# Hopelessness in Young People: a Twin Study

Eileen Walsh Doctorate in Clinical Psychology University College London ProQuest Number: U642681

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#### Abstract

Background: Suicide ranks among the leading causes of deaths in young people. The central role of hopelessness in the development of suicidal ideation has been supported by empirical research. Research on youth hopelessness has focused mainly on clinical populations, where suicidality has already been identified. Aetiological influences and correlates of hopelessness are at present poorly understood. This study investigated hopelessness in a community sample of adolescent twin and sibling pairs, in order to elucidate the relative contributions of genetic and environmental influences.

**Method:** Twin and sibling pairs (aged 15-23) and their primary caregiver provided ratings of depressive symptoms and externalising behaviour problems, associated cognitions (hopelessness, attributional style) and environmental stressors (life events, social adversity), by mailed self-report and parent-report booklets. Phenotypic and behavioural genetic analyses were undertaken.

Results: Individual differences in hopelessness showed no evidence of a genetic contribution, instead showing substantial non-shared environmental influence and some shared environmental influence. Hierarchical regression analyses indicated that (i) attributional style did not moderate the relationship between dependent negative life events and hopelessness, and (ii) a depressogenic attributional style and aggressive behaviour problems contributed uniquely to hopelessness, over-and-above the effects of social adversity, parental depression symptoms and prior youth depression symptoms.

Conclusions: The present study represents an initial examination of the genetic and environmental contributions to variance in hopelessness. Individual-specific environmental influence was the most important contributor to hopeless cognitions.

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#### **Chapter 1: Introduction**

#### 1.1 Overview

This study was designed to examine risk factors for hopelessness from a developmental genetic perspective. It will examine the impact of emotional and behavioural problems, cognitive style and life events on risk for hopelessness in late adolescence and early adulthood, in the context of a genetically sensitive study design. The first section will define hopelessness and describe its importance in cognitive theories of depression and suicidal behaviour. The main empirical findings with both adult and adolescent populations are reviewed. This is followed by an account of theories and research that address the development of hopelessness, leading to the current research questions.

#### 1.2 Definition of hopelessness

Hopelessness has been defined as negative expectancies concerning oneself and one's future life (Beck, Weissman, Lester & Trexler, 1974). Beck introduced the concept of hopelessness to refer specifically to one of the cognitive features of depression and suicidal behaviour. It is an important aspect of depression as both a common feature of the depressive syndrome and a hypothesised aetiological factor (Abramson, Metalsky & Alloy, 1989). Hopelessness has been described as an essential element of suicidality (Malone, Oquendo, Haas, Ellis, Li et al., 2000), even more so than depression (Beck et al., 1985; 1990; 1993). Conceptualised as having both 'state' and 'trait' features (Young, Fogg, Scheftner et al., 1996), a significant level of hopelessness can also be present when a person is not depressed (e.g. Haatainen, Tanskanen, Kylma et al., 2003).

## 1.3 Hopelessness and theories of depression

Individuals vary in their responses to negative life events. Investigators have attempted to understand individual differences in the response to stress in terms of biological, psychological and social processes. From the cognitive perspective, the meaning people give to their experiences influences whether or not they will become depressed and whether they will be vulnerable to recurrent, severe or long lasting depressive episodes. Two of the major cognitive theories of depression, Beck's (1967) theory and the hopelessness theory (Alloy, Abramson, Metalsky & Hartlage, 1988; Abramson et al., 1989) are vulnerability-stress models that conceptualise individual variability in the response to stress in terms of a set of unhelpful cognitive patterns. Both theories posit that particular cognitive styles increase an individual's likelihood of developing hopelessness and episodes of depression, specifically a cognitively mediated subtype of depression (Abramson et al., 1989), when they encounter negative life events. People who possess such depressogenic cognitive styles are vulnerable to depression because they tend to generate interpretations of their experiences that have negative implications for themselves and their futures.

Beck (1967) derived his cognitive theory from extensive observation of people showing depressive symptoms. According to Beck's (1967) theory, an individual's beliefs revolving around themes of inadequacy, failure, loss and worthlessness are hypothesised to provide this cognitive vulnerability to depressive symptoms. The notion of a cognitive vulnerability rests on the schema concept. Schemas are cognitive structures that hold core beliefs. These beliefs are usually out of a person's awareness until triggered by a life event, at which time they emerge accompanied by

strong emotion. It is this emotion that the person usually notices first. Such negative self-schemata are often presented as a set of dysfunctional attitudes or self-worth contingencies such as "If I fail my exam, I am a complete failure". When they encounter negative life events that impinge on their cognitive vulnerability, individuals exhibiting such dysfunctional attitudes are hypothesised to develop negatively biased constructs of the self, world and future (hopelessness), and, in turn, depressive symptoms. Beck's (1967) definition of the "cognitive triad" – the negative view of the self as a failure, the world as harsh and overwhelming, and the future as hopeless – encapsulates the themes apparent in depressogenic beliefs. The latter part of the triad, of the future being hopeless, was observed most specifically in those showing suicidal behaviour.

The hopelessness theory of depression (Abramson et al., 1989) aims to understand the interacting roles of cognitive and environmental factors in the development of hopelessness and hence depression. Taking a process-oriented perspective, it explicitly describes a pathway to hopelessness as the cause of "hopelessness depression". The theory posits that a depressogenic attributional style acts as a distal high risk factor for hopelessness, fully activated in the presence of life stress. Abramson et al. (1989) identify two components to hopelessness: an expectation that highly desired outcomes will not occur or that aversive ones will occur (negative outcomes expectancy), and that nothing is going to change things for the better (helplessness expectancy). The concept of hopelessness includes helplessness (i.e. the inability to control outcomes, whether good or bad), and adds the expectation that negative outcomes will occur (Abramson et al., 1989).

According to the hopelessness theory, individuals with cognitive vulnerability, when experiencing negative life events of high importance, that relate semantically to their underlying beliefs, are likely to become hopeless. The distal influence of an underlying depressogenic attributional style is hypothesised to trigger negative attributions of events, with these attributions mediating the pathway from negative life events to hopelessness (Abramson et al., 1989). Negative inferences regarding stressful events increase the likelihood of an individual developing hopelessness. This sense of hopelessness will lead to symptoms of a specific cognitive subtype of depression, labelled as hopelessness depression. Importantly, hopelessness is described as a proximal cause of depressive symptoms, sufficient to lead to depression, but not necessary. Additionally, the authors allow for other causal, contributory pathways to hopelessness, not accounted for by this model.

Brown & Harris (1978) conceptualise hopelessness as the key factor in the genesis of clinical depression. They hypothesise that loss of important sources of reward or positive value as the most likely cause of profound hopelessness. In certain vulnerable individuals, factors such as low socio-economic status and ongoing low self-esteem lead to generalised hopelessness, which results in clinical depression.

### 1.4 Hopelessness and suicidal behaviour

Hopelessness is also considered to be an important variable in the aetiology of suicidal behaviour. Beck (1967) studied depressed suicidal patients, observing the common theme of hopelessness underlying their suicidal wishes. The critical role that hopelessness plays in suicide is illustrated in the sequence of events that leads a depressed person to commit suicide (Beck, 1967). According to Beck (1967), the

person systematically construes their experience in a negative way and anticipates dire outcomes for their difficulties, until such point as they are drawn to the idea of suicide as a way out of their "insoluble problems".

Both of the cognitive theories of depression address the relationship between hopelessness and suicidal behaviour. Beck's (1974) theory postulates that hopelessness increases the probability of suicidal behaviour to the extent that it is activated by stressful events. Once the hopelessness schema is activated, the person's ability to perceive, generate and implement effective coping resources to rectify the crisis or problem is impaired. Consequently, the person can see no way to resolve the crisis or problem effectively. Compounding this is their expectation that the future will not improve, thus the person may come to view suicide as the only option (Bonner & Rich, 1991). The "hopelessness model" of suicidal risk (Abramson, Alloy et al., 1998), posits that a negative cognitive style acts as a distal risk factor for the development of hopelessness and the symptoms of hopelessness depression, including suicidal ideation.

Empirical evidence has shown that hopelessness has substantial clinical utility for suicide risk assessment and prediction. It is one of the strongest and most consistently found predictors of a) suicidal ideation (Beck, Steer, Beck & Newman, 1993), b) seriousness of intent in unsuccessful suicide attempts (e.g. Beck, Kovacs & Weissman, 1975), and c) completed suicide. Dixon, Rumford, Heppner & Lips (1992) evaluated different sources of stress (negative life events and hassles) as predictors of both hopelessness and suicidal ideation. Their findings suggested that hopelessness mediates the relationship between stress and suicidal thoughts. In an

investigation of protective factors against suicidal acts in major depression, Malone et al (2000) found that scores for hopelessness were significantly higher for suicide attempters. Beck, Brown, Berchick & Stewart (1990), in a prospective study with psychiatric outpatients, found that self-reported hopelessness as measured using the Beck Hopelessness Scale (BHS) (Beck et al., 1974) was significantly related to eventual suicide. A scale cut-off score of 9 or above identified 16 (94.2%) of the 17 patients who eventually committed suicide. Beck et al. (1974) reported that the BHS correlated highly (r=.74) with clinical ratings of hopelessness.

Brown, Beck, Steer & Grisham (2000) report on a twenty-year prospective study exploring risk factors for suicide, using a sample of 6,891 psychiatric outpatients. They identified that hopelessness along with severity of depression and suicidal ideation were significant risk factors for eventual suicide. Beck et al (1999) investigated predictors of eventual suicide in psychiatric outpatients (n=3,701), finding that self-reported hopelessness as measured using the BHS, had an odds ratio of 6.43 in predicting suicide. Abramson et al (1998a) found that hopelessness, as measured by the Beck Hopelessness Scale, completely mediated the relationship between cognitive vulnerability and prospective suicidality as measured by both questionnaire and interview over a two-year follow-up, even when controlling for prior history of suicidality.

#### 1.5 Hopelessness and depression in adolescents and young adults

The hopelessness theory may be especially useful in explaining why many individuals become depressed for the first time in mid to late adolescence. Some of the key aetiological factors featured in the theory, such as cognitive vulnerability and

hopelessness, have become developmentally operative (Kashani, Reid & Rosenberg, 1989), at the same time as the number of stressful negative life events is increasing (Ge, Lorenz, Conger & Elder, 1994). The next section will outline the current understanding of hopelessness in adolescents and young adults with depression.

#### 1.5.1 Depression in young people

Early-onset depression is increasingly identified as a major public health concern. From low levels in childhood, rates of depression rise significantly during adolescence (Angold et al., 2001). By late adolescence, between 10% and 20% of young people are reported to have experienced an episode of depression (Harrington, 2002). Even those with sub-clinical symptoms may have significant impairment and are frequently referred to services (Angold et al., 1999). Developmental findings suggest that late teens and early twenties may be the peak risk period for depression in women throughout the life course (Maughan, 2002). Once established, adolescent depression shows significant continuity into adulthood in both clinical (Fombonne, Wostear, Cooper, Harrington, & Rutter, 2001) and community (Pine, Cohen, Gurley et al., 1998) samples, with up to 70% of depressed children having further episodes of depression in adult life (Harrington et al., 2000). Individual consequences can be wide-reaching. Interpersonal relationships, academic achievements and work functioning may all also be compromised (Ferguson & Woodward, 2002), the ramifications of which may be particularly significant during the developmental transition to adulthood. Depression is also a strong predictor of suicide, which is now the second most common cause of death in those under 35 years of age (DoH, 1999). Young men have been identified as a risk group of particular concern. Rates of suicide among young men (15-24 years of age) have risen by 75% since 1982 (DoH, 1993).

## 1.5.2 Hopelessness and cognitive development

Research has increasingly addressed the role of cognitions in early-onset depression. A relationship between attributional style and depressive symptoms in adolescence is a consistent finding in the literature (Joiner Jr & Wagner, 1995). This suggests that cognitive theories of mood disorder are appropriate for conceptualising depression in young people. Further consistent findings cross-sectionally associate attributional style with self-reported depression and clinical depression and support the relation of attributional style to increases in depression over time (reviewed by Joiner Jr & Wagner, 1995).

Adolescent hopelessness has been associated with extent of cognitive development. Hopelessness is believed to be strongly related to cognitive capacities necessary to form a perception of the future (Cicchetti & Schneider-Rosen, 1985). Thus, it is important to take a developmental perspective on research in the area of hopelessness (Kazdin, 1983). Specific cognitive reasoning skills, such as time perspective and probability assessment, along with the necessary components of affect, are vital developmental precursors to reaching a state of hopelessness (Siomopoulos & Inamdar, 1979). However, research findings are inconclusive, with some showing evidence for hopelessness as young as mid childhood. Kashani et al (1989) investigated hopelessness at three age levels (8, 12 and 17 year olds) in 210 children and adolescents from a community sample, finding that hopelessness did not increase from preadolescence to adolescence. This suggests that hopelessness cognitions, and

therefore the cognitive ability to become hopeless, may be present at a younger age than previously assumed.

