School of Psychology

The Genetic Architecture of Reactive and Proactive Aggression: Relations to Disruptive Behaviour Problems Through Development

James Hanly

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Declaration

To the best of my knowledge and belief this thesis contains no material previously published by any other person except where due acknowledgment has been made.

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university.

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Abstract

Over the past two decades there has been increasing interest in the distinction between reactive (RA) and proactive aggression (PA; Card & Little, 2006; Polman, Orobio de Castro, Koops, van Boxtel & Merk, 2007). RA describes aggression that is defensive, impulsive and affect-laden, while PA comprises instrumental, calculated and typically unemotional aggressive behaviours (Vitaro, Brendgen & Tremblay, 2002). There is growing consensus that developmental models of RA and PA may help clarify risk pathways associated with the three disruptive behaviour disorders (DBD), attention-deficit hyperactivity disorder, oppositional defiant disorder and conduct disorder (Kempes, Matthys, de Vries & van Engeland, 2005; Raine et al., 2006; Waschbusch, Willoughby & Pelham, 1998). However, some confusion remains regarding etiological influences responsible for the differential shaping of aggression subtypes (Baker, Raine, Liu & Jacobson, 2008).

Contributing the first elucidation of developmental relations between aggression subtypes and DBDs, caregiver ratings were ascertained from a large community sample of families of twins (aged 6-18 years) using the Australian Twin Behaviour Rating Scales (ATBRS, n=2082), and at 9-month follow-up via an online electronic version of the ATBRS (n=511). These data were partitioned according to two age cohorts (6-10 years, and 10-18 years) and subsequently submitted to a series of univariate and multivariate cross-sectional and longitudinal analyses.

Consistent with previous research (e.g., Baker et al., 2008), cross-sectional models indicated strong influence of genes on both RA and PA—with genes showing greater

effects on PA compared to RA, particularly in the older cohort. Multivariate longitudinal analyses revealed a substantial differential role for genes in the continuity of aggression subtypes over time for both RA and PA in the younger cohort, and for PA in the older cohort. At odds with etiological models that posit differential psychosocial factors underpinning RA and PA (Crick & Dodge, 1996; Dodge, 1991), no evidence was found for shared environmental effects on persistence in RA over time. Conversely, shared environmental effects explained a significant portion of covariance in childhood PA across time points. However, contrary to predictions derived from psychosocial explanations (e.g., Dodge, 1991), these shared environmental influences were mediated through a general risk for (undifferentiated) overt physical aggression.

Data provide preliminary, albeit modest, evidence for a recent sequential model of RA and PA (i.e., Vitaro & Brendgen, 2005) which implicates RA as an early developmental precursor to PA. This model reformulates the role of social learning mechanisms invoked by seminal psychosocial formulations (i.e., Dodge, 1991). Specifically, the sequential hypothesis postulates that early RA which is reinforced in childhood, may increasingly come to be used operantly (i.e., instrumentally). In this way, RA is effectively converted to PA-type aggression. As predicted by this model, current data revealed the longitudinal relationship from RA to PA was predominantly explained by shared environmental influences (over and above a general risk for overt aggression) in the younger cohort. By contrast, the pathway from RA to PA in the older cohort was explained mainly by the influence of genes. These data provide some support for the suggestion that putative social learning mechanisms impact on the sequential pathway in childhood but not adolescence.

The expected high levels of phenotypic overlap between aggression subtypes and DBDs were consistently explained by genes and nonshared environmental influences with the former accounting for the majority of covariance in most bivariate models. Age cohort differences in multivariate models of aggression subtypes and DBDs were in line with the smorgasbord hypothesis suggesting the effects of genes generally increase, while the effects of shared environment generally decrease, as a function of age. This pattern was most consistent and pervasive in RA-related models. Notable exceptions to this pattern occurred in bivariate models involving PA on the one hand, and hyperactivity or CD on the other—with these models showing greater influence of shared environment in the older versus younger age cohort. Additionally, the current research indicates a trend towards greater segregation of genetic effects across aggression subtypes and DBDs as a function of age, while conversely, shared environmental effects were more likely to simultaneously affect multiple syndromes in the older versus younger cohort.

In regards to clinically specified risk pathways, relevant longitudinal analyses suggested that impulsivity confers only limited risk for future RA, while partial support was obtained for recent suggestions that both hyperactivity and PA are important cofactors in risk pathways associated with ODD and CD. Overall, the data that include DBDs broadly support RA and hyperactivity as key early markers of long-term risk.

The online component of the study also included two neuropsychological tasks adapted for the internet and completed by 310 twin siblings. Representing an attempt to integrate multiple explanatory frameworks, this latter study evaluated

differerential putative neuro-biogenic mechanisms underpinning RA and PA. Results from this study were inconclusive and issues pertaining to the delivery of neuropsychological tasks online are considered. In contrast to results from the adapted neuropsychological tasks, the online electronic ATBRS yielded higher data integrity and higher scale reliabilities than its original paper-and-pencil counterpart.

All results are discussed and implications for future research and clinical practice relating to childhood and adolescent aggression are considered. Finally, limitations of the current research project are examined.

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Chapter 1.
Introduction

1.1 Overview

This chapter provides a synopsis of the content contained in the current thesis. An outline of each of the main eight chapters is provided below.

1.2 Chapter Synopses

1.2.1 Synopsis of Chapter 2

Chapter 2 reviews current developmental perspectives on antisocial behaviour and aggression, with a particular emphasis on research efforts to define clinically useful etiological models of normative versus non-normative aggressive behaviour. It examines the distinction between reactive (RA) and proactive (PA) aggression subtypes, and further considers the etiological relationship between these aggression subtypes and extant clinical diagnostic entities including attention-deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD) and conduct disorder (CD).

Particular focus is afforded to the role of quantitative behaviour genetics in elucidating core clinical mechanisms in risk pathways associated with antisocial and disruptive behaviour generally, and RA and PA more specifically. Research concerning neurospychological correlates of RA and PA is also presented in an attempt to articulate and integrate a multiple levels perspective within a common empirical framework (Hinshaw, 2002). Finally, a rationale is provided for utilising

internet technologies in collecting genetically informative data.

A formulation of the current research agenda is presented at the end of this chapter and is stated in terms of objectives and aims associated with the current research. This involves objectives and a set of aims for each of the three main studies involved.

It is worth briefly commenting the terminology chosen to describe these studies. The current research project involved two main measurement occasions. For clarity, these are labelled Study1 and Study 2 respectively. Study 1 included caregiver rated data only. However, data from the second measurement occasion included both caregiver rated data, as well as data from a set of neuropsychological tasks completed by twins. These latter two data sets were analysed separately. As such these separate analyses were labelled Study 2a and Study 2b respectively.

1.2.2 Synopsis of Chapter 3

Chapter 3 provides information concerning the methodology and design features underpinning the current research project. The first section describes the broader research context within which the present series of studies were undertaken. A description of participant characteristics follows. The process of implementation (i.e., the design) of the two main studies is then outlined in section 3.4 (p. 87). Next, a number of consecutive sections detail the assessment protocols used and the measures derived from these instruments. The remainder of the chapter introduces some technical aspects of behaviour genetics modeling.

1.2.3 Synopsis of Chapter 4

Chapter 4 provides sample characteristics and descriptives for data obtained at each of the two main measurement occasions. Study 1 data was derived from the pencil-and-paper protocol version of the Australian Twin behaviour Rating Scale (ATBRS, administered as part of a large research project at Wave One). Study 2 data was collected exclusively over the internet and involved two separate online protocols.

The first protocol was an electronic short-form version of the caregiver-rated ATBRS. The second protocol represented two neuropsychological tasks adapted for delivery over the internet. Both these protocols were embedded in a software package delivered on CD-ROM to participant families. This integrated software allowed families to use their own home computers to login to a secure database which collected and temporarily stored all participant data.

1.2.4 Synopsis of Chapter 5

Chapter 5 presents results from preliminary genetic analyses of aggression subtypes and the disruptive behaviour disorders (DBDs). The first section revisits the model fitting process introduced in Chapter 3 providing more detail regarding the statistical procedures employed. Findings from the basic univariate models of RA and PA are then presented and key aspects discussed. Next, a series of univariate statistical models are considered with the aim to elucidate sex differences in underlying etiology of aggression subtypes. Finally, basic multivariate analyses that include DBD symptom dimensions as covariates are provided.

1.2.5 Synopsis of Chapter 6

Chapter 6 reports on longitudinal analyses relevant to Study 2a. This latter study involved the recruitment of a subset of 510 caregivers who completed the ATBRS as part of a larger Curtin research project (which encompassed the current Study 1) and who additionally consented to participate in the second study. All 24 items of the short-from ATBRS-ES questionnaire utilised in Study 2a derive directly from the 48 items used in Study 1 and both utilise the same four-point Likert scale. The quantitative genetic analyses of these longitudinal data are the focus of this chapter.

The length of time between Wave One and two data collection varied for different participant families. Data from the Wave One phase was collected approximately betwen january 2006 and December 2006. Data collection for Wave Two occurred between March 2007 and June 2007. A minimum time period of nine months between Wave One and two data collection was imposed such that only families who's *Wave One* data was collected prior to July 2006, were included for the longitudinal analyses. Because the majority of families had submitted their data prior to June 2006 (n=1815), only 77 of the 551 families (15%) who went on to consent to participation in the follow up online study were omitted from longitudinal analyses.

It is reiterated that, unlike the initial measurement occasion, Study 2 data was obtained exclusively in electronic format via the internet. This involved adapting the ATRBRS into electronic format using a web-application software program called Flash. The rationale, technology and procedures underpinning this methodology are provided in Chapter 7.

After a brief literature review and explanation of method, results are reports on longitudinal relationships revealed in the data with particular reference to the main objective and three aims of Study 2a.

1.2.6 Synopsis of Chapter 7

Chapter 7 reports on the online component of the current research, i.e., Study 2. This includes attempts to evaluate the reliability and validity of the online measures. Related technical and methodological issues unique to collecting psychological data online are also considered.

As noted in earlier chapters, caregiver ratings derived from the online questionnaire were used in longitudinal analyses (Study 2a) reported in Chapter 6. Chapter 7 examines more closely the data derived from the two neuropsychological tasks completed by twins (Study 2b). As explained herein, this neuropsychological data failed to meet basic assumptions regarding expected associations with key behavioural indices measured by the online questionnaire. A brief synopsis of the task data is provided and possible reasons for the null results are explored.

1.2.7 Synopsis of Chapter 8

Chapter 8 provides a summary of the main findings and discusses their implications for both research and clinical practice. Suggestions for future research are examined and limitations associated with the current research project are presented.

Chapter 2.

Etiology of Reactive and Proactive Aggression: A Literature Review

2.1 Developmental Perspectives on Antisocial Behaviour, Disruptive Behaviour, and Aggression

Life-course persistent antisocial behaviour presents a major challenge for tiered intervention services (Coie & Dodge, 1997; Mitchell, Colledge, Leonard & Blair, 2002; Waschbusch, 2002). A recent government report on offending behaviour identified the considerable scope of social and economic impacts of crime in Australia (Mayhew, 2003). The paper estimates that, in addition to the intangible and often tragic personal costs of violent crime, violent offending in 2003 cost the Australian community around $5 billion per annum (or nearly 0.8% of the national gross domestic product). Impacts were identified across multiple sectors including health, insurance, security and judicial systems. These estimates dovetail with statistics reported for other developed countries such as the United States (Anderson, 1999) and the UK (Brand & Price, 2000) and highlight the role of social research in elucidating risk factors for antisocial behaviour to inform effective intervention.

In socio-cultural terms, antisocial behaviour (ASB) describes any transgression of basic social norms or laws (Lahey, Waldman & McBurnett, 1999). From an interpersonal perspective, the term refers to behaviour that deliberately disadvantages others (Kempes, Matthys, de Vries & van Engeland, 2005), and/or behaviour that is experienced as aversive by others (Kiesner, Dishion & Poulin, 2001). Antisocial behaviour thus covers a diverse spectrum of both non-aggressive and aggressive

actions that vary considerably in nature and frequency across the life-span (Rutter, 1996). From a clinical perspective, non-aggressive ASB includes a constellation of symptoms including rule-breaking such as stealing, lying, truancy and other statutory offences, while aggressive ASB includes a wide range of social violations pertaining to verbal and physical (including sexual) assault and threatening behaviour as well as property damage (Simonoff et al., 1998). This distinction is discussed in more detail later in this chapter.

Of the various behaviours associated with life-course persistent ASB, it is overt physical aggression that has most consistently emerged as a primary risk factor linking the early childhood disruptive behaviour with life-course persistent antisocial outcomes (Broidy et al., 2003; Denham et al., 2000; Kokko & Pulkkinen, 2000; Lahey et al., 1999; Loeber & Hay, 1997; Loeber, Green, Lahey & Kalb, 2000; Nagin & Tremblay, 1999; Putallaz et al., 2007). Unsurprisingly then, the early identification of maladaptive physical aggression in childhood is considered critical for intervening effectively in antisocial pathways (Lynam, 1998).

While physical aggression is relatively normative in infancy (Pettit, 1997), research has shown that prevalence declines substantially in mid-to-late childhood and adolescence as inhibitory capacities develop (Tremblay et al., 2004; Vitaro, Brendgen & Barker, 2006). However, non-normative (i.e., persistent) trajectories typically become clinically salient during the preschool years (Tremblay et al., 2004). Indeed, early emergence of aggressogenic behaviour highlights the value of developmental models of aggression in refining taxonomic approaches to childhood, adolescent and adult antisocial behaviour (Loeber, Green, Keenan & Lahey, 1995;

Loeber, Green, Lahey, Frick & McBurnett, 2000; Vitiello & Stoff, 1997).

Aggression is broadly defined as behaviour enacted with the intent to cause harm or damage (Dodge & Schwartz, 1997; Pettit, 1997). Notional definitions such as this, however, belie the pervasive heterogeneity underpinning this etiologically complex phenotype. Indeed, recent advances across a range of research domains have emphasised the importance of conceptualising aggression as both *non-unitary* and *multi-determined* (Merk, de Castro, Koops & Matthys, 2005; Pliszka, 1999; Raine et al., 2006; Weinshenker & Siegel, 2002b). This work shows aggressive behaviour is associated with multifarious topology, i.e., different forms, (Crick, Casas & Ku, 1999; Keenan, Coyne & Lahey, 2008; Ostrov & Godleski, 2009; Putallaz et al., 2007), divergent functions (Crick & Dodge, 1996; Little, Jones, Henrich & Hawley, 2003; Marsee & Frick, 2007) and multiple risk pathways (Hinshaw, 2002). These findings have reinvigorated efforts to develop a clinically-relevant subtypology of aggression with the goal to inform and refine current early intervention practices (Bushman & Anderson, 2001; Keenan et al., 2008). More broadly, such efforts represent an opportunity to improve extant diagnostic taxonomies for antisocial behaviour—particularly in relation to the childhood disruptive behaviour disorders (DBD) articulated within the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV, American Psychiatric Association, 1994) and International Classification of Diseases (ICD-10, World Health Organisation, 1990)—through elucidating associated etiological factors and risk pathways (Brendgen, Vitaro, Boivin, Dionne & Pérusse, 2006; Kempes et al., 2005; Waschbusch, Willoughby & Pelham, 1998).

DBD symptomatology is organised according to three distinct but often co-occuring

syndromes; attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD) and conduct disorder (CD). ADHD consists of three symptom categories related to persistent (1) attentional difficulties, (2) motor hyperactivity and/or (3) impulsive behaviour (Lahey & Loeber, 1997). See Table 2.1 (p. 10) for symptom criteria. These symptom dimensions comprise three clinical subtypes of ADHD; inattentive symptoms (ADHD-IA), predominantly hyperactivity and impulsivity (ADHD-HI), and combined type ADHD (ADHD-C) which includes behaviours from all three symptoms dimensions. Research has generally confirmed the clinical reliability of these subtypes (Faraone, 2005) which have been associated with familial clustering of same-subtype combinations under substantial genetic control (Rasmussen et al., 2004).

Table 2.1

DSM-IV Diagnostic Criteria for Attention Deficit Hyperactivity Disorder

Inattentive Subtype

1. Often does not give close attention to details or makes careless mistakes in schoolwork, work, or other activities
2. Often has trouble keeping attention on tasks or play activities
3. Often does not seem to listen when spoken to directly
4. Often does not follow instructions and fails to finish schoolwork, chores, or duties in the workplace
5. Often has trouble organizing activities
6. Often avoids, dislikes, or doesn't want to do things that take a lot of mental effort for a long period of time
7. Often loses things needed for tasks and activities (e.g. toys, school assignments, pencils, books, or tools)
8. Is often easily distracted
9. Is often forgetful in daily activities

Hyperactivity Subtype

1. Often fidgets with hands or feet or squirms in seat
2. Often gets up from seat when remaining in seat is expected
3. Often runs about or climbs when and where it is not appropriate
4. Often has trouble playing or enjoying leisure activities quietly
5. Is often "on the go" or often acts as if "driven by a motor"
6. Often talks excessively

Impulsivity Subtype

1. Often blurts out answers before questions have been finished.
2. Often has trouble waiting one's turn.
3. Often interrupts or intrudes on others (e.g., butts into conversations or games).

ADHD, like ODD and CD is classed as a developmental disorder due to its predominance in childhood and adolescence. The most recent report on the population prevalence of ADHD suggests it affects around 6.8% of Australian children (Sawyer et al., 2001). However, the heterogenous nature of ADHD includes significant changes in presentation across the developmental continuum. While ADHD symptoms generally decrease with age (Willoughby, 2003), some estimates

suggest up to 50% of related symptomatology persists into adulthood (Biederman & Faraone, 2005). To further complicate the developmental picture, age-related reductions in symptomatology may be associated with a disproportionate increase in impairment incurred by accumulated primary and secondary adolescent symptoms (Ingram, Hechtman & Morgenstern, 1999). Specifically, while it appears that hyperactivity-related behaviours (i.e., excessive motor activity) show general improvement with age, (from childhood to adolescence), impaired attentional capacities and impulsivity have been shown to persist alongside residual secondary problems that result from poor scholastic achievement and other adverse outcomes of childhood ADHD (Hay, McStephen & Levy, 2001a; Ingram et al., 1999). Perhaps presenting the most significant challenge to researchers is the likelihood that symptoms occurring in adulthood are significantly phenotypically distinct from ADHD behaviours identified in childhood (Ingram et al., 1999). Ramsay and Rostain (2008) suggest that adult symptoms are primarily characterised by self-dysregulation and executive functioning deficits, but note that there is much empirical work required to adequately distinguish between childhood versus later forms of ADHD. Methodological issues include the fact that many studies have relied on retrospective measures of ADHD and there is certainly a need for more prospective longitudinal research. Nonetheless, it is clear that the potential persistence of underlying ADHD-related pathology into adulthood has become widely accepted in the literature.

ODD and CD have been referred to as the 'antisocial disorders' (e.g., Rutter, 2001). ODD is characterised by negativistic, disobedient and defiant behaviour. CD involves a constellation of aggressive and delinquent behaviours of a pervasively antisocial nature. See Tables 2.2 and 2.3 for relevant symptom criteria. The

population prevalence for ODD in Australia ranges between 6-10% (Sanders, Gooley & Nicolson, 2003) and is one of the major causes of hospitalisation in Australian children under 14—accounting for around 16% of this group (Al-Yaman, Bryant & Sargeant, 2002). This figure is similar to the 17% of child hospitalisations resulting from activity/attention disorders. In their (2001) report on prevalence of mental health disorders amongst 4083 6- to 17-year-old Australian youth, Sawyer et al. found around 3% met criteria for conduct disorder (3.2% for males and 2.8% for females).

Table 2.2

DSM-IV Diagnostic Criteria for Oppositional Defiant Disorder

1. Often loses temper
2. Often argues with adults
3. Often actively defies or refuses to comply with adults' requests or rules
4. Often deliberately annoys people
5. Often blames others for his or her mistakes or misbehavior
6. Is often touchy or easily annoyed by others
7. Is often angry and resentful
8. Is often spiteful or vindictive

Table 2.3

DSM-IV Diagnostic Criteria for Conduct Disorder

1. Often bullies, threatens, or intimidates others
2. Often initiates physical fights
3. Has used a weapon that can cause serious physical harm to others (for example, a bat, brick, broken bottle, knife, gun)
4. Has been physically cruel to people
5. Has been physically cruel to animals
6. Has stolen while confronting a victim (for example, mugging, purse snatching, extortion, armed robbery
7. Has forced someone into sexual activity
8. Has deliberately engaged in fire setting with the intention of causing serious damage
9. Has deliberately destroyed others' property (other than by fire setting)
10. Has broken into someone else's house, building, or car
11. Often lies to obtain goods or favors or to avoid obligations (in other words, "cons" others)
12. Has stolen items of nontrivial value without confronting a victim
13. Often stays out at night despite parental prohibitions, beginning before age 13 years
14. Has run away from home overnight at least twice while living in parental or parental surrogate home
15. Is often truant from school, beginning before age 13 years

The high degree of overlap between the DBDs remains a complex issue for clinical researchers (Lahey et al., 2009; Waldman et al., 2001). Notably, prevalence rates suggest that between 45% and 84% of children and adolescents with ADHD are diagnosed with ODD and/or CD (Barkley, 2006). The observation that both ODD and CD include aggressogenic symptoms (Greene & Doyle, 1999; Lahey et al., 1999), as well as the fact that aggression represents a considerable risk factor associated with comorbid ADHD+CD (Lynam, 1997) and negative long-term outcomes (Loeber et al., 1995; Loeber et al., 2000), has prompted renewed interest in the role of aggression in clarifying comorbidity and etiology in DBDs (Kempes et al., 2005). From an etiological perspective, ODD typically precedes CD and this has led

some researchers to postulate a developmental progression from ODD to CD (Lahey, Loeber, Quay, Frick & Grimm, 1992; Loeber et al., 2000). This model is discussed in more detail in a later section but serves to emphasise that *etiological comorbidity* is fundamental to understanding concurrent comorbidity in DBDs (Hinshaw, 2002).

While ODD, like CD, is a polytypic disorder (e.g., the constellation of symptoms may vary from child to child), the condition is not associated with any formal subtypology (indeed, the ICD-10 classifies this condition as a less severe variant of conduct disorder). Some have suggested that children with ODD who go on to develop CD may represent a distinct subgroup compared to their ODD peers who show no developmental progression (Rutter, 2001) however, this remains the subject of debate.

On the other hand, a number of subtypologies have been proposed for CD. For example, an earlier version of the DSM (DSM-III; American Psychiatric Association, 1980), as well as the current ICD-10, have employed Quay's (Quay, 1987; Quay, 1993) taxonomy which distinguishes between *undersocialised* and *socialised* aggressive conduct disorder. At the core of this distinction is the capacity of the conduct disordered child to regulate affect and form close relationships with others (including integrating into peer groups). Specifically, correlates differentiating this bimodal distinction suggest undersocialised CD is associated with a lack of social integration and poor affect regulation as well as earlier onset and poorer long-term outcomes (Deutsch & Erickson, 1989). While this taxonomy is still used in ICD-10 it was discontinued in the DSM-IV replaced instead by a focus on the age of onset of the disorder (Moffitt, Caspi, Harrington & Milne, 2002). Briefly, research

has found that onset of CD symptoms prior to the age of 10 years substantially increases risk for life-course persistent ASB (Lahey et al., 1998; Loeber et al., 2000; Masi et al., 2008). Conversely, CD symptoms that first emerge in adolescence (dubbed the "late-starter pathway") show a different set of correlates including less severe symptomatology and more transient pathology.

Simonoff et al., (1998) notes that the diversity of proposed subtypes for CD reflects broadly on the challenge of categorising hetereogeneity in DBD symptomatology. In their genetic analysis of CD subtypes these investigators utilised a subtypology proposed by Frick and colleagues (Frick et al., 1993) that parses ODD and CD symptoms according to two dimensions; overt-covert and destructive-nondestructive. These dimensions were derived from factor analytic methods applied to a meta-analysis of relevant studies. Categorising ODD and CD symptoms using this method results in four subtypes; property violations (covert, destructive), status violations (covert, nondestructive), oppositional behaviour (overt, nondestructive), aggression (overt, destructive).

Highlighting the complexity of this area of study, the application of the overt-covert distinction has also been applied specifically to the construct of aggression (Little et al., 2003; Willoughby, Kupersmidt & Bryant, 2001). In this context, *overt aggression* is direct and confrontational in nature whereas *covert aggression* is typically indirect and/or concealed from the target of aggression (Hinshaw, 2005; Klein, 2002; Ohan & Johnston, 2007; Simonoff et al., 1998). Importantly, more than any other proposed subtypology for aggression, this approach fundamentally parses the concept of aggression on the basis of *form* (Little et al., 2003). Form in this sense refers to any

set of phenotypic or topographical features (of aggression) that are observed as *co-occurring* on the basis of surface features alone, and preclude any explanation for their co-occurrence in terms of cause or effect. Other proposed subtypes for aggressive behaviours include *social* and *relational* aggression represent which are distinguished by way of communicative features—e.g., body language; (Card & Little, 2006)—in the former case, and manipulation of social allegiances to harm or damage the social integrity of another in the latter (Conway, 2005; Grotpeter & Crick, 1996).

Despite the range of proposed subtypologies for aggression, none have been formally recognised within the DSM-IV or ICD-10 (Barratt & Slaughter, 1998; Connor, 2002). From a clinical perspective, these empirical phenotypes have tended to constitute descriptive rather than explanatory constructs (Hinshaw, 2002). Specifically, these various distinctions have lacked a developmental framework to help explain the mechanisms underpinning emergence and maintenance of problematic aggressive behaviour (Kempes et al., 2005). Indeed, growing interest in the etiological role of aggression in antisocial behaviour has renewed debate for progressing a clinically meaningful subtypology that is amenable to developmental formulations.

The most frequently cited candidate taxonomy in the contemporary developmental literature is Dodge and colleagues' (Price & Dodge, 1989) proposed distinction between *reactive* (RA) and *proactive* (PA) aggression (Kempes et al., 2005). Reactive aggression refers to any aggressive behaviour enacted in direct response to provocation or threat (Vitaro, Brendgen & Tremblay, 2002). Typically, RA is viewed

as being accompanied by negative emotionality (e.g., anger) and disregulated affect (e.g., low frustration tolerance). Proactive aggression (PA), on the other hand, is defined as being instrumental or goal-oriented in nature (Card & Little, 2006). The latter subtype is most clearly distinguished from RA by the absence of both antecedent provocation and overt emotionality (Marsee & Frick, 2007). Contrasting the affective aspects of these two subtypes, RA has been referred to as hot-blooded aggression and PA as cold-blooded aggression (Card & Little, 2006).

Importantly, the RA-PA framework defines aggression in functional terms. That is, phenotypic characteristics are categorised on the basis of differential motivational underpinnings (i.e., *function*) rather than being based on a constellation of topographical features (i.e., *form)*. From this perspective, RA serves a protective function while PA is used to seek personal advantage, or advance one's social agenda (Merk et al., 2005). Functional explanations of RA and PA are particularly amenable to developmental formulations, as highlighted by Petite's (1997) description of RA as being "pushed" by intrinsic motivational factors to (i.e., to protect oneself), and PA as "pulled" by extrinsic rewards.

