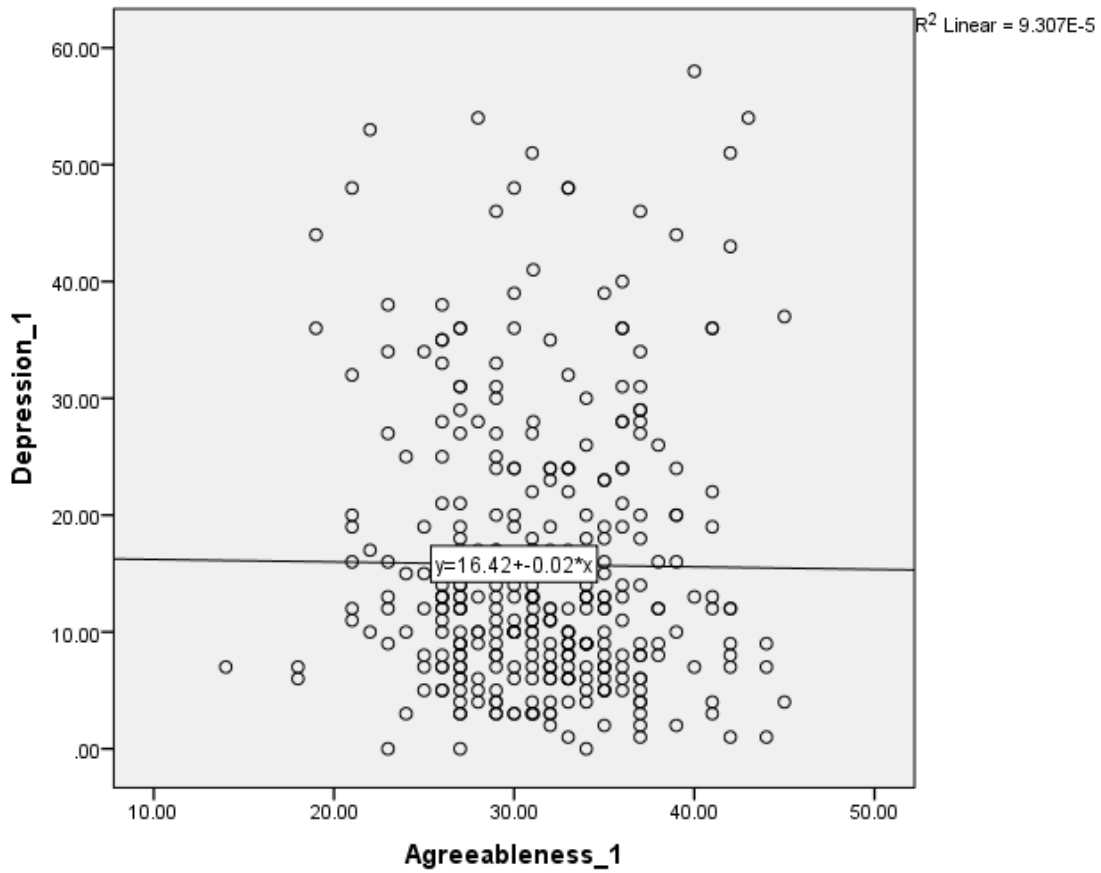




Scatter plot of Depression and Stress Time 1

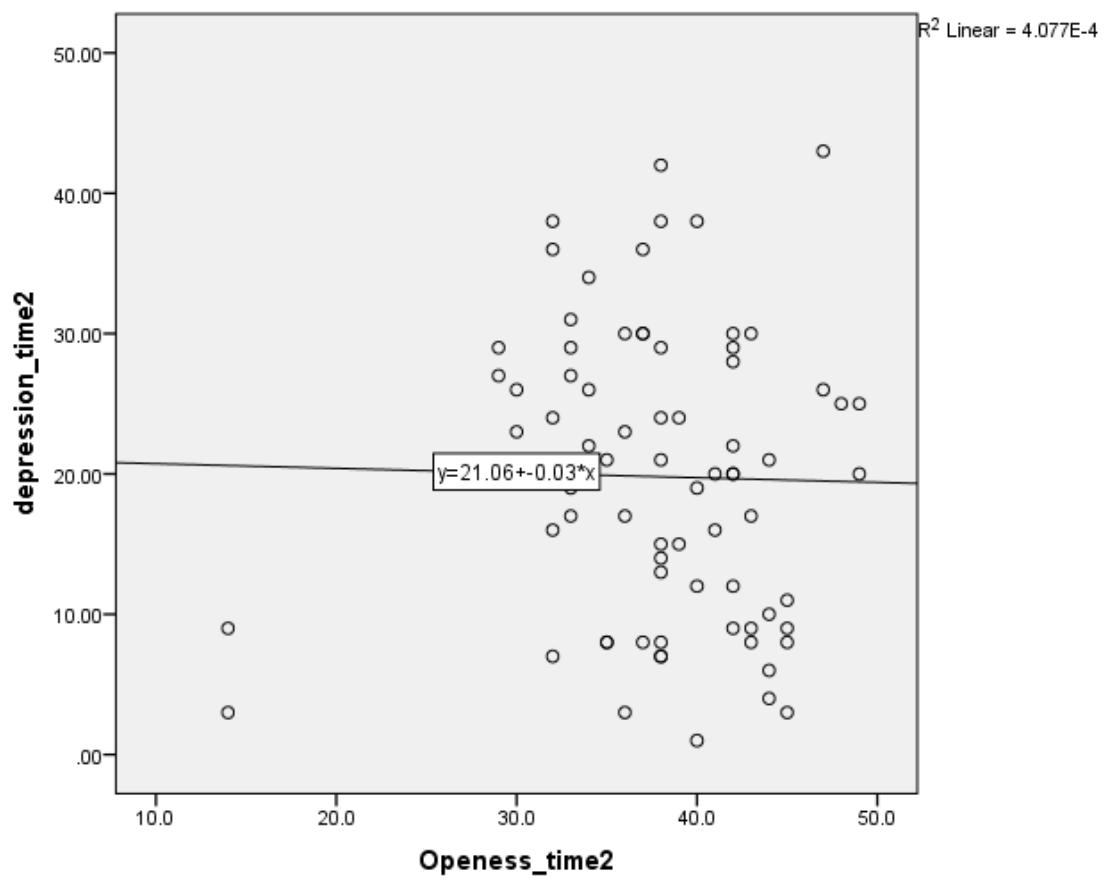
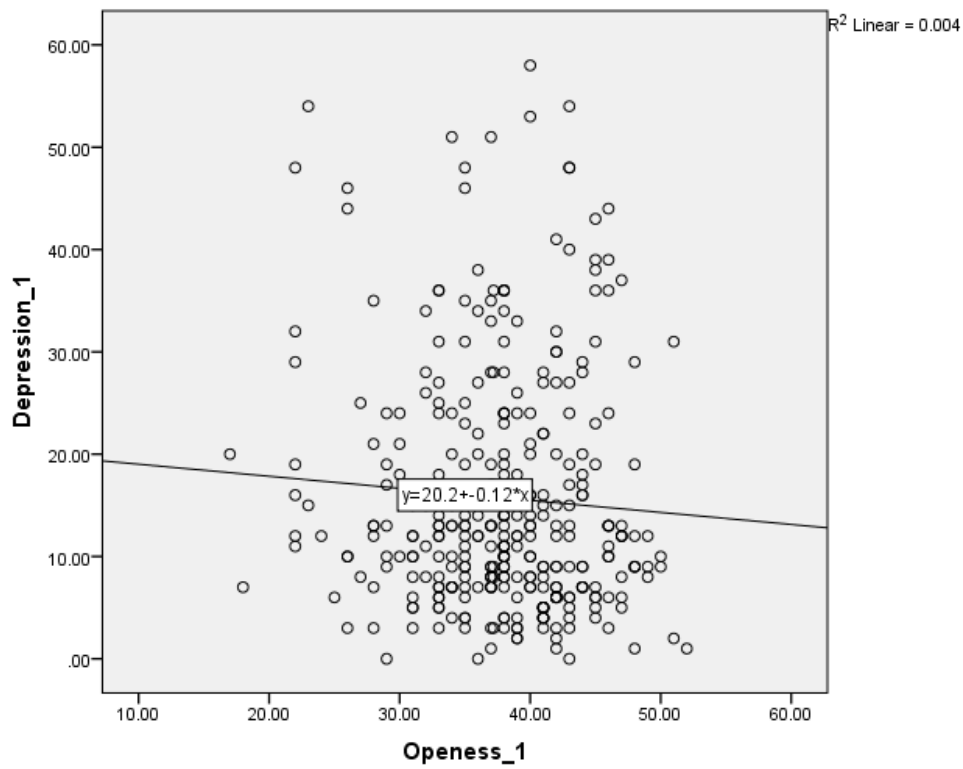


