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Personality and adjustment

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ABSTRACT

FACULTY OF MEDICINE, HEALTH AND LIFE SCIENCES
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Personality and psychopathology are associated in adults (e.g. Krueger, Caspi, Moffitt, 2000). There have also been studies on continuities between early childhood characteristics such as temperament and later psychopathology (Caspi, 2000) however, it is unclear what produces this relationship. For example, personality may reflect a dimension of behaviour at the extreme of which is psychopathology (Graham & Stevenson, 1987) or personality may act as a risk factor for psychopathology (Rutter, Sillberg, O'Conner, Simonoff, 1999). These issues can be addressed using genetic methodologies, but there has been relatively little research to date. To look at this personality and adjustment were assessed in a sample of adolescent and young adult twins and their siblings (n = 609) using a personality measure specifically designed to avoid overlap with adjustment measures. While genetic and environmental factors contributed to variation in both personality and adjustment measures, genetic factors accounted for the majority of variation in the relationship between personality and adjustment. Findings of common genetic influences on personality and adjustment are consistent with Graham & Stevenson's (1987) suggestion that variation in adjustment may reflect extremes in personality.

Table of contents **Page**

List of tables **Page**

List of figures **Page**

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CHAPTER ONE
REVIEW OF THE LITERATURE

1.1 TEMPERAMENT AND PERSONALITY

Definitions of temperament and personality

Personality refers to general behavioural styles, including social presentation of self, attitudes, beliefs, and emotional expression. However, a distinction is made between early child temperament and more adult-like personality characteristics. Temperament can be defined as individual differences in behavioural regulation. These are present from birth, and assumed to be biologically based (Thomas, Chess, Birch, Hertzig, Korn, 1964). Personality is seen as more differentiated than temperament, including non-behavioural aspects such as plans and intentions (Rutter, 1987) and until recently temperament and personality have been treated as two separate research areas (Plomin, Defries, McLearn, Rutter,1997; Digman & Shmelyov, 1996).

Theoretical models of personality

There have been several different approaches to personality. These include attachment, social learning, psychoanalytic, phenomenogical self-concept, cognitive, and trait theories (see Rutter,1987; McAdams, 1994; Strelau, 2001). Trait theories are the most relevant to understanding the relationship between personality and psychopathology, within a genetic methodology. This approach views personality in terms of a set of basic behavioural dimensions (e.g. extraversion, neuroticism) which are assumed to reflect underlying biological influences on behavioural variation, and be relatively stable over time and situations. Traits are usually conceptualised in bipolar terms (e.g. most anxious to least anxious) with individual differences situated along a normally distributed continuum for each trait (McAdams, 1994).

1.1.2 TEMPERAMENT

Contemporary research into temperament was initiated by Thomas and Chess in the New York Longitudinal Study, which started in the 1950's (Rutter, 1987). At that time, there

were two main theoretical approaches to psychological function. Individual differences were assumed to be directly due to differences in underlying physiology, or alternatively, as differences in responses which were directly due to different environmental experiences. Both approaches were assumed to be mutually exclusive and there was little emphasis on the relationship between child and environmental characteristics (Thomas, et al, 1964).

Development model

Thomas, et al (1964) suggested that both child, and environmental characteristics, contributed to personality development. Therefore the aim of the New York study was to identify the relationship between early behavioural variation, and later psychological organisation. This was done by assessing the consistency of behavioural characteristics from infancy onwards, and addressed two questions; can infants be distinguished on the basis of behavioural regulation tendencies? , and is there a relationship between early and later behaviour?

New York Longitudinal Study (Thomas, et al, 1964: Thomas, Chess, Birch, 1968)

The sample consisted of 140 infants, 133 of which have been followed into adult life. For a subset of the sample detailed behavioural characteristics were obtained through interviews with parents. The validity of parent reports was confirmed through comparison to observational data, and information regarding environmental factors was also obtained for each child. Content analysis of this information led to the development of nine temperament categories (activity level, rhythmicity, approach/withdraw, adaptability, intensity, threshold, mood, distractibility, persistence). Behaviour was rated at regular intervals from infancy onwards with each temperament category assessed on a three-point scale (high, medium, low) (see table 1.1.2.1).

Figure 1.1.2.1 NYLS Temperament categories

Activity level (general level of motor activity, including diurnal periods of activity and inactivity, and motor activity across bathing, eating, playing, etc, as well as information regarding the sleep-wake cycle, reaching, crawling, walking).
Rhymicity (the extent the behaviours were predictable).
Approach or withdrawal (the nature of responses to novel stimuli, including objects, foods, and people).
Adaptability how frequently responses to novel or altered stimuli were successfully adjusted)
Intensity of reaction (the energy level of responses, irrespective of quality or direction)
Threshold of responsiveness (the intensity level of sensory stimulation, or stimulation from objects and social contact needed to evoke a behavioural response)
Quality of mood (the amount of pleasant, joyful, friendly behaviour compared to upleasant, crying and unfriendly behaviour)
Distractability (the extent external stimuli interfered with or altered the direction of ongoing behaviour)
Attention span and persistence(the length of time an activity is pursued by the child, attention span, and continuation of the activity despite obstruction, persistence)

Thomas, Chess, et al, 1964

For each child temperament was characterised by the most frequent rating for each category over time. The children could be classified into three broad groups, 'easy' (rhythmic, i.e. regular sleeping and eating patterns, friendly, adaptable and easy to distract) 'difficult' (irregular sleeping and eating patterns, irritable, inflexible, intense reactions to new situations, and hard to distract) and 'slow to warm up' (low activity, mild withdrawal from new situations and slower to adapt).

Evidence was consistent with the model of development suggested by Thomas, et al (1964). There was a relationship between temperament categories and environmental responses. The children showed different behavioural responses to the same environmental influences, which made them relatively easy or difficult to parent. This implies a reciprocal relationship between child characteristics and environmental effects (Thomas, et al, 1964).

The measures developed by Thomas and Chess have been widely used in research, and influential on the subsequent development of other models (Digman & Shmelyov, 1996). However, there have been some criticisms.

Maternal reports of temperament

Temperament categories were derived from maternal reports of infant behaviour, and there are issues relating to the extent that maternal reports of infant temperament accurately

reflect characteristics of the child. For example, evidence shows a) differences between maternal and observational ratings (Seiffer, Sameroff, Basset, Kratchuk, 1994) b) that maternal ratings correspond more to the mother's own characteristics than the child's (Manseldorf, Gunner, Kestenbaum, Lang, Andreas, 1990) and c) that child temperament can be predicted by maternal perceptions in pregnancy (see Graham & Stevenson, 1987; Belsky, Hsieh, Cornic, 1998).

However, these studies were based on questionnaire data, and not on the methods used by Thomas,et al, 1964 (see Graham & Stevenson, 1987). Stable behavioural variation in infants has been shown using behavioural measures of stress responses e.g. physiological coping resources involving the hypothalamic-pituatary-adrenal axis, the sympathetic nervous system, the neurotransmitter system, and the immune system, which are likely to be enduring and related to temperament and environmental experience (see Glaser, 2000 p 103; Boyce, Barr & Zeltzer, 1992). For example, Lewis (1992) used three dimensions, threshold (intensity of stimulation required to produce a stress response) dampening (ability to inhibit the stress response) and reactivation (ability to become aroused following dampening) to capture variation in stress responses. Threshold and dampening measures were stable from birth to two months, and this was more pronounced in highly reactive infants.

In addition, differences between maternal ratings and ratings by other informants may not just be due to measurement error as a) correspondence between maternal perceptions in pregnancy and child temperament may reflect similarity in personality characteristics due to genetic relatedness, and b) evidence suggests that lack of convergence between different informants may largely reflect the fact that they are reporting on different aspects of the child's behaviour. For example, Presley & Martin (1994) directly compared parent and teacher ratings of items from The Temperament Assessment Battery for Children (TABC) for over 2000 children aged between three and seven years. This inventory has six scales designed to assess activity level, adaptability, approach/withdrawal, emotional intensity, distractibility, and persistence. Items for parent and teacher report scales are selected to tap similar items, except for distractibility where items on the parent form assess the extent distraction can be used to divert the child from inappropriate behaviour, and items on the teacher form assess the interruption of attention from noise/other children etc. Factor analysis showed that items from parent reports loaded onto five factors, however, items

from teacher reports loaded onto three factors (see figure 1.1.2.2). This suggested that situational factors may contribute to differences between ratings by different informants, rather than lack of agreement on the same behavioural aspects of the child (see Presley & Martin, 1994).

Figure 1.1.2.2 Factor solutions of parent and teacher ratings of temperament

	Factors identified from TABC items
Parent ratings	Social inhibition
	Negative emotion
	Adaptability
	Activity
	Persistence
Teacher ratings	Persistence
	Inhibition
	Negative emotionality

(adapted from Presley & Martin, 1994)

Validation of temperament categories

Factor analysis of category items from the NYLS has not shown the expected pattern of distinct clusters for the category items (Buss & Plomin, 1984; Graham & Stevenson, 1987). For example, a meta-analysis of the factor structure across middle childhood, found evidence for only seven dimensions, five of which did correspond to the original categories (see Presley & Martin, 1994).

Broad Trait model

A broader trait based approach to temperament was suggested by Buss & Plomin (1984). Three temperament traits, (activity, sociability, and emotionality) were identified through factor analysis of stable characteristics that were present in the first year (see figure 1.1.2.3).

Figure 1.1.2.3 EAS temperament traits

Activity (consisting of two highly correlated aspects; vigour, which refers to intensity or amplitude of behaviour, and tempo, which refers to pace of behaviour).
Sociability (the extent the child prefers closeness, attention, responsiveness and social stimulation).
Emotionality (the tendency to become physiologically aroused easily, and intensely, in upsetting situations).

(Buss & Plomin, 1984)

These measures correspond to more adult-like personality traits (Shiner, 1998) and form the basis of the Emotionality, Activity, and Sociability (EAS) Temperament Survey, with separate versions for children and adults. Measures have been validated in children aged between one and nine years, and need to be validated in older children and adolescents, and across different samples (Mathiesen & Tambs, 1999; Boer & Westenberg, 1994; Gibbs, Reeves & Cunningham, 1987; Shiner, 1998). A fourth trait of impulsivity was not included in the EAS Temperament Survey, due to overlap with the activity factor, however, more recently evidence of a separate genetic influence on impulsivity has been found (Shiner, 1998, Carver & Scheier, 2000).

In support of the model there is some agreement in the literature that emotionality, sociability, and activity, are important temperament dimensions as some aspects of these traits are identified across several different models of temperament and adult personality (see Rutter, 1987). For example, factor analysis of different temperament scales has identified fear, irritability/anger, positive affect/approach, activity level, attention, and persistence as main dimensions (Sanson & Rothbart, 1998). There is also evidence from a meta-analysis that implies different temperament scales are measuring the same basic constructs (Rothbart & Bates, 1998).

1.1.3 PERSONALITY

There are several trait models of personality, with some dispute over whether cognitive factors like intelligence, cognitive processing, and motivation form part of personality, and the number of traits needed to capture personality adequately (Rutter, 1987; Eysenck, 1990).

Five-factor model

The five-factor model is predominant in the trait approach. The NEO Personality Inventory (Costa & McCrae, 1992) was based on factor analysis of natural language trait terms. The model suggests personality is hierarchically organised, with five main traits (extraversion, agreeableness, conscientiousness, neuroticism, and openess) each consisting of six minor traits (see figure 1.1.3.1).

Figure 1.1.3.1 NEO personality traits

Extraversion (active, assertive, energetic, enthusiastic, outgoing, talkative)
Agreeableness (appreciative, forgiving, generous, kind, sympathetic, trusting)
Conscientiousness (efficient, organised, planful, reliable, responsible, thorough)
Neuroticism (anxious, self-pitying, tense, touchy, unstable, worrying)
Openness (artistic, curious, imaginative, insightful, original, wide interests)

(Costa & McCrae, 1992)

There is quite wide empirical support for the model. The five factors have been found either singly, or in combination, in the majority of broad spectrum personality inventories (Costa & McCrae, 1992). The NEO-PI Inventory has also been validated with adults across several languages (McCrae & John, 1991).

However, there are indications that the phenotypic structure of the five factor model may not accurately reflect underlying biological variation. There is some evidence of overlap where alcohol and anti-anxiety drugs produce lower scores for both neuroticism and introversion, implying a shared biological origin (see, Cloninger, Sravkic, Przybeck,1993).

Psychobiological model

The psychobiological model (Cloninger, et al, 1993; Cloninger, 1994) is a genetic model of personality structure, and development. Personality is defined as learning or adaptation, with complex information processing and behavioural regulation gradually emerging from more basic processes. Personality dimensions are related to the functional organisation of brain, learning and memory systems. Based on neuroanatomical evidence and comparison of the phylogeny of learning across species, the model suggests two main learning and memory systems. The first is the perceptual learning system (automatic or non-conscious sensory, motivational and associative processes) which underlies emotion regulation, reflected in four temperament dimensions. The second is the conceptual learning system (conscious or aware processing like logic, construction, evaluation based on conceptual representations of information) which underlies insight learning, reflected in three character dimensions (see Cloninger, 1994: Cloninger, et al, 1993) (see figures 1.1.3.2 and 1.1.3.3).

This model suggests personality development arises through intensive integration across temperament and character dimensions. For example, harm avoidance interacts with

novelty seeking and reward dependence by inhibiting responses to novel stimuli and social attachments (Svrakic, Svrakic, Cloninger, 1996, p 256).

Temperament traits are manifest in early life and stable across development (Sigvarsson, Bohman, Cloninger, 1987: Svrakic, et al, 1996). Character traits mature in adulthood and are influenced by both temperament dimensions and systematic socio-cultural factors (Cloninger, Svrakic, Svrakic,1997).

Two different personality inventories have been developed from this model, the Tridimensional Personality Questionnaire (TPQ) and the Temperament and Character Inventory (TCI). A modified version of the TCI has been developed for child self-report (see Luby, Svrakic, McCallum, Przybeck, Cloninger, 1999). Factor analysis has confirmed measures in adults and children, with traits normally distributed in representative samples and different cultures (see Cloninger, et al ,1993: Sigvardsson, et al, 1987: Heath, Cloninger, Martin, 1994: Stallings, Hewitt, Cloninger, Heath, Eaves, 1996: DeFruyt, DeWiele, van Heeringen, 2000).

Figure 1.1.3.2 Temperament and character dimensions

Temperament dimensions	High	Low
Harm avoidance	Pessimistic, Fearful Shy, Fatigable	Optimistic, Daring Outgoing, Energetic
Novelty seeking	Exploratory, Impulsive Extravagant, Irritible	Reserved, Deliberate Thrifty, Stoical
Reward dependence	Sentimental, Open Warm, Appreciative	Detached, Aloof Cold,Independent
Persistence	Industrious, Determined Enthusiastic, Perfectionist	Lazy, Spoiled Underachiever Pragmatist
Character dimensions		
Self-directedness	Responsible, Purposeful, Resourceful Self-accepted, Disciplined	Blaming, Aimless Inept, Vain Undisciplined
Cooperativeness	Tender-hearted, Emphatic,Helpful, Compassionate Principled	Intolerant, Insensitive, Hostile,Revengeful, Opportunistic
Self-transcendent	Spontaneous, Intuitive Acquiesent, Spiritual Idealistic	Contrived, Unimaginative Controlling,Materialistic Conventional

Figure 1.1.3.3 Model structure

Personality dimensions	Functional organisation of brain systems, which underlie learning and memory	
Temperament		
Novelty seeking	Behavioural activation system	(dopaminergic activity)
Harm avoidance	Behavioural inhibition system	(serotonergic activity)
Reward dependence	Behavioural dependence system	(noradrenergic activity) (links BAS/BIS)
Persistence	Behavioural maintenance system	
Character		
Self-directedness	conceptual system	hippocampal formation and cerebral neocortex
Co-operativeness		
Self-transcendence		

1.1.4 STABILITY OF TEMPERAMENT AND PERSONALITY

Studies of stability over infancy

NYLS findings

Evidence from the New York Longitudinal Study showed that behavioural differences were stable from infancy until two years. Stability was assessed both in terms of cross-time frequency of ratings for each category, also by the frequency of agreement between ratings in any two periods. In addition, for each child it was predicted that the most frequent rating for each temperament category would be the same at time one and time five (see figure 1.1.4.1). Children with atypical scores showed the same pattern of age to age stability as the whole group (see figure 1.1.4.2).

Figure 1.1.4.1 Stability of temperament - Category scale point at time five predicted by scale point at time one

Temperament category	Total	Confirmed	Inaccurate	Binomial test
Activity	62	24	38	0.8*
Rhythmicity	71	49	22	6.2
Adaptability	75	66	9	9.9
Approach	67	50	17	6.1
Threshold	72	42	30	4.4
Intensity	73	50	23	6.3
Mood	67	44	23	5.6
Distractability	61	45	16	6.5
Persistence	63	51	12	8.0

All significantly different to chance at $p < 0.001$, *except activity level
(Thomas, Chess, 1964)

Figure 1.1.4.2 Stability of atypical temperament measures from birth to two years

Temperament	Percentage stable
Low activity	14.3
Irregular	11.0
Nonadaptive	12.5
Withdrawal	27.3
High threshold	69.2
Intense	77.7
Negative mood	52.9
Nondistractable	64.7
Nonpersistent	40.0

(Thomas & Chess, 1964)

As well as looking at the most frequent or typical responses for each category, the hierarchical order of the number of items scored in each scale point for each category was analysed in a ranking model, which showed the same pattern of constancy for the majority of the children. There was though some variation in the degree to which children showed consistency across each category, and rhythmicity, adaptability, threshold and intensity were the most consistent characteristics over time (see figure 1.1.4.3).

Figure 1.1.4.3 Rank order stability of temperament, (Friedman 2-way analysis of variance)

Temperament category	Percentage of children with interperiod stability
Activity	27.5
Rhymicity	65.0
Adaptability	83.8
Approach	81.2
Threshold	41.2
Intensity	87.5
Mood	92.5
Distractability	36.2
Persistence	65.0

(Thomas & Chess, 1964)

Mathiesen & Tambs (1999)

The same pattern of stability and some normative change was found for measures of temperament using the EAS scales, in a large sample of Norwegian children, aged 18, 30 and 50 months. The factor structure was the same for each age group, however, with increasing age, scores for activity and sociability decreased, and scores for emotionality and shyness increased (Mathiesen & Tambs, 1999).

Lemery, Goldsmith, Klinnert, Mrazek (1999)

Lemery, et al (1999) obtained maternal ratings of four temperament dimensions (positive emotionality, fear, distress-anger, and activity) for 180 infants at 3,6,12,18,24,36, and 48 months. For infants (3 - 18 months) the relationship between measures of temperament at time one and time three depended on measures of temperament at time two, suggesting progressive change. For toddlers/preschool children (24 - 48 months) factor loadings were similar at different time points implying stability. Overall, findings were similar for each of the four dimensions, for example for three to six months correlations were between 0.55 and 0.59, however, over time continuity was stronger for distress-anger and weaker for activity level, and for 24 to 48 months correlations were around 0.70. This is in line with evidence from several large research projects that stability of temperament measures increase from infancy onwards, and become much more consistent by two years (Lemery, Goldsmith, 1999). Although, for broad temperament groupings measures of activity and startle reactivity at one month predicted category membership at three and a half years (Askan, Goldsmith, Snider, Essex, Clark, Hyde, et al, 1999).

Developmental context of change

Developmental change makes it difficult to compare earlier and later personality (Sigvardsson, et al, 1987; Rutter, 1987; Svrakic, et al, 1996) and a key issue in the literature is whether the temperament characteristics identified in children are related to personality characteristics in adulthood (Nigg, 2000). For example, with increases in age there are increases in motor skills, language, cognition, self-awareness, and self concept. By two years of age children are usually self-aware, express shame, have an understanding that the 'self' is separate from other people, and use verbal communication (Svrakic, et al, 1996). In particular cognition becomes more flexible and adult-like at around five to seven years, and this improved executive function appears to be related to increased myelination of the frontal lobes, and more co-ordinated electrical activity in the brain (Janowsky & Carper, 1995; Thatcher, 1994).

Shiner (1998) suggests this allows a wider range of personality characteristics to be displayed, and possibly an increased ability to self-regulate emotion, which creates more differentiation, and hierarchical integration of temperament. Therefore, the expression of traits in infancy may not relate to the expression of traits in middle childhood and adolescence, as some behaviours may not be able to be expressed at an earlier age. Consistent with this statistical modelling has shown that temperament becomes more stable at an age associated with the development of emotional self-regulation and language (Lemery, Goldsmith, Klinnert, Mrazek,1999).

In summary there is a meaningful relationship between measures of early and later temperament. This relationship becomes stronger over time, consistent with an increase in cognitive abilities during development. Patterns are the same for children with scores in the normal and extreme range.

Studies of stability over the lifespan

There have been several reviews of rank-order stability which show a convergent pattern of relatively high trait consistency over time (see Roberts & Delvecchio, 2000). A recent meta-analysis by Roberts & Delvecchio (2000) focused more specifically on age and trait

consistency by using data from longitudinal studies on temperament and personality, and categorising test-retest correlations into age ranges associated with developmental changes.

Overall, evidence suggested increasing stability of traits over time. Test-retest correlations were approximately 0.31 in childhood, 0.54 during late adolescence and early adulthood, 0.64 at age 30, and 0.74 between age 50 and 70 years. However, increases showed a step-wise pattern, with distinct changes in the strength of test-retest correlations in early childhood, young adulthood and later middle-age. In early childhood, consistency was 0.35, between 0 and 2.9 years, and 0.52 between 3 and 5.9 years. Estimates were similar up to young adulthood, between 22 to 29 years, consistency was 0.57, and in later middle age (between 50 - 59 years) consistency was 0.74.

Findings are consistent with evidence that correlations in traits at different times are higher for shorter periods between testing. Therefore Roberts and DelVecchio suggest that temperament stability may have been underestimated in the review, as different measures were used at different ages, and the period between testing was at least a year in each study.

Dunedin multidisciplinary Health & Development Study

A prospective longitudinal study of a New Zealand birth cohort (Dunedin Multidisciplinary Health and Development Study) showed that temperament category at age three years predict personality structure at age eighteen years (Caspi, 2000). At three, cognitive and motor skills were assessed, and temperament ratings were based on items from the Bayley Infant Behaviour Record. Following cluster analysis of behavioural ratings three main temperament categories were identified for analysis ('undercontrolled', 'inhibited', and 'well-adjusted'). At age eighteen, personality was assessed by the MultiDimensional Personality Questionnaire. Children rated as 'undercontrolled' at three had low scores on measures related to constraint (indexes traditionalism, harm avoidance, self-control) and high scores related to negative emotionality (indexes aggression, alienation, stress reaction) at eighteen. Children rated as 'inhibited' at three, had high scores on measures related to constraint, and low scores on measures related to positive emotionality (indexes achievement, social potency, well-being, social closeness) at eighteen. Children rated as 'well-adjusted' at three, were well adjusted at eighteen.

Moreover, self-report measures of personality at eighteen, corresponded to personality ratings by someone who knew the individual well at age twenty-one (Caspi, 2000).

Individual variation

There is evidence of individual variation in stability of personality, with some individuals, showing much more consistency across time than others (Thomas, et al, 1968; Roberts & DelVecchio, 2000; Robins, Fraley, Roberts, Trzesniewski, 2001). This may be related to emotional stability (Roberts & DelVecchio, (2000) as general population samples have shown more consistency than clinical samples (Schuerger, Zarella, Hotz, 1989).

In summary personality remains consistent across time, with relationships between measures in early childhood to late adulthood. These relationships become stronger over time. However, there is also individual variation with some individuals showing less consistency than others.

1.2 PSYCHOPATHOLOGY

Psychopathology can be broadly defined as psychological disturbance, which includes both clinical disorders, and psychologically disturbed behaviour, (Raine, 1993, p2). It has been conceptualised differently within psychiatric and psychological approaches. The psychiatric approach assumes disordered behaviour is qualitatively different to normal behaviour and can be classified on a categorical basis. The psychological approach assumes disordered behaviour is quantitatively different to normal behaviour and can be classified on a dimensional basis.

1.2.1 PSYCHIATRIC APPROACH

The psychiatric approach is based on a medical model of disease. Disorder is defined and classified on a categorical basis within clinically derived classification systems. The two major systems are the American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders, (DSM IV-R), (American Psychiatric Association, 1994), and The Tenth Revision of the International Classification of Diseases, (ICD-10), (World Health Organisation, 1992).

Diagnostic criteria, is based on several factors within the systems. For example, DSM-IV-R, criteria are classified along five axes. Clinical disorders are assessed by Axis I, and mental retardation and personality disorders are assessed by Axis II. Within both axes, main categories of disorders are subdivided to distinguish subtypes. Medical history is assessed by Axis III, psychosocial and environmental factors are assessed by Axis IV, and overall level of functioning by Axis V. Disorders normally diagnosed in infancy, childhood, and adolescence are: mental retardation, learning disorders, motor skills disorders, communication disorders, pervasive development disorders, separation anxiety disorder, attention deficit and disruptive behaviour disorders, feeding and eating disorders of infancy and early childhood, tic disorder, elimination disorder, and other disorders of infancy, childhood, or adolescence. However, the classification systems were developed to increase clinical efficiency and there are issues regarding diagnostic categories, boundaries between normal and abnormal behaviour, heterogeneity of symptoms, comorbidity, and distinctiveness of Axis I and II disorders.

Diagnostic categories and the underlying structure of disorder

Categories are atheoretical as they were based on clinical descriptions of behavioural symptoms, rather than theoretical considerations regarding the underlying ontological or latent structure of disorder (the underlying patterns of neurological, anatomical, biochemical, and environmental factors which actually produce disorder). This means there may be no relationship between behavioural symptom clusters and the structural level of disorder (Raine, 1993; Sonuga-Barke, 1998) (Nigg & Goldsmith, 1998). However, Sameroff (2000) suggests that there has been an assumption that classification systems reflect reality, which has led to the use of evidence of clinical validity to address issues regarding the ontological status of disorders. Also, that this has implications where it is assumed that identifiable causal factors underlie disease categories, that the same causal factors produce the same disorder, that similar symptoms at different development stages are reflecting the same disorder, and that there is specificity in disorder from child to adulthood.

The boundaries between normal and abnormal behaviour

Categorisation implies discontinuity between normal and abnormal psychological function, although, evidence of non-linearity of symptom severity and clinical aetiological factors is needed to imply categorical distinctions at the structural level (see Caron & Rutter, 1991; Widiger & Clark, 2000).

Heterogeneity and co-morbidity

Two phenomena suggest that diagnostic categories may not reflect the underlying structure of disorder. There is evidence of heterogeneity of symptoms with a lot of variation in within category defining features, and symptom clusters that fall between categories (Clark, Watson, Reynolds, 1995) which, suggests that categories are too broad, and may not be distinguishing between symptom clusters, which have different underlying aetiologies (Sonuga-Barke, 1998). In addition, epidemiological studies show that comorbidity rates are very high, and not accounted for by the base rates of separate disorders, which suggests that there may be aetiologically distinct comorbid groups (Caron & Rutter, 1991).

Caron & Rutter (1991) suggest comorbidity could be due to a) referral bias b) overlap in category symptoms c) artificial sub-division of syndromes d) the fact that symptoms of one disorder represent early manifestations, or are secondary to another or e) where disorder acts as a risk factor for another. In particular, a meta-analysis by Angold, Costello & Erkanli (1999) concludes that a) evidence to date suggests methodological factors are not contributing to comorbidy rates as possible methodological explanations do not account for all combinations of disorders, and b) that there is evidence for categorical boundaries within child psychopathology (Angold, et al, 1999, p 78).

Distinctiveness of Axis I and Axis II disorders

There is evidence that the distinction made between Axis I and Axis II disorders may not reflect the true underlying structure of disorder. For example, some Axis I disorders (dysthymia and generalised social anxiety) have an early onset, are chronic, and hard to distinguish from personality disorders, which are characterised in trait terms, and assumed

to have a continuous influence on behaviour without remission (Widiger & Trull, 1992). Moreover, schizotypal, borderline and avoidant personality disorders can be conceptualised as variants of Axis I disorders, and there is evidence of abnormalities in the personalities of individuals with Axis I disorders (see Widiger & Trull, 1992). This lack of distinctiveness between Axis I and Axis II disorders suggests it may be difficult to distinguish between some symptom clusters and enduring behavioural traits.

This has implications where there has been less research on child disorders (Nigg & Goldsmith, 1998). In particular, behaviour is more diffuse in younger children, and developmental change means that symptoms may be expressed differently at different ages. So there is less diagnostic specificity in children and if broad diagnostic groupings are used as operational definitions, groups may contain children with different underlying disorders. There are also issues regarding the suitability of current diagnostic systems for very young children (see Zeanah, Boris, Scheeringa,1997).

Acknowledgement of problems within the psychiatric approach means that prototypic categorisation has become increasingly influential. For example, rather than just using diagnostic criteria, diagnosis is based on the extent that symptoms correspond to a category exemplar or 'prototype' of symptoms (Sonuga-Barke, 1998). So individuals with the same diagnosis may differ in both the presenting symptoms and possibly in the underlying etiology (Raine, 1993). However, this has implications for operational definitions used in research where accurate phenotypes are needed (Nigg & Goldsmith, 1998; Krueger, et al, 2000). Therefore, it has been suggested that a more conceptually based approach to classification is needed, where rather than assuming diagnostic categories exist, they are treated as hypothetical constructs, and empirically tested (see Meehl, 1995; Sonuga-Barke, 1998).

1.2.2 PSYCHOLOGICAL APPROACH

The psychological approach conceptualises psychopathology differently to that in psychiatry. It assumes continuity between normal and disordered behaviour, with disorder just representing extremes of the normal range of behaviour. In line with this, evidence shows that dimensional behavioural rating scales do differentiate between internalising and externalising behaviour problems. For example, The Child Behaviour Checklist (CBCL)

(Achenbach, 1991) was developed from clinical and normal samples, and has been widely used in epidemiological, developmental and clinical research (Goodman & Scott, 1999). In addition, factor analysis of items separates out internalising and externalising behavioural problems for adults, showing four broad dimensions of psychopathology, a) affective disorder, b) anxiety disorder (internalising) c) substance abuse disorder, and d) conduct disorder (externalising) (Krueger, et al, 2000).

1.2.3 COMPARISON OF CATEGORICAL AND DIMENSIONAL MEASURES

Categorical and dimensional measures have been directly compared within the same samples. Several studies have found a relationship between Child Behaviour Checklist scores and structured diagnosis criteria (see Jensen & Watanabe, 1999). However, it is not clear if caseness based on dimensional measures is equivalent to caseness based on diagnostic classification. For example, dimensional measures do not provide information about how enduring symptoms are, or if they are associated with significant impairment (Jensen & Watanabe, 1999).

In order to address this Jensen & Watanabe (1999) directly compared scores on the CBCL and diagnosis based on DSM criteria by looking at four groups of children. These were classified as true-positive (children who met criteria for DSM diagnosis and had high scores on the CBCL) true negative (children who did not meet criteria for either assessment method) and children who met criteria for only one assessment method (false positive; high CBCL scores only, or false negative; DSM criteria only). Children in the true-positive group differed significantly from children in the false-positive group in terms of developmental delay and use of mental health services (Jensen & Watanabe, 1999) which implies CBCL measures are less strongly associated with external validators of clinical severity. However, CBCL scores were significantly different between the four groups implying that CBLC and diagnostic criteria measures were comparable. In particular high scores on the CBLC, but no DSM diagnosis, was related to greater impairment (difficulties with concentration, contact with school counsellor) than low scores on the CBLC and no diagnosis.

This implies quantitative rather than qualitative differences between disorder classified by dimensional and categorical measures. Consistent with this, almost half the children from

the Australian Temperament Project identified by dimensional measures of behaviour received a clinical diagnosis, and at risk children as a group (ignoring diagnostic status) were more disadvantaged than control children (Prior, Sanson, Smart, Oberklaid, 1999).

1.2.4 STABILITY OF PSYCHOPATHOLOGY

Evidence suggests a relationship between early and later psychopathology. This will be discussed first in relation to associations between psychopathology found at different ages during the childhood years and then secondly into adulthood.

Stability of psychopathology in childhood

Numerous studies have shown that early behavioural problems predict later psychopathology (see Schmitz, Fulker, Plomin, Zahn-Waxler, Emde, DeFries, 1999 p334). For example, regulatory disorders in infancy (difficulties in emotion regulation, sensorimotor processing, behaviour problems) have been associated with behaviour problems at four years (DeGangi, Borges, Sickel, Greenspan, 1993). Behaviour problems in pre-school children predict behaviour problems at school age (Campbell, 1995) and show similar patterns of stability in normal and clinical samples (Deater-Deckard, Dodge, Bates, Petit, 1998). Emotional and behaviour problems at age seven predict serious emotional disturbance in adolescence (Costello, Angold, Keeler, 1999). There is diagnostic continuity for ADHD symptoms for children and adolescents across multiple measures (Biederman, Faraone, Taylor, Sienna, Williamson, Fine, 1998) and adolescent bipolar disorder has been associated with child behaviour problems and substance abuse (Carlson,Bromet, Sieves, 2000). In particular evidence from the Dunedin study showed continuity in externalising behaviours across both parent and teacher measures at 5, 7, 9, 11, and 15 years (Caspi, 2000).

Stability of psychopathology into adulthood

There is evidence of a relationship between early psychopathology and psychopathology in adulthood. Early behaviour problems are associated with an increased risk of adult psychopathology, and dimensional measures showed rank order stability for internalising and externalising problems in young adults between age eighteen and twenty-one years

(Krueger, Caspi, Moffitt, Silva, McGee, 1996). In particular, a review of studies by Zeitlin (2000) suggests approximately 70% of children and adolescents with depressed mood, or psychotic disorders, show depressed mood or psychosis in adulthood, with follow-up studies showing that relapse rates increased substantially over time. To look at relationships between early and later psychopathology studies of attention deficit hyperactivity disorder (a child onset disorder) and schizophrenia (an adult onset disorder) will be discussed along with complexities in reviewing evidence, neurobehavioural deficits and treatment responses.

Attention-deficit hyperactivity disorder (ADHD)

Follow up studies of children with ADHD have independently shown higher than general population rates of psychiatric disorders, anti-social behaviour and problems with inter-personnel relationships in adulthood which remain after the effects of co-occurring conduct disorder are accounted for (Swanson,Seargeant, Taylor, Sonuga-Barke, Jensen, et al,1998).

Schizophrenia

Around 50% of adult schizophrenics had childhood emotional problems, including anxiety and aggression, together with neurological dysfunction and odd behaviour (see Zeitlin, 2000: Rutter, et al, 1999: Gilvarry, Russell, Helmsley, Murray, 2001). Consistent with this there is evidence of early behavioural antecedents of schizophrenia in adulthood from both home videos, and from teacher reports of adaptation in childhood from a follow-up study of 15,000 individuals born in March 1958 (see Pearlson, 2000).

Complexities

Inconsistencies in evidence may be due to several factors. Firstly, evidence of early behavioural antecedents of adult onset disorders implies that patterns of continuity will only be apparent if children are followed into adulthood. For example, follow-ups of children who developed schizophrenia as adults showed that boys had more externalising problems at seven, but that girls had more internalising problems in early adolescence (see Pearlson, 2000). Secondly, high rates of comorbidity make it difficult to establish how specific continuity is over time, and in particular comorbidity can be 'concurrent' or

'successive' (see Angold, et al, 1999). Studies also show that comorbidity is particularly associated with anxiety and personality disorders, with evidence of both homotypic (disorders from similar diagnostic categories) and heterotypic (disorders from different diagnostic categories) continuity of psychopathology (see Graham & Stevenson, 1987; Foley,Pickles, Simonoff, Maes, Sillberg, Hewitt, et al, 2001; Angold, et al, 1999).

Stable neurobehavioural deficits

Psychopathology is associated with a wide range of stable neurobehavioural deficits. For example, children and relatives of schizophrenics have significantly poorer neurobehavioural functioning relative to controls, which shows continuity from early childhood to adolescence. Sustained attention deficits and neuromotor impairment are present in schizophrenic probands and their relatives, and are present during remission (Chen & Faraone, 2000; Goodman, 1988). Differences have also been found in individuals with affective psychosis and their relatives, with relatives of both schizophrenic and affective psychosis individuals showing schizophrenia spectrum traits (Gilvarry, et al, 2001; Egan, Goldberg, Gscheidle, Weirich, Rawlings, Hyde, et al, 2001). Similarly, poor neurobehavioural function in early childhood is associated with psychopathology in adolescence (Gilvarry, et al, 2001; Hans, Marcus, Nechterlein, 1999).

Treatment responses

There is some evidence that responses to treatment differentiate between internalising and externalising disorders. For example, there is overlap in drug treatments across disorders, but in contrast behavioural treatments do show differences between internalising and externalising disorders. For internalising disorders, cognitive-behavioural therapy, which uses strategies to alter the perspectives, and thoughts believed to be important in internalising disorders, has been shown to significantly improve functioning (Kendall & Southam-Gerow, 1996; Kendall, Flannery-Schroeder, Panichelli-Mindell, Southam-Gerow, Henin, Warmen, 1997). Whereas, for externalising disorders, cognitive-behavioural therapy is not effective, and consequently, there is more emphasis on social skills training, and behavioural intervention in school, and in the home. There is some evidence of improvement, particularly where there is early intervention (Sonuga-Barke, Daley, Thompson, Laver-Bradbury, Weeks, 2001; Barkley, Shelton,Crosswait, Moorehouse,

Fletcher, Barrett, 2000). However, it is very difficult to assess treatment effectiveness (Harrington, Whittaker, Shoebridge, 1998) and follow-up studies tend to be over relatively short periods.

In summary although psychopathology has been conceptualised differently in psychiatric and psychological approaches, categorical and dimensional measures of psychopathology are comparable. There is a relationship between early and later psychopathology. This is consistent with the presence of stable neurobehavioural deficits associated with psychopathology. Patterns are similar for externalising and internalising disorders, but there are complexities in assessing how specific continuity is.

1.3 PERSONALITY AND PSYCHOPATHOLOGY

1.3.1 MEASUREMENT ISSUES

Before discussing the association between personality and psychopathology issues of measurement overlap need to be discussed. Definitions of personality and psychopathology clearly distinguish between the two phenotypes (personality refers to enduring dispositional behaviours, and psychopathology refers to psychologically disturbed behaviours). However, there are problems with the extent that empirical measures of personality and psychopathology are capturing different behaviours.

Face validity of measures

The content of personality and psychopathology measures is similar. For example, items reflecting neuroticism refer to behaviours assessed as symptomatic of anxiety, and items reflecting novelty seeking refer to behaviours assessed as symptomatic of hyperactivity. Therefore any relationship between personality and psychopathology may reflect contamination of measures rather than any true phenotypic association (see Lahey, Waldman, Applegate, in press: Shiner & Caspi, 2003).

Construct validity

Issues regarding the extent that personality and psychopathology represent different behaviours have not been well addressed in the literature, with some studies using aspects of temperament as adjustment measures. One difficulty is the way that personality influences a wide range of psychological variables (e.g. Eysenck, 1990). However, psychopathology is associated with many factors (developmental instability, neurobehavioural deficts, medical conditions, social class, psychological distress, etc.) not characteristic of personality, although there seem to have been few direct tests. There is some evidence of differential associations for personality and psychopathology with other variables. For example, Rijsdijk, Sham, Shore, Purcell, Farmer, Goldberg, et al (2001) found neuroticism was less strongly associated with life events than anxiety, and in particular, while evidence from genetic studies show overlap in genetic influences, there are differential patterns of genetic and environmental influences on both phenotypes, which will be discussed.

1.3.2 PERSONALITY AND PSYCHOPATHOLOGY IN ADULTS

Theoretical perspectives

The Eysenck Personality Questionnaire (EPQ) (Eysenck & Eysenck, 1975) includes three orthogonal traits (neuroticism, extraversion, and psychoticism) which reflect biological variation underlying personality related to vulnerability to psychopathology. In particular neuroticism and psychoticism represent vulnerability to disorder in terms of underlying predispositions which make an individual vulnerable under stress. Evidence from conditioning studies, and drug responses, has been consistent with this theory.

Figure 1.3.2.1 EPQ traits

Neuroticism	Extraversion	Psychoticism
extent of physiological reactivity - high scores linked to chronic overarousal/neurosis	extent of balance between inhibition/excitation - high scores linked to under-arousal	possible links to male sex hormones - high scores linked to lack of empathy, aggression, criminality/psychosis

Neuroticism or emotional instability has also been more specifically linked to psychopathology. Based on reviews of studies of anxiety and depression Clark & Watson (1991) suggested a tripartite model to account for the high rates of comorbidity between these two disorders. General distress or high levels of negative affect is common to both anxiety and depression, but physiological arousal is specific to anxiety disorders, and absence of positive affect is specific to depressive disorders.

Empirical evidence

Studies have used a variety of different personality instruments, however, two key aspects of personality (emotionality and constraint) can be summarised, which have been consistently associated with psychopathology. Emotionality broadly refers to the tendency to become physiologically aroused and experience negative affect, this includes components of stress reactivity, anxiety, fearfulness, anger/aggression, and alienation/victimisation. Constraint broadly refers to the tendency to regulate/inhibit behaviour, which includes self-control, planfulness, lack of impulsiveness/sensation seeking, and persistence.

Emotionality

Both cross-sectional and prospective studies show that negative affect or trait neuroticism is associated with anxiety and depression (see Clark, Watson, Mineka, 1994; Wilhelm, Parker, Dewhurst-Savellis, Ashgari, 1999; Bienvenu, Brown, Samuels, Liang, Costa, Eaton, et al 2001; Bienvenu, Nestadt, Samuels, Costa, Howard, Eaton, 2001; Jorm, Christensen, Henderson, Jaccomb, Korton, Rodgers, 2000; Cox, Enns, Walker, Kjerniskil, Pidubny, 2001). Similar patterns have been obtained when state anxiety and state depression measures were controlled for (Tanaka, Sakamoto, Kiji, Kikamura, 1999) in clinical and general population samples, and across categorical and dimensional measures (see Bienvenu, Nestadt, et al, 2001; Krueger,et al, 2000).

Negative affect has also been specifically associated with panic disorder, social phobia, agoraphobia and major depression (Bienvenu, Brown, et al, 2001) suicide behaviour (Verona, Joiner, Patrick, 2001) obsessive compulsive disorder (Lyoo, Lee, Kim, Kung, Kwan, 2001) conduct disorder and substance abuse, risk-taking, and interpersonal violence

(Krueger, et al, 2000) and is the most important independent predictor of personality disorder (Ball, Tennen, Poling, Kranzier, Rounsaville, 1997).

Constraint

Similarly personality traits of constraint, self-directedness and impulsive sensation seeking have also been specifically related to disorders. These include risk-taking behaviours (alcohol, sex, smoking, drugs, driving, gambling) interpersonnel violence, crime, substance abuse, conduct disorder, and obsessive compulsive disorder (Krueger, et al, 2000; Zuckerman & Kuhlman, 2000; Lyoo, et al, 2000). In particular high novelty seeking scores were specifically associated with casual and abusive levels of ecstasy use, relative to a control group made up of non-drug users and users of drugs other than ecstasy (Dughiero, Schifano, Forza, (2001). However, chronic ectasy use was also associated with low harm avoidance scores, and ectasy users as a group scored significantly higher on the psychoticism scale of the Hopkins Symptoms Distress Checklist.. There is also evidence of a relationship between personality characteristics and age of first offence, and frequency of lifetime arrests (Donnellan, Ge, Wenk (2001). Normative values and orientation scores differentiated among offenders at age of first arrest, with more conforming behaviour and self-control associated with older age of first arrest. Similarly this group showed significantly more interest in intellectual matters.

In particular, Nigg, John, Blaskey, Huang-Pollock, Willcutt, Hinshaw, et al (2002) found a relationship between ADHD symptoms and personality across six studies. Inattention-disorganised symptoms were associated with low constraint and neuroticism, and hyperactivity-impulsivity-oppositional symptoms were associated with low agreeableness.

Personality and personality disorders

Research has also focused on the relationship between normal personality traits and personality disorders. However, there are difficulties in the diagnosis and conceptualisation of personality disorders (Hill, Fudge, Harrington, Pickles, Rutter, 2000). Morey (1997 p938) suggests that there appears to be no qualitative difference between personality disorder and normal personality traits in that there is no natural boundary or discontinuity, which implies the same underlying structure. For example, personality

disorders have been modelled both by the five-factor personality inventory (Widiger & Trull, 1992; Miller, Lynam, Widiger, Leukefeld, 2001) and the temperament and character inventory (Cloninger, Bayon, Svrakic, 1998).

Reciprocal relationship between emotionality and constraint

There is also some evidence of a reciprocal relationship between emotionality and constraint and association to psychopathology. For example, John, Caspi, Robins, Moffitt, Stouthamer-Loeber (1994) found low agreeableness and conscientiousness scores in delinquent boys, and Heaven (1996) found correlations between agreeableness, conscientiousness and neuroticism with delinquency.

In particular, psychopathy can be represented by specific combinations of lower order five-factor personality dimensions. Miller, et al (2001) found that scores on an expert based NEO PI Psychopathy Resemblance Index significantly related to low scores of agreeableness, and conscientiousness. For extraversion psychopathy resemble corresponded to high scores on assertiveness and excitement seeking, and low scores on warmth, and for negative affect psychopathy resemblance corresponded to high scores on angry hostility, and impulsivity, and low levels of anxiety, depression, self-consciousness and vulnerability.

1.3.3 CHILD TEMPERAMENT AND PSYCHOPATHOLOGY

Graham & Stevenson (1987) suggested some types of psychopathology could be conceptualised as a continuum of normal temperament traits. For example, dimensional measures of behavioural problems in twins have a similar three factor structure to temperament traits of activity, emotionality and sociability. Moreover, when children with extreme scores were excluded from analysis, a similar factor structure was obtained. Therefore hyperactivity, emotional and antisocial disorders, can be conceptualised as expressions of very extreme forms of ordinary non-pathological behaviours.