There has been a range of operational indices proposed to measure RA and PA (Brown, Atkins, Osborne & Milnamow, 1996; Dodge, Lochman, Harnish, Bates & Pettit, 1997; Kempes, Matthys, Maassen, van Goozen & van Engeland, 2006; Polman, de Castro, Thomaes & van Aken, 2009; Pulkkinen, 1996; e.g., Raine et al., 2006). While all are questionnaire protocols, some have been designed specifically for different informant groups such as, teachers (Dodge et al., 1997; Polman et al., 2009; Pulkkinen, 1996; Raine et al., 2006), caregivers (Kempes et al., 2006; Raine et

al., 2006), self-report (Raine et al., 2006), and peer nominations (Pulkkinen, 1996). These instruments also differ in the number of items included for indexing aggression subtypes (for a concise synopsis of approaches to measuring aggression subtypes see Kempes et al., 2005). Despite this diversity, all proposed measures comprise two subscales, one each for RA and PA. Arguably the most frequently used protocol is the 6-item Aggression Scales (Table 2.4) devised by Dodge and colleagues (e.g., Dodge et al., 1997). Characteristics of this scale are discussed in more detail in section 3.2 (p. 97).

Table 2.4

The Aggression Scales: Operational criteria for aggression subtypes

Reactive Aggression
1. Reacts angrily to accidents
2. Blames others for fights
3. When teased will strike back
Proactive Aggression
1. Threatens and bullies others
2. Uses physical force to dominate others
3. Gets others to gang up on peers

The RA-PA distinction has its roots in an earlier body of comparative research concerned with the socio-neurobiological features of animal aggression (Weinshenker & Siegel, 2002a). These early functional specifications made frequent reference to *affective* and *instrumental* subtypes of aggression (Knutson & Hynan, 1973; Krames, Milgram & Christie, 1973; Mast, Blanchard & Matsumoto, 1974)(Moyer 1968 cited in, Weinshenker & Siegel, 2002b)—a distinction largely homologous to reactive and proactive aggression. See Weinshenker and Siegel (2002a) for a detailed comparison.

Other terms used in the human developmental literature that share conceptual similarities with RA include *retaliatory*, *hostile* or *impulsive* aggression, all of which are generally considered largely synonymous with RA (Frick & Ellis, 1999)—although again, the interested reader is referred to Weinshenker and Siegel for a more detailed comparison.

Worth noting here is a difference in theoretical emphasis associated with the concept of impulsive aggression (Barratt & Felthous, 2003; Barratt & Slaughter, 1998) when compared to typical formulations of RA. Specifically, research into the former construct has raised the question of primacy, i.e., the question of whether impulsivity or aggression is the primary pathology (e.g. Barratt & Slaughter, 1998; Coccaro, Bergeman, Kavoussi & Seroczynski, 1997; Hinshaw, 2002). Nothwithstanding risk factor research which has evaluated overlap between ADHD symptomatology and RA, (e.g., Price & Dodge, 1989; Waschbusch et al., 1998), there has been surprisngly little sustained research effort to resolve this issue in the developmental litertaure. Indeed, disentangling the shared etiology of impulsivity and RA remains a significant challenge to aggression researchers (Raine et al., 2006).

Additional terms that share conceptual overlap with PA include *predatory* (Serin, Mailloux & Malcolm, 2001), *cold-blooded* and *premeditated* (Barratt, 1991) aggression as well as the construct of *callous/unemotional traits* (C/U) which shares some clinical features with PA ((Frick & Ellis, 1999)Blair). C/U behaviours represent one of three core symptom dimensions that have been associated with *psychopathy*—with the other two dimensions representing narcissism and impulsive/

conduct problems (Barry, Frick, DeShazo, Ellis & Loney, 2000; Frick, Bodin & Barry, 2000; Frick, O'Brien, Wooton & McBurnett, 1994). Psychopathy is explained in more detail further below, however C/U traits are worth briefly introducing here. This set of behaviours include muted concern for others' feelings, lack of guilt and remorse and a more general lack of emotionality (Frick et al., 2000). A recent study demonstrated that children rated high on psychopathic- and C/U-related traits were rated higher on PA scores (Frick, Cornell, Barry, Bodin & Dane, 2003). The dimension of C/U traits (and psychopathy more broadly (Frick & Ellis, 1999) share with construct of PA the theoretical absence of empathic responsivity (Blair, Colledge, Murray & Mitchell, 2001; Marsee & Frick, 2007). A number of clinical models have been proposed to account for this putative underlying deficit and some of these are explored in more detail later.

At the same time, there is a degree of overlap in symptomatology between the aggression subtypes (as indexed by the Aggression Scales) and the antisocial DBDs in particular. For example, both RA and ODD include symptom criteria that indexes maladaptive responding to accidents or mistakes and both behaviour dimensions emphasise anger in interpersonal interactions. There is also operational overlap between PA and CD. In fact, these latter two behaviour dimensions share a similar criterion; "Threatens and bullies others" for PA and "Bullies, threatens, or intimidates others" for CD. Given that the PA scale comprises a mere three items, this likely represents a non-trivial degree of operational overlap. This apparent redundancy across symptom sets that has prompted some to question the clinical utility of the aggression subtypes (Waschbusch et al., 1998). However, to reiterate, a core difference between the RA-PA framework and the DBDs, is that the former

taxonomy priorities motivational features of key maladaptive behaviour, while the latter prevailing set of clinical entities reflect an attempt to classify heterotypic topographical features of disruptive, antisocial and aggressive behaviour (Kempes et al., 2005).

Focusing on the motivational underpinnings of aggression has rendered the RA-PA framework particularly amenable to clinical research precisely because it appeals directly to empirically-established developmental processes to explain the etiology of divergent maladaptive aggressive behaviours (Baker, Raine, Liu & Jacobson, 2008; Fite, Stoppelbein, Greening & Gaertner, 2009; Kempes et al., 2005; Polman et al., 2009; Scarpa, Tanaka & Chiara Haden, 2008). Certainly, within the clinical and developmental literatures, the RA-PA distinction has led to the exploration of a diverse range of proposed aggressogenic risk mechanisms (Baker et al., 2008; Blair, 2009; e.g., Dodge, 1991; Fite, Colder, Lochman & Wells, 2008; Kempes, de Vries, Matthys, van Engeland & van Hooff, 2008; Scarpa et al., 2008). Importantly, these mechanistic accounts have allowed researchers to go beyond simple pathway models of aggression to postulate co-relations between putative psychosocial, cognitive and biogenic pathogens (Brendgen et al., 2006; Kempes et al., 2005). At the same time, these models have begun to shed empirical light on behavioural and etiological hetereogeneity in disruptive behaviour disorders (Polman et al., 2009). This emerging focus on manifold mechanisms echoes earlier calls to integrate a wider range of paradigmatic perspectives on multiple risk pathways associated with aggression including increased focus on the genetics of divergent aggressogenic mechansims to complement and broaden the existing evidence base (Hinshaw, 2002).

Consistent with these calls, research concerning the psychosocial and socio-cognitive etiology of RA and PA has recently been augmented by a number of seminal studies assessing the relative role of environmental and genetic factors in the emergence of RA and PA (Baker, Jacobson, Raine, Lozano & Bezdjian, 2007; Baker et al., 2008; Brendgen et al., 2006). The present thesis attempts to extend this work by examining familial (and non-familial) contributions to continuity across key periods in development with particular reference to diagnostic disruptive behavioural symptomtatology. However, before considering the current research agenda in more detail, extant literature examining the etiology of RA and PA is briefly reviewed.

2.2 Etiology of Reactive and Proactive Aggression

2.2.1 Socio-Cognitive Model of Reactive and Proactive Aggression

In his seminal etiological formulation of RA and PA, Dodge (1991) appealed to the social learning perspectives of Berkowitz (Berkowitz, 1993; Berkowitz & Buck, 1967) and Bandura (Bandura, 1973; Bandura, 1983; Bandura, Ross & Ross, 1961) in proposing differential socio-cognitive mechanisms underpinning aggression subtypes. Specifically, Dodge refers to Berkowitz's frustration-aggression hypothesis in explaining the emergence of RA in childhood. According to this model, RA constitutes a disregulated defensive response to perceived threat. Dodge's formulation identifies coercive and/or harsh parenting practices as formative in the development of problematic RA due to its association with (child-perceived) threatening behaviour (Vitaro et al., 2002; Vitaro, Gendreau, Tremblay & Oligny, 1998). At the socio-cognitive level, persistent exposure to threat is thought to ultimately compromise the encoding of relevant social information by increasing the

likelihood that ambiguous social cues are misinterpreted as aggressive or hostile (Dodge & Schwartz, 1997). Crick and Dodge (1996) called this putative social information-processing deficit a *hostile attribution bias* and posited that the resulting hypervigilence to threat can maintain RA behaviour across multiple social settings.

Conversely, Dodge and colleagues suggest PA is the developmental product of imitation learning, as described in Bandura's preeminant work (Schwartz et al., 1998), in which aggression is modeled as a means to achieve desired social goals. In Dodge's socio-cognitive specification of RA-PA, this modeling is postulated to contribute to late-stage (i.e., controlled) social information-processing deficits reflecting positive outcomes expectancies associated with PA (Card & Little, 2006; Crick & Dodge, 1996; Dodge & Schwartz, 1997; Schwartz et al., 1998). As the term suggests, positive outcome expectancies operate as a maintaining mechanism potentially eliciting social interactions that positively reinforces PA behaviour.

Dodge and colleagues' socio-cognitive model of RA and PA has received considerable research attention over the past two decades. Investigations of socio-cognitive deficits and associated psychosocial correlates have yielded generally supportive, albeit, mixed results (Merk et al., 2005). Although a detailed discussion of findings is beyond the scope of this text, a brief review of this literature is provided below.

Evidence of differential socio-cognitive correlates derive from tests of Dodge and colleague's social information processing model of RA-PA. Briefly, studies suggest children evidencing predominantly RA behaviours are more likely than their non PA-

dominant counterparts, to interpret ambiguous social cues (e.g., accidently being pushed in a queue) as hostile (Crick & Dodge, 1996; Dodge & Coie, 1987; Schwartz et al., 1998). Children with a PA-dominant behavioural style, on the other hand, are more likely than their non PA-dominant counterparts to show positive outcome expectancies for the use of aggression (Dodge & Schwartz, 1997; Dodge et al., 1997; Schwartz et al., 1998; Smithmyer, Hubbard & Simons, 2000) and to evidence unrealistically positive self-evaluations of social competence that are disputed by peers (Orobio de Castro, Brendgen, Van Boxtel, Vitaro & Schaepers, 2007). However, see Kempes et al. (2005) for a failure to replicate Dodge et al.'s (1997) finding on positive outcome expectancies.

2.2.2 Psychosocial Features of Reactive and Proactive Aggression

The broad assertion of subtype-specific etiologies is supported by research into concurrent correlates of psychosocial adjustment (Card & Little, 2006; Raine et al., 2006). A number of indices of maladjustment have been identified. For example, children exhibiting predominantly RA behaviours have been shown to evidence deficits in social problem solving to a greater extent than their PA-dominant or pervasively aggressive peers (Dodge et al., 1997; Schwartz et al., 1998; Vitaro et al., 1998). In this context, the term *pervasively aggressive* refers to those who show high levels of both RA and PA. Moreover, RA-dominant children show elevated risk of exhibiting anger when frustrated (Hubbard et al., 2002; Mc Auliffe, Hubbard, Rubin, Morrow & Dearing, 2006), as well as heightened risk of peer rejection (Polman, Orobio de Castro, Koops, van Boxtel & Merk, 2007; Poulin & Boivin, 2000). Similarly, RA deficits appear to extend to difficulties coping with teasing and failure and negotiating compromise (Day, Bream & Paul, 1992; Hubbard et al., 2002).

Conversely, children who exhibit a PA-dominant behavioural style tend to associate with children who share similar proactive aggressive tendencies (Poulin & Boivin, 2000). Moreover, PA is often associated with higher overall levels of aggression (Vitaro et al., 2002; e.g., Vitaro et al., 1998) and with psychopathic-type traits (Beatty, Dodge, Dodge, White & Panksepp, 1982; Blair, 2003; Blair, Peschardt, Budhani, Mitchell & Pine, 2006; Mitchell et al., 2006; Nouvion, Cherek, Lane, Tcheremissine & Lieving, 2007). A number of studies have provided an empirical link between the divergent social problems experienced by RA versus PA and socio-cognitive deficits posited by Dodge (Crick & Dodge, 1996; Dodge & Coie, 1987; Orobio de Castro et al., 2007; Schwartz et al., 1998; Smithmyer et al., 2000).

In their recent meta-analytic review of 42 studies involving over 20,000 children, Card and Little (2006) examined eight indices of maladjustment (i.e., internalising problems, emotion disregulation and ADHD-type symptoms, delinquency, low prosocial behaviour, low social preference and peer acceptance, higher peer rejection, and victimisation) and found that, after controlling for PA, RA alone was independently and positively correlated with all factors included in their review. In contrast, PA showed a positive independent association with only two measures (i.e., delinquency and peer rejection). Consistent with other reports (Price & Dodge, 1989; Salmivalli & Helteenvuori, 2007), PA was significantly related to lower levels of peer rejection.

2.2.3 Developmental Correlates of Reactive and Proactive Aggression

As noted above, Dodge's model postulates relatively discrete developmental trajectories for RA and PA. However, retrospective and longitudinal analyses have

yielded equivocal results in regards to psychosocial precursors and long-term outcomes associated with subtypes.

In their seminal retrospective analysis of RA and PA in a population sample of school aged children, Dodge et al. (1997) found that RA measured in Grade 3 was uniquely associated with an early history of physical abuse (including harsh discipline). RA was also linked to early onset of behaviour problems. By contrast, children with high levels of PA did not differ in relation to their non-aggressive peers on any of the measures of early experience, however the data suggested no history of parental abuse in this group.

In a second accompanying study involving older children (mean age of 13 years) with a history of either reactively (RV) or proactively (PV) violent behaviour, Dodge et al. (1997) found the former group initiated problematic behaviour at around 4.5 years—two years earlier than the PV group. This revealed consistency across samples concerning early onset of problem behaviours in RA-dominant groups. At the same time, early family problems and trauma did not distinguish RV and PV groups. It is worth noting that the retrospective reporting of behaviours, along with the more salient nature of reactive aggression, warrant a degree of caution in interpreting results (Vitaro & Brendgen, 2005).

Increasingly, the study of RA and PA has been informed by longitudinal research. A recent prospective study by Salmivalli and Helteenvuori (2007) found that in a population sample of 10-13 year old children, RA but not PA predicted future victimisation in boys (but not girls) measured in three Waves over a 12 month

period. Moreover, RA predicted future PA for both boys and girls, however the inverse was not true for PA. Interestingly, like results reported by Dodge et al. (1997) in a similar aged-sample, neither RA nor PA was differentiated on retrospective measures of earlier exposure to adverse psychosocial factors, i.e., harsh parenting and trauma in the case of Dodge et al., and peer victimisation in Salmivalli and Helteenvuori's study. In another investigation, McAuliffe et al., (2006) assessed 57 second-grade children over a period of 12 months and found that neither subtype predicted the other, from the first to the second wave. Notably, this study used residualised measures of RA and PA which involves deriving more theoretically "pure" indices of each subtype by statistically partialling out the influence of the other subtype - a technique which some have queried on statistical grounds (e.g., Miller & Lynam, 2006; Raine et al., 2006). For example, Raine et al. (2006) point out that this technique increases the error variance and reduces the reliability of the measures involved. To add to the inconsistency in the literature, other longitudinal studies have indicated prospective relationships between RA and PA that are contrary to those discussed above. This research is discussed in more detail below.

The above retrospective and prospective studies have generally identified RA as being most strongly associated with long-term adverse social outcomes. However, there are a number of well-cited studies that have found a quite different pattern of results. Notably, in a large-scale prospective study, Pulkkinen et al. (1996) reported that PA, determined at 14, was associated with externalising problems and criminality at 27 years. More specifically, at each of the three data collection Waves, adolescents who exhibited higher levels of PA showed poorer adjustment than peers with high levels of RA. At the same time, compared with the RA group, participants

rated highly on PA evidenced heightened levels of aggression measured earlier at the age of 8. Thus, in this latter study, PA was consistently associated with greater conduct problems, from childhood into adolescence. Pulkkinen et al.'s (1996) results were generally replicated by those of Vitario et al. (1998) who reported that in a community sample of at-risk children, PA and not RA at age 12 was strongly predictive of later Conduct Disorder, measured in mid-adolescence (average age of 14.9 years). These authors observe that, "the 'predatory' nature of CD problems may explain why proactive rather than reactive aggression was a significant predictor" (Vitaro et al., 1998, p. 383). These investigators also found that PA measured at the first wave predicted later ODD.

Unlike Dodge et al.'s (1997) findings which suggest children rated highly on both RA and PA (i.e., pervasively aggressive children) are at greatest risk of global behavioural problems (in Grade 3), Vitaro et al. (1998) found that pervasively aggressive adolescents were less at risk of delinquency than their 'pure' PA-rated peers. They speculated that this might be because children showing high levels of RA may be at increased risk of experiencing high levels of concurrent anxiety, regardless of whether or not they also evidenced high levels of PA. Thus, RA was found to mediate the link between PA and delinquency such that higher levels of RA was associated with lower levels of delinquency. However, RA did not mediate the link between PA and the antisocial behaviour disorders (i.e., ODD and CD).

In a later study, Vitaro et al. (2002) found all three aggressive subgroups evidenced higher scores than controls on early maternal ratings of physical aggression. However, contrary to Dodge et al. (1997), PA (regardless of concomitant RA) was

associated with higher levels of aggression than RA. By age 13 years, only PA (and not RA) group membership predicted higher levels of aggression than those exhibited by control children. The authors concluded that PA-rated children were most at risk for delinquency. Consistent with earlier findings (i.e., Vitario et al., 1998), the authors reported that RA-only children evidenced higher levels of anxiety at age 6 years. The authors interpreted this finding as supporting Vitario et al.'s (1998) earlier speculation that anxiety may work to inhibit delinquency. The 2002 study suggests inhibitive effects of anxiety may extend to aggression.

At the same time, strong links between anxiety, ADHD and conduct problems (Baldwin & Dadds, 2008) raise the possibility that the relationship between RA and anxiety implicates comorbid ADHD symptomatology. Determining the role of anxiety in either increased or reduced delinquency requires distinguishing between affect-regulation versus fear-based aspects of anxiety (Dadds & Salmon, 2003). In this context, affect-regulation refers to the capacity to modulate negative emotionality such as worry and sadness while fear-based aspects of anxiety refer to basic autonomic responsivity to fear-inducing situations. It is possible that the former may help explain desistance in long-term antisocial pathways (Dadds & Salmon, 2003). Conversely, a lack of fear-responsivity may be implicated in persistence of aggressive behaviour (Dadds & Salmon, 2003; Frick & Ellis, 1999).

More recently, Raine et al. (2006) reported results from a longitudinal study in which they assessed developmental precursors in a community sample of young males at age 7 in order to elucidate relationships with RA and PA at age 16. A range of psychosocial (e.g., poor peer relationships, permissive parenting, academic

motivation, parental education, parental substance use, SES status), personality (e.g., psychopathy, stimulation-seeking) as well as psychological and behavioural (e.g., anxiety, impulsivity, Hyperactive-Inattentive problems, initiation of fights, strong-arm tactics, delinquency, serious violent offending) measures were collected at the first and second waves. RA and PA were evaluated at the second wave using a novel 26-item questionnaire allowing for a retrospective analysis of aggression subtypes. PA (at age 16), and not RA was associated with greater maladjustment at the first wave. Specifically, PA measured at the second wave was uniquely related to initiation of fights, excessive fighting, strong-arm tactics, delinquency, poor school motivation, poor peer relationships, single parent status, psychosocial adversity, parental substance abuse, and hyperactivity at the earlier Wave.

Conversely, RA (at age 16) was not independently associated with any of the earlier measures apart from excessive fighting. These findings are at odds with earlier retrospective studies that found RA was associated with earlier behaviour difficulties (Dodge et al., 1997; Salmivalli & Helteenvuori, 2007).

PA at age 16 was uniquely associated with a number of concurrent features including psychopathic traits, blunted affect, stimulation seeking, delinquency and serious violent offending while RA was independently correlated with concurrent hostility, social anxiety, lack of close friends, stimulation seeking and impulsivity. It is worth noting that, the unique relationship between RA and impulsivity concurs with Dodge et al. (1997).

Indeed, it turns out that results linking aggression subtypes with attentional and

impulse problems are more consistent across studies than many of the broader ASB-related outcomes highlighted above. For example, Vitario et al. (2002) identified distinct socio-emotional antecedents and consequents associated with subtype group membership measured annually from age 10 to 13 years in a population sample, revealing that at age 13, children rated as high in RA behaviours, regardless of whether or not they also exhibited PA, were rated (at age 6 years) by their mothers as more reactive and inattentive than their PA-only and non-aggressive peers. Antecedent correlates were obtained from data collected at an earlier Wave when participants were aged 6 years.

These results corroborate the earlier findings of Dodge et al. (1997) that suggest "RA children are concurrently more inattentive and impulsive than proactive or non-aggressive children" (Vitaro et al., 2002, p. 502). However, in contrast to Dodge et al., Vitario et al., also found that those children showing RA-only scored more highly than their RA/PA peers on both these measures, suggesting a potential moderating effect of PA (Vitaro et al., 2002).

2.2.4 Risk Pathway Formulations for Reactive and Proactive Aggression

At first blush, the original findings of Dodge et al. (1997), as well as those reported by Salmivalli and Helteenvuori (2007), appear at odds with a number of recent etiological investigations (Pulkkinen, 1996; Raine et al., 2006; Vitaro et al., 2002; i.e., Vitaro et al., 1998). That is, the former body of work implicates RA as a primary risk indicator for persistant ASB while the latter suggests that PA is more strongly associated with antisocial outcomes.

One aspect differentiating longitudinal studies that implicate RA versus PA as the predominant risk factor for ASB, is that the former body of research has tended to involve the assessment of pre-adolescent data. By contrast, research that has found stronger associations between PA and problem behaviour has generally used follow-up data collected relatively later in development, i.e., mid-adolescence (e.g., Vitaro et al., 2002), late-adolescence (e.g., Raine et al., 2006) and adulthood (e.g., Pulkkinen, 1996). Interestingly, in their study of psychiatrically referred children ranging in age from 5 to 18 years, Connor et al. (2004) found that age was significantly negatively correlated with RA which they speculated reflects increases in inhibitory control over the course of development (see also Connor, 2002; Kempes et al., 2008; Vitaro et al., 2006). From a longitudinal perspective, this finding leads to the relatively uncontroversial prediction of declining prognostic power associated with RA over the course of development.

Taken together, it is empirically feasible that RA has greater prognostic value than does PA at earlier stages in the developmental spectrum, with this pattern potentially reversing from mid-adolescence onwards as planned and goal-oriented aggression becomes an increasingly salient marker of adverse long-term outcomes (Connor, 2002; Connor et al., 2004; Lahey et al., 1994). Vitaro and Brendgen extend this speculative formulation in proposing their recent integrative developmental model of RA and PA (Vitaro & Brendgen, 2005).

These latter authors have proposed a sequential model of RA and PA (Vitaro & Brendgen, 2005). Consistent with reports that RA typically emerges prior to PA in

development (e.g., Dodge et al., 1997; Vitaro et al., 2006), the authors posit the former subtype as developmentally precursory to the latter subtype. They go on to cite a range of evidence supporting suggestions that RA is fundamentally influenced by child factors such as temperament and neurobiological factors. PA, on the other hand, is posited as being the product of positively reinforced (i.e., operantly conditioned) RA behaviour. In other words, this model suggests that RA comes to be used used instrumentally in cases where RA has lead to net social gains for the aggressor. In this sense, RA becomes converted to PA. This model neatly resolves much of the apparent ambiguity of the RA-PA distinction which has lead to some previous criticisms of the distinction (e.g., Bushman & Anderson, 2001).

It is presumed here the sequential model implies RA behaviour which persists in the absence of concomitant PA is predominantly maintained by endogenous characteristics rather than external contingencies. The authors are more explicit in speculating that complete transition from RA to PA (i.e., where RA ceases to be an observable facet of the phenotype) can occur due to increasingly autonomous or permissive environments, or in cases where the aggressor increasingly possesses personal characteristics (e.g., physical stature) that reduce the need for retaliation, or likelihood of goal-frustration. Vitaro and Brendgen (2005) note that their formulation of pure PA does not suggest *propensity* for RA desists, it merely posits reduced exposure to environmental conditions likely to elicit it.

For the benefit of clarity, it is noted that the current thesis takes a multifinality view of aggression subtypes. While considerable interest is placed here in the sequential hypothesis as one pathway to PA, later sections presented within this chapter

highlight a broader range of etiological contributors to PA. The clinical usefulness of any discrete pathway model is ultimately determined empirically, but invariably single pathway explanations provide only a partial etiological picture.

2.2.5 Summary of Longitudinal Perspectives on Aggression Subtypes

In summary, evidence for differential etiologies reveals inconsistencies in the RA and PA literature and makes definitive conclusions premature. Certainly, a majority of studies have focused on concurrent factors and more longitudinal data are needed (Vitaro & Brendgen, 2005). As the previous section highlights, the goal of clarifying discrete versus shared trajectories raises complex questions about transmission of risk. Findings from risk factor (i.e., correlational) studies make it difficult to disentangle (1) causal risk factors associated with RA and PA (i.e., parameters which affect change in outcomes), let alone (2) the means by which they create risk (Hinshaw, 2002; Rutter, 2001; Rutter, 2006).

According to Hinshaw (2002), achieving such goals requires two things: Firstly, it is imperative that research be guided by "conceptual frameworks for understanding the process of transmitting risk processes". On this point, Hinshaw notes that some of the most convincing evidence concerning modes of transmission is derived from the use of 'experiments of nature' such as twin (i.e., behavioural genetic) studies. Secondly, "elaboration of intervening variables and mechanisms that provide the explanation of how and why risk variables exert their effects" (p.436) is equally essential. As an example of the complexities involved in this latter task, Hinshaw (2002) highlights the elusive but ultimately necessary goal of reconciling the various levels of

explanation applicable for the RA versus PA distinction, including their different etiological, psychobiological and psychosocial features.

To date, the majority of RA-PA research has focused on putative psychosocial and socio-cognitive mechanisms underlying the subtypes. However, increasingly, investigators are hypothesising and testing broader, and arguably more fundamental, developmental pathways. For example, two recent studies have provided the first empirical analysis of the role of genes in RA and PA (Baker et al., 2008; Brendgen et al., 2006). These studies are reviewed further below. Preceding this review is a brief introduction to the behavioural genetics paradigm which provides an overview of relevant concepts and terminology.

2.2.6 Behavioural Genetics of Reactive and Proactive Aggression

2.2.6.1 The Behavioural Genetics Paradigm

The field of quantitative behavioural genetics (BG) is concerned with estimating the relative contribution of genetic and environmental factors to individual differences in behaviour (Levy & Hay, 2001). The ubiquitous classical twin design compares behavioural similarity in monozygotic (i.e., identical) and dizygotic (i.e., fraternal) twins with reference to the fact that monozygotic twins share 100% of their segregating genes while dizygotic twins and non-twin siblings share, on average, only 50% (Hay, 1985; Rutter, 2006). This observation leads to the fundamental tenet of the BG model. That is, when environmental conditions are equal, greater behavioural similarity between monozygotic (MZ) twins compared to dizygotic (DZ) twins is attributed to the effect of genes.

Genetic influences are typically categorised as being additive or non-additive in nature. Additive genetic influences are those in which the overall effect of the putative gene set is equivalent to the sum of the effects of each constituent gene considered independently. *Non-additive* genetic effects refer to the synergistic influence of multiple genes which can occur by either dominance or epistasis (Neale & Cardon, 1992). Synergistic influences reflect effects that deviate from the simple summed impact of the relevant constituent genes. Dominance refers to the interaction of alleles (i.e., the two halves of a gene) at the same locus on the chromosone while epistasis occurs when the effect of a gene at one locus depends on "which genotype is expressed at a different locus" (Neale & Cardon, 1992, p. 10).