# 1.5.3 Role of hopelessness in youth suicidal behaviour

There is now accumulating evidence that hopelessness is a key cognitive variable in youth suicidal behaviour. Some level of suicidal ideation is relatively common during the adolescent years. Community-based studies indicate that up to 12% of children and adolescents have some form of serious suicidal ideation (Pfeffer, Conte, Plutchik et al., 1980). Suicidal young people are more hopeless than nonsuicidal psychiatric controls (Asarnow, Carlson & Guthrie, 1987). Also, hopelessness appears to differentiate children and adolescents with low levels of suicidal ideation from those who plan or attempt suicide (Brent, Kalas, Edelbrock & Costello, 1986). It increases with severity of suicidal ideation in child and adolescent psychiatric inpatients (Kazdin, 1983). More hopeless adolescents made premeditated rather than impulsive suicidal attempts (Brown et al., 1991), which are more likely to result in threat to life (Brent, 1987). Indeed, more hopeless adolescents report attempting suicide in order to die (Boergers, Sprito & Donaldson, 1998). Hopelessness has also been shown to predict suicidal outcomes when depressed mood is controlled (Nock & Kazdin, 2002). Gender differences may also be important in the consequences of adolescent hopelessness, playing a more active role in female suicidal ideation, alongside depression (Rich, Kirkpatrick-Smith, Bonner & Jans, 1992).

## 1.5.4 Hopelessness and behaviour problems in young people

Hopelessness has been associated with behaviour difficulties in children and adolescents (Kashani, Suarez, Allan & Reid, 1997). This is likely to be a result of an interaction between their commonly adverse environmental contexts and features of the individual expressed in externalising problems. Research has clearly demonstrated that youth who have one disorder often have one or more co-occurring disorders as well. Children and adolescents with conduct problems have been found to be at risk of late adolescent depression (Moffitt, Caspi, Rutter & Silva, 2001), which may make them vulnerable to hopelessness at this challenging developmental stage.

Cognitive difficulties frequently occur in young people with behaviour problems, particularly those with aggression since childhood who meet diagnostic criteria for an externalising disorder (Moffitt, 1990a). Individuals with conduct problems are likely to be differentially exposed to stress as a result of adverse background circumstances and the effect of their own behaviours (Rutter et al., 1997). Cognitive deficits may render children more vulnerable to these adverse home environments, thus exacerbating existing behaviour problems (Moffitt et al., 1990b). Additionally, biased cognitions associated with conduct problems increase vulnerability in the face of the environmental stressors experienced by this group (Moffitt et al., 2001). Together, both poor cognitive skills and biased attributions may contribute to problem-solving difficulties in the context of stressful negative life events, leading to a young person more vulnerable to developing hopelessness (e.g. Bonner & Rich, 1991).

The relationship between hopelessness and aggressive behaviour has not been examined. However, young people with behaviour problems have been identified as a high-risk group for suicidal behaviour (Bonner et al., 1992). A lifetime history of suicidal thoughts or attempts has been reported in over one third of young people diagnosed with conduct disorder (Ruchkin, Schwab-Stone, Koposov et al., 2003). Retrospective research on youths who committed suicide identified that up to 67% had demonstrated various types of antisocial behaviour, and about 50% could be retrospectively diagnosed as having conduct disorder (Shaffer et al., 1996). In the suicide literature, adolescents who have attempted suicide tend to use verbal and physical aggression to deal with their negative mood state (Lehnert, Overholser & Spirito, 1994). Given the relationship between suicidality and hopelessness, aggression may be an important variable to examine in relation to hopelessness in a community sample.

## Summary

Hopelessness has been both theoretically and empirically linked to depression and suicidal behaviour in adults. Cognitive theories of depression and suicidality identify hopelessness as a proximal factor in depression and suicidal behaviour. Increasingly, similar findings have been established with adolescents, in clinical and community samples. Cognitive development may play a role in development of youth hopelessness. In young people, hopelessness has been associated with high levels of psychopathology, which may involve both internalising and externalising disorders.

### 1. 6 Development of hopelessness

#### 1.6.1 Overview

While the role of hopelessness in suicidal behaviour has received extensive theoretical and empirical attention, little work has been done to determine what factors influence an individual to become vulnerable to the development of hopelessness expectations (Bonner & Rich, 1991). It is well documented that a range of psychopathological outcomes are implicated for those who develop hopelessness cognitions, but the pathways to hopelessness are unclear. At least three groups of risk factors seem likely to be involved: environmental stress, cognitive style and genetic risk.

#### 1.6.2 Environmental stressors

Both distal and proximal environmental risks have been implicated in the development of hopelessness. Research on poverty in the UK describes "powerlessness, loss of self-esteem, depression and anger" as common feelings of those marginalized by low income (Beresford et al, 1999). Socio-economic disadvantage, such as low educational level, poor financial circumstances, being a 'blue-collar' worker and unemployment were associated with both developing and continued hopelessness in adults (Haatainen et al., 2003). Adverse neighbourhood with weak social networks (Perez-Smith, Spirito & Boergers, 2002), have predicted hopelessness independently of depression. Interpersonal relationships, both past and current, are also influential. A history of poor peer relationships (Kashani, Dandoy & Reid, 1992) and perceived lack of support from peers and family (Kashani et al.,

1997) have shown significant associations with hopelessness in adolescents. These findings indicate that social adversity is a major contributor to hopelessness.

Early experiences may contribute to later vulnerability for hopelessness. Suffering multiple adverse childhood experiences have been linked to hopelessness in adult women, even without a mental health disorder (Haatainen et al., 2003). Studies have also identified a relationship between levels of hopelessness and a history of childhood maltreatment (Gibb, Alloy, Abramson et al., 2003). Proximal negative life events have also been identified as a precursor to the development of hopelessness (Abramson et al., 1989). Adult sexual victimisation predicted current hopelessness among female students (Stepakoff, 1998). Interpersonal conflicts and notable worsening of financial situation were associated with development of hopelessness (Haatainen et al., 2003).

The psychological consequences of different environmental stressors, particularly life events, may be related to the range of likely interpretations available to the individual. Distinguishing between different types of life events may be an informative way of investigating this. Dependent life events are those over which a person has a greater level of control, e.g. failing an exam, and independent life events are those resulting independently of the person e.g. being a victim of an earthquake (Brown & Harris, 1978). Of note, negative experiences, which people bring about through their own behaviour have been recognised as a psychopathological risk (Rutter, Silberg, & Simonoff, 1993).

### 1.6.3 Cognitive style

Cognitive theories hypothesise that environmental stressors impact on hopelessness by shaping cognitive style (e.g. Abramson et al, 1989). Theoretical models of hopelessness and suicidal behaviour are based primarily on a stress-vulnerability framework, with stressors imposed on a set of cognitive risk factors resulting in high hopelessness and eventual suicidality. They propose that these risk factors pose a vulnerability to suicide that becomes apparent during adverse conditions. The development of hopelessness is conceptualised as the penultimate step in this pathway towards suicidal behaviour.

Early models focused on cognitive skills, such as interpersonal problem-solving (Schotte & Clum, 1982), whereby individuals deficient in interpersonal problem-solving are at risk of hopelessness and suicidal behaviour in stressful situations. This vulnerability means that affected individuals are unprepared to effectively perceive, generate, and implement coping responses to difficulties, thus becoming hopeless. Research findings have supported a relationship between problem-solving and hopelessness (Schotte & Clum, 1982), but show an improvement in skills when depressive symptoms abate, implicating problem-solving deficits as a concomitant of hopelessness, rather than a cause (Schotte, Cools & Payvar, 1990). More recently, a person's appraisal of their problem-solving effectiveness, rather than their actual skill, was found to be the significant factor in predicting hopelessness (Bonner & Rich, 1992), implicating a role for cognitive style.

Incorporating cognitive and environmental factors, Bonner & Rich (1987) proposed a 'transactional stress-vulnerability' model. This posits that emotional alienation,

cognitive distortions and deficient adaptive resources create a vulnerability, whereby the individual is unable to cope in stressful situations, and is thus at risk for depression and suicidal ideation. With repeated exposure to stress over time, these vulnerable individuals are presumed to reach a point of hopelessness and move into more overt lethal suicidal behaviour. Their research findings supported both proximal and distal aspects of this model. The interaction of negative life stress with cognitive mediators predicted hopelessness in college students (Bonner et al., 1991) and prison inmates (Bonner et al., 1992). Distal risk factors identified include loneliness, irrational beliefs and few reasons for living (Bonner et al., 1991).

The hopelessness theory (Abramson et al., 1989), outlined earlier, describes a similar pathway to depression and suicidality, but posits attributional style as the distal vulnerability factor, which becomes fully realised in the presence of life stress. Stability across periods of depressed mood and remission is an important component of attributional style, if it is to be regarded as a vulnerability factor. It has alternatively been suggested that a negative cognitive style is merely a confounding symptom of depressed mood (as reviewed by Alloy et al., 1999). Research has found evidence of the stability of attributional style. A study by Steinberg et al. (1998a) found that cognitive vulnerability did not worsen as a function of intervening episodes of depression, supporting its 'trait-like' conceptualisation. Evidence for stability of attributional style has also been found for adolescents. Similar scores on attributional style were reported for young people upon hospital admission with depression and on discharge when remitted, even in the context of sharp declines in depressive symptoms (Voelz, Walker, Pettit et al, 2003).

A recent body of research has identified social and familial aetiological contributors to attributional style, further strengthening the case for a trait-like cognitive vulnerability (as reviewed in Alloy, Abramson, Whitehouse, et al., 1999).

Cognitive vulnerability has been linked to incidence, prevalence and severity of depressive episodes. Individuals who exhibited negative cognitive styles, as compared with those without, were more likely to develop depressive symptoms when they experienced negative life events (e.g. Nolen-Hoeksema, Girgus & Seligman, 1986; 1992). Longitudinal investigations have shown that individuals exhibiting a negative cognitive style have a greater lifetime prevalence of major depressive disorder, with triple the rate of hopelessness depression (Alloy, Abramson, Hogan et al., 1998a). Cognitive vulnerability also has implications for illness recurrence. Comparing participants with a past history of depression, individuals identifies as high-risk were more likely than the low risk group to develop recurrences of major depressive disorder (27% as compared with 6%) and hopelessness depression (52% as compared with 22%) (Alloy, Abramson, Whitehouse, et al., 1998d).

Research evidence also supports the posited relationship between depressogenic attributions and hopelessness, with these attributions mediating negative life events in predicting hopelessness (Alloy et al., 1998). Of note, the nature (and therefore meaning) of life stressors appears important, specifically whether there is a match between the content of the person's negative attributions and the stressful life event. Individuals with an attributional style-stressor match exhibited higher levels of

hopelessness as compared to depressed patients without this match (Spangler et al., 1993).

In attempting to address the developmental origins of attributional style, research findings have focused mainly on social learning and parental psychopathology. Children may learn their cognitive styles in part from significant others. One possible mechanism is that children model the cognitive styles of their care-givers - if so, their cognitive styles should correlate with those of their mothers or fathers. Research findings on this are inconclusive. Seligman et al (1984) reported that 8-13 year old children's attributional styles for negative events correlated with those of their mothers, but not their fathers. However, other studies have failed to replicate parent-child correlations in attributional styles (Kaslow et al., 1988; Oliver et al., 1992).

However, parental psychopathology seems to play an important role. Children of women with mood disorders evidenced more negative attributional styles and cognitions about themselves (Hammen, 1992). Longitudinal findings on mothers of high-risk, but non-depressed, participants, found that they were more likely to have a history of major depressive disorder (Abramson et al., 1998d), had more depressogenic cognitive styles (Alloy et al., 1998b) and provided more depressogenic feedback about causes and consequences of stressful events that happened to their child. This suggests that, in addition to modelling, the feedback parents provide to their children about causes and consequences of negative events in the young person's life may contribute to their cognitive risk for depression.