Scatterplot of Depression and Agreeableness Time 1 and 2

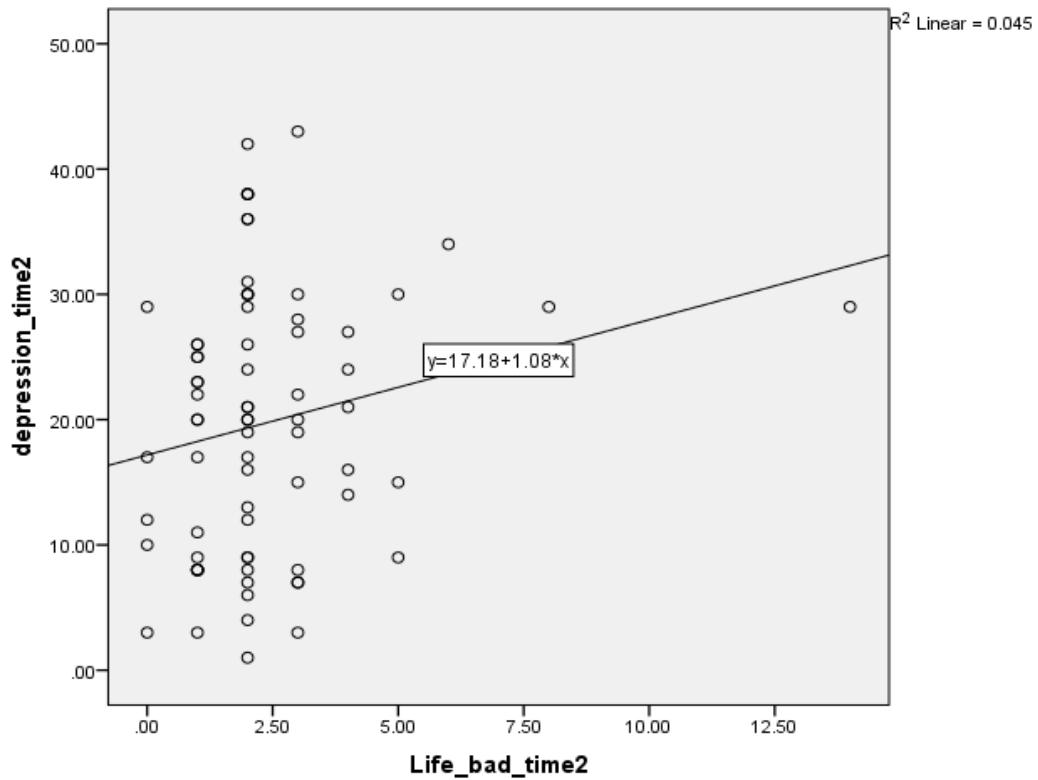
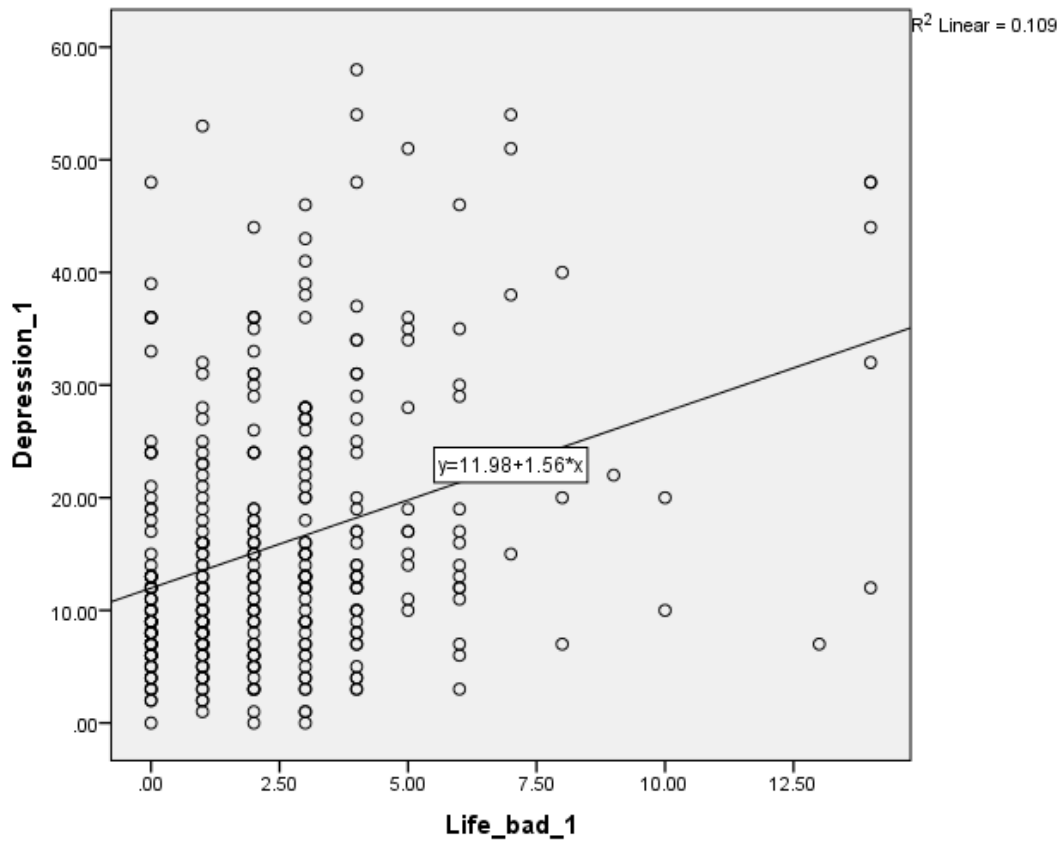