Consistent with this typology, early temperament characteristics have been associated with later psychopathology. The concept of 'difficult temperament' (low adaptability, withdrawal from novel stimuli, high intensity, negative mood, and low rhythmicity) was developed in the New York Longitudinal Study, and associated with behaviour problems in childhood, and, from age three with psychiatric adjustment in adulthood (Thomas, et al, 1968). Difficult temperament has been related to regulatory disorder in infancy, and predicts regulatory disorder in early childhood, early temperament predicts problem behaviour at age three in boys, and there is evidence that a fussy demanding temperament in infancy predicts psychiatric symptoms in adolescence, although classification of disorder in infancy is problematic, where the behaviour of very young children is diffuse (see Zeanah, Boris, Larrieu,1997; Earls & Jung, 1987; Teerikangas, Aronen, Martin, Huhunon, 1998; Graham & Stevenson, 1987). Difficult temperament at age two till four years is more strongly associated with stable rather than transitory behaviour problems (Prior, Smart, Sanson, Pedlow, Oberklaid, 1992).

Continuity of the relationship between temperament and psychopathology

Difficult temperament in early childhood appears to be a strong predictor of psychiatric problems in older children. There is evidence that difficult temperament at four contributes to clinical outcome (Maziade, Cotes, Bernier, Boutin, Thivierge, 1989). Emotion regulation and behaviour problems at age three has been linked to externalising disorders at age eight, with emotional problems linked to internalising symptoms (Sonuga-Barke, Thompson, Stevenson, Viney, 1997). In particular, a prospective study of a large cohort sample in Dunedin, New Zealand linked temperamental factors at age three to psychological adjustment at age 21. Individuals with an undercontrolled temperament at three showed more externalising problems in middle childhood, adolescence and early adulthood. Individuals with an inhibited temperament showed more internalising problems in adolescence and early adulthood (Caspi, 2000). For children followed from infancy in the Australian Temperament Project, previous behaviour problems were the strongest predictor of psychological adjustment at twelve years. In addition, emotionality, inflexibility and poor task persistence in middle childhood were associated with increased risk of behaviour problems at twelve years (Prior, Smart, Sanson, Obkerlaid, 2000).

Externalising problems

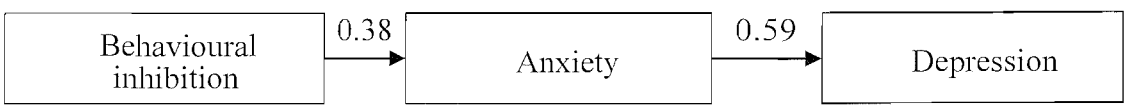
Early temperament traits predict later externalising problems. For example, stimulation seeking and fearfulness at age three predicted aggression at age eleven independently of gender or ethnicity (Raine, Reynolds, Venables, Mednick, Farrington, 1998). Similarly, undercontrolled temperament at three is associated with violent and non-violent self-reported offences at eighteen (Henry, Caspi, Moffit, Silva, 1996). Consistent with this management difficulties, activity, and temper tantrums at age three is associated with adult violence convictions independently of family and social circumstances (Stevenson & Goodman, 2001). Trait impulsivity was significantly related to measures of psychopathy in boys aged twelve and thirteen. Cognitive impulsivity accounted for 10% of behavioural impulsivity and for 35% of the variance in psychopathy, and this was not related to general cognitive ability (Lynam, 1997).

Internalising problems

A series of studies by Kagan and colleagues has linked behavioural inhibition to later anxiety problems. For example, childhood behavioural inhibition has been associated with social phobia in adolescence, and is related to negative affectivity (Hayward, Killen, Kraemer, Taylor, 1998) and highly reactive temperament in infancy is related to anxiety symptoms in childhood (see Prior, et al, 2000; Kagan, Snidman, 1999) and adult anxiety disorders (Kagan & Snidman, 1999). Negative affect has been associated with major depression in 11 to 16 year olds (Goodyer, Ashby, Althan, Vize, Cooper, 1993).

Self-reported behavioural inhibition was associated with anxiety and depression symptoms in a sample of 968 adolescents, and the best fitting model showed that behavioural inhibition leads to anxiety which results in depression (see Muris, Merckelbach, Schmidt, Gadet, Bogie, 2001).

Figure 1.3.3.1 The relationship between behavioural inhibition and anxiety and depression



(Muris,Merckelbach, Schmidt, Gadet, Bogie, 2001)

Factor analysis showed structural similarity in the relationship between personality traits and behaviour problems in male adolescents in a Russian Juvenille Correction Centre and controls (Ruchkin, Eisemann, Cloninger, 1998). Although, both groups differed significantly on personality and behavioural and emotional problems, with higher novelty seeking, harm avoidance and self-transcendence, and lower self-directedness and co-operativeness for the clinical group, patterns of interrelationships were similar. Emotional problems were positively related to harm avoidance, and negatively related to self-directedness, and aggressive and delinquent behaviour was positively related to novelty-seeking, and negatively related to co-operativeness. There was also evidence of a relationship between novelty seeking and attention problems in both groups. Similarly, a significant relationship between personality and delinquency was found for dimensional measures of delinquency across three groups (adolescent males attending school, adolescent females attending school, and institutionalised adolescent males) (Romero, Luengo, Sobral, 2001).

Similar relationships between personality and behaviour problems were found in children and adolescents (7 to 17 years) (Huey & Weisz, 1997). Ego-undercontrol was positively correlated with externalising problems and negatively correlated with internalising problems, ego-resiliency was negatively associated with both internalising and externalising symptoms. This implies an overlap between personality related to impulse control and psychopathology (Huey & Weisz, 1997).

1.3.4 BIOLOGICAL CORRELATES

In support of evidence of a relationship between personality and psychopathology at the behavioural level, there is also evidence of a relationship between biological correlates of personality and psychopathology.

Internalising problems

Similar patterns of exaggerated fear responses have been shown in both behaviourally inhibited and clinically anxious children. Research with animals has shown that the amygdala is responsive to novel stimuli and events, and is important to the acquisition of conditioned fear (see Kagan, Snidman, 1999; LeDoux, 1996). There is evidence that some

children with reactive temperaments (indexed by vigorous limb movement and distress to novel stimuli in infancy, and fearfulness and avoidance in young children) implying a low threshold in amygdala responsivity, show psychological and biological signs of stress to threat including indices of sympathetic activity. Physiological reactivity associated with behavioural inhibition has been associated with narrow facial skeletons, which implies a genetic origin. Cells of the neural crest (a necklace of ectodermal cells that appear around three weeks after conception as the neural tube is forming) migrate to form sensory ganglia, bones of the skull and face and the autonomic nervous system (see Kagan, Snidman, 1999 p 1537; LeDoux, 1996).

Consistent with this there is evidence that children with anxiety disorders show an exaggerated amygdala response to fearful faces, whereas children with depression show a reduced amygdala response to fearful faces (Thomas, Drevets, Dahl, Ryan, Birmater, Eccard,et al, 2001). Children classified as belonging to high or low reactive temperament groups in infancy, could be differentiated by brainstem auditory evoked responses in late childhood (aged between ten and twelve years) (Woodward, McManis, Kagan, Deldin, Snidman, Lewis, et al (2001).

However, there is some evidence that there are separate neural systems underlying fear and anxiety as a differential association was found between both traits with norepinephrine and dark-induced pupil reactivity (White & Depue, 1999). Specific norepinephrine induced pupil reactivity was significantly associated with measures of Harm avoidance (MPQ) and dark-induced pupil reactivity was associated with negative emotionality, which imply a double dissociation between fear and anxiety.

Externalising problems

There is evidence of a relationship between biological markers of personality and externalising disorders. In particular similar patterns of low autonomic reactivity have been shown in children with low trait constraint and externalising disorders. Low resting heart-rate, together with body size at age three, is associated with fearfulness and stimulation seeking, and later aggressive behaviour (Raine, Venables, Mednick, 1997). Psychopathy and under-socialised aggressive conduct disorder has been associated with electrodermal hyporeactivity , which is hypothesised to reflect a weak behavioural inhibition system, and

there is evidence of potential executive functioning deficit in psychopaths (see Fowles, 2000).

Individuals with low self-directedness had significantly reduced P300 amplitudes, which may reflect a common factor personality factor in several different types of disorder (Vedeniapin, Anokhin, Sirevaag, Rohrbaugh, Cloninger, 2001). In line with this reduced amplitude of the P300 event-related brain potential is thought to represent brain dysfunction, and has been associated with schizophrenia, bipolar depression, borderline and anti-social, personality disorders.

Pharmacological responses

There is evidence that drugs like anti-depressants show broad-band efficacy and therefore may be influential through their effects on personality (see Krueger, Caspi, et al, 2000). Consistent with this personality traits have been linked with drug responses. Individuals with high scores of reward dependence, and low scores of harm avoidance showed a better outcome following drug trials where serotonergic re-uptake inhibitors were used to shorten the timescale of effects from anti-depressants (Tome, Cloninger, Watson, Issac, 1997).

In summary theoretical considerations suggest a relationship between personality and psychopathology. Evidence is consistent with this and shows personality traits of emotionality and low constraint are associated with psychopathology. High emotionality is associated with psychopathology in general, while low constraint is more specific to externalising disorders. This pattern is the same for adults and children, and for behavioural and physiological measures.

1.4 THE GENETICS OF TEMPERAMENT, PERSONALITY AND PSYCHOPATHOLOGY

1.4.1 BACKGROUND

Genetic methodology can be used to identify factors underlying the association between personality and psychopathology. Quantitative genetic research uses biometric modelling to estimate genetic, shared and non-shared environmental contributions to behaviour. Molecular genetic research identifies specific genetic variation associated with behavioural variation. Factors underlying variation in personality, psychopathology and cognitive function have been widely addressed, however there has been much less research into factors underlying the relationship between personality and psychopathology.

Gene structure and function

Genes are pieces of deoxyribonucleic acid (DNA) which consists of two anti-parallel strands of nucleotide bases wrapped round into a double helix. There are four possible bases, adenine (A) thymine (T) guanine (G) and cytosine (C) and these bond together in specific pairs. Adenine always pairs with thymine, and guanine always pairs with cytosine, which means one strand is the mirror image of the other and acts as a template for replication when the molecule comes apart during cell division. The genetic code is contained in triplet sequences of the base pairs coding for specific amino acid in proteins. Some sequences act as stop and start signals and regulate when the gene is operative, so genetic influences are dynamic and can change over time.

During protein synthesis the genetic code is expressed and translated into sequences of amino acids which form protein structures. This happens in two stages, the cell makes a complementary copy of one strand of the DNA molecule by synthesising a strand of ribonucleic acid (here the sugar is ribose instead of deoxyribose, and uracil replaces thymine in the base sequences) called messenger ribonucleic acid (mRNA). mRNA then leaves the nucleus of the cell and enters the cytoplasm where transfer ribonucleic acid translates the sequence of amino acids into a protein structure on the ribosomes.

Only a small percentage of DNA is transcribed into RNA (approximately 2%) (Wahlsten, 1999). Introns are parts of genes that are transcribed but then spliced out before the RNA leaves the nucleus so they do not code for proteins. Their function is unclear but in some cases they regulate the transcription of other genes. The parts of genes that are spliced back together are called exons, these leave the cell nucleus and are expressed as amino acid sequences which form proteins.

Changes or mutations in the triplet base sequences can give qualitatively different gene products. For example, if bases are deleted, inserted or altered in some way this will change the amino acid sequence, which affects how the protein folds and in turn affects how well it functions. This has implications for behaviour, for example, through the impact of genetic variation on brain structure, neural connectivity, metabolism in the central nervous system, and the efficiency of neuro-transmission.

Neurotransmission

Nerve cells communicate with each other through synapses (the point of junction between the axon of one neuron and the cell body or dendrite of another). Neurotransmitters are chemical messages that diffuse from a presynaptic neuron across the synaptic cleft to the membrame of the postsynaptic cell, which it binds to and stimulates.

Monamines

Molecular research has focused on monamines, which include substances like dopamine, serotonin, epinephrine and norepinephrine. Dopamine and serotonin are phylogentically ancient neurotransmitters related to brain function and behaviour (Cravick & Goldman, 2000, and see review by Gainetdinov & Caron, 2002). Dopamine is associated with the activation and intensity of response to reward underpinning the tendency to actively explore/approach novel stimuli, and serotonin is associated with inhibition of behaviour and emotional responses. Behavioural and pharmacological responses imply these are important to behavioural traits.

Dopamine

The dopamine D4 receptor genes show extensive variation, and are unusual as some marked variants do not result in changes in protein sequences (Ebstein, Benjamin, Belmaker, 2000). The dopamine transporter (DAT1) is functionally significant to the regulation of dopaminergic neurotransmission by mediating the active reuptake of dopamine from the synapse into the presynaptic terminal (Giros & Caron, 1993). DAT1 produces a protein present in the presynaptic membrame of dopamine containing nerve cells which recycle dopamine from the intracellular region that makes synaptic connections. It regulates the temporal and spatial availability of dopamine for neurotransmission. It is also the molecular site for several psychoactive drugs including amphetamine and cocaine. In particular cocaine appears to block dopamine transporter function, whereas amphetamine appears to stimulate the release of dopamine and block its re-uptake (see Rowe, Stever, Gard, Cleveland, Sanders, Abramowitz, 1998 p 216).

There are different behavioural effects associated with dopamine genes in mice. Mice lacking in dopamine D1 receptors show deficits in spatial learning, movement initiation and reaction to stimuli. They also drink less alcohol, and show reduced amphetamine induced locomotor activity. Mice lacking in dopamine D2 receptors show symptoms of Parkinsons disease and reduced spontaneous movement. Mice lacking in dopamine D3 receptors show increased motor activity, and reduced anxiety responses in behavioural tests. Mice lacking in dopamine D4 receptors show locomotor sensitivity to ethanol, cocaine and amphetamines. Mice lacking the DRD5 receptor, also show a reduced behavioural response to novel stimuli, and are more sensitive to ethonal (see Cravick & Goldman, 2000). There is also evidence that if the dopamine transporter gene (DAT1) is deactivated, mice become spontaneously hyperactive, retain extremely high dopamine levels and show increased activity in novel environments (Giros, Jaber, Jones, Wightman, Caron, 1996; Gainetdinov, Wetsel, Jones, Levin, Jaber, Caron, 1999).

Serotonin

Serotonergic neurons are located in the brainstem with projections to virtually every part of the central nervous system. In the adult brain serotonergic raphe neurons project to regions

linked functionally to emotion, cognition and motor function, and are important to behavioural regulation as their activity modulates responses to other neurotransmitters.

Transcription of the serotonin transporter gene (5-HTT) is modulated by a common polymorphism (5-HTT linked polymorphic region; 5 HTTLPR) for which ten novel sequence variants have been identified (see Nakamura, Ueno, Sano, Tanabe, 2000; Lesch & Mossner, 1998). 5HT is known to be important to early embryonic development, and is dysregulated in a variety of complex behavioural traits, including obsessive compulsive disorders, bipolar disorder, depression, anxiety, substance abuse and neurodegnerative disorders.

Although little is known about the function of serotonin 5HT receptors, there is a known high affinity for antipsychotic drugs, and antidepressant drugs. Mice lacking in these receptors show increased anxiety in behavioural tests, and more aggressive behaviour, and drink more alcohol (see Cravick & Goldman, 2000).

The 5HTTLPR promoter region polymorphism has been focused on in research into many disorders (panic disorder, bipolar disorder, obsessive compulsive disorder, late onset Alzheimer disease, schizophrenia, substance abuse, seasonal affective disorder, mood disorder, and smoking) (see Manor, Eisenberg, Tyano, Sever, Cohen, Ebstein, 2001 p93).

Drug effects

Dopamine receptors and 5HT receptors belong to the same family of G proteins, and are known to have a similar structure. Drugs used for depression, schizophrenia, obsessive compulsive disorder, panic disorder, generalised anxiety disorder, and social phobia act on brain 5HT and dopamine activity (Lesch & Mossner, 1998). For example, dopamine D2 receptors are the main targets of antipsychotic drugs, Dopamine D3 receptors are only in the limbic area and may be associated with dopaminergic control of cognition and emotion functions of antipsychotic drugs. Dopamine D4 receptors have a high affinity for clozapine which is used to treat schizophrenia if typical antipsychotic drugs are ineffective.

Gene transmission

Mendelian principles of segregation and independent assortment are general laws of inheritance which can be applied to both single gene and complex polygenic traits. There are two copies of each gene for any trait which are arranged in the same linear order on matching chromosome pairs. During reproduction each chromosome pair is separated. For each pair, offspring inherit one chromosome from each parent, which means the genes on separate chromosomes are independently or randomly transmitted to the offspring. Genes on the same chromosomes are linked (i.e. transmitted together). However, this linkage can be broken by crossing-over (the breakage and re-assembly of chromosomes during meiosis). The frequency of this breaking of linkage between two genes is used as a measure of distance apart of position on chromosomes.

Recombination of chromosome pairs during meiosis

Errors can arise during recombination of chromosome pairs in meiosis. These include nondisjunction (an uneven split of the pairs of chromosomes) inversion (part of the chromosome is inverted with respect to the rest of the chromosome) deletion (part of a chromosome is missing) duplication and translocation (a portion of one chromosome is broken off and attached to another chromosome).

Genes responsible for individual variation are polymorphic, they have two or more forms (alleles) and the two copies can be the same (homozygous) or different (heterozygous). These give ratios for the expected frequencies of possible phenotypes (expressed genetic influences) which can be used to ascertain genetic influences on behavioural variation.

Transmission of genetic information can be complex. For example, genetic influences can be additive, or non-additive (dominance, and epistasis). Additive influences are where the total effect of alleles are cumulative. Dominance influences are where there is an interaction between alleles at the same chromosomal location (loci). Epistasis is where interactions between alleles at different loci modifies their expression. Dominance and epistasis are non-additive influences and represent the difference between expected and actual genotype values (see Plomin, et al, 1997).

1.4.2 QUANTITATIVE GENETIC RESEARCH

Quantitative genetic research looks at the relative magnitude of genetic and environmental factors, which contribute to behavioural variation. Statistical modelling techniques are used to separate out components and provide estimates of the strength of genetic influences, and patterns of transmission of traits. Models are based on group differences and assume that underlying genetic and environmental liabilities are continuously distributed, so phenotype values represent variations from the population mean for a trait. For categorical traits a liability threshold is estimated as a cut-off point for distinguishing if a trait is present. In models phenotype variance is partitioned into three components. These are genetic influences, shared environmental or common influences on family members, and non-shared environmental or unique influences on each family member. Environmental influences include anything that is not genetically influenced. Components are estimated by comparing differences in phenotypes across different levels of genetic relatedness and environmental experiences using family, twin, and adoption study designs.

Family studies

In family studies the frequency of a trait across different degrees of family relatedness is compared to the frequency of the trait in the general population. For example, it can be assumed that on average full siblings, and parents and offspring, share 50% of their genes, whereas grandparents and grandchildren, half-siblings, uncles and nephews, etc. share 25% of their genes, and first cousins share only 12 ½ % of their genes. Therefore, patterns of relative risk for a trait can be assessed across family members. If a trait is genetically influenced then there should be a linear decrease in phenotypic similarity from siblings to first cousins etc. However, as family members also share a common environment, family resemblance may also be due to environmental effects.

Twin studies

Twin studies can separate out the genetic and environmental components contributing to behavioural variation, and allow children of the same age to be compared, which is important as expression of traits varies by age, and different genes may influence behaviour at different ages (Goodman & Stevenson, 1989; Martin, Boomsma, Machin,

1997). The analysis of this data is based on the differences between genetic relatedness in monozygotic (MZ) and dizygotic (DZ) twins. Both types of twins share a common environment, but MZ twins share all their genes, whereas DZ genes share on average only half, and differences in similarities give estimates of heritability. Higher similarity of a behavioural trait for MZ than DZ twins implies a genetic influence, as MZ twins share both additive and non-additive effects genetic effects.

Concordance

Concordance refers to the presence of a particular trait (usually dichotomous) and can be pairwise or probandwise. Pairwise concordance rates refer to the number of twin pairs where if one twin is affected the other one is. Probandwise concordance rates refer to the number of probands in concordant pairs in comparison to the total number of probands. For example, out of 100 twin pairs where 30 sets of twins are concordant and 70 sets of twins are disconcordant for a trait, pairwise concordance is 30% (30/100) and probandwise concordance is 46% (60/130).

Correlations

Genetic, shared and non-shared environmental influences contribute to observed behaviour. Both types of twins share a common environment (so the correlation for shared environmental influences is 1.00) but MZ twins share all their genes (so the correlation for genetic influences is 1.0) and DZ twins share only half on average (so the correlation for genetic influences is 0.5). Non-shared environmental influences will be unique to each twin (so the correlation for non-shared environmental influences will be 0). Therefore simultaneous equations allow genetic and environmental parameters to be estimated. In particular structural equation modelling (Neale & Cardon, 1992) allow simultaneous testing and estimation of genetic and shared environmental influences. Twin methodology will be discussed in detail in Chapter two.

DF extremes analysis

In DF extremes analysis (DeFries & Fulker, 1985) family resemblance is based on dimensional scores rather than presence/absence rates, and estimated as the extent to which

average trait scores for co-twins of probands regress back to the population mean relative to the proband mean. If means for DZ co-twins regress further back to the proband mean, this implies genetic influences on aetiology. This gives a group rather than an individual heritability estimate, based on the assumption that variation in factors underlying the trait, are continuously distributed. If data is available on a general population sample of twins, DF extremes analysis can be used to test whether the mix of A, C and E is different for extreme group membership (e.g. highly anxious individuals) for a trait than for variation in the normal range (eg. Stevenson, Batten, Cherner, 1991; Gjone, Stevenson, Sundet, 1996; Dale, Simonoff, Biship, Eley, Oliver, Price, et al, 1998). If individual and group heritability estimates differ for a trait this implies different etiological factors underlie a disorder to those which underlie differences within the normal range. However, if individual and group estimates are the same this implies that a disorder may be an extreme of normal behavioural variation (see Deater-Deckard, et al, 1997).

Adoption studies

Adoption studies separate out genetic and environmental influences. Resemblance between adopted children and their adoptive parents is assumed to be produced by shared environmental influences. Resemblance between adopted children and their biological relatives is assumed to be produced by genetic influences. Therefore differences in correlations of scores on a trait between biological and non-biological relatives can be used to separate out genetic and environmental influences.

1.4.3 MOLECULAR GENETIC RESEARCH

Recent advances in the availability of genomic information, laboratory and analytic techniques mean molecular research can be applied to locating and identifying the genes responsible for polygenic traits. Research aims to locate and identify the genes responsible for behavioural variation, and the main methodologies are association and linkage analysis.

Association analysis

Association studies look at the correlation between the frequency of genetic markers (identified polymorphisms in DNA), and a trait or disorder in the general population, to see

if a candidate gene, or biologically relevant marker, is more frequent in affected individuals than controls. If the marker is close to the gene responsible for the disorder very small effects can be detected, and if candidate genes are selected on the basis of a hypothesised role in the pathophysiology of a disorder statistical power is increased (see Malhotra & Goldman, 1999). However, associations can occur due to natural allelic variation in populations where the presence of certain alleles is not related to the disorder, and is just due to stratification across ethnic groups.

Linkage analysis

Linkage studies looks at the co-inheritance of genetic markers and a trait or disorder in families. This is based on the assumption that if a marker and the gene responsible for a disorder are very close together on a chromosome, there will be little breakage or recombination of chromosomal material during gamate formation. The less the distance between the marker and the gene, the less times recombination will occur. Therefore the location of the gene can be estimated. In parametric studies the parameters of the genetic mechanisms involved are known (eg. whether a trait is sex-linked, dominant or recessive). This allows a recombination fraction to be estimated, and lod scores (the log odds of the likelihood of linkage based model parameters divided by the likelihood of no linkage) give the strength of linkage or how close the genes are together on the chromosome. By convention a lod score of three and above is taken as evidence for linkage (but see Burmeister, 1999).

As the mode of transmission is unknown for most behavioural traits, non-parametric linkage studies are based on the comparison of shared alleles among affected family members to expected frequencies of independent transmission of alleles. Important here is if a mutation is influencing a trait in a specific chromosomal region in a high proportion of families, then it is expected that two affected individuals from the same family will share alleles at a marker locus more frequently than chance. As this may differ from family to family then only the number of alleles shared is compared. If there is no linkage the ratio of shared alleles is 1:2:1 and departure from this indicates that the marker is linked to the gene influencing the trait. In linkage analysis only a strong effect will be detected, but this can be for loci some distance from the marker if they co-segregate.

Transmission disequilibrium tests and haplotype relative risk strategies

Most studies now use transmission disequilibrium tests (TDT) or haplotype relative risk (HHR). TDT is where association and linkage are combined by looking at the frequency of alleles transmitted across parents and affected child trios to see if this is different to chance levels. At least one parent needs to be heterozygote in order to compare the ratios of expected phenotypes, and non-transmitted parental alleles act as controls. This overcomes the problem of spurious associations due to population stratification (see Plomin & Crabbe, 2000).

Haplotypes are a combination of closely linked groups of alleles inherited together as a unit, and the proportion of transmitted alleles is compared to the proportion of non-transmitted alleles in family trios consisting of parents and an affected child. Haplotype relative risk strategies overcome stratification bias for association studies.

Complexity of genetic influences on behavioural traits

Behavioural traits are complex and are thought to be influenced by multiple genes of small effects (quantitative trait loci) which contribute both additively and interchangely to probalistic risk factors. If there are multiple factors contributing to a trait then this implies that the trait will be distributed quantitatively, which may be important to conceptualisation of psychopathology (see Plomin & Crabbe, 2000, p 808), and in particular to assessing the relationship between personality and psychopathology.

Quantitative trait loci

Quantitative trait loci (QTL) are genes of various effect sizes in multiple-gene systems which influence continuous variation in a phenotype (Plomin, et al, 1997). QTL linkage analysis has been applied in animal studies and human studies. For example, several quantitative trait loci have been identified for trait emotionality in mice using measures defined by covariation of two traits, which have no physiological links (open field activity and defecation). Selective breeding (breeding for a phenotype over several generations by selecting parents with high/low scores on the phenotype, and mating them) showed heritability estimates of 0.26 and 0.11 respectively, but a genetic correlation of -0.86.

Quantitative trait loci were mapped for three mouse chromosomes related to this trait, and behavioural tests (covered and open maze), for the two separate lines showed the loci contributed significantly to variance in open field activity. Evidence from drug responses, electrophysical and lesion experiments suggest conservation of common neural substrates (Flint, Corley, DeFries, Fulker, Gray, Milleret al, 1995 p1434).

Phenotype measures

Many molecular studies use dimensional measures of behavioural traits as phenotype markers, rather than clinical diagnosis. Dimensional measures differentiate symptom severity for affected sibling pair designs, and genetic liability may be expressed as traits shared by several disorders (see Serretti, Maccardi, Catalano, Bellodi, Smeraldi, 1999). In addition endophenotypes (markers of a trait present in unaffected relatives) can be used in analysis. For example, neurobehavioural deficits and other behavioural measures (e.g. specific verbal short term memory, gross motor skills, and attention measures, which are significantly associated with offspring of schizophrenics, relative to controls, and offspring of affective disorder probands) which may predict schizophrenia in adulthood, have been identified in the New York High Risk Project.

1.4.4 RESEARCH FINDINGS FOR TEMPERAMENT AND PERSONALITY

Temperament

In a meta-analysis Goldsmith, Buss, Lemery (1997) looked at findings separately from studies using temperament scales based on categories from the New York Longitudinal Study, and studies using the Emotionality, Activity Sociability, scales. Weighted intra-class correlations were estimated by transforming the correlations into z scores and weighting by sample size across studies.

Measures based on NYLS categories showed moderate heritability of temperament from infancy. For children aged between six months to five years (Colorado Twin Project, and the Louisville Twin Study) correlations were higher for MZ than DZ twins for all categories except for rhythmicity.

Figure 1.4.4.1 Results from twin studies using the Revised Infant Temperament Questionnaire, the Toddler Temperament Scale and the Behaviour Style Questionnaire

	Weighted intra-class correlations across studies	
Temperament dimension	MZ twins	DZ twins
Adaptability	0.66	0.39
Approach/withdrawal	0.67	-0.03
Mood	0.60	0.41
Distractability	0.76	0.55
Activity	0.59	0.05
Rhythmicity	0.86	0.79
Intensity	0.62	0.30
Persistence	0.70	0.41
Threshold	0.77	0.52

(Goldsmith, Buss, Lemery, 1997).

Similar patterns of genetic and non-shared environmental contributions to temperament were found across eight twin studies (see Goldsmith, et al, 1997) using the EASI Temperament Survey, the EAS Questionnaire, and the Colorado Childhood Temperament Inventory (CCTI) for children aged between three years six months, to nine years six months. The CCTI was developed from factor analysis of EAS dimensions and temperament categories identified in the New York Longitudinal Study. For all of the studies DZ correlations were near zero or negative.

Figure 1.4.4.2 Results from twin studies using the EASI Temperament Survey, the EAS Questionnaire, and the Colorado Childhood Temperament Inventory

	Weighted intra-class correlations across studies	
Temperament dimensions	MZ twins	DZ twins
Emotionality	0.57	0.11
Activity	0.64	-0.08
Sociability	0.69	0.10
Impulsivity	0.66	0.15

(Goldsmith, Buss, Lemery, 1997)

Saudino, McGuire, Reiss, Hetherington, Plomin (1995) found similar patterns for EAS Questionnaire measures in an adolescent sample, which included twins, full siblings, half siblings and step-siblings

However, a study by Goldsmith, Lemery, Campos, Buss (1999) showed that shared environmental factors may contribute to temperament in infants. Parent ratings and behavioural assessment showed shared environmental and additive genetic factors

contributed to both smiling and laughter and duration of orienting in 3 to 16 month old twins.

Figure 1.4.4.3 Intraclass correlations for twins adjusted for age and gender

	MZ twins	DZ twins
Infant behaviour questionnaire		
Smiling and laughter	0.72	0.52
Duration of orienting	0.69	0.45
Soothability	0.53	0.61
Distress to limits	0.66	0.28
Activity level	0.54	0.28
Distress to novely	0.59	0.28
factors		
positive emotionality	0.74	0.55
negative emotionality	0.64	0.30

(Goldsmith, Lemery, Buss, 1999)

There have been fewer adoption studies of temperament, findings are similar to twin studies, however heritability estimates are lower (see Loehlin, 1992; Plomin, DeFries, et al, 1997; Rowe 1997). In particular, self report data from the Colarado Adoption Project, for children between nine and sixteen years, showed heritability estimates of 14% (Plomin, Corely, Caspi, Fulker, DeFries, 1998).

Findings are similar in studies using behavioural measures. For example, correlations for activity level in infants (measured by actometers) were larger for MZ than DZ twins, but showed a less extreme contrast, with no negative DZ correlations (Saudino & Eaton, 1991). Observational ratings of inhibition showed MZ correlations of between 0.55 - 0.82, and DZ correlations of 0.23 - 0.47 (see Goldsmith, Buss, Lemery, 1997).

Personality - NEO Personality Inventory

A lot of quantitative genetic research has focused on the NEO PI five factor traits in adolescents and adults. Loehlin (1992) reviewed evidence from twin and adoption studies. Five major twin studies (24,000 pairs of twins in total) from different countries showed correlations for extraversion and neuroticism of 0.50 for MZ twins, and 0.20 for DZ twins. Adoption studies suggest less genetic influence on personality and model fitting showed some variation between traits in the amount of variance accounted for by genetic and non-shared environmental factors. Shared environmental factors maybe more important for neuroticism than extraversion, and maybe more important for females than males. For all

of the five dimensions (summarised across different scales) genetic estimates were between 30 and 50%, and non-additive genetic effects contributed to about 14% of the variance (see figure 1.4.4.4.).

Figure 1.4.4.4 Correlations for extraversion and neuroticism scores across twin and adoption studies

	Extraversion	Neuroticism
MZ twins reared together	0.51	0.46
DZ twins reared together	0.18	0.20
MZ twins reared apart	0.38	0.38
DZ twins reared apart	0.05	0.23
Non-adoptive parents and offspring	0.16	0.13
Adoptive parents and offspring	0.01	0.05
Non-adoptive siblings	0.20	0.09
Adoptive siblings	-0.07	0.11

(Loehlin, 1992)

More recent studies have confirmed this pattern for all of the five factor traits, and their facets, using NEO PI scales. Bergeman, Chipuer, Plomin, Pedersen, McLearn, Nesselroade, et al (1993) found similar patterns of heritability and non-shared environmental effects for openness and conscientiousness (0.40 and 0.29 respectively) in a sample of over 500 twin pairs, reared together and apart. However, for agreeableness, genetic influences only accounted for 12% of the variation, and shared environmental factors accounted for 21% of the variation (see figure 1.4.4.5).

Figure 1.4.4.5 Twin concordance for NEO-PI dimensions

	Openness	Conscientiousness	Agreeableness
MZ twins reared together	0.51	0.41	0.47
MZ twins reared apart	0.43	0.15	0.19
DZ twins reared together	0.14	0.23	0.11
DZ twins reared apart	0.23	-0.03	0.10

(Bergeman, Chipuer, et al , 1993)

This was more consistent with theoretical considerations which had suggested different traits had different sources. For example, the distinction made between temperament and personality suggested a genetic basis for neuroticism and extraversion, and an

environmental basis for openness, agreeableness and conscientiousness (see McCrae, Jang Livesley, Riemann, Angleitner, 2001). However, McCrae, et al (2001) found similar patterns of phenotype and genotype variance for all of the NEO PI five-factor traits, and no evidence of shared environmental contributions, using estimated cross-correlations for MZ and DZ twins from different raters.

Lower order traits

In a sample of 250 twin pairs, genetic influences accounted for around 48% of the variance in personality overall, with little effect of shared environment, and similar patterns were found for minor traits, although for several of these non-additive genetic variance was predominant (Jang, Livesley, Vernon, 1996) (see figure 1.4.4.6).

Figure 1.4.4.6 Twin correlations for NEO PI higher and lower order dimensions

	MZ twins	DZ twins
Neuroticism	0.41	0.18
anxiety	0.26	0.13
angry hostility	0.37	-0.01
depression	0.33	0.14
self-consciousness	0.38	0.19
impulsiveness	0.36	0.21
vulnerability	0.45	0.17
Extraversion	0.55	0.13
warmth	0.43	0.14
gregariousness	0.56	0.19
assertiveness	0.42	0.10
activity	0.29	0.14
excitement-seeking	0.42	0.02
positive emotions	0.38	0.24
Openness	0.58	0.21
fantasy	0.32	0.22
aesthetics	0.60	0.14
feelings	0.44	0.35
actions	0.42	0.21
ideas	0.53	0.09
values	0.49	0.27
Agreeableness	0.41	0.26
trust	0.27	0.21
straight-forwardness	0.47	0.17
altruism	0.34	0.18
compliance	0.33	0.10
modesty	0.30	0.36
tender-mindedness	0.41	0.15
Conscientiousness	0.37	0.27
competence	0.37	0.13
order	0.25	0.23
dutifulness	0.42	0.23
achievement-striving	0.41	0.18
self-discipline	0.30	0.30
deliberation	0.26	0.26

(Jang, Livesley, Vernon, 1996)

Comparison of German and Canadian twin samples showed that patterns of genetic and environmental contributions to lower order personality traits are the same across different

cultures. There was no evidence for shared environmental effects in broad traits, however there was for some facets (Jang, McCrae, Angleitner, Riemann, Livesley, 1998).

One study using observational ratings of personality found some evidence of shared environment contribution to personality variation. Self-report, peer report, and observational ratings were made for 300 twin pairs in Germany. Aggregate observations came from 60 minutes of videotapes of participants in 15 different settings (for example, reading aloud, telling a joke, telling a story, introducing themselves, introducing a stranger, problem solving, memory recall, making and refusing requests). Model fitting showed that for observational ratings about 40% of the variance in personality came from genetic factors, 35% from non-shared environmental factors, and 25% from shared environmental factors (Borkenau, Riemann, Angleitner, Spinath, 2001). However, there are limitations with observational measures of personality where continuity of behaviour across time and situations cannot be assessed (see figure 1.4.4.7).

Figure 1.4.4.7 Median twin correlations for averaged NEO PI trait scores

	MZ twins	DZ twins
Self-report	0.45	0.26
Peer-report	0.42	0.13
Observational rating	0.60	0.38

(Borkenau, Riemann, Angleitner, Spinath, 2001).

Psychobiological model

Heath, Cloninger & Martin, (1994) compared patterns of genetic and environmental contributions for the Tridimensional Personality Questionnaire (TPQ) and the Eysenck Personality Questionnaire (EPQ) in 2680 Australian twin pairs. For both EPQ and TPQ dimensions, additive and non-additive genetic, and non-shared environmental factors contributed to personality variation. There was also evidence of gender differences for genetic contributions to neuroticism, and findings were similar to those of previous studies. Multivariate analysis was used to assess the extent that the two personality scales assessed the same dimensions of genetic and environmental variation. For genetic influences there was both common and specific variance, and factor analysis implied at least five or six dimensions were needed to assess genetic variance. For environmental influences there was both common and specific variance, and factor analysis implied six or seven dimensions were needed to assess environmental variance (see figure 1.4.4.8).

Figure 1.4.4.8 Twin correlations for TPQ and EPQ-R scales

TPQ	MZ female	DZ female	MZ male	DZ male	DZ opp. sex
Harm Avoidance	0.44	0.20	0.42	-0.03	0.09
Novelty Seeking	0.42	0.14	0.35	0.06	0.07
Reward dependence	0.38	0.11	0.39	0.18	0.06
EPQ-R					
Extraversion	0.48	0.20	0.50	0.19	0.16
Neuroticism	0.45	0.22	0.34	0.04	0.10
Social conformity	0.43	0.28	0.27	0.20	0.16
Tough-mindedness	0.34	0.21	0.36	0.19	0.14

(Heath, Cloninger, Martin, 1994).

Subsequently, Stallings, Hewitt, Cloninger, Heath, Eaves (1996) assessed the revised four factor psychobiological model in approximately 3500 twins. The model assumes temperament dimensions reflect independent neurobiological systems, with any phenotypic correlation being due to socio-cultural influences. In line with this genetic factors contributing to each temperament dimensions were independent, and phenotype covariances were largely environmentally determined. However, model fitting suggested gender differences in personality structure as only three dimensions accounted for genetic variance in males (implying persistence is largely environmentally mediated) whereas four dimensions accounted for genetic variation in females. This needs to be addressed further where the sample was predominantly female (see Stallings, et al, 1996) (see figure 1.4.4.9).

Figure 1.4.4.9 Twin correlations for the revised TPQ scales

	MZ twins		DZ twins		
	Male	Female	Male	Female	Opposite sex
Novelty seeking	0.51	0.50	-0.01	0.26	0.14
Harm avoidance	0.33	0.44	0.11	0.23	0.07
Reward dependence	0.40	0.37	0.04	0.18	0.24
Persistence	0.35	0.22	0.35	0.10	0.03

(Stallings, Hewitt, Cloninger, Heath, Eaves, 1996)

The review by Loehlin (1992) showed different patterns for age to age stability, with genetic factors contributing to change in childhood, but environmental factors contributing to change in later adolescence and adulthood. There was no evidence of genetic influence at birth, but there was by one year, and by two years heritability estimates were of the same magnitude as traits in adults. For example, in the Louisville Twin Study five dimensions (orientation, affect-extraversion, activity, auditory-visual awareness, and motor-coordination) were derived from factor analysis of tester ratings for the Infant Behaviour Record. For each, MZ correlations were approximately twice as large as DZ correlations for infants at 3, 6, 9, 12, 18 and 24 months, although activity ratings at 9 months and below showed similar correlations for MZ and DZ twins. At 12 months, MZ concordance for fearfulness was 0.48, but at 18, 24 and 30 months MZ concordance was 0.70 (Matheny, 1985).

Across studies there were also normative trends of lower mean neuroticism and extraversion scores over time, which remained when the fact that family members were at different ages when tested was controlled for (Loehlin, 1992). Similarly, Carmichael & McGue (1994) looked at mother-offspring correlations for extraversion and neuroticism in a 19 year longitudinal study. Personality was first assessed when the offspring were 16 years and again at 35 years. A main effect of time was significant for extraversion, neuroticism and lie scales, with magnitude of mean change around $^{2}/_{3}$ of a standard deviation. Offspring means were closer to mother means at time two than at time one, however correlations remained at around 0.12. If age did make a difference then this would have been reflected in greater mother offspring similarity at retest. There was also evidence that change in offspring personality was not related to mother's personality, but stability in offspring personality was significantly related to mothers personality for neuroticism measures.

Evidence also shows genetic factors continue to contribute to stability of personality across the period from late adolescence to early adulthood. For measures at age twenty, and thirty there was some evidence of a normative decrease in mean neuroticism, an increase in mean constraint, and no change in positive emotionality. Mean cross-time twin correlations

showed much higher levels of correspondence for identical than fraternal twins, with genetic factors accounting for over 80% of stability (McGue, Bacon, Lykken, 1993).

Longitudinal data from middle aged and older twins reared together, and apart, showed correlations for genetic influences of nearly one between measures ten years apart, implying that the same genes act across adulthood, correlations for nonshared environment influences were 0.50 reflecting differences in life events, and phenotype correlations were 0.70. As correlations for genetic influences were higher, this implies that genes contribute more to trait stability than environmental factors (Pedersen, Plomin, McLearn, Friberg, 1988).

More recently, model fitting has demonstrated no significant change for the contribution of genetic and environmental factors assessed by EPQ scales across adulthood. For a total of 5400 twin pairs, cross-sectional data came from three samples, average ages, 23, 37, and 61 years and longitudinal data from two samples retested either during young/middle adult, or during middle/late adulthood. Measures of extraversion, neuroticism, psychoticism, and lie (conformity) were analysed separately by sex for MZ twins, DZ same and opposite sex twins. For all groups correlations were higher for MZ than DZ twins, and similar across gender and the three age groups (young, middle, old). Genetic and non-shared environmental factors contributed to extraversion and neuroticism, for psychoticism and lie, DZ correlations were more than half the MZ correlations implying some effect of shared environment. There was also some shared environmental effect for conformity, but findings were similar to studies in Finland, and America (see Loehlin & Martin, 2001).

Methodological issues

Discrepancies between the findings of twin and adoption studies of personality seem to be accounted for by non-additive genetic effects as correlations between MZ twins (who share both additive and non-additive genetic influences) are high, and correlations between DZ twins (who may share only some or none non-additive genetic effects are low) but similar to sibling correlations (see Plomin, Corley, Caspi, Fullker, DeFries, 1998).

Estimates based on parent report

There has been a consistent finding of low or negative correlations for parent reports of temperament in DZ twins. This may be due to contrast or assimilation effects. Contrast effects are where estimates are based on the behavioural contrast between members of a twin pair, and act to magnify measured behavioural differences. This has more impact on DZ estimates as they are behaviourally less alike than MZ twins. Assimilation effects are where similarities in twin behaviour are emphasised, which would over-estimate similarity in MZ twins in particular. Rater bias is important where this may cause the effects of shared environmental influences to be under or over estimated (Saudino, et al, 1995).

Neale & Stevenson (1989).

Neale & Stevenson (1989) directly addressed issues of rater bias by looking at whether parents consistently over-estimated or under-estimated the similarity of personality in twins. However, although there was some evidence of bias, this did not give the best fitting model, and the data was more in line with contrast effects.

Goldsmith, Buss, Lemery (1997).

Goldsmith, et al (1997) suggest contrast effects could also be related to psychometric properties of questionnaires as more specific questions may reduce the tendency to contrast twin behaviour. For example, there were no negative or zero correlations in analysis of infant data using the Infant Behaviour Record from the Louisville Twin Study (Matheny, 1995). Moreover, two adoption studies which used the Infant behaviour record showed genetic estimates of around 44% (Braungart, Plomin, DeFries, Fulker, 1992).

Van der Valk, vand den Oord, Verhullst, Boomsma (2001).

Van der Valk, et al (2001) directly addressed the extent that behavioural ratings reflect the child's phenotype in a large sample of three year old twins. Rater bias and psychometric models were fitted to mother and father ratings of behaviour problems. Rater bias models assume both parents assess the same behaviours in the child, whereas psychometric models assume that in addition to assessing common behaviours, each parent assesses a unique aspect of the child's behaviour. Consistent with studies in older children (see Hewitt, Sillberg, Neale, Eaves, Erikson, 1992; Rowe & Kandel, 1997) the psychometric model provided a significantly better fit to the data, with common behaviour accounting for 75%

of the variance. In addition 50% of the variance in common behaviours (75% was accounted for by genetic factors. For externalising problems 18% of the variance was accounted for by shared environmental factors, for unique behaviour, genetic, shared and non-environmental factors each contributed around 8% of the variance.

Peer measures of personality.

Generally, there has been high validity of peer-report measures. For a sample of nearly 1000 twin pairs, correlations for peer and self-report ratings were 0.55 and correlations for ratings by two different peers were 0.63, implying strong agreement. Genetic influences on peer ratings were similar to genetic influences on self-report ratings, and the evidence suggested that the same genetic factors contributed to self-report and peer ratings (Angleitner, Riemann, Strelau, 1995). However, bias has been found for extraversion measures, shown by differences in the mean and variance of self-report and co-twin ratings as a function of zygosity. Extraverts tended to underestimate, and introverts tend to over-estimate extraversion for their co-twin, although self-report and co-twin ratings were in agreement (Heath, Neale, Kessler, Eaves, 1992).

Non-additive genetic effects

High MZ correlations for personality do not appear to be artifactual, as the same pattern is found for twins reared together or apart, and self-report and peer reports are similar (DiLalla , Carey, Gottesman, Bouchard, 1996; Plomin, Corley, et al, 1998) . Discrepancies between twin and adoption studies are expected as adoption studies give narrow (additive genetic effects) estimates of heritability, whereas twin studies give broad (additive and non-additive genetic effects) (Plomin, et al, 1997; Rowe, 1997; Plomin, Corley, et al, 1998). Lower than expected parent-offspring correlations are not due to age differences in adoption studies as recent estimates from the Colorado Adoption Project are based on children of around sixteen, which is close to the age when their biological mothers were assessed (see Carmichael & McGue, 1994;Plomin, et al, 1998). However, correlations are similar for both DZ twins, and non-twin siblings, who both have a 25% chance of inheriting the same set of alleles at a locus, and could show some resemblance for both additive genetic variance, and non-additive dominance effects (Plomin, et al, 1998).