The BG approach generally assumes additive effects where the MZ correlation (i.e., similarity) does not exceed twice the DZ correlation on the trait(s) under investigation (Baker et al., 2008). Conversely, MZ correlations that do exceed half the DZ correlations suggests the possible presence of non-additive effects.

As above, a core assumption of the BG model is that environmental factors do not exert any *differential* influence on MZ, DZ or non-twin siblings. This has been called the equal environments assumption (EEA, |Hettema 1995itchell 2007). Trait-relevant environments have been shown to be appropriately correlated for a wide range of psychiatric syndromes, confirming the general validity of this broad assumption (e.g., Hettema, Neale & Kendler, 1995; Kendler, Neale, Kessler, Heath & Eaves, 1993). While there are many ways to test the EEA, a recent study by Mitchell et al. (2007) validates use of model invariance in determining whether this asumption is

either met or violated. The model invariance method is explained in more detail in Chapter 4.

EEA does not contradict the profound impact of environment on behaviour. It merely implies that whatever influence environmental factors exert, the impact is equivalent for both twins. Environmental influences are typically partitioned into *shared* and *non-shared* effects. The former represents exogenous influences that operate to make twin pairs more alike, regardless of zygosity (Rutter, 2006). As previously mentioned, these shared influences are often considered to reflect the effects of factors such as parenting practices. Conversely, non-shared environmental influences represent exogenous factors that operate to make twins less alike, regardless of zygosity. Rutter (2006, p. 108) makes the useful clarification that *shared* and *non-shared* effects "concern inferences regarding the consequences of environmental influences and not the nature of environments as observed". It is also useful to adopt Rutter's terminology to emphaise that the equal environments assumption simply refers to the expectation that whatever the consequences of environment influences, the nature of environments observed are assumed to be the same for both MZ and DZ twins.

A final word in regards to terminology related to the BG literature. It is common for researchers to use a short-hand form of abbreviation for each of main sources of variance mentioned above. The letter '**A**' is commonly used to refer to additive genetic effects, '**C**' for shared environmental effects (also frequently called *shared environmental* effects) and, '**E**' for non-shared environmental influences. These abbreviations are adopted in the current text.

2.2.6.1.1 The role of adoption studies in behaviour genetics

It is worth emphasising that the methodology described above is based predominantly on the twin design. However, adoption studies have also made an important contribution to the field of behaviour genetics. One of the key strengths of this approach is the natural separation of genetic factors from environmental factors. This separation occurs where biological parents have had very little or no role in rearing the adopted-out child, while the adopting parents who provide this care have no genetic relationship with the child (Rutter, 2007). This effectively removes the problem of *passive gene-environment correlations*—a concept which typically refers to the fact that parents with a genetic vulnerability for pathology often provide corresponding risky environments.

Importantly, adoptee designs have a number of limitations. Particularly problematic for studies of aggression are the potential effects of assortive mating (Rutter, 2007). Assortive mating occurs when an adult selects a partner who shares trait-relevant similarities in terms of their genotype or phenotype (i.e., antisocial behaviour). Assortive mating in biological parents can lead to a "double dose" of genetic effects in the adoptee design. This is a problem for aggression research because of the relatively high prevalence of assortive mating associated with this trait (Rutter, 2007).

2.2.6.1.2 Gene-environment interplay in behaviour genetics

The issue of assortative mating raises the broader issue of interplay between genes and environment relating specifically to gene-environment (GE) correlations and gene-environment (GxE) interactions.

GE correlations occur when a person's genotype influences the types of environments in which they interact—and, in turn, increases the likelihood that preferred (i.e., genotype-consistent) environments are sought out and/or maintained (Caspi & Moffitt, 2006; Moffitt, 2005b; Moffitt, Caspi & Rutter, 2006; Rutter, Moffitt & Caspi, 2006).

GxE interactions occur when the ultimate effects of environmental conditions depend on the underlying genotype, or vice versa (Rutter, 2006). For example, Sheese and colleagues (Sheese, Voelker, Rothbart & Posner, 2007) recently demonstrated that the presence of the DRD4 7-repeat allele (a specific variant of genetic structure) in 18-21 month old infants mediated the relationship between the infant's temperament (i.e., indexed as increased sensation seeking behaviour) and quality of parental interaction (i.e., indexed by a range of observed behaviours such as "supportive presence" and "stimulation of cognitive development"). Specifically, levels of sensation seeking were inversely proportional to quality of parental interaction for infants with this allelic combination. Conversely, the absence of this allele was associated with an absence of any relationship between these two measured variables.

In the behavioural genetic twin study, gene-environment interactions and gene-environment correlations that operate to make twins more alike are not measured directly but instead are typically indexed by the heritability coefficient—thus inflating genetic estimates while underestimating the effects of shared environment (Moffitt, 2005a; Moffitt, 2005b). Indeed, Moffitt (2005a) has suggested this fact contributes to low estimates of environmental effects in studies of ASB and

aggression.

Gene-environment interactions have become increasingly of interest to researchers and are likely to have considerable relevance to the genetic study of aggression (Moffitt, 2005b). A detailed discussion of this phenomenon is reserved for a later chapter.

2.2.6.2 The Genetics of reactive aggression and proactive aggression

A considerable body of evidence attests to the influence of genetic factors on individual differences in ASB (Goldsmith & Gottesman, 1995; Rhee & Waldman, 2002; Rutter, 2006) and aggression (Miles & Carey, 1997). Behavioural genetic research has consistently revealed moderate to high heritability estimates for aggression. Most notably, in their widely cited meta-analytic review of the genetic architecture of aggression, Miles and Carey (1997) concluded that up to half the phenotypic variation in aggression is attributable to genetic influences. At the same time, these authors noted that there exists considerable variation in the magnitude of estimates across genetic studies of aggression. A range of moderating factors potentially impacting on reported estimates were assessed, including age and sex of participants. Significant, albeit weak, effects were found for both these latter variables. More specifically, heritability estimates were (marginally) higher for males, while studies involving younger participants were associated with lower heritability estimates. Both these sets of findings are discussed in more detail in a later section.

Another factor potentially contributing to inconsistency in heritability estimates

across studies, is variation in the conceptualisation/operationalisation of aggression (Coccaro et al., 1997; Miles & Carey, 1997). In concluding their review, Miles and Carey suggested more studies are required to investigate differential liability associated with distinct subtypes of aggression. This agenda has recently been advanced by Brendgen et al. (2006) as well as Baker et al. (2008).

In their seminal analysis of the architecture of RA and PA in childhood, Brendgen et al. (2006) assessed both aggression subtypes in a community sample of 6-year-old twins (n=172) via a standard teacher-report measure. Separate teacher ratings were obtained for each twin pair (i.e., twin and co-twin attended separate classrooms) as a result of the fact that Canadian government policy requires twins to be taught in separate classrooms. The investigators reported that genes play a role in the emergence of both RA and PA.

These authors included a generic measure of physical aggression in their analyses to complement the relatively standard measures of RA and PA. This trivariate design allowed the investigators to effectively control for the common *form* of aggression shared by both RA and PA (i.e., physical aggression) as recommended by Little et al. (Little et al., 2003). In this way, Brendgen et al.'s study was theoretically able to disentangle influences affecting form versus function (see also Card & Little, 2006).

The analyses reported by Brendgen et al. suggested that genetic factors account for 39% and 41% of the variance in RA and PA respectively. Shared environmental effects were found to lack explanatory power in initial univariate modeling and were subsequently omitted from the multivariate model. The authors noted that the

apparent lack of shared environmental influences on both RA and PA is somewhat at odds with the seminal etiological model developed by Dodge and colleagues. Indeed, the key aggressogenic factors posited by Dodge (1991) in his etiological model consitute parental rearing practices, and it is precisely these kinds of parenting practices which are commonly considered to exert shared environmental effects (e.g., Rutter 2006). At the same time, non-shared environmental effects represented the largest single source of variance, accounting for 61% and 59% of respectively.

Results also confirmed that a substantial proportion (76%) of the variance accounted for by genetic factors underpinned the overlap between RA and PA. In other words, a majority of genetic influences responsible for RA, were also found to be responsible for PA. Importantly, this common variance overlapped entirely with variance associated with the measure of overt physical aggression. These findings speak directly to the underlying common form hypothesis (i.e., Little et al., 2003; Polman et al., 2006) in suggesting that the frequently reported phenotypic overlap between RA and PA is the result of a common underlying genetic liability for (undifferentiated) physical aggression. Subsequently, only a small proportion of genetic influences contributed to divergence in functional subtypes. More specifically, around 11% of variance in RA was attributable to trait-specific genetic effects, while 7% of variance in PA was accounted for by trait-specific genetic influences. Thus, despite the substantial genetic influences identified, these results indicate genes play only a marginal role in shaping different functional subtypes of aggression.

Conversely, 42% of variance in RA, and 51% of variance in PA was attributable to

trait-specific non-shared environmental influences, with only a small proportion of overall non-shared environmental effects (6%) common to both subtypes. This led Brendgen and colleagues to conclude that non-shared environmental effects may play a formative role in differentially shaping RA and PA behaviour. In considering these results, the authors point out the conceptual link between peer group influence and non-shared environmental effects in speculating that social environments outside the home may be more important in contributing to the differentiation of subtypes than previously recognised. In particular, they note that the learning mechanisms proposed by Dodge (1991) would be expected to operate in peer groups as they do in family environments. While this is certainly a viable proposition, there are a number of issues worth considering in relation to the specific genetic and environmental architecture architecture as reported by Brendgen—particularly as it relates to the lack of **C** (i.e., shared environmental) effects found in this study.

Certainly, a number of previous genetic studies have reported an absence of shared environmental effects on overt forms of aggression. Edelbrock, Rende, Plomin and Thompson (1995) asked 181 parents to rate their twins, aged 7-12 years, on both overt aggression and non-aggressive delinquent behaviour using the Child Behavior Checklist (CBCL). Substantial genetic influences, but negligible shared environmental effects, were found for ratings on aggression. Non-aggressive delinquent behaviours, however, were associated with both genetic and shared environmental effects. This general pattern of results has been replicated across a number of subsequent studies (Button, Scourfield, Martin & McGuffin, 2004; Tackett, Krueger, Iacono & McGue, 2005; Tuvblad, Eley & Lichtenstein, 2005) and has led to the formulation of a dual pathway model distincguishing aggression and

non-aggressive delinquency are distinguishable on the basis of these etiological features (Tuvblad et al., 2005). It is interesting to note that these studies incorporate a range of measures for ASB including the Instrument of Antisocial Behaviour (Olweus, 1989, cited and utilised in Button et al., 2004), the CBCL (Achenbach, 1991; used in Tuvblad et al., 2005) and DSM criteria for CD sub-grouped according to the two key dimensions of interest (used in Tackett et al., 2005).

Nonetheless, despite this convergence across studies, a number of methodological factors can impact on the capacity to detect **C** effects (Neale & Cardon, 1992; Rutter, 2006). Of particular note, Rutter (2006) emphasises the large number of participants required to obtain adequate statistical power to effectively elucidate **C** effects. Indeed, model fitting procedures, which are predominantly informed by the principal of parsimony, often result in the dropping of the **C** component due to large error estimates (i.e., wide confidence intervals) associated with this latent variable—intervals that very often include 0 within their bounds (Rutter, 2006). For example, despite a substantial sample size of 1226 participants (average age of 8.46), Tuvblad found very low reliability for **C** effects—with (95%) confidence intervals that ranged between 0.00 to over 0.60 for both males and females. This lack of reliability was reflected in the fact that dropping **C** effects from the full model (i.e., including all variance components) did not lead to a significant reduction in model fit. Although Brendgen et al. (2006) did not report confidence intervals for their univariate models, they nonetheless caution that their findings were based on a modest sample size—an observation that underscores the value of replication in this area of research.

Another potential limitation associated the Brendgen et al. (2006) study is the

separate teacher rater design. As discussed in more detail in the next chapter, the literature suggests that separate teacher ratings are prone to a range of biases (e.g., Hartman, Rhee, Willcutt & Pennington, 2007; Vierikko, Pulkkinen, Kaprio & Rose, 2004) including reduced twin correlations (Simonoff et al., 1998; Towers et al., 2000; Vierikko et al., 2004). Rater bias refers to the systematic under- or over-estimations of twin similarity and occurs when rater perceptions of child behaviour are based on anything other than population levels, i.e., base rates, of the behaviour being measured (Simonoff et al., 1998; Simonoff et al., 1995). In a recent study by Hartman et al. (2007), separate teacher ratings of ADHD in a community sample of children were found to be more prone to rater bias than other informant sources (including single teacher, and caregiver ratings). In this latter study, rater bias was associated with inflated **C** effects due to general over-estimates of behavioural similarity. In fact, both separate teacher and caregiver ratings were associated with this bias, however it was the former informant group who showed the most pronounced bias. While Brendgen et al. found no evidence of **C** effects, the broader literature nonetheless indicates caution when interpreting separate teacher ratings.

More recently, in a large-scale multi-informant study involving 1, 211 twins (and 18 triplets) aged 10 years, Baker et al. (2008) reported substantial genetic effects for both RA and PA for teacher-rated aggression (accounting for 20% and 45% of variance respectively) and caregiver-rated aggression (26% and 32% respectively). However, unlike Brendgen et al., these authors demonstrated significant shared environmental effects for both RA and PA in both teacher (43% and 14% respectively) and caregiver (27% and 21% respectively) rated aggression. The possibility that higher **C** effects derived from teacher ratings reflected rater bias was

considered. However, controlling for this possibility, by accounting for bias effects in the relevant genetic models, Baker et al. (2008) reported that the presence of non-biased **C** effects remained significant, accounting for 30% of variance in RA (C=0.30, 95% CI=0.11, 0.46), and 14% of variance in PA (C=0.14, 95% CI=0.02, 0.30). This brought the estimates more closely into line with estimates derived from caregiver ratings.

Baker et al. (2008) note that it is not possible to methodologically or statistically disentangle true **C** effects from rater bias in single caregiver-rated data and this latter analysis was not undertaken for this informant group (Baker et al., 2008). The invariable confound between single caregiver rater bias and true **C** effects occurs because, unlike teachers, caregivers contribute exhaustively to both. This confound can be partly circumvented by inclusion of a two-caregiver rater model (i.e, including both maternal and paternal ratings for each twin pair) which introduces the requisite variance for which to make intra-familial comparisons (see also Rutter, 2006; Simonoff et al., 1995). Nonetheless, Baker et al. concluded that **C** effects are important in RA and PA, although their impact is of greater magnitude for the former subtype compared to the latter.

Broadly in agreement with the findings of Brendgen et al., Baker and colleagues found the high phenotypic correlation between RA and PA was largely accounted for by genes. This led the investigators to conclude that genes play only a marginal role in the developmental differentiation of subtypes although they did note that genetic influences appear to be more important in the development of PA relative to RA. At the same time, a sizable proportion of overlap between subtypes was attributable to

C effects (Baker et al., 2008). Like, Brendgen et al., this is generally at odds with the proposition that family factors operate to differentially shape aggression subtypes (i.e., Dodge).

A more complicated picture emerged when self-report data was considered (Baker et al., 2008). Most notably, strong gender effects were found in participants self-reported aggression such that aggression rated by boys showed a strong genetic influence while no such effects were found for female aggression. By contrast, no sex differences were found for teacher or parent rated aggression. This latter result is generally consistent with Brendgen et al. who found no sex effects in their teacher reported data. Barker et al. note the considerable challenge of making theoretical sense of divergent results across multiple informant groups.

Nonetheless, both the Brendgen et al. and Baker et al. studies highlight the important role of genes in the co-occurrence of RA and PA. Specifically, these early studies suggest that genetic effects are more strongly associated with undifferentiated aggression as opposed to being responsible for influencing specific subtype behaviour. However, it should be noted that the Baker study did not control for common form of physical aggression. Thus, there exists no large-scale genetic study that has attempted to disentangle form and function. Little et al. (2003) have demonstrated that after controlling for underlying common form of aggression, the high level of overlap between subtypes disappeared.

At the same time, the presence of shared environmental effects remains somewhat equivocal given the Brendgen et al. and Baker et al. studies reported divergent

findings relating to this issue. More data are needed to help clarify the role of C effects in aggression subtypes.

Both issues noted above represent an important focus of the current research.

2.2.6.3 Longitudinal Genetics and reactive aggression and proactive aggression

Meta-analytical approaches can be particularly useful in shedding empirical light where individual studies result in inconsistent findings. This approach involves pooling data from relevant studies thereby increasing the power to elucidate the size and nature of reported effects. In their meta-analytic review of undifferentiated aggression, Miles and Carey (1997) weighted 24 studies related to the genetics of aggression on the basis of reliability—a method which involved statistically controlling for the moderating effects of sample size. They also assessed for the potential moderating effects of age on reported results. Using this approach, the authors identified moderate influence of **C** effects for studies involving children and adolescents. However, they found the magnitude of these effects declined to negligible amounts in adult samples. Conversely, genetic effects were found to increase significantly with age.

The authors cited the smorgasbord model in speculating on this apparent pattern. In short, this latter model proposes that the effects of family environment are most influential earlier in a child's life and it suggests that increasing autonomy across development ultimately results in "the adult genotype...choosing the environments most compatible with the genotype" (Miles & Carey, 1997, p. 214). The net effect of

this GE correlation is putative age-related decreases in shared environmental effects accompanied by inverse increases in genetic effects. Such results have been reported in genetic studies of ASB (Miles & Carey, 1997; Rutter, 1996).

It is noted that the Brendgen et al. study (which found no **C** effects) involved 8-year-old participants while twins in the Baker et al. study (which found substantial **C** effects) were around 9 to10 years old—a pattern of results which is somewhat contradictory to that predicted by the smorgasbord model. However, the considerable difference in sample size between these two studies, and the very similar age ranges used, precludes any robust evaluation of the smorgasbord hypothesis by way of these two studies. A more comprehensive test of this proposition would be for a single large-scale population study to evaluate longitudinal data derived from stratified age groups that clearly distinguish childhood and adolescent cohorts. To reiterate, this represents a focus of the current research.

A final word regarding study design. In discussing optimal methodology aimed at increasing power to detect **C** effects, Rutter (2006) has noted the advantage of the longitudinal design over the cross-sectional design. Specifically, compared to longitudinal designs, cross-sectional analyses are generally disproportionately sensitive to idiosyncratic factors that (1) influence a trait at the specific point of measurement, but (2) do not contribute to continuity of that trait over time (Van den Oord & Rowe, 1997). Conversely, longitudinal designs are more sensitive to factors that consistently contribute to continuity of a trait over time—including, most notably, those factors associated with family environment (Rutter, 2006). This is an important point because understanding the factors that contribute to continuity, or

stability, of psychopathology is central to the goal of targeting effective interventions (van Beijsterveldt, Bartels, Hudziak & Boomsma, 2003).

At the same time, like all methodologies, longitudinal designs have specific limitations. One of the major difficulties with attempting to track behaviour in a population sample over time is the issue of *selective attrition*. Selective attrition occurs when participants who initially agree to take part in a study make a decision not to complete the study. Data from these participants are usually incomplete and hence are frequently left out of subsequent analyses. In addition to the loss of statistical power this incurs, it also represents a possible source of bias where those who withdraw from the study are relatively homogeneous in regards to a factor or variable known to impact on the trait under investigation. While longitudinal researchers invariably make considered efforts to minimise the impact of this problem, a certain degree of attrition is usually unavoidable.

Nonetheless, the information provided by longitudinal designs is well suited to clinically focused research due to its focus behaviour change both in the emergence and resolution of psychopathology. This clinical perspective also highlights the fact that the broader success of research into RA and PA is closely tied to its capacity to clarify etiological mechanisms relevant to existing clinical taxonomies such as the DBDs. The next section of this literature review will consider the potential for behaviour genetic perspectives on RA-PA to enhance the etiological evidence base from which clinical nomenclature is informed.

2.2.7 Etiological Overlap between Aggression Subtypes and Disruptive Behaviours

2.2.7.1 The role of comorbidity and heterogeneity in antisocial pathways

While aggression is amongst the most common referral issue for children presenting to psychiatric and mental health clinics, there is currently no diagnostic system that explicitly recognises disorders of aggression (Barratt & Slaughter, 1998; Connor, 2002). Nonetheless, two principal diagnostic systems exist for the classification of broader disorders of behaviour; the ICD-10 used extensively in Europe and the American DSM-IV. The following section will focus predominantly on the three DSM-IV-based Disruptive Behaviour Disorders (DBD) due to (1) the ubiquitous use of this diagnostic system in the behavioural genetics literature, and (2) the frequent reference to DBD symptomatology in RA-PA research. Nonetheless, where relevant, reference will be made to convergence or divergence in the ICD-10 versus DSM-IV schemes.

CD, like ASB, reflects a broad range of concomitant aggressive and delinquent behaviours (Lahey et al., 1999). However, two key differences distinguish CD from ASB. Firstly, CD represents only a subset of ASB behaviours. More specifically, the syndrome tends to apply to a comparatively restricted range of the life span. To illustrate, CD prevalence rates generally peak in adolescence with the disorder being less common in either childhood or adulthood (Loeber et al., 2000). It is noted, however, that those children with ADHD are at considerably higher risk of evidencing CD earlier in childhood (Loeber et al., 2000). Furthermore, identifying higher prevalence rates in adolescence does not minimise the importance of early

onset of CD as a critical marker for life-course persistent conduct problems (Lahey et al., 1999).

Secondly, being a diagnostic entity, CD represents a categorical distinction between affected and non-affected individuals, while ASB is most commonly conceptualised dimensionally (Rutter, 2001). It is worth noting here the contribution of a recent etiological study which suggests that extreme CD may be etiologically distinct from less severe forms CD (Levy, Bennett, Hartman, Hay & Sergeant, 2006). Specifically, Levy et al. (2006) found that when CD was classified into three distinct levels of severity—mild, moderate and severe—using a dimensional scale developed by Hartman and Sergeant, (cited in Levy et al., 2006), correlations between concurrent comorbid disruptive behaviours symptoms including inattention and hyperactivity/ impulsivity were significantly greater when compared to the less severe groups. Moreover, the mild and moderate CD groups showed substantially higher inter-class correlations with one another than with the severe group.

ODD symptoms are generally more congruent with aggressive than delinquent behaviour (Frick et al., 1993). However, differences in how aggression is defined determine whether ODD symptomatology is viewed as directly indexing aggression (Greene & Doyle, 1999; Lahey et al., 1999), or simply non-compliance (Shaw, Owens, Giovannelli & Winslow, 2001; Waslick, Werry & Greenhill, 1999). Nonetheless, few disagree that ODD behaviours "contain the seeds of aggressive responding" (Waslick et al., 1999, p.456). Prevalence rates for ODD generally peak earlier in development than CD, although, as previously noted, there exists considerable overlap between these two disorders (Loeber et al., 2000). Longitudinal

studies have confirmed that comorbidity involving ODD and CD appears to have an etiological dimension (Beiderman et al., 1996; Lahey & Loeber, 1994; McGee & Williams, 1999; Rowe, Maughan, Pickles, Costello & Angold, 2002; Rutter, Giller & Hagell, 1998). Specifically, a significant proportion of children who develop CD have a prior history of ODD while it is rare for CD to precede ODD (e.g., Farone, Biederman, Jetton & Tsuang, 1997).

In light of this work, Loeber and colleagues have proposed a risk pathway model in which ODD is posited to be a developmental precursor of CD (Burke, Loeber & Birmaher, 2002; Lahey & Loeber, 1994; Loeber & Hay, 1994). Rutter (2001) has also questioned the strict distinction between these two DBDs, agreeing that where ODD precedes CD developmentally, a single underlying disorder is likely. Nonetheless, Rutter (2001) offers an important qualification of the ODD-to-CD hypothesis in acknowledging that ODD that does not lead to CD may represent a disorder that is distinct from ODD that does. This caveat is important as some research indicates that up to two thirds of children with ODD do not go on to develop CD, (Greene & Doyle, 1999). Indeed, Loeber and colleagues emphasise the need to investigate the possibility of distinct subgroups of ODD with respect to onset of CD (Loeber et al., 1995). These commentators have further proposed that such a subtyping may be usefully based on the concomitant presence (versus absence) of comorbid ADHD symptomatology as a risk indicator for subsequent CD. Loeber et al.'s (1995) proposed subtyping of ODD underscores an important empirical and clinical fact, that comorbidity amongst DBDs is the rule rather than the exception (Hinshaw, 2002; Kadesjoe & Gillberg, 2001; Quay, 1999; Waldman, Rhee, Levy & Hay, 2001).

Indeed, around half the children who develop ADHD also evidence ODD or CD (Waschbusch, 2002). The issue of comorbidity in ADHD has been somewhat complicated by the fact of high within-disorder heterogeneity with the distinction between the three subtypes of ADHD supported on etiological grounds, including studies showing differential genetic liabilities (McLoughlin, Ronald, Kuntsi, Asherson & Plomin, 2007; Nadder, Silberg, Rutter, Maes & Eaves, 2001; Thapar, Hervas & MCGuffin, 1995; Waldman et al., 2001).

Although ADHD symptoms are less obviously indicative of ASB, a large volume of research confirms the developmental relationship between these two broad syndromes. Specifically, children with ADHD are at considerably greater risk for antisocial behaviour (Lahey & Loeber, 1997; Moffitt, 1990; Taylor, Chadwick, Hepinstall & Danckaerts, 1996), and aggression in particular (McKay & Halperin, 2001), in later life than children without ADHD. This comorbidity has led to protracted debate concerning whether comorbid ADHD and antisocial behaviour (typically viewed in terms of CD) is best considered a discrete clinical entity (i.e., ADHD+CD) with specific etiological features (Waschbusch, 2002). There is growing consensus, however, that evidence does not support concomitant ADHD and CD as a unique taxonomic category (Waschbusch, 2002) . Most recently, a behavioural genetic analysis of competing models provided compelling evidence that concomitant ADHD and CD is best understood as two distinct risk factors, rather than a qualitatively distinct disorder (Rhee, Willcutt, Hartman, Pennington & DeFries, 2008).

Certainly, at the clinical level the ICD-10 recognises hyperkinetic disorder with conduct problems as a relatively discrete diagnostic category. Hyperkinetic disorder is roughly equivalent to the ADHD syndrome articulated in the DSM-IV. However, it focuses predominantly on two subcategories, *impaired attention* and *overactivity*. The former category is synonymous with the inattention category in DSM-IV. Overactivity represents symptoms consistent with the composite hyperactivity-impulsive subtype in DSM.

Criteria for both ICD-10 subcategories need to be met for a diagnosis of hyperkinetic disorder. The condition is then mandatorily classified as being *with* or *without* comorbid conduct disorder.

This taxonomic approach illustrates differences between the managed care goal of providing tiered intervention services to address psychopathology and the broader research goal of better understanding etiological factors contributing to syndromal pathology. Invariably, progress in ASB research has been marked by attempts to disentangle intra-syndrome heterogeneity. As such, there has been a trend in the literature over the past decade to look more closely at the distinct role of subtypes of ADHD (i.e. hyperkinetc disorder) in ASB risk pathways. The role of inattention, hyperactivity and impulsivity in ASB are considered below.