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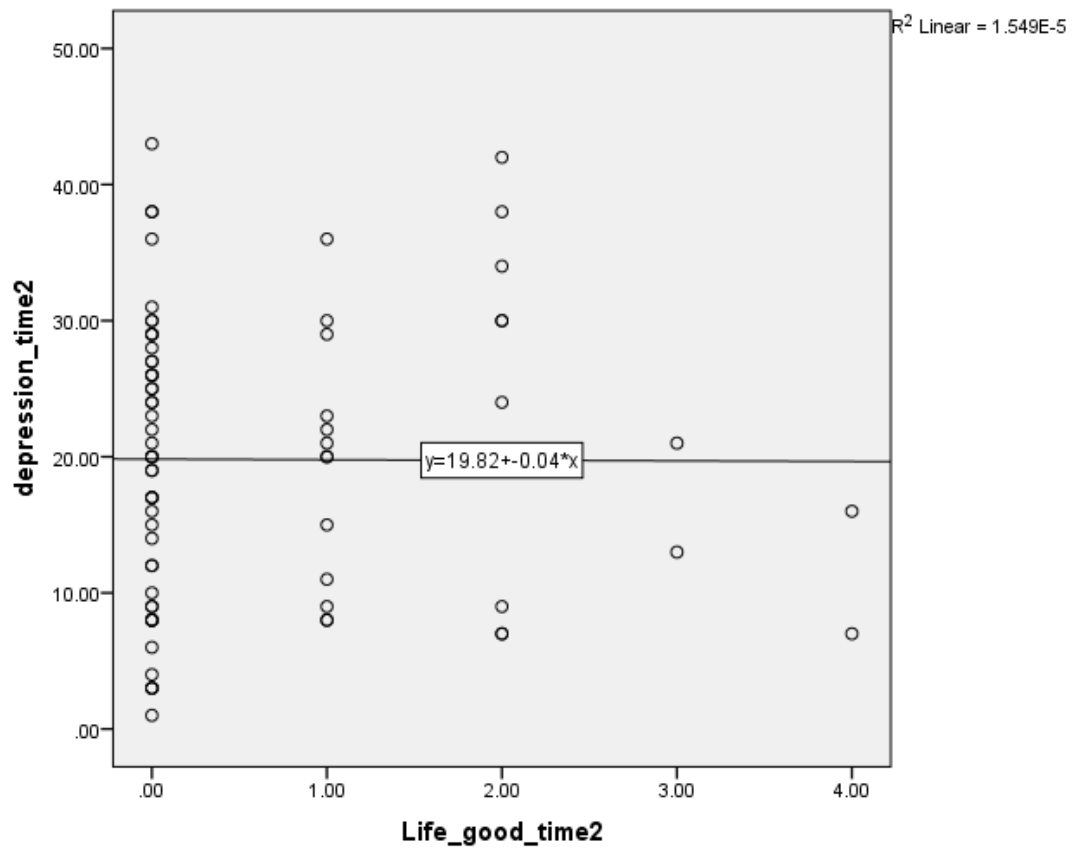
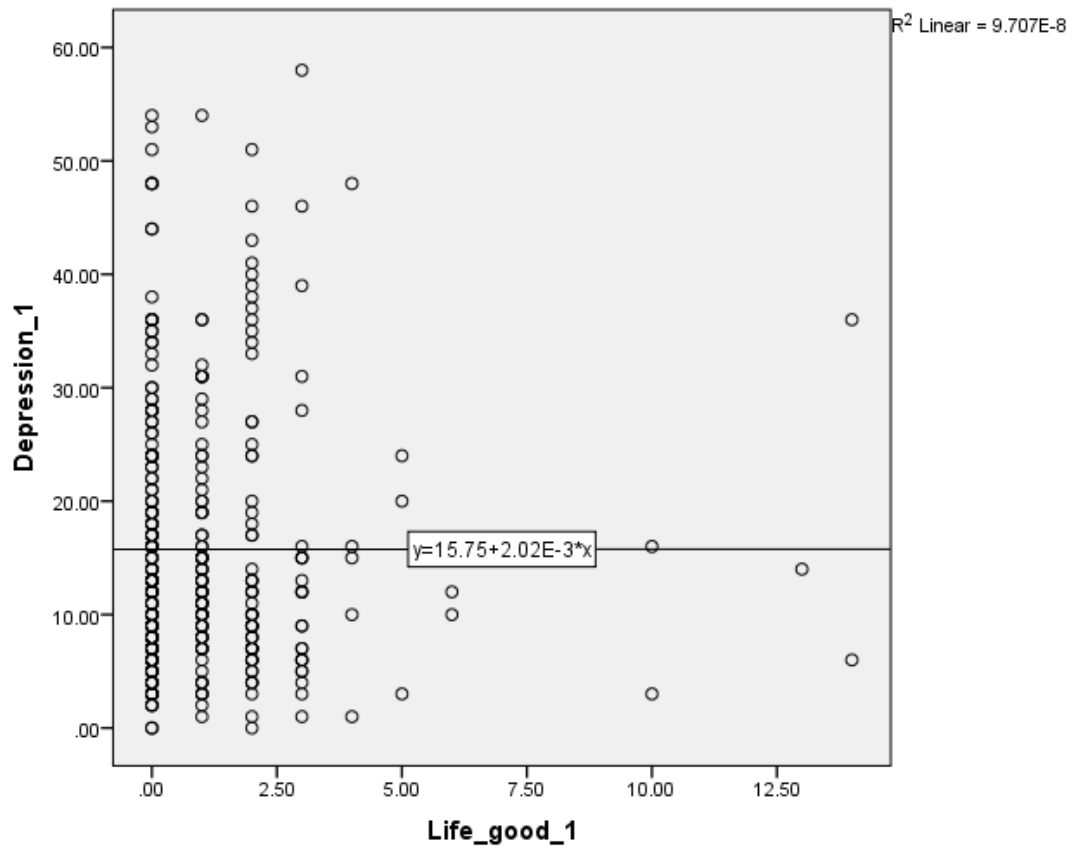
Scatterplot of Depression and Openness Time 1 and 2