Consistent with this some non-additive genetic variance has been identified for all five factor traits (Bouchard, 1994) with the majority of studies showing MZ twin correlations which are more than twice DZ correlations (Plomin, Corley, et al, 1998). There is also evidence which implies that non-additive genetic effects contribute to sex differences. Heath, et al (1994) found that non-additive genetic effects contributed to neuroticism in females, when rater bias was controlled for, and, Finkel & McGue (1997) found evidence of non-additive genetic variance effects for males, not females, in a sample of over 1200 twin families.

Estimates of environments

Theoretical considerations suggest gene-environment correlations and interactions may be important to personality development and there is evidence that estimates of environmental effects may be reflecting genetic effects (Rowe, 1997; Plomin & Bergeman, 1991). For example, genetic influences contribute to controllable life events for females that are mediated by personality factors, and personality and life events are correlated (Saudino, Pedersen, Lichtensteing, McLearn, Plomin, et al, 1997). However, specific environmental factors which may be important to personality development are unknown. Most research on environmental influences has not been within a genetic design and quantitative genetic methodologies estimate variance accounted for by shared and non-shared environmental factors rather than directly assessing specific effects. For example, non-shared environmental influences are normally calculated as a residual and estimates include random error, and systematic bias, as well as true non-shared environment variance (McCrae, et al, 2001 p 515).

Environmental influences can also be modelled in terms of direction of effect. For example, 'vertical environmental transmission' specifically refers to environmental influences transmitted from one generation to the next, as opposed to environmental influences from peers or siblings. Vertical environmental transmission can be subdivided into direct phenotypic transmission (specific parent-offspring influences) and socio-cultural transmission (parent-offspring similarity from social and cultural influences outside the family) (Goldsmith, Gottesman, Lemery, 1997 p368).

Molecular genetic research

Cloninger's psychobiological model predicts that dopamine, serotonin, and norepinephrine genes should account for the majority of the variance in novelty seeking, harm avoidance and reward dependence respectively.

Molecular genetic studies have shown a relationship between personality measures and genetic variation in genes coding for neurotransmitter systems which support the model, although findings are complex. Some studies have looked at specific polymorphisms, and others have looked at the effects of multiple genes.

High scores on TPQ measures of novelty seeking have been associated with the long form of the D4 dopamine receptor gene (Ebstein, Novick, Umansky, Priel, Osher, Blaine, et al, 1996; Benjamin, Li, Patterson, Greenberg, 1996; Ebstein, Nemanov, Klotz, Ansenko, Belmaker, 1997; Ono, Manki, Yoshimura, Muramsitsu, Higuchi, Yagi, et al, 1997; Noble, Ozkaragoz, Ritchie, Zhang, Belin, Sparkes, 1998; Strobel, Wehr, Michel, Brocke, 1999). Patterns of findings are complex though as there have also been associations of novelty seeking to the short form of the the DRD4 gene (Malhotra, Virkkunen, Rooney, Eggert, Linnoila, Goldman, 1996;Gelernter, Kranzler, Coccoro, Siever, New, Mulgrew, et al, 1997; Ekelund, Lichtermann, Pelhonen, 1999) and non-replications (Jonsson, Nothen, Gustavsson, Neidt, Brene, Tylec, et al, 1997; Sander, Harms, Lesch, Dufeu, Kuhn, Hoebe, 1997; Sullivan, Fifield, Kennedy, Mulder, Sellman, Joyce, 1998; Kuhn, Meyer, Nothen, Gansicke, Papassotiropoulus, Maier, et al, 1999; Pogue-Geile, Ferrell, Deka, Debski, Manuck, 1998; Vanderbergh, Zonderman, Wang, Uhl, Costa, 1997; Herbst, Zonderman, McCrae, Costa, 2000). Inconsistencies could be due to several methodological or genetic phenomena, however, there is also the possibility that the initial association may have been due to linkage disequilibrium between the 48 repeat polymorphism, and another polymorphism in the DRD4 gene (see Ebstein, et al, 2000 p206).

There have been replications of associations for other dopamine genes. These include the dopamine receptor, DRD2 and the dopamine transporter (DAT) although again evidence is inconsistent (see Comings, Gade-Andavolu, Gonzales, Nobleman, Blake, Mann, et al, 2000). A polymorphism of the DRD3 receptor gene has also been associated with higher neuroticism and behavioural inhibition scores, also with a trend to higher anxiety and

depression scores although this was not significant, and was not replicated (see Ebstein, et al, 2000).

Harm avoidance and neuroticism have been associated with a region of the serotonin transporter promoter region (5-HTTLPR) (Lesch, Bengel, Heils, Sobol, Benjamin, Greenberg, Hodge, Sowinski, Nicoll, 1996; Lesch & Mossner, 1998;Ricketts, Hamer, Sage, Manowitz, Feng, Menza, 1998; Greenberg, Tolliver, Huang, Li, Bengel, Murphy, et al, 1999; Murakami, Shimomura, Kotani, Ikawa, Nanba, Adachi, 1999; Katsugari, Kunugi, Sano, Tsutsumi, Isogawa, Nanko, et al, 1999) although there have been some non-replications (Ebstein, Gritsenko, Nemarov, Frisch, Osheler, Belmaker, 1997; Mazzanti, Lappalainen, Long, Bengel, Naukkarrinen, Essurt, et al, 1998; Gelernter, et al, 1997; Kumakiri, Kodama, Shimizut, Yamanouchi, Okada, Noda, et al, 1999;de Brettes, Berlin, et al, 1998; Herbst, et al, 2000).

There is also some evidence that implies gender differences in the contribution of genes to personality. Association between the short allele of 5-HTTLPR and neuroticism was replicated only in males, not females. Moreover there was a significant effect of gender on mean neuroticism and agreeableness scores, but not of 5-HTTLPR genotype on either trait. This may explain inconsistencies in previous studies (Du, Bakish, Hrdina, 2000, abstract). There is also evidence that found that impulsiveness may be partially related to TPH and 5HT2A variance in males, not females (Evans, Reeves, Platt, Leibenau, Goldman, Jefferson, et al, 2000). However, Jorm, Prior, Sanson, Smart, Zhang, Easteal (2000, abstract) carried out a longitudinal study to look at whether development stage may be related to association of serotonin candidate genes and behavioural traits. For 660 children temperament was assessed from four to eight months to fifteen to sixteen years, and behaviour problems assessed from three to four years to fifteen to sixteen years. No significant associations were found at most ages, however, between 13 - 14 and 15 to 16 the long/long allele of 5HTTLPR was associated with higher anxiety, which is the opposite pattern to other studies.

Two studies have also shown an association between the serotonin-2C receptor and reward dependence (Ebstein, et al, 1997; Kuhn, et al, 1999). However, evidence is mixed for hypothesised links between reward dependence and the central noradrenergic system. A recent study did not find any association between adrenoreceptors linked to epinephrine

function and the reward dependence dimension of the TPQ scales (see Tsai, Wang, Hong, 2001).

Gene x gene interactions

There is evidence of epistatis (gene-gene interactions) effects between DRD2 and DRD4 polymorphisms associated with personality traits (Noble, 1998). Boys with three minor (A1, B1 and Intron 6 1) alleles of the DRD2 gene had significantly higher novelty seeking scores than boys without any of these alleles. The presence versus absence of the 7 repeat DRD4 allele, was also associated with significantly higher novelty seeking scores, but the greatest difference in novelty seeking scores was between boys with all three minor DRD2 alleles and the 7 repeat DRD4 allele and boys without any of these alleles. There has been replication of epistasis between 5HT and DRD4 polymorphisms in TPQ measures. Presence of both polymorphisms accounted for 30% of phenotype variance for persistence, and 13% of variance in reward dependence. Also between DRD4, 5-HTTLPR and catechol-o-methyltransferase for novelty seeking, the absence of the short 5-HTTLPR allele combined with the presence of the catechol-o-methyltransferase val/val genotype, is associated with novelty seeking if the DRD4 7 repeat is present. In a sibling design, siblings with identical patterns for these genes had significantly correlated novelty seeking scores, where siblings with dissimilar genotypes did not. In addition TAQ1A and a D2 receptor polymorphism were associated with harm avoidance scores in alcoholics (see Ebstein, Benjamin, Belmaker, 2000).

Significant associations between specific alleles of the DAT1 gene differentiated individuals with low scores for the dependence/independence facet of reward dependence, and individuals with 1/s or s/s for 5HTT had significantly lower scores for the first facet of harm avoidance. Norepinephrine transporter (NET) alleles were not significantly associated with any of the dimensions (Samochowiec, Rybakowski, Czerksi, Zakrezwski, Steplen, Pelka-Wyslecha, et al, 2001)

In a longitudinal design by Ebstein, Levine, Geller, Auerback, Gritsenko, Belmaker (1998) neonate behaviour measures (which were hypothesised to show less influence of caretaking environment) and temperament measures at two months, have been associated to DRD4 and 5-HTTLPR alleles. At two weeks infants with long alleles of DRD4 scored

higher for orientation, motor organisation and state regulation scales of the Brazelton Neonatal Behavioural Assessment Scale relative infants with the short alleles. There were also interaction effects, infants with short DRD4 and homozygous short alleles of 5HTTLPR had low scores for orientation relative to infants with long DRD4 and s/s 5HTTLPR alleles. Similar patterns accounted for 13% of the variance in novelty seeking in an adult sample. At two months long DRD4 alleles were associated with less negative emotionality relative to short DRD4 alleles, and s/s 5HTTLPR alleles were associated with more negative emotionality than either 1/1 or 1/s alleles (Auerbach, Geller, Lezer, Shinwell, Belmake, Levine, 1999).

At 12 months the infants were observed in a series of temperament tests designed to elicit fear, anger, pleasure, interest and activity. Long DRD4 alleles were associated with less interest in structured block play, less anger during mild physical restraint, and more activity in free play. Infants homozygous for short 5HTTLPR alleles were less fearful/distressed to stranger approach and showed less pleasure to structured play than infants with 1/1 or 1/s 5HTTLPR alleles. The duration of looking in structured play showed a significant interaction between DRD4 and 5HTTLPR alleles, and was shorter for infants with long DRD4 and s/s 5HTTLPR allele (Auerbach, Forey, Ebstein, Katania, Levine, 2001).

Burt, McGue, Iacono, Commings, McMurray, (2001) looked at the relationship between DRD2 and DRD4 alleles and MPQ personality measures at an individual, and family level in families from the Minesotta Twin Family Study. There was a significant sex by DRD4 interaction for the Harm Avoidance scale, long alleles were associated with high scores in males, and low scores in females. The A1 allele of DRD2 was associated with higher control scale scores however, there was no significant association of either polymorphism with the novelty seeking scale.

To overcome confounds from epistasis effects some research is now focusing on scans for multiple candidate genes. One study has looked at the relationship between groups of functional genes (59 candidate genes in total) and TCI traits. In an analysis which included all 59 genes, over 25% of the variance in reward dependence was accounted for by norepinephrine, however for novelty seeking serotonin accounted for more variance (22%) than dopamine (12.5%) and for harm avoidance all three groups of genes were equally

represented. In analysis which just included dopamine, serotonin and norephinephrine genes the ratio of dopamine to the other groups was highest for novelty seeking, and the relative ratio of norepinephrine was highest for reward dependence (Comings, Gade-Andavolu, Gonzalez, Liu, Muhle, Blake, et al, 2000).

1.4.5 RESEARCH FINDINGS FOR PSYCHOPATHOLOGY

1.4.5.1 INTERNALISING DISORDERS

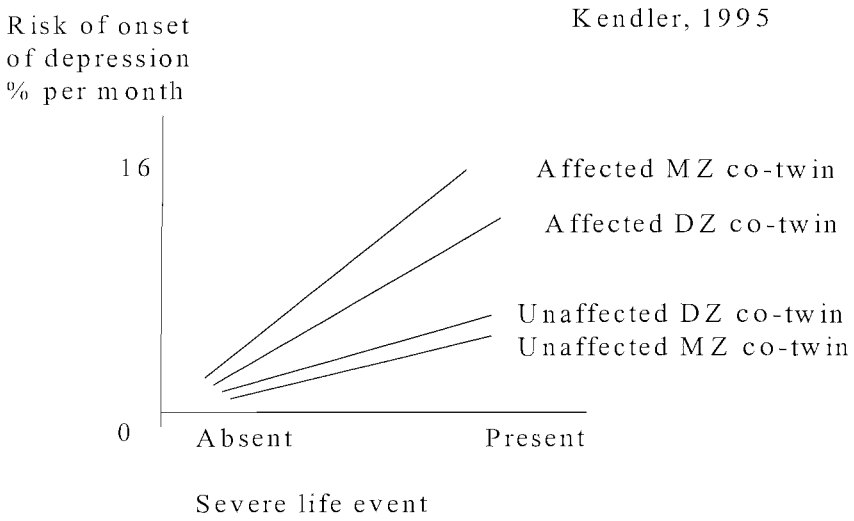
Depression

In adults heritability estimates for bipolar depression are approximately 80%, and for major depression 20 - 50%, however, increased rates of depressive symptoms in offspring of depressed parents are similar for both, and heritability of major depression may be under-estimated due to its episodic nature. There is some evidence of genetic anticipation (where a trait is more severe, or has earlier onset in subsequent generations) for bipolar depression, higher liability from maternal transmission, and higher genetic loading for offspring in families with multiply affected individuals.

The genetic liability for depression is broad with high rates of anxiety disorders in offspring, and common genetic contribution to both. There is also evidence of higher rates of alcoholism, substance abuse and anti-social behaviour in relatives of depressed children. Generally, separate assessment of maternal and paternal psychiatric histories, suggest that both parental concordance and comorbidity are influential on the relative risk of psychopathology in juvenile twins (Foley, Pickles, et al, 2001). However, parental depression, not comorbid, is still associated with significantly increased levels of depression and overanxious symptoms in offspring, the risk is higher for females, for maternal depression, and if both parents are depressed (see reviews by Rutter, et al, 1999; Kendler, 2001; Foley, Pickles, et al, 2001; Boomsma, Beem, van den Berg, Dolan, Koopmans, Vink, et al 2002). A follow-up study found some evidence of family specificity in comorbidity of anxiety and depression in children found evidence of some family specificity and stability for comorbidity of depression and anxiety alone (Avenevoli, Stolar, Dierker, Merikangis, 2001).

Much of the liability to depression is mediated by stressful life events. Higher rates of life events are found in families with a depressed proband, and twin studies have shown a genetic contribution to life events which is carried by personality (see Rutter, et al, 1999;Roberts & Kendler, 1999; Kendler, 2001). For example, Kendler, Kessler, Waites, McClean, Sham, Neale, et al (1995) compared onset of depression with the absence or presence of a serious life event during the preceding month in individuals at different genetic risk. For co-twins with a depressed proband, risk of onset increased in the presence of a life event and this increase was greater for MZ than DZ cotwins (see figure 1.4.5.1.1.).

Figure 1.4.5.1.1 Risk of onset of depression for affected and unaffected co-twins



In addition Kendler & Karkowski-Shuman (1997) looked at whether genetic liability to depression was associated with differential risk for life events. They found genetic liability to major depression in females was associated with a significantly increased risk for life events (assault, serious marital problems, divorce/break-up, job loss, serious illness, financial problems and trouble getting along with relatives/friends) which was not due to life events occurring during depressive episodes. In contrast genetic liability to alcoholism in females was associated with an increased risk for legal problems.

There have been no twin studies of clinically diagnosed childhood depression (Rutter, et al, 1999) but dimensional measures from the Virginia Twin Study showed heritability of around 48%. However, findings have been inconsistent. For children and adolescents (aged eight to sixteen) a genetic influence was only found for adolescent girls

(concordance rates were 0.37 for MZ twins and 0.09 for DZ twins) implying that genetic factors accounted for 28.3% of the variance in females, and that long term consistency of depression in females was mainly due to genetic factors. For males genetic factors decreased at adolescence. Genetic influences accounted for between 49 and 91% of the variance of measured life events, and there was a significant effect of life events on depression, which was stronger for females, and increased with age. There was also evidence of some common genetic influences on the two measures, implying that part of the genetic liability for depression is mediated through a genetic liability to life events. Findings were consistent with patterns found in adults (Silberg, Pickles, Rutter, Hewitt, Simonoff, Maes, et al, 1999; Thapar, Harold, McGuffin, 1998) and there is also evidence that loss events are associated with depression and threat events are associated with anxiety in children and adolescents (see Eley, Stevenson, 2000).

Anxiety disorders

Generally findings are similar across anxiety disorders. For broadly defined panic and generalised anxiety heritability estimates are approximately 43% and 37% respectively, in clinical and population samples. There has been less research on childhood anxiety and findings have been inconsistent. However, there were higher MZ than DZ correlations in seven year old twins for both physiological and social anxiety symptoms, suggesting a genetic contribution (Warren, Schmitz, Emde, 1999). Twin and family studies have shown heritability estimates of between 35 - 60% for eating disorders. Shared environmental influences were not important in late adolescence, although there is some evidence that they may be important in childhood (see review Kendler, 2001). Heritability estimates for fears and phobias range between 43 and 67% in adults twins (Kendler, Karrowski, Prescott, 1999) and are around 29% in children, with no evidence of differences between extreme and moderate fears (see Stevenson,Batten, Cherner, 1992). There is also consistent evidence that social phobia is familial, that family aggregation is fairly specific, also that the more severe generalised social phobia may be more heritable (see Stein, Chartier, Lizak, Jang, 2001).

Eley & Stevenson (1999) found higher genetic loadings in males than females, in self-reports for anxiety and depression. Univariate analysis showed significant effects of age and gender on the variance in both anxiety and depression. For depression heritability was

higher and shared environment less important for males relative to females, however heritability estimates were higher and shared environmental factors less important for adolescents relative to children. This was accounted for by higher heritability estimates for depression in adolescent males, and higher shared environmental factors for adolescent females relative to males. For anxiety genetic, shared and non-shared environmental factors contributed equally to the variance for children and adolescent males, however, for adolescent females both genetic and shared environmental factors were larger. Bivariate analysis showed that common genetic influences contributed to anxiety and depression for all four groups, which is consistent with other studies.

Molecular genetic studies

Serotonin

Serotonin (5HTTLPR and VNTR-17) polymorphisms are associated with internalising disorders. Twin studies have shown that 5HT uptake is genetically controlled and there is evidence of lower function in first degree relatives of depressed probands. 5HT variants have been associated to bipolar disorder, obsessive compulsive disorders, bipolar depression, anxiety, and substance abuse. Combined population and family studies on two separate groups show association to traits related to anxiety symptoms and depression, accounting for 3 - 5% of the total variance, and 7 - 9% of the genetic variance, although there have been inconsistencies (see Lesch, et al, 1996; Lesch & Mossner, 1998). There is also evidence that the density of 5-HT2A receptors are altered in brain regions of depressed suicide victims, suicide subjects, individuals with major depression and schizophrenia (see Du, et al, 2000). An association between the c allele of 102T/C polymorphism in the 5-HT2A receptor gene and schizophrenia has been replicated, and Du, et al (2000) found significant association between this allele and genotype frequency and depression if suicide ideation was included as a marker. Manipulation of serotonin 5HT in both animals and humans results in changes in feeding behaviour. Preferential transmission of the A allele of a polymorphism of the 5HT2A receptor gene promoter region (-1438A/G) with anorexia nervosa has been replicated, but findings are inconsistent (see Nishiguchi Matsushita, Susuki, Murrayami, Shirawaka, Higuchi, (2001).

Dopamine

Pharmacological evidence also suggests a role for dopamine in bipolar depression, and mania in particular. For example, cocaine and amphetamine increase dopamine concentration by acting at DAT to inhibit dopamine reuptake. The effects of psychostimulants resemble mania, and chronic use of psychostimulants can trigger mania in bipolar patients, and psychosis in nonbipolar patients. Moreover, L-dihydroxyphenylalanine increases dopamine transmission and has been observed to precipitate mania, and antipsychotic drugs which treat mania block dopamine receptors (see Greenwood, Alexander, Keck, McElroy, Sadovnick, Rennick, et al, 2001 p 145).

There is some evidence of linkage between the DAT1 locus on chromosome 5p15.3 and bipolar disorder implying that DAT may be important in some familial transmission of bipolar disorder. A second study by the same research group found evidence of linkage disequilibrium for a haplotype marker consisting of 5 single polymorphism nucleotides (SNP's) of the same region of the DAT gene in parent-proband triads (Greenwood, et al, 2001). Significant linkage of markers for the p16 region of the short arm of chromosome 4, which the dopamine type 5 receptor (DRD5) maps to, has been established for bipolar disorder, and schizoaffective disorder, although there have been inconsistencies (see Muir, Thomson, McKeon, Mynett-Johnsen, Whitton, Evans,et al, 2001).

Rowe, Stever, et al (1998) looked at the relationship between DAT1 and internalising disorders (generalised anxiety, major depression, obessive-compulsive, panic, separation anxiety, social phobia, specific phobia, tourettes) in clinical probands and their first degree relatives relative to controls and their first degree relatives. Between-family association was tested by the association of DAT1 genotypes with disorder symptoms in the population. For all eight disorders symptoms increased with a greater number of 10 repeat alleles. Linkage and within-family association tested using quantitative transmission disequilibrium testing (QTDT) was indicated by increased symptom scores in children who received 10 repeat alleles from heterozygous parents relative to children who received 9 repeats.

There have been conflicting results from studies which have looked at links between X chromosomal regions and bipolar affective disorder. However preferential transmission of

the c allele of a polymorphism in the pseudoautosomal X-linked gene SYBL1 in BPAD probands has been shown in an American and German sample (Muller, Schulze, Jahnes, Cichon, Krauss, Kasner, et al, 2002). However, Kornberg, Brown, Sadovnick, Remick, Keck, McElroy, et al (2000) looked at parent-of-origin effects in a sample of affected proband families (over 1000 individuals) participating in a linkage studies. They found no significant differences in the rate of illness of mothers and fathers of affected probands, but the rate of bipolar affective disorder was significantly higher in offspring of fathers with BPAD than in the offspring of mothers with BPAD implying a possible parent-of-origin effect.

Smoller, Rosenbaum, Biederman, Susswein,, Kennedy, Kagan (2001) used behavioural inhibition as a phenotype measure of anxiety disorder and found an inverse association between behavioural inhibition and the glutamic acid decarboxylase gene (65 kDA isoform) which encodes an enzyme involved in GABA synthesis, in families with children classified as behaviourally inhibited. This is consistent with evidence that GAD65 knock-out mice express increased anxiety related behavioural inhibition (see Kash, Tecott, Hodge, Baekksekovl, 1997) and mice heterozygous for the GABA receptor $\gamma 2$ subunit show similar behaviour to anxiety disorder indiviuals, as they avoid threat, and treat ambiguous cues as threatening, showing more sensitivity to anxiolytic drugs. In particular these mice show a 33% reduction in $\gamma 2$ subunits in the hippocampus, cingulate cortex and prefrontal cortex, and only a 5 - 15% reduction in other areas of the brain. Based on this Crestani, Loez, Baer, Essrich, Benke, Laurant, et al (1999) hypothesise that impairment of septo-hippocampal function underlies the effects of anxiolytic drugs on behavioural inhibition and influences other brain regions that underlie hippocampal processing.

However, McNaughton (1999) suggests that there is a need to distinguish between anxiety disorders due to differences in the underlying neural structures which mediate anxious behaviours (see McNaughton, 1999: Crestani, et al, 1999).

Gratacos, Nadd, Martin-Santos, Pujana, Gago, Peral, et al (2001) looked at the co-occurrence of panic and phobic disorders with joint laxity (patients with joint laxity show an increased rate of panic disorder/agoraphobia/simple phobia relative to controls) in multiply affected families. They identified an interstital duplication of chromosome 15q 24-26 (DUP25) which was significantly associated with panic disorder/agoraphobia/social

phobia and joint laxity, and with panic disorder in non-familial cases. 90% of patients with one or several anxiety disorders had the duplication, all panic disorder and social phobia patients did, and DUP25 was present in 87% of joint laxity patients. There was linkage with significant lod scores if all the disorders were included, also an association between anxiety and DUP25, which increased if joint laxity was included. Findings were replicated in 70 non-familial cases, DUP25 was present in 68 patients, but only in 7% of controls.

1.4.5.2 EXTERNALISING PROBLEMS

Attention deficit hyperactivity disorder

Family, twin, and adoption studies have shown a significant genetic contribution to the etiology of ADHD, with heritability estimates of around 0.70 to 0.90 (ranging from 0.54 to 0.98) (see Rutter, et al, 1999; Rhee, Waldman, Hay, Levy, 1999). There is an increased risk for first degree relatives (parents and siblings) of ADHD children for both ADHD and other psychiatric disorders (Silberg , Eaves, Simonoff, Maes, Murelli, Pickles, et al 1997) Prevalence for ADHD is around 5 times higher than the population risk, and for anti-social disorders around 3 times higher than the population risk (Biederman, Faraone, Keenan, Benjamin, et al, 1992). There is no evidence though of increased risk for second degree relatives, (grandparents, uncles etc.) of ADHD children except for grandmothers (Faraone et al, 1994, in Rutter et al, 1999).

The relative risk for first degree relatives of females with ADHD is less clear. Some evidence suggests that this is similar to patterns of risk among relatives of males (Faraone, Biederman, Freedman, 2000). Although, other studies have shown a higher familial loading is needed for relatives of females. In affected sibling pairs 55% had at least one lifetime ADHD parent, but for pairs with at least one female this was 64%, compared to 43% for pairs with at least one male (Smalley, McGough, DelHomme, NewDelman, Gordon, Tae, et al, 2000) which is more consistent with the lower prevalence of ADHD found in females. There is also some evidence that shared environmental factors may be more important for females, and that there may be dominance effects for males (Rhee, Waldman, et al, 1999).

Twin studies have shown large differences in concordance rates between MZ and DZ twins (Goodman et al,1989; Gillis, Gilger, Pennington, DeFries, 1992; Stevenson, Pennington,Gilger, DeFries, Gillis, 1993; Hudziack, Rudiger, Neale, Heath, Todd, 2000) with average heritability of around 0.80 (Faraone, 2000). This suggests a strong genetic component for ADHD. Genetic and environmental influences on AHDH are relatively stable across childhood and adolescence (Gjone, Stevenson, Sundet, 1996). Also, common genetic liability has been found for males and females (Goodman et al, 1989; Gjone, et al 1996; Rhee et al, 1999), and for comorbidity with spelling difficulties, and comorbidity with conduct disorders (Stevenson, et al, 1993; Levy, Hay, McStephen, Wood, Waldman, 1997, Silberg et al, 1996). There is evidence though that genetic liability may get more specific in adolescence (Silberg Rutter, Meyer, Maes, Hewitt, Simonoff, et al, 1996). Estimates of heritability do not differ for either continuous or categorical phenotype definitions (Silberg et al, 1996; Levy et al, 1997) or for broad or narrow phenotype definitions (Silberg et al, 1997, in Rutter et al, 1999). Generally, non-shared environmental influences account for most of the environmental influences on ADHD (Waldman, Rowe, Abramowitz, Kozal, Mohr, Sherman, et al, 1998). However ADHD and hyperactivity scales, especially when parent-reported tend to show sibling competition effects (see Stevenson, Asherson, Hay, Levy, Swanson, et al, 2005).

There is also evidence of no differences in etiology for specific behaviour problems. Attention problems assessed by the Child Behaviour Checklist showed substantial genetic influences for both males and females across children (5 to 9 years) and adolescents (12 to 15 years) in a large general population sample. Heritability estimates were between 0.73 and 0.76, with the best fitting model including additive genetic and non-shared environmental variance. There was no significant difference in the magnitude of genetic variance for mild or severe symptoms when cerebral palsy, epilepsy and low birth weight were controlled for. Heritability estimates were similar to those found in other studies which used dimensional measures (Gjone, Stevenson, Sundet, 1996; Goodman & Stevenson, 1989; Gillis, et al, 1992).

Early adoption studies of ADHD showed results consistent with other research methods, although there were some methodological problems (Tannock, 1998). More recent evidence suggests rates of ADHD for biological parents of ADHD children are significantly increased in comparison to rates for both adoptive parents of ADHD children,

and non-adoptive parents of unaffected children (Sprich, Biederman, Crawford, Mundy, Faraone, 2000).

Multifactorial inheritance for ADHD is implied by the continuous nature of behavioural symptoms, and in complex seggragation analyses which imply multiple genetic influences. Hetereogeneity is implied by the high rate of comorbidity between ADHD and other cognitive and psychiatric disorders (see Smalley, McCracken, McGough, 2001 p31). However, there is no evidence of family clustering of DSM-IV subtypes (Faraone et al, 2000) even for affected sibling pairs which would be expected to be more genetically similar (Smalley, et al, 2000). There is also debate about distinctions between ADHD, conduct disorder and oppositional defiance disorders given the extremely high rates of comorbidity between these disorders, and high concordance between behavioural ratings of ADHD and CD. To date results from studies which have looked at factors underlying comorbidity have been inconclusive. For example, Burt, Krueger, McGue, Iacono (2001) found shared environment factors accounted for comorbidity in 11 year old twins, whereas, other studies by Nadder, Sillberg, Eaves, Maes, Meyer (1998) and Young, Smolen, Stallings, Corley, Hewitt (2000) in older individuals found covariance was mainly due to genetic factors (see Rutter, Silberg, et al, 1999; Burt, Krueger, et al, 2001).

Several large studies by the same research group suggest a sub-type of ADHD which is comorbid with either conduct-disorder, or bipolar depression. This sub-type acted as a risk factor for poor outcome and was more familial with 84% of adult ADHD probands having at least one affected child, and 52% two or more affected children (Faraone, Biederman, Mennis, Russell, Tsuang, 1998). Three potential models for familial clustering of ADHD subtypes, comorbid learning disorders and conduct disorder in affected sibling pair families were tested in the ongoing UCLA ADHD Genetic Study (Smalley, et al, 2001). These were 1) ADHD and the comorbid condition is due to a specific set of genes that produce the phenotype expression of both conditions, 2) the relationship between ADHD and the comorbid condition is due to non-genetic phenomena, and 3) the relationship between ADHD and the comorbid condition is due to common susceptibility genes. Results implied that CD may reflect a subtype of ADHD with a distinct etiology, also that common genes seemed to contribute to ADHD subtypes. However it was unclear if comorbidity between ADHD and learning disorders was due to either model 1 or model 2.

Molecular genetic evidence

Dopamine is particularly implicated in the aetiology of disorders like ADHD (Swanson, Oosterlaan, Murias, Shick, Flodman, Spence, et al 2000). Several studies have replicated an association between the 7 repeat 48 bp allele of exon III of the dopamine D4 receptor gene with ADHD (Mill, Curran, Kent, Richards, Gould, Virdee, et al, 2001 and see personality section), and there have been replications of an association between ADHD and a polymorphism of the dopamine transporter gene (DAT1) (Cook, Stein, Cragowski, Cox, Olken, Keiffer, et al, 1995; Gill, Daly, Heron, Hawi, Fitzgerald , 1997; Waldman et al, 1998; Daly, Hawi, Fitzgerald, Gill, 1999). In particular Todd & Omalley (2001) showed replication of 5' 120 bp tandem duplication polymorphism in the gene encoding for the dopamine DRD4 receptor. Payton, Holmes, Barrett, Hever, Fitzpatrick, Trumper, et al (2001) found increased frequency of DRD4 7 repeat alleles and DAT1 48 bp repeat associated with high scoring concordant MZ twins in a population twin sample.

A specific region of DAT (3') has been implicated in the etiology of ADHD, psychosis in cocaine abusers and alcoholism. Moreover, family studies have suggested a possible genetic relationship between ADHD and bipolar disorder, which implies that the 3' region of the DAT gene may be linked to several behavioural syndromes (see Greenwood, Alexander, et al, 2001 p 150).

One study has shown an association between a common 44 bp deletion in the promoter region of the serotonin transporter (5-HTTLPR) and ADHD. A haplotype relative risk design was used with 98 parent-child triads. A significant decrease in the short/short 5-HTTLPR genotype was found relative to controls, although this was only significant for the 'combined' subtype (Manor, et al, 2001).

Fisher, Francks, McCracken, McGough, Marlow, McPhee et al (2002) carried out a genomewide scan for loci involved in ADHD. This implied that any single gene or X-linked effect was unlikely in that sample. Lod scores greater than 1.5 were found for regions on 5p 12, 10q 26, 12q 25 and 16p 15. Quantitative trait analysis of ADHD symptom counts implied a region on 12p 13 (lod score of 2.6). However of 36 candidate genes only DRD5, 5HTT and CALCYON coincided with sites of positive linkage and 2q 24 and 16p 13 coincided with linkage reports in genome scans of autistic sibpairs.

Conduct disorder and oppositional defiant disorder

Genetic research into CD and ODD had been conflicting, with some studies showing a strong genetic component for each, and others that shared environmental factors are more important (see Rutter, Silberg, et al, 1999; Eaves, Sillberg, Meyer, Maes, Simonoff, Pickles, et al, 1997; Slutske, Heath, Dinwiddie, Madden, Bucholz, Dunn, et al, 1998, Silberg et al, 1996, Nadder, Sillberg, Eaves, Maes, Meyer, 1998). Distinctions have been made between life-course persistent and adolescent antisocial behaviour (Moffit, 1993; Rutter, et al, 1999). CD in childhood with or without comorbid ADHD is associated with later aggressive behaviour, delinquency, and substance abuse, whereas attentional problems alone are not (see Burt, Krueger, et al, 2001). A study which looked at the covatiation between ADHD, CD, ODD, in a large sample of eleven year old twins did find specific genetic and environmental effects for each disorder. For all three MZ correlations were significantly greater than DZ correlations, (heritability estimates were 0.57, 0.65, 0.39 respectively). However, for ODD and CD in boys, genetic contributions were lower implying shared environmental factors contributed more. There was also evidence that common genetic liability accounted for 32%, 35%, and 22% of the covariance between CD and ODD, between AHDH and CD, and between ADHD and ODD, respectively. A single shared environmental factor accounted for more covariance in symptoms, 47% of covariance between ADHD and CD, 59% of the covariation between ADHD and ODD, and 50% of the covariance between CD and ODD. Non-shared environmental factors contributed to around 18% of covariances (Burt, Krueger, et al, 2001).

Antisocial behaviour

For juvenille antisocial behaviour, genetic, shared and non-shared environmental factors, were found to contribute equally to variance when both retrospective, and later self-report measures were combined (Jacobson, Prescott, Kendler, 2000). Twin and adoption studies have shown that suicidality and impulsive aggression (behaviours associated with personality disorders) are heritable. Heritability estimates for suicidality are 45%, and between 20 - 62% for impulsive aggression, and between 0.30 and 0.70 for impulsivity and aggression, with mainly non-shared environmental factors being important. There is also evidence that the phenotypic covariation between aggression and impulsivity measures is genetically mediated. Increased aggression in serotonin 1B knock-out mice have

implicated serotonin abnormalities in their etiology (see New, Gelernter,Goodman, Mitropolou, Koenigsberg, Silverman, Siever, 2001: Manuck, Flory, Ferrell, Mann, Muldoon, 2001).

A meta-analysis of twin and adoption studies into aggression related personality traits by Miles & Carey (1997) showed that both genetic (50%) and shared environmental factors contributed to variance, however evidence suggested that genetic influences increased and shared environmental factors decreased over development which is a similar pattern found to studies of adult antisocial behaviour and criminality. (see Miles & Carey, 1997: Billig, Hershberger, Iacono, McGue, 1996; Finkel & McGue, 1997).

Gene-environment interactions

There is evidence of gene-environment interactions for criminal behaviour. Data from over 14,000 adoptions in Denmark showed a higher rate of criminal convictions in adoptees whose biological parents had criminal records, also that this increased if the adoptive parents also had a criminal record (Mednick, Gabrielli, Hutchings, 1984). Similarly, Bohman (1996) found a gene-environment interaction between type of criminality, which interacted with alcohol abuse. In another study antisocial behaviour was increased for adolescent adoptees, who had biological parents with antisocial behaviour, and also experienced negative adoptive environments (see Cadoret, Yates, Troughton, Woodsworth, Stewart, 1995).

Comorbidity

Genetic factors account for around 50% of the covariance between ADHD, CD, and ODD (see Nadder, et al, 1998, Silberg, et al, 1996). However, combordity causes problems where there is evidence of assortitative or non-random mating for people with anti-social disorders (Rutter et al, 1999). This has implications for research in terms of inflating genetic estimates (Krueger, Moffitt, Caspi, Benke, Silva, 1998).

Molecular genetic evidence

In general, animal and human studies have shown an association between low serotonin activity and impulsiveness, aggression, and disinhibited behaviour. Physiological measures in children and adolescents have established that serotonergic brain pathways play a role in aggression and conduct disorder and both conduct and oppositional defiant disorder are commonly comorbid with ADHD. Caspi, McClay, Moffitt, Mill, Martin, Craig,et al (2002) found significant interactions between variation in the promoter region of the monamine oxadise A (MOAO) gene and the effects of maltreatment in early childhood (assessed across four separate measures) in the development of anti-social behaviour in individuals from the Dunedin Multidisciplinary Health and Development Study.

Substance abuse

Evidence from twin, adoption and family studies show strong evidence for a genetic contribution to substance abuse. Children of parents with substance abuse disorders are at increased risk of developing early onset substance abuse. Children of alcoholics, are approximately four to five times more likely than children of non-alcoholics to develop alcoholism, and are at increased risk of substance abuse, children of parents with cocaine and opioid dependence are also at increased risk of substance abuse. Heritability estimates range from 0.4 to 0.6 (see Iacono, Carlson, Taylor, Elkins, McGue, 1999). There are also increased rates of drug disorders in first degree relatives of drug addicts relative to controls, in adopted offspring of biological parents with substance disorder, with no increase when adoptive parents have substance abuse problems (see Iacono, et al , 1999; Noble, 2000; Duaux, Krebs, Loo, Pilier, 2000) (see figure 1.4.5.2.1).

Figure 1.4.5.2.1 Heritability for substance use in adolescents

	Male	Females	Combined
Alcohol	0.59	0.11	0.36
Tobacco	0.60	0.10	0.35
Other drugs	0.33	0.11	0.23

(Iacono, Scott, Carlson, Taylor, Elkins, McGue, 1999)

Familial transmission of substance abuse shows a generalised rather than specific pattern, with heterogeneity of symptoms and high rates of comorbidity both between substance

abuse disorders, and between substance abuse disorders and other psychopathology (Bierut, Dinwiddie, Begleiter, Crow, Hesselbrock, Nurnberger, et al, 1998; Kessler, Crum, Warner, Nelson, Schulsber, Anthony, 1997). However, data from the Minnesota Twin Study suggests comorbidity of substance abuse disorders may be due to common environment factors which accounted for 63% of the covariance between alcohol, tobacco and other drug use. There is also evidence of differences in heritability between early and late onset alcoholism, with genetic factors accounting for 90% and 40% of variance respectively (Sigvardson, Bohman, Cloninger, 1996).

Common genetic factors contribute to comorbidity between alcohol dependence and retrospective reports of CD in childhood in adult twins (17% and 35% for men and women respectively, and 11% and 23% respectively for total liability to alcohol dependence). The rest of the variance came from non-shared environmental factors (Slutske, Heath, et al, 1998).

Molecular genetic evidence

Variants of the DRD1, DRD2, DRD3, DRD4 genes and serotonin genes have been associated with substance abuse, although there have been non-replications, and high comorbidity between alcoholism and bipolar depression was not accounted for by DRD2 variants (see Duaux, et al, 2000; Gorwood, Bellivier, Ades, Leboyer, 2000, de Brettes, Berlin, et al, 1998). A meta-analysis of studies focusing on substance abuse disorders has shown that Taq1 D2 dopamine receptor A1 alleles are significantly more frequent in severe alcoholics (a threefold increase) but that they do not differentiate between less severe alcoholics and controls, which may account for some of the discrepancies. Variants of Taq1B alleles which are in linkage disequilibrium with the Taq1A site are also associated with alcoholism, and in general the DRD2 gene has been associated with cocaine, nicotine and opioid dependence, together with obesity and gambling, has been implicated in Tourettes syndrome, post-traumatic stress disorder, and specific symptoms linked to affective disorders and schizophrenia. Other variants have been linked to Parkisons disease and certain movement disorders.

Genetic linkage has been shown between alcohol abuse associated with antisocial behaviour and the serotonin 5 - HTIB receptor gene associated with aggression and

impulsivity in two populations (Lappalainen, Virkkunen, Dean, Ozaki, Linoila, Goldman (1998). There has also been association shown between a polymorphism of the gene for tryptophan hydroxylase, (TPH), and aggressiveness, also suicidality and hostility (see Manuck, Flory et al, 2000; Cloninger, 1987).

1.4.5.3 DIMENSIONAL MEASURES OF INTERNALISING AND EXTERNALISING SYMPTOMS IN CHILDREN

Population studies of behavioural difficulties in children show little difference between, heritability of continuous behavioural traits and extreme symptoms.

Gjone, Stevenson, Sundet, Eilertson (1996)

In a sample of children and adolescents (5 - 15 years) from five birth cohorts, these showed high heritability estimates for both internalising and externalising disorders. There was some evidence of differential patterns of etiology with severity. For externalising behaviour heritability was slightly higher and shared environment factors less important with increasing severity. For internalising behaviour this pattern was only shown for children aged 5 - 6, and 8 - 9 years. This is consistent with other studies which have found higher genetic contributions to more severe psychopathology. However, overall differences in heritability were non-significant (Gjone, et al, 1996).

Deater-Deckard, Reiss, Hetherington, Plomin (1997)

Deater-Deckard, et al (1997) compared individual and group heritability for adolescents in a twin/step family design. An extreme group was selected based on high scores, (one standard deviation above the mean), on the Child Behaviour Checklist. For the unselected individuals intra-class correlations were higher for MZ than DZ twins, across both interalising and externalising symptoms. A model with genetic, shared and non-shared environmental parameters estimated genetic contributions to behaviour problems of around 60%, and a shared environmental contribution of 10%. In contrast to the study by Gjone, et al (1996) genetic estimates for group heritability of the selected sample were slightly lower, and shared environmental contributions were slightly higher, which implies

environmental contributions may be more important for severe symptoms, but again the difference was not significant.

Group heritability was estimated by both liability threshold and DF extremes models. In a liability threshold model a cut-off point is estimated in a continuous underlying distribution, and liability correlations represent the proportion of difference between the proband and the population mean accounted for by differences between the proband sibling mean and population mean. If genetic or environmental factors are influential on a trait relatives of probands will have greater liability. In contrast there is no assumed cut-of point in DF extremes analysis which estimates family resemblance by the extent that the co-twin mean regresses back to the population mean. Very similar patterns were obtained for both a DF extremes model, and a liability threshold model, which is consistent with dimensional measures representing the underlying distribution assumed in the liability threshold model (Deater-Deckard, et al, 1997 p522).

Gjone & Stevenson (1997)

Models with genetic, shared and non-shared environmental parameters account for comorbidity between dimensional measures of internalising and externalising symptoms. In four groups of children, (males 5 to 9 years, females 5 - 9 years, males 12 to 15 years, females 12 - 15 years), covariance between internalising and externalising disorders was between 0.51 and 0.58, and mainly accounted for by shared environmental factors, particularly in the younger age groups (Gjone & Stevenson, (1997).

Van den Oord, Boomsma, Verhulst (2000)

Van den Oord, et al (2000) looked at the co-occurance of behaviour problems (oppositional, withdrawn/depressed/ aggressive, anxious, overactive, and sleep problems), in three year old twins. The best fitting model showed that genetic factors accounted for 37.3%, shared environmental factors accounted for 51.2%, and non-shared environmental factors accounted for 11.4% of the variance in phenotype correlations. This shows that shared environmental influences were important to comorbidity of behaviour problems, and there was also evidence that genetic and environmental factors were associated with different clustering of disorders. Factor analysis of CBCL scores showed that the highest

loadings on an externalising factor were for oppositional, aggressive and overactive behaviours. The highest loadings on an internalising factor were withdrawn/depressed and anxious symptoms. However, there were substantial cross-loadings for oppositional, and withdrawn/depressed had a higher factor loading on the externalising factor, which suggests a broad distinction of internalising and externalisations includes some overlap, and may explain some of the discrepancies in other studies.

Hudziack, Rudiger, et al (2000)

Hudziack, et al (2000) looked at dimensional measures of problem behaviour in twins (n = approximately 500) aged eight to twelve years. Heritability estimates for attention problems were 60- 68%, for aggression, 70 - 77%, and for 61 - 65% anxious/depressed behaviours. Rater bias terms were included, but the best fitting models had genetic and non-shared environmental parameters, except for aggressive behaviour in boys which showed some shared environmental effect.

1.4.5.4 NEURODEVELOPMENTAL DISORDERS AND COGNITION

As discussed earlier, psychopathology is associated with variation in cognitive ability and neurobehavioural deficits. To look at this, genetic studies of specific neurodevelopmental disorders and cognitive abilities will be reviewed, together with theoretical arguments regarding the role of early development in understanding the relationships between psychopathology and cognitive development.

Schizophrenia

Genetic research into schizophrenia is adult based given the age of typical onset. However, there is evidence of neurobehavioural and social difficulties in children later diagnosed as schizophrenic, and family studies show impaired attention is developmentally stable and predictive of future disorder in offspring of schizophrenic probands (Cornblatt & Malhotra, 2001).