While some research has identified attentional difficulties as a unique correlate in risk factor research (Dodge et al., 1997; Vitaro et al., 2002), scientific resources have predominantly been directed to the study of hyperactivity (i.e., overactivity) and impulsivity as the main prognostic variables of interest in ASB research. This tendency to divert focus away from inattention may be due to concomitant risk

factors associated with this ADHD subtype dimension including, perhaps most notably, poor academic performance and general classroom impairment (e.g., Herman, Lambert, Ialongo & Ostrander, 2007; Waschbusch et al., 1998) which tends to have negative cascading effects that may undermine the unique prognostic value of inattention in subsequent conduct problems (Hay et al., 2001a; Ingram et al., 1999). Unfortunately, to the author's knowledge there is no systematic review of research into the role of inattention in ASB pathways to shed light on this speculation. The scant research that does exist suggests this syndrome dimension is more closely associated with RA than PA (Dodge et al., 1997; Vitaro et al., 2002l; Waschbusch et al. 1998). Interestingly, these findings have not been clearly integrated into subsequent RA-PA research. At the same time, as more is known about the neuropsychological underpinnings of attention deficits, this paradigm will likely constitute a growing area of clinical enquiry in ASB research (see Blair & Mitchell, 2009; Dadds & Rhodes, 2008; Dadds et al., 2006).

Nonetheless, the somewhat narrowed research interest on hyperactivity and impulsivity as developmental markers of risk certainly represents clinical advances that reflect the fact that heterogeneity in DBDs requires careful and parsimonious delineation of risk phenotypes (Friedman-Weieneth, Harvey, Youngwirth & Goldstein, 2007; Hinshaw, 2002). Certainly, research has generally converged to recognise the combined hyperactivity and impulsivity (HI) subtype dimension as more closely aligned to risk pathways than the broader ADHD phenotype (Hinshaw, 2002). At the same time, there is mounting evidence that this subtype dimension does not enjoy unique prognostic value once physical aggression is accounted for (Broidy et al., 2003).

In their extensive six site study that involved over 6000 children followed into adolesence, Broidy et al. (2003) concluded that the combined hyperactivity/ impulsivity dimension failed to retain its status as an independent risk factor when other conduct problems (including oppositional behaviour and physical aggression) were taken into account. These findings concur with more recent research which has shown that neither genetic or environmental etiological factors associated with intattentive-hyperactivity or oppositional behaviour uniquely predict later conduct problems when early conduct problems are controlled for (Lahey et al., 2009). Importantly, Broidy et al. (2003) found that only physical aggression independently increased risk for future maladaptive behaviour after controlling for co-occuring disruptive behaviours such as hyperactivity or oppositional behaviour. Interestingly, oppositional behaviour, like hyperactivity/impulsivity, did not contribute risk over and above that accounted for by aggression. These results replicated those found in an earlier study reported by Nagin and Tremblay (Nagin & Tremblay, 2001).

Notable in all this research is the use of the composite hyperactivity/impulsivity (HI) measure. There is very little evaluation of etiological homogeneity associated with this scale. The question is thus raised whether the prognostic value of the HI scale is undermined by differential contributions to risk by hyperactivity and impulsivity symptoms. This is particularly pertinent in aggression research which seeks to evaluate the differential etiological role of aggression subtypes.

The above observation raises the broader issue regarding inconsistent operationlisation of disruptive behaviour syndromes in clinical research. Indeed it seems some investigators use the terms ADHD, Hyperactivity-impulsivity and

hyperactivity interchangeably as key research constructs (Raine et al., 2006; e.g., Vierikko et al., 2004). This is exemplified by the recent Raine et al. (2006) study which evaluated and discussed the unique role of *hyperactivity* in RA and PA pathways while providing methodological descriptions indicating their measure of hyperactivity in fact combined hyperactivity, impulsivity and attention difficulties into a single HIA scale. It is likely that etiological models will benefit from disentangling rather than conflating ADHD symptom dimensions. However, this is not to minimse the valuable research that clearly identifies children with ADHD at increased risk of long-term conduct problems.

2.2.7.2 Etiological overlap between aggression and disruptive behaviour in risk pathways

Given the established concurrent relationship between ADHD and aggression on the one hand (McKay & Halperin, 2001; Waschbusch & Willoughby, 2008), and the significant contribution of aggression to continuity in problem behaviour on the other, it is reasonable to propose aggression as a candidate developmental marker in attempts to refine diagnostic phenotypes (Kempes et al., 2005). Thus, from the perspective of the current review, an important question concerns whether aggression subtypes constitute problem behaviours that mediate the long-term risk conferred by specific DBD behaviours. An important first step in answering this question is to begin to understand the etiological relationships between these variables.

However, understanding the unique etiological role of aggression in DBDs is complicated by the fact the behavioural genetic literature has mixed findings regarding the etiology of comorbidity between the DBDs (Tuvblad, Zheng, Raine &

Baker, 2009). For example, Burt, Kreuger, McGue and Iacono (2001) found that the overlap between all three DBDs was explained predominantly by **C** effects. The authors suggested that this may reflect the effects of coercive parental practices. Conversely, and somewhat more consistent across studies, a number of investigators report that genes contribute substantially to overlap in DBD symptomatology (Dick, Viken, Kaprio, Pulkkinen & Rose, 2005; Tuvblad et al., 2009; Waldman et al., 2001).

In their trivariate analysis of ADHD, ODD and CD, Waldman et al. (2001) derived heritability estimates for ADHD and ODD of $h^2 = .85$ and $h^2 = .90$ respectively with no **C** effects present on the former and only marginally on the latter. By contrast, CD showed only moderate heritability ($h^2 = .51$) but showed significant **C** effects $h^2 = .34$. Non-shared environmental effects were universally weak across all DBDs. The Cholesky design used by these authors (explained in Chapter 3) provided strong indications that genes, and to a lesser extent non-shared environmental effects contributed significantly to comorbidity. Specifically, 93% of overlap between ADHD versus the two antisocial DBDs was attributable to genes with the remaining 7% accounted for by nonshared environmental (**E**) effects. When overlap between ODD and CD were considered independently, around 79% of shared variance was the result of genetic factors. The only **C** effects present in the trivariate model operated simultaneously on ODD and CD. As noted, for ODD, these effects were marginal.

Simonoff et al. (1998) isolated aggressive from non-aggressive behaviours from amongst ODD and CD symptoms using Frick et al.'s suggested dual continuum

model (Frick et al., 1993). Specifically, the authors derived four ASB subtypes from antisocial DBD symptomatology in a community sample of adolescent males including; property violations, status violations, oppositional behaviour and aggression. Their analysis of caregiver-rated data pointed to specific genetic influences on aggression with the authors concluding that "aggressive-destructive" behaviours had the greatest unique genetic liability and hence may represent an etiologically distinct component of ASB. These results reflect support for the dual pathway model discussed earlier in which aggressive versus non-aggressive delinquency are differentiated on the basis of core etiological influences (Tuvblad et al., 2005). However, the maternal ratings reported in the Simonoff et al. study suggested marginal **C** effects operating independently on all subtypes aggression except status violations—and these **C** estimates were generally equivalent or smaller to the **C** component that explained the overlap between all four subtypes. In this way, the latter results diverged from the dual pathway model which suggests that **C** effects are substantially more important in non-aggressive than aggressive delinquency (Tuvblad et al., 2005).

As noted above, the aggression items used in Simonoff et al.'s (1998) study were classified on the basis of being overt and destructive. As such, they did not distinguish functional subtypes. In this regard their index represents undifferentiated aggression.

Studies that have looked at the co-occurrence of ADHD behaviours and undifferentiated physical aggression have found mixed results. For example, while Vierikko et al. (Vierikko et al., 2004) reported substantial impact of genetic and

shared environmental factors on the overlap between hyperactivity/impulsivity (HI) and aggression, around half of the variance in the former composite measure was trait-specific, suggesting that HI and aggression have at least partially divergent etiologies. Conversely, Seroczynski, Bergeman and Coccaro (1999) found that impulsivity and reactive-type aggression share a substantial portion of genetic and environmental variance leading the authors to conclude that reactive-type aggression and impulsivity share similar etiologies. Curiously, despite their reference to it, reactive aggression was not measured directly in this latter study. From a somewhat related perspective, New, Goodman, Mitropoulou and Siever (2002) reviewed the genetic literature on impulsive aggression and concluded that a moderate amount of shared variance (i.e., 20%-60%) in this behaviour is attributable to genetic factors.

In their recent longitudinal (non-genetic) investigation of psychosocial correlates associated with aggression in adolescence, Raine et al. (2006) found differential relations between residualised RA and impulsivity, on the one hand, and residualised PA and hyperactivity on the other. They postulated that the relationship between PA and hyperactivity reflects a shared underlying association with developmental psychopathy. Developmental psychopathy refers to a range of symptoms, including callous/unemotional traits, that are consistent with adult psychopathy but are measured in childhood and adolescent populations (Salekin & Frick, 2005). Consistent with the poor outcomes associated with this latter construct, Raine et al. noted that developmental correlates of PA at age 16 included psychopathic personality traits, blunted affect and serious violent offending. This study provides an interesting complement to Vitaro et al.'s (2002) speculation that PA (and not RA) is associated with the ODD to CD risk pathway.

The Raine et al. (2006) study raises the possibility that reported associations between hyperactivity and increased risk for life-course peristant ASB, may be underpinned by etiologically distinct associations between aggression subtypes and risk. If the risk conferred by hyperactivity and oppositional behaviour is mediated by RA, PA or both subtypes, then it is interesting to speculate the nature of the mechanisms shared by the DBDs and aggression.

The above literature review has culminated in a range of key objectives and research aims guiding current attempts to elucidate the role of aggression subtypes in ASB pathways. This research agenda is set out in section 2.4 and presents a range of hypotheses and proposed exploratory investigations. The following (and penultimate) section represents an attempt to integrate multiple levels of explanation in understanding the origins of complex behaviours such as aggression. Specifically, an endophenotype approach is proposed that examines putative neuropsychological features of RA and PA and their possible role in mediating between geneotype and phenotype.

2.2.8 Neuropsychobiology of Reactive and Proactive Aggression

Advances in measurement of neurocognitive processes and neuroimaging have led to an increasing focus on endophenotypes in behaviour genetic research (Viding & Blakemore, 2007). An *endophenotype* is a phenotype that mediates between etiological factors and behavioural traits (Kuntsi, McLoughlin & Asherson, 2006; Rommelse et al., 2008a). Specifically, endophenotypes are "quantitative indices of disease liability or risk" that are considered relatively more proximal to genetic function than the identified behavioural phenotype (Castellanos & Tannock, 2002, p.

619). Critical characteristics of any endophenotype include the requirement that it is continuously quantifiable and predictive of the behavioural trait or phenotype (Castellanos & Tannock, 2002). Moreover, in the case that genetic mechanisms are being investigated, the endophenotypes must demonstrate heritability (Castellanos & Tannock, 2002; Gottesman & Gould, 2003).

A frequent assumption of this model is the ability to identify and measure implied rather than directly observed processes—e.g., the indexing of neuropsychological or cognitive functioning (see Castellanos & Tannock, 2002). The complexity of gene expression and its relationship to cognitive processes cannot be over-emphasised and underpins a fundamental limitation of the endophenotype approach. That is, this method relies principally on a good understanding of how best to theoretically parse complex neuropsychological functions. It further requires development of a valid and reliable measures of these identified functions. There is general consensus that further progress is required to adequately met these goals (Rommelse et al., 2008b), and a persistent challenge is that neither of the requirements can be resolved without progress in the other. Nonetheless, this dilemma is certainly not new to the scientific paradigm and the basic endophenotype approach offers one way forward in a relatively new, and somewhat fractured, field of enquiry. In the current state of the science, this approach is typically consitutes the application of behavioural genetic methodology to the field of neuroscience and cognitive neuropsychology (Castellanos & Tannock, 2002).

The study of RA and PA represents a potential area for endophenotype research for two related reasons. Firstly, the influence of genes on aggression is empirically

robust and consistently reported to be of substantial magnitude (Miles & Carey, 1997). Importantly, there exists emerging evidence that genes contribute to differentiation of aggression subtypes (Baker et al., 2008). Secondly, the RA-PA framework has its theoretical and empirical roots in the neurobiological literature which has consistently converged on the suggestion of differential neural substrates underpinning aggression subtypes (Blair, 2004; Mitchell, Avny & Blair, 2006). It is worth noting that Blair's recent formulation concerning the neuropscyhology of RA-PA presents the prospect of a partial double dissociation between identified processes associated with neurual substrates subserving each aggression subtype. These substrates are considered in turn.

2.2.8.1 Orbital Frontal Function and reactive aggression

The link between orbital frontal cortex (OFC) function and RA has been established in both adult (Bechara, Damasio, Damasio & Anderson, 1994) and childhood samples (Anderson, Bechara, Damasio, Tranel & Damasio, 1999; Pennington & Bennetto, 1993). In the clincial neuroscience literature, related deficits have been associated with a syndrome called *sociopathy* (Damasio, 1994). Adult sociopathy, is an acquired form of psychopathy which results from selective trauma to the orbito frontal area of the brain. A defining behavioural feature of sociopathy in adults is dramatic post-morbid increase in RA.

Psychopathy itself is defined by a number of constituent behavioural dimensions including a propensity to engage in impulsive and conduct problem behaviours (I/CP), and the presence of callous/unemotional (C/U) traits (Barry et al., 2000; Frick & Ellis, 1999; Frick et al., 1994) – see Appendix A for specific operational criteria.

Like sociopathy, seminal treatises on clinical features of psychopathy derive from the adult psychopathology literature. Certainly, related investigations have confirmed a strong association between psychopathic personality traits and violent and life-course persistant antisocial behaviour in adult criminals (Cornell et al., 1996; Edens, Skeem, Cruise & Cauffman, 2001; Lynam, 1997; Woodworth & Porter, 2002).

More recently, Blair and others (Blair, 2003; Blair, Lawrence, Clark & Smith, 1997; Frick et al., 2000; Mitchell & Blair, 2000) have proposed a developmental model of psychopathy that represents an attempt to identify and understand emergent psychopathic traits and behaviours in childhood. However, unlike causal models of adult sociopathy which emphasise the role of selective brain trauma, the latter literature suggests the possibility that developmental psychopathy is potentially the result of congenital factors. According to Blair (2001) candidate congenital factors include frontal lobe pathology and genetic abnormalities involving, for example, the 7 repeat dopamine 4 receptor gene. It is beyond the scope of this text to elaborate on this proposed mechanism—and indeed Blair has not yet articulated a fully specified model. Other candidate congenital factors include the potential effects of prenatal maternal smoking (e.g., Button, Thapar & McGuffin, 2005). At the same time, while research may yet uncover specific perinatal risk mechanisms that can be linked to developmental psychopathy, it is worth reiterating that complex behavioural phenotypes, such as that described by developmental psychopathy, are unlikely to be the result of any one factor, or even one set of factors, operating in isolation at a single time point.

Blair (2004) provides a useful summary of the emprical link between OFC function

and RA from a developmental perspective. The research of Colledge and Blair (2001) suggests OFC (and by association RA) is more closely associated with the I/ CD dimension of developmental psychopathy than with the C/U dimension. It should be noted that such distinctions are not dichotomus. More recently, Coccaro, McCloskey, Fitzgerald and Phan (2007) reported diminished OFC activity in adults with clinical levels of impulsive aggression (i.e., Intermittent Explosive Disorder), on tasks requiring participants to recognise facial expressions of emotion.

This is an emerging and complex field of enquiry and it need to be emphasised that the precise role of OFC in antisocial behaviour remains unclear (Blair, 2004). Nonetheless, neuropsychological models of adult sociopathy/psychopathy (e.g., Damasio, 1994; Hare & Neumann, 2008), may provide a starting point for understanding possible mechanisms involved in OF-related behavioural deficits in childhood. Specifically, *response reversal*, an executive function linked to damaged OFC (Bechara, 2004), may be relevant in a developmental context (Crone, Bunge, Latenstein & van der Molen, 2005; Garon & Moore, 2004; Garon & Moore, 2007b; Kerr & Zelazo, 2004). Response reversal refers to the capacity to modulate responding according to changes in reward value associated with specific contingencies. For example, adaptive modulation may involve the extinction of operant responding due to increased negative reinforcement (i.e., punishment) associated with the previously rewarded behaviour. Bechara and others associated with the Iowa research team (Bechara, 2004; Bechara, Damasio, Tranel & Damasio, 2005; Brand, Recknor, Grabenhorst & Bechara, 2007) developed the computer-based Iowa Gambling Task (IGT) to show that, compared with non-affected participants, OFC patients are less sensitive to long-term (net) losses associated with short-term

high value rewards (see Chapter 3 for an explanation of this task). By contrast, those without OFC deficits showed a tendency to adapt their responding style to preference short-term low value rewards associated with long-term net gains—although see Blair (2004), and Manes et al. (2002) regarding the alternative viewpoints on this task and its capacity to index OFC function.

The IGT has also been adapted for children (Crone & van der Molen, 2007; Garon & Moore, 2004; Garon & Moore, 2007a; Huizenga, Crone & Jansen, 2007; Kerr & Zelazo, 2004). In this latter developmental context, maturation of brain function, rather than brain injury, has been investigated—although see reports by Happaney and colleagues (Happaney & Zelazo, 2004; Happaney, Zelazo & Stuss, 2004) for a critique of extinction of operant responding as an index of OFC function in childhood.

It has been hypothesised that OFC function may be under a degree of genetic control (da Rocha et al., 2008). Although speculative, it therefore possible that OFC represents an empirically useful endophenotype for RA. Interestingly, reporting on a sample of children aged between 3 and 5 years old, (Happaney & Zelazo, 2004) provide preliminary support for associations between extinction learning, measured using an adaption of the Iowa Gambling Task, and temperament-related characteristics including anger, impulsivity and attentional difficulties. It is worth pointing out that all three of these temperament-related features have been shown to be under some level of genetic control (e.g., Giegling, Hartmann, Möller & Rujescu, 2006; Rasmussen et al., 2002; Stevenson et al., 2005), and all three are correlates of RA (e.g., Dodge et al., 1997; Vitaro et al., 1998). Thus, while OFC function has not

hitherto been identified as a candidate endophenotype for RA, there is some evidence to support preliminary research in this area.

2.2.8.2 The Limbic System and proactive aggression

In their developmental model of psychopathy, Blair and colleagues (Blair, 2003; Blair, 2006; Blair & Mitchell, 2009; Mitchell & Blair, 2000) posit lymbic system dysfunction as a key contributing factor in the early acquisition of PA type behaviour. According to this aspect of their model, sub-optimal amygdala functioning constitutes an ultimate mechanism in the development of callous-unemotional traits associated with childhood psychopathy (Viding, Blair, Moffitt & Plomin, 2005).

At the socio-emotional level, the relationship between limbic system deficits and developmental psyhopathy is thought to be mediated by empathic responsivity (Blair, 2006). Specifically, compromised amygdala function is hypothesised to lead to a selective impairment in autonomic responding to distress cues, e.g., facial expressions of fear and sadness (Blair, 2003). The model suggests this deficit disrupts socialisation by undermining the normal process of aversive conditioning that associates innate dysphoric responsivity to social distress cues with behaviours that elicit those distress cues. When functioning normally (over the course of development), this aversive learning theoretically works to reduce the probability of aggressive responding and antisocial behaviour. The model has received considerable empirical support (Blair & Mitchell, 2008).

For instance, Blair and colleagues have reported that both clinic-referred, as well as

non-clinic referred, children who scored highly on the Psychopathic Screening Device (PSD) showed selective deficits in recognising sad and fear facial expressions of emotion (Blair et al., 2001). The deficit appears to be particularly pronounced, or more consistent, for facial expressions of fear (see also Blair, 2004; Mitchell et al., 2006). The task used in this research paradigm is a computer-based program called MultiMorph, developed by Mitchell and colleagues (Mitchell et al., 2002; Mitchell, Richell, Leonard & Blair, 2006). The test requires participants to discriminate graded changes in intensity of facial expressions of emotion (see Chapter 3 for more information on this task).

Mitchell and Blair (2000) make the point that developmental psychopathy, and the C/U trait dimension in particular, phenotypically resembles PA. This observation has been borne out by subsequent research (Moffitt et al., 2008; Raine et al., 2006) underscores the possibility that children with PA may show a selective deficit in perceptual sensitivity to facial expressions of sadness and fear.

The more speculative assertion that sensitivity to specific emotional cues consitsutes an endophyenotype indexing putative genetic mechanisms responsbile for PA is based on the following proposed theoretical assumptions. Firstly, as above, the proposition emphasises the link between emotional recognition and empathic ability (Blair, 2003) on the one hand, and the link between empathic ability and callous/unemotional (C/U) traits (Blair, 2006) on the other. Blair (2001) calls this the *empathy position*, "that stresses the aspects of psychopathy related to the reduced sensitivity to the emotional signals of others". Secondly, there is a well-cited line of evidence that psychopathy, and the C/U trait dimension in particular, is less strongly

related to psychosocial correlates than other antisocial behaviours (Wooton, Frick, Shelton & Silverthorn, 1997). In their influential study involving sample of 6- to 13-year-old clinic referred boys, Wooton et al (1997) distinguished between boys low, versus high, on C/U traits - as measured by the child Psychopathy Screening Device (Frick et al., 2000; Frick et al., 1994). The investigators found that maladaptive parenting was associated with conduct problems only in the group who were low on C/U traits. Conversely, conduct problems in boys rated highly on the C/U trait dimension were not associated with quality of parenting suggesting an important role for non-environmental factors in the C/U dimension.

This latter hypothesis has received growing support in the behaviour genetics literature (Viding et al., 2005; e.g., Viding, Jones, Frick, Moffitt & Plomin, 2008). For example, Viding et al. (Viding et al., 2005) found a substantial genetic component in a sample of early school aged children rated highly on C/U traits, while Dworkin et al. (1976, cited in Rhee, Waldman, Hay & Levy, 2001) reported significant genetic influences on adolescent psychopathy. Consistent with Blair's own conceptualisation, Lahey et al. (1999) speculate that a callous/unemotional temperament may predispose to PA and these authors propose this behavioural propensity is genetically mediated.

The various findings outlined above point to the possibility that genes, or at least congenital factors, are implicated in the emotion recognition substrate implicated in C/U related empathic deficits (Jones, Laurens, Herba, Barker & Viding, 2009). If, as suggested by theorists such as Blair and Frick, PA symptomatology overlaps with C/U traits, in either an operational or theoretical sense, it is feasible this commonality

extends to neurobiological substrates and their corresponding etiological pathways.

A final point is worth making here. The suggestion that the link between temperament and antisocial behaviour is, in part, mediated by perinatal environmental factors that impact on brain development (van Goozen, Fairchild, Snoek & Harold, 2007), is not inconsistent with Blair's proposition of culmulative developmental problems precipitated by congenital deficits (Mitchell et al., 2006). At the same time, Tarquis' (Tarquis, 2006) thesis that chronic maltreatment in early childhood may adversely impact neural substrates (including amygdala) responsible for fear conditioning is a reminder of the complex and multiple pathways involved in developmental psychopathology. Crucially, from a multiple pathways perspective, perinatal and early childhood environmental risk pathways do not necessarily preclude the role of genetic vulnerability in synergistically increasing risk, as highlighted by the growing evidence base supporting genetics as a risk factor for psychopathy and associated traits (Blair et al., 2006).

To reiterate, coordinated multifaceted research programs that take full advantage of contemporary innovations in the field of science and technology are needed to unravel complex etiological pathways (Haworth et al., 2007). From a theoretical perspective, there are increasing calls for multimodal research programs that acknowledge the full scope of genetic, biochemical and electrophsyiological correlates relevant to the neurobiology of antisocial behaviour (e.g., Hinshaw, 2002), and psychopathy more particularly (e.g., Dolan, 1994). Blair's own (2001) multimodal model of developmental psychopathy has provided a useful foundation for inclusive pluralistic research.

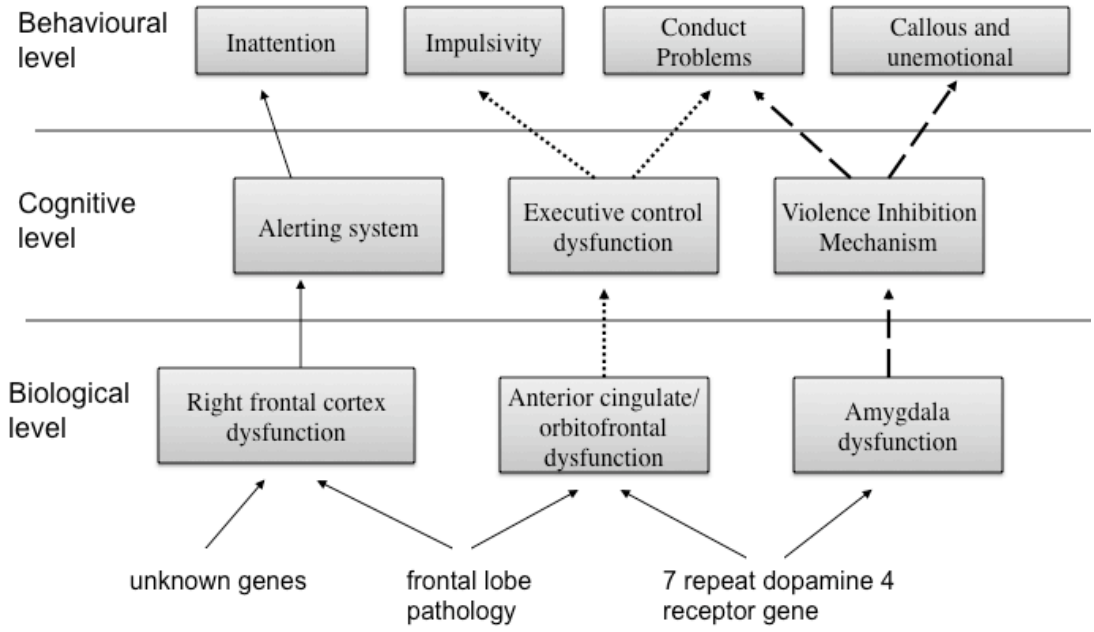


Figure 2.1 Schematic of Blair's (2001) proposed multi-level explanatory framework for understanding the etiology of developmental psychopathy

The study of endophenotypes represents a useful research model for integrating pluralistic mechanistic accounts. However, a current obstacle to this research is gathering enough behavioural and neuropsychological data to conduct relevant genetic analyses. The next section considers the use of contemporary online technologies as a means of addressing this issue.

2.2.8.3 The Role of Online Technologies in Endophenotype Research

The internet was originally conceived and developed as a tool to disseminate scientific information amongst research groups (Hewson, 2003). From its inception, there was an emphasis on the timely transmission of empirical data between research facilities separated by large distances. It is perhaps no suprise then that as the internet has evolved, its use as a tool for researchers has expanded to include 'real-time' participant data collection in a way that has reduced the impact of geographical amd

resource limitations on researchers (Fraley, 2007; Smith & Senior, 2001).

This technological revolution has had important implications for behavioural genetics research. Indeed, Haworth et al. (2007) note the potential value of the internet in BG research as a tool to connect with the large numbers of participants required for genetic studies. This issue is particularly relevant in countries, such as Australia, where the population is dispersed over vast geographical areas.

Chapters 3 and 7 present a novel online methodology utilised in the current research that afforded the online self-administration of the two neuropsychological tasks; The Iowa Gambling Task, and MultiMorph mentioned above. These tasks were adapted for delivery over the internet using web-based protocols. A short-form behaviour questionnaire based on the ATBRS was also presented online for caregivers to complete. Details of these tools and their implementation are provided in Chapters 3 and 7 along with preliminary results obtained using this approach.