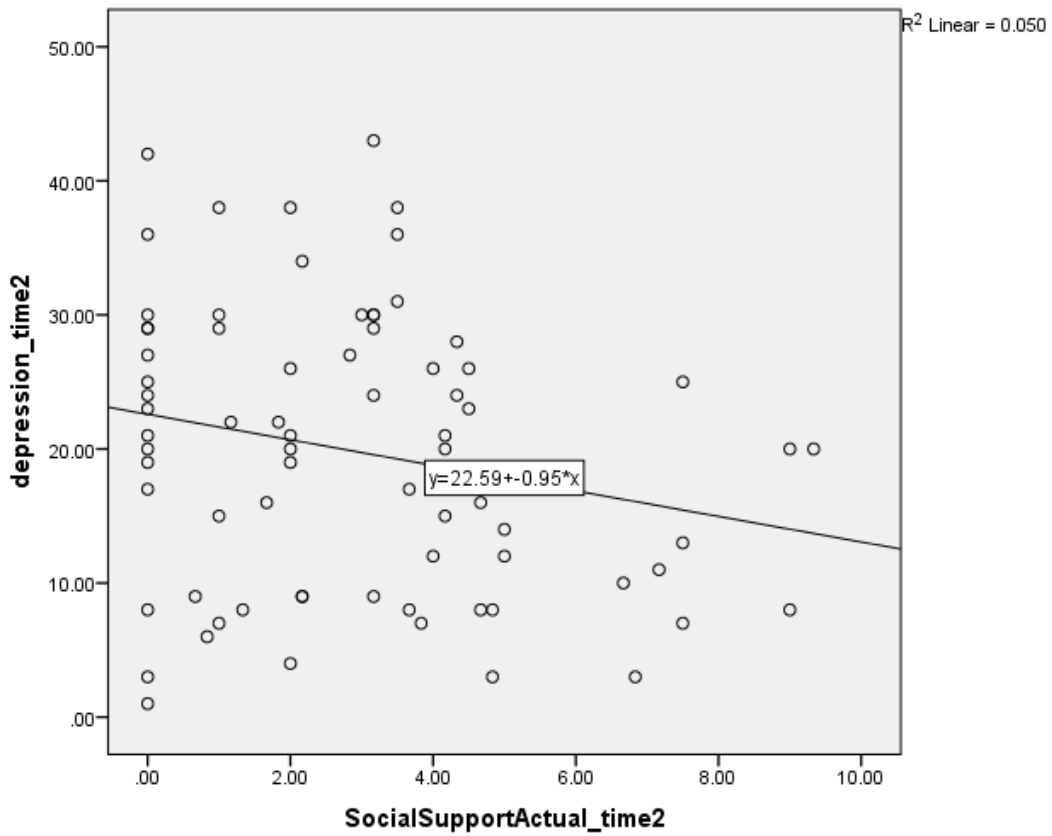
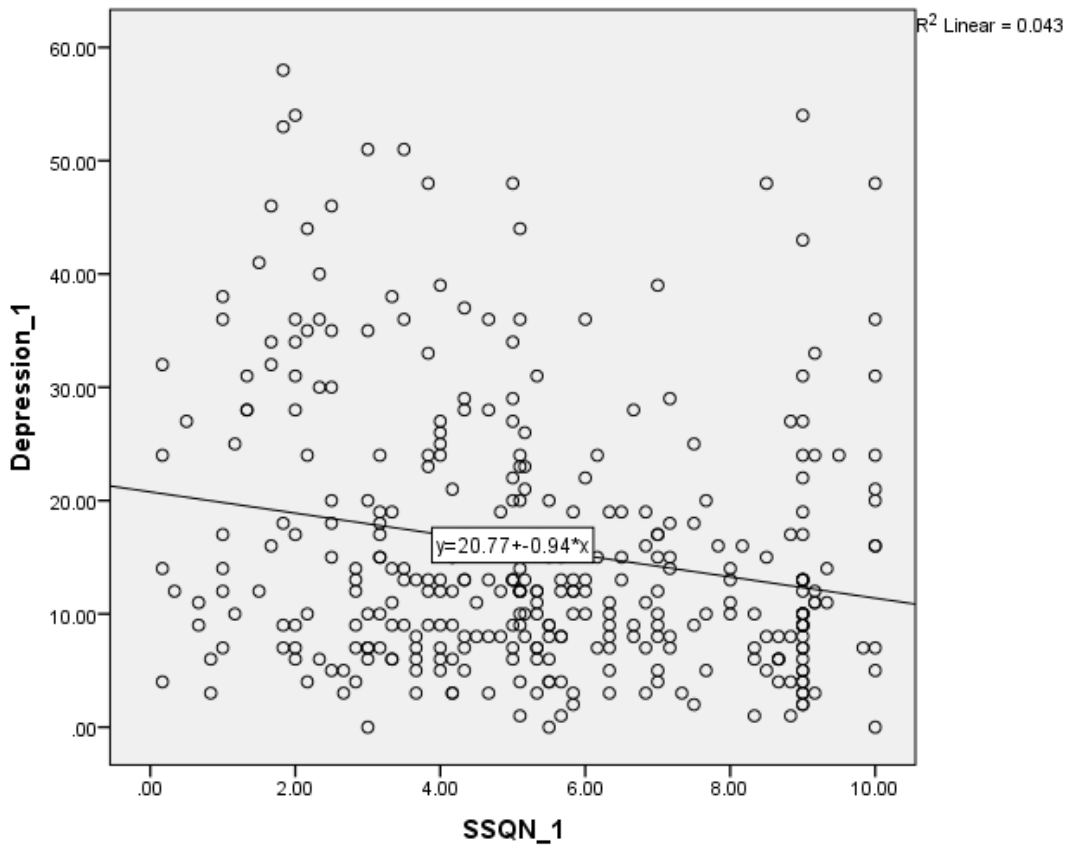
Scatterplot of Depression and Bad Life Events Time 1 and 2



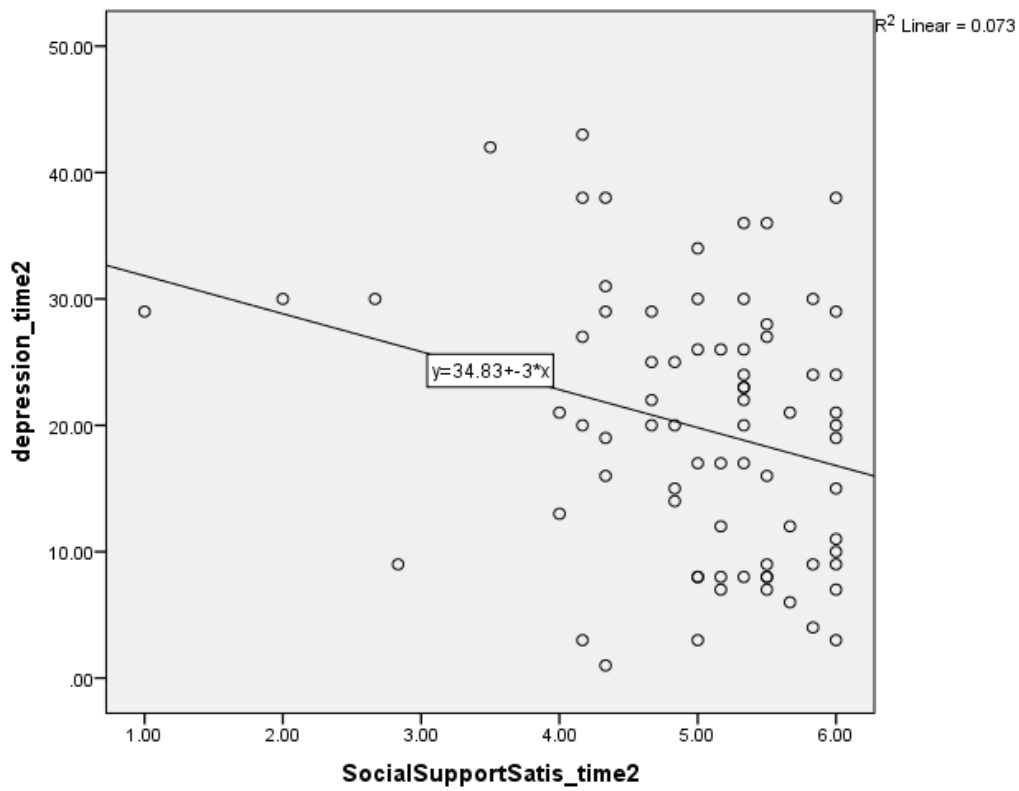
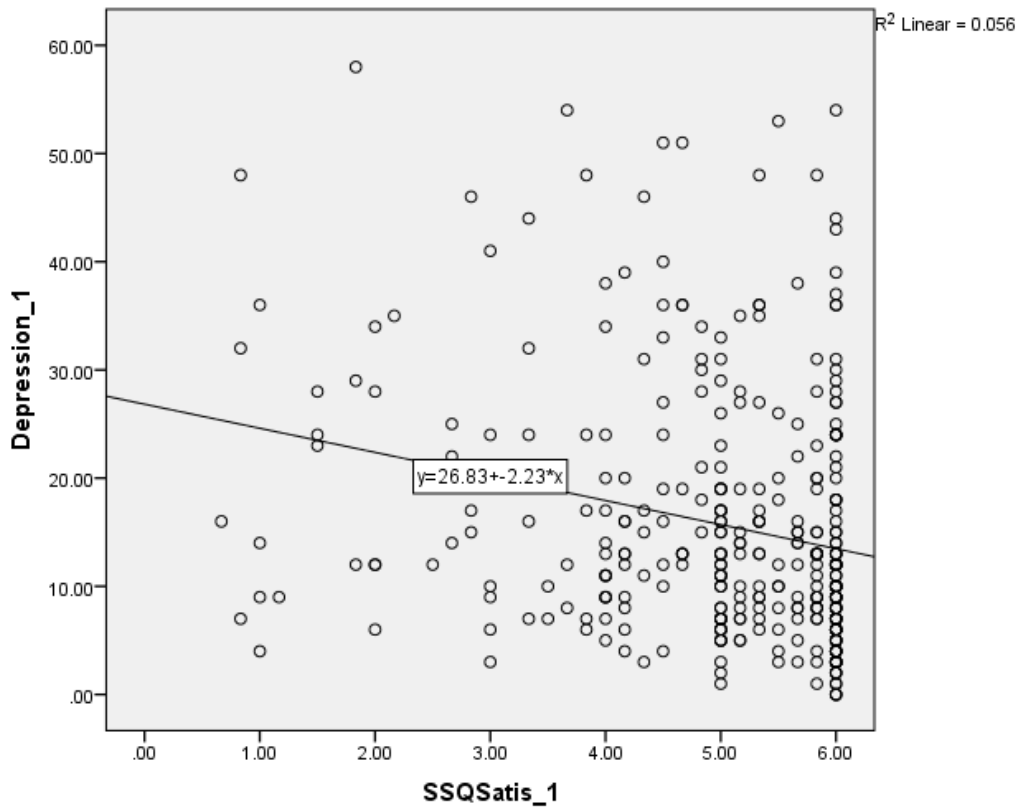
Scatterplot of Depression and Good Life Events Time 1 and 2