Family and twin studies

A study by Heston (1966) showed an 11% risk for schizophrenia in adopted-away offspring of schizophrenic mothers compared to a 0% risk in adopted-away offspring whose parents had no mental health problems. Later studies have confirmed this as rates of schizophrenia are considerably higher in the biological relatives of schizophrenic probands, and in adopted away offspring, but not for adoptees with schizophrenic adoptive parents and no biological family history of schizoprenia. Across twin studies concordance is 0.46 and 0.14 respectively for MZ and DZ twins, and heritability estimates overall average 0.75 - 0.84. Evidence also consistently shows that familial loading for schizophrenia includes delusional, schizo-affective, and schizotypal disorders although there have been some contradictory findings (see reviews Rutter, Silberg, et al, 1999; Kendler, 2001; Cardno, Gottesman, 2000: Ingraham & Kety, 2000).

There is also evidence of variation in brain asymmetry and genetic transmission of schizophrenia. A functional magnetic resonance imaging study by Sharma, Lancaster, Sigmundson, Lewis, Takei, Gurling, et al (1998) showed relatives of schizophrenic probands from the Maudsley Family Study, who appear to be transmitting liability had a loss of normal brain asymmetry. Similar to the probands, presumed obligate carriers lacked asymmetry in the prefrontal sensorimotor occipito-parietal cortical regions, while presumed non-obligate carriers only showed a lack of asymmetry in the occipito-parietal region.

Prenatal and perinatal factors associated with schizophrenia

A relationship between pregnancy, birth complications (PCB) and schizophrenia has been found. There is higher concordance for schizophrenia in MZ twins discordant, relative to concordant for handedness (Davis & Phelps, Bracha, 1995). There was a significant increase of both low birthweight children and schizophrenia spectrum disorders for children born during the Dutch famine in the 1940's (see Saugsted, 1998). Hultman, Sparen, Takel (1999) found schizophrenia was associated with multiparity, maternal bleeding during pregnancy, and later winter birth in three population based case control studies drawn from a cohort of Swedish children. Relative risk was higher for males small for gestational age (odds ratio 3.2) fourth or more in birth order (odds ratio 3.6) and where

maternal bleeding had occurred in late pregnancy (odds ratio 4.0). However, PCB's are weak predictors of adult schizophrenia, and the prevalence of schizophrenia is not consistent with variation in rates of PCB's across cultures and Goodman (1988) suggests in many cases PCB's may be an effect, rather than a cause, of schizophrenia.

Molecular genetic findings

Linkage for schizophrenia markers has been established at different chromosomes. There have been replications, but findings have been very inconsistent implying complex patterns of transmission (see Freedman, Leonard, Olinay, Kaufman, Malspinna, Cloninger, et al, 2001).

Figure 1.4.5.4.1 Molecular genetic findings for schizophrenia

	Findings
DRD4	7 repeat 48 base pair linearly associated with high delusional scores, Serretti, Maccairdi, et al(2001) Association to first psychotic episode, Rinetti, Camarena, Cruz, Apiquian, Frezan, Paez,et al,(2001)
DRD5	Differences in allele distribution and significant association of markers that map close to the DRD5 gene, Muir, et al (2001)
Chromosome 15q14	Significant genome wide linkage in both African and European families, Freedman, et al (2000)
Chromosome 6q25	Significant linkage (lod score 7.7) in a twelve generation member pedigree, Lindholm, Ekholm, Shaw, Jalonen, Johansson, Petterson, et al (2001)
Chromosome 1q42	Significant linkage for schizophrenia (lod score 3.6) and affective disorders (lod score 4.5) and depression and schizophrenia (lod score 7.1) Blackwood, Fordyce, Walker, St Clair, Porteous, Muir,et al (2001)

Austism

Twin, adoption and family studies have shown that autism is highly heritable with estimates of a genetic contribution of at least 90%, Prevalence rates in siblings of autistic probands are very high in comparison to the general population. Three large twin studies (Folstein & Rutter; 1977; Bailey, LeCouteur, Gottesman, Bolton, Simonoff, Yuzda, Rutter,

1995; Steffenburg, Gillberg, Hellgren, Anderson, Gillberg, Jakobsson, Bohman,1989) showed similar patterns of greater MZ than DZ concordance (0.60 - 0.90, and less than 0.05) respectively. There is also evidence that non-autistic co-twins of MZ autistic probands show autistic type social and communicative difficulties, a finding replicated in family studies (see Bailey, Phillips, Rutter, 1996; Smalley, 1997;Bailey, Palferman, Heavey, LeCouteur, 1998;Rutter, Silberg, et al, 1999).

Silverman, Smith, Schmeidler, Hollander, Lawlor, Fitzgerald, et al (2002) looked at families with multiply affected siblingships (including a proband with autistism and at least one sibling with significant deficits in autism symptom domains). Variance in siblings was reduced for both repetitive behaviour, and useful speech. Using only the diagnosis of autism siblingships were ranked for each domain independently, and these together with nonverbal communication behaviour provided evidence of familiality, the identified features were also familial in autistic spectrum conditions.

Molecular findings

Heterogeneity in expression has made it difficult to identify potential genetic variants related to etiology, as different aspects of symptomology may be genetically independent (see International Molecular Genetic Study of Autism Consortium (IMGSAC) 2001). A subset of autism cases are associated with a broad range of chromosomal abnormalities, in particular chromosome 15 anomalies, Prader-Willis and Anglemann syndrome have been mapped to this region, and a subset of individuals with these syndromes show autistic like behaviour (see Lui, Nyholt, Magnussen, Parano, Pavone, Geschwind, et al, 2001).

Serotonin

Several studies have reported increased whole blood platelet 5HT in autistic children, and pharmacological studies show that 5HTT reuptake inhibitors reduce repetitive behaviour aggression and language use. 5HT is also associated to a range of behaviours frequently disturbed in autistic disorders, for example aggression, impulsivity, anxiety, obsessive compulsive symptoms and social interaction and affiliation (see Lesch & Mossner, 1998). There is also evidence that implies serotonin reuptake inhibitors improve autistic symptoms in a subset of individuals (see Lui, et al, 2001).

Family based association studies using TDT have shown linkage to autistic disorder and there are similar patterns of association from haplotype analysis. Two studies found preferential transmission of the long 5HTTLPR allele, in autistic probands and their relatives, although one study showed preferential transmission of the short allele, and there has been a non-replication, there is also some suggestion of a relationship between 5HT and obsessive compulsive disorder (see Manor, Eisenberg, et al, 2001; Yirmiya, et al, 2001). A genomewide screen showed significant linkage to autism and autism spectrum disorders with markers on chromosomes 5 and 8, with suggestive linkage to chromosome 19 (Lui, et al, 2001). In particular evidence from full genome scans has identified chromosome regions which may be important. The IMGSAC (1998) reported positive lod scores for linkage to chromosome 4, 7, 10, 16, 19 and 22, with the highest lod score for C7, this latter finding has been replicated (IMGSAC, 1999) and the IMGSAC (2001) has replicated linkages on chromosomes 7q and 16p in an additional sample, with two regions of linkage identified on chromosomes 2q (lod score of 3.20) and 17q. The Paris Autism Consortium Research International Sibpair study found a trend for linkage at 11 chromosomes, four of which (2q, 7q, 16p and 19p) overlapped with the IMGSAG findings. Risch, Spiker, Lotspeich, Nouri, Hinds, Hallmayer, et al (1999) found maximum likelihood scores greater than on chromosomes 17p, 7p, 18q, and 1p, and Lui, et al (2001) found significant linkage for markers on chromosomes 5 and 8, with suggestive linkage evidence for chromosome 19.

Genetic variation has also been identified which may be related to social cognitive aspects of autistic behaviour. Research evidence from studies of Turner's syndrome (where one X chromosome is partially or wholly deleted in females) implies that there is a genetic locus for social cognition which is imprinted, and not expressed from the maternally derived X chromosome, which is line with evidence that 46XY males (whose single X chromosome is maternally derived) have a higher incidence of developmental and language disorders than 46 X X females (Skuse, Jones, Bishop, Coppins, Dalton, Aamodt-Leeper, et al, 1997 p705).

Language impairment

Specific language impairment is highly familial and twin studies have shown greater concordance for MZ than DZ twins for specific language impairment (see Williams,

Stevenson, 2001). Dale, Simonoff, Bishop, Eley, Oliver, Price, et al (1998) compared heritability for language impairment in a large sample of two year old twins. Group heritability was around 73% for the lowest performing 5%, wheras it was 25% for the entire sample, implying that early language delay is a distinct disorder. Segragation analysis from multiply affected families, and a point mutation identified in an individual with severe speech and language disorders of specific genetic variation implies a link to mechanisms involved in the developmental of speech and language (Fisher, Francks, MacCracken, McGough, Marlow, McPhee, et al, 1998;Lai, Fisher, Hurst, Vargharkhadens, Monaco, 2001). In addition The Specific Language Impairment Consortium (2002) has reported significant lod scores for measures of language difficulty to chromosomes 16 and 19.

Cognitive ability

A wide literature has shown that different aspects of cognitive ability (e.g. verbal ability, spatial ability, memory and speed of information processing) are typically inter-correlated with assessment of specific abilities contributing to general cognitive ability or g (see Plomin & DeFries, et al, 1999). Genetic studies of cognitive ability have focussed on general and specific abilities, school attainment, brain structure and birth weight.

General cognitive ability

Evidence from family, twin and adoption studies show genetic factors contribute to around 50%, and shared environmental factors to around 25% of the variance in general cognitive ability (see de Geus, Wright, Martin, Boomsma, 2001; Plomin, Kosslyn, 2001: Bouchard & McGue, 2003). However, genetic contributions are lower in infancy, and increase over the lifespan, with a corresponding decrease in shared environmental factors. Similar patterns have been found in longitudinal twin studies (age five to twenty nine years) and of older adults. While there is a general decline in fluid intelligence and speed of information processing after age seventy, comparison of heredity in older twins (seventy-five plus) showed no difference in factors underlying heritability in cognitive function in normal and impaired twins, implying influences on general cognition are separate to genetic influences on dementia (McGue & Christensen, 2001). There is also no evidence of differential patterns for extremely high cognitive ability.

Multivariate analysis has shown very high genetic correlations among specific cognitive abilities (Plomin, Hill, Craig, McGuffin, Purcell, Sham, et al, 2001; Plomin, Kosslyn, 2001). For example, genetic contributions to working memory, which includes executive functions, show genetic overlap for both spatial and verbal components (Ando, Ono, Wright, 2001). Posthuma, deGeus, Boomsma (2001) found that genetic factors contributed to 46% of total variance in perceptual speed, with significant phenotypic correlations between perceptual speed and verbal and performance IQ (0.19 and 0.27 respectively) produced by a common genetic influence. Luciano, Wright, Smith, Geffen, Geffen, Martin, et al (2001) found a common genetic influence on information processing speed, working memory and IQ, and Rijsdijk, Vernon, Boomsma (1998) found common genetic influences on reaction time and IQ.

Similar patterns of intercorrelations have been found in adoption studies. Alarcōn, Plomin, Fulker & DeFries (1999) compared parents (biological and adoptive) and offspring in the Colorado Adoption Project. They found phenotypic correlations of around 0.48 between verbal and spatial ability and perceptual speed, and around 0.27 for memory. Genetic factors contributed around 0.76 and 0.50 respectively to these relationships, and heritability increased with age.

School attainment

Studies have shown correlations of around 0.60 between performance on a range of school tests, also that these are highly correlated with general cognitive ability (see Plomin, et al, 1997). Evidence shows genetic factors contribute to around 30% of variation in test performance in younger children, increasing to around 60% in adolescence, with a corresponding decrease in common environment influences.

Studies which have looked at genetic and environmental influences on school performance

Thompson, Detterman, Plomin (1991) found heritability of around 0.30, and common environment influences of around 0.60 in reading, language and maths performance in 6 – 12 year olds. However, in a large sample of 13 year old twins Hussēn (1959) (in Plomin, et al, 1997) found heritabilities of around 0.60 for history, reading, writing and maths, and Loehlin & Nichols (1976) found heritabilities of around 0.40 across a range of subjects.

Wadsworth, Corley, Hewitt & DeFries (2001) found genetic influences contributed to stability in variation for reading across childhood, adolescence.

Multivariate genetic analysis shows substantial genetic correlations between subjects, and evidence that genetic influences on general cognitive ability also influence performance on school tests (see Plomin, et al, 1997). For example, Alarcōn, Knopik, DeFries (2000) found heritabilities for variation in maths and general cognitive ability were 0.90 and 0.80 respectively, with around 90% of the covariation between the two phenotypes due to common genetic factors. In addition, evidence of increases in heritability for school performance are similar to patterns of genetic influences of general cognitive ability.

Molecular studies

Plomin, Hill, et al (2001) have carried out a genome wide scan of 1842 markers of general cognitive ability using a five stage design with DNA pooling and extreme selected groups. Two markers (D4s2460 and D14565) met criteria in two independent case control samples, but were not replicated in TDT analysis.

Heritability of brain structure

Twin studies have shown high heritability for brain region volume (see Vernon, Wickett, et al, 2000 for a review) with similar patterns found across different measures (e.g gray matter volume) (Thompson, Cannon, et al, 2001: White, Andreasen, Nopoulos, 2002). In addition Thompson, et al (2001) found differences in frontal gray matter were significantly linked with differences in Spearmans g (similar to IQ this measure taps intellectual function common to multiple cognitive tests and has been shown to be highly heritable, $h^2 = 0.70 \pm .17$ in this sample). These findings are consistent with findings in 28 pairs of 12 year old MZ twins (see Thompson, Cannon, et al, 2001) and with findings of a relationship between brain volume and g from studies involving several hundred individuals, which indicated that larger brain volume is associated with higher cognitive ability (see Vernon, Wickett, Banzana, Stelmack, 2000).

Birth weight and cognitive ability

There is evidence of a relationship between low birth weight (under 2500g) and cognitive ability (Matte, Breshchan, Begg, Susser, 2001). Bohm, Katz-Salamon, Smedler, Lager, Cranz, Forssberg, et al (2002) found that very low birth weight children from the Stockholm Neonatal Project were disadvantaged on IQ tests relative to controls with a larger decrement on performance IQ (measures of visual perception and spatial reasoning) and that there was a significant correlation between birth weight ratio, performance and full IQ except for verbal ability. There is also evidence of atypical brain asymmetry in low birth weight (<1000 g) infants (Dugdale, Mohay, O'Callaghan, 1987) and an absence of typical gender differences in cognitive ability were found for children under 2500g at birth in the study by LaBarthe (1997).

In addition Matte, et al (2001) found a relationship between cognitive ability and birth weight in the normal range in a cohort sample of 3485 same-sex siblings. When socio-economic and other environmental factors were controlled for mean IQ at age seven years increased monotonically with increases in birth weight. Within siblingships the relationship between IQ and birth weight was stronger for males than females, with 1000g increase corresponding to an increase of 4.6, and 2.8, in IQ respectively.

This relationship appears to be due to genetic influences. Boomsma, van Beijskerveldt, Rietveld, Bortels, van Baal, et al (2001) found that dizygotic twins with the lowest birth weight had low IQ's relative to their co-twins, whereas mean IQ was the same for both low and high birth weight monozygotic twins, implying differences between IQ for DZ twins was a function of both genetic and environmental influences.

Cognitive variation and psychopathology

Cognitive variation is associated with psychopathology. There is a higher frequency of psychiatric disorders in individuals with IQ below 70 (Scott, 1994) and in particular cognitive impairment is associated with autism, with seventy-five percent of autistic individuals showing intellectual impairment. There is a relationship between IQ in the normal range and behavioural problems. Increased behaviour problems are associated with lower IQ in adolescents (Goodman, Simonoff, Stevenson, 1995) and with lower scores on

tests of verbal and non-verbal cognitive development in toddlers (Plomin, Price, Eley, Dale, Stevenson, 2002). Academic underachievement has also been shown to be associated with psychopathology (e.g. American Academy of Pediatrics, 2000: Prior, et al, 2000).

Genetic studies

Boomsma (1998) looked at heritability of cognitive failures (failures in memory, perceptual and motor control associated with psychiatric symptoms, but not related to IQ) and found genetic and non-shared environmental factors contributed roughly equally.

P300

The P300 amplitude indexes cognitive processing of novel stimuli, for example, allocation of attentional resources during tasks which involve working memory, and reduced P300 amplitude is a common marker of psychopathology. In particular, van Baal, Boomsma, de Geus (2001) found a significant association between EEG coherence (a measure of brain connectivity) and IQ in a twin sample, which was mainly genetically mediated.

Estimates of family resemblance for P300 are between 0.30 to 0.81. However, van Beijsterveldt, van Baal, Molenaar, Boomsma (2001) found stability for P300 during adolescence was accounted for by genetic factors in males, and shared environmental factors in females. There is also high heritability for background electroencephalogram (EEG) power spectrum, and Anokhim, van Baal, van Beijstervaldt, de Geus, Grant, Boomsma (2001) found high genetic correlations (0.54 - 0.74) with 30% of the total P300 variance explained by genetic factors influencing EEG in males, and 45% of P300 explained by shared environmental factors in common with EEG in females.

Twin studies

Plomin, Price, Eley, Dale, Stevenson (2002) looked at the association between behaviour problems and verbal and non-verbal cognitive abilities in a community sample of twins born in 1994 and 1995. For the entire sample correlations between behaviour problems

and verbal and non-verbal cognitive abilities were less than 0.30. Associations increased across assessment at age 2, 3, and 4 years, and were stronger for males than females at the extremes of the distribution. Multivariate genetic analysises showed genetic and shared environmental factors contributed to the association between behaviour problems and cognitive ability, and that genetic links between these phenotypes may be stronger at the extremes of the distribution.

Theretical explanations

Theoretical explanations for the relationship between brain anomalies and risk for psychopathology concentrate on specific brain structures. However, it has been hypothesised that there is a more general relationship between cerebral lateralisation and risk for psychopathology (Geschwind & Galaburda, 1985; Annett, 1985; Crow, 2000; Yeo, Gangested, Daniels, 1993). It is suggested that brain asymmetry underlies many aspects of cognitive variation, and that atypical brain development contributes to risk for both learning disabilities and psychopathology.

Cerebral lateralisation

The human brain is asymmetrical with the planum temporale of the left cerebral hemisphere typically two thirds wider than the right (Hellige, 1993). Morphological brain asymmetries are thought to underlie cognitive and behavioural lateralisation of brain organisation. The sensory and motor cortex both show contra-lateral control (areas in the right hemisphere send and receive information from the left side of the body, and areas in the left hemisphere send and receive information from the right side of the body) (Beatty, 1995). Approximately, 90% of the population are right-handed,97% of which show predominant left hemisphere language localisation, however only around 60% of left-handed individuals show left hemisphere language localisation (Geschwind, Miller, DeCarli, Carmelli, 2002).

Androgens and fetal development

The role of androgens in fetal development has been considered to be important. Based on neuroanatomical evidence and a series of studies Geschwind & Galaburda (1985) proposed

that fetal testosterone levels modify neural development, immune development, and neural crest development (see Geschwind & Galaburda, 1985; Geschwind & Galaburda, 1987 for a full discussion). To look at this genetic studies of cerebral lateralisation, developmental instability, and fluctuating asymmetry (associated with psychopathology) are reviewed.

Genetic studies of cerebral lateralisation

Genetic contributions to cerebral lateralisation are implied as the probabality of left-handedness increases if one biological parent is left-handed (especially the mother) and is higher if both parents are left-handed. The probability of right-handed parents having a left-handed child is 0.02. If one parent is left-handed the probability of a left-handed child is 0.17. If both parents are left-handed the probability of a left-handed child is 0.46 (see McManus, 2002; Springer & Deutcsh, 1998). Similar patterns have been obtained across twin, family, adoption and cross-fostering studies (see Geschwind, et al, 2002; Annett, 1999).

Total sidedness.

Reiss (1999) looked at the heritability of lateral asymmetries (handedness, footedness, eyedness, earedness, hand clasping, arm folding, and leg crossing) in a family study consisting of 292 parent-offspring triads and 36 sibling pairs. The frequency of left-sidedness increased with the number of left-sided parents. Associations were significant except for footedness and handclasping. However, genetic correlations for lateral asymmetry were relatively imperfect, which implies multigenetic determinants.

X-linkage.

Several models of genetic effects on cerebral lateralisation are based on X-linkage, with some disagreement on whether gender differences are due to additive or recessive gene effects (see Corballis, 2001, Jones, Martin, 2001: McKeever , 2000).

Higher frequency of left-handedness in monozygotic twins.

The frequency of left-handedness in monozygotic twins is estimated to be around twenty percent, and evidence of discordance in these twins implies intra-uterine influences on neuro-development. This may be due to greater prenatal and perinatal difficulties associated with multiple births, and processes underlying mirror imaging (for example

asymmetry reversals in handedness, direction of hair whorl, facial features, etc resulting from delayed zygotic splitting) (see Kee, Cherry, Neale, McBride, Segal, 1998).

However, there is no evidence of difference to singletons in performance asymmetries (e.g. dichotic listening, finger tapping and visual discrimination) (see Kee, Cherry, et al (1998), and imaging studies have shown normal patterns of handedness differences in planum temporale asymmetry for discordant twin pairs, (see Steinmetz, Herzog, Schlaug, 1995; Procopio, 2001).

There is evidence of differences in genetic and environmental factors underlying cerebral lateralisation in monozygotic twins. Geschwind, Miller, DeCarli, Carmelli (2002) used MRI to assess genetic and environmental influences on the volumes of left and right cerebral cortex in a large cohort of ageing twins. Genetic influences contributed to changes in lobar volume that occur with ageing, and shared environmental factors (which the authors suggest is likely to represent in utero influences) were twice as strong for the left, than the right hemisphere. When twin pairs concordant for right-handedness were compared to twin pairs with at least one left-handed twin, genetic factors contributed twice as much to left and right cerebral hemispheric volumes in right-handed twin pairs, suggesting less genetic control for non-right handed twin pairs, which is consistent with models of a right-hand/left-hemisphere bias. Similarly, measures of left and right hemispheric volumes in a subset of this sample showed high heritability for most structures, but MZ interclass correlations for right-handed pairs were significantly higher than for non right handed pairs (see Geschwind & Miller 2002).

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Molecular genetic findings.

Roubertoux, LeRoy, et al (2001) independently measured direction (preferential use of the left/right paw) and degree (absolute differences between the left and right paw) of laterality in mice. QTL analysis showed lod scores of 5.6 (forepaw) and 7.2 (hindpaw) for association between degree of laterality and chromosome 4, and the map position was consistent with influences of gonadal steroid influences on the degree of laterality.

Yeo, Shaw, Thoma, Daniel (1996) looked at the relationship between the human leucocyte antigen (HLA) antigens and hand preference in 664 individuals. They predicted left-handed individuals would have a higher frequency of A1, B8 and DR3 alleles, and

predictions were generally supported, with left-handers more likely to possess B8 and DR3 alleles, and possess the A1/B8 haplotype, relative to right handers.

Common influences on brain and body asymmetry

There is a relationship between cerebral lateralisation and physical development. Kulaksiz, Gozil (2002) found left hand shape index and right hand shape index were phenotypic indicators of hand preference as increases in left hand shape index and right hand shape index correlated with increases in left and right hand laterality scores respectively. Also, asymmetries of the skull are more pronounced in right-handed individuals (LeMay, 1997) and the right hand is usually larger than the left (Wood, Ward, Morris-Kay, 1996). This asymmetry is more pronounced in right-handed individuals, which implies that different influences may contribute to the development of body asymmetry in left and right-handed individuals.

Homeobox gene effects

Manning,Scott, Wilson, Lewis-Jones (1998) suggests that the role of androgens in cerebral lateralisation is due to common influences of the Hox genes. The Hox genes are a family of 39 homeobox containing genes arranged in four chromosmal clusters, which share organisational and sequence homologies. They are highly conserved transcription factors, implying a role in embryonic development. For example, region specific Hox genes influence the initial body plan by providing positional information along the anterior-posterior body axis and the limb axis (see Redline, Neish, Holmes, Collins,1992; Ponsuksili, Wimmers, Adjaye, Schell, Ander, 2001). The Hox gene sequence also link pathways of segmentation, and early development in the limb, gut and vertical column (Podalzek, Douboule, Bushman, 1997). There is also evidence that Hox genes are involved in brain development in Drosphila (see Hirth, Therianos, Loop, Gehring, Reichert, Furukubo-Tokunaga, 1995).

In a series of studies Manning and colleagues have found a relationship between digit ratio (the ratio between the length of the second and fourth fingers) on the left and right hand, androgen levels and cerebral lateralisation. This ratio is formed by around the 14th week in utero (Garn, et al, 1975) and known to be stable across development. It is sexually

dimorphic (males have longer index than ring fingers, and mean ratios are lower for males than females) and negatively correlated with testosterone, and positively correlated with oestrogen in adults (see Manning, Barley, Walton, Lewis-Jones, Trivers, Singh, 2000: Manning, Scott, et al, 1998: Peters, McKenzie, Bryden, 2002). This index provides an index of prenatal testosterone levels and unlike handedness or cognitive task performance is not influenced by social learning. Manning, et al (1998) suggests the association between digit ratio and reproduction/sexual orientation comes from common effects of the Hox genes with the production of testosterone and oestrogen affecting differentiation of the digits (see Piechel, Prabhakaran, Vogt, Fradeau, Zakony, Duboule, 1997: Herault, Fradeau, Zakany, Duboule, 1997: Chui & Hamrick, 2002: Redline, et al, 1992: Ponsuksili, Wimmers, et al, 2001: Podalzek, et al, 1997).

Developmental instability and fluctuating asymmetry

Developmental instability refers to an organisms degree of vulnerability to genetic and environmental stresses during development, i.e. chromosome anomalies, mutations, pathogens, extreme temperature, maternal stress, radiation (see Thornhill & Moller, 1997 for a review). Fluctuating asymmetry is a measure of general developmental disruption (defined as deviation from bilateral symmetry, which results from genetic stress as the corresponding sides of a bilateral symmetrical trait are encoded by the same genes) (see Thornhill & Moller, 1997: Yeo, Gangsted, Thoma, Shaw, Reva, 1997) and is often used to evaluate developmental homeostasis (see Rahman & Wilson , 2002; Pechenkina, Benfer, Vershousskaya, Kozlova , 2000). Fluctuating asymmetry and other indices of developmental instability are associated with psychopathology (see review by Thornhill & Moller, 1997). In a review of thirty-four studies of different species (including humans) Moller & Thornhill (1997) found significant heritability for fluctating asymmetry.

1.4.6 PERSONALITY AND PSYCHOPATHOLOGY

Genetic analyses of the relationship between personality and psychopathology allow issues of causation to be addressed. For example, it is not clear if genetic and environmental factors which influence personality also influence psychopathology (bivariate heritability) or whether expressed behaviour resulting from genetic and/or environmental influences on

personality variation increases risk for psychopathology (phenotypic causation model) (see Simonoff, 2000; Carey & DiLalla, 1994).

Figure 1.4.6.1 Bivariate heritability model

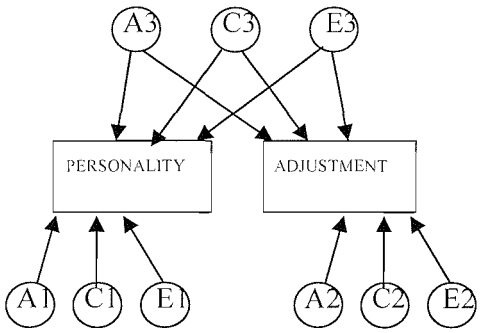
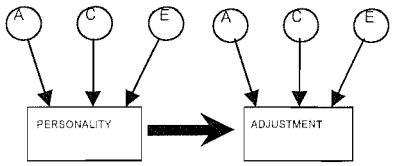


Figure 1.4.6.2 Phenotypic causality model



Bivariate heritability (see discussion of this construct on pages 89 and 190) implies that the risk for psychopathology will vary according to underlying genotype or environmental factors (e.g. individuals with an AA genotype will be at greater risk than individuals with an Aa or aa genotype). Phenotypic causation implies that the risk for psychopathology is the same for everyone with the same phenotypic value regardless of genetic influences (e.g. aa, Aa, and AA) (Carey & DiLalla, 1994).

Yale Family Study of Comorbidity of Substance Abuse and Anxiety

Similar temperament dimensions differentiated diagnostic groups in parents and their children in the Yale Family Study of Comorbidity of Substance Abuse and Anxiety. In both adults and children anxiety and depression were associated with low scores on adaptability and approach/withdrawal, whereas externalising or substance abuse disorders were associated with low attention and higher activity scores. Comorbidity was associated

with both temperament clusters and greater clinical severity, and in particular children of parents with internalising disorders were less active, and children of parents with externalising disorders were more active and scored lower on rhythmicity (Merikangas, Swendsen, Preisig, Chazan, 1998).

Giancola (2000)

Giancola, (2000) compared the relationship between temperament and anti-social behaviour in pre-adolescent boys (10 - 12 years) with or without a family history of substance abuse. In general low scores on rhythmicity, behavioural regulation and positive affect were related to antisocial behaviour, however, there were differences in specific patterns of temperament traits related to disorders in both groups. Boys with a family history of psychiatric problems had low behavioural regulation scores and greater levels of aggression and delinquency scores, and a greater number of symptoms for oppositional defiant disorder (ODD) and conduct disorder (CD). For this group only low positive affect was associated with delinquency, low behavioural regulation was associated with CD, and low rhythmicity was associated with ODD only in the presence of low positive affect. Low behavioural regulation and low rhythmicity was also associated with increased teacher ratings of aggression, and low scores on all three temperament dimensions were separately linked to greater levels of externalising disorders.

Roberts & Kendler (1999)

Self-esteem and neuroticism are personality traits theoretically linked to depression, however the relationship between self-esteem and depression is non-significant when neuroticism is controlled for. Neuroticism predicts major depression in adult females, and covariation between self-esteem, neuroticsim and depression is largely due to genetic factors (Roberts & Kendler, 1999).

Gillepse, Johnston, Gillespie, Johnstone, Boyce, Heath, Martin (2001)

There is evidence that genetic factors related to temperament dimensions are associated with a measure of depression (Interpersonal Sensitivity Measure) (IPSM) overlap. In a sample of over 3000 adult twin pairs, measures from both the Eysenck Personality

Questionnaire, and the Tridimensional Personality Questionnaire accounted for a large proportion of the genetic variation in the IPSM (Gillespie, et al, 2001).

Gjone & Stevenson (1997)

Gjone, Stevenson (1997) looked at the longitudinal covariance of temperament and behaviour problems in a sample drawn from five national cohorts of same-sex twins aged 7 to 17 years at two-year follow-up. EAS temperament traits, emotionality, activity and sociability, and behaviour problems, anxious/depressed, delinquent, aggressive behaviour and attention problems (Parent Report Child Behaviour Checklist) (CBCL) (Achenbach, 1991) were assessed at two time points.

The aims of the study were to look at 1) the extent temperament predicted behaviour problems in children and adolescents, 2) specific associations between EAS temperament and behaviour problems as suggested by Graham & Stevenson (1987) and 3) whether genetic factors contributed to covariance of temperament and behaviour problems.

High emotionality predicted anxious/depressed, delinquent, aggressive behaviour, and attention problems. Aggression was further predicted by high activity, in particular in young children (see table 1.4.6.1).

Figure 1.4.6.3 Correlations between time 1 EAS and time 2 CBCL by sex and age

	Anxious/depressed		Attention problems		Delinquent		Aggressive	
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
Emot								
Group 1	0.37**	0.35**	0.19	0.32**	0.35**	0.24*	0.50**	0.40**
Group 2	0.36**	0.36**	0.39**	0.48**	0.28**	0.11	0.51**	0.50**
Group 3	0.28**	0.39**	0.37**	0.45**	0.27**	0.36**	0.41**	0.49**
Group 4	0.54**	0.34**	0.51**	0.23**	0.49**	0.19*	0.50**	0.40**
Soc.								
Group 1	0.03	0.22*	0.04	0.33**	0.04	0.14	0.15	0.32
Group 2	0.18	0.18	-0.05	0.19	0.13	0.13	0.10	0.12
Group 3	-0.03	0.13	0.07	0.14	0.08	0.13	0.10	0.18
Group 4	0.12	0.04	0.10	-0.06	0.06	-0.06	0.23	0.04
Activity								
Group 1	0.05	0.04	0.11	0.27*	0.08	0.17	0.22*	0.22*
Group 2	0.15	0.04	0.04	0.12	0.27**	0.18	0.30**	0.25**
Group 3	-0.20*	-0.07	-0.05	-0.05	-0.00	0.03	0.04	0.06
Group 4	0.05	-0.15	-0.00	-0.21*	-0.07	-0.24**	0.10	-0.11

Group one = 7 to 8 years, group two = 10 to 11 years, group 3 = 14 to 15 years, group four = 16 to 17 years
(Gjone & Stevenson, 1997)

Genetic factors contributed significantly to the covariance between emotionality and attention problems, and the covariance between emotionality and aggressive behaviour. There was no evidence of significant common environment influences on any of the associations between temperament and behaviour problems.

Temperament was assessed at 14, 20, 24 and 30 months by the Colorado Childhood Temperament Inventory, and behaviour problems assessed at four years by the Child Behaviour Checklist. For all four ages emotionality was significantly associated with total problem scores, internalising and externalising groupings, and shyness was significantly associated with internalising groupings. MZ correlations were higher than DZ correlations for temperament measures, but they were less so for behaviour problem correlations. The best fitting models for temperament included genetic and non-shared environmental parameters, and the best fitting models for behaviour problems included genetic, shared and non-shared environmental parameters. As shared environmental influence was not important for variance in temperament dimensions, covariation between temperament and behaviour problems was mainly gentically mediated. Genetic factors accounted for 94% of the covariance between emotionality and internalising disorders, and 76% of the covariance between shyness and internalising disorders (Schmitz, et al, 1999).

Table 1.4.6.4 Twin correlations for temperament and behaviour problems

	MZ twins	DZ twins
Emotionality		
14 months	0.42	0.04
20 months	0.51	0.04
24 months	0.37	0.02
36 months	0.39	-0.13
Shyness		
14 months	0.42	-0.19
20 months	0.42	0.01
24 months	0.39	-0.02
36 months	0.52	-0.08
Externalising problems		
4 years	0.77	0.60
Internalising problems		
4 years	0.66	0.59
Total problem score	0.80	0.78

(Schmitz, Fulker, Plomin, Zahn-Walker, Ende, DeFries, 1999)

Goldsmith & Lemery (2000)

Goldsmith, Lemery (2000) looked at the covariation between temperament and anxiety symptoms. The best fitting model included genetic and non-shared environmental parameters, with independent non-shared environment influences contributing to early fear measures and overanxiety, implying that covariation was due to genetic influences. For fearfulness and separation anxiety the best fitting model included genetic, shared and non-shared environmental factors. 29% of the variance in separation anxiety due to shared environmental factors was shared with fearfulness measures, and 13% was unique. A similar pattern was obtained for early shyness and separation anxiety, but 40% of shared environmental factors were common to both variables.

DiLalla, Kagan, Reznick (1994)

Comparison of dimensional and categorical measures of behavioural inhibition in 24 moth old twins showed that heritability estimates were higher for extreme scores, although this was not significant. For the entire sample using dimensional measures MZ correlations were higher than DZ correlations (0.82 and 0.47 respectively). Extreme groups analysis showed that heritability was higher for the top 20%, also that this increased with higher thresholds for differentiating probands (DiLalla, et al, 1994).

Iacono, Carlson, Taylor, Elkins, McGue (1999)

Iacono, et al (1999) suggest early onset alcoholism is characterised by personality traits, behavioural disorders, and psychophysiological indicators that reflect a general disposition to behavioural disinhibition. Two personality dimensions consistently linked are negative emotionality and behavioural disinhibition. Recent reviews have shown behavioural disinhibition assessed by the novelty seeking scale of the Tridimensional Personality Questionnaire, and other scales, predicts alcohol abuse and criminality, also discriminating alcoholics with and without antisocial behaviour. There is also evidence from longitudinal studies that personality differences are present prior to onset of SA, and predict other externalising psychopathology (see Iacono, et al, 1999).

In support of this genetic factors contributing to psychophysiological indicators of externalising behaviours associated with behavioural disinhibition overlap with genetic factors linked to SA. These include extreme responses in the autonomic nervous system, and central nervous system. Children and adolescents at risk of developing anti-social and aggressive behaviour show autonomic hyporeactivity reflected by low resting heartrate and skin conductance, and poor classical conditioning of these responses. Resting heart rate is lower in male children with conduct disorder and oppositional defiant disorder, and skin conductance orienting responses are lower in male children with ADHD and CD. Variation in P3 amplitude has been associated with antisocial behaviour. Autonomic hyopactivity in early childhood predicts antisocial behaviour and criminality in adolescence and adulthood (see Raine, 1996; Raine ,Venables, Mednick, 1997, Fowles, 2000).

Stein, Chartier, Lizak, Jang (2001)

Stein, et al, (2001) suggest that genetic vulnerability to social phobia comes via transmission of temperament factors rather than genetic influences directly on social phobia. To look at this, first degree relatives of generalised social phobia probands were compared to relatives of a control group in a family study. The first degree relatives of GSP scored significantly higher than relatives of the control group on measures of trait anxiety, social anxiety and Harm Avoidance subscale of the TPQ. Correlations between these measures ranged from 0.56 to 0.81, and a single factor explained more than 83.5% of the variance in the measures.

Boomsma, Beem, van den Berg, Dolan, Koopmans, Vink, et al (2002)

There is evidence that association between neuroticism and anxiety/depressive disorders is genetically mediated. Factor-analysis of scores for a sub-sample of participants from the Netherlands Twin Family study of Anxious depression of families, where at least two siblings had extreme scores identified three main components which accounted for 55% of the variance. The first had high loadings of anxiety, neuroticism, somatic anxiety and depression (29%) the second had high loadings for sensation seeking traits. The third had high loadings for Type A behaviour, extraversion and trait anger. Similar patterns were observed for combined data from measures two years apart, and measures taken five years

later, implying stability. Model fitting was used to identify common genetic or environmental factors. Correlations for all variables were higher for MZ than DZ twins. Univariate analysis showed heritability estimates of approximately 50% for anxiety, depression, neuroticism and somatic anxiety. Family resemblance was explained by genetic factors, with higher heredity estimates for males than females. This was due to greater genetic variance in females, and findings were similar to other large twin studies in America (Kendler, Neale, Kessler, Heath, Eaves, 1992; Kendler, Neale, Kessler, Heath, Eaves, 1993) and Australia (Kendler, Heath, Martin, Eaves, 1986). All genetic covariation between the measures could be accounted for by a common genetic factor which explained a large proportion of the phenotype variance (see Boomsma, Beem, 2002).

Table 1.4.6.5 Heritiability estimates of common and specific genetic factors in males and females

	Females		Males	
	Common	Unique	Common	Unique
Anxiety	0.46	0.04	0.42	0.03
Neuroticism	0.45	0.10	0.33	0.10
Somatic anxiety	0.28	0.17	0.20	0.11
Depression	0.46	0.07	0.35	0.11

(Boomsma, Beem, 2002).

Molecular genetic findings

Generally, some genes associated with personality traits (DRD4, 5-HTTLPR, and catechol-o-methyltransferase) appear to be pleiotropic and affect several disorders, including hyperactivity/attention problems, addiction, obsessive-compulsive behaviour, depression and anxiety, parkinsons disease, schizophrenia, hypnotizability. It is feasible that common influences of genes may be through modulation of neurotransmitter systems related to higher order cortical and limbic systems (executive functions) shown through regulation of impulsiveness, attention processes common to both personality and disorders like autism, ADHD, schizophrenia and addiction (see Ebstein, Benjamin, Belmaker, 2000p 210).

General methodological issues

There are several methodological issues underlying genetic research, which have implications for findings. These include model assumptions underlying twin and adoption studies, biological typicality of twins, and complexity of genetic transmission (see Plomin, DeFries, et al, 1997: Martin, Boomsma, Machin, 1997: Plomin & Crabbe, 2000: Bouchard & McGue, 2003). However, evidence is convergent across family, twin, adoption and molecular studies and implies specific method issues are not creating a systematic bias in research findings (see Tannock, 1998).

1.5 SUMMARY AND DISCUSSION

Temperament and personality

Personality is a stable individual difference which can be reliably distinguished from infancy. Temperament and personality have been modelled separately, and there are some developmental issues regarding the extent that measures of early temperament correspond to measures of adult personality (Shiner, 1998). However, theoretical considerations (e.g. Cloninger, 1993) suggest continuity in personality over development. Consistent with this evidence from a variety of methodologies, samples and cultures suggests early temperament characteristics are meaningfully related to later personality traits. Evidence from family, adoption, twin, and molecular genetic studies shows genetic influences contribute to stability in personality from an early age, with non-shared environmental influences contributing to change (e.g. Loehlin, 1992).

Psychopathology

Psychopathology has been conceptualised differently within psychiatric and psychological approaches. Within the psychological approach it is assumed that there is continuity between normal and disordered behaviour, and evidence suggests overlap between caseness identified by dimensional and categorical measures (Jensen & Watanabe, 1999). Evidence from family, adoption, twin and molecular genetic studies show genetic and non-shared environmental influences contribute to variation in psychopathology. Also, that common environmental influences are more evident in externalising disorders. Heritability

estimates are similar across both categorical and dimensional measures (e.g. Rutter, et al, 1999: Bouchard & McGue, 2003). Molecular genetic evidence shows multiple gene effects underlie variation in most types of psychopathology, which is consistent with a continuous distribution, and that there is considerable overlap between genetic influences on disorders.

Psychopathology and cognitive variation

Psychopathology is associated with cognitive variation. There is a relationship between intellectual impairment and psychiatric disorder, and both cognitive variation in the normal range of IQ, and school attainment have been associated with adjustment problems (Scott, 1994: Goodman, et al, 1995: Prior, et al, 2000). Theoretical considerations suggest these relationships may be due to variation in brain asymmetry which underlies many aspects of cognitive variation. General developmental disruption is also associated with both cognitive variation and psychopathology.

Personality and psychopathology

Theoretical models suggest variation in personality is associated with adjustment (Eysenck & Eysenck, 1975: Clark & Watson, 1991: Cloninger, et al, 1997) and that this relationship is phenotypically driven (genetic and environmental influences on personality act to increase risk for psychopathology) (Carey & DiLalla, 1994). However, Graham & Stevenson (1987) suggest some psychopathology can be conceptualised as a continuum of normal temperament variation, implying common aetiology. Consistent relationships have been found between personality and adjustment. Similar patterns of emotionality and low constraint have been associated with psychopathology in both clinical and population samples, and both adults and children, which implies a fairly non-specific relationship. High emotionality is associated with psychopathology in general, with low positive affect more specific to depression and psychosis, and low constraint more specific to externalising disorders. There is also evidence of a relationship between biological correlates of personality and psychopathology. More specific associations between personality and psychopathology have been found using minor facets of broad personality traits. However, it is difficult to compare studies where different personality inventories and measures of psychopathology have been used.

Different patterns of genetic and environmental effects on personality and psychopathology suggest the two phenotypes can be distinguished (Nigg & Goldsmith, 1998). However, findings from molecular genetic studies imply that some genes associated with personality traits may be pleiotropic and affect several disorders (e.g. Ebstein, et al, 2000; Jang, Hu, Livesley, Angleitner,Riemann, et al, 2001). Consistent with this evidence from quantitative genetic studies suggests specific associations between personality and psychopathology are familial (Merikangas, et al, 1998: Giancola, 2000: Stein, et al, 2001). Also common genetic effects contribute to the relationship between personality and psychopathology in adults (Gillespie, et al, 2001: Roberts & Kendler, 1999: Boomsma, et al 2002) and to the relationship between temperament and behaviour problems in children (e.g. Gjone & Stevenson, 1997: Schmitz, et al, 1999: Goldsmith & Lemery, 1999).

Definitions of personality and psychopathology clearly distinguish between the two phenotypes. Psychopathology measures are associated with many factors not characteristic of personality, however key methodological issues are the extent that measures of both phenotypes may have similar content (e.g. Lahey, et al, in press) and that some studies have used aspects of temperament as adjustment measures.

CHAPTER TWO
PERSONALITY AND ADJUSTMENT IN TWINS AND SIBLINGS

2.1 AIMS

The literature suggests common genetic influences contribute to the relationship between personality and psychopathology. Also, that influences underlying cognitive variation may be important to this relationship. However, these relationships have not been studied widely in children/young people, and the aim of this study is to look at the extent common genetic and environmental influences contribute to the relationship between personality and behavioural adjustment.

2.2 DESIGN/METHODS

A twin/family design was used. This analysis is based on differences in genetic relatedness between family members. For example, both MZ and DZ twins share a common environment. MZ twins share all their genes, whereas DZ twins, share on average only half, and unique environmental influences are not shared between twins. Simultaneous equations allow genetic and environmental parameters for a trait to be estimated.