A developmental history of maltreatment and neglect may also contribute to the formation of cognitive risk for depression (Gibb et al, 2001; 2003). Research demonstrating 'depressive realism' (e.g. Alloy & Abramson, 1988) suggests that depressives may not be as irrational as originally portrayed in Beck's cognitive distortion theory of depression (Alloy et al., 1999). The negative cognitive styles that confer risk for depression might be the internal representations of maltreatment or adverse environments depressives actually experienced, rather than cognitive distortions (Rose & Abramson, 1992). This is consistent with models formulated by several theorists (e.g. Joiner et al., 1992) that emphasise the nature of the individual's interpersonal environment in contributing to vulnerability to depression. Specific instances of emotional maltreatment have been identified as a key contributor to the development of negative inferential styles (Gibb, Alloy, Abramson, Rose, Whitehouse et al., 2001; 2003).

## Summary

A variety of environmental stressors have been linked to the development and maintenance of hopelessness expectations. These range from distal socio-economic factors to familial factors and proximal negative life events. These stressors may be (i) aetiologically linked, and (ii) moderated by an individual's cognitive style. A depressogenic attributional style confers vulnerability to hopelessness and the development and recurrence of hopelessness depression. Aetiological influences are hypothesised to involve social learning and be related to parental psychopathology and child maltreatment. Alternatively, the observed relationships between environmental and cognitive factors in predicting hopelessness may reflect underlying genetic influence.

## 1.6.4 Genetic influence

One hypothesis is that young people develop hopelessness expectations because they inherit a genetic liability for hopelessness (i.e. via "nature"). We know of no studies that have tested this hypothesis, but raise it for several reasons, as outlined in this section.

There is strong evidence for a genetic role in depression (McGuffin & Katz, 1989), particularly when onset occurs before adulthood (Rice, Harold & Thapar, 2002). There is consensus from family and twin studies that depression is both familial and heritable (McGuffin & Katz, 1989). There have been at least 10 independent twin studies of the genetic aetiology of childhood and adolescent depression, most of which used parental and self-report measures (reviewed by Rice et al., 2002). These studies have shown heritability estimates between 30 and 75% according to parental measures, and between 15 and 80% for child self-report measures.

Family and epidemiological research indicates that adolescent-onset depression may be more strongly genetically influenced than adult-onset, with both having distinct origins (Jaffee, Moffitt, Caspi, Fombonne, Poulton et al., 2002). Firstly, although a significant proportion of depressed children become depressed adults, most individuals who experience depression in adulthood were not depressed as children. Some family studies have found an increased risk of major depressive disorder in the relatives of early-onset cases, as compared with adult-onset (Jaffee et al, 2002). Increased familiality has been reported in the offspring of earlier-onset major depressive disorder (onset before 30 years) compared with the offspring of later-

onset cases (Rice, Harold, & Thapar, 2002). Children of depressed parents are at high risk for juvenile-onset major depressive disorder compared with children of non-depressed parents – and this association is explained by early parental age at onset of depressive illness (Rice et al., 2002).

There is also evidence of increasing genetic influence in depression with age (towards mid to late adolescence), suggesting that developmental change may be important. Thapar & McGuffin (1994), using the Mood and Feelings Questionnaire, showed that shared environmental factors accounted for most of the variance for children's symptoms (8-11 years). In contrast, their findings for adolescents, both self and maternally rated, showed that genetic factors were substantial. Rice et al. (2002), using maternal-reported scores from the Mood and Feelings Questionnaire, found that genetic influence increased from zero in 8-10 year olds to 29% in 11-17 year olds. Mid to late adolescence is also indicated as a developmental period when cognitive skills have advanced. Increased prevalence of hopelessness expectations from mid adolescence onwards may reflect this increased genetic influence.

Gender may influence the effect of genetic influence on adolescent depression. Eley & Stevenson (1999) examined self-reported scores from the Child Depression Inventory in 490 twin pairs aged 8-16, finding that the importance of additive genetic factors increased with age in males but decreased with age in females. Heritability has been shown to increase with age in adolescent girls (Silberg, Pickles, Rutter, Hewitt, Simonoff, et al., 1999). Scourfield, Rice, Thapar, Harold, Martin et al. (2003) examined genetic and environmental influences on depressive symptoms in a population based twin sample aged 5-17. In both cross-sectional and longitudinal

analysis, shared environmental effects had significant influence in younger children but not in adolescence, when depression scores were significantly more heritable. Significant new genetic influences emerged in adolescence but no new shared environmental influences. Some sex differences were found, with girls showing greater genetic influence than boys, but only from parent-reported data. There is also some evidence for gender differences in hopelessness, with reports of higher scores in females. This may reflect genetic influence on cognitions.

Genetic influence appears to be stronger with severe levels of depression (Gjone et al., 1996). This severe sub-group are also most at risk of hopelessness, both precipitating and concurrent with their depressive illness, which may reflect a genetic component to the development of hopelessness.

Broad spectrum internalising and externalising disorders, in general, and depressive and antisocial behaviour disorders, in particular, share some common genetic liability (O'Connor, Neiderhiser, Reiss, Hetherington & Plomin, 1998). Jaffee et al (2002) reported differences in early childhood risk factors for juvenile and adult onset depression. Juvenile groups had similar high-risk profiles on childhood measures. They showed more perinatal insults and motor skill deficits, caretaker instability, criminality, psychopathology in family of origin, behavioural and socioemotional problems. There is evidence for a higher degree of psychopathology in 'high hopelessness children', which may reflect a genetic liability. High hopelessness scores in children have been linked to more withdrawn behaviour, higher negative mood and lower levels of adaptability to their environment (Kashani et al., 1991). Children with high hopelessness had lower cognitive ability, "difficult

child" temperament characteristics, more anxiety, lower self-esteem and a higher degree of psychopathology than the low hopelessness group (Kashani, Vaida, Soltys & Dandoy, 1991).

The effects might also reflect a combination of genetic and environmental factors.

For example, the same genes influencing parental depression may also contribute to susceptibility for depression in their offspring. As the young person becomes the recipient of both 'depressogenic' rearing environments and 'depressogenic' genes, these risks are confounded, an effect known as a gene-environment correlation (Scarr & McCartney, 1983). This could arise when the parental genetic make up influences both the child's genotype and the quality of the relationship with the child, a passive gene-environment correlation. Alternatively, the child's genetic propensities may, through certain behaviours and cognitions, elicit certain reactions from the parent, an evocative gene-environment correlation. For example, externalising behaviour problems in the child may influence a punitive response by a depressed parent, leading to deterioration in their relationship quality. Thus the child may have less support available to buffer the effects of their own low mood, and be more likely to become hopeless.

There is also some evidence for the heritability of skills such as attributional style, problem solving and coping with stress, deficits in all of which have been linked to the development of hopelessness. Higher concordances for attributional style among adult monozygotic twins have been found, as compared with dizygotic, indicating genetic effects (Schulman, Keith & Seligman, 1993). Twin/adoption analysis by Plomin, Scheier, Bergeman & Pederson, (1992) revealed significant heritability

estimates for both optimism and pessimism; multivariate analyses of their sample suggested that genetic factors contributed appreciably to associations between optimism/pessimism and mental health. There is also evidence for a substantial genetic component in correlated factors, such as the ability to cope with stressful events (Kendler, Kessler, Heath & Neale, 1991).

Research on suicidality has shown a familial relationship with genetic influences. A controlled familial study of adolescent suicides showed that suicide runs in families (Brent et al., 1996). There are twin studies indicating that genetic factors are important for suicidality (Gustavsson et al., 1996b).

The extensive evidence base for genetic influences in depression, along with a growing body of research on genetic factors in suicidality and attributional style, indicate that genetic influences may be relevant for understanding hopelessness. Also, the frequently complex extent of social and psychopathological correlates found in people who experience high levels of hopelessness may reflect an underlying genetic vulnerability. A genetically sensitive study design facilitates an understanding of the relative contribution of these factors, while investigating genetic risk.

## 1.7 Behaviour genetics methodology

One of the main aims of this investigation is to explore whether individual differences in experiencing hopelessness can be partly explained by inherited factors. The behaviour genetics methodology will be described briefly. To date, there have been no behaviour genetic studies of hopelessness in children or adolescence.

Adoption and twin studies aim to investigate genetic and environmental influences on behaviour. The principle underlying both is that there are differentially genetically related individuals growing up together in the same family. For example, while identical or monozygotic (MZ) twins share all of their genes (as they are the result of one feritilised egg splitting into two), fraternal or dyzygotic (DZ) twins share only half of their additive genes and one quarter of their genetic dominance on average, the same as normal sibling pairs. Additionally, when reared in the same family, both types of twin will experience similar environments. Environmental differences that result in within-pair similarity are called shared or common environment, whereas those that are child-specific, resulting in differences between the pair, are called nonshared environment. As such, similarity between an MZ pair is assumed to be due to sharing the same genes and sharing, by definition, the same shared environment. In contrast, within pair similarity for DZ twins is said to be due to them sharing half their additive genetic influence, one quarter of genetic dominance, but again, by definition, all the shared environment. Thus, if you have a group of MZ twins and a group of DZ twins, the difference between their correlations is relative to the size of the genetic influence (i.e., increased genetic similarity is assumed to account for the greater similarity of MZ pairs than DZ pairs). Thus, the extent to which the DZ

correlation is lower than the MZ correlation indicates the level of additive and non-additive (dominant) genetic influence.

One important assumption of the twin design is that assortative mating is negligible. This assumes that parents select each other on random character traits, rather than selecting an individual for a specifically similar trait. This assumption is important because we would expect a higher genetic correlation between siblings from parents who are particularly similar for a measured trait.

## Summary

Hopelessness has been identified as a strong predictor of depression and suicidal behaviour in clinical populations. Adolescents with high levels of hopelessness have shown a range of other psychopathologies, linked to a variety of risk and vulnerability factors. However, the development of hopelessness is poorly understood. This study aims to further elucidate the development of hopelessness and its relationship with genetic and environmental risk factors.

#### 1.8 Research aims

- The first research aim is to test the proximal pathway to hopelessness as
  posited by the hopelessness theory of depression (Abramson et al., 1989).

  Based on the current literature, which suggests that negative life events are
  moderated by a depressogenic attributional style, the first hypothesis tests this
  relationship.
  - Hypothesis 1: Attributional style will moderate the relationship between negative life events and hopelessness
- 2. The second research aim is to examine influence of earlier risk factors, social, familial and individual-specific, which have been linked to both hopelessness and psychopathology.
  - Hypothesis 2: The association between depressogenic attributional style
     and hopelessness is accounted for by social adversity
  - Hypothesis 3: The combined effects of social adversity and current parental depression show an association with young people's hopelessness

- Hypothesis 4: Depressogenic attributional style has an effect on hopelessness after controlling for social adversity and current parent depression
- Hypothesis 5: There is a relationship between externalizing behaviour problems and hopelessness, independently of social adversity, current parental depression and depressogenic attributional style
- Hypothesis 6: A depressogenic attributional style predicts hopelessness,
   over and above the effects of social adversity, current parental depression,
   externalizing behaviour problems and prior depression
- 3. The third research aim is to investigate the role of genetics in the aetiology of hopelessness. Based on the literature showing genetic influence in associated variables, such as attributional style, depression and suicidal behaviour, it is hypothesised that genetic influence may play a role in development of hopelessness.
  - Hypothesis 7: Hopelessness is influenced by genetic transmission of risk.

#### **Chapter 2: Method**

#### 2.1 Overview

This section describes the GENESIS 1219 study in detail. GENESIS stands for 'the Genetic Environmental Nature of Emotional States in Siblings' and 1219 (12-19) refers to the age range of participants when they first took part. First, the methods used for recruiting the twin and sibling pairs of adolescents will be described. Next, the behaviour genetics design used in this study is reviewed. The data collection procedure is outlined and the measures used are then presented. This section concludes with an account of the statistical analyses undertaken.

## 2.2 Participants

The sample used in this study consists of adolescent and young adult twin and sibling pairs (n=1323), along with one of their parents, most commonly their biological mother (n=662). The twin and sibling pairs were recruited to the study from different sources, as outlined below.

The participants in the GENESIS 1219 study were recruited in two ways: either from the GENESIS study, or from twin registers held by the Office of National Statistics. The initial cohort from the GENESIS study did not include twin pairs. Adolescent siblings aged 12 to 19 were recruited from parents who had participated in the GENESIS study. This is a community sample of approximately 40,000 adults aged 20-55 years taking part in a questionnaire-based study of depression and anxiety. If GENESIS participants indicated that they had children living with them, they were

contacted about GENESIS 1219, and told that this was an extension of the main GENESIS study for adolescents.

The twin pairs were recruited via Health Authorities and General Practitioners, on behalf of the study, by the Office of National Statistics. This sample consisted of a random selection of 1,000 live twin births in each year cohort between 1985 and 1988. Of the 4,000 families contacted, 1,419 initial-stage responses were received, generating a twin sample of 2,830 (1,439 females, 1,337 males), with a mean age of 14.5 years.