2.3 Summary of Literature Review

Acceptance of RA and PA as a valid clinical subtypology is highlighted by consideration for its inclusion in the pending fifth revision of the DSM international diagnostic system. However, to demonstrate a clear contribution to formal taxonomies, this distinction will need to show a capacity to clarify existing diagnostic entities rather than simply provide yet another alternative way to classify key antisocial behaviours. The potential strength of the RA and PA framework lies in its inherent appeal to etiological mechanisms. Indeed, the existing disruptive behaviour syndromes have been empirically rather than theoretically derived and

have tended to lack coherent explanatory frameworks. From this perspective, the behaviour genetics paradigm offers a valuable empirical backdrop to developmental theories of aggression and their relevance to existing clinical constructs. To the author's knowledge the current research is the first to simultaneously examine the genetic architecture of the aggressive subtypes and the disruptive behaviour disorders. Due to previous meta-analytic research that has suggested the possibility of age-related changes in the genetic architecture of aggression (Miles & Carey, 1997), the ascertainment of data that represents both childhood versus adolescent (including pre-adolescent) cohorts was considered important.

Within the broader research context, inconsistencies deriving from existing behaviour genetics studies of RA and PA need to be considered. Specifically, extant ambiguities regarding the role of shared environment (**C**) in the differential etiology of RA versus PA—as might be predicted by the pre-eminent psychosocial model (i.e., Dodge et al., 1997)—have not yet been resolved and require complementary data. A useful means to increase the capacity to detect **C** effects is to utilise a repeated-measures design (Rutter, 2006). The current methodology, which is discussed in more detail in the following chapter, incorporates a 9-month follow-up testing occasion as a key design feature in order to contribute additional data on this issue. While this length of time to follow-up is modest, the quasi-longitudinal design also provided the opportunity for the preliminary evaluation of etiological factors associated with continuity (and discontinuity) of aggression over time. To the author's knowledge, there has been no repeated-measures behaviour genetics studies of RA and PA to date. A brief word on terminology is warranted here. While the term *longitudinal* is typically associated with methodology that involves three or

more measurement occasions (over time), the term is used throughout the remaining text as interchangeable with the term *repeated-measures*.

Finally, the importance of integrating multiple levels of explanation represents a challenging but vital component of any comprehensive theory of problematic aggression and antisocial behaviour. To this end, the current research also aims to investigate two candidate endophenotypes of RA and PA using novel online methodology designed to increase the capacity for reaching the large numbers of participants required for genetic research.

The specific objectives associated with the present project are outlined in the following section.

2.4 Objectives of the Current Research

The current research aims to provide the first elucidation of developmental relations between aggression subtypes and DBDs. Caregiver ratings were ascertained from a large community sample of families of twins (aged 6-18 years) using the Australian Twin Behaviour Rating Scales (ATBRS, n=2082), and at 9-month follow-up via an online electronic version of the ATBRS (n=511). These data were partitioned according to two age cohorts (6-10 years, and 10-18 years) and subsequently submitted to a series of univariate and multivariate cross-sectional (Study 1) and longitudinal (Study 2a) analyses. The online component of the study also included two neuropsychological tasks adapted for the internet and completed by 310 twin siblings (Study 2b). Figure 3.1 (p. 88), as well as Tables 6.3 (p. 243) and #.# (p. #) illustrate the relationship between studies and data collection Waves.

The following section sets out a series of research questions concerning the basic genetic architecture of RA and PA that aim to complement and contribute to existing findings in this relatively new field of enquiry. Sections 2.3.2 - 2.3.4 describe the aims associated with Study 2a and 2b respectively. The final sub-section considers aims associated with the use of innovative online technologies to assess putative endophenotypes in genetically-sensitive research design.

2.4.1 Study 1 Research Objective (1): Evaluate the Genetic Architecture of Aggression Subtypes in a Large Population Sample

2.4.1.1 Aim 1: Assess for differences in architecture across childhood and adolescent cohorts

The current research provides an opportunity to assess the potential relevance of the smorgasbord hypothesis (i.e., Miles & Carey, 1997) to aggression subtypes. Indeed, in their widely-cited meta-analysis of the genetics of aggression, Miles and Carey (1997) speculated that important shifts may occur in the genetic architecture of aggression over the course of development. Specifically, this proposition represents the testable prediction of increased magnitude of genetic effects in older versus younger cohorts, with an inverse corresponding reduction in shared environmental effects. The research of Brendgen further suggests the predominance of non-shared environmental effects with age.

2.4.1.2 Aim 2: Evaluate shared environmental effects in a large-scale population sample

Analyses conducted on data from the current sample aim to shed light on previous inconsistent findings which have failed to clarify the role of shared environmental

effects in the development of RA and PA. At the same time, the current methodology seeks to ensure that common underlying form of aggression (i.e., physical aggression) is controlled for (Brendgen et al., 2008; Kempes et al., 2005; Little et al., 2003). To the author's knowledge, this aspect of methodology did not occur in the Baker et al. (2008) study.

2.4.1.3 Aim 3: Assess for sex differences in and reactive aggression and proactive aggression

The extant literature is somewhat equivocal concerning sex differences in RA and PA (see Chapter 5 for a more detailed review). The current study aims to use two complementary approaches to elucidate the role of genes in sex differences in aggression subtypes. Specifically, more data are needed to help answer the question of whether differences in prevalence of RA and PA amongst male versus female cohorts are due to different levels of liability or different etiological mechanisms. The advantage of the current design is that this question can be applied to different age cohorts reducing the risk of sex effects being masked due to any changes in genetic architecture occurring over the key developmental stages.

2.4.2 Study 1 Research Objective (2): Evaluate Etiological Relations between Aggression Subtypes and Disruptive Behaviour Disorders

This objective reflects the broad goal of elucidating etiological relationships between aggression subtypes and DBDs. While the role of RA and PA in DBD risk pathways has been established, it remains unclear how the etiologies of aggression subtypes interact with other disruptive behaviour problems such as hyperactivity, impulsivity, ODD and CD. To the author's knowledge, this series of bivariate genetic models presented in relation to this objective will constitute the first reported analyses of the

genetic relationship between the aggression subtypes and clinical syndrome dimensions.

It is noted that these analyses involve separate analyses for hyperactivity and impulsivity. While it is common practice in the literature to utilise scales which combine these two ADHD symptom dimensions to reflect the clinical subtype of ADHD-HI, the current approach recognises recent work that supports a somewhat more circumscribed view of ADHD symptom dimensions (Raine et al., 2006). Certainly, the current research is informed by the assumption that ongoing efforts to parse heterogeneity in aggression in a clinically meaningful way are assisted by efforts to reduce the potential confounding effects of heterogeneity in other DBDs, and ADHD in particular.

A final note concerns the inclusion of inattention in basic bivariate modeling. While the literature is suggestive of unique relations between inattention and RA (Dodge et al., 1997; Vitaro et al., 2002), there have been few theorists who have addressed the specific role of attention deficits in DBD or ASB risk pathways (although see Blair & Mitchell, 2009; Dadds & Rhodes, 2008; Dadds et al., 2006). This latter work indicates the possibility that attention difficulties will increasingly become an area of focus in ASB research. The current research hopes to contribute preliminary indications of etiological relationships that explain the overlap between this ADHD subtype dimension and aggression subtypes.

2.4.3 Study 2a Research Objective (1): Assess Etiological Contributions to Continuity in Aggression Subtypes and Disruptive Behaviour Disorders

The first aim stated below relates to predictions derived from the sequential hypothesis suggesting RA is a developmental precursor to PA. The second aim of this objective is to provide a series of empirical tests of assumptions and hypotheses derived from the extent aggression subtype and DBD literature.

2.4.3.1 Aim 1: Evaluate the degree to which reactive aggression predicts future proactive aggression in both childhood and adolescent cohorts after controlling for underlying common form of aggression (i.e., physical aggression)

This aim is effectively the sequential hypothesis stated in empirical terms. The inclusion of two age cohorts in the current study provides the added advantage of testing the assumption that any transitional relationships revealed in a specific cohort are linear across major stages of development. Moreover, this analysis involves controlling for common form of aggression which has been demonstrated to confound relationship between RA and PA (Brendgen et al., 2008; Kempes et al., 2005; Little et al., 2003).

2.4.3.2 Aim 2: Assess whether common versus unique etiological relations between aggression subtypes, hyperactivity, impulsivity and ODD contribute to continuity in aggression subtypes over time

This goal represents a series of aims related to the broad question of shared etiological factors in aggression subtypes and the DBDs that contribute to persistence in aggression over time. It involves assessing the degree of etiological overlap between DBDs and aggression subtypes measured at time 1 alongside the corresponding longitudinal relationship between subtypes measured at time 2.

Specifically, it comprises a number of aims related to questions raised by the extant literature on aggression subtypes and DBDs with the purpose being to:

2.4.3.2.1 Aim 2a. Contribute data bearing on the relationship between impulsivity and reactive aggression

2.4.3.2.2.1 *The role of impulsivity in the emergence of reactive aggression*

Some researchers have proposed that reactive types of aggression (such as RA) are secondary to an underlying impulsivity (Barratt & Slaughter, 1998; e.g., Coccaro et al., 1997). This suggests a shared etiology between impulsivity and RA in which the former is causally implicated in emergence of the latter, over and above any existing propensity for overt physical aggression. As noted above, risk factor research has shown a high degree of phenotypic overlap between these two behaviours—with, for example, impulsivity being associated with RA (and not PA) in sample of adolescent boys (Raine et al., 2006). However, this literature has also revealed that the overlap is certainly less than unity with RA explaining unique variance in impairment and problem behaviour in the classroom over and above both attention problems and impulsivity (Dodge et al., 1997; Waschbusch et al., 1998). This leads to the possibility of divergent underlying etiological characteristics. Thus a third aim of the current objective is to examine concurrent and prospective etiological links between impulsivity and RA. The lack of research in this area precludes any specific expectations in regards to the relative etiological independence of this pathway.

2.4.3.2.2 Aim 2a. Contribute data on etiological relationships between hyperactivity, oppositional behaviour and proactive aggression

2.4.3.2.2.2 *Assessing the hyperactivity-to-proactive aggression risk pathway*

A recent large scale collaborative study, involving six sites, questions any unique

role of hyperactivity/impulsivity in long-term ASB outcomes (Broidy et al., 2003). At the same time, the frequent association between hyperactivity, in particular, and serious long-term aggression is unlikely to be spurious. Raine et al., recently found that childhood hyperactivity predicted adolescent PA (and not RA) and speculated that this may represent a unique risk pathway associated with later psychopathy. This latter analysis raises the possibility that the longitudinal pathways between hyperactivity and PA shows some etiological independence after accounting for the longitudinal effects of RA. Such a finding would have implications for the sequential hypothesis (Vitaro & Brendgen, 2005).

2.4.3.2.2.3 *Evaluating independent versus shared pathways involving hyperactivity, oppositional behaviour and proactive aggression*

The proposed hyperactivity-to-PA pathway raises empirical questions concerning PA's putative role in the widely cited ODD-to-CD risk pathway (Pulkkinen, 1996; Vitaro et al., 2002). From the perspective of facilitating better early identification of problem behaviour, a preliminary question concerns the the etiological independence versus overlap between hyperactivity, ODD and PA in refining clinical markers of risk. Evaluating these relationships represent a final aim of the current objective.

2.4.4 Study 2b Research Objectives (1): Evaluate Online Technologies for Obtaining Genetically Relevant Information on the Neuropsychological Features of Aggression Subtypes

This research objective has two related aims. Firstly, novel methodology is used in an attempt to obtain a large sample of genetically informative data via the implementation of online data collection technologies. This includes the adaption and implementation of two computer-based neuropsychological tasks and the creation of

a relatively simple web-based caregiver-rated behaviour questionnaire. The primary aim is to determine the viability and relevant methodological issues of using online methodologies in behaviour genetic/endophenotype research. A second aim, which is contingent on successful outcomes associated with the first aim, is to analyse resultant neuropsychological data in an attempt to inform putative endophenotypic models of RA and PA.

2.4.5 Structure of Thesis Chapters relating to Research Objectives

For the benefit of clarity, Chapter 5 presents results from the two main objectives (and related aims) associated with Study 1. Chapter 6 deals with the main objective (and related aims) underpinning Study 2a. Chapter 7 considers results and implications derived from the Study 2b objective. Below is a table outlining the research objectives and aims for easy reference.

Table 2.5

Research Objectives and Aims

Study	Objective
1	(1) **Evaluate the genetic architecture of aggression subtypes in a large population sample**
	Aim 1 Assess for differences in architecture across childhood and adolescent cohorts
	2 Evaluate shared environmental effects in a large scale population sample
	3 Assess for sex differences in aggression subtypes
	(2) **Evaluate etiological relations between aggression subtypes and disruptive behaviour disorders**
	Aim 1 Assess common versus unique etiological relations between aggression subtypes, hyperactivity, impulsivity, ODD and CD
2a	**Assess etiological contributions to continuity in aggression subtypes (including contributions associated with disruptive behaviour disorders)**
	Aim 1 Evaluate the degree to which RA predicts future PA in both childhood and adolescent cohorts
	2 Assess whether common versus unique etiological relations between aggression subtypes, hyperactivity, impulsivity, and ODD contribute to continuity in aggression subtypes over time
	2a: Contribute data on the question of distinct impulsivity pathways to RA
	2b: Contribute data bearing on distinct hyperactivity and ODD pathways to PA
2b	**Evaluate use of online technologies to obtain genetically relevant information on the neuropsychological features of aggression subtypes**

Chapter 3.
Methodology

3.1 Overview

This chapter provides information concerning the methodology and research design underpinning the current research. The first section describes the broader research context within which the present series of studies were undertaken. A description of participant characteristics follows. The process of implementation (i.e., the design) of the two main studies is then outlined in the 3.4 (p. 87). Next, a number of consecutive sections detail the assessment protocols used and the measures derived from these instruments. The remainder of the chapter introduces some technical aspects of behaviour genetics modeling.

3.2 The Australian Twin ADHD Project

The Australian Twin ADHD Project (ATAP) is a large-scale nation-wide twin research program established by David Hay (Curtin University) and Florence Levy (University of New South Wales) to examine etiological characteristics of ADHD with a focus on quantitative and molecular genetics (Hay, McStephen, Levy & Pearsall-Jones, 2002). The high participation rates as well as the longitudinal scope associated with ATAP has seen the program make significant contributions to the field in a range of areas (Bennett et al., 2006). In addition to its elucidation of ADHD (Hay, McStephen & Levy, 2001; Levy & Hay, 2001; Levy, Hay, McStephen, Wood & Waldman, 1997; Rasmussen et al., 2002), the program has shed valuable light on common comorbid conditions such as reading disability (Sheiki, 2008), substance use disorder (Yewers, 2006), developmental coordination disorder (Piek, Pitcher &

Hay, 1999) and agression-related disruptive behaviour disorders, i.e., ODD and CD (Waldman et al., 2001). Particularly relevant to the current research is the program's contribution to debate concerning the clinical classification of disruptive behaviour symptomatology (Hay et al., 2002; Levy et al., 2006; Rasmussen et al., 2004; Stevenson et al., 2005).

ATAP has been running for almost two decades and has included four Waves of data collection (Levy & Hay, 2001). The first three Waves constitued a large-scale longitudinal study involving a cohort of over 2400 participant families of twins. Details of this longitudinal study can be found in (Bennett et al., 2006; Hay et al., 2002; Levy & Hay, 2001).

Wave four involved a second nation-wide cohort derived also from the Australian Twin Registry (ATR) alongside other contributing sources including the Western Australian Twin Registry (Bennett et al., 2006). The ATR is a national voluntary database of twins and higher-order multiple birth families born in Australia. This government-funded organisation enlists families of twins from a number of sources including the Australian Multiple Birth Association, maternity hospitals and other medical centres, schools, and community promotion. Through its affiliation with widely published research groups such as the ATAP team, the ATR has provided valuable clinical information on the epidemiology and etiology of a vast array of human disease and disorders (Hopper, 2002). This latest Wave of the ATAP study is described in (Bennett et al., 2006; Levy & Hay, 2001).

3.3 Participants

The current data collection started with information provided by families registered with the Australian Twin Registry who were originally contacted by mail to participate in the most recent ATAP study (i.e., Wave four mentioned above). This initial phase was undertaken by the ATAP research team at Curtin, of which the author is a member. Two thousand six hundred and ten of 3500 families contacted ultimately consented to participate in the fourth ATAP data collection occasion and were subsequently sent the Australian Twin Behaviour Rating Scales (Levy, Hay, McLaughlin & Wood, 1996) behaviour questionnaire which included relevant RA-PA items—see below for more details. At the time of the current research project, a total of 2082 of these 2610 participating families had completed and returned their questionnaires.

It is useful to emphasise that the *fourth ATAP data collection occasion* mentioned immediately above, refers to the sequencing of data collection phases relevant to the broader ATAP study but does not reflect the sequencing of data collection phases specific to the current study. When the current study is considered alone, this same Wave of data collection is (re)labelled as *phase 1* data collection and this phase includes *wave one* of the two time-point follow-up component of the current research. Because the first phase of data collection associated with the current research was encompassed by the broader ATAP project, families were only approached to contribute unique data to the current research at phase 2 of current data collection (as explained in more detail in section 3.4 below). Thus, of the 2082 families who had returned questionnaires under the auspices of the ATAP study,

1217 provided further consent to be approached for additional research projects that were ancillary to ATAP, but conducted by the members of the ATAP Curtin research team. All 1217 families were mailed out an information pack requesting participation in the second wave of the present research. The information pack can be found in Appendix B.

Consistent with previous Waves (Hay et al., 2002), eligibility for participation in the broader ATAP research program precluded those families with one or both twins who suffer from a major medical condition, developmental disorder (e.g., Autism or other disorders associated with significant neurological deficits), rare genetic condition or psychotic symptoms. Children born significantly pre-term and with low-birth weight were also precluded from the current research program—as were families who had participated in any previous long-term twin studies (Hay et al., 2002).

3.4 Research Design and Procedure

3.4.1 Research Design

A synopsis of the overall research design and accompanying procedure for the current project is outlined below. Specifically, Figure 3.1 below provides a schematic timeline outlining the types of data collected at the two main measurement occasions occurring respectively in 2006 and 2007.

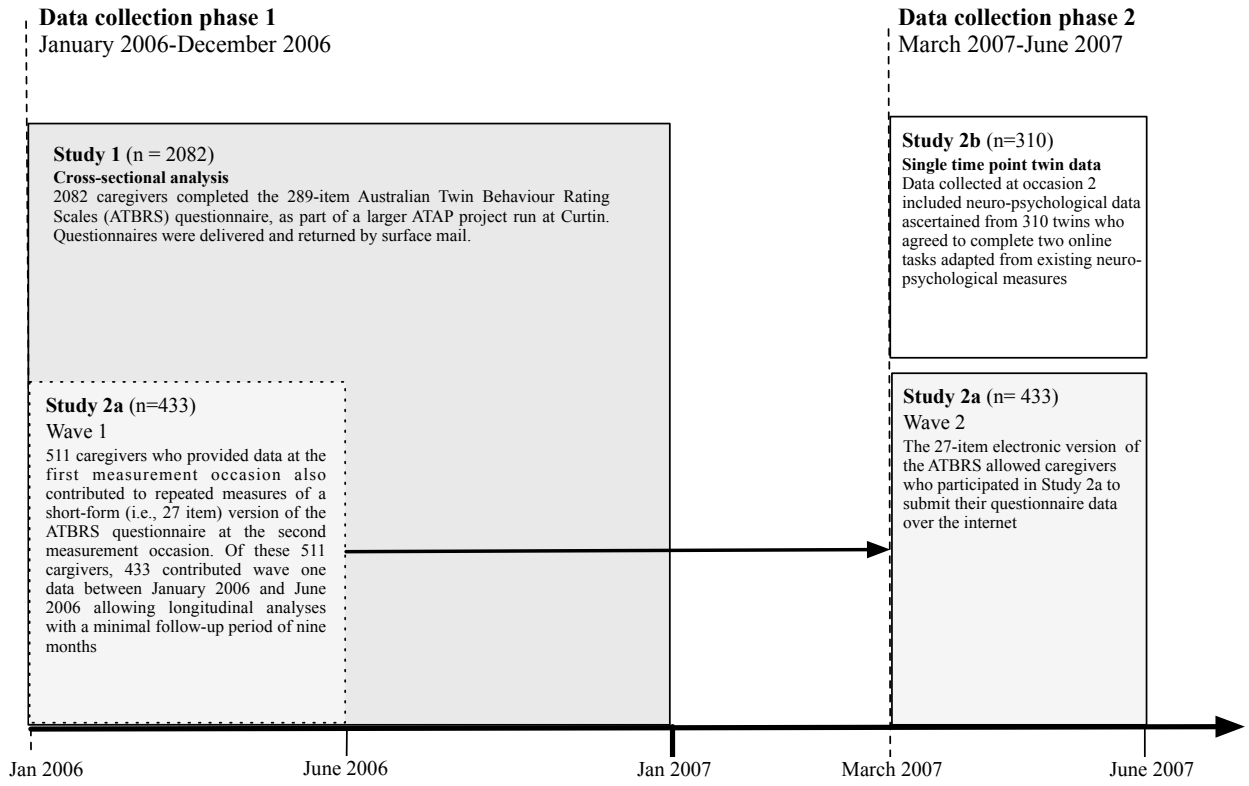


Figure 3.1 Timeline representing the two data collection phases

The repeated-measures component of the research is referred to as Study 2a. The 9-month period chosen for the length of time between measurement occasions represents a modest time-frame compared to typical longitudinal studies of clinically relevant behaviours. However, a key priority for the current design was to provide clarity regarding the presence or absence of shared environmental effects on RA and PA using a repeated-measures design to reduce error variance. Given that both childhood and adolescence are periods which encompass considerable developmental change, it was this important to determine a length of time that would maximally capture both stability (i.e., continuity) as well as developmental change (i.e., discontinuity).

3.4.2 Research Procedure

3.4.2.1 Synopsis of Procedure for Study 1

Three thousand and five hundred families were originally contacted by the ATAP Curtin research team to participate in the ATAP study. At the time of preparations for the current study, 2082 of these ATAP-approached families had returned completed pencil-and-paper ATBRS to the Curtin University research team. Twelve hundred and seventeen of these families returned consent forms confirming their availability to be approached for future studies. The returned questionnaire data was manually entered into local secure database using SPSS statistical software (*Statistical Package for the Social Sciences,* 2002).

3.4.2.2 Synopsis of Procedure for Study 2

The 1217 ATAP families who provided consent to be approached for additional research projects were contacted by mail requesting consent to participate in Study 2. Seven hundred and seventy four families consented to participate in Study 2. Consent was collected via email, while expressions of intent to decline participation were collected by surface mail.

The 774 families who agreed to participate in Study 2 were sent a CD-ROM containing the online applications (i.e., online questionnaire and neuropsychological tasks) presented within a common testing environment (i.e., online interface). Five hundred and eleven families successfully completed the electronic questionnaire via the web-ready interface supplied on the CD-ROM. This interface facilitated the passing of questionnaire data directly into an secure online database (Study 2a).

Additionally, 310 twins provided completed data sets for the two neuropsychological tasks, also delivered online (Study 2b).

Data for studies one and two were analysed using SPSS (Statistical Package for the Social Sciences, 2000), mx (Neale, 1994) and Lisrel (Joreskog & Sorbom, 1993), and results are reported within this text.

It should be noted that the six RA and PA items, described in Chapter 2, were specifically included in the ATAP pencil-and-paper version of the ATBRS questionnaire for the purpose of the current study to acknowledge the increasing importance of RA and PA in the developmental literature and the importance of obtaining data relating to these subtypes from large scale community samples of twins that include multiple age cohorts and at least short-term follow-up.

3.4.2.3 Follow-up Procedures

A series of follow-up contacts were made to ATAP families who had indicated a willingness to be involved in additional Curtin University-based research projects. Specifically, those ATAP families who had provided consent to be contacted for additional studies but who did not respond to the initial approach/information pack requesting participation in Study 2 received follow-up contact a total of two times—in general accordance with Dillman's protocol (Dillman, 1972) where initial and subsequent responses are not received. Consistent with Dillman, the first contact involved the mailout of a follow-up reminder card that requested feedback on families' decision to participate. The subsequent second follow-up involved the mailing of a letter outlining the original request for participation. Unlike the Dillman

protocol, however, the final reminder letter was not sent registered post.

Families who had provided consent to participate in Study 2 but who failed to login to the online assessment environment (within one month of receiving consent) were emailed and asked to provide feedback on their participation status. This latter process allowed the provision of support to families who were experiencing technical difficulties.

3.4.2.4 Ethics Approval Process

Ethics approval for the project was sought and obtained from Curtin University Ethics Committee as well the relevant ethics board at the Australian Twin Registry.

3.4.3 Measures and Instruments by Study and Wave

Table 3.1 below outlines the measures and instruments utilised in each study/Wave.

Table 3.1

Instruments by study and Wave

	data collected 2006	data collected 2007
DBD Measures	*used in:*	*used in:*
Hyperactivity	Study 1, **Study 2a**	**Study 2a**
Impulsivity	Study 1, **Study 2a**	**Study 2a**
Inattention	Study 1, **Study 2a**	**Study 2a**
ODD	Study 1, **Study 2a**	
CD	Study 1, **Study 2a**	
Aggression Measure		
Aggression Scales	Study 1, **Study 2a**	**Study 2a, Study 2b**
N.Psych Measures		
Iowa Gambling Task		**Study 2b**
MultiMorph		**Study 2b**

RA = Reactive aggression, PA = Proactive aggression, DBD = Disruptive behaviour Disorder, ODD = Oppositional defiant disorder symptom scale, CD = Conduct disorder dymptom scale; N.Psych = neuropsychological; Studies in emboldened text denote where measures were obtained via the internet

3.4.4 Procedural Protocols Concerning Medication

A specific note regarding procedure, and one that concerns the implementation of the neuropsychological tasks in particular is warranted. Although it is common to request that participants abstain from psychopharmacological medication in studies that examine phenotypic features of disruptive behaviour, the present study precluded any such request. The rationale for this omission primarily reflects attempts to minimise the burden of participation in the study. ATAP families have shown enormous generosity over the years in agreeing to support ongoing research conducted by Curtin and other ATR-affiliated groups. Many families have engaged in numerous studies and this commitment emphasises the collaborative nature of maintaining a large scale population cohort. It is encumbent on researchers to balance the ideals of methodological rigour with maxmising the likelihood that families will continue to offer their valuable time and input to important scientific

research. Moreover, as ADHD behaviour was not a primary focus of the investigation into the neuropsychological correlates of reactive and proactive aggression, effects of stimulant medication were not deemed a significant confound.

3.5 Determination of Zygosity

Zygosity was determined using a standard questionnaire format. Parents were asked to indicate if there had been DNA testing to determine zygosity. Where no previous testing was reported, parents completed a twin similarity questionnaire (Levy et al., 2001) based on empirically established guidelines (see Cohen, Dibble, Grawe & Pollin, 1975; Nichols & Bilbro, 1966). Results from such questionnaires have been shown to have good agreement with genetic markers (McGuffin, Owen, O'Donovan, Thapar & Gottesman, 1994). The questionnaire contains 12 questions such as "Does their mother ever confuse them in appearance?", "Do they have very similar personalities?", and "Do they have the same blood group?" to assess how similar the twins appear to be in terms of appearance, personality and biology. All opposite-sex pairs were assigned as dizygotic. See Appendix D for the complete zygosity questionnaire used in the ATBRS.

3.6 Behaviour Questionnaires and Neuropsychological Tasks

A number of different assessment protocols were used in the current research. Study 1 involved analysis of data from the ATBRS caregiver-rated questionnaire (Levy et al., 1996) which is a pencil-and-paper protocol delivered to, and returned by families via surface mail. Study 2 comprised two separate methodologies;

- **Study 2a** utilised a short-form 24-item version of the ATBRS adapted into electronic format capable of delivering caregiver-rated data over the internet,

and

- **Study 2b** involved the use of two computer-based neuropsychological tasks adapted to deliver participant data over the internet.