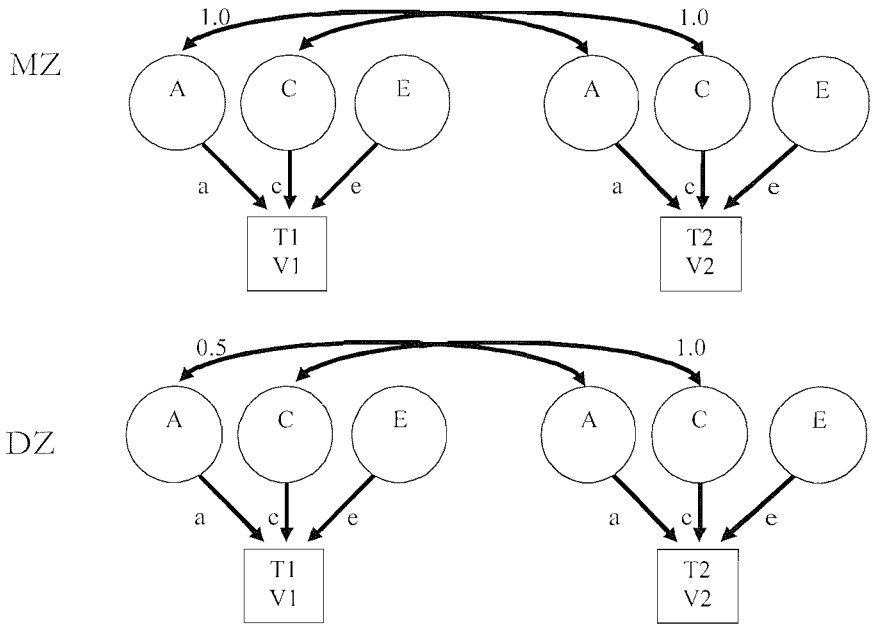
ACE models (Neale & Cardon, 1992)

In ACE models variables can be measured traits (squares) or latent variables (circles). Paths represent the effect of one variable on another, independent of the other variables. MZ and DZ covariance matrices provide the data against which the model is tested. The covariance set between the additive genetic (A) terms is 1.0 and 0.5 for MZ and DZ twins respectively. The covariance set between the common environment (C) terms is 1.0 for both MZ and DZ twins. The covariance set between the non-additive genetic (D) terms is 1.0 and 0.25 for MZ and DZ twins respectively. There is no covariance between the E terms, which also contains variance due to error. Full siblings also share half their genes and can be included in models using a correlation matrice based on mid-twin correlations. Within the models latent genetic and environmental influences are represented by uni-directional arrows. Correlations are represented by double-headed arrows. Path coefficients are represented by lower case letters. Squared path values give estimates of

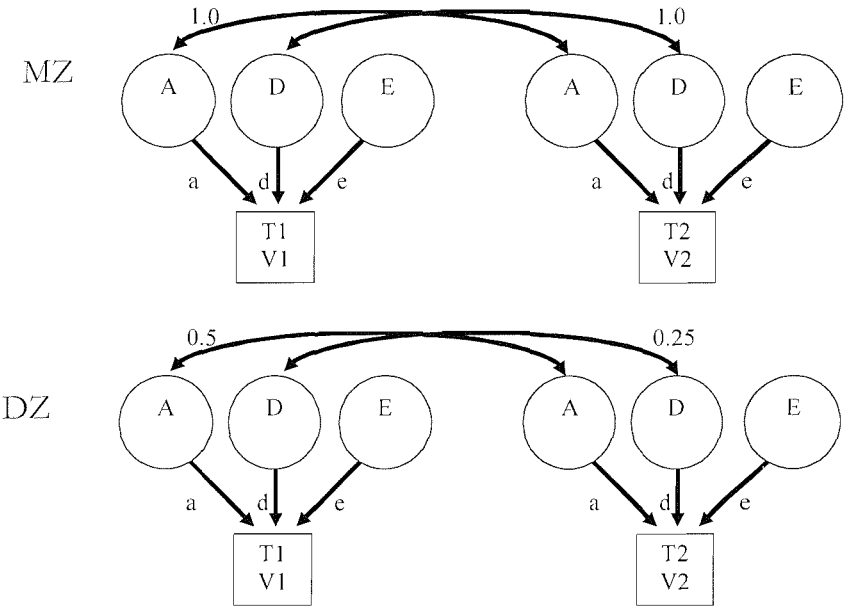
variance explained, for example, the proportion of phenotype variance due to genetic influences - heritability (see Plomin, 2001)(see figure 2.1.1).

Figure 2.1.1, ACE and ADE models

ACE model



ADE model



* A = non-additive genetic variance, C = common environment variance, E = non-shared environment variance, D = additive genetic variance (dominance).

Materials

Personality

Child and Adolescent Temperament Scale (Lahey, Waldman, Applegate, in press)

Studies of personality and psychopathology to date have used standard personality instruments, which contain items that show some overlap with psychopathology scales. Therefore the association between personality and psychopathology may reflect contamination of measures (Lahey, Waldman, Applegate, in press). To overcome this, the authors developed The Child and Adolescent Temperament Scale, specifically for use in assessing personality/psychopathology relationships. 50 items were selected from a literature review of social and emotional characteristics associated with psychopathology in children and adults. These items a) included personality characteristics associated with psychopathology, that were not well represented in most current temperament and personality scales, and did not include items not associated with psychopathology, and b) did not include items reflecting symptoms of psychopathology.

These items were hypothesised to reflect at least three temperament dimensions important to variation in psychopathology:

'Prosociality/conscientiousness'

This dimension reflects concern for others, respect for rules, and guilt, and reflects agreeableness and conscientiousness dimensions of the NEO Personality Inventory (Costa & McCrae, 1992) and the reverse pole of psychoticism in the Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975) (see Lahey, et al, in press).

'Daring'

This dimension reflects novelty seeking (Cloninger, 1987) sensation seeking (Zuckerman & Kuhlman, 2000) both positively correlated with extraversion, and may reflect the negative pole of inhibition (Kagan, Snidman, 1999) (see Lahey, et al, in press).

'Negative emotionality'

This dimension refers to experiencing negative emotions frequently, intensely, and with little provaction, and reflects neuroticism dimensions in both the Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975) and the NEO Personality Inventory (Costa & McCrae, 1992). (see Lahey, et al, in press).

Reliability and construct validity of the scales were tested in two cohort samples.

First study - Lahey, Waldman, Applegate (in press)

The aim of the study was to identify aspects of temperament concurrently linked to emotional and behaviour problems in children/adolescents. Factor analysis of caretaker ratings of 1,358 children (aged four to seventeen) and self ratings of 826 children (aged nine to seventeen) produced the three hypothesised dimensions (prosociality/conscientiousness, daring, negative emotionality). Split-half analysis suggested a very stable factor structure. Test-retest reliability (over seven to fourteen days) was high. There were some gender differences in the internal consistency and reliability of unit-weighted factor scores and inter-correlation among factors.

These temperament dimensions were concurrently associated with emotional and behavioural adjustment.

Study two - Lahey, Waldman, Applegate (in press)

Confirmatory factor analysis of 2000 twin pairs confirmed the pattern of results from study one, and these factors were correlated with psychopathology scores. In addition, these factors showed a high genetic influence with little shared environmental influence in a sample of 118 adult twins reared apart.

In addition there has been a tendency in the literature to study personality separately in children and adults, which makes comparison between findings of a relationship between personality and psychopathology in children and adults, difficult. However, there are theoretical grounds and empirical evidence, which suggests the same temperament/personality dimensions can be captured across development (see Cloninger,

1997; Luby, et al, 1999; Constantino, et al, 2002) and The Child and Adolescent Temperament Scale has been developed across a wide age range.

The Child and Adolescent Temperament Scale (parent report) contains 53 items, assessed on a four point scale (1 = 'Not at all', 2 = 'Just a little', 3 = 'Pretty much/prettyoften, 4 = 'Very much/very often'.

The Child and Adolescent Temperament Scale (youth report) contains 58 items, assessed on a four point scale (1 = 'Not at all', 2 = 'Just a little', 3 = 'Pretty much/prettyoften, 4 = 'Very much/very often'.

For the current study items from the Child and Adolescent Temperament Scale (parent and youth report) were converted into questionnaire items. For both versions of the questionnaire, two items were excluded ('Is he/she more interested in sex than other children his/her age' and 'Would he/she think it would be fun to watch two dogs fight') as they were thought unsuitable for a volunteer sample, and had received factor loadings of less then 0.3 in the American sample. Three additional items were included ('Do you daydream a lot', ''Do you believe that spiritual forces (e.g. God) sometimes direct life', and 'Do you make decisions quickly because you don't like to wait'). These were based on items from character dimensions from The Junior Temperament and Character Inventory (Luby, et al, 1999) and assumed to reflect aspects of personality reflecting character development which has a stronger cognitive component) (see Cloninger, et, al, 1993). To avoid order effects (see Lahey, et al, in press) question items were randomised, in addition, half the sample received the questionnaire in reverse order.

Behavioural adjustment

Strengths and Difficulties Questionnaire – p4 - 16 (Goodman, 1997)

The Strengths and Difficulties Questionnaire (SDQ) (Goodman, 1997; Goodman & Scott, 1999) is a brief behavioural screening questionnaire, suitable for children aged between four and sixteen years. This has both Parent and Teacher versions, and has been widely used in clinical, developmental and epidemiological research. The scale contains both positive and negative attributes regarding behaviour, emotions and relationships. The

items cover five scales (conduct problems, inattention/hyperactivity, emotional symptoms, peer problems, and prosocial behaviour). There are five items for each scale, scored on a 3 point scale ('somewhat true' is always 1, 'not true', and 'certainly true' are scored either as 0 or 2 across different items). The maximum score for each scale is 10 and the scores can be used as continuous variables. Totals for the first four scales are combined into a total difficulties score.

Figure 2.1.2, Caseness from symptom scores

	Normal	Borderline	Abnormal
Total difficulties score	0 – 13	14 – 16	17 -40
Emotional symptoms score	0 – 3	4	5 – 10
Conduct problems score	0 – 2	3	4 – 10
Hyperactivity score	0 – 5	6	7 – 10
Peer problems score	0 – 2	3	4 – 10

In the current study parent completed SDQ scores were used as a measure of behavioural adjustment.

General health Questionnaire – 12 item (Goldberg, 1972)

The General health Questionnaire is a brief screening instrument for adjustment in adult samples. Factor analysis has identified dimensions related to anxiety/depression, social dysfunction, and loss of confidence and self-esteeem (see Wernecke, Goldberg, Yalcin, Ustun, 2000).

Reliability.

For the General Health Questionnaire test-retest reliability is 0.75 - 0.90, internal consistency is 0.83 - 0.95 and inter-rater disagreement was only 4% of symptom scores in 12 interviews.

Validity.

For the General Health Questionnaire criterion validity was 0.76 - 0.81 with the Clinical Interview Schedule, 0.73 with clinical depression scores and 0.67 with clinical anxiety (see McDowell, Newell, 1987).

Cognitive variation

School attainment (Stevenson & Pit-ten Cate, Child Health and Development Study, April 1999 – March, 2000)

Variation in cognitive ability was assessed by parent report of school attainment across nine items (reading, spelling, handwriting, maths, art, music, computers, science, pe and games) used in the Child Health and Development Study (Stevenson, Pit-ten Cate, in press) on a three point scale (above average = 3, average = 2, below average = 1). These items were validated against other measures of school achievement/cognitive variation.

School attainment measures have been shown to correlate highly with each other and with measures of general cognitive ability, with the literature suggesting correlations between tests is due to common genetic influences (see Martin, Jardine & Eaves, 1984; Plomin, et al, 1997).

Influences on early brain development

Theories of cerebral lateralisation suggest brain asymmetry underlies many aspects of cognitive variation, and that atypical brain development contributes to risk for both learning disabilities and psychopathology (Geschwind & Galaburda, 1987; Annett, 1985; Crow, 2000; Yeo, Gangested, Daniels, 1993). While influences on early brain development cannot be directly assessed, handedness, digit ratio and presence of immune disorder were assessed as indirect indices of influences on early brain development.

Handedness (preference and skill)

Hand preference is a correlate of both learning disability and variation in cognitive performance (e.g Geschwind & Galaburda, 1987). Theoretical considerations and empirical evidence suggest handedness is an index of functional brain asymmetry, and therefore reflects something about underlying brain morphology, and given the non-random relationship between hand preference and language lateralisation, may reflect something about language lateralisation (see Springer & Deutch, 1993; Hellige, 1998; Geschwind, Miller, et al, 2002).

Hand preference was assessed by both parent report and behavioural tasks.

Parent report.

Parent report of hand prefence was assessed by items from the Edinburgh Handedness Inventory (Oldfield, 1972) which is a widely used instrument (see Geschwind, et al, 2002; Bishop, 2001; Williams, 1991). Parents reported on 4 items (writing, holding a toothbrush, throwing/catching a ball, holding a knife, without a fork) using a a 5 point scale, (always left = -2, usually left = -1, both hands equally = 0, usually right = 1, always right = 2). Scores for items are summed, positive score = right handed, negative total score = non-right handed.

Hand preference measures are highly correlated with behavioural measures like the pegboard test, finger tapping, and long pegboard test (see Chapman & Chapman, 1987; Bryden, Pryde, Roy, 2000; Peters, 1998).

Behavioural tasks.

1. Based on a study by Niswander & Gorden (1972) children were asked to draw the letter X three times, with a different colour pencil each time. Parents reported on which hand the child uses each time (e.g Left, 1, 2, 3, Right, 1, 2 ,3). Left hand values are scored as negative values, (e.g. -1, -2, -3) right hand values are scored as positive values (e.g. +1, +2, +3).

2. Based on a study by Zucker, Beaulieu, Bradley, Grimshaw, Wilcox, et al (2001) children were asked to copy four shapes (based on items taken from a developmental neuropsychological assessment (NEPSY) (Psychological Corporation, 1992). Parents reported on which hand the child used during the task (e.g. always left = -2, usually left = -1, both hands equally = 0, usually right = 1, always right = 2).

3. There is a distinction made within the literature between hand preference and hand skill (see Bryden, et al, 2000). Participants were asked to check as many boxes (within a 10 x 11 grid) as they could in 15 seconds, first with the right hand, then with the left hand. This task has little cognitive load and reflects the relative skill between the right and the left hand. The number of boxes checked is summed for each hand, then scores for the left hand are subtracted from scores for the right hand, and divided by the total number of

boxes checked (R - L/R + L). A positive score reflects an advantage for the right hand, and a negative score reflects an advantage for the left hand.

Immune disorder

Wamboldt, Schmitz, Mrazek, (1998) found cross-correlations of 0.26 for MZ twins and 0.04 for DZ twins between atopy symptoms and behaviour problems, 77% of this covariance was accounted for by common genetic factors. Immune disorder is also a correlate of cognitive variation and handedness (see Geschwind & Galaburda, 1987) with molecular genetic evidence suggesting a relationship between allelic variation in immune system antigens and handedness (see Yeo, et al, 1996).

Presence of immune disorder was assessed by parent report on six items (asthma, hayfever, eczema, rheumatoid artheritis, ulceritive colitis, other) used by Geschwind and colleagues in a series of studies (see Geschwind & Galaburda, 1987). For each item, there was a dichotomous scale (yes = 1, no = 0). Scores for each item were summed to give total scores for the scale.

Digit ratio

Participants were provided with written instructions and diagrams which enabled them to produce photocopies of each hand in the correct position. Digit ratio is a highly repeatable measure (Manning, et al, 2000) reviews of studies by Peters, et al (2002) suggest consistent sex differences (not due to the larger size of hands in males) but some variability in samples, effect sizes are small so power to detect an effect is low. In particular, measures from photocopies of the hands have been compared to direct measures in 30 subjects and found to be essentially the same (see Manning, Barley, et al, 2000).

Two measures of digit ratio were calculated:

2D/4D ratio.

2D/4D ratio is based on measures from the tip of the both the second and fourth finger to the first basal crease, the length of the second finger is then divided by the length of the fourth finger (Manning, et al, 1999).

Distal extent ratio.

Distal extent ratio – distal extent is the distance between the tip of the index and ring fingers and the middle finger tip, relative to the middle finger. This is measured by drawing a line on the photocopy from the midline of the middle finger, then a line at right angles to this midline across the tip of the middle finger to form a 'T'. The distance from the tip of the middle finger to the tip of the index and ring finger can then be measured. The ratio is calculated by dividing the tip extent of the ring finger by the tip extent of the index finger (4D/2D ratio) as a shorter 4D measure indicates greater distal extent of the ring finger (see Peters, et al, 2002).

For both measures a value of less than one indicates that the ring finger is longer than the index finger.

Peters et al (2002) found both measures of digit ratio have good reliability and inter-observer agreement. In their sample there was a higher proportion of individuals with ring fingers greater than index fingers (see table 2.1.3) However, this was significantly more marked in males than females. Tip measures showed more consistent gender differences than length measures, which were more marked for the left hand in males, and the right hand in females.

Figure 2.1.3 Percentage of individuals where the ring finger is longer than the index finger

	Left hand	Right hand
Males	87.8	86.9
Females	73.1	74.3

Although digit ratio is highly correlated with gender studies have shown that this measure can explain variation in behavioural markers in males (i.e. spatial task performance, musical ability, handedness) and in females (see Manning & Trivers, 2000: Austin, Manning, McInroy, Matthews, in press: Yeo & Gangsted, 1993).

Procedures

1150 families from the Twin Register (Centre for Psychological Research, University of Southampton) were contacted by mail. In addition, emails were forwarded to students

from the Medical School and School of Nursing at Southampton University. Individuals who replied to this email were then contacted by mail.

Questionnaires were mailed to the twin families with instructions for completion.

- Adolescents (individuals aged eighteen and under in October 2002) were asked to complete Parent report CATS, Youth report CATS, SDQ, Parent report school attainment, Parent Report handedness, Parent report presence of immune disorder, behavioural handedness measures (see Appendix A, B, D &E).
- Adults (individuals aged between nineteen and twenty-five years in October 2002) were asked to complete – Youth report CATS, self report GHQ, and a behavioural handedness measure for a subset of the sample (see Appendix A, & D).

For both groups a photocopy of the hands was requested (see Appenix F).

Participants

In total 296 families responded (29%) to the mailing. The adolescent group (n = 340) consisted of 158 males and 182 females. Mean age 13.86 years, sd = 3.52 years. This included 134 twin pairs (46 monozygotic pairs, 21 male and 25 female, 88 dyzgotic pairs, 21 male, 23 female, 44 mixed) and 72 siblings. The adult group (n = 269) consisted of 94 males and 175 females. Mean age 22.25 years, sd =2.03 years. This included (107 twin pairs, (48 monozygotic, 14 male,34 female, and 59 dizygotic pairs, 15 male,29 female, 15 mixed, 5 siblings, 55 unpaired twins).

Non-respondents

A total of 881 families did not respond to the mailing. These included 344 monozygotic pairs, 175 male, 169 female, and 537 dyzotic pairs, 166 male, 165 female, 206 mixed. Mean age 16.49 years, sd = 4.88.

Consent

For twins recruited via The Twin Register, consent had previously been obtained. Twins recruited via email, were asked to complete a consent form (parental consent was obtained, for individuals aged eighteen years and under).

Zygosity determination

For twins recruited via The Twin Register zygosity was established, For twins recruited by email zygosity was established using the Twin Similarity Questionnaire (Nichols & Bilbro, 1966) (see Appendix G). This is a brief questionnaire containing items regarding the physical similarity (e.g. eye colour, hair colour, weight, height) and physical confusability (e.g. 'do you ever confuse them') of the twins, which is completed by parents. For some items parents rate their twins on a dichotomous scale (yes/no) for other items on a scale from 0 to 2 (e.g. 'not at all', 'somewhat', 'exactly'). The higher the total score, the more similar in appearance the twins are. The maximum score is 20, and the general rule used was twins who scored 13 or more were classified as monozygotic, twins who scored 12 or less, were classified as dyzgotic (see Kuntsi & Stevenson, 2001). Zygosity determination by questionnaire has been shown to identify 96% of twins accurately (see Willcutts, Pennington, DeFries, 2000). In the sample recruited via email all monozygotic twins scored 16 all dyzogtic twins scored 8 or less.

2.3 PREDICTIONS

Personality and behavioural adjustment

1. Conscientiousness will be negatively associated with behavioural adjustment scores.
2. Daring will be positively associated with behavioural adjustment scores.
3. Negative emotionality will be positively associated with behavioural adjustment scores.
4. Scores for daring and negative emotionality may not contribute independently to scores for externalising dimensions.

Personality and school attainment

1. Conscientiousness will be positively associated with school attainment scores.
2. Daring will be negatively associated with school attainment scores.
3. Negative emotionality will be negatively associated with school attainment scores.

Psychopathology and school attainment

1. Behavioural adjustment scores will be negatively associated with school attainment scores.

Personality and digit ratio/hand preference/immune disorder

1. Digit ratio will be positively associated with negatively emotionality (females tend to score higher on trait neuroticism) and conscientiousness (the inverse of psychoticism, which males tend to score higher on) and negatively associated with daring (males tend to score higher on novelty/sensation seeking).

Psychopathology and digit ratio/hand preference/immune disorder

1. Behavioural adjustment scores will be negatively associated with digit ratio differences.
2. Behavioural adjustment scores will be positively associated with immune disorder.

School attainment and digit ratio/hand preference/immune disorder

1. School attainment scores reflecting verbal ability will be positively associated with digit ratio (females tend to score higher on verbal abilities).
2. School attainment scores reflecting spatial ability should be negatively associated with digit ratio (males tend to score higher on spatial ability) and positively associated with non-right handedness and immune disorder.
3. There will be larger digit ratio differences in non-right handed individuals.

Univariate analysis

1. Variance in personality will be accounted for by genetic and non-shared environmental influences.
2. Variance in adjustment, school attainment and developmental markers will be accounted for by genetic, shared and non-shared environmental influences.

1. Genetic influences will contribute to the associations between personality and adjustment, personality and school attainment, personality and developmental markers, adjustment and developmental markers.

2.4 FACTOR ANALYSIS OF PERSONALITY ITEMS

Principal Components Analysis using varimax rotation was conducted on the 50 items from each questionnaire which were included in analysis by Lahey, et al (in press) (see Appendix H). Kaiser's (1970) measure of sampling adequacy is a ratio of the sum of squared correlations to the sum of squared correlations plus the sum of squared partial correlations. For the parent report questionnaire items, the value was 0.863, and for the self report questionnaire items, the value was 0.838 indicating small partial correlations and therefore that the data was suitable for factor analysis.

For each questionnaire (parent and youth report) the same three principle factors were obtained as in the American population samples. (Parent report – factor one = conscientiousness, factor two = negative emotionality, factor three = daring, Youth report, factor one = conscientiousness, factor two = daring, factor three = negative emotionality). (see table 2.4.1).

Table 2.4.1, Eigenvalues, % of variance and cumulative % for factor analysis of parent and self report personality items.

	Eigenvalues	% of variance	Cumulative %
Parent report			
Conscientiousness	6.27	13.06	13.06
Negative emotionality	4.09	8.53	21.59
Daring	3.63	7.55	29.15
Self report			
Conscientiousness	3.91	8.15	8.15
Daring	3.40	7.08	15.23
Negative emotionality	2.79	5.81	21.04

2.5 PRELIMINARY ANALYSIS

Scores for the personality, adjustment and school attainment variables were relatively normally distributed, and given the large sample sizes no outliers were removed. For personality variables, where there were one or two missing scores, these were replaced with the median value. Standardised scores for each personality item were then totalled to create scores for each dimension. Prior to model fitting for each of the variables effects of age and gender were partialled out through multiple regression analysis. DZ mixed pairs were included in the analysis.

CHAPTER THREE

UNIVARIATE BEHAVIOUR GENETIC ANALYSIS

Full ACE models (Neale & Cardon, 1992) were fitted to each of the personality, adjustment, and cognitive variation variables*. These were then compared with AE and CE models. Fit of the models was assessed by the Comparative Fit Index (CFI) where values of 0.9 and greater indicate excellent fit. AE and CE models are nested within the main model (subsets of free parameters in these models are contained in the full model) and change in chi-square values was used to determine which model gave the most parsimonious fit. Chi-square values for the full model are subtracted from chi-square values for the sub-model, and the difference is evaluated with degrees of freedom equal to differences between degrees of freedom for the two models. In addition, ADE models were fitted where MZ correlations were more than twice DZ correlations, and for non-nested models (e.g ACE vs ADE) fit was compared using the Akakike's Information Criteria (AIC). This is based on the ratio of chi-square to the degrees of freedom and small, preferably negative values, indicate good fit (see Dunn, Everitt, Pickles, 1993). Typically parameters that do not significantly contribute to model fit are dropped (i.e. no significant change in chi-square values between models). However, given that estimates are based on small sample sizes full models were retained if these gave a reasonable fit (even where there was no significant change in chi-square values between models) since there was low power to detect significant effects.

Heritabilities (squared path values) are given in summary tables. Models marked with an * are based on correlation matrices as on inspection bad fit was due to variance differences in the two zygosity groups. However, similar path values for models using both covariance and correlation matrices justify the interpretation of path values.

* Full ACE models are not always reported here because on occasion they gave a very poor model of fit, or there was failure to converge.

3.1 PERSONALITY MODELS

3.1.1 PERSONALITY DIMENSIONS (ADOLESCENTS)

Conscientiousness

For parent report data the change in chi-square value was non-significant from the ADE model to the AE model $\Delta\chi^2$ (1) = 0.77 n.s. The chi-square value for the CE model was large relative to the degrees of freedom. AIC values indicate that the AE model and ADE models provide a better fit for the data than the CE model. As the ADE model did not significantly improve fit, the AE model was considered to be the most parsimonious. For self report data the change in chi-square value was non-significant from the ADE model to the AE model $\Delta\chi^2$ (1) = 1.06 n.s. The chi-square value for the CE model was large relative to the degrees of freedom. AIC values indicate that the AE and ADE models provide a better fit for the data. As the ADE model did not significantly improve fit, the AE model was considered to be the most parsimonious.

Daring

For parent report data the change in chi-square value was non-significant from the ACE* model to the AE model $\Delta\chi^2$ (1) = 2.80 n.s , and to the CE model $\Delta\chi^2$ (1) = 1.99 n.s. with the ACE model giving the most parsimonious fit based on number of degrees of freedom. (For comparison of path values between the ACE and ACE* models see Appendix I). For self report data the change in chi-square value was non-significant from the ACE model to the AE model $\Delta\chi^2$ (1) = 0.54 n.s , and to the CE model $\Delta\chi^2$ (1) = 0.09 n.s indicating that all the models fitted the data, with the ACE model giving the most parsimonious fit based on number of degrees of freedom.

Negative emotionality

For parent report data the change in chi-square value was non-significant from the ADE* model to the AE* model $\Delta\chi^2$(1) = 2.01. n.s. The chi-square value for the CE* model was large relative to the degrees of freedom. AIC values indicate that the AE and ADE models provide better fit for the data. As the ADE model did not significantly improve fit,

the AE model was considered to be the most parsimonious. For self report data the change in chi-square value was significant from the ACE* model to the AE model $\Delta\chi^2$ (1) = 5.96 p < .05, and to the CE model $\Delta\chi^2$ (1) = 4.23 p < .05 indicating that the full ACE* model is needed. (For comparison of path values between the models fitted to covariance and correlation matrices, see Appendix I).

Table 3.1.1.1. Fit of the models for personality dimensions (adolescents)

	X^2	Df	P	CFI	AIC
Parent conscientiousness					
ADE	1.30	3	.73	1.00	-4.69
AE	*2.07*	*4*	*.72*	*1.00*	*-5.93*
CE	19.25	4	.001	0.69	11.25
Self conscientiousness					
ADE	1.24	3	.74	1.00	-4.76
AE	*2.30*	*4*	*.68*	*1.00*	*-5.70*
CE	10.32	4	.03	0.74	2.32
Parent daring					
*ACE**	*0.00*	*3*	*1.0*	*1.00*	*-6.00*
AE	2.80	4	.58	1.00	-5.16
CE	1.99	4	.74	1.00	-6.00
Self daring					
ACE	*0.49*	*3*	*.92*	*1.00*	*-5.51*
AE	1.03	4	.90	1.00	-6.97
CE	0.58	4	.96	1.00	-7.42
Parent negative emotionality					
ADE*	0.0	3	1.0	1.00	-6.00
*AE**	*2.01*	*4*	*.73*	*1.00*	*-5.98*
CE*	27.96	4	.001	0.59	19.96
Self negative emotionality					
ACE*	*0.0*	*3*	*1.00*	*1.00*	*-6.0*
AE	5.96	4	.20	1.00	-2.03
CE	4.23	4	.37	1.00	-3.76

Italic font indicates a best fitting model, * indicates model fitted to correlation matrice

3.1.2 PERSONALITY DIMENSIONS (ADULTS)

Conscientiousness

The change in chi-square value was non-significant from the ACE model to the AE model $\Delta\chi^2$ (1) = 3.51 n.s, and to the CE model $\Delta\chi^2$ (1) = .46 p n.s , indicating that the ACE model gives the most parsimonious fit based on numbers of degrees of freedom.

Daring

The change in chi-square value was non-significant from the ACE model to the AE model $\Delta\chi^2$ (1) = 3.33 n.s. AIC values suggest that the ACE and AE models give a better fit than the CE model, with the ACE model gives the most parsimonious fit based on the number of degrees of freedom.

Negative emotionality

The change in chi-square value was non-significant from the ADE* model to the AE model $\Delta\chi^2$ (1) = 1.36 n.s. AIC values indicate that the AE and ADE models provide a better fit for the data than the CE model. As the ADE model did not significantly improve fit, the AE model was considered to be the most parsimonious. (see table 3.1.2.1) (For comparison of path values between the ADE and ADE* models see Appendix I).

Table 3.1.2.1, Fit for the models for personality dimensions (adults)

	χ^2	df	P	CFI	AIC
Conscientiousness					
ACE	*0.31*	*3*	*.98*	*1.00*	*-5.68*
AE	3.82	4	.43	1.00	-4.18
CE	0.77	4	.94	1.00	-7.25
Daring					
ACE	*1.08*	*3*	*.78*	*1.00*	*-4.92*
AE	4.41	4	.35	0.98	-3.59
CE	1.08	4	.89	1.00	-6.92
Negative emotionality					
ADE*	0.0	3	1.0	1.00	-6.00
AE	*1.36*	*4*	*.85*	*1.00*	*-6.63*
CE	4.89	4	.30	0.95	-3.11

Italic font indicates a best fitting model, * indicates model fitted to correlation matrice

3.1.3 PERSONALITY DIMENSIONS (COMBINED SCORES FOR ADOLESCENTS AND ADULTS)

Conscientiousness

The change in chi-square value was non-significant from the ADE model to the AE model $\Delta\chi^2$ (1) = 0.18 n.s. The chi-square value for the CE model was large relative to the degrees of freedom. AIC values indicate that the AE and ADE models provide a better fit for the data .than the CE model. As the ADE model did not significantly improve fit, the AE model was considered to be the most parsimonious.

Daring

The change in chi-square value was non-significant from the ACE model to the AE model $\Delta\chi^2$ (1) = 2.65 n.s., and to the CE model $\Delta\chi^2$ (1) = 0.28 n.s., indicating that the ACE model gives the most parsimonious fit based on number of degrees of freedom.

Negative emotionality

The change in chi-square value was non-significant from the ACE* model to the AE model $\Delta\chi^2$ (1) = 0.19 n.s., and to the CE model $\Delta\chi^2$ (1) = 0.85 n.s., indicating that the ACE model gives the most parsimonious fit based on the number of degrees of freedom. (For comparison of path values between the ACE and ACE* models see Appendix I).

Table 3.1.3.1, Fit for the models for personality dimensions (combined)

	χ^2	Df	p	CFI	AIC
Conscientiousness					
ADE	0.85	3	.84	1.00	-5.15
AE	*0.67*	*4*	*.95*	*1.00*	*-7.33*
CE	8.54	4	.07	0.89	0.54
Daring					
ACE	*0.47*	*3*	*.92*	*1.00*	*-5.52*
AE	3.12	4	.54	1.00	-4.88
CE	0.75	4	.94	1.00	-7.25
Negative emotionality					
*ACE**	*0.00*	*3*	*1.0*	*1.00*	*-6.00*
AE	0.19	4	.99	1.00	-7.80
CE	0.85	4	.14	0.90	-1.14

Italic font indicates a best fitting model, * indicates model fitted to correlation matrice

3.1.4 SUMMARY OF FINDINGS FOR PERSONALITY DIMENSIONS

It can be noted that variances for Mz pairs were slightly greater than for Dz pairs, however these differences were not significant. Additive genetic and unique environmental influences contribute to variation in scores for conscientiousness and negative emotionality. There was also some evidence of common environment influences on conscientiousness in the adult group, and for negative emotionality for self report scores in the adolescent group. For daring, additive genetic, common and unique environmental influences contributed to variation, with unique environmental influences accounting for the largest part of the variance.

Table 3.1.1 Summary of personality dimensions

	h^2	c^2	e^2	d^2
Conscientiousness				
Adolescents (parent report)	.77		.23	
Adolescents (self report)	.59		.41	
Adults	.21	.24	.55	
Combined	.54		.46	
Daring				
Adolescents (parent report)	.13	.20	.67	
Adolescents (self report)	.10	.18	.71	
Adults	.01	.44	.55	
Combined	.10	.28	.62	
Negative emotionality				
Adolescents (parent report)	.84		.16	
Adolescents (self report)	.13	.22	.65	
Adults	.53		.47	
Combined	.38	.07	.54	

Table 3.1.2, Total number of twins pairs, correlations, and standard deviations for personality models

	MZ				DZ			
	pairs	R	SD_1	SD_2	pairs	R	SD_1	SD_2
Conscientiousness								
Adolescents (pr)	44	.80	1.02	1.04	83	.29	0.90	0.97
Adolescents (sr)	44	.66	1.04	1.07	81	.18	0.93	0.98
Adults	47	.45	1.02	0.95	59	.35	0.97	0.99
Combined	91	.56	1.03	1.01	140	.25	0.94	0.98
Daring								
Adolescents (pr)	45	.33	0.91	0.92	84	.26	0.95	1.08
Adolescents (sr)	44	.27	0.89	0.95	80	.24	0.95	0.97
Adults	47	.45	1.00	1.00	59	.45	1.08	0.95
Combined	91	.37	0.94	0.97	139	.33	1.00	0.96
Negative emotionality								
Adolescents (pr)	45	.84	1.13	1.20	83	.28	0.91	0.82
Adolescents (sr)	43	.35	1.15	1.05	82	.29	0.98	0.88
Adults	48	.56	1.10	1.02	59	.23	1.01	0.94
Combined	91	.45	1.13	1.03	141	.27	0.99	0.91

* Parent report = pr, Self report = sr.

3.2 BEHAVIOUR ADJUSTMENT MODELS

For the emotion problems subscale of the SDQ the change in chi-square value was significant from the ADE model to the AE* model $\Delta\chi^2$ (1) = 5.49 $p < .05$. The chi-square value for the CE model was large relative to the degrees of freedom. AIC values indicate that the AE and ADE models provide a better fit for the data than the CE model. The chi-square value of the ADE model was large relative to the degrees of freedom, therefore the AE* model was considered to fit the data best. For the conduct problems subscale of the SDQ the change in chi-square value was significant from the ADE model to the AE* model $\Delta\chi^2$ (1) = 19.79 $p < .001$. The chi-square value for the CE model was large relative to the degrees of freedom. AIC values indicate that the ADE and CE models fit the data less well than the AE* model. For the hyperactivity subscale of the SDQ the change in chi-square value was significant from the AE* model to the CE* model $\Delta\chi^2$ (1) = 19.07 $p < .001$. The chi-square value for the CE model was large relative to the degrees of freedom.

For the peer problems subscale of the SDQ AIC values indicate that the CE models fit the data less well than the AE* model. For the total scores on the SDQ AIC values indicate that the CE models fit the data less well than the AE* model. For the total scores on the GHQ both AE and CE models fit the data well, however, AIC values suggest that the CE model fits the data less well than the AE model. For the combined adjustment scores AIC values suggest that the CE model fits the data less well than the AE model indicating that

the AE* model gave the best fit to the data. However the chi-square value for this model was just above the significance level, which indicates a poor fit. For the prosocial subscale of the SDQ AIC values indicate that the CE* models fit the data less well than the AE* model. (see table 3.2.1) (For comparison of path values between the AE and AE* models see Appendix I).

Table 3.2.1. Fit for the models for behavioural adjustment

	X^2	Df	P	CFI	AIC
Emotion problems(sdq)					
ADE	6.59	3	.08	0.91	0.59
*AE**	*1.10*	*4*	*.89*	*1.00*	*-6.89*
CE	21.09	4	.001	0.56	13.09
Conduct problems (sdq)					
ADE	23.65	3	.001	0.40	17.65
*AE**	*3.86*	*4*	*.42*	*1.00*	*-4.13*
CE*	18.69	4	.001	0.57	10.68
Hyperactivity (sdq)					
*AE**	*10.03*	*4*	*.03*	*0.76*	*2.02*
CE	29.10	4	.001	0.01	21.10
Peer problems (sdq)					
*AE**	*3.40*	*4*	*.49*	*1.00*	*-4.59*
CE*	10.83	4	.002	0.64	2.83
Total SDQ score					
*AE**	*3.40*	*4*	*.49*	*1.00*	*-4.59*
CE*	28.76	4	.001	0.54	20.76
Total GHQ score					
*AE**	*1.26*	*4*	*.89*	*1.00*	*-6.74*
CE*	2.01	4	.73	1.00	-5.98
SDQ prosocial					
*AE**	*1.12*	*4*	*.89*	*1.00*	*-6.88*
CE*	16.84	4	.00	0.70	8.83

Italic font indicates a best fitting model, * indicates model fitted to correlation matrice

3.2.1 SUMMARY OF BEHAVIOURAL ADJUSTMENT MODELS

Additive genetic and unique environmental influences contributed to variation in scores for all the SDQ subscales, with similar patterns for the prosociality subscale. However, for GHQ total scores estimates of genetic influences were much smaller, with the majority of variation accounted for by unique environmental influences (see table 3.2.2).

Table 3.2.2 ,Summary of heritabilities for behavioural adjustment models

	h^2	c^2	e^2	d^2
Adolescents				
Emotion problems	.72		.27	
Conduct problems	.71		.29	
Hyperactivity	.58		.42	
Peer problems	.53		.47	
Total SDQ scores	.83		.17	
SDQ Prosocial scores	.75		.24	
Adults				
Total GHQ scores	.14		.86	

Table 3.2.3, Total number of twins pairs, correlations, and standard deviations for behavioural adjustment models

	MZ				DZ			
	pairs	r	SD_1	SD_2	pairs	R	SD_1	SD_2
Adolescents								
Emotion problems	43	.75	0.97	1.09	85	.27	0.89	1.14
Conduct problems	43	.74	1.34	1.07	85	.17	0.82	0.75
Hyperactivity	43	.69	1.10	1.04	85	-.03	0.77	1.08
Peer problems	43	.61	1.07	0.93	84	.10	0.87	1.11
Total SDQ scores	43	.84	1.28	1.18	83	.24	0.81	0.85
SDQ prosocial	43	.77	1.15	1.05	86	.28	0.97	0.89
Adults								
Total GHQ scores	46	.22	0.74	0.82	58	-.06	1.02	1.12
Combined								
Adjustment scores	89	.64	1.04	1.00	141	.08	0.90	0.97

3.3 SCHOOL ATTAINMENT MODELS

For reading the change in chi-square value was non-significant from the ACE* model to the AE model $\Delta\chi^2$ (1) = 2.30 n.s. but significant to the CE model $\Delta\chi^2$ (1) = 10.53 $p < .01$ indicating that the ACE* and AE models fit the data best, with the ACE* model giving the most parsimonious fit based on number of degrees of freedom. For spelling the change in chi-square value was non-significant from the ADE model to the AE* model $\Delta\chi^2$ (1) = 1.42 n.s, with both models fitting the data well. For the CE model the chi-square value was large relative to the degrees of freedom. As the ADE model did not significantly improve fit, the AE* model was considered to be the most parsimonious. For handwriting the chi-square value for the CE model was large relative to the degrees of freedom. AIC values indicate that the AE* model gives the most parsimonious fit. For maths the change in chi-square value was non-significant from the ACE model to the AE model $\Delta\chi^2$ (1) = 1.49 n.s. but significant to the CE model $\Delta\chi^2$ (1) = 10.51 $p < .01$ indicating

that the ACE and AE models fit the data best, with the ACE model giving the most parsimonious fit based on number of degrees of freedom. For art the chi-square value was large relative to the degrees of freedom for the CE model. AIC values indicate that the AE* model gives the most parsimonious fit. For music the change in chi-square value was non-significant from the ACE model to the AE model $\Delta\chi^2$ (1) = 0.31 n.s. but significant to the CE model $\Delta\chi^2$ (1) = 5.67 $p < .05$ indicating that the ACE and AE models fit the data best, with the ACE model giving the most parsimonious fit based on number of degrees of freedom. For computers the chi-square value was large relative to the degrees of freedom for the AE* model. AIC values indicate that the CE* model gives the most parsimonious fit. For science the change in chi-square value was significant from the ACE to the AE model $\Delta\chi^2$ (1) = 7.2 $p < .01$ but not to the CE model $\Delta\chi^2$ (1) = 0.87 n.s, indicating that ACE and CE models fit the data best, with the CE model being the most parsimonious. For pe/games the change in chi-square value was non-significant from the ACE to the AE model $\Delta\chi^2$ (1) = 0.30 n.s. but significant to the CE model $\Delta\chi^2$ (1) = 15.9 $p < .01$, indicating that ACE and AE models fit the data best, with the ACE model being the most parsimonious based on number of degrees of freedom. (see table 3.3.1). (For comparison of path values between models fitted to covariance and correlation matrices, see Appendix I).

Table 3.3.1, Fit for the models for school attainment

	X^2	Df	p	CFI	AIC
Reading					
*ACE**	*0.0*	*3*	*1.0*	*1.00*	*-6.00*
AE	2.30	4	0.68	1.00	-5.69
CE	10.53	4	0.03	0.84	2.53
Spelling					
ADE	1.09	3	.78	1.00	-4.90
AE	*2.51*	*4*	*.64*	*1.00*	*-5.49*
CE	10.83	4	.02	0.71	2.83
Handwriting					
*AE**	*2.02*	*4*	*.73*	*1.00*	*-5.97*
CE*	7.33	4	.12	0.75	-0.60
Maths					
ACE	*2.77*	*3*	*0.43*	*1.00*	*-3.22*
AE	4.26	4	0.37	0.99	-3.73
CE	13.28	4	0.01	0.71	5.27
Art					
*AE**	*7.27*	*4*	*0.12*	*0.91*	*-0.73*
CE*	25.43	4	0.001	0.44	17.43
Music					
ACE	*0.48*	*3*	*0.92*	*1.00*	*-5.52*
AE	0.79	4	0.94	1.00	-7.20
CE	6.15	4	0.18	0.94	-1.84
Computers					
AE*	9.64	4	.04	.86	1.63
*CE**	*0.19*	*4*	*.99*	*1.00*	*-7.80*
Science					
ACE	0.45	3	.93	1.00	-5.55
AE	7.65	4	.10	0.89	-0.35
CE	*1.32*	*4*	*.86*	*1.00*	*-6.68*
Pe/games					
ACE	*1.30*	*3*	*.72*	*1.00*	*-4.69*
AE	1.60	4	.81	1.00	-6.40
CE	17.20	4	.001	0.81	9.20

Italic font indicates a best fitting model, * indicates model fitted to correlation matrice

3.3.1 SUMMARY OF SCHOOL ATTAINMENT MODELS

Additive genetic and unique environmental variance contributed to variation in scores for reading, spelling, handwriting, maths, music, art, and pe/games. There was also some evidence of a modest common environmental influence on reading, maths, music and pe/games. Variation in science and computers was largely explained by common and unique environmental influences, with a very small genetic contribution to science (see table 3.3.2).

Table 3.3.2, Summary of heritabilities for school attainment models

	h^2	c^2	e^2	d^2
Reading	58	.12	.30	
Spelling	.61		.38	
Handwriting	.48		.51	
Maths	.58	.21	.20	
Music	.57	.11	.32	
Art	.75		.25	
Computers		.53	.47	
Science	.005	.49	.50	
Pe/games	.72	.10	.18	

Table 3.3.3 Total number of twins pairs, correlations, and standard deviations for school attainment

	MZ				DZ			
	pairs	r	SD_1	SD_2	pairs	r	SD_1	SD_2
Reading	42	.70	0.96	0.91	83	.41	1.08	0.97
Spelling	42	.64	1.05	0.93	84	.20	1.03	1.03
Handwriting	41	.55	1.03	0.85	84	.11	1.00	1.05
Maths	42	.83	1.09	0.96	84	.49	0.96	0.94
Music	42	.68	1.03	0.97	84	.40	1.03	0.99
Art	42	.79	0.95	1.03	84	.12	1.02	0.98
Computers	42	.50	0.91	1.02	84	.55	1.05	0.97
Science	42	.54	0.99	1.04	84	.49	0.96	0.90
Pe/games	42	.85	1.05	1.05	84	.44	0.96	0.91

3.4 DIGIT RATIO MODELS

Univariate models were fitted for both the average and difference measures for both 2d4d and distal extent ratios. For 2d/4d ratios the change in chi-square value was non-significant from the ACE to the AE model $\Delta\chi^2$ (1) = 0.51 n.s, and to the CE model $\Delta\chi^2$ (1) = 2.29 n.s, indicating that ACE and AE models fit the data best, with the ACE model being the most parsimonious based on number of degrees of freedom. For 2d/4d ratio difference both AE* and CE* models fitted the data well. AIC values indicate that the CE* model fitted the data less well than the AE* model. For distal extent average the change in chi-square value was non-significant from the ACE* to the AE* model $\Delta\chi^2$ (1) = 0.26 n.s, and to the CE model $\Delta\chi^2$ (1) = 1.81 n.s, indicating that all models fit the data well, with the ACE* model being the most parsimonious based on number of degrees of freedom. For distal extent difference both AE* and CE* models fit the data well. AIC values indicate that the CE* model fits the data less well than the AE* model. (see table 3.4.1). (For comparison of path values between models fitted to covariance and correlation matrices, see Appendix I).

Table 3.4.1, Fit for the models for digit ratio(combined)

	X^2	Df	p	CFI	AIC
2d/4d ratio (average)					
ACE	*0.26*	*3*	*.97*	*1.00*	*-5.74*
AE	0.74	4	.94	1.00	-7.22
CE	2.55	4	.63	1.00	5.44
2d/4d ratio difference					
*AE**	*2.79*	*4*	*.59*	*1.00*	*-5.21*
CE*	3.93	4	.41	1.00	-4.07
Distal extent (average)					
*ACE**	*0.00*	*3*	*1.0*	*1.00*	*-6.00*
AE*	0.26	4	.99	1.00	-7.73
CE*	1.81	4	.77	1.00	-6.19
Distal extent difference					
*AE**	*0.95*	*3*	*.92*	*1.00*	*-7.04*
CE*	1.89	4	.76	1.00	-6.11

Italic font indicates a best fitting model, * indicates model fitted to correlation matrice

3.4.1 SUMMARY OF FINDINGS FOR DIGIT RATIO

Additive genetic, common and unique environmental influences contributed to variation in digit ratio (2d4d and distal extent). For digit ratio differences (discrepancies between ratios for the left and the right hand) there was no evidence of common environment influences (see table 3.4.2).