Once the initial questionnaire was received, a more in-depth wave 2 questionnaire was sent to both cohorts (n=4030), which yielded a 73% response rate. Approximately 2 years after wave 2 data collection, wave 3 questionnaires were sent to each wave 2 respondent, along with a parent booklet and a parent-report booklet about each young person. We received 1323 replies (55% response rate). Please see Appendix 2 for details of the sample recruitment and response rates.

Any participants who missed out more than 5 items from the hopelessness scale were discounted from analysis. Table 2.1 below shows the number of participants for each sibling group used in the analysis.

Table 2.1 Number of participants in each group that completed the Hopelessness Scale

Group	N (total=1323)	% of total N	
MZ males	128	10	
MZ females	195	15	
DZ males	104	8	
DZ females	222	17	
DZ-opposite sex	376	28	
Full sibling pairs males	60	4.5	
Full sibling pairs females	98	7	
Sib-pairs – opposite sex	140	10.5	

### 2.3 Design

The current study used a longitudinal combined twin and sibling design. This facilitated disentangling similarity across siblings that could be attributed to shared genetic heritage from shared or non-shared environment. According to this design, if the correlation between siblings for the Hopelessness Scale is the same for all sibling types, then shared environment will be an important factor. If genetic influences are important, the correlations will show the following pattern: MZ twins > DZ twins = full siblings. Data collection took place over three time points in a three year period, which enabled investigating precipitators of current emotional and behavioural problems.

#### 2.4 Procedure

The present study utilises data gathered at three different time points over a period of approximately 3 years, as illustrated below. At wave 1, each adolescent received a booklet explaining the purpose of the study and was asked to complete the short form of the Moods and Feelings Questionnaire. Parents were also asked to participate. Those who consented were sent a booklet containing questions on personal mental health such as depression symptoms and neuroticism, plus scales relating to the family and wider social context, such as threatening life events and social adversity.

Three-four months after wave 1 data was received, each adolescent was sent a questionnaire that comprised the measures described above at wave 2. The following protocol was observed for wave 2:

- send the wave 2 questionnaire with information directly to participants aged
   over 16 and to parents of participants under 16 years
- 2) Send follow-up reminder letter to non-responders 6 weeks later
- 3) Send a second reminder with another copy of the questionnaire 12 weeks after the initial mailing

The wave 3 measures were sent two years after wave 2. For wave 3, the same protocol was observed, with the addition of a final reminder 4 weeks after the second reminder.

Participants were provided with a freephone number and advised to make contact if they had queries or wished to discuss anything further.

#### 2.5 Measures

# Youth and parental hopelessness

Beck Hopelessness Scale (Beck, Weissman et al., 1974)

The Beck Hopelessness Scale (BHS) is a 20-item true-false, self-report questionnaire that assesses the degree to which a person holds negative expectations about the future. Nine of the items are keyed false and 11 true. The items are summed to obtain a hopelessness score (range 0-20). Internal consistency has been reported at .93, concurrent validity, in terms of agreement with clinical ratings of hopelessness, at .74, and, in terms of agreement with other scores of hopelessness, at .60 (Beck et al., 1974). The scale has also demonstrated good retest reliability (Holden et al., 1988). In the current study the Cronbach alpha coefficient was .82 for the young people and .85 for parent hopelessness. If a response was missing up to 25% of items (n=5), it was replaced with an arithmetic mean of the subject's other responses, while in the

case of more missing items, the BHS was regarded as missing data and excluded from the study.

### Youth and parental depression symptoms

Short Moods and Feelings Questionnaire (Angold et al., 1987)

Adolescent and parent depressive symptoms were assessed using the Short Moods and Feelings Questionnaire (MFQ) (Angold et al., 1987). The short MFQ assesses core depressive symptoms. The measure consists of 13 statements such as "I feel unhappy" which are rated on a 3-point Likert scale for frequency over the past two weeks. Summation of responses was used to create a total depression score. The scale was designed to provide a brief screen of core symptoms of depression and has good psychometric properties (Thapar & McGuffin, 1998).

### Young person's attributional style

The Children's Attributional Style Questionnaire Revised (CASQ-R) (Kaslow & Nolen-Hoeksema, 1991) contains 24 forced-choice items that assess each of the three dimensions of attributional style (internal-external, global-specific and stable-unstable). Each item describes a positive or negative event (e.g. "I get an A on a test") followed by two possible causes of the event (e.g. "I am clever" or "I am good at the subject the test was in"), from which the individual must choose. Each set of response options holds constant two of the three dimensions of attributional style, whilst varying the third, allowing for independent assessment of that dimension. A composite score is computed by summing all responses. Of note, lower composite scores indicate more negative attributional styles. Adequate indices of reliability and validity have been reported (Thompson, Kaslow & Weiss, 1998).

### Life events measures

The Life Events Scale for Adolescents (LES-A) (Coddington, 1984) was used to assess negative life events over the past year. This is a simple count of the number of life events a young person has experienced from a list of 50 items. It has been shown to be reliable and valid (Coddington, 1984). The measure is designed to include 3 subgroups. The first include adverse family events over which the young person has little control, including events such as the death of a parent or loss of a job by parent. This subgroup of items was used as a measure of 'independent life events' as their occurrence is likely to be independent of the young person's actions. The remaining subgroups comprise desirable and undesirable extra-familial events. Undesirable extra-familial events include breaking up with a boy/girl friend and suspension from school. The majority of these were used as a measure of 'dependent life events', as occurrence of these is likely to be at least partly attributable to the young person's own behaviour. One item 'death of a close friend' was excluded, as it is unlikely to be behaviourally dependent.

#### Externalising behaviour problems

The Child Behaviour Checklist (CBCL; Achenbach, 1991b) and Young Adult Behaviour Checklist (YABCL; Achenbach, 1997) are parent questionnaires for assessing behaviour problems in 4- to 18-year olds (CBCL) and young adults over 18 (YABCL). Each item relates to a specific behaviour characteristic, such as 'threatens to hurt other people' or 'doesn't feel guilty after doing something wrong'. The response format is 0=not true, 1=somewhat or sometimes true and 2=very true or often true. The measures are composed of eight syndrome scales, of which two are

used in this study: 'Delinquent behaviour' and 'Aggressive behaviour', which together constitute the 'Externalising' grouping of syndromes. As the sample ranged from mid-adolescents to adults aged 23, both scales were included in the questionnaire and the appropriate items scored for each age group. Both scales have been shown to have good reliability and validity (Achenbach, 1991; 1997).

### Social Adversity

Social Problems Questionnaire (SPQ) (Corney, 1988)

The SPQ was used to assess social adversity. It has a four-point rating scale from "none" to "severe difficulties" and assesses problems relating to finances, housing, work, relationships and social activities.

Parental education level was used as an estimate of social disadvantage. It was assessed using a scale ranging from no qualifications to a postgraduate degree.

### Demographic factors

Information was also gathered on important demographic variables. The young people provided information on their educational level, current occupation / education, accommodation and ethnic origin. Their parents reported on their family income and occupation (from which a measure of socio-economic status was estimated).

Participants completed measures over three time points, as outlined in Table 2.

	Time 1	Time 2	Time 3
Adolescent	Depression	Depression	Depression
variables	symptoms	symptoms	symptoms
		Attributional style	Negative life events
			Hopelessness Scale
Adolescent			Accuracion
Audiescent			Aggression
variables-parent			Delinquency
report			

Parent variables

Family variables

Social adversity

Parent education

Depression

symptoms

Hopelessness Scale

### 2.6 Statistical analysis

#### 2.6.1 Phenotypic analyses

Linear regression was used to test the association between concurrent depression and hopelessness in young people, adjusted for sex and age. The baseline model was estimated as,

Model I:  $HOPELESSNESS = \beta_0 + \beta_1 (ATTRIB) + e$ ,

where HOPELESSNESS refers to the young person's level of hopelessness,  $\beta$  refers to the intercept, ATTRIB refers to the young person's attributional style, and e refers to error. Regression results are based on the sandwich or Huber/White variance estimator (Gould & Scribney, 1999), a method available in STATA 7.0 (StataCorp, 2001), which adjusts estimated standard errors to account for the dependence in the data due to analysing sets of twins and provides results that are robust to model assumptions (Lumley et al., 2002). Multivariate regression analyses built on this basic model to investigate the association between hypothesised risk factors and hopelessness, while controlling for the effects of depressive symptoms.

The Goodman (1) version of the Sobel Test was then used to assess mediational effects. Mediators are variables that account for the relation between predictor and outcome. The on-line Sobel Test Calculator was used for these analyses (Preacher & Leonardelli, 2003).

### 2.6.2 Genotypic analyses

The differing levels of genetic resemblance between siblings were employed to disentangle genetic and environmental influences for hopelessness. To estimate the relative contribution of genetic and environmental factors to levels of hopelessness, intraclass correlations and univariate model-fitting analyses were conducted. Intraclass correlations provide an estimate of the magnitude of similarity between siblings. If genetic influences are important sources of individual variation, the pattern of sibling intraclass correlations for hopelessness will correspond to the siblings' genetic relatedness. In the case of the GENESIS 1219 study, the pattern of sibling correlations expected if genetic influence is important is MZ twins > DZ twins = full siblings.

If shared environment makes a substantial contribution, the sibling correlations will be large and similar across all sibling types. Nonshared environmental influence is implicated to the degree that genetic and shared environmental sources of variance cannot account for all of the observed variation. If hopelessness is mainly influenced by environmental factors that are not shared by siblings reared in the same family, the sibling correlations observed will be low and will not differ according to sibling type. The most direct estimate of nonshared environmental factors is the lack of similarity between MZ twins.

Maximum likelihood model-fitting analyses offer a more precise method to estimate the contribution of genetic (A), shared environmental (C), and nonshared environmental (E) factors by partitioning the variance of a phenotype into these three components simultaneously. To illustrate, in this study we estimate the heritability of

hopelessness by comparing correlations between different types of family members. We describe the correlation observed between MZ and DZ twins and full siblings in terms of the components of variance they share. For example, we expect the correlation between full siblings to represent half the additive genetic variance and, by definition, all the shared environmental variance but none of the nonshared environmental variance. The observed data that are modelled are the variance-covariance matrices for the twin and sib pairs. Maximum likelihood model fitting also provides estimates of a fit between an assumed model and the observed data, and allow hypothesis testing with different models (Neale & Cardon, 1992). Univariate maximum likelihood model-fitting analyses were conducted for this sample using the standard ACE model of the MX structural equation modelling package (Neale & Cardon, 1992).

As can be seen from figure 2.1, the ACE model assumes that the variance of a particular phenotype (in this case, hopelessness) is due to the three latent factors A (additive genetic variance), C (shared or common environment variance) and E (nonshared environmental variance plus measurement error). The following calculations were used to provide estimates for model-fitting. The within-pair phenotypic correlation for A (ra) is fixed according to the genetic relatedness of the sibling pairs: 1 for MZ twins and .5 for DZ twins and full siblings. The within-pair phenotypic correlation for C (rc) is set to 1 for all sibling types because, by definition, shared environmental factors account for sibling similarity above and beyond that resulting from shared genetic heritage. Therefore,

$$r_{MZ} = a^2 + c^2$$
, and  $r_{DZ} = a^2/2 + c^2$ .

Subtracting the second equation from the first gives:

$$r_{MZ} - r_{DZ} = a^2 - a^2/2 + c^2 - c^2 = a^2/2$$

$$a^2 = 2(r_{MZ} - r_{DZ})$$

Heritability is calculated as twice the difference between the correlations observed for MZ and DZ twin pairs. The proportion of shared environment is estimated as the difference between the MZ correlation and the heritability ( $c^2 = r_{MZ} - a^2$ ). The E parameter is uncorrelated because nonshared environmental factors account for differences between siblings, above and beyond those resulting from genetic differentiation. However, if they represent proportions,  $a^2$ ,  $c^2$  and  $e^2$  must sum to 1, so:  $a^2 + c^2 + e^2 = 1$ 

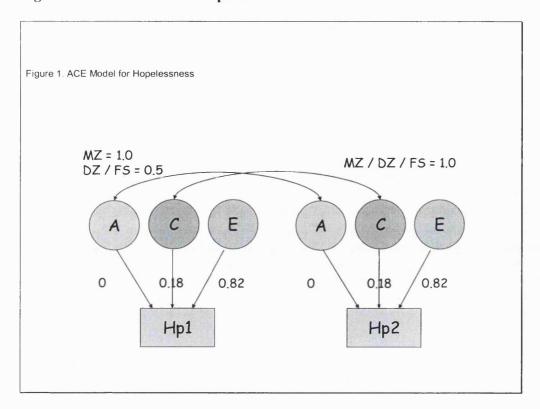
$$[2(r_{MZ}-r_{DZ})] + [r_{MZ}-2(r_{MZ}-r_{DZ})] + e^2 = 1,$$

$$\therefore \mathbf{r}_{MZ} + \mathbf{e}^2 = 1$$

$$\therefore e^2 = 1 - r_{MZ}$$

The fit between the ACE model and the observed data was assessed using  $\chi_2$  (small and nonsignificant  $\chi_2$  indicating a good fit) and Akaike's Information Criteria (AIC ( $\chi_2$ -2df); large and negative AIC indicating a good fit). To establish the best fit for the data, two alternative models were tested. The first model allowed the parameters to differ between males and females, to test for quantitative differences in genetic and environmental effects. The second was a "no sex effects" model, where the parameters for males and females were constrained to be the same. A comparison between the fit of these two models was used to test for the significance of sex effects. The relative improvement or worsening of the fit was assessed on the basis of a change in  $\chi_2$  relative to the change in degrees of freedom.