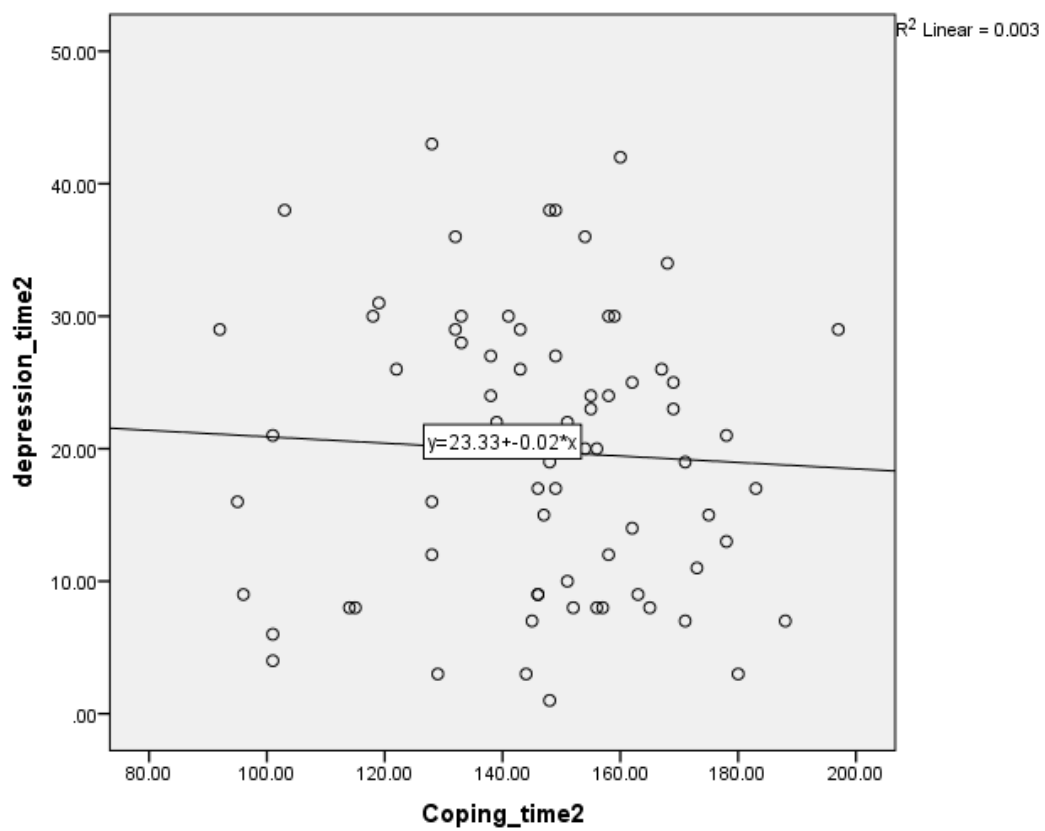
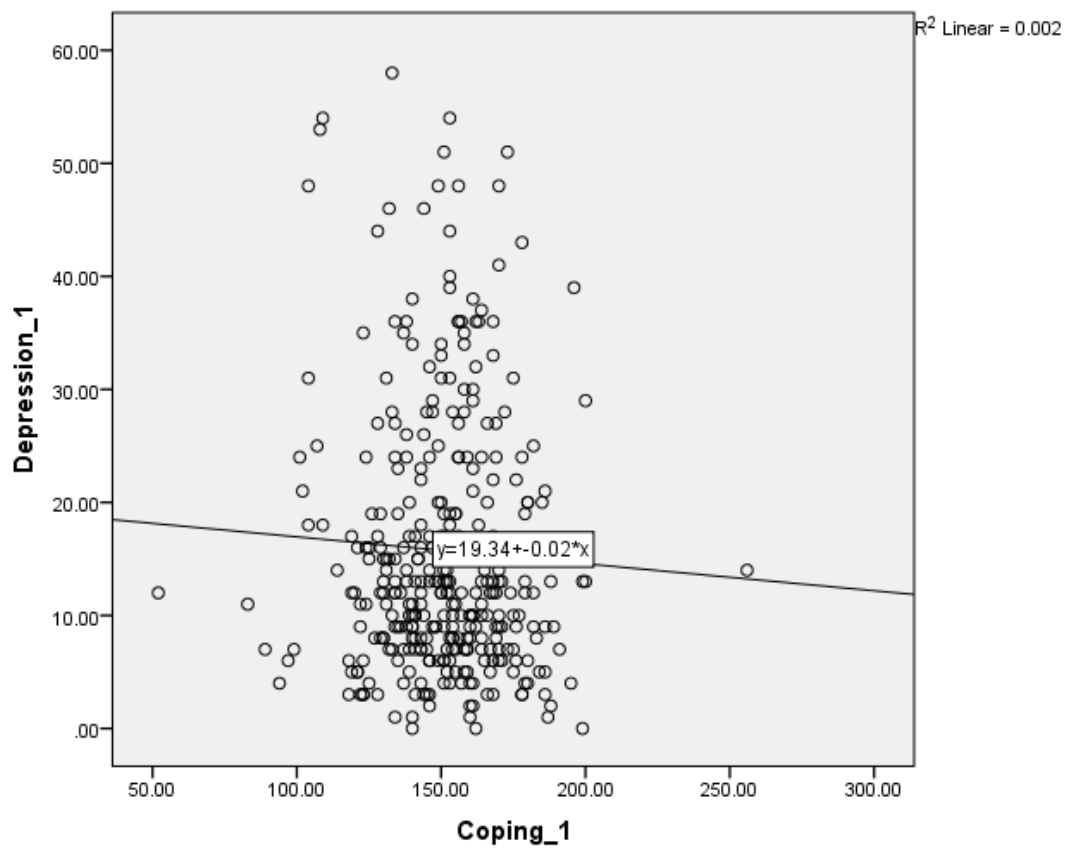
Scatterplot of Depression and Actual Social Support Time 1 and 2



Scatterplot of Depression and Social Support Satisfaction Time 1 and 2



Scatterplot of Depression and Coping Time 1 and 2





**APPENDIX D:**

**Study Two Information Sheet and Lotto**

Thank you for participating in this follow up study conducted by Kate Fenton from the Australian National University. You will be asked to complete a survey you have previous done and again answer questions about your mood, how you cope with stress, who your friends are and how supportive you find the people around you.