Table 3.4.2, Summary of heritabilities for digit ratio models

	h^2	c^2	e^2	d^2
Combined				
2d4d ratio (average)	.39	.16	.44	
2d4d ratio difference	.15		.85	
Distal extent (average)	.39	.13	.48	
Distal extent difference	.31		.72	

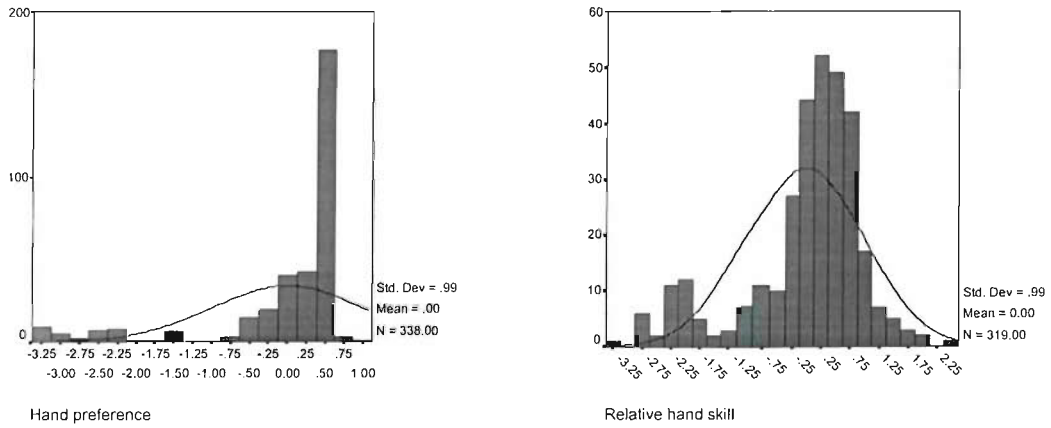
Table 3.4.3, Total number of twins pairs, correlations, and standard deviations for digit ratio

MZ					DZ			
	pairs	r	SD_1	SD_2	pairs	r	SD_1	SD_2
Combined								
2d4d ratio (average)	56	.55	0.81	0.84	69	.36	0.82	0.85
2d4d ratio difference	56	.25	0.86	0.84	69	-.10	0.83	0.90
Distal extent (average)	50	.52	0.92	1.12	58	.33	0.73	0.92
Distal extent difference	50	.36	0.96	0.58	58	.04	0.70	0.88

3.5 HANDEDNESS AND IMMUNE DISORDER MODELS

Geschwind & Galaburda's (1985) model of handedness suggests a basic evolutionary pattern of leftward brain asymmetry, with intra-uterine environmental influences underlying a shift to less asymmetry and non-right-handedness. This implies common influences underlie both right and non-right handedness. However, genetic models of handedness (e.g. Annett, McManus) imply different influences underlie the distribution of handedness in right and non-right handed individuals as they suggest genotype differences between the two groups. For the sample the distribution of scores for handedness showed a typical rightward shift, however the distribution for scores for hand skill was bimodal (see figure 3.5.1) and independent t-tests showed significant mean differences between scores for hand skill for right-handed and non-right-handed individuals $t_{(304)} = -16.95$ $p = < 0.001$.

Figure 3.5.1, Distribution of standardised scores for hand preference and hand skill



Therefore to look at genetic and environmental influences underlying variation in handedness the sample was divided into two groups based on scores for hand preference. Individuals scoring less than zero were classified as non-right handed (n = 39, 11.71% of the sample) and individuals scoring zero and above were classified as right-handed (n = 294).

Of the sample 119 twin pairs were concordant for right-handedness, one pair was concordant for left-handedness, and 11 pairs were discordant for handedness. Univariate genetic analysis for relative hand skill was only carried out on the concordant RH pairs as there were inadequate numbers of non-right-handed pairs.

For hand skill (concordant RH pairs) both AE* and CE* models fit the data well with AIC values indicating that the AE* model was the most parsimonious . For immune disorder (adolescents) only an AE model would converge. (see table 3.5.1)

Table 3.5.1, Fit for the models for handedness (combined) and immune disorder

	X^2	df	p	CFI	AIC
Hand skill(combined)					
*AE**	*1.20*	*4*	*.88*	*1.00*	*-6.80*
CE*	2.09	4	.72	1.00	-5.91
Immune disorder(Adolescents)					
*AE**	*3.22*	*4*	*52*	*1.00*	*-4.77*

3.5.1 SUMMARY OF FINDINGS FOR HANDEDNESS AND IMMUNE DISORDER

Variance in hand skill among right handed individuals showed a small additive genetic influence, but was mainly accounted for by unique environmental influences. Variation in immune disorder was accounted for by additive genetic and unique environmental influences (see table 3.5.2).

Table 3.5.2, Summary of handedness and immune disorder scale models

	h^2	c^2	e^2	d^2
Hand skill	.18		.83	
Immune disorder (adolescents)	.63		.36	

Table 3.5.3, Total number of twins pairs, correlations, and standard deviations for handedness (RH pairs) and immune disorder

MZ					DZ			
	pairs	r	SD_1	SD_2	pairs	r	SD_1	SD_2
Adolescents								
Hand skill	35	.27	0.58	0.64	63	-.03	0.63	0.77
Immune disorder	41	.68	1.00	0.91	83	.15	0.91	1.09

3.6 TWIN AND SIBLING ANALYSIS

To control for any effects of bias in the twin data models were fitted to the personality and adjustment variables for the entire adolescent sample. These models are presented in

addition to the twin models in order to demonstrate a consistent pattern of same effects for twins and siblings and twins alone.

3.6.1 PERSONALITY VARIABLES

Conscientiousness

For parent report the chi-square value for the CE* model was large relative to the degrees of freedom. AIC values indicate that the AE* model provides a better fit than the CE model. For self-report the chi-square value for the CE model was large relative to the degrees of freedom. AIC values indicate that the AE model provides a better fit than the CE model.

Daring

For parent-report the change in chi-square value was non-significant from the ACE model to the AE model $\Delta\chi^2$ (1) = 0.09 n.s. and to the CE model $\Delta\chi^2$ (1) = 0.95 n.s indicating that the ACE model gives the most parsimonious fit based on number of degrees of freedom. For self-report both AE and CE models fit the data well. AIC values indicate that the AE model provides a better fit than the CE model.

Negative emotionality

For parent-report the chi-square value for the CE model was large relative to the degrees of freedom. AIC values indicate that the AE model provides a better fit than the CE model. For self-report the change in chi-square value was non-significant from the ACE to the AE model $\Delta\chi^2$ (1) = 0.71 n.s. and to the CE model $\Delta\chi^2$ (1) = 0.18 n.s. The ACE model gives the most parsimonious fit based on number of degrees of freedom.

Table 3.6.1.1 Fit for the personality models

	χ^2	Df	p	CFI	AIC
Conscientiousness (parent-report)					
*AE**	*17.56*	*7*	*.01*	*0.78*	*3.56*
CE*	36.56	7	.00	0.40	22.56
Conscientiousness (self-report)					
AE	*13.08*	*7*	*.07*	*0.74*	*- 0.92*
CE	22.32	7	.00	0.36	8.23
Daring (parent-report)					
ACE	*3.10*	*6*	*.80*	*1.00*	*- 8.90*
AE	3.19	7	.87	1.00	-10.81
CE	4.05	7	.77	1.00	- 9.94
Daring (self-report)					
AE	*3.74*	*7*	*.81*	*1.00*	*-10.26*
CE	4.55	7	.71	1.00	- 9.44
Negative emotionality (parent-report)					
*AE**	*11.67*	*7*	*.11*	*0.92*	*- 2.33*
CE*	37.94	7	.00	0.46	23.94
Negative emotionality (self-report)					
ACE	*8.37*	*6*	*.21*	*0.77*	*- 3.63*
AE	9.08	7	.24	0.80	- 4.92
CE	8.55	7	.28	0.85	- 5.44

3.6.2 SUMMARY OF FINDINGS FOR PERSONALITY DIMENSIONS

Additive genetic and unique environmental factors contribute to variation in both parent and self-report conscientiousness. There was evidence of common environmental influences on parent-report daring only, also for negative emotionality self-report. This replicates the models fitted to the twin data alone, except for daring self-report where there was evidence of common environmental influences in the model fitted to twin data.

Table 3.6.1.2 Summary of heritabilities for personality dimension models

	h^2	c^2	e^2
Conscientiousness			
Parent-report	.70		.29
Self-report	.51		.49
Daring			
Parent-report	.33	.06	.61
Self-report	.29		.70
Negative emotionality			
Parent-report	.81		.19
Self-report	.12	.17	.70

Table 3.6.1.3 Total number of twin and twin-sibling pairs, correlations and standard deviations for the personality dimensions

	MZ Pairs	r	sd1	sd2
Conscientiousness				
Parent-report	44	0.80	1.02	1.05
Self-report	44	0.66	1.04	1.07
Daring				
Parent-report	45	0.33	0.92	0.92
Self-report	44	0.28	0.89	0.95
Negative emotionality				
Parent-report	45	0.84	1.13	1.20
Self-report	43	0.35	1.15	1.05
	DZ Pairs	r	sd1	sd2
Conscientiousness				
Parent-report	84	0.29	0.90	0.96
Self-report	83	0.17	0.92	0.98
Daring				
Parent-report	85	0.24	0.96	1.07
Self-report	81	0.24	0.94	0.97
Negative emotionality				
Parent-report	84	0.26	0.91	0.82
Self-report	84	0.29	0.97	0.88
	Twin-sibling pairs			
	Pairs	r	sd1	sd2
Conscientiousness				
Parent-report	71	-0.06	0.95	1.10
Self-report	59	-0.03	0.78	1.04
Daring				
Parent-report	71	0.22	1.02	1.07
Self-report	58	0.03	0.97	1.07
Negative emotionality				
Parent-report	70	0.09	1.08	1.07
Self-report	58	0.11	1.00	0.83

3.6.3 ADJUSTMENT VARIABLES

For emotion problems, AIC values indicate that the AE model provides a better fit than the ADE or AE models. For conduct problems, hyperactivity, peer problems and prosociality sub-scales, and the total SDQ the chi-square value for the CE models was large relative to the degrees of freedom. AIC values indicated that the AE models provide a better fit than the CE models.

Table 3.6.3.1 Fit for the SDQ total and subscale models

	χ^2	df	P	CFI	AIC
Emotion problems					
AE	*15.70*	*7*	*.03*	*.80*	*1.70*
ADE	14.87	6	.02	.80	2.86
CE	28.48	7	.00	.51	14.48
Conduct problems					
*AE**	*10.87*	*7*	*.14*	*.88*	*- 3.12*
CE*	24.24	7	.00	.49	10.24
Hyperactivity					
*AE**	*13.50*	*7*	*.06*	*.74*	*- 0.47*
CE*	22.31	7	.00	.38	8.31
Peer problems					
AE	*13.50*	*7*	*.06*	*.62*	*- 0.50*
CE	20.22	7	.00	.23	6.22
Total SDQ					
AE	*16.25*	*7*	*.02*	*.84*	*2.52*
CE	35.24	7	.00	.53	21.23
Prosociality SDQ					
AE	*9.22*	*7*	*.24*	*.95*	*- 4.77*
CE	24.49	7	.00	.59	10.49

3.6.4 SUMMARY OF FINDINGS FOR ADJUSTMENT

Additive genetic and unique environmental factors contributed to variation in all the sub-scale and total SDQ scores. This replicates the models based on twin data alone.

Table 3.6.3.2 Summary of heritabilities for behavioural adjustment models

	h^2	c^2	e^2
Emotion problems	.69		.31
Conduct problems	.62		.37
Hyperactivity	.48		.52
Peer problems	.43		.57
Total SDQ	.75		.24
Prosociality SDQ	.67		.33

Table 3.6.3.3 Total number of twin and twin-sibling pairs, correlations and standard deviations for the adjustment dimensions

	MZ Pairs	r	sd1	sd2
Emotion problems	43	.75	0.97	1.09
Conduct problems	43	.74	1.34	1.07
Hyperactivity	43	.69	1.10	1.04
Peer problems	43	.61	1.07	0.93
Total SDQ	43	.84	1.28	1.18
Prosociality SDQ	43	.77	1.15	1.05
	DZ Pairs	r	sd1	sd2
Emotion problems	86	.27	0.89	1.14
Conduct problems	86	.17	0.81	0.75
Hyperactivity	86	-.02	0.80	1.07
Peer problems	85	.10	0.87	1.11
Total SDQ	84	.23	0.80	0.86
Prosociality SDQ	86	.28	0.97	0.89
	Twin-sibling pairs			
	Pairs	r	sd1	sd2
Emotion problems	69	.30	0.78	0.89
Conduct problems	69	.04	1.11	1.15
Hyperactivity	69	.07	1.07	1.00
Peer problems	69	.01	0.90	0.98
Total SDQ	66	.34	1.04	1.04
Prosociality SDQ	69	.10	1.00	1.00

3.6.5 SCHOOL ATTAINMENT, DIGIT RATIO AND IMMUNE DISORDER VARIABLES

School attainment

For reading, spelling, maths and art, the chi-square values for the CE models were large relative to the degrees of freedom. AIC values indicate that the AE models provide a better fit than the CE models. For handwriting AIC values indicate that the AE model provides a better fit than the CE model. For music the change in chi-square value was significant from the ACE to the CE model $\Delta\chi^2$ (1) = 6.81 $p < 0.05$, but not to the AE model $\Delta\chi^2$ (1) = 0.35 n.s. The ACE model provides the most parsimonious fit based on number of degrees of freedom. For computers the change in chi-square value was significant from the ACE to the AE model $\Delta\chi^2$ (1) = 5.93 $p < 0.05$, but not to the CE model $\Delta\chi^2$ (1) = 0.25 n.s. The ACE model provides the most parsimonious fit based on number of degrees of freedom. For science the change in chi-square value was non-significant from the ACE model to the AE model $\Delta\chi^2$ (1) = 2.98 n.s. and to the CE model $\Delta\chi^2$ (1) = 0.98 n.s. The

ACE model gives the most parsimonious fit based on number of degrees of freedom. For pe/games the change in chi-square value was significant from the ACE to the CE model $\Delta\chi^2$ (1) = 21.59 $p < 0.05$, but not to the AE model $\Delta\chi^2$ (1) = 2.48 n.s. The ACE model provides the most parsimonious fit based on number of degrees of freedom.

Digit ratio and immune disorder models

For mean 2d4d ratio the AE and the CE models fit the data well. Based on AIC values the AE model provides the best fit. For immune disorder, the chi-square value for the CE model was large relative to the degrees of freedom. AIC values indicate that the AE model provides the best fit.

Table 3.6.5.1 Fit for the school attainment, digit ratio and immune disorder models

	X^2	df	p	CFI	AIC
Reading					
AE	*7.22*	*7*	*.41*	*.99*	*- 6.78*
CE	19.26	7	.00	.70	5.26
Spelling					
AE	*10.52*	*7*	*.16*	*.85*	*- 3.47*
CE	18.62	7	.01	.49	4.62
Handwriting					
AE	*5.80*	*7*	*.56*	*1.00*	*- 8.20*
CE	11.18	7	.13	.77	- 2.81
Maths					
AE	*11.23*	*7*	*.12*	*.94*	*- 2.76*
CE	30.91	7	.00	.66	16.91
Art					
AE	*10.58*	*7*	*.16*	*.91*	*- 3.42*
CE	27.63	7	.00	.50	13.63
Music					
ACE	*1.54*	*6*	*.96*	*1.00*	*- 10.46*
AE	1.89	7	.96	1.00	-12.11
CE	8.35	7	.30	.97	- 5.65
Computers					
ACE	*3.98*	*6*	*.68*	*1.00*	*- 8.02*
AE	9.91	7	.19	.94	- 4.09
CE	4.23	7	.75	1.00	- 9.77
Science					
ACE	*7.73*	*6*	*.26*	*.96*	*- 4.27*
AE	10.71	7	.15	.91	- 3.29
CE	8.71	7	.27	.96	- 5.29
Pe/games					
ACE	*10.64*	*6*	*.10*	*.93*	*- 1.36*
AE	8.16	7	.32	.98	- 5.84
CE	32.23	7	.00	.64	18.23
Mean 2d4d ratio					
AE	*2.69*	*7*	*.91*	*1.00*	*-11.30*
CE	7.34	7	.39	.98	- 6.66
Immune disorder					
AE	*8.93*	*7*	*.26*	*.93*	*- 5.07*
CE	19.69	7	.00	.57	5.69

3.6.6 SUMMARY OF FINDINGS FOR THE SCHOOL ATTAINMENT, DIGIT RATIO AND IMMUNE DISORDER MODELS

Additive genetic and unique environmental factors contributed to variation in reading, spelling, handwriting, maths, art, mean 2d4d ratio, and immune disorder. For music, computers, science and pe/games there were also common environmental influences. This replicates findings based on twin data alone, except for evidence of no common

environment influences on reading and maths, and for evidence of genetic influences on computers.

Table 3.6.5.2 Summary of school attainment, digit ratio and immune disorder models

	h^2	c^2	e^2
Reading	.70		.30
Spelling	.50		.50
Handwriting	.50		.50
Maths	.80		.20
Art	.72		.28
Music	.60	.09	.31
Computers	.12	.39	.49
Science	.25	.28	.47
Pe/games	.84	.007	.15
Mean 2d4d ratio	.67		.32
Immune disorder	.64		.36

Table 3.6.5.3 Total number of twin and twin-sibling pairs, correlations and standard deviations for the school attainment, digit ratio and immune disorder variables

	MZ Pairs	r	sd1	sd2
Reading	42	.70	0.96	0.91
Spelling	42	.64	1.05	0.93
Handwriting	41	.55	1.03	0.85
Maths	42	.83	1.09	0.96
Art	42	.79	0.95	1.03
Music	42	.68	1.03	0.98
Computers	42	.50	0.91	1.02
Science	42	.54	0.99	1.04
Pe/games	42	.85	1.05	1.05
Mean 2d4d ratio	24	.69	0.84	0.80
Immune disorder	41	.68	1.00	0.91
	DZ Pairs	r	sd1	sd2
Reading	84	.41	1.08	0.96
Spelling	85	.21	1.03	1.02
Handwriting	85	.11	1.00	1.05
Maths	85	.50	0.96	0.93
Art	85	.13	1.04	0.98
Music	85	.40	1.05	0.99
Computers	85	.53	1.04	0.97
Science	85	.48	0.96	0.90
Pe/games	85	.43	0.96	0.91
Mean 2d4d ratio	41	.37	0.74	0.74
Immune disorder	83	.15	0.91	1.09
	Twin-sibling pairs			
	Pairs	r	sd1	sd2
Reading	70	.12	0.97	1.02
Spelling	70	-.04	0.89	0.97
Handwriting	70	.28	1.05	0.98
Maths	70	.19	0.95	1.07
Art	70	.22	1.04	0.96
Music	70	.38	1.07	0.97
Computers	70	.36	1.04	0.96
Science	70	.33	0.96	1.13
Pe/games	70	.21	0.92	1.07
Mean 2d4d ratio	31	.28	0.90	0.84
Immune disorder	66	.28	1.12	0.99

CHAPTER FOUR
PHENOTYPE CORRELATIONS AND ASSOCIATIONS BETWEEN CONSTRUCTS

4.1 PHENOTYPE CORRELATIONS

Pearson product moment correlations were conducted for each of the personality, adjustment, school attainment, handedness and digit ratio variables.

4.1.1 PERSONALITY

For each personality dimension, parent and self report measures were more highly correlated with each other, than with any other dimension, which suggests a level of agreement between informants (see table 4.1.1.1).

Table 4.1.1.1, Phenotype correlations for personality dimensions

	P cons	P dar	P neg	S cons	S dar	S neg
Adolescents						
P cons	1.00					
P dar	0.05	1.00				
P neg	-0.35**	0.11	1.00			
S cons	0.44**	-0.07	-0.18**	1.00		
S dar	-0.10	0.50**	0.10	-0.08	1.00	
S neg	-0.08	-0.10	0.32**	-0.09	-0.01	1.00
Adults						
Cons				1.00		
Dar				-0.12*	1.00	
Neg				-0.05	-0.12	1.00

* $p < 0.05$ ** $p < 0.01$

4.1.2 BEHAVIOURAL ADJUSTMENT

All four problem sub-scales were significantly correlated with the total difficulties score for the SDQ. Emotion and conduct problems was significantly correlated with all other problem subscale scores, however, there was no relationship between hyperactivity and peer problems. Scores for the prosociality subscale were significantly negatively correlated with all the problem subscale dimensions (see table 4.1.2.1).

Table 4.1.2.1, Adjustment subscale correlations

	Emot	Cond	Hype	Peer	TSDQ	SDQPR
Emot	1.00					
Cond	0.34**	1.00				
Hype	0.26**	0.53**	1.00			
Peer	0.25**	0.20**	0.06	1.00		
TSDQ	0.71**	0.75**	0.75**	0.48**	1.00	
SDQPR	-0.11*	-0.45**	-0.26**	-0.18**	-.36**	1.00

* $p < 0.05$ ** $p < 0.01$

4.1.3 SCHOOL ATTAINMENT

There were significant positive relationships between the school attainment scores. However scores for pe/games were not related to reading, spelling or maths, and scores for art were not related to reading, maths, science or computers (see table 4.1.3.1).

Table 4.1.3.1, School attainment correlations

	Read	Spll	Hwrit	Maths	Art	Mus	Comp	Scie	Pe
Read	1.00								
Spll	0.62**	1.00							
Hwrit	0.38**	0.47**	1.00						
Maths	0.42**	0.43**	0.22**	1.00					
Art	0.08	0.13*	0.29**	0.09	1.00				
Mus	0.14**	0.12*	0.14*	0.12*	0.12*	1.00			
Comp	0.21**	0.20**	0.24**	0.16**	0.02	0.12*	1.00		
Scie	0.34**	0.29**	0.18**	0.48**	0.05	0.13*	0.31**	1.00	
Pe	-0.11	0.03	0.14*	0.04	0.13*	0.12*	0.11*	0.12*	1.00

* $p < 0.05$ ** $p < 0.01$

4.1.4 HANDEDNESS

There were significant positive relationships between each of the handedness measures, which suggests a very high agreement between parent report and behavioural measures of hand preference. Correlations between scores for relative hand skill and the hand preference measures were slightly lower and differed between right and non-right handed individuals (see tables 4.1.4.1, 4.1.4.2, and 4.1.4.3) suggesting that hand preference and relative hand skill may reflect different aspects of handedness

Table 4.1.4.1, Correlations for handedness measures

	Hand p	Copy X	Shapes	Hand sk	Immune
Hand pr	1.00				
Copy X	0.90**	1.00			
Shapes	0.90**	0.99**	1.00		
Hand sk	0.72**	0.76**	0.76**	1.00	
Immune	-0.03	-0.01	0.00	0.02	1.00

* $p < 0.05$ ** $p < 0.01$

Table 4.1.4.2 Correlations between handedness measures for non-right-handed individuals

	Copy x	Shapes	Handskill
Copy x	1.00		
Shapes	1.00**	1.00	
Handskill	0.76**	0.76**	1.00

** significant at the 0.01 level

Table 4.1.4.3 Correlations between handedness measures for right-handed individuals

	Copy x	Shapes	Handskill
Copy x	1.00		
Shapes	0.97**	1.00	
Handskill	0.44**	0.43**	1.00

** significant at the 0.01 level

4.1.5 DIGIT RATIO

Measures for 2d/4d and distal extent ratios were significantly correlated (see table 4.1.5.2). Means for both measures of digit ratio were comparable to findings from a literature (see table 4.1.5.1) review by Peters, et al (2002), in that study the mean for males was 0.92 – 0.98, and for females 0.92 – 1.00, and means for distal extent measures were 10.3 – 12.7 for males, and 10.2 to 10.8 for females. It was not possible to analysise digit ratio measures by gender due to the sample size.

Table 4.1.5.1 Digit ratio means(combined)

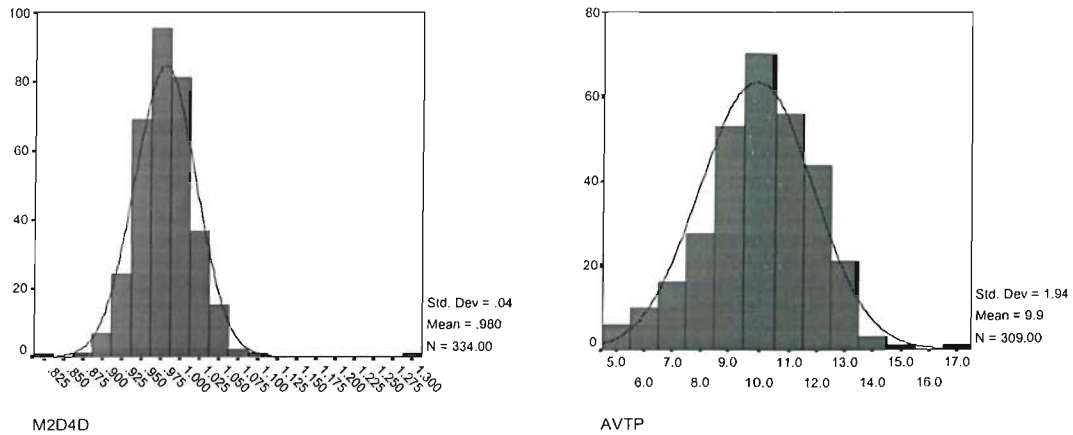
	Mean	sd
2d4d L	0.98	0.05
2d4d R	0.98	0.04
Dext L i	9.54	2.72
Dext L r	10.50	2.87
Dext R i	9.15	2.91
Dext R r	10.45	2.94

Table 4.1.5.2, Digit ratio correlations(combined)

	2D4D L	2D4D R	DEXT L	DEXT R
2D4D L	1.00			
2D4D R	0.45**	1.00		
DEXT L	0.48**	0.31**	1.00	
DEXT R	0.33**	0.46**	0.51**	1.00

* $p < 0.05$ ** $p < 0.01$

Figure 4.1.5.1 distributions for 2d4d and distal extent measures



For each variable independent t-tests showed no significant mean differences in scores between: twins and siblings; between MZ and DZ twins; between right and non-right handed individuals.

4.2 ASSOCIATIONS BETWEEN CONSTRUCTS

To look at the relationship between the personality and the adjustment measures, Pearson Product moment correlations were conducted between each of the three personality dimensions and the adjustment and school attainment measures. To control for multiple testing Bonferroni corrections were applied separately to each correlation table.

4.2.1 PERSONALITY AND ADJUSTMENT

Conscientiousness

Variation in conscientiousness was negatively associated with adjustment in the adolescent group. Individuals who scored higher on parent report conscientiousness had lower scores for conduct problems, hyperactivity, and the total difficulties subscale of the SDQ, and showed more prosocial behaviour. Individuals who scored higher on self report conscientiousness had significantly lower scores for conduct problems, hyperactivity, peer problems, and the total difficulties subscale of the SDQ, and showed more prosocial behaviour. However, variation in conscientiousness was not associated with emotion problems. There was no relationship between conscientiousness and adjustment in the adult group (see tables 4.2.1.1, 4.2.1.2 & 4.2.1.7).

Table 4.2.1.1, Correlations between conscientiousness (parent-report) and SDQ dimensions

	Pcns	Emot	Cond	Hype	Peer	Tsdq	Pssdq
Pcns	1.00						
Emot	-0.05	1.00					
Cond	-0.51**	0.34**	1.00				
Hype	-0.37**	0.26**	0.53**	1.00			
Peer	-0.09	0.25**	0.20**	0.06	1.00		
Tsdq	-0.37**	0.71**	0.75**	0.75**	0.48**	1.00	
Pssdq	0.69**	-0.11	-0.45**	-0.26**	-0.18**	-0.36**	1.00

* $p < 0.05$ ** $p < 0.01$ (after Bonferroni corrections)

Table 4.2.1.2, Correlations between conscientiousness (self-report) and SDQ dimensions

	Scns	Emot	Cond	Hype	Peer	Tsdq	Pssdq
Scns	1.00						
Emot	-0.10	1.00					
Cond	-0.26**	0.34**	1.00				
Hype	-0.27**	0.26**	0.53**	1.00			
Peer	-0.18**	0.25**	0.20**	0.06	1.00		
Tsdq	-0.30**	0.71**	0.75**	0.75**	0.48**	1.00	
Pssdq	0.34**	-0.11	-0.45**	-0.26**	-0.18**	-0.36**	1.00

- $p < 0.05$ ** $p < 0.01$ (after Bonferroni corrections)

Daring

Variation in daring was positively associated with adjustment in the adolescent group. Individuals who scored higher on self report daring had significantly higher scores for conduct problems and hyperactivity. However, variation in parent report daring showed a significant negative association with peer problems, and there was no relationship between

either measure of daring and the total difficulties subscale of the SDQ. There was no relationship between daring and adjustment in the adult group (see tables 4.2.1.3, 4.2.1.4 & 4.2.1.7).

Table 4.2.1.3, Correlations between daring (parent-report) and SDQ dimensions

	Pdar	Emot	Cond	Hype	Peer	Tsdq	Pssdq
Pdar	1.00						
Emot	-0.16**	1.00					
Cond	0.10	0.34**	1.00				
Hype	0.17**	0.26**	0.53**	1.00			
Peer	-0.18**	0.25**	0.20**	0.06	1.00		
Tsdq	-0.03	0.71**	0.75**	0.75**	0.48**	1.00	
Pssdq	0.17**	-0.11	-0.45**	-0.26**	-0.18**	-0.36**	1.00

* $p < 0.05$ ** $p < 0.01$ (after Bonferroni corrections)

Table 4.2.1.4, Correlations between daring (self-report) and SDQ dimensions

	Sdar	Emot	Cond	Hype	Peer	Tsdq	Pssdq
Sdar	1.00						
Emot	-0.09	1.00					
Cond	0.20**	0.34**	1.00				
Hype	0.21**	0.26**	0.53**	1.00			
Peer	-0.16**	0.25**	0.20**	0.06	1.00		
Tsdq	0.07	0.71**	0.75**	0.75**	0.48**	1.00	
Pssdq	-0.10	-0.11	-0.45**	-0.26**	-0.18**	-0.36**	1.00

* $p < 0.05$ ** $p < 0.01$ (after Bonferroni corrections)

Negative emotionality

Variation in negative emotionality was positively associated with adjustment in the adolescent group. Individuals who scored higher on parent report negative emotionality had significantly higher scores for emotion problems, conduct problems, hyperactivity, peer problems and the total difficulties subscale of the SDQ, and scored higher on the prosociality subscale of the SDQ. Individuals who scored higher on self report negative emotionality had significantly higher scores for emotion problems, conduct problems, hyperactivity and the total difficulties subscale of the SDQ. There was a significant positive association between negative emotionality and adjustment in the adult group. Individuals who scored higher on self report negative emotionality had higher total GHQ scores (tables 4.2.1.5, 4.2.1.6 & 4.2.1.7).

Table 4.2.1.5, Correlations between negative emotionality (parent-report) and SDQ dimensions

	Pne	Emot	Cond	Hype	Peer	Tsdq	Pssdq
Pne	1.00						
Emot	0.45**	1.00					
Cond	0.73**	0.34**	1.00				
Hype	0.54**	0.26**	0.53**	1.00			
Peer	0.18**	0.25**	0.20**	0.06	1.00		
Tsdq	0.71**	0.71**	0.75**	0.75**	0.48**	1.00	
Pssdq	-0.38**	-0.11	-0.45**	-0.26**	-0.18**	-0.36**	1.00

* p< 0.05 ** p < 0.01 (after Bonferroni corrections)

Table 4.2.1.6 Correlations between negative emotionality (self-report) and SDQ dimensions

	Sne	Emot	Cond	Hype	Peer	Tsdq	Pssdq
Sne	1.00						
Emot	0.22**	1.00					
Cond	0.24**	0.34**	1.00				
Hype	0.18**	0.26**	0.53**	1.00			
Peer	0.07	0.25**	0.20**	0.06	1.00		
Tsdq	0.27**	0.71**	0.75**	0.75**	0.48**	1.00	
Pssdq	-0.10	-0.11	-0.45**	-0.26**	-0.18**	-0.36**	1.00

* p< 0.05 ** p < 0.01 (after Bonferroni corrections)

Table 4.2.1.7 Correlations between personality and adjustment (adults)

	TGHQ	CONS	DAR	NE
TGHQ	1.00			
CONS	-0.11	1.00		
DAR	-0.12	-0.12	1.00	
NE	0.32**	-0.05	-0.12	1.00

* p< 0.05 ** p < 0.01 (after Bonferroni corrections)

4.2.2 PERSONALITY AND SCHOOL ATTAINMENT

Variation in conscientiousness and daring was positively assoociated with school attainment in the adolescent group. Individuals who scored higher for self report conscientiousness had significantly higher scores for maths. Individuals with higher scores for both measures of daring had significantly higher scores for Pe/games. Variation in negative emotionality was negatively associated with school attainment. Individuals who scored higher on parent report negative emotionality had significantly lower scores for reading, maths and science. Individuals who scored higher on self report negative emotionality had significantly lower scores for science and pe/games (see tables 4.2.2.1, 4.2.2.2, 4.2.2.3, 4.2.2.4 & 4.2.2.5).

4.2.3 ADJUSTMENT AND SCHOOL ATTAINMENT

Behavioural adjustment scores were negatively associated with school attainment. Individuals with higher scores on emotion problems had significantly lower scores for reading, spelling, maths, science and pe/games. Individuals with higher scores on conduct problems had significantly lower scores for maths. Individuals with higher scores for hyperactivity had significantly lower scores for reading, spelling, handwriting, maths and science. Individuals with higher scores on peer problems had significantly lower scores for pe/games. Individuals with higher scores on the total difficulties subscales had significantly lower scores for reading, spelling, handwriting, maths and science. However, there was a significant positive association between peer problems and computers. There was no relationship between school attainment and the prosociality subscale of the SDQ (see tables 4.2.3.1, 4.2.3.2, 4.2.3.3, 4.2.3.4, 4.2.3.5 & 4.2.3.6).

4.2.4 PERSONALITY, ADJUSTMENT AND DEVELOPMENTAL MARKERS

There were trends for associations as predicted between handedness and developmental markers with some of the personality and adjustment measures. However, these relationships did not remain significant following bonferroni corrections.

Table 4.2.2.1 Correlations between daring (parent-report) and school attainment

	Pdar	Read	Spll	Hwt	Maths	Art	Music	Comp	Scie	Pe
Pdar	1.00									
Read	-0.05	1.00								
Spll	-0.03	0.62**	1.00							
Hwt	-0.02	0.38**	0.47**	1.00						
Maths	-0.07	0.42**	0.43**	0.22**	1.00					
Art	-0.03	0.08	0.13	0.29**	0.09	1.00				
Music	0.09	0.14*	0.12	0.14*	0.12*	0.12*	1.00			
Comp	0.10	0.21**	0.20**	0.24**	0.16**	0.02	0.12*	1.00		
Scie	0.05	0.34**	0.29**	0.18**	0.48**	0.05	0.13*	0.31**	1.00	
Pe	0.45**	-0.03	0.03	0.14*	0.04	0.13*	0.12*	0.11*	0.12*	1.00

- $p <$ ** $p < 0.01$ (after Bonferroni corrections)

Table 4.2.2.2 Correlations between negative emotionality (parent-report) and school attainment

	Pneg	Read	Spll	Hwt	Maths	Art	Music	Comp	Scie	Pe
Pneg	1.00									
Read	-0.19**	1.00								
Spll	-0.14	0.62**	1.00							
Hwt	-0.14	0.38**	0.47**	1.00						
Maths	-0.20**	0.42**	0.43**	0.22**	1.00					
Art	-0.02	0.08	0.13	0.29**	0.09	1.00				
Music	-0.05	0.14*	0.12	0.14*	0.12*	0.12*	1.00			
Comp	-0.15	0.21**	0.20**	0.24**	0.16**	0.02	0.12*	1.00		
Scie	-0.25**	0.34**	0.29**	0.18**	0.48**	0.05	0.13*	0.31**	1.00	
Pe	-0.07	-0.03	0.03	0.14*	0.04	0.13*	0.12*	0.11*	0.12*	1.00

* $p <$ ** $p < 0.01$ (after Bonferroni corrections)

Table 4.2.2.3 Correlations between conscientiousness (self-report) and school attainment

	Scns	Read	Spll	Hwt	Maths	Art	Music	Comp	Scie	Pe
Scons	1.00									
Read	0.15	1.00								
Spll	0.08	0.62**	1.00							
Hwt	0.15	0.38**	0.47**	1.00						
Maths	0.20**	0.42**	0.43**	0.22**	1.00					
Art	0.08	0.08	0.13	0.29**	0.09	1.00				
Music	0.10	0.14	0.12	0.14	0.12	0.12	1.00			
Comp	-0.03	0.21**	0.20**	0.24**	0.16	0.02	0.12	1.00		
Scie	0.16	0.34**	0.29**	0.18	0.48**	0.05	0.13	0.31**	1.00	
Pe	0.02	-0.03	0.03	0.14	0.04	0.13	0.12	0.11	0.12	1.00

* p < ** p < 0.01 (after Bonferroni corrections)

Table 4.2.2.4 Correlations between daring (self-report) and school attainment

	Sdar	Read	Spll	Hwt	Maths	Art	Music	Comp	Scie	Pe
Sdar	1.00									
Read	-0.04	1.00								
Spll	-0.11	0.62**	1.00							
Hwt	-0.04	0.38**	0.47**	1.00						
Maths	-0.12	0.42**	0.43**	0.22**	1.00					
Art	-0.01	0.08	0.13	0.29**	0.09	1.00				
Music	0.09	0.14	0.12	0.14	0.12	0.12	1.00			
Comp	0.09	0.21**	0.20**	0.24**	0.16	0.02	0.12	1.00		
Scie	0.03	0.34**	0.29**	0.18	0.48**	0.05	0.13	0.31**	1.00	
Pe	0.36**	-0.03	0.03	0.14	0.04	0.13	0.12	0.11	0.12	1.00

* p < ** p < 0.01 (after Bonferroni corrections)

Table 4.2.2.5 Correlations between negative emotionality (self-report) and school attainment

	Sneg	Read	Spll	Hwt	Maths	Art	Music	Comp	Scie	Pe
Sneg	1.00									
Read	-0.02	1.00								
Spll	-0.05	0.62**	1.00							
Hwt	-0.08	0.38**	0.47**	1.00						
Maths	-0.11	0.42**	0.43**	0.22**	1.00					
Art	-0.09	0.08	0.13	0.29**	0.09	1.00				
Music	-0.01	0.14	0.12	0.14	0.12	0.12	1.00			
Comp	-0.13	0.21**	0.20**	0.24**	0.16	0.02	0.12	1.00		
Scie	-0.21**	0.34**	0.29**	0.18	0.48**	0.05	0.13	0.31**	1.00	
Pe	-0.21**	-0.03	0.03	0.14	0.04	0.13	0.12	0.11	0.12	1.00

* p < ** p < 0.01 (after Bonferroni corrections)

Table 4.2.3.1 Correlations between conduct problems and school attainment

	Cond	Read	Spll	Hwt	Maths	Art	Music	Comp	Scie	Pe
Cond	1.00									
Read	-0.08	1.00								
Spll	-0.08	0.62**	1.00							
Hwt	-0.15	0.38**	0.47**	1.00						
Maths	-0.18**	0.42**	0.43**	0.22**	1.00					
Art	-0.04	0.08	0.13	0.29**	0.09	1.00				
Music	0.03	0.14	0.12	0.14	0.12	0.12	1.00			
Comp	-0.14	0.21**	0.20**	0.24**	0.16	0.02	0.12	1.00		
Scie	-0.18	0.34**	0.29**	0.18	0.48**	0.05	0.13	0.31**	1.00	
Pe	-0.06	-0.03	0.03	0.14	0.04	0.13	0.12	0.11	0.12	1.00

* p < ** p < 0.01 (after Bonferroni corrections)

Table 4.2.3.2 Correlations between hyperactivity and school attainment

	Hype	Read	Spll	Hwt	Maths	Art	Music	Comp	Scie	Pe
Hype	1.00									
Read	-0.25**	1.00								
Spll	-0.23**	0.62**	1.00							
Hwt	-0.32**	0.38**	0.47**	1.00						
Maths	-0.26**	0.42**	0.43**	0.22**	1.00					
Art	-0.12	0.08	0.13	0.29**	0.09	1.00				
Music	-0.17	0.14	0.12	0.14	0.12	0.12	1.00			
Comp	-0.15	0.21**	0.20**	0.24**	0.16	0.02	0.12	1.00		
Scie	-0.31**	0.34**	0.29**	0.18	0.48**	0.05	0.13	0.31**	1.00	
Pe	0.02	-0.03	0.03	0.14	0.04	0.13	0.12	0.11	0.12	1.00

* p < ** p < 0.01 (after Bonferroni corrections)

Table 4.2.3.3 Correlations between peer problems and school attainment

	Peer	Read	Spll	Hwt	Maths	Art	Music	Comp	Scie	Pe
Peer	1.00									
Read	-0.04	1.00								
Spll	-0.04	0.62**	1.00							
Hwt	-0.05	0.38**	0.47**	1.00						
Maths	0.02	0.42**	0.43**	0.22**	1.00					
Art	-0.01	0.08	0.13	0.29**	0.09	1.00				
Music	-0.04	0.14	0.12	0.14	0.12	0.12	1.00			
Comp	0.27**	0.21**	0.20**	0.24**	0.16	0.02	0.12	1.00		
Scie	-0.04	0.34**	0.29**	0.18	0.48**	0.05	0.13	0.31**	1.00	
Pe	-0.19**	-0.03	0.03	0.14	0.04	0.13	0.12	0.11	0.12	1.00

* p < ** p < 0.01 (after Bonferroni corrections)

Table 4.2.3.4 Correlations between SDQ total and school attainment

	TSDQ	Read	Spll	Hwt	Maths	Art	Music	Comp	Scie	Pe
TSDQ	1.00									
Read	-0.20**	1.00								
Spll	-0.20**	0.62**	1.00							
Hwt	-0.25**	0.38**	0.47**	1.00						
Maths	-0.24**	0.42**	0.43**	0.22**	1.00					
Art	-0.04	0.08	0.13	0.29**	0.09	1.00				
Music	-0.11	0.14	0.12	0.14	0.12*	0.12	1.00			
Comp	-0.15	0.21**	0.20**	0.24**	0.16	0.02	0.12	1.00		
Scie	-0.28**	0.34**	0.29**	0.18	0.48**	0.05	0.13	0.31**	1.00	
Pe	-0.12	-0.03	0.03	0.14	0.04	0.13	0.12	0.11	0.12	1.00

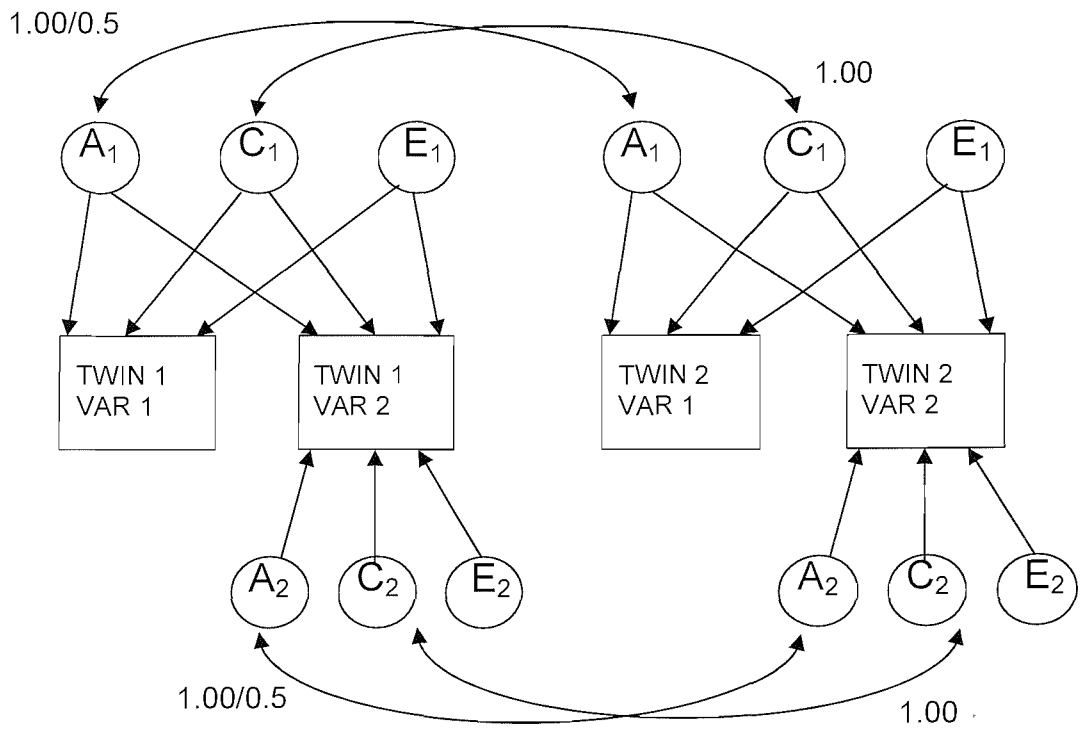
* p < ** p < 0.01 (after Bonferroni corrections)

CHAPTER FIVE
MULTIVARIATE ANALYSIS

To look at the relative extent genetic and environmental factors contributed to associations between constructs multivariate behaviour genetic analysis was carried out. This was for all relationships where correlations were significant and moderate, and also for any significant relationships between variables where there independent measures (e.g. self-report personality and parent-report adjustment).

The genetic and environmental architecture underlying the relation between variables was investigated using a multivariate analysis. The basic logic from the univariate analyses are extended to identify the origins of the pattern of relationships between these two variables (see figure 5.1).