Figure 2.1 ACE model for hopelessness



### **Chapter 3: Results**

# 3.1 Socio-demographic characteristics

Table 3.1 gives the socio-demographic characteristics of the study sample. In general the sample showed a reasonable distribution in terms of characteristics such as education, employment and accommodation. As almost all participants were white, the sample is not representative of every ethnic background in the UK. As is frequently the case with studies of this type, the families participating were more likely to be from socio-economic classes 1 and 2 (59.5%), more likely to have an above-average household income (over 50% had a total household income of £30k + per year), and were more likely to own their own home (90.1%). Socio-economic class was estimated from parental occupations, based on classification categories used by the Office of National Statistics. Parents' highest education level ranged from no qualifications to postgraduate degree, showing a slight bias towards more educated, with a disproportionate 17.4% of parents having degree-level qualifications.

Table 3.1 Sociodemographic characteristics of the young people in the study

Sociodemographic characteristics	% of total sample (N=1323)
Age, yr	
15	18.8
16	23.9
17	23.1
18	20.5
19	4.7
20	3
21	2.3
22	1.5
23	0.8
Gender	0.0
Female	59.8
Male	39.2
Family Ethnic Background	37.2
White	98
Black-Caribbean	.7
Black-African	.1
Indian	.3
Mixed Race	.1
Current occupation	.1
Studying at school	30.5
Studying at college	33.1
Studying at conege Studying at university	9.9
Working full-time	11.4
-	10.2
Working part-time On a government benefit	1.0
	.3
Full time parent Other	2.7
Other	2.7
Family characteristics	
Parent highest education level	
No qualifications	11.2
CSE	23.6
O'level/GCSE	18.5
A/AS/S level	12.2
HNC	3.3
HND	2.7
Degree	17.4
Postgraduate	5.9
Pamila and annual alam	
Family socio-economic class	567
1 (professional & managerial)	56.7
II (associated professional & technical)	2.8
full-time student	.3
Family Annual Income	•
less than £15k	17.9
£16 - £20k	12.5
£21 – £30k	19
	15
	11.1
£51 – £60k	24.5
less than £15k £16 - £20k £21 - £30k £31 - £40k £41 - £50k	12.5 19 15 11.1

### 3.2 Descriptive statistics

The mean score for all 20 items on the hopelessness scale was 4.89, with a standard deviation of 1.83 (N=1323) (see table 2). This indicates a positive skew (1.44) with more responders having low or no hopelessness scores, and positive kurtosis (2.16). The reliability of the total scale for this sample using Cronbach's alpha was .82, indicating that the items had high internal consistency.

# Sex and age effects for hopelessness

Females scored significantly higher on hopelessness than males, with means of 5.01 (SD=1.85, N=866) and 4.72 (SD=1.74, n=562) respectively ( $t_{1426} = -3.21$ , p<.01). There was no significant correlation between age and hopelessness (r=-.02. p=.389).

In subsequent analyses, age and sex effects were regressed from each measure according to standard procedures (McGue & Bouchard Jr, 1984). As the distribution of scores was positively skewed, a log transformation ( $\ln (x + 1)$ ) was applied to approximate normality.

Table 3.2 Means and standard deviations for hopelessness scores across gender and zygosity.

Group	N	Hopelessness Scale
		mean (SD)
Whole sample	1323	4.89 (1.83)
MZ males	128	4.56 (1.62)
MZ females	195	4.82 (1.61)
DZ males	104	4.59 (1.68)
DZ females	222	5.31 (2.08)
DZ-opposite sex	376	4.93 (1.83)
Full sibling pairs males	60	5.13 (2.05)
Full sibling pairs females	98	4.83 (1.73)
Sib-pairs-opposite sex	140	4.66 (1.64)

## 3.3 Phenotypic analyses

The data were analysed with a series of univariate and multivariate linear regression analyses using the statistical analysis package STATA (Stata Corporation, 2002), using the "robust cluster" option, which takes into account clustering within a sample. This was necessary due to the sample consisting of twin and sibling pairs, whose responses are not independent of each other. The first phase of analyses involved multiple univariate linear regressions, with hopelessness at time 2 as the dependent variable.

### Predicting hopelessness

A significant association was found between current depression symptoms and current hopelessness. Previous history of depression also predicted current hopelessness, but accounted for slightly less of the variance. A significant association was found between both self-reported measures of depressogenic attributional style and hopelessness and "dependent" negative life events and hopelessness. Parent-reported aggression and delinquency were also significantly associated with adolescent self-reported hopelessness (see table 3.3).

Parents' self-reported current depression symptoms were significantly associated with adolescent current hopelessness. There was no relationship between parents' self-reported current hopelessness and adolescent current hopelessness.

Parents' education level was significantly associated with young people's hopelessness, with offspring of more educated parents more likely to be hopeless. Social adversity showed a association with hopelessness at 'trend' level (p=.056).

Table 3.3 Associations between young people's depression symptoms, depressogenic attributional style, negative life events, behaviour problems and current hopelessness

Measure	Robust	Robust	t.	95%	6 CI	$\mathbb{R}^2$
	B Coef.	SE				
Current depression symptoms	.095	.010	9.36***	.075	.115	.08
Depression two years ago	.004	.001	6.43***	.003	.005	.04
Depression three years ago	.004	.001	5.42***	.002	.005	.04
Depressogenic attributional style	009	.001	-8.83***	012	007	.07
Negative life events-dependent	.04	.014	2.68**	.010	.067	.02
Negative life events-independent	.017	.018	.94	018	.052	.01
Parent reported aggression	.06	.010	6.48***	.047	.087	.04
Parent reported delinquency	.01	.002	5.69***	.006	.012	.04

<sup>\*</sup>p<.05 \*\*p<.01 \*\*\*p<.001

Table 3.4 Associations between parent self-reported depression, hopelessness and social factor and young people's hopelessness

Measure	Robust	Robust	t	95%	6 CI	R <sup>2</sup>	
	B Coef.	SE					
Parent depression symptoms	.026	.011	2.30*	.003	.048	.006	
Parent hopelessness	.025	.015	1.70	004	.054	.004	
Parent education level	0003	.0002	-2.15*	001	000	.011	
Social adversity	.007	.004	1.91	0002	.0136	.012	

<sup>\*</sup>p<.05

Hypothesis 1: Depressogenic attributional style will moderate the relationship between dependent negative life events and hopelessness

Dependent negative life events were significantly associated with a depressogenic attributional style (b=-2.1, SE=.378 ,t <sub>(3, 765)</sub> =5.54, p<.001); and depressogenic attributional style was significantly associated with hopelessness

(b=-.009, SE=.001, t  $_{(3,765)}$  = -8.83, p<.001). After controlling for attributional style, dependent negative life events showed no unique contribution to hopelessness.

Interaction analyses revealed no significant interaction between attributional style with dependent negative life events on hopelessness (b=-.002, SE=.004,  $t_{(5,762)}$  = -0.43, ns).

Hypothesis 2: The association between depressogenic attributional style and hopelessness is accounted for by social adversity

After controlling for social adversity, depressogenic attributional style continued to independently predict hopelessness at a 2 year follow-up (b=.007, SE=.004, t  $_{(5, 709)}$  = 2.01, p<.05) (Table 3.7, Model I). Parent education level showed a significant negative association with hopelessness (b=.0003,SE=.0001, t  $_{(5, 709)}$  =-2.25, p<.05), meaning that young people whose parents had more education were more likely to be hopeless. This model accounted for 7.6% of the variance in hopelessness scores.

Hypothesis 3: The combined effects of social adversity and current parental depression show an association with young people's hopelessness

There was no significant association between maternal depression and hopelessness, when controlling for social adversity and parent education (Table 3.7, Model II). Social adversity or parent education did not show a significant association with hopelessness when parental depression was included in the model. This model accounted for 2% of the variance in hopelessness scores.

Hypothesis 4: A depressogenic attributional style has an effect on hopelessness after controlling for social adversity and current parent depression

Depressogenic attributional style continued to be significantly associated with hopelessness, after controlling for the effects of social adversity, parent education and current parent depression (b=.01,SE=.001, t  $_{(6, 626)}$  =-8.15, p<.001) (Table 3.7, Model III). This model accounted for 8% of the variance in hopelessness scores.

Hypothesis 5: There is a relationship between externalizing behaviour problems and hopelessness, independently of social adversity, current parental depression and depressogenic attributional style

Aggression shows a significant relationship with hopelessness, independently of social adversity, parent education, current parent depression, delinquency and depressogenic attributional style (b=-.003, SE=.001,  $t_{(8, 625)}$  =2.61, p<.01) (Table 3.7, Model IV). Delinquency did not show a significant relationship with hopelessness, when the other factors were included in the model. This model accounted for 9.98% of the variance in hopelessness scores.

Hypothesis 6: A depressogenic attributional style predict hopelessness, over and above the effects of social adversity, current parental depression, externalizing behaviour problems and prior depression

Depressogenic attributional style was significantly associated with hopelessness, over and above the effects of social adversity, parent education, current parental depression, externalizing behaviour problems and prior depression (b=-.01, SE=,.001,  $t_{(9, 625)}$  =-5.61, p<.001) (Table 3.7, Model IV). This model accounted for 10.4% of the variance in hopelessness scores. Parent-reported youth aggressive behaviour continued to show a unique contribution to hopelessness (b=.002, SE=.001,  $t_{(9, 625)}$  =2.41, p<.05).

# 3.4 Genotypic analyses

Hypothesis 7: Hopelessness is influenced by genetic transmission of risk

The analyses conducted in this section investigated the genetic, common environment and non-shared environment components of the hopelessness scale. Table 3.5 shows the intraclass correlations between sibling pairs for hopelessness.

Hopelessness similarity was comparable for MZ and DZ twins and for full siblings. These correlations suggest that there is unlikely to be any meaningful genetic influence on hopelessness. However, it is unclear whether the differences in male and female scores reflect a gender difference in heritability. Also, we do not know the role of environment that is shared by siblings, as compared with their non-shared environment. To elucidate the influences of genetic and both common and non-shared environmental influences, including gender differences, univariate model fitting analyses were used.

Univariate model fitting for hopelessness

The model fitting was conducted using Mx – a behaviour-genetic computer-modelling package developed by Neale (1999). A sex-limited univariate model (Neale & Cardon, 1992) for hopelessness was computed to examine the patterns from the sibling correlations.

Table 3.6 displays the components of variance and chi-square values for univariate model fitting. The second "no sex effects" model showed the best fit, with a higher AIC.

This indicates that there are no significant sex differences in the proportion of hopelessness accounted for by shared and nonshared environmental factors.

The findings show that genetic transmission does not seem to play a role in young people's hopelessness. Shared environmental factors play a partial role, accounting for 18% of influence. The most important factor appears to be non-shared environmental influences, which account for 82% of influence.

Table 3.5 Sibling intraclass correlations for hopelessness

Group	Hopelessness
MZ males	.21
MZ females	.08
DZ males	06
DZ females	.25*
DZ-opposite sex	.26*
Sib-pairs males	.09
Sib-pairs females	.12
Sib-pairs opposite sex	.10

Table 3.6. Components of variance and chi-square values for univariate model fitting for hopelessness.