To participate you respond to questions on the computer screen by selecting the appropriate answer with your mouse. There are no right or wrong answers. Please try to answer as honestly and quickly as possible.

Your responses will be anonymous and the researcher will make no effort to link you to the survey. Your participation is voluntary and you do not have to complete this survey. Throughout the survey you may withdraw at any time by closing your internet browser. This research is approved by the ANU Ethics Office number 2011/032.

**Please make sure you have already completed this survey. If you haven't completed this study before please email the researcher on [kate.fenton@anu.edu.au](mailto:kate.fenton@anu.edu.au) and a link can be sent to you to complete the first part which will allow you to go into the draw to win an IPAD.**

**I understand that by clicking the 'NEXT' button I indicate that I have read and understood this information and both myself and my parent/guardian have provided written consent.**

**Thank you for taking part in this research. Your participation is much appreciated and will contribute to the knowledge of policy makers and health professionals in order to understand the unique experience of adolescence.**

**To go into the draw to win an IPAD send an email to [kate.fenton@anu.edu.au](mailto:kate.fenton@anu.edu.au) notifying her that you have completed the study. Please include the date and time you finished the survey. The prize will be drawn by the end of the year and if you win the IPAD will be delivered to you.**

The purpose of this study was to explore various aspects of adolescent depression, and the link between depression, personality and available social support. How you cope with stress and changes in your life was also explored. The researcher is gathering data from young people around Australia. Once the data is collected the results will be analysed to determine the relationship between adolescent depression, personality, coping and social support. If you have any further questions regarding the study please contact Kate Fenton on [kate.fenton@anu.edu.au](mailto:kate.fenton@anu.edu.au). This research was approved by the Australian National University's Human Research Ethics Committee, protocol number 2011/032. If you have any ethical concerns please contact the Australian National University's Human Ethics Committee on 6125 7945 or [Research.Office@anu.edu.au](mailto:Research.Office@anu.edu.au).

**If you feel you need to speak to a professional regarding any psychological concerns in the first instance contact your school counsellor. Kid's Helpline are there to listen at all hours on 1800 55 1800 FREE if you need to talk to someone outside of school time.**

**APPENDIX E:**

**CASE STUDY**

# Case Study: Comparison of two Adolescents Treated for a Major Depression Episode using Cognitive Behavioural Therapy (CBT) and Interpersonal Therapy (IPT)

Kate Fenton, Prof Don Bryne, Dr Fiona Perrett.

## Abstract

**Objective:** To assess the change in depressive symptoms in two adolescents using current best practice therapies, and to investigate if personality and psychosocial functioning had an impact on treatment. **Method:** Two female adolescents participated in the study. They were randomly assigned to either CBT or IPT treatment. Depression was assessed via standardised measures pre-treatment, weekly and also post treatment. Personality, social support and stress were also assessed pre and post treatment. **Results:** CBT outperformed IPT in symptom reduction. The two young people had significant psychosocial and personality differences. The young person treated with CBT was significantly different on measures of neuroticism and social support. **Conclusions:** CBT demonstrated higher treatment efficacy in treating the straightforward case of depression, not complicated by personality vulnerabilities.

Current best practice standards recommend the use of Cognitive Behavioural Therapy (CBT) or Interpersonal Therapy (IPT) as the first line of treatment for adolescent depression (NICE, 2005; 2015). Both CBT and IPT are manualised, time limited treatments, with robust evidence bases, including random control trials (Curry, 2011; David-Ferdon & Kaslow, 2008). Where the two therapies differ, is in their key treatment focus and methodology. CBT focuses on teaching the link between thinking, feelings and behaviour and how they can function as maintaining factors, whereas IPT aims to improve interpersonal functioning by addressing areas such as interpersonal sensitivity and grief (Moreau, Mufson, Weissman, & Klerman, 1991; Mufson, Dorta, Moreau, Weissman, 2004; Spirito et al., 2011).

The provision of sound timely intervention is of high importance, as a major depressive episode in the formative years is associated with functional impairment, risk of suicide and relapse (Kessler al., 1997). Optimal treatment reduces the chance of disruption to the young person achieving their developmental milestones (Curry, 2011). Comparison studies have generally shown CBT to be superior in symptom reduction and maintenance of treatment gains (Rossello, Bernal, & Rivera-Medina, 2012). However, no sound therapeutic guidelines exist to assist clinicians in their choice of therapy (NICE, 2005).

Studies have suggested that individual aspects of the therapy candidate, such as personality and attachment style may impact on therapeutic outcome. Specifically, McBride and Colleagues (2010) found when comparing young people with an anxious-avoidant attachment style, to securely attached youth, CBT was superior to IPT in treating the depressive episode. Whereas, Bagby and

Collegues (2008) found that depressed individuals high on neuroticism fared better with pharmacological treatment than CBT. Overall the results suggest that individual aspects may impact on treatment and as such, their variables should be considered in the choice of therapy.

While studies have begun to address the concept that individual aspects may inform treatment choice, no studies to date have compared CBT and IPT, with knowledge of individual differences, in Australia adolescents. As such, the current study aimed to complete two case studies of two Australia adolescents experiencing a major depressive episode, and analyse if individual personality and psychosocial differences impact treatment outcome.

## **METHOD**

### **Participants**

Two adolescent females, JB (aged 13 years) and AS (aged 15 years) participated in the study. AS was referred to local child and adolescent mental health services based in NSW by the hospital emergency department, whereas JB was referred by her mother. Both participants met the DSM V criteria for a Major Depressive Episode- Severe- First Episode (APA, 2013). Both young people were also being treated concurrently with psychopharmacological treatment. AS was treated with Fluoxetine (SSRI antidepressant), while JB was prescribed Ritalin (stimulant medication, to treat established diagnosis of ADHD). With the exception of JB's historic diagnosis of ADHD there were no other comorbid disorders. The two young people were similar in their family's socio-economic status, and they were both students at the local public high school. Neither of the young people had any major physical illnesses or severe childhood trauma.

### **Procedures**

Permission was obtained from the relevant ethical boards prior to commencing the study. The participants and their parents also provided written consent to take part in the study. Two senior psychologists, of doctorate level or higher, supervised the registered psychologist who provided the therapy. Participants were randomly assigned to the therapy via a random draw.

Prior to commencing therapy the participants completed pre measures via an online survey. The link was provided to them after discussing the study and obtaining consent. They used the same platform to complete the post measures. Qualtrics software was used for the online platform and the rights were purchased by ANU.