Figure 5.1Cholesky decomposition



Genetic influences affecting the two variables are implicated when the MZ cross-correlation (the correlation between one twin's score on a variable with the other twin's score on a second variable) is greater than the DZ cross-correlation. Conversely, if the

cross-correlation is similar across MZ and DZ twins, there is evidence for common shared environmental effects

A Cholesky decomposition was used to model the genetic and environmental factors underlying the relationship between personality and psychopathology. A Cholesky decomposition is a triangular decomposition. In this model, the first set of genetic and environmental factors (A1,C1,E1) represents factors common to both variables. The second set of factors underlies only the psychopatholgy measure (A2, C2,E2).

The total portion of variability ascribed to genetic and non-shared environmental influences can be calculated by summing the squared path estimates across each row. The genetic correlation (r_g) and environmental correlations between each of the variables can be calculated as well. For example, the genetic correlation can be calculated with the following formula: $r_g = GCOV_{xy}/SQRT\ (V_{Gx}V_{Gy})$, where $GCOV_{xy}$ is the genetic covariance of X and Y and V_{Gx} and V_{Gy} is the genetic variance of X and Y, respectively.

5.1 PERSONALITY AND ADJUSTMENT

Conscientiousness (parent report) and conduct problems

Table 5.1.1 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. Since there was no evidence from the univariate analyses of a shared environment effect on these variable, only A and E terms were used. These totals correspond closely to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables.

The genetic factor common to the two variables shows substantial genetic loadings (.91,.67). There is a modest genetic factor effecting conduct problems alone (.42). This indicates that over two thirds (72%) of the genetic factors influencing conduct problems are shared with conscientiousness. The non-shared environmental factor on conscientiousness (.41) also had a small effect on conduct problems. However the major part of the non-shared environmental influence on conduct problems was specific to that variable (.58). The genetic correlation of conscientiousness and conduct problems is: .91 x .67/sqrt(.82 x .62) = .85. The non-shared environmental correlations is .41 x .21/sqrt (.17

x .38) = .33. The genetic correlation between these variables is substantially greater than the correlation for the non-shared environment.

Table 5.1.1 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Conscientiousness (parent)	.91		.82
Conduct problems	.67	.42	.62
	e1	e2	e^2
Conscientiousness (parent)	.41		.17
Conduct problems	.21	.58	.38

Table 5.1.2, Total number of twin pairs, correlations and standard deviations for conscientiousness (parent) and conduct problems

	sd	T1PCNS	T1COND	T2PCNS	T2COND
MZ (n = 42 pairs)					
TIPCNS	1.01	1.00			
T1COND	1.30	- 0.63*	1.00		
T2PCNS	1.06	0.81**	- 0.58**	1.00	
T2COND	1.03	-0.41**	0.72**	- 0.43**	1.00
DZ (n = 84 pairs)	Sd				
TIPCNS	0.90	1.00			
T1COND	0.82	-0.55**	1.00		
T2PCNS	0.96	0.29**	- 0.04	1.00	
T2COND	0.74	0.03	0.18	-0.37**	1.00

* P<0.05, **P<0.01

Conscientiousness (self report) and conduct problems

Table 5.1.3 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. Since there was no evidence from the univariate analyses of a shared environment effect on these variable, only A and E terms were used. These totals correspond closely to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables.

The genetic factor common to the two variables shows a substantial genetic loading for conscientiousness (.79). The negative genetic loading for conduct problems (-.34) implies that genetic influences which increase conscientiousness scores act to decrease scores for conduct problems. There is a substantial genetic factor effecting conduct problems alone (.76). The non-shared environmental factor on conscientiousness (.62) also had a small

effect on conduct problems. However the major part of the non-shared environmental influence on conduct problems was specific to that variable (.55).

The non-shared environmental correlation between the two variables is .62 x .06/sqrt(.38 x .31) = 0.11.

Table 5.1.3 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Conscientiousness (self)	. 79		.62
Conduct problems	-.34	.76	.69
	e1	e2	e^2
Conscientiousness (self)	.62		.38
Conduct problems	.06	.55	.31

Table 5.1.4, Total number of twin pairs, correlations and standard deviations for conscientiousness (self) and conduct problems

	sd	T1SCNS	T1COND	T2SCNS	T2COND
MZ (n = 41 pairs)					
TISCNS	1.06	1.00			
T1COND	1.37	- 0.35*	1.00		
T2SCNS	1.11	0.67**	- 0.02	1.00	
T2COND	1.08	-0.45**	0.74**	- 0.12	1.00
DZ (n = 81 pairs)	Sd				
TISCNS	0.92	1.00			
T1COND	0.82	-0.40**	1.00		
T2SCNS	0.96	0.20	- 0.22*	1.00	
T2COND	0.77	-0.11	0.18	-0.05	1.00

* P<0.05, **P<0.01

Conscientiousness (self report) and hyperactivity

Table 5.1.5 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. Since there was no evidence from the univariate analyses of a shared environment effect on these variable, only A and E terms were used. These totals correspond closely to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables.

The genetic factor common to the two variables shows a substantial genetic loading for conscientiousness (.77). The negative genetic loading for hyperactivity (-.30) implies that

genetic influences which increase conscientiousness scores act to decrease scores for hyperactivity. There is a substantial genetic factor effecting hyperactivity alone (.66). The non-shared environmental influences appear to be specific to each variable, with the negative loading for hyperactivity (-.04) suggesting non-shared environmental influences which increase conscientiousness scores, decrease scores for hyperactivity.

Table 5.1.5 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Conscientiousness (self)	. 77		.59
Hyperactivity	-.30	.66	.52
	e1	e2	e^2
Conscientiousness (self)	.64		.41
Hyperactivity	-.04	.68	.46

Table 5.1.6, Total number of twin pairs, correlations and standard deviations for conscientiousness (self) and hyperactivity

	Sd	T1SCNS	T1HYPE	T2SCNS	T2HYPE
MZ (n = 41 pairs)					
TISCNS	1.06	1.00			
T1HYPE	1.11	- 0.53**	1.00		
T2SCNS	1.11	0.67**	- 0.24	1.00	
T2HYPE	1.03	-0.35**	0.68**	- 0.11	1.00
DZ (n = 81 pairs)	Sd				
TISCNS	0.92	1.00			
T1HYPE	0.81	-0.25*	1.00		
T2SCNS	0.96	0.20	- 0.10	1.00	
T2HYPE	1.06	-0.03	-0.10	-0.21	1.00

* P<0.05, **P<0.01

Conscientiousness (self report) and peer problems

Table 5.1.7 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. Since there was no evidence from the univariate analyses of a shared environment effect on these variable, only A and E terms were used. These totals correspond closely to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables.

The genetic factor common to the two variables shows a substantial genetic loading for conscientiousness (.78). The negative genetic loading for peer problems (-.23) implies that genetic influences which increase conscientiousness scores act to decrease scores for peer

problems. There is a substantial genetic factor effecting peer problems alone (.68). The non-shared environmental influences appear to be specific to each variable, with the negative loading for peer problems (-.04) suggesting non-shared environmental influences which increase conscientiousness scores, decrease scores for peer problems.

Table 5.1.7 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Conscientiousness (self)	. 78		.61
Peer problems	-.23	.68	.51
	e1	e2	e^2
Conscientiousness (self)	.63		.40
Peer problems	-.04	.70	.49

Table 5.1.8, Total number of twin pairs, correlations and standard deviations for conscientiousness (self) and peer problems

	sd	T1SCNS	T1PEER	T2SCNS	T2PEER
MZ (n = 41 pairs)					
TISCNS	1.06	1.00			
T1PEER	1.09	- 0.19	1.00		
T2SCNS	1.11	0.67**	- 0.10	1.00	
T2PEER	0.93	- 0.20	0.60**	- 0.09	1.00
DZ (n = 80 pairs)	Sd				
TISCNS	0.92	1.00			
T1PEER	0.86	-0.23*	1.00		
T2SCNS	0.97	0.20	- 0.03	1.00	
T2PEER	1.05	-0.08	-0.11	-0.22	1.00

* P<0.05, **P<0.01

Conscientiousness (parent report) and total difficulties (SDQ)

Table 5.1.9 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. Since there was no evidence from the univariate analyses of a shared environment effect on these variable, only A and E terms were used. These totals correspond closely to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables.

The genetic factor common to the two variables shows a substantial genetic loading for conscientiousness (.88). The negative genetic loading for total difficulties (-.23) implies that genetic influences which increase conscientiousness scores act to decrease scores for

total difficulties. There is a substantial genetic factor effecting total difficulties alone (.82). The non-shared environmental influences appear to be specific to each variable, with the negative loading for total difficulties (-.16) suggesting non-shared environmental influences which increase conscientiousness scores, decrease scores for total difficulties.

Table 5.1.9 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Conscientiousness (parent)	.88		.77
Total difficulties(SDQ)	-.29	.82	.76
Conscientiousness (parent)	.48		.23
Total difficulties(SDQ)	-.16	.47	.25

Table 5.1.10, Total number of twin pairs, correlations and standard deviations for conscientiousness (parent) and total difficulties (SDQ)

	sd	T1PCNS	T1TSDQ	T2PCNS	T2TSDQ
MZ (n = 42 pairs)					
TIPCNS	1.01	1.00			
T1TSDQ	1.18	- 0.52**	1.00		
T2PCNS	1.06	0.81**	- 0.48**	1.00	
T2TSDQ	1.15	-0.29	0.83**	- 0.37**	1.00
DZ (n = 82 pairs)	Sd				
TIPCNS	0.90	1.00			
T1TSDQ	0.81	-0.24*	1.00		
T2PCNS	0.97	0.28	0.01	1.00	
T2TSDQ	0.86	0.02	0.23*	-0.26*	1.00

* P<0.05, **P<0.01

Conscientiousness (self report) and total difficulties (SDQ)

Table 5.1.11 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. Since there was no evidence from the univariate analyses of a shared environment effect on these variable, only A and E terms were used. These totals correspond closely to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables.

The genetic factor common to the two variables shows a substantial genetic loading for conscientiousness (.78). The negative genetic loading for total difficulties (-.38) implies that genetic influences which increase conscientiousness scores act to decrease scores for total difficulties. There is a substantial genetic factor effecting total difficulties alone (.83).

The non-shared environmental factor on conscientiousness (.62) had a negligible effect on total difficulties (.02) with the major part of non-shared environmental influence specific to this variable (.41). The non-shared environmental correlation is .62 x.02/sqrt(.38 x .17) = 0.05.

Table 5.1.11 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Conscientiousness (self)	.78		.61
Total difficulties(SDQ)	-.38	.83	.83
Conscientiousness (self)	.62		.38
Total difficulties(SDQ)	.02	.41	.17

Table 5.1.12, Total number of twin pairs, correlations and standard deviations for conscientiousness (self) and total difficulties(SDQ)

	sd	T1SCNS	T1TSDQ	T2SCNS	T2TSDQ
MZ (n = 41 pairs)					
TISCNS	1.06	1.00			
T1TSDQ	1.30	- 0.45**	1.00		
T2SCNS	1.11	0.67**	- 0.17	1.00	
T2TSDQ	1.17	-0.50**	0.84**	- 0.22	1.00
DZ (n = 79 pairs)	Sd				
TISCNS	0.91	1.00			
T1TSDQ	0.81	-0.36**	1.00		
T2SCNS	0.97	0.18	- 0.15	1.00	
T2TSDQ	0.84	-0.10	0.28*	-0.15	1.00

* P<0.05, **P<0.01

Conscientiousnes (parent report) and prosociality (SDQ)

Table 5.1.13 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. Since there was no evidence from the univariate analyses of a shared environment effect on these variable, only A and E terms were used. These totals correspond closely to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables.

The genetic factor common to the two variables shows substantial genetic loadings (.88,.62). There is a modest genetic factor effecting prosociality alone (.55). This indicates that over half (56%) of the genetic factors influencing prosociality are shared with conscientiousness. The non-shared environmental factor on conscientiousness (.48) also

had a small effect on prosociality. However the major part of the non-shared environmental influence on prosociality was specific to that variable (.47).

The genetic correlation of conscientiousness and prosociality is: .88 x .62/sqrt(.77 x .69) = .75. The non-shared environmental correlations is .48 x .29/sqrt (.23 x .30) = .53. The genetic correlation between the variables is greater than the correlation for the non-shared environment.

Table 5.1.13 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Conscientiousness (parent)	.88		.77
Prosocial (SDQ)	.62	.55	.69
	e1	e2	e^2
Conscientiousness (parent)	.48		.23
Prosocial (SDQ)	.29	.47	.30

Table 5.1.14 Total number of twin pairs, correlations and standard deviations for conscientiousness (parent) and prosocial (SDQ)

	sd	T1PCNS	T1PR	T2PCNS	T2PR
MZ (n = 42 pairs)					
TIPCNS	1.01	1.00			
T1 PR	1.12	0.79**	1.00		
T2PCNS	1.06	0.81**	0.67**	1.00	
T2 PR	1.00	0.55**	0.75**	0.69**	1.00
DZ (n = 84 pairs)	sd				
TIPCNS	0.90	1.00			
T1 PR	0.98	0.70**	1.00		
T2PCNS	0.96	0.29**	0.19	1.00	
T2 PR	0.88	0.24**	0.29**	0.62**	1.00

* P<0.05, **P<0.01

Conscientiousnes (self report) and prosociality (SDQ)

Table 5.1.15 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. Since there was no evidence from the univariate analyses of a shared environment effect on these variable, only A and E terms were used. These totals correspond closely to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables.

The genetic factor common to the two variables shows a substantial genetic loading for consicientiousness, and a moderate genetic loading for prosociality (.77,.39). There is a substantial genetic factor effecting prosociality alone (.75). This indicates that around one fifth of the genetic factors influencing prosociality are shared with conscientiousness. The non-shared environmental factor on conscientiousness (.64) also had a small effect on prosociality. However the major part of the non-shared environmental influence on prosociality was specific to that variable (.51).

The genetic correlation of conscientiousness and prosociality is: .77 x .39/sqrt(.59 x .71) = .46. The non-shared environmental correlations is .64 x .13/sqrt (.41 x .28) = .24. The genetic correlation between personality and psychopathology is substantially greater than the correlation for the non-shared environment.

Table 5.1.15 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Conscientiousness (self)	.77		.59
Prosocial (SDQ)	.39	.75	.71
	e1	e2	e^2
Conscientiousness (self)	.64		.41
Prosocial (SDQ)	.13	.51	.28

Table 5.1.16, Total number of twin pairs, correlations and standard deviations for conscientiousness (self) and prosocial (SDQ)

	sd	T1SCNS	T1PR	T2SCNS	T2PR
MZ (n = 41 pairs)					
TISCNS	1.01	1.00			
T1 PR	1.11	0.57**	1.00		
T2SCNS	1.11	0.67**	0.28	1.00	
T2 PR	1.07	0.43**	0.77**	0.27	1.00
DZ (n = 81 pairs)	sd				
TISCNS	0.92	1.00			
T1 PR	0.96	0.48**	1.00		
T2SCNS	0.96	0.20	0.04	1.00	
T2 PR	0.88	0.22*	0.34**	0.26*	1.00

* P<0.05, **P<0.01

Negative emotionality (parent report) and emotion problems

Table 5.1.17 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. Since there was no evidence from the univariate analyses

of a shared environment effect on these variable, only A and E terms were used. These totals correspond closely to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables.

The genetic factor common to the two variables shows a substantial genetic loading for negative emotionality, and a moderate genetic loading for emotion problems (.90,.51). There is a substantial genetic factor effecting psychopathology alone (.64). This indicates that over one third (39%) of the genetic factors influencing emotion problems are shared with negative emotionality. The non-shared environmental influences appear to be specific to each variable, with the negative loading for emotion problems (-.05) suggesting non-shared environmental influences which increase negative emotionality scores, decrease scores for emotion problems. The major part of non-shared environmental influence on emotion problems was specific to that variable (.57).

The genetic correlation of negative emotionality and emotion problems is: .90 x .51/sqrt(.81 x .67) = .62.

Table 5.1.17 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Negative	.90		.81
emotionality(parent)	.51	.64	.67
Emotion problems			
	e1	e2	e^2
Negative emotionality	.44		.19
(parent)	-.05	.57	.33
Emotion problems			

Table 5.1.18, Total number of twin pairs, correlations and standard deviations for negative emotionality(parent) and emotion problems

	sd	T1PNE	T1EMOT	T2PNE	T2EMOT
MZ (n = 43 pairs)					
TIPNE	1.16	1.00			
T1EMOT	0.97	0.58**	1.00		
T2PNE	1.22	0.84**	0.56**	1.00	
T2EMOT	1.09	0.55**	0.75**	0.58**	1.00
DZ (n = 84 pairs)	sd				
TIPNE	0.91	1.00			
T1EMOT	0.89	0.46**	1.00		
T2PNE	0.82	0.26*	0.21	1.00	
T2EMOT	1.14	0.30**	0.26*	0.29**	1.00

- P<0.05, **P<0.01

Negative emotionality (self report) and emotion problems

Table 5.1.19 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond closely to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables. Since there was evidence of a shared environmental effect for negative emotionality from the univariate analyses, A,C, and E terms were included for this variable.

The genetic factor common to the two variables shows a modest genetic loading for negative emotionality, and a substantial genetic loading for emotion problems (.36,.67). There is a moderate genetic factor effecting emotion problems alone (.50). This indicates that over two thirds (68%) of the genetic factors influencing emotion problems are shared with negative emotionality. The non-shared environmental influences appear to be specific to each variable, with the negative loading for emotion problems (-.04) suggesting non-shared environmental influences which increase negative emotionality scores, decrease scores for emotion problems. The major part of non-shared environmental influence on emotion problems was specific to that variable (.54).

The genetic correlation of negative emotionality and emotion problems is: .36 x .67/sqrt(.13 x .70) = .80.

Table 5.1.19 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Negative emotionality (self)	.36		.13
Emotion problems	.67	.50	.70
	c1		c^2
Negative emotionality (self)	.47		.22
Emotion problems	n/a		n/a
	e1	e2	e^2
Negative emotionality (self)	.80		.64
Emotion problems	-.04	.54	.29

Table 5.1.20, Total number of twin pairs, correlations and standard deviations for negative emotionality(self) and emotion problems

	sd	T1SNE	T1EMOT	T2SNE	T2EMOT
MZ (n = 40 pairs)					
TISNE	1.16	1.00			
T1EMOT	0.91	0.22	1.00		
T2SNE	1.07	0.40*	0.12	1.00	
T2EMOT	1.08	0.26	0.72**	0.17	1.00
DZ (n = 82 pairs)	Sd				
TISNE	0.98	1.00			
T1EMOT	0.90	0.26*	1.00		
T2SNE	0.88	0.29*	0.21	1.00	
T2EMOT	1.16	0.18	0.28*	0.22*	1.00

- P<0.05, **P<0.01

Negative emotionality (parent report) and conduct problems

Table 5.1.21 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond closely to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables. Since there was no evidence of a shared environment effect on these variables from the univariate analyses, only A, and E terms were used.

The genetic factor common to the two variables shows substantial genetic loadings (.91,.67). There is a modest genetic factor effecting emotion problems alone (.42). This indicates that over two thirds (72%) of the genetic factors influencing conduct problems are shared with negative emotionality. The non-shared environmental factor on negative emotionality (.41) also had a small effect on conduct problems. However, the major part of non-shared environmental influence on conduct problems was specific to that variable (.57).

The genetic correlation of negative emotionality and emotion problems is: ..91 x .67/sqrt(.83 x .62) = .85. The non-shared environmental correlation is .41 x 21/sqrt (.17x .37) = 0.34. The genetic correlation between personality and psychopathology is substantially greater than the correlation for the non-shared environment.

Table 5.1.21 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Negative	.91		.83
emotionality(parent)	.67	.42	.62
Conduct problems			
	e1	e2	e^2
Negative	.41		.17
emotionality(parent)	.21	.57	.37
Conduct problems			

Table 5.1.22, Total number of twin pairs, correlations and standard deviations for negative emotionality (parent) and conduct problems

	sd	T1PNE	T1 COND	T2PNE	T2 COND
MZ (n = 43 pairs)					
TIPNE	1.16	1.00			
T1COND	1.34	0.82**	1.00		
T2PNE	1.22	0.84**	0.71**	1.00	
T2COND	1.07	0.71**	0.74**	0.84**	1.00
DZ (n = 84 pairs)	sd				
TIPNE	0.91	1.00			
T1COND	0.82	0.57**	1.00		
T2PNE	0.82	0.26*	0.17	1.00	
T2COND	0.76	0.27*	0.17	0.69**	1.00

* P<0.05, **P<0.01

Negative emotionality (self report) and conduct problems

Table 5.1.23 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables. There was evidence of a shared environment effect on negative emotionality, however, a model including C terms for negative emotionality would not converge, only A, and E terms were used in the bivariate model.

The genetic factor common to the two variables shows a substantial genetic loading for negative emotionality, and a modest genetic loading for conduct problems (.66,.36). There is a substantial genetic factor effecting conduct problems alone (.74). This indicates that around one fifth (19%) of the genetic factors influencing conduct problems are shared with negative emotionality. The non-shared environmental factor on negative emotionality (.75) had a negligible effect on conduct problems (.02) with the major part of the non-shared environmental influence specific to this variable (.57).

The genetic correlation of negative emotionality and conduct problems is .66 x .36/sqrt(.43 x .68) = .44. The non-shared environmental correlation is .75 x .02/sqrt (.56 x .45) = 0.03. The genetic correlation between personality and psychopathology is substantially greater than the correlation for the non-shared environment.

Table 5.1.23 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Negative emotionality(self)	.66		.43
Conduct problems	.36	.74	.68
	e1	e2	e^2
Negative emotionality(self)	.75		.56
Conduct problems	.02	.57	.32

Table 5.1.24, Total number of twin pairs, correlations and standard deviations for negative emotionality(self) and conduct problems

	sd	T1SNE	T1 COND	T2SNE	T2 COND
MZ (n = 40 pairs)					
TISNE	1.16	1.00			
T1COND	1.32	0.31	1.00		
T2SNE	1.07	0.40**	0.13	1.00	
T2COND	1.05	0.43**	0.72**	0.31*	1.00
DZ (n = 82 pairs)	Sd				
TISNE	0.98	1.00			
T1COND	0.83	0.17	1.00		
T2SNE	0.88	0.29*	-0.08	1.00	
T2COND	0.77	0.28*	0.18	0.28*	1.00

Negative emotionality (parent-report) and hyperactivity

Table 5.1.25 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses ; the

multivariate model allowed for specification of the genetic and environmental links between the variables. Since there was no evidence of a shared environment effect on these variables from the univariate analyses, only A, and E terms were used.

The genetic factor common to the two variables shows a substantial genetic loading for negative emotionality, and a moderate genetic loading for hyperactivity (.91, .47). There is a moderate genetic factor effecting conduct problems alone (.43). This indicates that over half (55%) of the genetic factors influencing hyperactivity are shared with negative emotionality. The non-shared environment factor on personality (.40) also had a small effect on hyperactivity. However, the major part of the non-shared environmental influence on hyperactivity was specific to that variable (.75).

The genetic correlation of negative emotionality and hyperactivity is .91 x .47/sqrt(.83 x .40) = 0.74. The non-shared environmental correlation is .40 x .18/sqrt (.16 x .59) = 0.23. The genetic correlation between negative emotionality and hyperactivity is substantially greater than the correlation for the non-shared environment.

Table 5.1.25 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Negative	.91		.83
emotionality(parent)	.47	.43	.40
Hyperactivity			
	e1	e2	e^2
Negative	.40		.16
emotionality(parent)	.18	.75	.59
Hyperactivity			

Table 5.1.26, Total number of twin pairs, correlations and standard deviations for negative emotionality (parent) and hyperactivity

	sd	T1PNE	T1 HYPE	T2PNE	T2 HYPE
MZ (n = 43 pairs)					
TIPNE	1.16	1.00			
T1HYPE	1.10	0.72**	1.00		
T2PNE	1.22	0.84**	0.61**	1.00	
T2 HYPE	1.04	0.60**	0.69**	0.73**	1.00
DZ (n = 84 pairs)	sd				
TIPNE	0.91	1.00			
T1HYPE	0.81	0.59**	1.00		
T2PNE	0.82	0.26*	0.14	1.00	
T2 HYPE	1.05	0.20	- 0.32	0.18	1.00

* P<0.05, **P<0.01

Table 5.1.27 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables. There was evidence of a shared environment effect on negative emotionality, however, a model including C terms for negative emotionality would not converge, only A, and E terms were used in the bivariate model.

The genetic factor common to the two variables shows moderate genetic loadings (.66, .36). There is a more substantial genetic factor effecting hyperactivity alone (.43). This indicates around a fifth (23%) of genetic factors influencing hyperactivity are shared with negative emotionality. The non-shared environmental influences appear to be specific to each variable, with the negative loading for hyperactivity (-.03) suggesting non-shared environmental influences which increase negative emotionality scores, decrease scores for hyperactivity. The major part of non-shared environmental influence on hyperactivity was specific to that variable (.67).

The genetic correlation of negative emotionality and hyperactivity is .66 x .36/sqrt(.43 x .55) = 0.49.

Table 5.1.27 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Negative emotionality(self)	.66		.43
Hyperactivity	.36	.65	.55
	e1	e2	e^2
Negative emotionality(self)	.75		.56
Hyperactivity	-.03	.67	.45

Table 5.1.28, Total number of twin pairs, correlations and standard deviations for negative emotionality(self) and hyperactivity

	Sd	T1SNE	T1 HYPE	T2SNE	T2 HYPE
MZ (n = 40 pairs)					
TISNE	1.16	1.00			
T1HYPE	1.07	0.28	1.00		
T2SNE	1.07	0.40**	0.25	1.00	
T2 HYPE	1.04	0.43**	0.69**	0.43**	1.00
DZ (n = 82 pairs)	Sd				
TISNE	0.98	1.00			
T1HYPE	0.81	0.30**	1.00		
T2SNE	0.88	0.29*	0.10	1.00	
T2 HYPE	1.05	0.01	- 0.01	-0.02	1.00

* P<0.05, **P<0.01

Negative emotionality (parent) and peer problems

Table 5.1.29 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables. Since there was no evidence of a shared environment effect on these variables from the univariate analysis only A, and E terms were used.

The genetic factor common to the two variables shows a substantial genetic loading for negative emotionality, and a more modest genetic loading for peer problems (.90,.31). There is a more substantial genetic factor effecting peer problems alone (.60). This indicates that around a quarter (21%) of the genetic factors influencing peer problems are shared with negative emotionality. The non-shared environmental influences appear to be specific to each variable, with the negative loading for peer problems (-.26) suggesting non-shared environmental influences which increase negative emotionality scores, decrease scores for peer problems. The major part of non-shared environmental influence on peer problems was specific to that variable (.70).

The genetic correlation of negative emotionality and hyperactivity is .90 x .31/sqrt(.81 x .45) = 0.46.

Table 5.1.29 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Negative	.90		.81
emotionality(parent)	.31	.60	.45
Peer problems			
	e1	e2	e^2
Negative	.44		.19
emotionality(parent)	-.26	.70	.56
Peer problems			

Table 5.1.30, Total number of twin pairs, correlations and standard deviations for negative emotionality(parent) and peer problems

	sd	T1PNE	T1 PEER	T2PNE	T2 PEER
MZ (n = 43 pairs)					
TIPNE	1.16	1.00			
T1PEER	1.07	0.44**	1.00		
T2PNE	1.22	0.84**	0.52**	1.00	
T2 PEER	0.93	0.43**	0.61**	0.43**	1.00
DZ (n = 83 pairs)	sd				
TIPNE	0.91	1.00			
T1PEER	0.81	0.13	1.00		
T2PNE	0.82	0.26*	0.12	1.00	
T2 PEER	1.05	0.13	0.10	- 0.36	1.00

* P<0.05, **P<0.01

Negative emotionality (self) and peer problems

Table 5.1.31provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables. There was evidence of a shared environment effect on negative emotionality, however, a model including C terms for negative emotionality would not converge, only A, and E terms were used in the bivariate model.

The genetic factor common to the two variables shows a substantial genetic loading for negative emotionality, and a more modest genetic loading for peer problems (.66,.19). There is a more substantial genetic factor effecting peer problems alone (.68). This indicates that only a very small proportion of the genetic factors influencing peer problems are shared with negative emotionality. The non-shared environmental influences appear to be specific to each variable, with the negative loading for peer problems (-.06) suggesting

non-shared environmental influences which increase negative emotionality scores, decrease scores for peer problems. The major part of non-shared environmental influence on peer problems was specific to that variable (.71).

The genetic correlation of negative emotionality and hyperactivity is .66 x .19/sqrt(.43 x .50) = 0.27.

Table 5.1.31 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	A2	a^2
Negative emotionality(self)	.66		.43
Peer problems	.19	.68	.50
	e1	e2	e^2
Negative emotionality(self)	.75		.56
Peer problems	-.06	.71	.50

Table 5.1.32, Total number of twin pairs, correlations and standard deviations for negative emotionality(self) and peer problems

	sd	T1SNE	T1 PEER	T2SNE	T2 PEER
MZ (n = 40 pairs)					
TISNE	1.16	1.00			
T1PEER	1.06	0.07	1.00		
T2SNE	1.07	0.40**	0.41**	1.00	
T2 PEER	0.92	0.19	0.57**	0.48**	1.00
DZ (n = 81 pairs)	sd				
TISNE	0.98	1.00			
T1PEER	0.86	0.02	1.00		
T2SNE	0.84	0.27*	-0.05	1.00	
T2 PEER	1.12	-0.00	0.17	- 0.05	1.00

* P<0.05, **P<0.01

Negative emotionality (parent) and total difficulties score

Table 5.1.33 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables. Since there was no evidence of a shared environment effect on these variables from the univariate analysis only A, and E terms were used.

The genetic factor common to the two variables shows substantial genetic loadings for both variables (.85,.73). There is a more modest genetic factor effecting total difficulties alone (.40). This indicates that over three quarters (77%) of the genetic factors influencing peer problems are shared with negative emotionality. The non-shared environmental factor on negative emotionality (.53) also had a small effect on total difficulties. However, the major part of the non-shared environmental influence on total difficulties was specific to that variable (.54).

The genetic correlation of negative emotionality and hyperactivity is .85 x .73/sqrt(.72 x .69) = 0.88. The non-shared environmental correlation is .53 x .11/sqrt (.28 x .30) = .20. The genetic correlation between negative emotionality and total difficulties is substantially greater than the correlation for the non-shared environment.

Table 5.1.33 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Negative	.85		.72
emotionality(parent)	.73	.40	.69
Total difficulties score			
	e1	e2	e^2
Negative	.53		.28
emotionality(parent)	.11	.54	.30
Total difficulties score			

Table 5.1.34 Total number of twin pairs, correlations and standard deviations for negative emotionality (parent) and total SDQ

	sd	T1PNE	T1 SDQ	T2PNE	T2 SDQ
MZ (n = 43 pairs)					
TIPNE	1.16	1.00			
T1 SDQ	1.28	0.84**	1.00		
T2PNE	1.22	0.84**	0.78**	1.00	
T2 SDQ	1.18	0.75**	0.84**	0.86**	1.00
DZ (n = 82 pairs)	sd				
TIPNE	0.89	1.00			
T1 SDQ	0.81	0.69**	1.00		
T2PNE	0.83	0.25*	0.24*	1.00	
T2 SDQ	0.86	0.30*	0.22*	0.41**	1.00

- P<0.05, **P<0.01

Negative emotionality (self) and total difficulties

Table 5.1.35 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables. There was evidence of a shared environment effect on negative emotionality, however, a model including C terms for negative emotionality would not converge, only A, and E terms were used in the bivariate model.

The genetic factor common to the two variables shows moderate genetic loadings for both variables (.62,.46). There is a more substantial genetic factor effecting total difficulties alone (.73). This indicates that over one quarter (28%) of the genetic factors influencing total difficulties are shared with negative emotionality. The non-shared environment factors on negative emotionality (.78) and total difficulties (.50) are specific to each variable.

The genetic correlation of negative emotionality and total difficulties is .62 x .46/sqrt(.38 x .74) = 0.54. The non-shared environmental correlation is .78 x .00/sqrt(.61 x .25) = 0.00.

Table 5.1.35 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Negative emotionality(self)	.62		.38
Total difficulties score	.46	.73	.74
	e1	e2	e^2
Negative emotionality(self)	.78		.61
Total difficulties score	.00	.50	.25

Table 5.1.36, Total number of twin pairs, correlations and standard deviations for negative emotionality(self) and total SDQ

	sd	T1SNE	T1 SDQ	T2SNE	T2 SDQ
MZ (n = 40 pairs)					
TISNE	1.16	1.00			
T1 SDQ	1.20	0.30	1.00		
T2SNE	1.07	0.40**	0.30	1.00	
T2 SDQ	1.14	0.45**	0.83**	0.45**	1.00
DZ (n = 80 pairs)	sd				
TISNE	0.98	1.00			
T1 SDQ	0.81	0.29**	1.00		
T2SNE	0.89	0.28*	0.09	1.00	
T2 SDQ	0.86	0.12	0.26*	0.18	1.00

* P<0.05, **P<0.01

Negative emotionality (self) and total GHQ

Table 5.1.37 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables. Since there was no evidence of a shared environment effect from univariate analyses only A, and E terms were used.

The genetic factor common to the two variables shows a substantial genetic loading for negative emotionality, and a more modest genetic loading for total GHQ (.72,.23). There is a modest genetic factor effecting total GHQ alone (.22). This indicates that half of the genetic factors influencing total difficulties are shared with negative emotionality. The non-shared environment factors on negative emotionality (.70) had a small effect on total difficulties. However, the major part of the non-shared environment influences on total GHQ, are specific to that variable (.92).

The genetic correlation of negative emotionality and GHQ is .72 x .23/sqrt(.52 x .10) = 0.73. The non-shared environmental correlation is .70 x .20/sqrt(.49 x .89) = 0.21. The genetic correlation between negative emotionality and total GHQ is substantially greater than the correlation for non-shared environment.

Table 5.1.37 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Negative emotionality	.72		.52
Total GHQ	.23	.22	.10
	e1	e2	e^2
Negative emotionality	.70		.49
Total GHQ	.20	.92	.89

Table 5.1.38, Total number of twin pairs, correlations and standard deviations for negative emotionality and total GHQ

	Sd	T1SNE	T1 GHQ	T2SNE	T2 GHQ
MZ (n = 46 pairs)					
TISNE	1.09	1.00			
T1 GHQ	0.74	0.53**	1.00		
T2SNE	1.00	0.54**	0.28	1.00	
T2 GHQ	0.82	0.20	0.22	0.22	1.00
DZ (n = 58 pairs)	Sd				
TISNE	1.02	1.00			
T1 GHQ	1.02	0.24	1.00		
T2SNE	0.92	0.23	0.03	1.00	
T2 GHQ	1.12	0.01	- 0.06	0.31*	1.00

* P<0.05, **P<0.01

Negative emotionality (parent report) and prosociality(SDQ)

Table 5.1.39 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables. Since there was no evidence of a shared environment effect from univariate analyses for both variables only A, and E terms were used.

The genetic factor common to the two variables shows a substantial genetic loading for negative emotionality (.90) The negative genetic loading for prosociality (-.37) implies that genetic influences which increase negative emotionality scores act to decrease scores for prosociality. There is a substantial genetic factor effecting prosociality (.75). The non-shared environmental influences appear to be specific to each variable, with the negative loading for prosociality (-.17) suggesting non-shared environmental influences which increase negative emotionality scores, decrease scores for prosociality. The major part of non-shared environmental influence on peer problems was specific to that variable (.71).

Table 5.1.39 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Negative emotionality(parent)	.88		.77
Prosociality(SDQ)	-.37	.75	.70
	e1	e2	e^2
Negative emotionality(parent)	.48		.23
Prosociality(SDQ)	-.17	.51	.26

Table 5.1.40, Total number of twin pairs, correlations and standard deviations for negative emotionality (parent) and prosociality (SDQ)

	Sd	T1SNE	T1PRS	T2SNE	T2PRS
MZ (n = 43 pairs)					
TISNE	1.16	1.00			
T1PRS	1.15	-0.44**	1.00		
T2SNE	1.22	0.84**	-0.35*	1.00	
T2PRS	1.05	-0.51**	0.77**	-0.53**	1.00
DZ (n = 84 pairs)	Sd				
TISNE	0.91	1.00			
T1PRS	0.98	-0.39**	1.00		
T2SNE	0.82	0.26*	-0.06	1.00	
T2PRS	0.89	-0.04	0.28*	-0.32**	1.00

* P<0.05, **P<0.01

Daring (self report) and conduct problems

Table 5.1.41 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses; the multivariate model allowed for specification of the genetic and environmental links between the variables. There was evidence of a shared environment effect from univariate analyses for daring, so a C term for this variable was included in the model.

The genetic factor common to the two variables shows moderate genetic loadings (.40,.45). There is a substantial genetic factor effecting conduct problems alone (.71). This indicates that over one quarter (28%) of the genetic factors influencing conduct problems are shared with daring. The non-shared environment factors were specific to both variables.

The genetic correlation of daring and conduct problems is .40 x .45/sqrt(.16 x .71) = 0.53.

Table 5.1.41 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Daring(self)	.40		.16
Conduct problems	.45	.71	.71
	c1	c2	c^2
Daring(self)	.39		.15
Conduct problems	n/a	n/a	n/a
	e1	e2	e^2
Daring(self)	.80		.69
Conduct problems	.00	.54	.29

Table 5.1.42, Total number of twin pairs, correlations and standard deviations for daring(self) and conduct problems

	sd	T1SDR	T1COND	T2SDR	T2COND
MZ (n = 41 pairs)					
TISDR	0.92	1.00			
T1COND	1.37	-0.04	1.00		
T2SDR	0.92	0.32*	0.33*	1.00	
T2COND	1.08	0.06	0.74**	0.50**	1.00
DZ (n = 80 pairs)	sd				
TISDR	0.95	1.00			
T1COND	0.82	0.12	1.00		
T2SDR	0.97	0.24*	0.07	1.00	
T2COND	0.77	0.14	0.20	0.20	1.00

* P<0.05, **P<0.01

Daring (self report) and hyperactivity

Table 5.1.43 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables. There was evidence of a shared environment effect from univariate analyses for daring, so a C term for this variable was included in the model.

The genetic factor common to the two variables shows moderate genetic loadings (.41,.63). There is a modest genetic factor effecting hyperactivity alone (.36). This indicates that three quarters (75%) of the genetic factors influencing hyperactivity are shared with daring. The non-shared environmental influences appear to be specific to each variable, with the negative loading for hyperactivity (-.06) suggesting non-shared environmental influences which increase daring scores, decrease scores for hyperactivity. The major part of non-shared environmental influence on hyperactivity was specific to that variable (.68).

The genetic correlation of daring and hyperactivity is .41 x .63/sqrt(.17 x .53) = 0.86. There is no correlation between non-shared environmental correlation influences.

Table 5.1.43 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Daring(self)	.41		.17
Hyperactivity	.63	.36	.53
	c1	c2	c^2
Daring(self)	.37		.14
Hyperactivity	n/a	n/a	n/a
	e1	e2	e^2
Daring(self)	.83		.69
Hyperactivity	-.06	.68	.46

Table 5.1.44, Total number of twin pairs, correlations and standard deviations for daring(self) and hyperactivity

	Sd	T1SDR	T1HYPE	T2SDR	T2HYPE
MZ (n = 41 pairs)					
TISDR	0.92	1.00			
T1HYPE	1.11	0.08	1.00		
T2SDR	0.92	0.32*	0.34*	1.00	
T2HYPE	1.03	0.13	0.68**	0.48**	1.00
DZ (n = 80 pairs)	Sd				
TISDR	0.95	1.00			
T1HYPE	0.82	0.23*	1.00		
T2SDR	0.97	0.24*	0.23*	1.00	
T2HYPE	1.05	0.12	0.01	0.12	1.00

* P<0.05, **P<0.01

Table 5.1.45 summarises the fit for the bivariate models of personality and adjustment.

Table 5.1.45, Fit for the bivariate models of personality and adjustment

	χ^2	Df	p	CFI	AIC
PCNXCOND*	26.56	14	.02	.95	- 1.44
SCNXCOND*	22.93	14	.06	.89	- 5.06
SCNXHYPE*	21.54	14	.10	.89	- 6.84
SCNXPEER	11.28	14	.66	1.0	-16.72
PCNXTSDQ	18.44	14	.19	0.96	- 9.56
SCNXSDQ*	14.21	14	.43	1.0	-13.78
PCNXPR	13.03	14	.52	1.0	-14.97
SCNXPR	15.18	14	.37	.99	-12.82
PNEXEMOT*	15.79	14	.33	.99	-12.21
SNEXEMOT	16.52	13	.22	93	- 9.48
PNEXCOND*	26.56	14	.02	.95	- 1.44
SNEXCOND*	13.97	14	.45	1.0	-14.03
PNEXHYPE*	50.39	14	.00	.78	22.39
SNEXHYPE*	20.47	14	.12	.86	- 7.53
PNEXPR*	15.29	14	.04	.98	-12.71
SNEXPR*	12.89	14	.53	1.0	-15.11
PNEXTSDQ	41.84	14	.00	.89	13.83
SNEXTSDQ	20.38	14	.12	.92	- 7.62
SNEXTGHQ	20.66	14	.11	.81	-7.34
SNEXPRS	15.32	14	.36	.99	-12.68
SDXCOND*	13.00	13	.45	1.0	-12.99
SDXHYPE*	17.98	13	.16	.88	- 8.02

* indicates model fitted to correlation matrice (for path values for models fitted to covariance matrice see Appendix J)

5.2 PERSONALITY AND SCHOOL ATTAINMENT

Conscientiousness (self-report) and maths

Table 5.2.1 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables. There was some evidence from the univariate analysis of a shared environmental effect for maths, however a model including this term would not converge and only A, and E terms were used.

The genetic factor common to the two variables shows a substantial genetic loading for conscientiousness and a modest genetic loading for maths (.79,.16). There is a more substantial genetic factor effecting maths alone (.88). This indicates that around 3% of the genetic factors influencing handwriting are shared with conscientiousness. The non-shared environment factors on conscientiousness (.61) had a small effect on maths. However, the

major part of the non-shared environmental influence on maths was specific to that variable (.44).

The genetic correlation of conscientiousness and reading is .79 x .16/sqrt(.62 x .80) = 0.18. The non-shared environmental correlation is .61 x .09/sqrt(.37 x .20) = 0.20.

Table 5.2.1 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Conscientiousness (self)	.79		.62
Maths	.16	.88	.80
	e1	e2	e^2
Conscientiousness (self)	.61		.37
Maths	.09	.44	.20

Table 5.2.2, Total number of twin pairs, correlations and standard deviations for conscientiousness (self) and maths

	Sd	T1SCNS	T1MATH	T2SCNS	T2MATH
MZ (n = 40 pairs)					
TISCNS	1.05	1.00			
T1MATH	1.11	0.46**	1.00		
T2SCNS	1.10	0.69**	0.30	1.00	
T2MATH	0.97	0.44**	0.82**	0.37*	1.00
DZ (n = 80 pairs)	Sd				
TISCNS	0.92	1.00			
T1MATH	0.97	0.07	1.00		
T2SCNS	0.96	0.22*	0.03	1.00	
T2MATH	0.89	- 0.18	0.49**	0.06	1.00

- P<0.05, **P<0.01

Daring (parent-report) and pe/games

Table 5.2.3 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables. There was some evidence from the univariate analysis of a shared environmental effect for both variables, however a model including C terms would not converge and only A, and E terms were used.

The genetic factor common to the two variables shows moderate genetic loadings for both variables (.58,.62). There is a more substantial genetic factor effecting maths alone (.66).

This indicates that around half (47%) of the genetic factors influencing pe/games are shared with daring. The non-shared environment factors on daring (.81) also had a small effect on pe/games (.16). However, the major part of non-shared environment influence was specific to that variable (.38).

The genetic correlation of conscientiousness and reading is .58 x .62/sqrt(.34 x .82) = 0.68. The non-shared environmental correlation is .81 x .16/sqrt(.65 x .17) = 0.39.

Table 5.2.3 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Daring (parent)	.58		.34
Pe/games	.62	.66	.82
	e1	e2	e^2
Daring (parent)	.81		.65
Pe/games	.16	.38	.17

Table 5.2.4, Total number of twin pairs, correlations and standard deviations for daring (parent) and pe/games

	sd	T1PDR	T1PE	T2PDR	T2PE
MZ (n = 42 pairs)					
TIPDR	0.91	1.00			
T1 PE	1.05	0.55**	1.00		
T2PDR	0.88	0.26	0.42**	1.00	
T2 PE	1.05	0.37*	0.85**	0.54**	1.00
DZ (n = 84 pairs)	sd				
TIPDR	0.95	1.00			
T1 PE	0.96	0.46**	1.00		
T2PDR	1.07	0.28**	0.22*	1.00	
T2 PE	0.90	0.29**	0.42**	0.58**	1.00

- P<0.05, **P<0.01

Daring (self-report) and pe/games

Table 5.2.5 provides standardized path estimates and total genetic and environmental effects for the Cholesky model. These totals correspond to the univariate analyses ; the multivariate model allowed for specification of the genetic and environmental links between the variables. There was some evidence from the univariate analysis of a shared environmental effect for both variables, however a model including C terms would not converge and only A, and E terms were used.

The genetic factor common to the two variables shows moderate genetic loadings for both variables (.56,.46). There is a more substantial genetic factor effecting pe/games alone (.78). This indicates that around one quarter (26%) of the genetic factors influencing pe/games are shared with daring. The non-shared environment factors on daring (.82) also had a small effect on pe/games (.15) However, the major part of non-shared environment influence was specific to that variable (.40).

The genetic correlation of daring and pe/games is .56 x .46/sqrt(.31 x .82) = 0.51. The non-shared environmental correlation is .82 x .15/sqrt(.67 x .18) = 0.35.