<del></del>	-	Males		]	<b>Temales</b>	<u> </u>	Goodness of fit tests				
Model	A <sup>2</sup> %	C <sup>2</sup> %	E <sup>2</sup> %	A <sup>2</sup> %	C <sup>2</sup> %	E <sup>2</sup> %	AIC	$\chi^2$	df	p	
1	0	15	85	0	20	80	-5.62	22.38	14	ns	
2	0	18	82	0	18	82	-11.41	22.59	17	ns	

Table 3.7 The association between depressogenic attributional style and hopelessness in young people, controlling for social risk, current parental depression, externalising problems, and young people's prior depression

	Model I: AS and			Model II: AS and Model III: Social and Model III: AS, Social						Social	Model	III: Inc	luding	Model IV: Including prior depression			
	Social risk		Familia	al risk		and Familial risk $R^2 = .08$			Externalising Problems $R^2 = .099$								
	$R^2 = .$	.076		$R^2 = .02$								$R^2 = .104$					
	b	SE	t	b	SE	t	ь	SE	t	b	SE	t	b	SE	t		
Sex	.02	.007	2.69**	.02	.01	2.51*	.02	.01	2.63**	.02	.01	3.01**	.02	.01	2.72**		
Age	00	.002	097	001	.002	4	002	.002	71	001	.002	61	002	.002	88		
Attributional style	01	.001	-8.31***				01	.001	-8.15***	01	.001	-6.54***	01	.001	-5.61***		
Social factors																	
Social adversity	.01	.004	2.01*	.01	.004	1.13	.01	.004	1.23	.01	.004	1.27	.004	.004	1.13		
Parent education	0003	.000	1 -2.25*	0003	.001	-1.86	0003	.001	-2.17*	0002	.001	-1.96	0002	.0001	-1.94		
Familial risk																	
Parent depression				.013	.01	.313	.01	.01	.83	.01	.01	.5	.004	.011	.33		
Externalising												and the second		16.4.2			
Aggression							ali di			.003	.001	2.691**	.002	.001	2.41*		
Delinquency										.003	.002	1.38	.002	.002	1.17		
Internalising																	
Depression													.001	.001	1.66		

# **Chapter 4: Discussion**

#### 4.1 Overview

This section evaluates the research findings with reference to the current body of knowledge on hopelessness. The first part describes the main findings; the second section outlines the descriptive statistics and relationship to theoretical models; the third section addresses the multivariate results. The limitations of the study are considered, and with these in mind, the strengths are also noted. Implications for future research and professional practice are outlined.

The aims of the present study were to:

- test the risk factors for hopelessness (negative life events as a proximal risk, moderated by the distal influence of a depressogenic attributional style), as posited by the hopelessness theory of depression
- 2. investigate other potential risk factors for hopelessness, in conjunction with attributional style
- examine the genetic and environmental contributions to adolescents' and young adults' hopelessness

Using a twin-sibling design, this study was the first to assess the relative contribution of nature and nurture in the risk for hopelessness in adolescents and young adults. The findings indicated that young people with a depressogenic attributional style were significantly likely to have hopelessness cognitions through a risk process that operates environmentally. In line with this, genotypic analyses revealed a substantial contribution of non-shared environment on youth hopelessness, accounting for 80%

of the variance. Shared environment accounted for the remainder of the variance (20%), with genetic influence showing no contribution to hopelessness. However, young people with hopelessness cognitions were significantly more likely to have previous depression symptoms and concurrent externalising behaviour problems, associated with hopelessness over-and-above the contribution of their attributional style.

### 4.2 Main findings

Based on the literature on hopelessness and its relationship with depression and suicidal behaviour, we hypothesised that there would be a genetic contribution to hopelessness. Contrary to the predicted hypothesis, variation in hopelessness showed no evidence of a genetic contribution, but instead showed substantial nonshared environmental influence. Univariate analyses were conducted for the hopelessness scale, incorporating tests for sex differences in the genetic and environmental contributions to the variance in the scale. Contrary to predictions, genetic factors were unimportant in explaining individual differences in hopelessness. The variance in scores was largely explained by nonshared environmental factors and measurement error, suggesting that individual-specific environmental influence is substantial. There were no gender differences in the relative contribution of each factor on hopelessness.

Showing no evidence of genetic influence in this investigation, hopelessness is in contrast with correlates such as attributional style (Lau et al., 2004), depression (Rice et al., 2002) and suicidality (Gustavsson et al., 1996b), all of which have shown some genetic component in behaviour genetics studies. Additionally, there was no

significant relationship between parent and child hopelessness. This also contrasts with research showing familiality for attributional style (Alloy et al, 1998b), depression (McGuffin & Katz, 1989) and suicidality (Gustavsson et al., 1996b). Both of these findings may indicate that genetic influence plays a role in cognitive style and psychiatric disorder.

Hopelessness has both 'state' and 'trait' features (Young et al., 1996), which may have bearing on findings in this study. This characteristic is poorly understood at present. One possibility may be a similar phenomenon to 'kindling' effects, i.e. intraorganismic changes that are brought about by the occurrence of an episode of a disorder, which have been reported for depression and other disorders (Post, 1992). Consequentially, with repeated episodes, the link between stress and depression becomes weaker. This may mean that the causal mechanisms for the first episode may not be identical to recurrences (Rutter, 2003). In the case of hopelessness, this could mean that becoming hopeless may, for example, require less negative life events in repeated episodes than at first onset. Of note, this phenomenon has also been reported for suicidal behaviour. Negative events were related to intensity and duration of suicidal crises among never- and first- attempters, but not among those with previous suicide attempts (Joiner Jr & Rudd, 2000). Capturing different conceptual features of hopelessness, such as hypothetical 'kindling' effects, would require sensitive, longitudinal investigation, to examine causes, and correlates of hopelessness cognitions over time.

Although we have failed to find evidence for a genetic role, more detailed examination may elucidate genetic influence for high hopelessness. Findings in the

depression literature indicate that genetic influence is most pronounced for early-onset cases (Rice et al., 2002) and more severe symptomatology (Gjone et al., 1996). Investigating hopelessness levels developmentally in a longitudinal study commencing in childhood, with particular attention to moderate-to-severe reports, may uncover some genetic contribution.

As mentioned earlier, this investigation found that non-shared environment was the most important contributor to hopelessness. Non-shared environment involves all components of environmental influence that are individual-specific, i.e. not shared between siblings. This may include siblings being exposed to different environments, for example, being in different school classes, having different peers. It may also incorporate more subtle non-shared components, such as experiencing different parenting styles, perhaps due to a combination of factors, such as gender or age. Individual-specific environment may also be brought about partly by genetic influence, with individuals shaping and selecting their environments through their behaviour, an example of a gene-environment correlation (Rutter et al., 1997). These experiences may contribute to cognitive features such as attributional style (Lau et al., 2004), an individual's response to stressful life events (Kendler et al., 1991) and disorders, such as depression (Rice et al., 2002).

As the most important contributor to hopelessness in this study, non-shared environmental influences seem to be reflected in the observed associations between hopelessness and both attributional style and dependent negative life events. Multivariate regression analyses revealed that factors more likely to reflect shared environment, such as social adversity and parental depression, showed no unique

contribution to hopelessness once more individual-specific variables such as attributional style and behaviour problems were considered alongside them. This finding supports the behaviour genetic findings, in that shared environment, although playing a significant role in hopelessness, accounts for only a small portion of variability.

It is surprising that, while highly correlated variables show a significant genetic component, hopelessness is influenced solely by environmental factors. One hypothesis to account for this discrepancy is that, while vulnerability factors such as cognitive style and previous depression symptoms may be genetically influenced, to develop an extreme state of hopelessness cognitions requires the added individual-specific negative life events. Dependent life events may be more likely to reflect non-shared environment, as compared with independent events, which may be more indicative of shared environment, at least as measured by the Life Events Checklist (see Appendix 6). Equally, dependent negative events may trigger more negative attributions, as they have not also happened to peers and family members. Distinguishing between those individuals who do and don't become hopeless may require longitudinal information about the nature and frequency of negative events, particularly in contrast to the life events of siblings and peers.

Related to this is the interesting finding that parent-reported aggressive behaviour shows a unique contribution to hopelessness, when social adversity, parental depressive symptoms, delinquency and prior depression are considered. In univariate analyses, parent-reported aggression and delinquency were both associated with youth hopelessness. This is consistent with the findings of Kashani et al. (1997), who

found that adolescents with high hopelessness showed more aggressive behaviour and depressive symptoms. This is also consistent with research documenting the adverse social consequences of behaviour problems (Rutter et al., 1997) and reports of high levels of comorbidity with internalising problems in young people with behaviour difficulties (Moffitt et al., 2001).

Aggression has been highlighted as a marker of more severe risk of poor outcomes generally (Moffitt, 1990a). The association between aggression and a depressogenic attributional style has several possible explanations. Firstly, it may reflect a common genetic liability for both internalising and externalising disorders (O'Connor et al., 1998), whereby young people with externalising behaviour problems are more likely to have cormorbid depression. Second, it may reflect the adverse environment of young people who use aggressive behaviour, combined with social responses to this behaviour, whereby the young person finds increasing evidence to develop a depressogenic cognitive style, perhaps in conjunction with hostile cognitions (Dodge & Frame, 1982). Such individuals may be at higher risk of experiencing a multitude of dependent negative life events, and thus more likely to become hopeless. Thirdly, this may be related to the developmental trajectory of young people with aggressive behaviour. As adolescence progresses, the increased sophistication of cognitive skills means that young people are more likely to think about and reflect on their experiences. Young people with a history of adverse experiences and negative life events, perhaps combined with poor relationships with parents and peer rejection (e.g. Rutter, 1997) may have few positive and many negative experiences to reflect upon. As a result, they could be more likely to develop internalising symptoms.

# 4.3 Hopelessness: descriptive statistics and relationship to theoretical models.

#### 4.3.1 Descriptive statistics

Hopelessness scores averaged 4.89 (SD=1.83) across the sample, which indicated that most participants reported none or low levels of hopelessness. This is comparable to adult investigations using non-clinical samples, in Ireland (4.45, SD=3.09) (Greene, 1981), France (4.83, SD=3.65) (Bouvard et al., 1992), and Finland (3.5, SD=3.1) (Haatainen et al., 2003). Adolescent and young adult nonclinical samples have reported similar self-reported hopelessness levels, for example 3.95 (SD=3.29) for USA undergraduate college students (Alloy & Clements, 1998) and 4.56 (SD=3.42) for Spanish students (Vinas Poch et al., 2004). Interestingly, a general population study in Japan reported notably higher average hopelessness scores of 8.6 (SD=3.9) (Tanaka et al., 1996), and Asian American students (5.43, SD=4.95) reported slightly higher average scores than Caucasian American (3.18, SD=3.3) (Chang, 1998). These findings suggest that cultural differences may play a role either in levels of hopelessness or reporting of hopelessness expectations. The participants involved in the current study were almost all white British (98%), so differences between cultural groups could not be explored. Of note, most non-clinical studies using adolescent and young adult samples involve USA undergraduate college students, who may be demographically quite different from a UK twin sample; however, both populations showed similar scores, suggesting that, any cultural differences may not be significant in relation to hopelessness cognitions.

### 4.3.2 Hopelessness and depression

Hopelessness was associated with self-reports of current and previous depressive symptoms. Since hopelessness is postulated as important in cognitive models of depression (Beck et al., 1974), the findings provide further validation of the Beck Hopelessness Scale. Hopelessness showed a stronger relationship with current depressive symptoms, accounting for more of the variance. This supports previous findings that levels of hopelessness are higher in those with active depressive symptoms, but that elevated levels are present in those with past depression, compared to never-depressed individuals (Young et al., 1996). As hopelessness was only investigated at one time point, it cannot be established from this study if it is a true precursor of depression, as hypothesised in the hopelessness theory of depression (Abramson et al, 1989), or part of the depressive symptomatology.

#### 4.3.3 Hopelessness and attributional style

A depressogenic attributional style predicted higher hopelessness levels. This finding is in line with the hopelessness theory (Abramson et al, 1989), which posits that a depressogenic cognitive style is a vulnerability factor for depression, fully expressed in the presence of negative life events. These finding are consistent with those of Spritio et al. (1988), who found a statistically significant correlation between the composite bad events score on the CASQ and the Hopelessness Scale for Children (Kazdin et al, 1983), modelled after Beck's Hopelessness Scale for adults (Beck et al, 1974). These relationships provide some evidence of the validity of the model. An additional consideration is that the relationship could be a result of the concept of hopelessness overlapping somewhat with the notion of attributional style, which would explain the significant association (Spirito et al., 1988).