3.6.1 Description of Behaviour Questionnaires

3.6.1.1 Australian Twin Behaviour Rating Scales (ATBRS, Levy et al., 1996)

3.6.1.1.1 Original pencil-and-paper version of the ATBRS

The original long-form pencil-and-paper ATBRS is a 289-item questionnaire designed to investigate disruptive behaviour in twins and their siblings (Levy et al., 1996). It integrates a number of established scales including DSM-IV (American Psychiatric Association, 1994) symptom critieria as well as the six-item Aggression Scales (Dodge & Coie, 1987) indexing reactive and proactive aggression. A range of developmental and learning disorders and perinatal health indicators peripheral to the current research are also included in this tool. The ATBRS also incorporates the zygosity questionnaire described above. Table #.# (p. #) shows the specific combination of behavioral scales (embedded within the ATBRS) utilised in each study.

ATBRS-measured behavioural dimensions relevant to the current research include all three disruptive behaviour disorders, ADHD (including inattentive, hyperactive and impulsive items), ODD, CD as well as RA and PA. Items are rated dimensionally on a four-point Likert scale (0=not at all, 1=somewhat, 2=pretty much, 3=very much). The form allows caregivers to provide behaviour ratings for four children arranged in adjacent columns (see Appendix E for an example page of this questionnaire). The

first two columns are used to provide twin and co-twin ratings with the remaining two columns available for non-twin sibling ratings (if relevant).

Only a subset of the 289 items of the pencil-and-paper ATBRS protocol were used in the current research. This subset included a total of 47 items representing the following behaviour syndromes and dimensions;

- Attention deficit hyperactivity disorder subtypes including:
    - inattentive (9 items),
    - hyperactive (6 items), and
    - impulsive (3 items) behaviours,
- Oppositional defiant disorder (8 items),
- Conduct disorder (15 items) as well as
- Aggression Subscale items including,
    - reactive aggression (3 items) and
    - proactive aggression (3 items) behaviours.

Behavioural criteria for each of the Disruptive Behaviour Disorders are provided below.

In the current study, caregivers were asked to rate their twins (and siblings) according to the frequency of each of the relevant behaviours "now or within the past 12 months". It is noted that this method precludes formal diagnosis and appeals to a broader chronological timeframe than utilised in the DSM-IV for Disruptive Behaviour Disorders. This approach allows a more flexible range of options in identifying problem behaviour. Methods include assessing for presence of symptoms

to either (1) approximate diagnosis and/or (2) provide information of how clusters of symptoms are naturally distributed in the normal population.

In regards to the former method, Levy et al. (Levy et al., 1996; Volk, Todorov, Hay & Todd, 2009) used a *symptom count* approach to analyse ATBRS data and found behavioural criteria agreed adequately with interview data collected using the Diagnostic Interview Schedule for Children. While kappa values obtained for ADHD ($\kappa = .53$), ODD, ($\kappa = .68$), CD ($\kappa = .66$) were modest, they provide a conservative estimate of the number of children meeting criteria for formal diagnoses. Indeed, questionnaires generally provide conservative estimates of prevalence when compared with the diagnostic interview (Hay, 1997; 2001).

An example of the latter approach are "bottom up" approaches that use factor analytic methods to evaluate the natural clustering of individual symptoms in population groups. Notably, the work of Todd and colleagues (Rasmussen et al., 2002; Rasmussen et al., 2004; Todd et al., 2001) have contributed to an evidence base that reveals familial clustering of ADHD subtypes differ in composition from clusters based on clinically defined criteria. This research has informed ongoing debate concerning whether ADHD symptoms cluster in continuous dimesions in the normal population or whether they are better represented by discrete clinically-defined categories. Specifically, there are a number of genetic studies supporting the conceptualisation of ADHD subtypes as continuous, with pathological manifestations representing the extreme ends of these continuums (Hudziak et al., 1998; Levy et al., 1997; Neuman et al., 1999). To an extent, these findings extend to DBD comorbidity (e.g., Faraone, Biederman, Keenan & Tsuang, 1991; Sterba, Egger & Angold, 2007),

although it is noted there is emerging evidence that extreme CD may be discontinuous with less severe CD (Levy et al., 2006).

The current research uses a continuum-based approach with the mean rated frequency of identified problem behaviours (associated with each of the specific DBD disorders and relevant subtypes) used to directly index individual differences in symptomatology. As above, the core assumption associated with this approach is that relevant syndromes (as defined by the DSM-IV) vary, to a significant extent, in the normal population (Levy et al., 2006).

3.6.1.1.2 The Aggression Scales

RA and PA were assessed using the Aggression Scales (AS, Dodge & Coie, 1987; Kempes et al., 2005). The AS contain a total of six items; three indexing RA and three measuring PA (see Table 2.4, p. 18). These items were integrated within the larger ATBRS. While the original AS used a 5-point rating scale (Dodge & Coie, 1987), the current research utilised the four-point Likert scale (0=not at all, 1=somewhat, 2=pretty much, 3=very much) employed in the ATBRS.

3.6.1.1.1.1 *Issues Relating to the Operationalisation of Reactive and Proactive Aggression*

Although a number of different methods for operationalising RA and PA exist (e.g., (Baker et al., 2008; Polman et al., 2007; Pulkkinen, 1996)), a majority of studies have utilised Dodge and colleague's original Aggression Scales.

The AS were originally designed to inform variable-centred research that

distinguished atypical levels of aggression. In this extreme groups approach, RA and PA and non-aggression groups were defined categorically using a cut-off of one standared deviation above the group mean on each sub-trait dimension (Dodge & Coie, 1987; Dodge et al., 1997). However, there has been growing interest in person-centered (i.e., dimensional) approaches in attempts to identify differential risk pathways in RA and PA (Baker et al., 2008; Brendgen et al., 2006; Kempes et al., 2005; Pulkkinen & Pitkanen, 1993; Vitaro & Brendgen, 2005; Waschbusch et al., 1998). In line with this latter approach, the current study investigates individual differences in aggression from both a behaviour genetic and neuropsychological perspective.

Regardless of which method is used, studies have consistently shown substantial co-occurence of subtypes. Specifically, intercorrelations between AS aggression subscales is typically high ranging between .41 and .83, with higher correlations usually associated with population samples (Kempes et al., 2005).

Such empirical overlap has led several commentators to question the clinical utility of the RA and PA distinction (Bushman & Anderson, 2001; Polman et al., 2007; Waschbusch et al., 1998). However, there is growing awareness that despite the RA-PA framework's theoretical prioritisation of function over form, conventional operationalisation of RA and PA (including assessments that utilise the AS) has tended to confound these aspects (Little et al., 2003; Polman et al., 2007). Polman et al. (2007) suggest that this confound may help explain the substantial empirical overlap between RA and PA reported in the literature. Notably, Little (2003) found that after statistically controlling for *form* of aggression (i.e., by distinguishing and

controlling for differences between overt versus indirect relational aggression), the correlation between subtypes disappeared.

Beyond controversy is that conventional measures of RA and PA (such as the AS) have demonstrated a capacity to reliably detect distinct behavioural dimensions. At the measurement level, exploratory and confirmatory factor analyses have been shown to provide a better fit than a single factor solution for RA and PA (Crick & Dodge, 1996; Day et al., 1992; Pellegrini, Bartini & Brooks, 1999; Poulin & Boivin, 2000; Raine et al., 2006). Moreover, numerous studies attest to the construct and discriminative validity of the distinction (Day et al., 1992; Dodge et al., 1997; Pellegrini et al., 1999; Poulin & Boivin, 2000; Price & Dodge, 1989; Pulkkinen, 1996; Raine et al., 2006; Vitaro et al., 2002; Vitaro et al., 1998; Waschbusch et al., 1998). The section below focuses on reliability and validity pertaining specifically to the AS.

3.6.1.1.1.2 *Validity and Reliability of the AS*

Despite the small number of items in this rating scale, confirmatory factor analysis has confirmed the relative independence of the two aggression subtypes (Poulin & Boivin, 2000). Specifically, Poulin and Boivin (2000) assessed RA and PA in a population sample of 149 9- to 11-year-old males using the AS. Chi-square tests of alternative nested models demonstrated the superiority of a two-factor solution over a single factor model. Firstly, the latter model showed a significant deterioration in fit relative to the saturated (i.e., non-constrained) model while the former did not. Secondly, the chi-square for the two-factor model was significantly lower than that of the single factor model ($\chi^2 = 19.68 - 5.57 = 14.11$, $df = 9 - 8 = 1$, $p < .001$). The

authors further demonstrated that the Comparitive Fit Index (CFI, Bentler & Bonett, 1980) and Parsimonious Normed-Fit Index (PNFI, Mulaik et al., 1989) confirmed the two-factor model as the best fit. Determining this statistical independence is critical to establishing construct validity due to the high co-occurrence of RA and PA in community samples (e.g., Dodge & Coie, 1987; Price & Dodge, 1989).

In a second study, Poulin and Boivin (2000) reported teacher- and caregiver-rated AS scores. In this second investigation, which included a demographically similar sample to the first study (n = 193), both the one- and two-factor models differed significantly from the sample data for both rater groups. However, the two-factor solution again revealed a chi-square that was significantly lower than the single factor model—for both rater groups. Moreover, the CFI and PNFI were both higher for the two-factor solution compared to the single factor model (indicating better fit) for both teacher and parent ratings.

Waschbusch et al. (1998) assessed criterion validity associated with the AS. The two criteria used were 'overall impairment' and 'classroom impairment'. The authors confirmed adequate criterion validity of both RA and PA with the former subtype significantly correlated with overall impairment (r = .51) and classroom behaviour (r = .34) after statistically controling for PA. After controlling for RA, PA concurrently predicted overall impairment (r = .16) only. It is useful to note that conventional clinical measures of disruptive behaviour derived from DSM-IV symptomatology were also included to assess the clinical usefulness of the RA-PA distinction. RA, but not PA, was found to provide additional clinical information over and above that determined by conventional clinical measures.

Internal consistency for the two AS subscales is typically high across studies. Poulin and Boivin (2000) reported Cronbach's alphas of .91 for both RA and PA respectively in the first study and .90 and .92 respectively in the second study. This is very similar to figures revealed by Dodge and Coie (1987) who obtained reliabilities of .90 and .91 for RA and PA respectively. In more recent genetic studies, reported Cronbach alphas were .88 and .72 respectively in teacher-rated RA and PA (Brendgen et al., 2006) study, and .83 and .77 for caregiver-rated aggression (Baker et al., 2008).

3.6.1.1.1.3 *Parent Ratings Versus Teacher ratings*

The AS was designed, and is obstensibly used, as a teacher-rated tool (Dodge & Coie, 1987). However, there has been an increasing tendency to use caregiver-rated data in studies of RA and PA e.g., (Poulin & Boivin, 2000). The current study exclusively utilised caregiver-rated data, reflecting the practical limitation of collecting multi-informant data in a large twin samples derived from a population twin registry. This section explores some of the strengths and limitations associated with data obtained from single-source rater input, and caregiver report in particular.

While caregiver report represents a ubiquitious and important source of information, a number of issues have been noted within the behavioural genetics literature (Hudziak, Rudiger, Neale, Heath & Todd, 2000; Neale & Cardon, 1992). A frequently cited concern pertains to rater bias. As previously noted, rater bias refers to the tendency of parents of twins to either under- or over-estimate differences in behavioural characteristics between probands leading to spurious estimates of

etiological effects (Hudziak et al., 2000).

Rater bias that significantly over-estimates behavioural similarity between twins produces inflated **C** estimates, and rater bias that systematically under-estimates similarity in DZ twins can mimic non-additive genetic effects (Eaves et al., 1997a; Hartman et al., 2007; Hay, Bennett, Levy, Sergeant & Swanson, 2007; Hudziak et al., 2000; Sherman, Iacono & McGue, 1997; Simonoff et al., 1998). It is the latter rater bias which has most commonly been associated with parental ratings of hyperactivity (Rutter, 2006). While the mechanisms of rater bias are not well known, one reason often cited as to why teachers tend to be less susceptible to rater bias than caregivers is they have greater opportunity to make more representative comparisons by comparing behaviours amongst a larger group of children (Hartman et al., 2007; Stevenson et al., 2005). There is also evidence that the type of rating scale impacts on rater bias—with conventional DSM-IV based scales being more prone to these effects than some alternative measures (Hay et al., 2007).

As noted above, teacher ratings have generally been found to be less susceptible to rater bias than caregiver report (Eaves et al., 1997b; Hartman et al., 2007; Silberg et al., 1996; Thapar et al., 1995). For example, Hartman et al. (2007) assessed ratings of ADHD symptoms in a population sample of eight to 18-year-old twins and concluded that while differences between parents and teachers are partly due to clinically-valid differences in the types of behaviour that informants reported to have observed (Hartman et al., 2007), they also found some evidence that parents are more biased than teachers in their ratings of ADHD. These authors did not discuss the possibility of sibling interaction in their analyses, which may be more salient in the

home environment where twins have more opportunity to interact freely. The concept of sibling interactions is discussed further below.

At the same time, concerns regarding teacher-rated data have also been raised. For example, Vitaro and Gagnon (1995) found teachers' rating style changed over time as a function of management style—emphasising the fact that reliability as well as the validity of ratings needs to be considered, particularly in longitudinal studies. Additionally, the Hartman et al. study provided a comprehensive evaluation of twin ADHD data where probands were rated separately by different teachers—a design employed in the Brendgen et al. (2006) study. Interestingly, although these authors found that while single-teacher ratings evidenced less rater bias than other informant sources, the separate teacher ratings approach showed substantially greater rater bias compared to both single-teacher and caregiver ratings. Other studies have found that separate teacher ratings lead to artifactually reduced twin correlations (Simonoff et al., 1998; Towers et al., 2000; Vierikko et al., 2004).

Importantly, there is general consensus that rater bias is less common in the field of aggression research (Hudziak et al., 2000; Rasmussen et al., 2002; Vierikko et al., 2004). However, in a recent challenge to this view, Vierikko et al. (2004) reported evidence from a large population based study of 11 to 12 year-olds suggesting the presence of possible rater bias in their best fitting univariate and bivariate models of caregiver-rated aggression and hyperactivity. At the same time, the authors note that their analyses were unable to disentangle rater bias from the possibility of sibling interaction effects.

Sibling interaction (SI) effects are empirically unrelated to rater bias and occur when the behaviour of one twin directly increases or decreases the likelihood of the same type of behaviour in the co-twin. These influences are typically referred to as cooperative versus competitive SI effects respectively (Neale & Cardon, 1992). While the literature suggests that SI effects are not common in aggression research (Vierikko et al., 2004), Vierikko et al. (2004) have reported some evidence for competitive SI effects in undifferentiated aggression. At the same time, it is possible that different aggression subtypes are associated with different sibling interaction effects. For example, a study by Waschbusch et al. (e.g., Waschbusch et al., 2002) illustrates that RA does differentiate boys (aged 9-13 years) with comorbid ADHD/ ODD/CD at lower levels of provocation. This raises the possibility that even at low levels, provocative aggressive behaviour is likely to elicit RA behaviour in conduct disordered children, which may set off a subsequent cycle of reciprocal aggressive interactions that are, by definition, predominantly RA in nature.

Cooperative SI effects mimic **C** influences while competitive SI effects mimic additive-genetic (**D**) effects (Rutter, 2006; Vierikko et al., 2004). Nonetheless, SI effects can be distinguished from genuine **C** or **D** influences because SI effects differentially impact on underlying variances as a function of relatedness (Neale & Cardon, 1992). Specifically, while cooperative SI and genuine **C** effects are both the result of a reduced differential between MZ and DZ correlations, cooperative SI effects lead to greater variance associated with MZ twin behaviour compared to DZ variance. By contrast, genuine **C** effects are predicated on the assumption of homogeneity of variance. Conversely, while competitive SI and **D** effects both result from an increased differential between MZ and DZ correlations, competitive SI

effects are associated with violations of homogeneity of variance such that variance associated with MZ twin behaviour is less than that found for DZ variance.

Modeling SI effects requires covariance matrices, rather than correlational matrices, in structural analysis because correlations standardise the variance of different groups of individuals, thus precluding comparison of between-group variances (Neale & Cardon, 1992). Correlational matrices were preferred in the current analyses because they are generally less susceptible to idiosyncratic deviations in the underyling covariance structure (Hay, 1985). Nonetheless, because SI effects result in deviant variances across zygosity groups, a preliminary test of SI effects simply involves evaluating the assumption of homogeneity of variance as typically conducted prior to model fitting (Jinks & Fulker, 1970; Neale & Cardon, 1992). Put more simply, the presence of SI effects can initially be indexed as a violation of the assumption of homogeneity of variance (Jinks & Fulker, 1970).

It is thus assumed that where basic homogeneity of variance is obtained amongst variables in the relevant analyses, this provides an reasonable indication that SI effects are not present.

The broader issue of interpreting rater agreement versus disagreement also presents somewhat of a challenge to the field of behaviour genetics. It is generally accepted that combining data from multiple informants provides a more comprehensive picture than single source ratings (De Los Reyes & Kazdin, 2005; Miles & Carey, 1997). Unfortunately, it remains unclear how best to integrate divergent response sets within a behaviour genetics context (Baker et al., 2008; Kerr, Lunkenheimer &

Olson, 2007; Simonoff et al., 1998; Simonoff et al., 1995). Certainly, elucidation of multi-informant data requires coordinated research programs to disentangle divergent ratings from multiple sources and settings.

In their analysis of rater effects on ASB (including RA and PA), Baker et al. (2007, p. 219) concluded that "parents, children, and teachers have only a partly 'shared view' and that the additional factors that influence the 'rater-specific' view of the child's antisocial behavior vary for different informants". In their large-scale twin study, inter-rater agreement between teachers and parents for RA (r=.15) and PA (r=.16) was low. However, 81% of the variance of caregiver ratings was attributable to the "shared view" combined index —a considerably greater proportion than found for the other rater types. This suggests that, if only one rater source is available, caregivers will tend to offer the most representative ratings for RA and PA. Certainly, Kempes (2006, p. 44) note the important role of caregiver report suggesting that "in future research into the distinction between RA and PA, the view of parents may be taken into account".

3.6.1.1.3 The ATBRS electronic short-form (ATBRS-ES)

The **ATBRS-ES** was developed specifically for the purposes of the current research. It includes the ADHD items (from all three subscales) as well as the six aggression subtype items from the Aggression Scales (Dodge et al., 1997). These items were chosen on the basis of their relevance to the current research. While it would have been ideal to include ODD and CD items in this follow-up, these syndromes were omitted due to the need to prioritise resources to reflect the core research questions under consideration—namely, the relationship between non-aggressive disruptive

behaviours and aggression. Omission of the ODD and CD items occurred also kept the length of the questionnaire to a minimum and thus reduced the burden of participation on the family.

The questionnaire utilised a number of design principles as discussed by Dillman and colleagues (Dillman, 2007; Dillman & Smyth, 2007). Namely, an uncluttered screen design was used, but one in which relevant instructions and information were readily clear and accessible. See Appendix I for a picture of the online questionnaire interface.

Each questionnaire item was presented alone on the screen, along with two rows of four *radio buttons* (which are similar to check boxes but restrict the user to only one choice from amongst a response set). The series of four radio buttons represented the ATBRS Likert scale and each button was labelled accordingly. The two rows were provided so caregivers could rate both twins, and these rows were thus clearly labeled for Twin One and Twin Two. The ATBRS instructions explain that caregivers should use birth order to ascertain assignment of twins. These instructions were also included in the ATBRS-ES.

Dillman and colleagues (Dillman, 2007; Dillman & Smyth, 2007) discuss the advantages and disadvantages of designing an electronic questionnaire which involves placing multiple questions across successive "pages" or screens versus placing all questions on the same screen. Dillman et al. note it is sometimes less practical to have the user click forward through successive pages as this can limit their ability to scan across the entire questionnaire quickly and make it difficult for

them to know where they are up to in the full sequence of items. The decision to have each item on a separate page reflects the relatively unique nature of this questionnaire. That is, the ABTRS-ES requires caregivers to rate both twins at the same time. Remaining mindful of the possibility that some families may be using small sized monitors (e.g., 15-17 inch screens) it was decided to try and achieve an uncluttered screen with the least possibility for confusion regarding which question pertained to which twin. Having to scroll down a screen can mean that the kind of overarching perspective that one has of an A4 sized questionnaire page is lost.

In an attempt to address some of the issues raised by Dillman et al. a cumulative item count was placed on screen at all times when the questionnaire was being used to help caregivers keep track of how many items had been completed and how many more were left to complete. Also, a function allowed users to "jump" between any two items in a discontiguous manner.

3.6.2 Description of Neuropsychological Tasks

3.6.2.1 Iowa Gambling Task (Bechara et al., 1994)

The Iowa Gambling Task (IGT) is a computer-based task originally designed to assess putative discrete functional pathways and processes associated with the orbital frontal cortex (OFC) in frontal patients (Bechara et al., 1994; Damasio, 1994). Specifically, damage sustained to this area of the brain has been associated with reduced sensitivity to changes in reward/punishment contingencies, as well as with increased reactive aggression (Damasio, 1994).

The task requires the user to pick a series of virtual cards freely from four card decks (labelled A to D) with the aim of winning points. The four decks are presented 'face-down' and the user is asked to pick 100 cards in succession from across the four decks in any order. Every card scores points for the user. The amount of 'winnings' varies across decks with two decks delivering equivalent high winnings and the remaining two decks delivering a more modest amount of points relative to their higher scoring counterparts. Winnings automatically accrue throughout the task. However, some cards additionally incur penalty points. Cards that deliver penalties are distributed in a quasi-random fashion across the four decks and their associated losses are automatically subtracted from the users score (the user is informed of all wins and losses as they occur and a running total is displayed at all times throughout the task).

Card decks A and B hold cards that deliver high rewards (i.e., winnings) but higher penalties (i.e., losses) such that all winnings are ultimately lost if one persists in picking only from these decks. Decks C and D deliver more modest rewards but associated penalties total less than the sum of these rewards. Thus a net gain is possible where selections favour these decks. This latter selection bias represents the optimum long-term strategy for obtaining winnings.

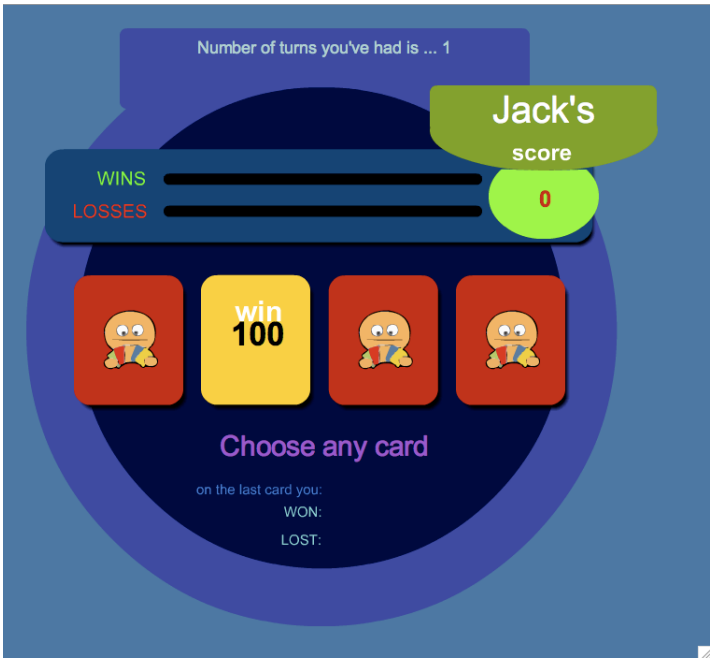


Figure 3.2 Screen-shot 1 of the adapted Iowa Gambling Task

Table 3.2 displays the values and frequencies of wins and losses associated with cards from each deck. Each deck holds a total of 40 cards. Every card from decks A and B scores 100 points, while all cards from decks C and D score 50 points each. Thus absolute winnings for decks A and B total 4000 each while total possible losses per deck is -5000. Decks C and D deliver potential total winnings of 2000 points each with possible losses for each deck equaling -1000 points. As mentioned above, losses are sequenced in a quasi-random manner with penalties assigned to Decks A and C distributed at higher frequencies than decks B and D. Thus, the amount lost in any given penalty delivered by a card in deck A is lower relative to the less frequent penalties associated with deck B. This same differential pattern applies to deck C (high frequency, low value penalities) versus deck D (low frequency, high value penalities).

Table 3.2

Trial structure for Iowa Gambling Task

Trial	Deck A (+100)	Deck B (+100)	Deck C (+50)	Deck D (+50)
1				
2	-150			
3			-50	
4	-300			
5			-50	
6	-200			
7			-50	
8	-250			
9	-350		-50	
10		-1250	-50	-250
11	-350			
12			-25	
13	-250		-75	
14	-200	-1250		
15				
16	-300			
17	-150		-25	
18			-75	
19				
20			-50	-250
21	-300	-1250		
22				
23	-350			
24			-50	
25	-200		-25	
26	-250		-50	
27	-150			
28				
29			-75	-250
30	-350		-50	
31	-200			
32	-250	-1250		
33				
34			-25	
35			-25	-250
36	-150			
37	-300		-75	
38				
39			-50	
40			-75	

Healthy adults have been shown to develop a tendency to favour selecting from the

'safe' decks (i.e., decks C and D) by around the 40th trial (Manes et al., 2002). Suboptimal responding (i.e., favouring decks A and B throughout the duration of the task) has been associated with patients who have sustained orbitofrontal damage (Bechara et al., 1994; Damasio, 1994). Indeed, OFC (along with the dorsolateral prefrontal cortex) has been implicated in a neural substrate that mediates performance on the IGT (Bechara, 2004; Bechara et al., 1994; Bechara et al., 2005; Bolla et al., 2003; Manes et al., 2002).

OFC function, as indexed by this task, has been found to correlate with psychopathy (Blair, 2004; Blair, Colledge & Mitchell, 2001; Blair & Cipolotti, 2000; de Bruin, van Oyen & Van de Poll, 1983; Eslinger, Flaherty-Craig & Benton, 2004; Happaney et al., 2004; Kerr & Zelazo, 2004; Manes et al., 2002; Rolls, 2004; van Honk, Hermans, Putman, Montagne & Schutter, 2002) as well as increased RA (Best, Williams & Coccaro, 2002). At least one study indicates that attentional capacities may mediate the relationship between psychopathy and poor performance on the IGT (Lösel & Schmucker, 2004). Malloy-Diniz, Fuentes, Leite, Correa and Bechara (2007) showed that adult participants with ADHD evidenced errors on the IGT. These latter findings relate predominantly to the impulsivity component of ADHD. The task has also shown to be sensitive to measures of impulsivity in normal adult populations (Franken, van Strien, Nijs & Muris, 2008; Sweitzer, Allen & Kaut, 2008). Interestingly, associations between task performance on the IGT and a gene variant associated with serotonin production have been reported (Homberg, van den Bos, den Heijer, Suer & Cuppen, 2008; Maurex et al., 2008; van den Bos, Homberg, Gijsbers, den Heijer & Cuppen, 2008).