Table 5.2.5 Parameter estimates for the bivariate Cholskey decomposition

			Total estimate
	a1	a2	a^2
Daring (self)	.56		.31
Pe/games	.46	.78	.82
	e1	e2	e^2
Daring (self)	.82		.67
Pe/games	.15	.40	.18

Table 5.2.6, Total number of twin pairs, correlations and standard deviations for daring (self) and pe/games

	sd	T1SDR	T1PE	T2SDR	T2PE
MZ (n = 40 pairs)					
TISDR	0.90	1.00			
T1 PE	1.06	0.37**	1.00		
T2SDR	0.95	0.25	0.17	1.00	
T2 PE	1.06	0.33**	0.84**	0.39*	1.00
DZ (n = 79 pairs)	sd				
TISDR	0.96	1.00			
T1 PE	0.95	0.37**	1.00		
T2SDR	0.98	0.24*	0.10	1.00	
T2 PE	0.90	0.27*	0.42**	0.41*	1.00

* P<0.05, **P<0.01

Table 5.2.7 shows the fit indices for bivariate models of personality and school attainment.

Table 5.2.7 Fit for the bivariate models of personality and school attainment

	χ^2	Df	p	CFI	AIC
SCNSXMTHS	16.81	14	.27	.97	-11.19
PDRXPE	8.92	14	.84	1.0	-19.07
SDRXPE	7.26	14	.92	1.0	-20.74

* indicates model fitted to correlation matrice (for path values for models fitted to covariance matrice see Appendix J).

CHAPTER SIX
DISCUSSION

6.1 SUMMARY OF FINDINGS

Analysis of the factor structure of parent and self report items from the Child and Adolescent Temperament Scale(CATS) items confirmed three personality dimensions (conscientiousness, daring, negative emotionality). Significant genetic influences were found for all three CATS personality dimensions, and the patterns of genetic and environmental influences were similar across parent and self report. In addition, significant genetic influences were found for adjustment, school attainment and development markers.

Variation in personality was associated with adjustment and phenotype correlations are largely consistent with predictions. There were significant negative correlations between parent report conscientiousness and conduct problems, hyperactivity, and total difficulties scores (-0.37 to -0.51). There were significant negative correlations between self report conscientiousness and conduct problems, hyperactivity, peer problems and total difficulties scores (-0.18 to -0.30). There were significant positive correlations between prosocial behaviour and both parent and self report conscientiousness (0.69, and 0.34 respectively). There were significant positive correlations between self report daring and conduct problems and hyperactivity (0.20, and 0.21 respectively) and also prosocial behaviour (0.17). There were significant positive correlations between parent report negative emotionality and emotion problems, conduct problems, hyperactivity, peer problems, and total difficulties scores (0.18 to 0.71). There were significant positive correlations between self report negative emotionality and emotion problems, conduct problems, hyperactivity scores, and total difficulties scores (0.22 to 0.27). Prosocial behaviour was significantly negatively associated with parent report negative emotionality (-0.38). For the adult group negative emotionality was significantly negatively associated with total GHQ scores (-0.32).

Variation in personality was associated with school attainment. There were significant positive correlations between parent and self report conscientiousness scores and maths (0.20). There were significant positive associations between both parent and self report daring and pe/games (0.45 and 0.36 respectively). Parent report scores for negative

emotionality were significantly negatively correlated with reading, maths and science (-0.19 to -0.25). Self report scores for negative emotionality were significantly negatively correlated with science and pe/games (-0.21 for both).

Variation in adjustment was associated with school attainment. All significant phenotype correlations were negative as predicted. Conduct problem scores were significantly associated with maths scores. Hyperactivity scores were significantly associated with reading, spelling, handwriting, maths and science scores. Peer problem scores were significantly associated with computer and Pe scores. Total SDQ scores were significantly associated with reading, spelling, handwriting, maths and science scores. There was some evidence of a trend to association between indices of early development and the other variables, but results are not significant.

Phenotype correlations between personality and adjustment were largely due to genetic factors. The bivariate heritabilities (the proportion of the phenotype correlation due to genetic influences) are moderate to high . The genetic correlations (a measure of the extent to which two traits are influenced by the same set of genes) range from moderate to high, indicating genetic effects on personality and adjustment overlap, with higher correlations indicating that these variables may be influenced by a common set of genes.

For personality and school attainment, bivariate heritabilities are low. The genetic correlations are modest, but do suggest some overlap between genetic effects on personality and school attainment. However, for both parent and self report daring and pe/games phenotype correlations were high, and largely due to genetic influences. The bivariate heritabilities were high, also the genetic correlations indicating that these variables may be influenced by a common set of genes.

6.2 COMPARISON OF FINDINGS TO STUDIES BY LAHEY, ET AL (IN PRESS)

Factor analysis of CATS dimensions replicates findings in the two American population samples. Lahey et al (in press) found a strong genetic contribution to the three personality dimensions in a study of adult twins reared apart. This pattern was replicated for conscientiousness and negative emotionality. However, heritability for daring in the

current study was low (ranging from .01 to .13) and there was more evidence of common environmental influences (ranging from .18 to .44).

The associations between personality and adjustment are largely consistent with the studies by Lahey et al (in press). They found that high negative emotionality, low conscientiousness, and high daring ('conduct disorder' personality profile) were associated with increased presence of adjustment problems, whereas low negative emotionality, high conscientiousness, and low daring, together with strong cognitive verbal skills ('antitype') were associated with absence of adjustment problems. Therefore, this suggests similar patterns of association between personality and adjustment for individuals of different nationality, and for both population and volunteer samples, even when item overlap is controlled for. In addition, the finding of a relationship between negative emotionality and adjustment has been shown for a wider age range (4 to 25 years) than in the American studies (4 – 17 years). It is not clear if the lack of association between the other two personality dimensions and adjustment in adults is due to age differences, or to the fact that GHQ measures reflect mainly internalising symptoms.

6.3 COMPARISON OF FINDINGS TO OTHER STUDIES

Personality

For negative emotionality and conscientiousness, heritability ranged from .13 to .84, and .21 to .77 respectively, and were similar to findings from studies of heritability of around .50 for trait extraversion and neuroticism (e.g. Loehlin, 1992; Bouchard & McGue, 2003). Patterns of heritability have been similar across temperament measures in early childhood and personality measures in adulthood (see section 1.4.4). However, estimates for the current study are based on individuals aged from early childhood to adulthood who all completed the same temperament scale, and therefore allow direct comparison.

The common environment influence on daring is not typical of findings of negligible shared environment influences on personality. For example, there is no evidence of common environment influences on neuroticism or extraversion (see review by Bouchard & McGue, 2003) and evidence of common environment effects ($c^2 = .21$) on agreeableness found by Bergman, Chippeur, et al, 1993 has not been replicated. This finding may just

reflect variation in sample estimates, however patterns were consistent across parent and self report for adolescents, and self report for adults suggesting this may not be the case.

Previous studies have shown common environmental influences on trait psychoticism (which shows some overlap with aspects of daring). However, this may also be due in part to the nature of the current volunteer sample. This sample has relatively higher than average socioeconomic status, a factor associated with higher scores for positive child rearing environments (see Rowe, 1994). Therefore, it may be that there is an interaction between family environment and phenotypic expression of daring, with a more positive child rearing environment in some way modifying expression of genetic influences on daring. For example, a child may learn to inhibit impulsive behaviour within a well-structured environment.

There is also evidence of environmental mediation of genetic influences on personality in a study by Boomsma, de Geus, van Baal, Koopmans (1999). They found an interaction between religious background and genetic influences for the disinhibition scale of sensation seeking in a sample of Dutch twins. Individuals from a religious background showed less genetic influence on disinhibition than individuals who were not from a religious background. Heritability for disinhibition for males from a religious background was zero, and was lower for females with a religious background than for others. As variation in religious background was entirely accounted for by common environmental influences in this sample, this suggests that common environmental influences may act to alter the expression of genetic factors on aspects of sensation seeking. However, this could be a direct effect of common environmental influences, or an indirect effect through gene x environment correlations.

This may be important as most theories regarding the relationship between personality and adjustment suggest aspects of personality reflecting constraint contribute to the development of behavioural regulation, and Kochanska (1997) hypothesises an interaction between parental behaviour and child characteristics in the development of self-regulation, and in support of this Kochanska, Murray & Harlan (2000) found an interaction between parental socialisation, child temperament and rule internalisation in pre-school age children.

However, restricted variation in family environment should act to reduce estimates of effects of common environment, and generally studies using genetic methodologies have consistently found that common environment, and in particular child-rearing behaviours are not important sources of influence on child outcome measures (Rowe, 1994, p 161). These findings are based on population samples, and may not reflect family influences on extremely disturbed children, raising issues regarding the threshold of liability whereby family influences may influence child outcomes more substantially. In addition, family environments may differentially impact on children in a family, and therefore contribute to estimates of unique environmental influences (see Rowe, 1994).

Genetic effects on environmental influences

There is also evidence of genetic effects on environmental influences which may explain the evidence of common environment factors in the current studies despite low variation in socioeconomic status. For example, twin and adoption studies show evidence of genetic influences on a wide range of different measures, including child rearing behaviours, and in particular parental personality characteristics have been related to child-rearing behaviour (see Plomin & Bergemann, 1991; Rowe, 1994).

This is important as even if variation in environmental influences is genetically influenced, it does not rule out the possibility that associations between environmental influences and behavioural outcomes are environmentally mediated. For example, convergent evidence from adoption and twin studies suggests that the association between parental IQ and child socioeconomic status is produced both through common genetic factors, and indirectly through genetic influences on family environment (see Rowe, 1994).

Adjustment

The mean scores for the SDQ problem scales are typical for parent reported adjustment and are well within the range for normal scores (e.g. Goodman, 1999). Genetic and environmental influences for adjustment in the adolescent group are largely consistent with findings from population studies using SDQ dimensions, and are consistent with findings from clinical populations. For example, heritabilities for emotion problems, conduct problems, hyperactivity, peer problems and total difficulties scores are .72,.71,.58,.53,.83

respectively. Typical heritability estimates are around .20 to .50 for internalising dimensions, and .20 to .65, and .70 to .90, for conduct problems and hyperactivity respectively (e.g Rutter, et al, 1999: Kendler, 2001) However, there was no evidence of common environmental influences on externalising problems, which has been found in other studies, with estimates for common environment for conduct problems typically around .25 (see Rutter, et al, 1999). Notably, the heritability estimates for total GHQ scores (.14) are much lower than those typically reported for internalising symptoms, but it is not clear why this occurred.

School attainment

Scores for the school attainment variables were very consistent with findings using the same measures from the Child Health and Development Study (Stevenson & Pit-ten Cate, 2000) The heritability estimate for spelling was .58 which is consistent with findings by Stevenson (1993) and generally heritability estimates for reading, spelling, handwriting, maths, music, art and pe/games (.57 to .75) are very consistent with the literature (e.g. Plomin, et al, 1997) and are typical of findings for general cognitive ability (e.g. Alarcon, Knopik, DeFries, 2000; Bouchard & McGue. 2003). Variance in computers and science scores was almost entirely accounted for by common and unique environmental factors. It should be noted that the estimates in the current study are based on a wide age range.

Developmental markers

The finding of no relationship between handedness and zygosity, and no evidence of significant concordance for non-right handedness in either MZ or DZ twins is consistent with the study by Geschwind, et al (2002). The univariate models for handedness (right handed only) are consistent with previous family, adoption and twin studies of increasing resemblance for handedness across increasing genetic relatedness (e.g. McManus, 2003). The finding of no common environment influences for handedness is consistent with previous studies (e.g. Annett, 2000), but not with findings by Bishop, et al (2001) of no genetic influences on handedness in children with and without specific language impairment.

There appear to be no other twin studies to date for digit ratio, however, estimates of genetic, common and unique environmental influences are consistent with theoretical considerations, suggesting that genetic influences on susceptibility to prenatal testosterone levels combined with maternal environment contribute to variation in cerebral lateralisation (e.g. Geschwind & Galaburda, 1985; Manning, et al, 2000). The same univariate models were obtained for measures of 2d4d ratio, and distal extent ratios and estimates of heritability were identical (.39). The univariate models for both measures of digit ratio differences (the difference between the ratios for the left and the right hand) are consistent with findings of significant heritability for measures of fluctuating asymmetry in a review of 34 studies based on animals and humans (see Thornhill & Moller, 1997).

6.4 ASSOCIATIONS BETWEEN VARIABLES

For personality and adjustment findings are comparable to those from other studies where high trait emotionality (which resembles negative emotionality) and low trait constraint (which resembles low conscientiousness and high daring) have been associated with adjustment in both clinical and population samples. Similar relationships have been found between child report temperament and parent and teacher report symptoms of ADHD, depression (Bussing, Gary, Mason, Leon, Sinha, Garvan, 2003).

In addition, findings for the current study are based on measures of the same temperament scale over a wide age range, extending findings of similar patterns of association between children and older adolescents using different measures. The associations between personality and school attainment and between adjustment and school attainment are consistent with findings from studies which have looked at variation in general and more specific cognitive abilities (see section 1.4.5.4).

Evidence of common genetic effects between personality and adjustment, and between personality and school attainment, are consistent with findings from molecular genetic studies (e.g. Ebstein, et al, 2000; van Baal, et al, 2001).

6.5 THEORETICAL IMPLICATIONS

Findings of common genetic influences contributing to the relationship between personality and adjustment are consistent with Graham & Stevenson's hypothesis that some psychopathology can be viewed as extremes of variation in temperament. For example, they suggested low sociability would increase risk for later antisocial behaviour, high emotionality would increase risk for later internalising problems, and high activity would increase risk for later hyperactivity. In the current study there were substantial genetic correlations between conscientiousness (similar to sociability) and conduct problems (-0.85) between negative emotionality (similar to emotionality) and emotion problems (.62) and between daring (similar to high activity) and hyperactivity (.86) indicating considerable overlap between genetic factors influencing these relationships.

6.6 METHODOLOGICAL ISSUES/LIMITATIONS

Comparison of parent and self report personality dimensions

Previous studies have shown high agreement between parent and child temperament ratings assessed by The Dimensions of Temperament Scale (a measure based on original measures used by Thomas & Chess) (Luby & Steiner, 1993). Means for parent and self report personality scores were not compared as the two versions of the CATS inventory contained slightly different items. However, the moderate correlations between parent and self report personality suggest that while there was agreement, to some extent informants may have reported on different aspects of personality.

The correlations across informants in this study (.44,.50,.32 respectively for conscientiousness, daring and negative emotionality) are comparable to other studies of different informant personality ratings. For example, Angleneitner, Riemann, Strelau (1995) compared personality scores for 1000 adult twins from two peers, and from each twin. Correlations between peer ratings were .63, and the correlation between averaged peer ratings and self report ratings of each twin were .55. Genetic influences on peer ratings were similar to genetic influences on self report, and multivariate genetic analyses showed common genetic influences across peer and self report scores.

Similarly, van der Valk, et al (2001) looked at processes underlying parental agreement in behavioural ratings of three year old twins finding common genetic and common environmental influences contributed to 75% of the common variance in behaviour, with around 8% of the variance in behaviour due to unique genetic and environmental influences.

The proportion of variance in personality accounted for by this measure was greater for parent than self report (29.15% and 21.04% respectively) suggesting that parents may have been better at reporting variance in personality.

Structural Equation models

On a number of occasions inequalities in variances gave difficulties in analysing covariance matrices, this required analysis of correlation matrices (where it was possible to do both path values were similar) however it is more desirable to fit to variance/covariance matrices.

Twin samples

In the current study there is no evidence that twins differ from singletons in their scores for personality, adjustment, school attainment, or indices of early development, as no significant mean differences in scores for any of the variables were found between twins and their siblings, and between MZ and DZ twins. This suggests that findings from this study are generalisable to non-twin samples. Findings were similar for univariate models based on twin data and for the entire adolescent sample (twins and siblings) except for less evidence of common environmental effects.

Cross-sectional data

In the current study personality and adjustment were assessed at the same time point and therefore temperament cannot be shown to predict adjustment problems. However, temperament remains relatively stable over time, and previous studies have shown that early temperament predicts later adjustment (e.g. Gjone & Stevenson, 1997).

Response rates

The mean age for non-respondents was slightly lower than for the participants however there was no difference between these groups in terms of zygosity or gender. As any effects of age were controlled for it seems unlikely that there was an effect of non-response on the pattern of results obtained. However, the low response rate had implications. The small sample size available, precluded systematic examination of any age-related change on relationships between variables. It was also not possible to carry out several analyses, including looking at variation in the digit ratio measures separately for males and females, and univariate analysis for non-right handed individuals. Also, if the sample size had been greater a more economic approach to the analysis would have been to fit multivariate (rather than univariate and bivariate) models.

Heritability estimates

Power to detect associations between personality measures and total adjustment scores, and between personality and total adjustment scores and indices of early development could have been increased by combing the adolescent and adult data. However, the very low heritability estimates for GHQ scores, in comparison to SDQ scores, meant that it could not be assumed variation in adjustment measured by both scales come from the same distributions. Due to the sample sizes, confidence intervals around parameter estimates are wide. However, in most cases heritability estimates are consistent with estimates from other twin studies with large samples.

Proportion of variance in personality accounted for by CATS dimensions

Generally, relationships between personality and adjustment measures were moderate suggesting that the measures were not just capturing similar behaviours. However, the relationship between parent report conscientiousness and prosocial behaviour was strong and did suggest that these two dimensions are reflecting very similar behaviours.

Multivariate analysis of the relationship between adjustment and school attainment

was not carried out as correlations were low and as both variables were parent report, may have arisen in part as a result of shared method variance.

Differentiation between bivariate heritability and phenotypic causality models

Within the current design it was not possible to distinguish between models of bivariate heritability (shared genetic and environmental risk factors, which directly influence both phenotypes, or are mediated by a third variable) and phenotypic causality models (one phenotype alters the risk for vulnerability to the other) (see Simonoff, 2000) (see section 1.4.6).

6.7 CONCLUSIONS

The association between personality and adjustment scores found in previous studies remains even when overlap in the content of measures is controlled for. Phenotype correlations were similar to findings in the literature based on both clinical and population samples, suggesting the relationship between personality and adjustment may not differ between normal and extremes of variation in behavioural adjustment. As there was a wide age range in the current studies this adds to the literature, where the same personality measure was used for children, adolescents and young adults. The finding of significant genetic influences in the association between personality and adjustment measures, and between personality and some school attainment measures, suggest biological influences on personality contribute, at least in part, to behaviour problems. This could be directly shared genetic influences, or the influence of personality could be mediated through gene-environment correlations by influencing the way individuals are exposed too, or select different environmental factors. The finding of a larger common environment influence on daring may be due to a mediation effect for personality. There was also some evidence of overlap between genetic influences for personality and school attainment, which is consistent with theories concerning the relationship between personality, cognition, and adjustment (e.g. Kochanska, 1997; Nigg, 2000). However, the current study has only shown associations between personality and adjustment, and the mechanisms underlying these associations need to be explored.

APPENDICES

APPENDIX A

Child and Adolescent Temperament Scale Parent Version (CATS-P) **A**

Name _____

Read the following questions and describe which choice best fits the child/adolescent you are describing in this questionnaire. When you answer these questions think about the way the person has been like over the last twelve months. To answer, tick the appropriate box.

1. Does he/she enjoy being with other children/people his/her own age?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

2. Do his/her moods change unpredictably?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

3. Does he/she try to cheer up other children/people his/her age who are sad or upset?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

4. Does he/she feel sorry for kids who get picked on?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

5. Does he/she enjoy bothering or hurting other children/people his/her own age?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

6. Is your child curious?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

7. Does he/she get upset easily?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

8. Is he/she jealous of what other children/people have?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

9. Is he/she energetic when he/she has a job to do?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

10. Is he/she calm and easy going?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

11. Does he/she care about other children/people's feelings?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

12. Is he/she friendly?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

13. Does he/she enjoy it when other people say he/she did a good job?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

14. Is he/she afraid of children/people his/her age who like to fight?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

15. Does he/she enjoy doing things that are risky and dangerous?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

16.Does he/she keep his/her true feelings to himself/herself?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

17. Does he/she enjoy learning about new and interesting things?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

18. Does he/she think it's funny when other children/people his/her age are upset?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

19. Does he/she try to do excellent work (in school/at work)?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

20. Does he/she want everyone to follow the rules, including himself/herself?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

21. Is he/she daring and adventurous?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

22. Would he/she get upset if he/she saw an animal being hurt?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

23. Does he/she like meeting new children/people his/her age?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

24. Is he/she concerned about what is right and wrong?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

25. Would he/she feel guilty if he/she did something that broke the law?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

26. Is he/she smooth and charming when he/she is trying to get his/her own way?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

27. Does he/she like TV, movies, comics or electronic games with a lot of violence in them?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

28. Is he/she easily embarrassed?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

29. Does he/she avoid situations where he/she might get hurt?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

30. Does he/she react with <u>little or no emotion</u> to both positive and negative things?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

31. Does he/she enjoy doing what he/she is told <u>not</u> to?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

32. Is she/she carefree?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

33. Does he/she like things that are exciting and loud?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

34. Does he/she make decisions quickly because he/she doesn't like to wait?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

35. Does he/she get bored easily?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

36. Does he/she daydream a lot?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

37. Does he/she share his/her things with other children/people his/her own age without being asked?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

38. Does he/she react intensely when he/she gets upset?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

39. Does he/she do things to help other children/young people without being asked?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

40. Is he/she enthusiastic about life?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

41. Would it bother him/her if he/she didn't have a close friend?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

42. Is he/she brave?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

43. Does he/she like rough games and sport?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

44. Does he/she exaggerate things and blow them out of proportion?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

45. Is he/she shy with other children/people his/her own age?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

46. Is he/she selfish?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

47..Does he/she like for things to stay the same and not change?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

48. Does he/she feel bad for other children/people his/her own age when they get hurt?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

49. Does he/she believe that spiritual forces (i.e. God) sometimes direct life?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

50. Is he/she good at telling lies that other people believe?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

50. Does he/she like to scare other children/people his/her age?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

52. Is he/she emotional?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

53. Is he/she cautious?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

Child and Adolescent Temperament Scale Youth Version (CATS-P) **A**

Name _____

Read the following questions and describe which choice bests describes you. When you answer these questions think about the way you have usually felt or acted over the last twelve months. To answer, tick the appropriate box.

1. Do you enjoy being with other children/people your own age?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

2. Do your moods change unpredictably?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

3. Do you try to cheer up other children/people your age who are sad or upset?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

4. Do you feel sorry for kids who get picked on?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

5. Do you enjoy bothering or hurting other children/people your age?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

6. Are you curious?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

7. Do you get upset easily?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

8. Are you jealous of what other children/people have?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

9. Are you energetic when you have a job to do?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

10. Do you make decisions quickly because you don't like to wait?

1. Not at all ☐
2. Just a little ☐
3. Pretty much/pretty often ☐
4. Very much/very often ☐

11 Are you calm and easy-going?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

12. Do you care about other children/people's feelings?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

13. Are you friendly?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

14. Do you enjoy it when other people say you did a good job?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

15. Are you afraid of children/people your age who like to fight?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

16. Do you enjoy doing things that are risky or dangerous?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

17. Do you keep your true feelings to yourself?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

18. Do you enjoy learning about new and interesting things?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

19. Do you think it's funny when other children/people your age are upset?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

20. Do you try to do excellent work (in school/at work)?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

21. Do you want everyone to follow the rules, including yourself?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

22. Are you daring and adventurous?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

23. Would you get upset if you saw an animal being hurt?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

24. Do you like meeting new children/people your age?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

25. Are you concerned about what is right and wrong?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

26. Would you feel guilty if you did something that broke the law?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

27. Are you smooth and charming when you are trying to get your own way?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

28. Are you proud of yourself?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

29. Do you like TV, movies, comics, or electronic games with a lot of violence in them?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

30. Are you cheerful?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

31. Are you easily embarrassed?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

32. Do you avoid situations where you might get hurt?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

33. Do you react with little or no emotion to both positive and negative things?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

34. Do you believe that spiritual forces (i.e. God) sometimes direct life?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

35. Do you enjoy doing what you are told not to do?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

36. Are you carefree?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

37. Do you like things that are exciting and loud?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

38. Are you a self-starter, who does the things you need to do without being told?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

39. Do you get bored easily?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

40. When you have something to do, are you determined to get it done?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

41. Do you share things with other children/people your age without being asked?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

42. Do you react intensely when you get upset?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

43. Do you do things to help other children/young people without being asked?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

44. Are you enthusiastic about life?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

45. Would it bother you if you didn't have a close friend?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

46. Are you brave?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

47. Do you like rough games and sports?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

48. Do you exaggerate things and blow them out of proportion?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

49. Are you shy with other children/people your age?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

50. Are you selfish?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

51. Do you like for things to stay the same and not change?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

52. Do you feel bad for other children/people your age when they get hurt?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

53 Do you feel confident that you can handle life's challenges?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

54. Are you good at telling lies that other people believe?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

55. Do you like to scare other children/people your age?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

56. Do you daydream a lot?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

57. Are you emotional?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

58. Are you cautious?

1. Not at all
2. Just a little
3. Pretty much/pretty often
4. Very much/very often

B

Strengths and Difficulties Questionnaire

For each item, please mark the box for Not True, Somewhat True or Certainly True. It would help us if you answered all items as best you can, even if you are not absolutely certain or the item seems daft! Please give your answers on the basis of your child's behaviour over the last six months.

Child's name	Not True	Somewhat True	Certainly True
1. Considerate of other people's feelings			
2. Restless, overactive, cannot stay still for long			
3. Often complains of headaches, stomach-aches or sickness			
4. Shares readily with other children (treats, toys, pencils etc)			
5. Often has temper tantrums or hot temper			
6. Rather solitary, tends to play alone			
7. Generally obedient, usually does what adults request			
8. Many worries, often seems worried			
9. Helpful if someone is hurt, upset or feeling ill			
10. Constantly fidgeting or squirming			
11. Has at least one good friend			
12. Often fights with other children or bullies them			
13. Often unhappy, down-hearted or tearful			
14. Generally liked by other children			
15. Easily distracted, concentration wanders			
16. Nervous or clingy in new situations, easily loses confidence			
17. Kind to younger children			
18. Often lies or cheats			
19. Picked on or bullied by other children			
20. Often volunteers to help others (parents/teachers/other children)			
21. Thinks things out before acting			
22. Steals from home, school or elsewhere			
23. Gets on better with adults than with other children			
24. Many fears, easily scared			
25. Sees tasks through to the end, good attention span			

Signature ..... Date .....

Parent/Teacher/Other (please specify)

General Health Questionnaire

Name _____

Please read this carefully:

We should like to know if you have had any medical complaints and how your health has been in general over the past few weeks. Please answer all the questions on the following page simply by underlining the answer which you think most nearly applies to you. Remember that we want to know present and recent complaints, not those you had in the past.

It is important that you try to answer **all** the questions.

Have you recently:

1. been able to concentrate on whatever you are doing?	Better than usual	Same as than usual	Less than than usual	Much less Usual
2. lost much sleep over worry?	Not at all	No more than usual	Rather more than usual	Much more than usual
3. felt you are a useful part in things?	More so than usual	Same as usual	Less useful than usual	Much less useful
4. felt capable of making decisions about things?	More so than usual	Same as usual	Less so than usual	Much less than usual
5. felt constantly under strain?	Not at all	No more than usual	Rather more than usual	Much more than usual
6. felt you couldn't overcome your difficulties?	Not at all	No more than usual	Rather more than usual	Much more than usual
7. been able to enjoy your normal day to day activities?	More so than usual	Same as usual	Less so than usual	Much less than usual
8. been able to face up to your problems?	More so than usual	Same as usual	Less able than usual	Much less than usual
9. been feeling unhappy and depressed?	Not at all	No more than usual	Rather more than usual	Much more than usual
10. been losing confidence in yourself?	Not at all	No more than usual	Rather more than usual	Much more than usual
11. been thinking of yourself as a worthless person?	Not at all	No more than usual	Rather more than usual	Much more than usual
12. been feeling reasonably happy, all things considered?	More so than usual	About the same as	Less so than usual	Much less than usual

Can you please complete this questionnaire for each child/adolescent D

Name _____ Age _____ Date of birth __/__/__

How does your son or daughter's performance on the following compare with the average for a child/adolescent of that age?

	Above average	About average	Below average
Reading	☐	☐	☐
Spelling	☐	☐	☐
Handwriting	☐	☐	☐
Maths	☐	☐	☐
Art	☐	☐	☐
Music	☐	☐	☐
Computers	☐	☐	☐
Science	☐	☐	☐
PE and games	☐	☐	☐

Has your son or daughter ever had any of the following immune disorders?

	Yes	No
Asthma	☐	☐
Hayfever	☐	☐
Eczema	☐	☐
Rheumatoid artheritis	☐	☐
Ulceritive colitis	☐	☐
Other (please specifiy)	☐	☐

Which hand does your son or daughter use for the following activities?

1. Writing?

Always left	Usually left	Both hands equally	Usually right	Always right
☐	☐	☐	☐	☐

2. Holding a toothbrush?

Always left	Usually left	Both hands equally	Usually right	Always right
☐	☐	☐	☐	☐

3. Throwing/catching a ball?

Always left	Usually left	Both hands equally	Usually right	Always right
☐	☐	☐	☐	☐

4. Holding a knife (without a fork)?

Always left	Usually left	Both hands equally	Usually right	Always right
☐	☐	☐	☐	☐

Please read the following questions and look at the last three pages. Then can you detach these and ask your son or daughter to perform each task (you will need a watch with a second hand for the last task).

After each task can you then answer the appropriate question.

I. Which hand did they use to draw each X?

	Left	Right
1.		
2.		
3.		

2. Which hand did they use to copy the shapes?

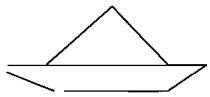
Always left	Usually left	Both hands equally	Usually right	Always right

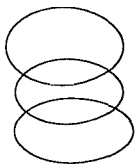
Can you please reattach the pages or write your son or daughter's name on each one.

Name _____ E

In the box below can you draw an X three times, with a different colour pencil each time.

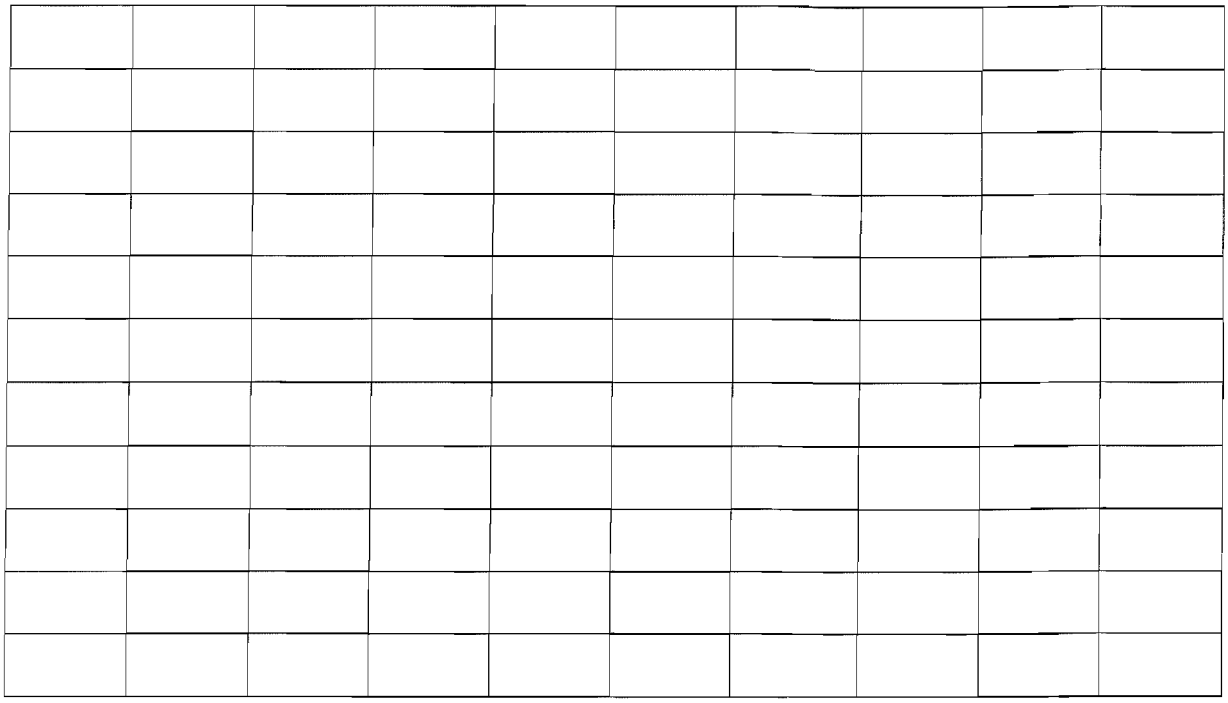
In the box below can you copy the shapes.



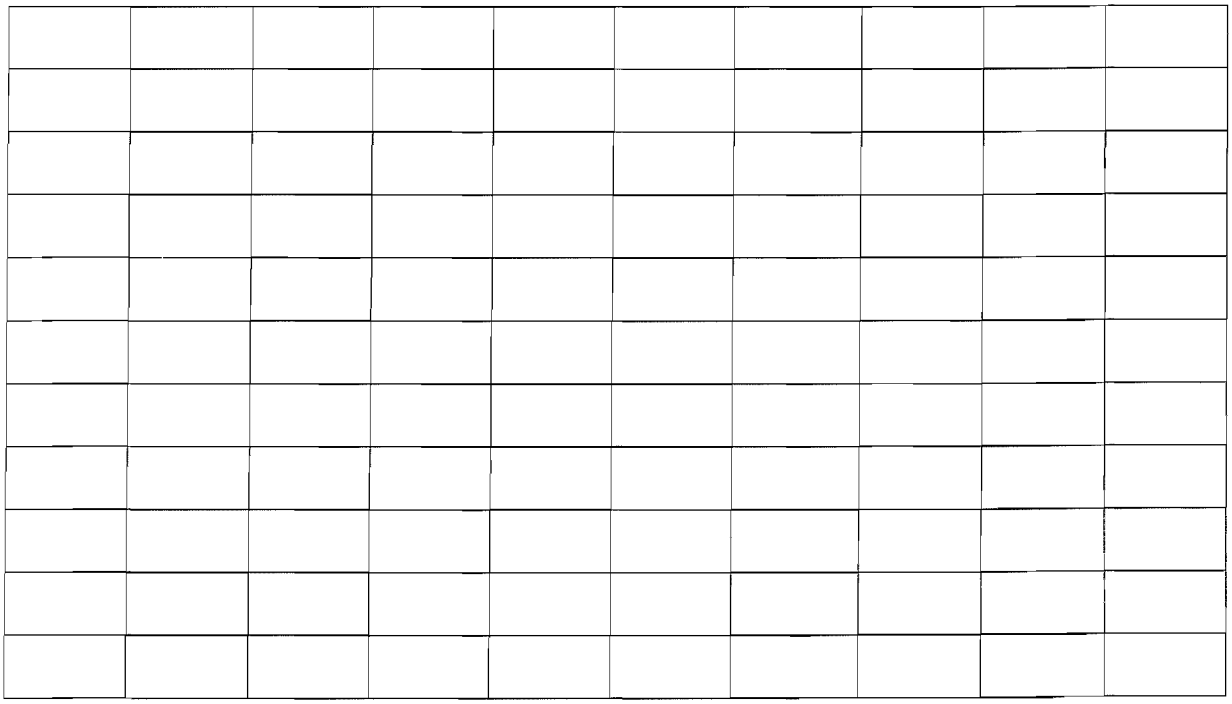


Name _____

Using your right hand, please tick as many boxes as you can in 15 seconds.

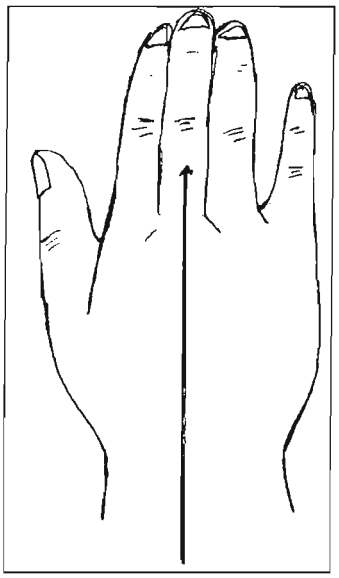


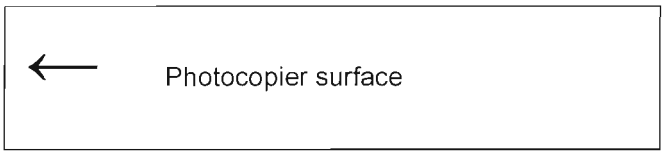
Using your left hand, please tick as many boxes as you can in 15 seconds.



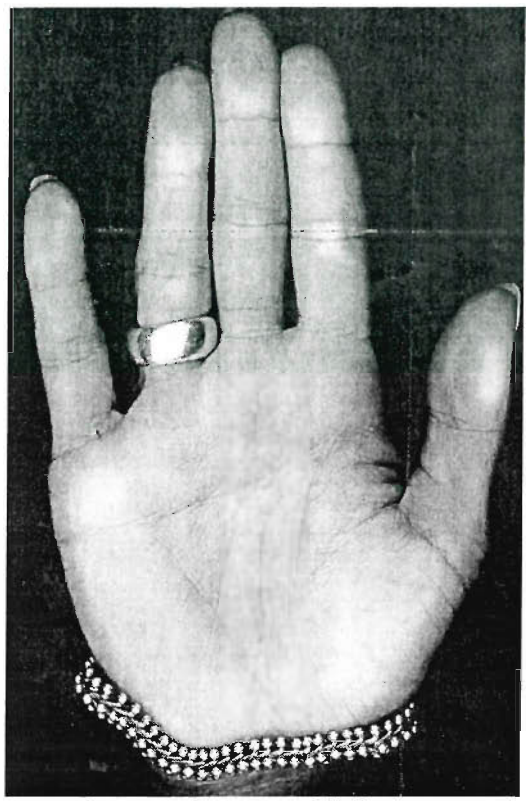
Hand position

Both hands need to be placed firmly on the surface of the photocopier palm-side down so full contact is made. For each hand the ring, middle and index fingers need to be closed, with the thumb and the little finger slightly relaxed. It is very important that the middle finger of each hand is in alignment with the forearm and that the wrist is straight. (see below) (It is quite hard to keep the arms straight so it may be easier to photocopy one hand at a time).





For accurate measurement the photocopy should clearly show the length of the index, middle and ring fingers, and the crease where the fingers join the palm. E.g.



****Could you please write your child's name on the back of the photocopy.**

TWIN SIMILARITY QUESTIONNAIRE **G**

Please ring the answer that is correct for your twins. If questions 1 – 6 are difficult to answer because of the twins age please enter N/A for not applicable.

1. Are the twins emotionally attached to each other? N/A Strongly Somewhat Not at all
2. Do the twins have the same friends at the house? N/A Strongly Somewhat Not at all
3. Do the twins argue? N/A Strongly Somewhat Not at all
4. Do the twins try to be different from one another? N/A Strongly Somewhat Not at all
5. Up to what age were the twins dressed alike? N/A Strongly Somewhat Not at all
6. Has one of the twins ever told you that they should not be dressed the same anymore? N/A Strongly Somewhat Not at all
7. To what extent are the twins similar at the moment for the following: -

Height	Not at all	Somewhat	Exactly
Weight	Not at all	Somewhat	Exactly
Facial appearance	Not at all	Somewhat	Exactly
Hair colour	Not at all	Somewhat	Exactly
Eye colour	Not at all	Somewhat	Exactly
Complexion	Not at all	Somewhat	Exactly

8. Do they look as alike as two peas in a pod? No Yes
9. Do you ever confuse them? No Yes
10. Are they sometimes confused by other people in the family? No Yes
11. Is it hard for strangers to tell them apart? No Yes

APPENDIX H

Comparison of factor loadings – parent report

	US sample n = 1358	Study one n = 326
Conscientiousness	F1	F1
Care about others feelings	.65	.76
Feel for others hurt	.64	.70
Spontaneously helps	.61	.80
Sorry for victims	.60	.74
Cheers others up	.59	.76
Concerned about right and wrong	.55	.52
Wants all follow rules	.54	.34
Guilty if broke law	.46	.50
Cautious	.44	.17
Tries to do excellent work	.42	.24
Upset saw animal hurt	.42	.40
Spontaneously shares	.42	.70
Enjoys learning interesting things	.41	.23
Negative emotionality	F2	F2
Upset easily	.69	.84
Reacts intensely	.61	.77
Moods change unpredictably	.56	.75
Blows out of proportion	.55	.60
Emotional	.54	.75
Enjoys non-complying	.50	.26
Believable lies	.48	.27
Jealous	.45	.36
Bored easily	.45	.28
Selfish	.44	.30
Easily embarrassed	.40	.19
Calm/and easy going	-.42	-.69
Daring	F3	F3
Daring and adventurous	.62	.74
Enjoys risky	.50	.75
Likes rough sport/games	.47	.49
Likes meeting people	.45	.13
Friendly	.43	.10
Brave	.43	.63
Likes exciting/loud	.40	.43
Shy	.48	-.08

Comparison of factor loadings – self report

	US sample n = 826	Study one n = 570
Conscientiousness	F1	F1
Feel for others hurt	.66	.52
Care about others feelings	.66	.60
Cheers others up	.65	.58
Sorry for victims	.63	.49
Spontaneously helps	.60	.73
Likes meeting people	.54	.37
Friendly	.49	.46
Enjoys praise	.48	.17
Guilty if broke law	.45	.11
Concerned about right and wrong	.45	.18
Enjoys being with others	.43	.05
Upset saw animal hurt	.42	.23
Want all follow rules	.41	.05
Tries to do excellent work	.40	.30
Daring	F2	F2
Enjoys risky	.60	.71
Daring and adventurous	.60	.72
Brave	.52	.63
Likes rough sport/games	.51	.76
Likes exciting/loud	.50	.45
Believable lies	.44	.09
Likes violent TV	.42	.50
Negative emotionality	F3	F3
Upset easily	.60	.66
Reacts intensely	.54	.75
Moods change unpredictably	.50	.73
Blows out of proportion	.47	.46
Easily embarrassed	.46	.12
Bored easily	.46	.19
Jealous	.44	.22
Emotional	.41	.56

Appendix I

Comparison of path values between models fitted to covariance and correlation matrices for univariate models

	Covariance			Correlation		
	a	c	e	a	c	e
Pneg	.881		.473	.915		.403
Sneg (comb)	.488	.416	.767	.616	.276	.738
Emot	.850		.527	.855		.519
Cond	.777		.629	.843		.537
Hype	.759		.651	.760		.650
Peer	.722		.692	.728		.686
SDQ	.866		.499	.912		.410
GHQ	.365		.934	.378		.926
SDQ prosocial	.844		.537	.870		.493
Read	.820	.241	.518	.759	.351	.549
Handw	.697		.717	.692		.722
Art	.855		.518	.867		.498
Comp		.770	.639	.	.730	.683
2d4dD	.382		.924	384		.924
Dextav	.723	.617	.750	.628	.359	.691
DextD	.525		.851	.557		.847
Hskll	.415		.910	.420		.908
Immune	.802		.598	.796		.606
Pcns (tsib)	.840		.543	.835		.538
Pneg (tsib)	.869		.495	.901		.433
Cond (tsib)	.744		.668	.791		.611
Hype(tsib)	.690		.724	.687		.727

Appendix J

Path values for bivariate AE models fitted to covariance and correlation models

	Covariance		Correlation			Covariance		Correlation	
	a1	a2	a1	a2		a1	a2	a1	a2
Pcon	.88		.91		Scon	.79		.79	
Cond	.48	.62	.67	.42	Cond	-.25	.73	-.34	.76
	e1	e2	e1	e2		e1	e2	e1	e2
Pcn	.47		.41		Scn	.64		.62	
Cond	.15	.60	.21	.58	Cond	-.04	.63	.06	.55
	a1	a2	a1	a2		a1	a2	a1	a2
Scon	.79		.77		Scon	.76		.78	
Hype	-.32	.67	-.30	.66	TSDQ	-.34	.79	-.38	.83
	e1	e2	e1	e2		e1	e2	e1	e2
Scon	.61		.64		Scon	.65		.62	
Hype	-.01	.67	-.04	.68	TSDQ	-.02	.50	.02	.41
	a1	a2	a1	a2		a1	a2	a1	a2
Pneg	.87		.90		Pneg	.87		.91	
Emot	.50	.64	.51	.64	Cond	.66	.37	.67	.42
	e1	e2	e1	e2		e1	e2	e1	e2
Pneg	.50		.44		Pneg	.49		.41	
Emot	-.02	.58	-.05	.57	Cond	.67	.59	.21	.57
	a1	a2	a1	a2		a1	a2	a1	a2
Sneg	.62		.66		Pneg	.88		.91	
Cond	.33	.69	.36	.74	Hype	.48	.43	.46	.43
	e1	e2	e1	e2		e1	e2	e1	e2
Sneg	.78		.75		Pneg	.48		.40	
Cond	.06	.65	.02	.57	Hype	.18	.74	.18	.75
	a1	a2	a1	a2		a1	a2	a1	a2
Sneg	.62		.66		Pneg	.86		.90	
Hype	.36	.68	.36	.65	Peer	.35	.57	.31	.60
	e1	e2	e1	e2		e1	e2	e1	e2
Sneg	.78		.75		Pneg	.51		.44	
Hype	-.02	.68	-.03	.67	Peer	-.25	.70	-.26	.69
	a1	a2	a1	a2					
Sneg	.62		.66						
Peer	.23	.66	.19	.68					
	e1	e2	e1	e2					
Sneg	.79		.75						
Peer	-.08	.71	-.06	.71					

Path values for bivariate ACE models fitted to covariance and correlation models

	Covariance		Correlation			Covariance		Correlation	
	a1	a2	a1	a2		a1	a2	a1	a2
Sdar	.45		.40		Sdar	.45		.41	
Cond	.40	.66	.45	.71	Hype	.55	.47	.63	.36
	c1	c2	c1	c2		c1	c2	c1	c2
Sdar	.36		.39		Sdar	.34		.37	
Cond	n/a	n/a	n/a	n/a	Hype	n/a	n/a	n/a	n/a
	e1	e2	e1	e2		e1	e2	e1	e2
Sdar	.82		.80		Sdar	.83		.83	
Cond	-.03	.62	.00	.54	Hype	-.06	.67	-.06	.68

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