## 4.3.4 Hopelessness and negative life events

Youth reports of dependent negative life events over the past year showed a significant association with hopelessness, whereas independent negative life events showed no relationship. Distinguishing two categories of life events is based on the work of Brown & Harris (1978), who divided life events into two types: 'dependent', i.e. events over which a person has a greater level of control, and 'independent', those which happen independently of the person's actions. This distinction has been utilised in the behaviour genetics literature, findings from which suggest that the occurrence of dependent life events show some genetic influence (Thapar, Harold & McGuffin, 1998). Dependent negative life events, in particular, have been identified as related to psychopathology (Rutter, 1997). The non-significant relationship between independent events and hopelessness may be related to the attributions made by the individual as to the cause of these events. Unlike dependent events, there may be less evidence available to the person to facilitate making negative internal, global and stable attributions about the event.

#### 4.4 Findings from multivariate analyses

Hypothesis 1: Depressogenic attributional style will moderate the relationship between negative life events and hopelessness

A depressogenic attributional style did not moderate the relationship between dependent negative life events and hopelessness. Independent negative life events showed no correlation with hopelessness. The former finding does not support the hopelessness theory of depression (Abramson et al, 1989), which describes a

negative cognitive style as a vulnerability factor for hopelessness and hopelessness depression, activated in the presence of stressful life events. A significant relationship exclusively between dependent negative life events and hopelessness may be related to an 'attributional style – stressor match' (Spangler et al., 1993). Individuals who had a match between the cognitive domains of their negative attributional style and a recently occurring negative life event exhibited higher levels of hopelessness compared with those without a match (Spangler et al., 1993). Dependent life events, likely occurring at least partly as a result of the person's own behaviour, may result in cognitions quite different from those of an independent event. These dependent event-related cognitions may provide more scope for making negative internal, global and stable attributions. For example, failing an exam may trigger thoughts of not being a clever person, whereas an independent event such loss of a job by a parent may be less likely to lead to self-critical attributions.

Another consideration here is the statistical methods used. Two assumptions are made in using a multiple regression to estimate a mediational model. First, that there's no measurement error in the mediator, and second, that the dependent variable doesn't cause the mediator (Baron & Kenny, 1986). As an internal, psychological variable, there is likely to be some measurement error in the attributional style questionnaire. Consequentially, the effect of attributional style may be underestimated and the effect of dependent negative life events on hopelessness may be overestimated (Judd & Kenny, 1981a). Also, as hopelessness was only measured at one time point, we cannot be sure that it didn't precede, and indeed cause, a depressogenic attributional style. Also, the concepts of hopelessness and depressogenic attributional style may overlap. For example, work on stable or 'trait'

hopelessness (e.g. Young et al, 1996) may be capturing cognitions that would also influence a measurement of attributional style.

Hypothesis 2: The association between depressogenic attributional style and hopelessness is accounted for by social adversity

Social adversity did not explain the relationship between having a depressogenic attributional style and hopelessness cognitions. Although social adversity is significantly associated with hopelessness, the influence of attributional style accounts for more of the variance in young people's hopelessness. This finding is also consistent with the hopelessness theory (Abramson et al, 1989), which places cognitive style as the most important vulnerability factor for hopelessness.

However, the model also revealed that social adversity, as estimated by the Social Problems Questionnaire (Corney, 1988) uniquely accounted for part of the variance in hopelessness levels at a 2-year follow-up. This supports previous findings that adverse neighbourhoods (Perez-Smith et al., 2002), poor financial circumstances, and unemployment (Haatainen et al., 2003) are associated with higher hopelessness.

Of note, a component of this measure involved satisfaction ratings of different components of social life, such as neighbourhood, finances, relationships and work. It is possible that attitudinal variables such as these may vary significantly according to mood, with less satisfaction linked to low mood.

Parental education level showed an association with hopelessness, meaning that young people from families with more educated parents were more likely to be hopeless. This was an unexpected finding, as lower levels of education have been identified as a risk factor for poor outcomes generally, and hopelessness specifically (Haatainen et al., 2003). Several possibilities may account for this finding. This sample contained a disproportionate amount of highly educated parents, with 17.5% holding a degree; this lack of variability may have resulted in an unusual result. Alternatively, this finding may reflect a real effect. One possible interpretation is that added pressure on young people from high achieving families, of whom more is expected in the late adolescent and early adulthood period, may leave them at a higher risk of hopelessness. In future studies, a composite measure of both parents' education level in conjunction with information on poverty would enable further clarification of educative and economic disadvantage, in relation to hopelessness.

Hypothesis 3: The combined effects of social adversity and current parental depression symptoms show an association with young people's hopelessness

Considering social adversity and current parental depression symptoms together, there was no significant association with hopelessness. This was a surprising finding, as it was expected that combining these factors would account for more of the variance. Depression in mothers has been linked to elevated levels of psychopathology in their offspring, including depression (e.g. Goodman & Gotlib, 2002) and antisocial behaviour (e.g. Hay et al., 2003). Although maternal depression

symptoms did show a significant correlation with hopelessness, when combined with social adversity, a greater association would have been expected.

Of note, the measure of parent depression symptoms only enquired about the two-week period preceding completion of the questionnaire. This may have resulted in a misrepresentation of parental depressive symptomatology, as there is no estimate of symptoms over an extended period of time. This would have provided a more detailed insight into the extent and duration of parent low mood. Additionally, information was only available on mood state of the parent who completed the questionnaire, so a full picture of both maternal and paternal depressive symptoms was not available for analyses.

Hypothesis 4: A depressogenic attributional style has an effect on hopelessness after controlling for social adversity and current parent depression

Depressogenic attributional style continued to show a significant association with hopelessness, when controlling for social adversity and current parent depression. Given that the measures of social adversity and current parent depression had not shown a significant association, this is perhaps an unsurprising result. Children of women with mood disorders have shown more negative attributional styles and cognitions about themselves (Hammen, 1992). Parental depression showed no unique contribution to hopelessness, when incorporating social adversity and attributional style. This finding suggests that the risk to offspring associated with parent depression may be specifically the influence it has on youngsters' cognitive style. This may be a result of both providing more depressogenic feedback related to their

child (Alloy et al, 1998b) and modelling, whereby the child learns a negative cognitive style from repeatedly hearing such cognitions in the home environment (e.g. Seligman, 1984).

Hypothesis 5: There is a relationship between externalizing behaviour problems and hopelessness, independently of social adversity, current parental depression and depressogenic attributional style

Aggression showed a unique contribution to hopelessness, when controlling for delinquency, current parental depression and attributional style. In contrast, delinquency showed no significant association with hopelessness when the other variables were included in the analyses. Aggressive behaviour has been identified as a marker of higher risk for adverse outcomes, as compared with delinquency, which during adolescence, is relatively normative (Moffitt, 1990a).

The association between aggression and hopelessness was partly influenced by attributional style. Therefore, young people with aggressive behaviour problems may be at risk of hopelessness both influenced by depressogenic attributions, but also independently of having a depressogenic attributional style. This suggests that other processes may also be operating to link aggressive behaviour to hopelessness. As noted earlier, the cumulative risk trajectory experienced by young people with aggressive behaviour, with greater psychopathology, repeated negative interactions and negative life events may in fact lead directly to hopelessness. Further studies should continue to seek other variables that might influence this relationship.

Hypothesis 6: A depressogenic attributional style predicts hopelessness, over and above the effects of social adversity, current parental depression, externalizing behaviour problems and prior depression

Depressogenic attributional style predicted hopelessness at a 2-year follow-up, over and above the effects of social adversity, current parental depression, externalising behaviour problems and prior depression. This is consistent with Abramson's (1989) hopelessness theory of depression, as it shows cognitive style as an important proximal vulnerability factor for hopelessness. With the sole exception of aggressive behaviour, no variables contributed to hopelessness independently of attributional style, although all had associations with hopelessness when considered individually. This is an interesting finding, as it suggests that the risk associated with these variables is operationalised through their effect on cognitive style.

#### 4.5 Limitations of the study

Sample

Several limitations to these findings must be noted. The first concerns the characteristics of the sample. For the initial recruitment, participants in the GENESIS study with adolescent offspring aged 12-19 were invited to participate. This may have excluded people with lower levels of literary and organisational skills, who had not remained involved with the GENESIS study. Additionally, female parents were over-represented. This meant that we could not extrapolate a full picture of family socio-economic status, as they were not asked about their partner's occupation and educational level.

The response rate also affected representativeness of the sample. In studies of this kind, biases arise in those who did and did not respond. In particular, those families where mental health concerns may have been a difficulty may have been more likely to take an interest and thus respond. Conversely, those currently suffering with depression, for example, may have found it more difficult to complete the booklets and return them. Replications would benefit from design considerations to facilitate a more representative sample, particularly with regard to involving families who may, as a result of mental health, educational and social disadvantage, opt out of postal studies.

Moreover, ranging in age from 15 to 23, the sample varied considerably in terms of developmental level; differences in cognitive development and social circumstances may differentiate the participants' reports of symptomatology. However, preliminary analyses found no significant age effects for hopelessness. Although developmental differences were not evident when the sample was analysed across age or when our results were compared with those of studies with different age-groups, further investigations are needed to confirm that developmental differences are not playing a significant role. Specifically, we didn't assess cognitive level, or subdivide the sample according to developmental stage, both of which may have yielded developmental effects.

### Research Design

There are also limitations in the twin/sibling design, partly as a result of necessary assumptions made in analysing this type of data. One assumption is that assortative mating is negligible. That is, the parents of the twin and sib pairs are no more alike in

hopelessness cognitions than random individuals. If there is selective mating, this would increase the non-MZ sibling correlation. Collecting hopelessness data from both parents could test this assumption.

Additionally, as with all twin and sibling designs, the analyses here are based on the equal environments assumption for MZ and DZ twins and full sibling pairs (Plomin et al., 1994). For example, it is assumed that twin and sibling pairs share common environment factors, such as similar family, community and social influences. It is possible that common environment is greater for MZ twins as compared with DZ's, and particularly for DZ's as compared with sibling pairs. This may be especially relevant in this sample, where the sibling pairs could vary in age as much as 7 years. If this were the case, it is possible to interpret the greater similarity of MZ twins as being due to environmental factors.

The equal environments assumption was reviewed by Bouchard & Propping (1993), who found that the assumption seems to be a reasonable one for most traits. For example, Bouchard studied MZ and DZ twins reared apart on a variety of personality measures, finding that there were little differences between MZ twins reared apart or together. He argues that shared environmental influences appear to be quite small for personality traits, and thus greater similarities for MZ twins are likely to be due to genetic factors.

Changes to the twin/sibling design would provide more robust methods of disentangling environment and genetic influences, such as including hopelessness measures for both parents. Collecting information on variables such as the similarity of each sibling's appearance and activities may offer some estimate of the extent of

environment shared by each sibling pair. Including half-siblings, step-siblings and adopted siblings into the genetic design would offer another way of looking at environmental effects.

#### Measures

The Beck Hopelessness Scale is the most widely used and statistically validated measure of hopelessness expectations. Although the scale has good sensitivity, it has not shown good specificity or positive prediction value (e.g. Beck et al., 1990). Hopelessness may be assessed with more certainty by additionally using a clinical interviewing procedure for research investigations. The advantage of this would be greater specificity regarding hopeless individuals, and hence more reliable research findings. Hopelessness symptoms were measured at a single time-point, so stability of hopelessness cognitions could not be established.

The measure of depressive symptoms was a brief self-reported questionnaire, chosen to maximise response rates. Thus, participants scoring highly could not be deemed with certainty to have reached criteria for major depressive disorder, although they would be at highly increased risk of such a diagnosis (Thapar & McGuffin, 1998). Also, as depressive symptom assessment covered only the two-week period immediately prior to the questionnaire completion, it is likely that we failed to detect episodes of depression that emerged between interviews. Further studies would benefit from the use of diagnostic interviews to examine these hypotheses with regard to major depressive disorder.

The measures of aggression and delinquency used were parent-reported (of which most were mothers). Some researchers have cautioned that depressed mothers may

over-report their children's problems, which artificially inflates the statistical association between their depression and their children's behaviour problems (e.g. Boyle & Pickles, 1997). Obtaining reports from fathers and an independent informant, such as the young people's school teachers, would provide more detailed information on this.

As all data were self-reported, a negative mood bias cannot be discounted. The validity of self-report measures has been criticised because of affective bias (Atkinson et al., 1997). However, a more recent investigation suggests that internalising problems, including negative cognitions and depression can be most accurately assessed through self-report (e.g. Merrell, McClun, Kempf & Lund, 2002). Data was collected at two time points, but no information was available between those time points. This means that episodes of psychiatric disorder, for example, may not be incorporated in our dataset, hence leading to less accurate findings.

### Results

These results are limited to a single, contemporary cohort of British adolescents and young adults. Although rates of youth hopelessness found with this UK sample match rates from US (Alloy & Clements, 1998) and Spanish (Vinas Poch et al., 2004) surveys, further studies are required to determine whether these results are generalisable to other times and places.