The above outcomes are tempered by evidence supporting the argument that the IGT confounds expectations of long-term outcomes on the one hand, and gain-loss frequencies on the other (Chiu et al., 2008). Also, education level seems to have a strong effect on task scores in adults (Davis et al., 2008). Perhaps more relevant to the current research is the argument that use of the IGT in child and adolescent populations is sensitive to changes in brain function that occur due to normative maturation processes (Crone & van der Molen, 2004; Hooper, Luciana, Conklin & Yarger, 2004; Huizenga et al., 2007). For example, using a simplified version of the IGT (Kerr & Zelazo, 2004) showed that a community sample of 4-year-old children outperformed their 3-year-old counterparts. This effect was more pronounced for female participants. Interestingly, sex differences in IGT scores have also been found in adolescent populations (Hooper et al., 2004; Overman, 2004) but not in mid-to-late childhood samples (Overman, 2004). The apparent stratified effects of age on sex differences notwithstanding, Hooper et al. (2004) suggest the kind of normative developmental maturation of frontal function indexed by the IGT continues well into late adolescence and early adulthood. Nonetheless, Blair et al. (2001) was able to show that boys (aged nine to 17 years) who scored highly on the Psychopathy Screening Device (Frick et al., 2000) also showed deficits on this task relative to non behaviour disordered peers.

Given the widespread use of this task, the lack of available reliability analyses related to the IGT is somewhat surprising. However, this tends to reflect the primary purpose for developing the task, i.e., to assess patients with specific frontal injuries—an inherently complex field of inquiry. Nonetheless, (Bechara et al., 1994) provide response patterns typical of healthy adult participants versus patients with OFC

deficits. This paper illustrates well a consistent finding in both the neuroscience and developmental literatures suggesting that later trials of the IGT are more sensitive to the relevant effects (that is, optimal versus suboptimal responding) as might be expected given that the IGT is effectively an implicit learning task.

3.6.2.1.1 Deriving an IGT measure

In the current study, a single IGT score was calculated for each participant. This score was derived by combining the total number of cards chosen from decks A and B and then subtracting this figure from the total number of cards chosen from decks C and D combined. This simple method provided an index of the degree to which participants chose from the safe decks moderated by the degree to which they selected from the unsafe decks.

3.6.2.2 The Emotional Expression Multimorph Task (Blair, 2001)

The Emotional Expression Multimorph Task (Blair et al., 2001; Coupland, Singh, Sustrik, Ting & Blair, 2003) is a computer-based protocol originally designed to evaluate the purported association between developmental psychopathy and selective deficits in processing sad and fearful facial expressions of emotion (Blair et al., 2001). It was devised as an alternative to traditional facial emotion recognition paradigms that involve showing participants photos of faces depicting archetypical expressions of emotion. One difficulty with this conventional approach is that most children become highly proficient at recognising basic archetypes of emotions by around middle childhood (Williams et al., 2009). As such, this traditional paradigm is relatively insensitive to individual differences in capacity for perceptual discrimination of emotional cues in late-childhood onwards (Blair et al., 2001).

The Emotional Expression Multimorph Task, or Multimorph (MM) for short, involves showing participants a series of photographs of faces in succession which gives the illusion of dynamic change from a neutral expression to display one of six emotional expressions (happiness, sadness, fear, anger, disgust and surprise). Figure 3.3 (p. 116) shows an example series of photos displayed in a specific trial that involves the morphing of a neutral facial expression into a fear expression.

Digital morphing procedures are used to create trial sets of 40 photographs that range in intensity (from 0% to 100% in 2.5% increments) for each emotion (Dent, 2001)—see also (Pollak & Kistler, 2002) for a similar approach. Each emotion is depicted separately by six different facial identities or actors (three adult males and three adult females) derived from Paul Eckman's frequently used photograph set (i.e., *Pictures of facial affect,* 1976). This results in 36 trial sets involving a total of 1440 photographs (i.e., 40 photographs per trial set)—72 photographs originally developed by Eckman and Friesen (1976), and 1368 photographs representing intermediate (digitally modified) versions of the originals.

As above, the first photograph in each trial set (i.e., 40 picture sequence) displays one of the six actors displaying a neutral expression (i.e., 0% emotion). Each subsequent photo, from the first to the last in the trial set, depicts incrementally increasing intensity of facial emotion from 0% to 100% intensity in 2.5% steps. The final photograph depicting 100% intensity of emotional expression represents the original Ekkman photograph of the relevant emotion (and actor). The initial 0% intensity photograph is also derived from the Ekkman set which includes neutral

emotion depicted by each of the actors.

Figure 3.3 Sequence of 40 photographs digitally manipulated to represent graded intensity of fear emotion in 2.5% increments.

The participant is presented with a static picture of a face model (selected by the computer program randomly) displaying neutral expression picture—see Figure 3.4. A trial commences when the user clicks a start button onscreen which triggers the sequential presentation of a trial-set of 40 photographs. Each of the 40 photos in the sequence is displayed for a duration of 500 milliseconds. Each trial thus lasts for a total of 20 seconds. From the participant's perspective the sequence of photographs resembles a slow motion video of a face morphing from a neutral emotional expression into an emergent emotional expression.

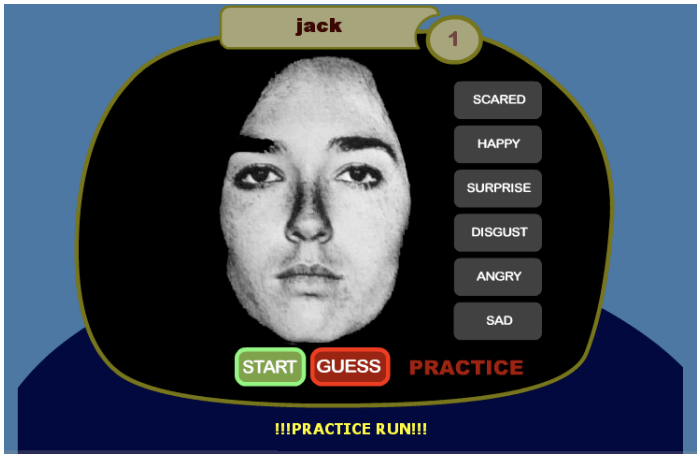


Figure 3.4 Screen-shot 1 of the adapted MultiMorph task (initial neutral expression).

The participant is asked to guess as soon as they think they know what the emerging emotion is. Figure 3.5 shows an example of a what the participant would see if they chose to stop the stream of photos (for the purpose of making a guess) while the 35% emotion photo was being displayed. If correct, the participant is scored according to the percentage of intensity at which the response was given. If incorrect, no score is awarded for the trial.

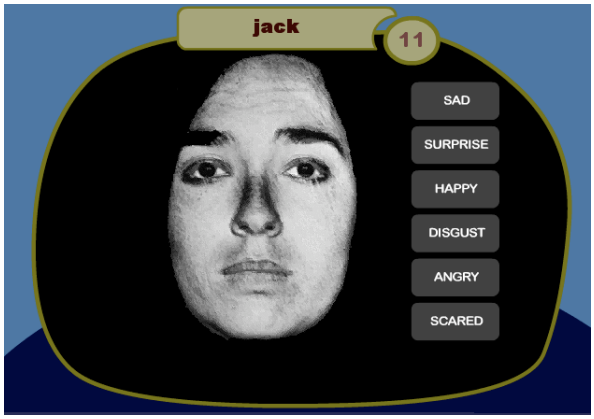


Figure 3.5 Screen-shot 2 of the adapted MultiMorph task (35% fear expression).

Like the IGT, there is no published information regarding the reliability and validity of the MM task. This likely reflects the relatively specialised purpose for which the

task was developed as well as the relatively sparse use of the paradigm in the literature. Nonetheless, a number of studies have reported successful use of the task (Blair et al., 2001; Blair & Curran, 1999; Coupland et al., 2003). Particularly relevant to the current research is the finding that children rated high on the Psychopathy Screening Device (PSD, Frick et al., 1994) required higher intensity emotional stimuli (relative to peers rated as low on the PSD) before achieving correct recognition on the MM task.

The MM task was adapted in a number of ways for the current research. Firstly, an effort was made to reduce the length of the task in order to assist participants in maintaining concentration. This was done because (1) the task was designed to be delivered over the internet and hence self-administered in the home environment—a method which precludes a qualified administrator present to monitor and encourage task engagement during administration, and (2) participant twins with ADHD were not asked to cease medication during the testing period. To achieve shorter task duration, a number of changes were made. Firstly, the number of emotions was reduced to the four principal emotions; happy, sad, fear and anger. Secondly, the duration of each of the 40 photos in each trial set was reduced to 200 milliseconds (from the 500 milliseconds used in previous implementations). Thus a totla of 960 photos were used and each trial lasted for a possible total of eight seconds rather than 20 as in the original specification.

Following Coupland et al. (2003), once a participant made their selection on a given trial, the 40th photo in that trial set (representing the full archetype emotion) was immediately displayed (indefinitely). The participant was then asked to name the

archetype emotion—effectively offering the participant a chance to confirm their most recent selection. This latter component simply represents a conventional emotional recognition paradigm and, in the current research, was included to provide additional information over and above that provided by the perceptual sensivity aspect of the task (for the purpose of testing the reliability and validity of the online task). Once the user makes this second confirmation selection of this static picture, the trial is ceased and the next trial is presented. The process is repeated until all 24 trials are complete.

3.6.2.2.1 Deriving the MultiMorph measure

Firstly, accuracy scores were determined for each of the four emotions; happy, sad, fear and anger. Establishing an accuracy score involved calculating the number of trials for which each respective emotion was correctly identified and dividing by the total number of trials for each emotion. As above, each of the four emotions was displayed a total of six times (i.e., once each with the six different actors). As a result, all total accuracy scores were divided by six. This accuracy score was then multiplied by 100 to maintain percentile scaling.

A sensitivity index was then ascertained for each emotion by dividing the accuracy score by the average percentage of intensity at which correct trials were achieved (and multiplying the result by 10 to maintain an easy to read score). Thus, *perceptual sensitivity* as used in the current research context represents a measure of accuracy for facial emotion recognition that is penalised by higher levels of intensity required to obtain that accuracy. To illustrate, two participants who both correctly identify on five out of the six trials involving facial expressions of sadness (83.3% accuracy)

will differ in perceptual sensitivity where one participant required those expressions to be displaying (on average) only 75% intensity where the other required to see up to the 40th photo (100% intensity) before correct recognition occurred. In this example, the former participant's sensitivity index score would be 83.3/75 (x 100) = 111 while the other would score lower, i.e., 83.3/100 (x 100) = 83.3.

The final score used in subsequent analyses was derived by the following process. Firstly, the sensitivity index scores for the distress related emotions (i.e, sad and fear indices) were combined to form a composite *distress cue* sensitivity index score. Next, the two sensitivity index scores for the non-distress related emotions (i.e., happy and anger indices) were combined to create a *non-distress cue* sensitivity index score (see Figure 3.6).

$$\textbf{\textit{distress cue}}\ \textbf{SIS} = SIS_{sadness} + SIS_{fear}$$

$$\textbf{\textit{non-distress cue}}\ \textbf{SIS} = SIS_{happy} + SIS_{angry}$$

Figure 3.6 Formula for deriving distress and non-distress cue scores. SIS denotes sensitivity index score

A difference score was then obtained by subtracting the distress cue sensitivity index from the non-distress cue sensitivity index. Where resulting set of difference scores possessed a negative value, all difference scores were adjusted by adding the lowest negative value to all scores. This ensured no negative values in the final difference index. Finally, the distress cue sensitivity index score was divided by the difference score (see Figure 3.7).

***distress cue* SIS**

***distress cue* SIS — *non-distress cue* SIS**

Figure 3.7 Formula for deriving the selective sensitivity index

This approach provided a distress sensitivity index that was moderated by divergence between perceptual sensitivity to distress cues (i.e., sad and fear cues) versus non-distress cues (i.e., happy and angry cues). This final index was called the selective sensitivity index (SSI). This latter refining of the final score was included because failing to integrate information regarding sensitivity to non-distress cues would preclude the ability to comment on the theoretical proposition of a selective distress cue-related deficit (e.g., Blair et al., 2001).

3.7 Behaviour Genetic modeling

A brief overview of statistical conventions and related theoretical assumptions associated with behaviour genetic analytical methods is provided below.

3.7.1 Univariate Behaviour Genetic Model

It is useful to introduce BG methodology be describing the initial classical approach to estimating heritability. This approach involves calculating correlations between relatives that can be used to derive ratios representing relative genetic and environmental influence (Jinks & Fulker, 1970). Specifically, genetic effects are implied when the magnitude of the MZ correlation is signficantly greater than the DZ correlation (with the previously mentioned qualification that the DZ correlation must be at least half the MZ correlation to assume *additive* genetic effects). The magnitude

of the genetic contribution is estimated by doubling the difference between MZ and DZ correlations, i.e., 2 x ($r_{MZ} - r_{DZ}$). Shared environment is estimated by twice the DZ correlation minus the MZ correlation, i.e., (2 x r_{DZ}) – r_{MZ}.

Biometrical genetical approaches represented a significant advance in the sophistication of statistical methods to estimate genetic versus environmental effects (Jinks & Fulker, 1970). This latter approach explicitly recognises *polygeny* which refers to the effects of multiple genes (across different loci) combining to influence an observed trait or phenotype. The biometrical model predicts that complex polygenic traits will be continuously distributed in the normal population. This approach opened up the field to the use of a broader range of parametric statistical techniques which fundamentally involved partitioning within- and between-family variance in order to estimate the relative proportions of phenotypic variance that result from genetic and environmental influences (Evans, Gillespie & Martin, 2002).

The variance components defined by biometrical genetics form the crude basis of the model fitting strategy. Indeed, one of the most important legacy of this approach is Jinks' formulation of the "maximum likelihood" statistic which extended the concept of likelihood initially introduced by Ronald Fischer (Neale & Cardon, 1992). This reflects the fact that contemporary quantitative genetic methods estimate model parameters but they also estimate the precision of the model being evaluated. That is, goodness-of-fit indices are used to evaluate the degree to which the observed model deviates from expected twin covariances as specified by the biometrical model of quantitative inheritance (Neale & Cardon, 1992).

Current methods represent the integration of biometrical genetical model fitting approaches and factor analytical methods that allow the specification of (directional) pathway models (Neale & Cardon, 1992). Specifically, this latter area provided a means to analyse *covariance structures* which could then be integrated within the biometrical model described above. These linear structural models constitute a more statistically sophisticated representation of the variance components defined by the biometrical genetic model. As Neale and Maes (1992, p.73) explain,

> "The advantage of the path method is that it goes beyond measuring the degree of association by the correlation coefficient or determining the best prediction by the regression coefficient. Instead the user makes explicit hypotheses about relationships between the variables which are quantified by path coefficients."

Key aspects of the path structure are dictated by the fundamental predictions of the twin model. Specifically, in the univariate case involving additive genetic effects, the genetic (**A)** component is defined within the covariance structure as being equal for MZ twins (r=1), and of half-magnitude (r=.5) for DZ twins. This statistically represents the fact that MZ twins share all their DNA while DZ twins share on average only half their DNA (Hay, 1985). Because shared environmental effects are assumed to be equivalent for all twins and siblings, the shared environment (**C)** term is specified as equivalent (i.e., set to 1 in the model) for both zygosity groups. The no-shared environment **(E)** term represents the remaining variance unaccounted for by **A** and **C** terms (encompassing both non-shared environmental effects as well as

model error). Together, this model is represented statistically as $1 = (1)a^2 + (1)c^2 + e^2$ for the MZ correlation and $1 = (.5)a^2 + (1)c^2 + e^2$ for the DZ correlation. Thus, non-shared environment effects are approximated by the degree to which the twin correlation is less than 1, i.e., $e^2 = 1 - a^2 - c^2$.

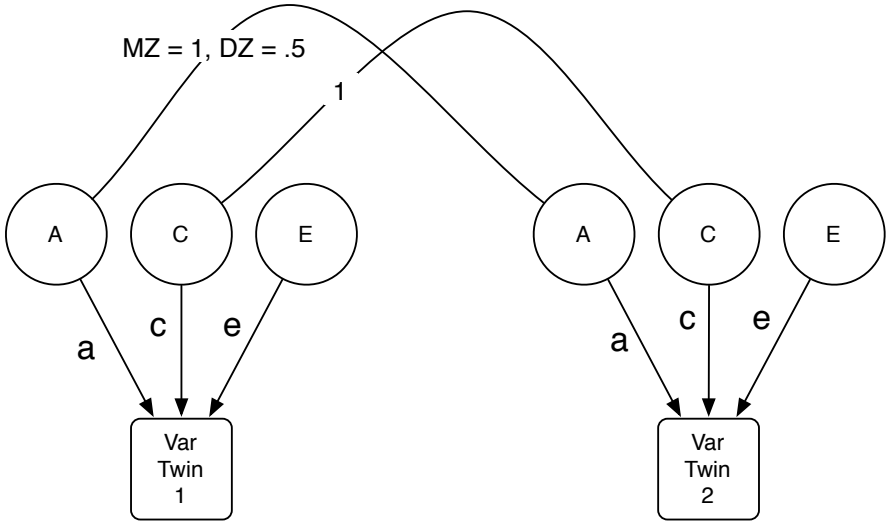


Figure 3.8 The basic univariate pathway diagram for the classical twin design. MZ and DZ denote monozygotic and dizygotic twins respectively. Var denotes variable. A denotes the latent variable representing genetic effects. C denotes the latent variable representing shared environmental effects. E denotes the latent variable representing non-shared environmental effects. a, c and e represent pathway coefficients for their respective latent variables.

The labeling of pathway coefficients in both Figure 3.8, and Figure 3.9, is instructive. Pathway coefficients which share the same label within each of these diagrams, indicate these paths are constrained to be equal. These coefficients provide estimates of the degree to which the latent variables A, C, and E (i.e., etiological factors) load on the variable(s) of interest. As indicated by the equations presented immediately above, squaring the estimates derived from these path coefficients produces the proportion of variance attributable to each respective latent factor.

3.7.2 Multivariate Behaviour Genetic Model

The multivariate model extends the univariate model by including the decomposition of covariance into cross-trait and trait-specific components. In this expanded model, twin 1's score on each variable is considered in relation to his or her co-twin's score on each of the covariates. Genetic effects that simultaneously operate on multiple traits are implied if the cross-trait MZ correlation exceeds the DZ cross-trait correlation. Heritability estimates for cross-trait effects are thus derived using the same basic set of formulas that define the univariate model above. Likewise, environmental effects common to multiple traits are implied if the magnitude of the MZ cross-trait correlation is less than twice the DZ cross-trait correlation. Also, similar to the above, nonshared environmental effects are estimated as the degree to which cross-trait correlations are less than unity. Estimates of trait-specific effects derive from the application of these same standard formula to residual intra-class correlations. Residual intra-class correlations preclude the effects of cross-trait covariation.

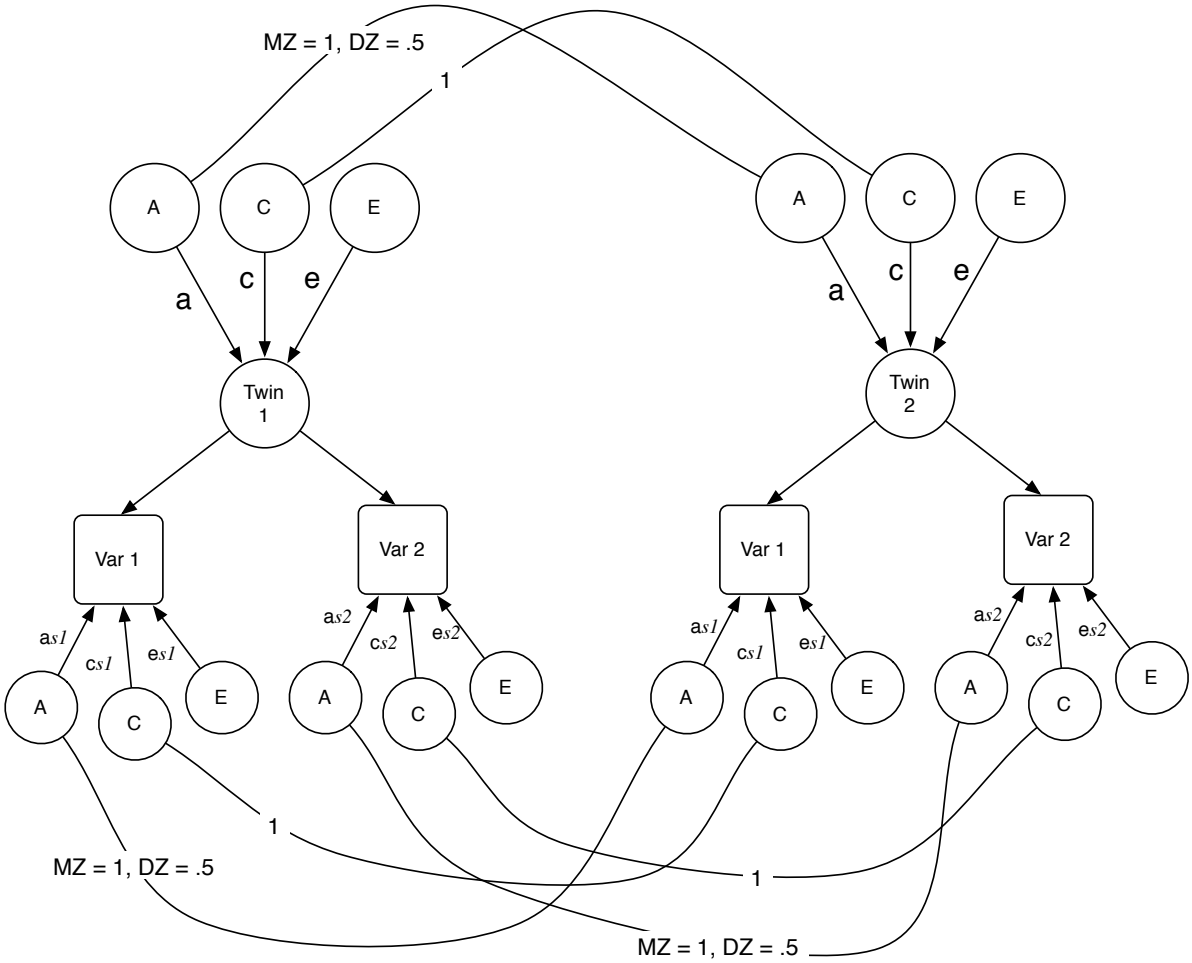


Figure 3.9 The basic bivariate pathway diagram for the classical twin design. MZ and DZ denote monozygotic and dizygotic twins respectively. Var denotes variable. A, C and E denotes latent variables representing cross-trait effects. a, c and e represent pathway coefficients for their respective latent variables. S denotes trait-specific variance components.

3.7.3 Quantitative Genetic Model Fitting

As indicated above, use of structural equation modeling (i.e., path analysis) enables researchers to evaluate the relative fit of multiple alternate models and thus test hypotheses about the relative importance of the three sources of variance noted above. In behavioural genetic research, this usually involves testing a set of nested models by systematically excluding one or more of the three main sources of variance (i.e., **A**, **C** and **E**) from the *full* (i.e., saturated) model to assess the most

parsimonious explanation of the data. In multivarite path analysis, individual parameters representing specific types of effects (i.e., **A**, **C** and **E**) operating on trait-specific versus cross-trait covariance are retained or removed to determine the best fitting model.

3.7.3.1 Goodness of Fit Indicators

Where similar acceptable fit estimates are obtained, parsimony ultimately favours a model with less parameters over one with a more complex specification (Cudeck & Henly, 1991). Model fit is ascertained using a set of *goodness of fit* indicators that typically provide information on which path structure best describes the data. In the current analyses three complementary fit estimates are used; the Chi Square (χ^2) staistic, Akaike's Information Criterion (AIC), and the Root Mean Square Error of Approximation (RMSEA) (Neale & Cardon, 1992). Each are described in turn below.

3.7.3.1.1 Chi Square Statistic

Chi-square (χ^2) is frequently used in behaviour genetic modeling to test the null hypothesis, i.e., that the refined (or target) model does not differ significantly from the saturated model. The χ^2 statistic is the product of the sample *df* and the F value derived from the Maximum Likelihood procedure (Kline, 2005). Nonsignificant χ^2 values ($p > .05$) indicate good model fit. In a related way, **s**maller χ^2 values are generally associated with better fit. Unlike the two goodness of fit indices discussed below, the χ^2 value approximates a central chi-square distribution (Kline, 2005). Limitations of this index include the fact that as a statistical benchmark it is based on

the implausible assumption of perfect fit. It is also susceptible to sample size. That is, larger sample sizes tend to increase the risk of rejecting the target model on the basis of relatively trivial failures of fit (Dick et al., 2005). It is noted that smaller sample sizes may have the opposite, but equally undesirable effect of increasing risk of false positives. Finally, this statistic cannot directly compare two competing models that do not share hierarchically nested structures. However, use of chi square is ubiquitious in the literature due to the fact that it forms the basis of many supplemental indices (Kline, 2005) such as the AIC index described below.

3.7.3.1.2 Akaike's Information Criterion (AIC)

The AIC statistic is used to compare the difference between the chi-square of the saturated and nested alternative models. The nested model with the smallest AIC value is considered to have superior fit (Dick et al., 2005). The statistic is *parsimoniously-adjusted* such that it favours less complex models over more complex ones (Kline, 2005). It is chi-square distributed with degrees of freedom (*df*) calculated as the difference in *df* of both comparison models (Van den Oord, Boomsma & Verhulst, 2000). Like the chi-square statistic, the AIC index is susceptible to sample size and so is commonly reported alongside the RMSEA statistic which is not (McDonald, 1989). The RMSEA is described immediately below.

3.7.3.1.3 Root Mean Square Error of Approximation (RMSEA)

RMSEA is based on a noncentral chi-square distribution (as is the AIC index) and thus does not require a true null hypothesis (Kline, 2005; McDonald, 1989). However, unlike the AIC index, the RMSEA does not assume that the fit of the

model in the population is perfect. In this way, the latter test does not validate the target model against a 'perfect fit' and thus remains unaffected by sample size. Additionally, the RMSEA is *parsimoniously-adjusted* meaning that where two models possess similar explanatory power, the more simple model is favoured by virtue of a 'built-in" correction that penalises greater complexity of specification (Kline, 2005).

RMSEA is calculated as follows (Kline, 2005):

$$\text{RMSEA} = \sqrt{\frac{\hat{\delta}_M}{df_M\,(N-1)}}$$

The M signifies "model" and the non-centrality parameter (i.e., δM) is calculated as the largest of the following two possible values; χ^2_M- df_M or 0. RMSEA estimates of < .05 reflect good model fit. Estimates falling between .05 and .1 indicate adequate fit, while estimates falling below .05 indicate very good fit (Hudziak et al., 2000). Conversely, estimates above .1 indicate poor or inadequate fit.

The model fitting process is explained in further detail in Chapter 5. This includes the application of the relevant concepts within the context of data analysis.

Chapter 4.
Sample and Scale Characteristics

4.1 Overview

This chapter provides sample characteristics and descriptives for data obtained at each of the two main measurement occasions. As detailed in the previous chapter, Study 1 data was derived from the pencil-and-paper protocol version of the Australian Twin behaviour Rating Scale (ATBRS, administered as part of a large research project at Wave One). Study 2 data was collected exclusively over the internet and involved two separate online protocols.

The first protocol was an electronic short-form version of the caregiver-rated ATBRS. The second protocol represented two neuropsychological tasks adapted for delivery over the internet. Both these protocols were embedded in a software package delivered on CD-ROM to participant families. This integrated software allowed families to use their own home computers to login to a secure database which collected and temporarily stored all participant data.

A note about the chronicity of the longitudinal data. As noted in Chapter 3, data from the Wave One phase was collected over approximately nine months leading up to December 2006. The second data collection phase occurred between March and June 2007. In order to ensure at least a 9-month period between measurement occasions, only families who submitted wave one data prior to July 2006 were chosen for inclusion in Study 2a analyses (n = 411).