The CBT intervention was based on the manual by Munzo & Miranda (2000), and the IPT was based on Mufson & Weissman's (2004) adaptation of IPT to IPT-A for adolescents. The intervention

consisted of 12 individual sessions for AS, and 10 individual sessions and 2 family sessions for JB (family sessions are an important component of manualised IPT). Treatment was delivered weekly for the first ten sessions, while the final two sessions were fortnightly bolster sessions. Session lasted for 50 minutes.

## Measures

Pre and post measures were completed on the entire battery of questionnaires. While changes in depression scores were measured weekly.

Depressed mood was monitored weekly using the Center for Epidemiology Depression Scale (CES-D). The CES-D is a commonly used self-report scale with normative data for adolescents in community and clinical samples (Hogue & Steinberg, 1995). The scale has high internal consistency of .85 in nonclinical and clinical samples, moderate test-retest coefficients ranging from .51 to .32 for time intervals varying between 2 weeks and 12 months and moderate correlations with convergent measures of depression (Caracciolo & Giaquinto, 2002; Radloff, 1991). Items on the scale reflect the four dimensions of depression, including depressed affect, lack of positive affect, somatic retarded activity, and interpersonal relations (Radloff, 1991). The scale consists of 20 questions and respondents indicate how often they have experienced symptoms of depression over the past week, on a 4-point scale ranging from 0 (rarely or none of the time) to 3 (most or all of the time) for questions such as 'I was bothered by things that usually don't bother me' and 'I did not feel like eating; my appetite was poor'. A score of 19 or higher is indicative of depressed mood, however as adolescents tend to inflate self-report questionnaires due the emotional instability in this period, a conservative cut off of 20 was utilized (Radloff, 1991).

The pre and post self-report measures were: The Junior Eysenck Personality Questionnaire (JEPQ ; Eysenck & Eysenck, 1975); The Adolescent Stress Questionnaire (ASQ; Byrne, Davenport & Mazanov, 2007) and The Short Form Social Support Questionnaire (SSQ; Sarason, Levine, Basham & Sarason, 1983). The JEPQ measured neuroticism and extraversion, while the ASQ measured the amount of subjective stress the young person reported. All three scales are well established, with moderate- high internal consistency, and test-retest reliability (Aluja et al., 2002; Caracciolo & Giaquinto, 2002; Kafetsios & Nezelek, 2012; Martinez & Lau, 2001; Murray, Byrne & Reiger, 2011; Radloff,1991).

## RESULTS

AS and JB's pre and post test scores on the measures are shown in Table 1 below. Table 1 contains the pre and post measurement scores, taken in the first and last week of therapy. As shown in Table 1, prior to treatment, AS and JB had very similar depression scores, both of which were in the severe range (>40). This is confirmatory with the DSM-V diagnosis made via clinical interview.

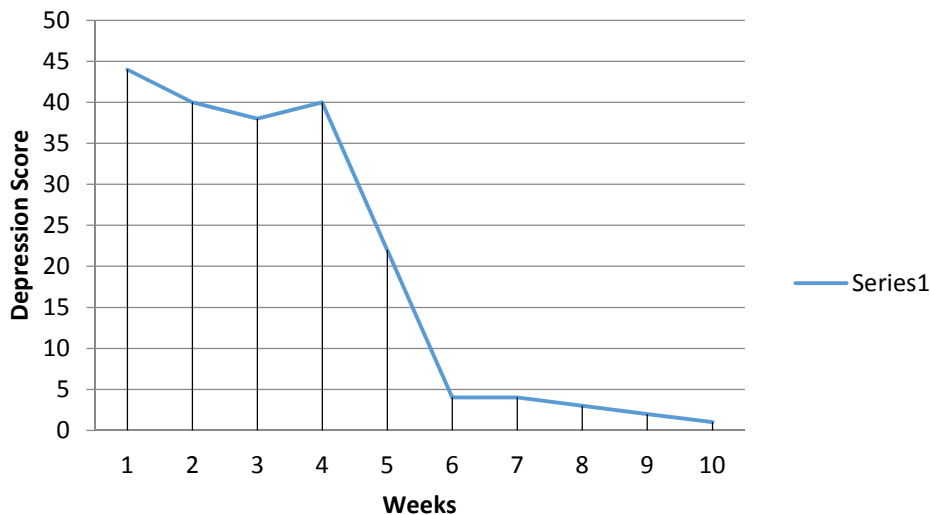
Following treatment the two participant's depression scores differed. While these did not reach statistical significance,  $p < .05$ , CI [-8.13, 29.13], the clinical implication of the results are very important. Specifically, at completion, AS no longer demonstrated any depressive symptoms above the CES-D threshold (<20). While JB's symptoms reduced during the course of therapy, she still reported depression symptoms at the end of treatment. At completion she had a score of 20 on the CES-D, which indicates a mild/moderate depressive illness.

*Table 1.* Pre and Post Measure Scores on the Main Variables

	Pre Treatment		Post Treatment	
	AS	JB	AS	JB
Depression	43	41	1	20
Neuroticism	19	30	2	25
Extraversion	10	17	21	19
Social Support	2.67	1.67	2	2
Stress	217	300	82	250

After 5 weeks of CBT treatment AS no longer met the criteria for depression (as classified by a score of <20 on the CES-D). As shown in Graph 1, she experienced a dramatic drop in symptoms after four weeks of therapy. This coincided with completing the behavioural activation component of CBT and beginning cognitive strategies at session five. Following completing behavioural activation and one session of cognitive therapy her CES-D score was <5. Following treatment AS demonstrated lower stress levels. However, her pre and post measure scores showed variability on the personality measures with a reduction in neuroticism from an average score of 19 (as compared to adolescent norms) to a score of 2; and an increase in extraversion from 10 to 21. Her social support satisfaction was similar pre and post treatment.

*Graph 1.* AS Scores on the CES-D during CBT Therapy



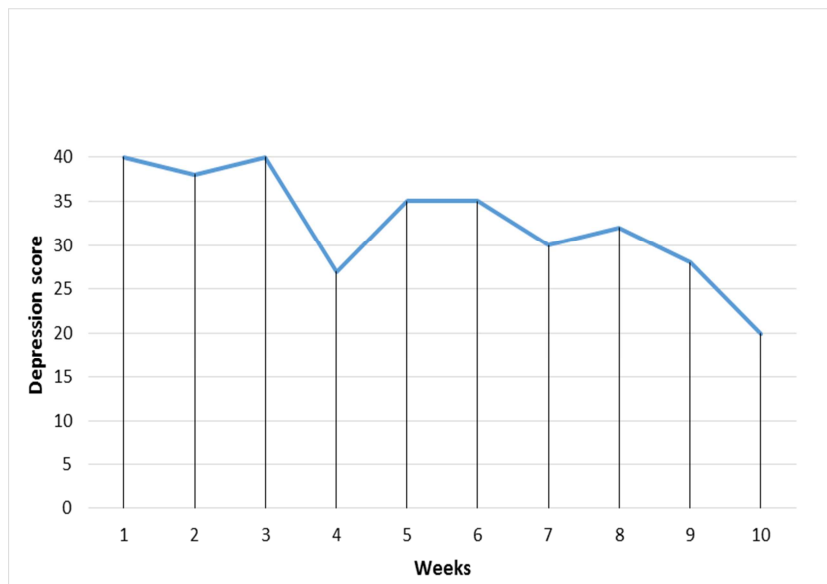
As shown in Graph 2 below, JB experienced depressive symptoms across the course of therapy. Interestingly after the family sessions in week 4 and week 7 her depressive symptoms increased. From week 8 onward her score on the CES-D demonstrated a downward trend. Week 8 coincided with strategies to address interpersonal functioning, and role plays addressing how to connect with friends. Friendships were a challenge for JB, as reflected in her pre therapy score on the SSQ (1.67) and also her clinical interview. Her friendships improved slightly using the skills she learnt in IPT and at the end of therapy she had a score of 2 on the measure, suggesting she was slightly more satisfied with her social support.