Researchers have indicated that rates of depression may be higher in mothers of twins than of singletons (Thorpe et al, 1991). Information on lifetime prevalence of

maternal depression was not requested in this study, to compare twin and sibling mothers. Therefore, we cannot be sure that the relationships observed here between parent depression and offspring hopelessness would apply to families with singletons.

The identification of potential risk factors for hopelessness in no way ensures their causal status (Kraemer et al., 1997). Hopelessness was only investigated at one time point; thus, the observed associations with externalising and internalising problems, although reported at a previous time point, could not be ruled out as consequences of hopelessness. Further research is needed to determine whether changes in these risk factors would decrease the likelihood of hopelessness, thus implying their causal status.

Additionally, the measures of hopelessness, depressive symptoms and negative life events were very positively skewed in this dataset. There was thus little variance in levels of hopelessness for the independent variables to explain. This reduced the power of the statistical tests and probably underestimated the magnitude of the various relationships. Although logarithm transformations were used, as several of the major variables used in analyses were skewed to a moderate extent, improvements of analyses with transformation may have been only marginal. Additional research would benefit from using a higher risk or clinical sample, which may offer greater variance in participants' levels of hopelessness, that could be investigated more usefully in relation to the independent variables.

This study examined hopelessness without controlling for current depression. This may be problematic, as the results may not have reflected levels of hopelessness independently of participants' concurrent depressive symptoms. Therefore, we cannot conclude that the findings regarding hopeless cognitions do not simply reflect depressive symptoms.

Finally, the vulnerability factors tested in this investigation could only explain up to 10% of the variance in hopelessness. While not unusual for this size of sample, nevertheless, most of the variance in hopelessness was unaccounted for by this study. With large samples, even very small differences between groups can become statistically significant. This does not mean that the difference has any practical or theoretical significance. To investigate the strength of associations, an effect size calculation would be appropriate with a sample of this size. Also, refinement of methodological techniques, as suggested above, plus comparison of high- and low-risk samples across longitudinal studies using path analysis techniques, may offer more insight into factors contributing to and protecting against hopelessness.

## 4.6 Strengths of the study

With these limitations in mind, the strengths of the study can be noted. The genetically sensitive longitudinal design offered several advantages. First, reporting of symptomatology was made prospectively, thus eliminating problems associated with long-term retrospective recall (Henry et al., 1994). Second, use of a community rather than clinical sample, meant that the observed associations between hopelessness and psychopathology were not caused by high-risk families selectively coming to clinical attention. Thirdly, utilising a mixed twin and sibling design

allowed examination of the relative contribution of genetic and environmental influences to the development of hopelessness. Finally, use of a large sample offered this study considerable statistical power, leading to more robust and reliable results.

#### 4.7 Directions for future research

As addressed above, methodological refinements in future investigations may offer more detailed findings. This investigation would be furthered by replication with a 'high-risk' stratified community sample. More detailed information on families' social context, along with protective factors against hopelessness, under-investigated at present (Needles & Abramson, 1990), would provide a more comprehensive picture of both risk and resilience for hopelessness.

Further research could examine thinking styles of young people with behaviour problems, particularly those with co-occurring aggressive behaviour and depression, through the pre-adolescent and adolescent periods, to further investigate depressogenic cognitive style, in addition to hostile attributional style, which has received much attention. Of particular importance is how this relates to their risk for hopelessness and suicidal behaviour, given that aggressive youths have been identified as a vulnerable group for suicidal behaviour (Kashani et al., 1997).

Clinical research may be useful for clarifying the role of hopelessness in psychopathology, by using a psychotherapeutic intervention specifically designed to reduce hopelessness. One interesting line of enquiry would be to explore 'state' and 'trait' features of hopelessness and how they operate over time, in response to life

stressors. This may provide further understanding of the risk of hopelessness in suicidality.

## 4.8 Clinical Implications

Associations found in this study between non-shared environmental factors and hopelessness have implications for clinical practice. Hopelessness, unlike other predictors of suicide, such as age, sex, or ethnicity, is a characteristic that can be modified (Beck et al., 1990). For example, depressed patients treated with cognitive therapy showed a more rapid reduction in hopelessness scores than a comparison group of depressed patients treated with an anti-depressant drug (Beck et al., 1990). Attributional style influenced the relationship between all examined risk factors and hopelessness. Thus, therapeutic interventions that target cognitive style, which have more recently been applied in a systematic way to adolescents, are appropriate, and should continue to be pursued. Additionally, when intervening with behaviour problems, attention should also be paid to depressogenic thinking styles, particularly with adolescent youth, who may be experiencing co-morbid internalising symptoms.

## 4.9 Conclusions

In summary, the present study represents an initial examination of the genetic and environmental contributions to variations in hopelessness. Non-shared environmental factors showed most influence on hopelessness, with common environment also showing some contribution. There was no evidence of a genetic contribution to hopelessness. Risk factors such as a depressogenic attributional style, co-occurring psychopathology, parental depressive symptoms and social risks all showed

individual associations with hopelessness. When considered together, aggression and attributional style offered a unique contribution to variability in hopelessness cognitions. These findings require further examination, particularly in relation to trait and state features of hopelessness within individuals over time. This study also offers support for clinical interventions targeting negative attributional style in youth who show comorbid aggressive and depressive symptoms.

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Institute of **Psychiatry** 

Ethical Committee (Research)

Research Ethics Co-ordinator Margaret M Chambers Msc

De Crespigny Park Denmark Hili London SE5 8AF Tel 020 7848 0797. Fax 020 7848 0147

P006, Room W109

Email m.chambers@iop.kcl.ac.uk



at The Maudsley

**University of London** 

## ETHICAL COMMITTEE (RESEARCH)

29 September 2003

Dr T Eley PO80 SGDP Research Centre Institute of Psychiatry

Dear Dr Eley

Warriers and Worriers: routes to adolescent depression (217/03) Re:

The Ethical Committee (Research) considered and approved the above study at its meeting on 19 September 2003.

Initial approval is given for one year. This will be extended automatically only on completion of annual progress reports on the study when requested by the EC(R). Please note that as Principal Investigator you are responsible for ensuring these reports are sent to us.

Please note that projects which have not commenced within two years of original approval must be re-submitted to the EC(R).

Any serious adverse events which occur in connection with this study should be reported to the Committee using the attached form.

Please quote Study No. 217/03 in all future correspondence.

Yours sincerely,

Margaret M Chambers

Research Ethics Coordinator

# Appendix 3 - Beck Hopelessness Scale (Beck, Weissman et al., 1974)

# How you see the future

The following are different ways that people describe how they see their future. Please put a cross in the 'false' or 'true' box to indicate whether you think each statement is true or false for you.

	. 0/80	Krie		المراد
I look forward to the future with optimism and hope		10	I don't think I will get what I really want .	
I might as well give up because I can't make things better for myself		11	When I look ahead to the future, I think I will be happier than I am now	
When things are going badly, I know they won't be bad all of the time		12	Things just won't work out the way I want them to	
I can imagine what my life will be like in 10 years time		13	I never get what I want, so it's foolish to want anything	
I have enough time to finish the things I really want to do		14	I don't think I will have any real fun in the future	
Someday, I will be good at doing the things I really care about		15	The future seems unclear and confusing to me	
I will get more of the good things in life than the average person		<u> </u>	I can look forward to more good times than bad times	
I don't have good luck, and there's no reason to think I will in the future		17	There's no use in really trying to get something I want because I probably won't get it	
All I can see ahead of me are bad things, not good things				

## Appendix 5 – Children's Attributional Style Questionnaire (Kaslow et al., 1991)

# How you react to situations

Here are some situations. Try to imagine that these situations have just happened to you. For each situation, there are also two possible reasons for why the situation might have happened. Put a cross in the box next to the most likely reason to explain why the situation happened to you.

1	You get an "A" on a test.	I am good in the subject that the test was in
2	Some people that you know say that they do not like you.	Once in a while people are mean to me
3	A good friend tells you that s/he hates you	My friend was in a bad mood that day
4	A person steals money from you.	That person is not honest
5	Your parents tell you something that you make is very good.	I am good at making some things
6	You break a glass.	I am not careful enough
7	You do a project with a group of others and it turns out badly.	I don't work well with people in that particular group
8	You make a new friend.	I am a nice person
9	You have been getting along well with your family.	I am usually easy to get along with when I am with my family
10	You get a bad mark in school / college	I am not a good student
11	You walk into a door and you get a bloody nose.	I wasn't looking where I was going
12	You have a messy room.	I did not clean my room that day
13	Your mother makes you your favourite dinner.	There are a few things that my mother will do to please me
14	A team that you are on loses a game.	The team members don't help each other when they play together
15	You do not get your chores done at home.	I was lazy that day
16	You go to an amusement park and have a good time.	I usually enjoy myself at amusement parks
17	You go to a friend's party and you fun.	Your friend usually gives good parties

# Appendix 6. Life Events Scale for Adolescents (Coddington, 1984)

# **Events in your life**

Here is a list of events that might have happened to you recently. Please put a cross in the box if the event has happened to you in the <u>past year</u>.

1	Outstanding personal achievement (special prize) . 26	Major increase in your parents' income
2	Finding an adult that really respects you	Loss of a job by your father or mother
3	Stopping the use of drugs	Hospitalisation of a brother or sister
4	Becoming involved with drugs	Birth of a brother or sister
5	Death of a close friend	Remarriage of a parent to a stepparent
6	Being hospitalised for illness or injury	Hospitalisation of a parent.
7	Being sent away from home	The death of a grandparent
8	Deciding to leave home	Marital separation of your parents
9	Becoming an adult member of a church	Divorce of your parents
10	Failing to achieve something you really wanted	The death of a brother or sister
11	Appearance in juvenile court	The death of a parent
12	Recognition for excelling in a sport or other activity .	Getting married
13	End of a problem between you and your parents 38	Getting pregnant or fathering a pregnancy
14	Start of a new problem between you and your 39 parents	Getting your first permanent job
15	Suspension from school	Getting your first summer job
16	Failing end of year exams	Being responsible for a road accident
17	Move to a new school district	Getting your first driver's license
18	Beginning the first year of GCSEs	Being invited to join a social organisation
19	Being told you are very attractive by a friend	Being accepted at the university of your choice
20	Mother beginning to work outside the home	Completing sixth form
21	A new adult moving into your home	Being told to break up with a boy/girl friend
22	Change in father's job so he has less time home 47	Finding a new boy/girl friend
23	End of a problem between your parents	Being invited by a friend to break the law
24	Start of a new problem between your parents	Breaking up with a boy/girl friend

Appendix 7: Social Problems Questionnaire (Corney, 1988)

The following questions are about different parts of your life.
The Items will ask you about your housing situation, your work situation, finances, and your relationships to friends and family. Some questions will not apply to you. Read the instructions before these questions to see if you should answer them.
Section A: Housing Please cross the box most relevant to you
Owned Rented Housing Association/Council Living in parent's home Other please specify
1. How satisfied are you with your present housing situation?
Satisfied Slightly Markedly Severely dissatisfied dissatisfied
2. Do you have any problems with your neighbours?  No Slight Marked Severe problems problems problems
Section B: Work  Do you have a job? Yes No (go to question 5)  If you have a job:
Please mark a cross in the one box most relevant to you  Manager or administrator  Professional (e.g. health; legal, teaching science)
Associate professional or technical Craft or manual related Service (e.g. travel. catering, security) Sales Plant or machine operative Unemployed Full time student Self employed Never worked
3a. How satisfied are you with your present job?  Satisfied Slightly Markedly Severely dissatisfied dissatisfied

	No Slight Marked Severe problems problems				
finances, ly to you. r them.	Do you look after a home as well as Yes No (go to Section C, finances) having a job?				
7	If you look after a home as well as having a job: 4. How satisfled are you with working and running a home?				
	Satisfied Slightly Markedly Severely dissatisfied dissatisfied				
	If you do not have a paid job:  5. How satisfied are you with not having a paid job?				
	Satisfied Slightly Markedly Severely dissatisfied dissatisfied				
	Section C: Finances				
<del></del>	This question is about your household gross (before tax) income per year including benefits (e.g. Housing Benefit, Child Benefit, etc.). Please mark a cross in the <u>one</u> box most relevant to you.				
	£15,000 £16,000- £21,000- £31,000- £41,000- £50,000 or less £20,000 £30,000 £40,000 £50,000 or more				
al, teaching,	6. Do you have any difficulties in meeting bills and other financial commitments?				
security)	No Slight Marked Severe difficulties difficulties				
	7. How satisfied are you with your financial position?				
	Satisfied Slightly Markedly Severely dissatisfied dissatisfied				