Her stress levels remained high at the end of the therapy and she reported that her home life was stressful. JB's score on extraversion increased slightly (from 17 to 19) across the course of therapy which may suggest she began to feel more confident interacting socially due to the skills she learn in IPT. Her neuroticism score was slightly reduced which may indicate a reduction in depressive symptoms.

Measures of the statistical difference between neuroticism scores for AS and JB did not find they significantly differed,  $p < .05$  CI [-9.03, 36.03]. No other significant differences were found on the main measures,  $p < .05$ . However, given the small sample size a significant difference on the main measures would be rare as overlapping confidence intervals are very common in small samples.



Graph 2. JB Scores on the CES-D during IPT Treatment



## DISCUSSION

The aim of the current study was to explore and compare the treatment outcomes of the two gold standard treatments for adolescent depression, CBT and IPT. A secondary aim was to explore if individual differences in the participants may have impacted on treatment outcome. Two Australian adolescents with depression were randomly assigned to 12 weeks of either therapy.

In line with a number of published studies, CBT was superior to IPT in the treatment of adolescent depression (Curry, 2001; Rossello, 2012). CBT resulted in not only remission of depression for the young person treated, but a quicker timeframe from treatment onset to symptom reduction. The largest treatment effect occurred following completion of the behavioural activation (BA) component of CBT. Studies have suggested that a large portion of the treatment effect found in CBT is the result of BA and the current study suggests confirmation of such a finding (Ekers et al., 2007; Jacobson et al., 1996; Lewinsohn, Biglan, & Ziess, 1976).

Between the two individuals there were differences on the main measures. While these didn't reach statistical significance they are clinically interesting. The young person treated with CBT had lower initial levels of trait neuroticism and stress, and higher social support prior to intervention. She also had an increase in extraversion following treatment. Interestingly, AS' neuroticism score reduced dramatically after the completion of treatment, whereas JB's score remained relatively static. As such, it is possible the AS' score was inflated due to depression. Studies have shown depression inflates the self report of neuroticism, and it is possible that JB's score may be more

indicative of her usual personality composition (Enns, Larsen & Cox, 2000; Karsten et al., 2012). Higher levels of neuroticism may have been one reason JB did not respond as well to therapy, as neuroticism is implication in poor treatment outcome, difficulty forming a therapeutic alliance and high rates of depression relapse (Mulder, 2001; Vittengl, Clark, Thase, & Jarrett, 2015).

Furthermore, JB retained elevated stress levels, and in particular family stress was particular potent for her. Interestingly when family based work was undertaken she experienced an exacerbation of depressive symptoms. As such JB may have benefited from a family systems approach to her treatment. However this is indicated as a second line of treatment after offering IPT or CBT (NICE, 2005; Restifo & Bogels, 2009). As such, young people with complex family systems may require a different treatment modality from the outset and this concept requires further exploration.

It is important to consider the results in light of AS also being treated with antidepressant medication. While the guidelines for the treatment of severe depression suggest the concurrent use of therapy and medication, it is possible that the treatment effect may have been the result of factors other than the therapy (NICE, 2005).

The current study was exploratory in nature and used a very small sample size. As such, caution should be used when interpreting the results. The study has merit in that it provides an interesting contrasting clinical case study which future research could replicate on a larger scale. It also draws attention to the ease of collecting information on individual differences and including them in any treatment study.

Overall the current study found CBT to be a superior treatment modality than IPT in terms of overall and also time related symptom reduction. While the current study was exploratory and is unable to be compared to a larger cohort of individuals, it provides a useful first exploration and could inform larger treatment studies. Specifically, future studies could build on the current research by undertaking a large scale study of CBT and IPT for depressed Australian adolescents. The current findings also suggest it would be useful to also survey individual differences to inform treatment decisions.

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**APPENDIX F:**

**CASE STUDY CONSENT FORM**

Research School of Psychology  
College of Medicine, Biology  
and the Environment  
Building No. 39  
Canberra ACT 0200 Australia  
Telephone: 61255545  
Facsimile: 61250499

Researcher: Kate Fenton (BSc Science (Psychology) Hons. / B Arts) Psychologist Doctorate Candidate
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Project: Adolescent Depression: Comparing two treatments

## Participant Consent Form

Dear Participant,

The current research is being conducted as part of a Clinical Doctorate by Kate Fenton from the Department of Psychology at the Australian National University.

The first part of the research consists of answering a questionnaire which aims to measure your mood and some aspects of your personality. There are no right or wrong answers. You respond by selecting the option which you think is the most like you. If your answers on this questionnaire mean you may benefit from learning some skills to help with your mood you and your parent/guardian will be notified and you may be invited to take part in a treatment which has scientific evidence.

The treatment would consist of therapy, which means attending weekly for approximately 12 weeks and talking about things which have happened either recently or in the past. The role of the therapist is to help you understand your experience and learn skills which would mean you felt more able to achieve your goals.

If you do not want to take part in this study you don't have to. You can stop at any time during the course of therapy without consequences. Your results will be kept private by the main researcher and no information which could identify you would be shared.

Name: ... ..

Signature: ... ..

Date: ... ..

Research School of Psychology  
College of Medicine, Biology  
and the Environment  
Building No. 39  
Canberra ACT 0200 Australia  
Telephone: 61255545  
Facsimile: 61250499

Dear Parent or Guardian,

Your child has been identified as potentially benefiting from a treatment for low mood. If you consent it would be beneficial for the author to monitor their progress across treatment to comment on the effectiveness of the treatment in improving their mood. The therapy would be conducted by Kate Fenton who is a Registered Psychologist and a Candidate in the Australian National University's Doctor of Clinical Psychology Degree. She is supervised by two Clinical Psychologists, Professor Don Byrne and Dr Fiona Perrett. The current research has also been approved by the ANU Ethics Committee.

Many psychologists aim to test the efficacy of therapy and conduct scientific analysis of treatment outcomes. No identifying information about your child or your family would be included in the scientific reporting of the treatment. The results will be de-identified and included in the authors Doctorate Thesis and potentially also in scientific academic journals. To begin with your child would be asked to complete a questionnaire designed to screen for low mood, to look at aspects of their personality and how supported they feel. The therapy program is based on two types of therapy which have been researched extensively and shown to be more effective than routine clinical care. The treatments utilise cognitive behavioural therapy (CBT) and interpersonal therapy (IPT) frameworks and if your child is eligible to participate they will be randomly assigned to one type of therapy. Treatment takes approximately 12 weeks and sessions occur weekly.

Please indicate in the space below if you give permission for your child's results to be written included in the authors study. If for any reason you object to your child taking part there will be no penalty and they will still receive routine clinical care. Your child will also have the opportunity to withdraw from participation at any time and if they do not want to participate. If you have any questions regarding this research please contact Kate on [kate.fenton@anu.edu.au](mailto:kate.fenton@anu.edu.au).

Yours sincerely,

Kate Fenton

-----> Please circle one option below

**I give permission** for my child to participate

Parent/Guardian Name: ... .. Signature: ... ..

Date: ... .. Contact Number.....