4.2 Sample Characteristics for Study 1

Study one participants initially included 2082 families registered with the Australian Twin Registry (ATR, http://www.twins.org.au) who were concurrently participating in a large population-based research project coordinated by Curtin University (Bennett et al., 2006; Hay et al., 2001a). Details of this broader project were presented in Chapter 3. In the following section, sample size varies from analysis to analysis. For example, zygosity information was not ascertained for 28 families, thus reducing all genetic analyses to (at most) 2054 twin pairs. Moreover, following common practice (Brendgen et al., 2006), a majority of genetic analyses involved monozygotic (n = 924) and dizygotic (DZ) same-sex pairs only (n = 626) with DZ opposite twin pairs (n = 504 twin pairs) omitted. As such, most descriptives statistics below are derived from this truncated data set of 1550 twin pairs.

4.2.1 Age as Grouping Factor

In the Wave One sample, age of twins ranged from 6.4 to 18.8 years (M = 13.4, SD = 3.3). The distribution of age (Figure 4.1) was significantly non-normal, $D(2058)=0.11$, $p<.05$. On inspection, data approximated a bimodal distribution organised around two modes occurring at 8 and 15.5 years. It is clear from Figure 4.1 that there are substantially less data points for participants aged between 9 and 12 years. While there are a number of likely reasons for such under-representation in this relatively narrow age range, it is clear that only a small proportion of first-time multiparous families completed registration with the ATR in the relevant 36 month period (between 1995-1998).

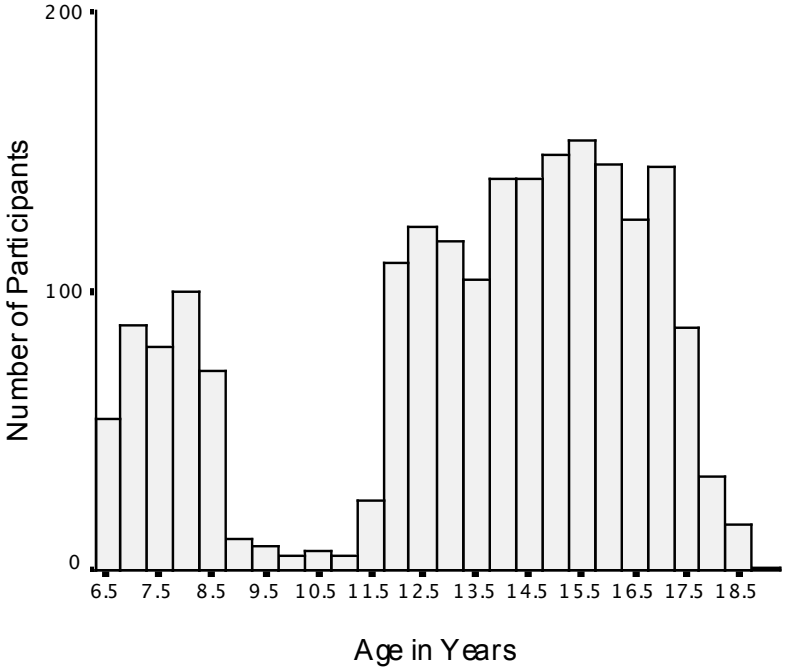


Figure 4.1 Frequency of age in years for participants in Study 1.

The decision to divide the cohort into two separate age cohorts had been established on a methodological basis prior to analysis of the frequency distribution. This decision was based on a number of issues. Most broadly, the decision reflects the need for developmentally sensitive sampling across the current wide age range. Most notably, it is generally accepted that aggression in childhood is etiologically distinct from aggression in adolescence—particularly in regards to prevalence (Barker, Tremblay, Nagin, Vitaro & Lacourse, 2006). Relatedly, and of particular relevance to the current research, the current research design (a priori) aimed to recognise the clinical convention that distinguishes the emergence of clinically impaired ASB before the age of 10 years as an etiologically significant pathway associated with increased risk of long-term adverse outcomes ((Moffitt et al., 2002); see Chapter 2).

It is noted that the current research was not able to incorporate a comprehensive

longitudinal design (that is, a three or more measurement occasions over a time period greater than 12 months) due to methodological limitations relating to time frames associated with the current research. As such, it was not possible to distinguish groups on the basis of aggression that first emerged prior to 10 years of age versus aggression that emerged after this age. Nonetheless, it is noted that the minimum age of criminal culpability in Australia is 10 years. In legal terms, a child of 10 years or more is considered to have the cognitive capacity to adequately determine the consequences of their behaviour in matters relating to criminal conduct—including the use of aggression and violence. As such, in the present research, the age of 10 years was used as a cut-off to divide the sample into a younger age cohort (consisting of all participants aged less than 10 years), and an older cohort (consisting of participants aged 10 years and over). Although this latter cohort included both pre-adolescent and adolescent children, for brevity, the cohort is sometimes referred to as the *adolescent cohort*.

Sub-grouping by age partitioned the sample into two separate, and relatively normal, age-related distributions. To validate this approach, a basic test of invariance for RA and PA was undertaken using age as a grouping factor. This analysis is discussed in more detail in section 4.1 (p. 146) below. Briefly, this modeling resulted in rejection of the null hypothesis that the factor structure (i.e., the natural grouping of items and the relative strength of items) associated with the six aggression subtype items was exactly the same for both age groups. This provided empirical support for partitioning the total sample on the basis of the specified age groups.

4.2.2 Descriptives for Younger Age Cohort in Study 1

Defining two age groups using an age cut-off of 10 years resulted in a younger age cohort sample of 620 participants (310 twin pairs excluding DZ opposite sex pairs) with a mean age of 7.68 years (*SD* = .76). A breakdown of participant age by sex and zygosity is presented in Table 4.1.

Table 4.1

Mean Age by Sex and Zygosity for Participants in Study 1

		Males		Females	
		Age (*SD*)	n	Age (SD)	n
Younger Cohort					
	MZ	7.74 (*0.07*)	200	7.69 (*0.07*)	176
	DZ_{SS}	7.55 (*0.67*)	148	7.66 (*0.80*)	108
	DZ_{OS}	7.66 (*0.07*)	102	7.66 (*0.07*)	102
Older Cohort					
	MZ	14.78 *(0.09)*	718	14.66 *(0.09)*	744
	DZ_{SS}	15.07 *(1.71)*	502	14.89 *(1.79)*	480
	DZ_{OS}	14.90 *(1.80)*	396	14.90 *(1.80)*	396

MZ = Monozygotic twins DZ_{SS} = Dizygotic same-sex twins, DZ_{OS} = Dizygotic opposite-sex twins, **NB** Numbers in the above table indicate number of individual twins not twin pairs.

4.2.3 Descriptives for Adolescent Cohort in Study 1

The adolescent group, included 2380 participants (1190 twin pairs excluding DZ opposite sex pairs) aged 10 years and above, with a resultant mean age of 14.81 years (*SD* = 1.79). A breakdown of participant age and sex is presented as a function of zygosity group in Table 4.1.

4.3 Scale Characteristics for Study 1 Behaviour Measures

This section will provide an overview of the main scales used for analysis in the first study. Scales comprise a set of behavioural measures including; the Aggression Scales, and five indices of DBD-related symptom dimensions representing the three

ADHD sub-scales, hyperactivity, impulsivity and inattention as well as ODD and CD criteria.

The six behavioural measures were derived from a Likert scale ranging between zero and three (described in Chapter 3). The maximum score possible for each behavioural measure is therefore the total number of scale items multiplied by three. The reactive and proactive aggression scales and impulsivity scale were each based upon three scale items with a resultant maximum score of nine. The Hyperactive scale was based on six scale items with a maximum possible score of 18. The upper limit score for impulsivity (three items) was nine, for the seven-item ODD scale the upper limit was 21, and for CD (15 items) the maximum possible score was 45. As above, all analyses are considered separately for younger and adolescent cohorts and are based on scale scores derived from Wave One data collection.

4.3.1 Scale Characteristics for Aggression Scales

While studies investigating RA and PA have generally treated the construct categorically (i.e., utilising a cut-off threshold to distinguish aggression subtypes, i.e., Dodge et al., 1997), an increasing number of investigators are complementing this method with a variable-centered (i.e., dimensional) approach (Baker et al., 2008; Brendgen et al., 2006; Raine et al., 2006; Vitaro et al., 2002). There are advantanges and disadvantages to both methods (Vitaro et al., 2002). The current research acknowledges both approaches where relevant, but continuous scales inform the bulk of analyses presented within this text.

4.3.1.1 Reliability Statistics for Aggression Scales

Chronbach's alpha for the RA scale derived from Wave One data was $\alpha = .68$. The reliability estimate for PA was also $\alpha = 0.68$. These reliabilities are lower than expected given previous research. Specifically, the Aggressions Scales have been used extensively in the literature and the majority of studies have reported good to excellent levels of reliability, i.e., typically deriving Cronbach's alpha values of between $\alpha = .75$ and $\alpha = .90$ (Brendgen et al., 2006; Card & Little, 2006; e.g., Dodge et al., 1997; Raine et al., 2006).

Chronbach's alpha indexes the degree to which scale items load onto the latent variable they purport to measure. For RA, the item with the lowest loading was "Reacts angrily to accidents". The corresponding item with the worst scale loading for PA was "Gets other to gang up on peers".

It is noted however, that the reliability obtained for RA that was submitted via the electronic questionnaire did reach a value equivalent to previous studies, i.e., $\alpha = 0.74$ (see Table 4.2, p. 138, and Table 4.9, p. 165). No such improvement was found for PA when online data were analysed.

Nonetheless, while reliability were certainly low, the estimates obtained here were considered at least adequate for the purpose of the current research project. The issue of reliability as it pertains to the current series of studies is discussed in more detail in later sections of this text.

4.3.1.2 Evaluation of Reactive and Proactive Aggression Distributions

Distributions deriving from dimensional indices of disruptive behaviours are commonly positively skewed (Levy & Hay, 2001). Distributions for reactive and proactive aggression are typically no exception (Poulin & Boivin, 2000). A skew index was derived from dividing the skewness statistic provided in SPSS by its standard error. A conventional cut-off of ± 2 was used to evaluate problematic skew associated with various distributions of subgroup scores (where subgroups were defined by age and zygosity). The skew index for Reactive Aggression scores ranged between approximately one and three representing a mild positive skew. The Proactive Aggression scale showed a high degree of positive skew with the relevant index ranging from approximately 10 to 25. This skew reflects the relatively lower frequency of PA-related behaviours within this population sample. Following previous studies (Brendgen et al., 2006) skewed variables were log transformed prior to analyses. Log transformations were conducted using algorithms as determined (and implemented by) LISREL version 8.2 (Joreskog & Sorbom, 1993).

4.3.1.3 Aggression Scale characteristics for younger age cohort

Because descriptives for RA and PA are typically reported using parametric methods on non-normalised data, Table 4.2 (p. 138), presents means and standard deviations for raw RA, PA hyperactivity, impulsivity, inattention, ODD and CD scores. Scores are presented by sex and zygosity. Note that raw scores were derived from a Likert scale—described in section 3.1 (p. 94)—indicating frequency of behaviours ranging from 0 ("Not at all") to 3 ("Very much/very Often").

Table 4.2

Descriptive statistics for all behaviour scales for both age cohorts in Study 1

	RA	PA	Hyp	Impulsivity	ODD	CD	n
Scale characteristics							
No. items	3	3	6	3	7	15	
Scale range	0-9	0-9	0-18	0-9	0-21	0-45	
Cronbach's alpha	.679	.679	.725	.703	.860	.780	
Younger cohort							
Males: Mean (*SD*)							
MZ	2.78 (*1.69*)	0.39 *(0.83)*	4.09 *(3.34)*	2.30 *(1.77)*	3.89 (*2.76*)	1.34 (*1.69*)	189
DZ_{SS}	2.31 *(1.60)*	0.30 *(0.70)*	3.34 *(2.64)*	2.14 *(1.63)*	3.52 (*2.96*)	1.13 (*1.45*)	138
DZ_{OS}	2.69 (*1.95*)	0.42 *(0.84)*	3.79 *(3.63)*	2.41 *(2.04)*	4.31 (*3.80*)	1.14 (1.*59*)	98
Females: Mean (*SD*)							
MZ	2.14 (1.48)	0.17 *(0.42)*	3.37 *(2.98)*	1.91 *(1.46)*	2.97 (*2.42*)	0.68 (*0.95*)	161
DZ_{SS}	2.25 (*1.39*)	0.25 (*0.72*)	2.32 (*2.18*)	1.95 (*1.62*)	2.91 (*2.63*)	0.64 (*1.22*)	106
DZ_{OS}	2.25 (*1.89*)	0.19 (*0.57*)	2.64 (*2.94*)	1.87 (*1.65*)	3.51 (*3.28*)	0.62 (*1.20*)	98
Older cohort							
Males: Mean (*SD*)							
MZ	2.25 (*1.91*)	0.37 *(0.94)*	2.29 *(2.42)*	1.55 *(1.65)*	3.30 (*3.43*)	1.13 (*2.18*)	667
DZ_{SS}	2.63 *(1.99)*	0.59 *(1.22)*	2.78 *(3.03)*	1.65 *(1.81)*	4.14 (*3.95*)	1.79 (*3.43*)	474
DZ_{OS}	2.44 (*2.01*)	0.48 *(1.06)*	2.75 *(3.09)*	1.68 *(1.73)*	3.90 (*3.74*)	1.48 (*3.00*)	359
Females: Mean (*SD*)							
MZ	2.15 (*1.82*)	0.23 (*0.77*)	1.81 (*1.99*)	1.27 (*1.48*)	2.82 (*3.11*)	0.74 (*1.96*)	674
DZ_{SS}	2.11 (*1.82*)	0.22 (*0.65*)	1.74 (*2.03*)	1.27 (*1.50*)	3.07 (*3.31*)	0.77 (*1.61*)	439
DZ_{OS}	1.93 (*1.64*)	0.11 *(0.42)*	1.76 *(2.09)*	1.23 *(1.44)*	2.76 (*2.82*)	0.63 (*1.57*)	358

RA = Reactive aggression, PA = Proactive aggression, Hyp = Hyperactivity symptoms ODD = Oppositional defiant disorder symptoms, CD = Conduct disorder symptoms, SD = Standard deviation, n = number of participants in sample. MZ = Monozygotic twins, DZss = Dizygotic same-sex twins, DZos = Dizygotic opposite-sex twins

Of note in Table 4.2 is the near-zero mean scores for PA amongst all sex-zygosity groups reflecting the fact that the large majority of participants were rated as evidencing no PA-related behaviours.

4.3.1.3.1 Sex differences in mean aggression subtype scores for younger age cohort

Using a non-parametric test of group differences, the modest difference between scores for male and female participants in the younger cohort was significant (i.e., higher for males) for RA, $Z(1, 427) = 1.57$, $p = .15$, but not PA, $Z(1, 430) = 1.35$, $p > .05$. All Kolmogrov-Smirnov tests were conducted using individual twin scores. To compensate for this double-entry procedure in which familial dependence across twin pairs was ignored, the *df* was adjusted to reflect the number of twin pairs. In a smaller community cohort of 6-year-olds, Brendgen et al. (2006) tested for sex differences using a structural equation model that constrained means for male versus females scores to be equal. These investigators reported a significant deterioration in model fit when equivalence was assumed for RA but not PA.

Following Brendgen et al. (2006), a test of invariance was applied to the covariance structure of RA and PA as a function of sex. This is explained in more detail with accompanying statistics in section 4.3 (p. 148). To summarise, despite differences in mean scores both RA and PA, no significant sex difference in covariance structure was found for either MZ or DZ twins in the younger cohort. However, it is noted that this finding of equivalence in covariance structure represented a marginal result. Specifically, while the analysis did not support the rejection of the null hypothesis (which suggests the factor structure for the six aggression subtype items is equivalent regardless of whether measured in males or females).

4.3.1.3.2 Analysis of aggression subtype group cut-off scores in the younger age cohort

Dodge and colleagues (e.g., Dodge et al., 1997) have established a convention for categorising group differences in aggression subtypes within child and adolescent

populations. Specifically, a cut-off of one standard deviation above the sample mean is used to identify presence of high levels of RA and/or PA. In the current study, cut-off scores were assessed for males and females separately with zygosity and twin status collapsed. In the younger cohort, 49 (14.4%) male participants scored above the cut-off of 4.32 for RA (M = 2.62, SD = 1.70). Despite the marked difference in mean levels of RA versus PA, 43 (12.6%) male participants scored above the cut-off of 1.13 for PA (M = 0.36, SD = 0.77). For females, the cut-off scores for RA (M = 2.19, SD = 1.62) and PA (M = 0.22, SD = 0.62) were 3.81 and 0.84 respectively. This equated to 41 (14.6%) and 42 (14.9%) female participants scoring above cut-off for RA and PA respectively. Point prevalence in the younger cohort was therefore 14.5% and 13.7% for RA and PA respectively.

It is worth noting that the proportion of female participants scoring above cut-off was similar to that of their male counterparts (and in fact was marginally higher) despite the significantly lower mean levels of both kinds of aggression in the former group. This is consistent with the notion that the shape of the underlying distributions for both males and females is similar (despite the apparent differences in means between males and females). This buttresses the invariance results reported immediately above which indicated no differences between males and females.

4.3.1.3.3 Overlap between aggression subtypes in the younger age cohort

It has been well established that RA and PA frequently co-occur (Kempes et al., 2005; Little et al., 2003). In the current research, a correlation of r=.47 (p<.05) was observed between RA and PA in the younger cohort (males, r=.47, p<.05; females, r=.45, p<.05) reflecting an expected moderate-to-strong association between the two

subtypes. To tease apart the overlap between RA and PA, four aggression groups were identified; 1) an RA-only group which included those who scored above cut-off on RA and below the PA cut-off, 2) a PA-only group which classified those exceeding the cut-off for PA but not RA, 3) an RA+PA group (often referred to as Pervasively Aggressive group in the literature) which included those who scored above cut-off on both RA and PA measures, and 4) a non-aggression group including those who scored below cut-off on both RA and PA. Results of group membership are presented in Table 4.3.

Table 4.3

Reactive and proactive aggression group membership in the younger cohort

	Males			**Females**		
	n (%)	Mean RA (SD)	Mean PA (SD)	n (%)	Mean RA (SD)	Mean PA (SD)
RA-only	31 (4.9%)	5.26 (*0.51*)	0.35 (*0.48*)	23 (3.7%)	4.17 (*0.49*)	0.00 (*0.00*)
PA-only	25 (4.0%)	3.12 (0.93)	2.16 (0.37)	24 (3.9%)	2.71 (*0.46*)	1.21 (*0.51*)
RA+PA	18 (2.9%)	5.77 (*0.81*)	2.39 (*0.50*)	18 (2.9%)	5.17 (*1.25*)	1.44 (*0.98*)
NA	266 (42.8%)	2.02 (*1.23*)	0.06 (*0.24*)	216 (34.8%)	1.61 (*1.00*)	0.00 (*0.00*)

RA = Reactive aggression, PA = Proactive aggression, RA-only = Reactive aggression only group, PA-only = Proactive aggression only group, RA+PA = Reactive and proactive aggression group, NA = Non-Aggressive group, **NB** Numbers in the above table indicate number of individual twins not twin pairs. Percentages derive from total number of participants in younger aged cohort regardless of sex.

Compared with previous research, the pattern of group membership shown in Table 4.3 reveals relatively low levels of RA and PA in primary-school aged males and females (e.g., Dodge and Coie, 1987; Samilvali & Nieminen, 2002; Washcbusch et al., 1998) although Waschbusch et al. (1998) only found 1.2% of 5th grade children

in their sample exhibited PA. A key difference between these latter studies and the current study is that the present results were obtained via caregiver-rated rather than teacher-rated questionnaires.

4.3.1.4 Aggression scale characteristics for the adolescent cohort

Table 4.2 presents means and standard deviations for raw RA and PA scores in the adolescent cohort by sex and zygosity. The scores observed for older participants in this table show a degree of variability in relation to childhood scores. That is, there is no definitive pattern across zygosity groups suggesting lower versus higher prevalence as a function of age or sex. While some previous studies have found prevalence in both RA and PA to trend downwards from childhood to adolescence (e.g. Brame, Nagin & Tremblay, 2001), others (McAdams, 2002) have reported PA as showing a higher degree of continuity in both childhood and adolescence when compared to RA. Omnibus tests of the difference in prevalence of RA and PA by age group (with sex and zygosity both collapsed) suggested that mean levels of RA in adolescence were significantly lower than childhood levels, $Z(1, 2027) = 2.62$, $p = .00$. However, there was no difference in mean levels of PA in older versus younger cohort mean scores, $Z(1, 2037) = .42$, $p = .99$.

Another point worth making here is that mean levels of disruptive- and aggression-related behaviours in the older cohort are typically accompanied by larger variance estimates relative to means derived from the younger cohort, suggesting greater variability around the mean in the former age group. This reflects the likelihood that floor effects (i.e., positive skew) are impacting to a greater extent on the younger cohort data—with its smaller smaller sample size— relative to older cohort data.

4.3.1.4.1 Sex differences in mean reactive and proactive aggression scores for the older cohort

Despite only small divergence on mean scores, females participants in the adolescent cohort scored significantly lower than males for both RA, $Z(1, 1599) = 1.62$, $p < .05$ and PA, $Z(1, 1606) = 3.15$, $p < .05$. As for the younger cohort, *df* was adjusted for number of twin pairs to compensate for double-entry of data.

4.3.1.4.2 Analysis of aggression subtype group cut-off scores in the older cohort

RA-PA group cut-off scores were determined separately for males and females with all other factors (i.e., zygosity, twin status) collapsed. The RA cut-offs for males and females were 4.18 (M = 2.32, SD = 1.86) and 4.08 (M = 2.19, SD = 1.89) respectively. For PA, the cut-off was 1.39 (M = 0.42, SD = .97) for males and 1.03 (M = 0.24, SD = 0.79) for females. These cut-off scores are similar to those obtained for the younger cohort. A total of 164 (13.8%) males and 228 (19.2%) females exceeded cut-off for RA. For PA, 141 (11.9%) males and 158 (13.3%) females scored above their respective identified thresholds. Compared with the younger cohort, the point prevalence of RA and PA was marginally lower for older males by 0.6% and 0.7% respectively. For females, levels of PA were lower in the older cohort relative to younger females by a magnitude of 1.6%. Conversely, levels of RA were higher in the older cohort by a magnitude of 4.6%. The higher prevalence of RA in older females, both in relation to their male counterparts and younger female counterparts is striking given the general expectation that within a normal population, levels of overt aggression will decrease during adolescence (e.g. Brame et al., 2001).

4.3.1.4.3 Overlap between aggression subtypes in the adolescent cohort

The correlation between RA and PA in the older cohort (r=.48, p<.05) was similar to that found for the younger age group (r=.47, p<.05). While this similarity in correlation across younger and older age groups is inconsistent with one previous meta-analysis (Card & Little, 2006) who found increasing correlations as a function of age, it is nonetheless generally consistent with the meta-analysis by Polman et al. (2007) who found no age effects on the correlations between RA and PA.

Males (r=.52, p<.05) evidenced a higher correlation than females (r=.43, p<.05) in the older sample. All correlations obtained in the current research are notably lower than the results revealed by Card and Little's meta-analysis in which a correlation of r=.67 (p<.05) was found between RA and PA at age 10 and was observed to increase linearly (BZr = .013) each year into adolescence.

Group membership for RA and PA in the older cohort is presented in Table 4.4.

Table 4.4

Reactive and proactive aggression group membership in older cohort

	Males			Females		
	n (%)	Mean RA (SD)	Mean PA (SD)	n (%)	Mean RA (SD)	Mean PA (SD)
RA-only	83 (3.5%)	5.95 (*1.06*)	0.42 (*0.90*)	126 (5.3%)	4.92 (*1.24*)	0.00 (*0.00*)
PA-only	60 (2.5%)	3.08 (*0.83*)	2.17 (*0.46*)	56 (4.7%)	2.35 (*0.72*)	1.32 (*0.61*)
RA+PA	81 (3.4%)	6.42 (*1.18*)	3.44 (*0.50*)	102 (2.4%)	5.42 (*1.31*)	1.88 (*1.39*)
NA	963 (40.5%)	1.72 (*1.20*)	0.11 (*0.31*)	905 (38.1%)	1.4 (*1.03*)	0.00 (*0.00*)

RA = Reactive aggression, PA = Proactive aggression, RA-only = Reactive aggression only group, PA-only = Proactive aggression only group, RA+PA = Reactive and proactive aggression group, NA = Non-Aggressive group, **NB** Numbers in the above table indicate number of individual twins not twin pairs. Percentages derive from total number of participants in younger aged cohort regardless of sex.

4.3.1.5 Evaluating model invariance for aggression subtypes

The current finding of divergence in RA and PA scores as a function of both age and sex underscores the importance of assessing for group differences to establish that variances and covariances underpinning the constructs being measured are homogenous (Jinks & Fulker, 1970). Tests designed to achieve this goal are often collectively referred to as tests of *model invariance*. Such tests are important in confirming that the underlying nature of the construct being measured is consistent across different groups. Three separate analyses of model invariance were applied according to three core assumptions associated with the underlying data including assumed equal factor structure for (1) child and adolescent participants, (2) zygosity groups, representing the equal environments assumption (Mitchell et al., 2007), and (3) males and females.

The approach utilised is generally consistent with that used by Brendgen et al. (2006). Identified groups were submitted to a simple model fitting procedure using LISREL version 8.2 (Joreskog & Sorbom, 1993) in which covariance structure was constrained to be equal across groups. In all cases, the model invariance test assessed whether the pattern of item weightings (i.e., the degree to which each item contributed to their respective scales) was the same across identified groups.

A chi-square difference test was used to compare resulting fit statistics with estimates derived from an unconstrained model in which covariance was allowed to vary freely. As in previous omnibus tests, a double-entry procedure was used such that the data for each twin were treated at the individual case level rather than pairing data as a function of twin status. To correct for the potential violation of the independence assumption problem, the *N* value (i.e., number of observations) specified within the evaluated models was adjusted to reflect the number of contributing twin pairs rather than the full number of cases entered into the relevant models (i.e., twice the number of twin pairs).

Each of the three main analyses are considered in turn.

4.3.1.5.1 Invariance of aggression subtypes as a function of age

As mentioned in section 4.1, p. 131, the total sample reported in the current project covers a broad age range and, developmentally speaking, one that spans considerable topographic diversity in relation to aggression. Indeed, the question has been raised regarding putative age-related differences in the expression of aggression (Miles & Carey, 1997). Equivalence in covariance factor structure was assessed as a function

of age group. Separate analyses were conducted for MZ and DZ zygosity groups.

A significant deterioration in model fit was found for MZ twins, χ^2(4, N = 188 younger twin pairs, 731 older twin pairs) = 19.76, p >.05 and DZ twins, χ^2(4, N = 129 younger twin pairs, 483 older twin pairs) = 16.22, p>.05 when the test of invariance was applied.

To reiterate from an earlier section, the implication of divergent patterns of loadings across age groups suggests (either or both) RA and PA operate differently in these different age groups. The result supports the partitioning of the sample into two different age groups. It is noted that the differences in item loadings are only modest.

4.3.1.5.2 Invariance of aggression subtypes as a function of zygosity

A test of equivalence in factor structure for the 6-item Aggression Subscales across zygosity groups was included to provide an evaluation of the equal environments assumption (Mitchell et al., 2007). In this analysis, two participant groups were established representing MZ and DZ twins (with age and sex collapsed as variables).

A chi square difference test indicated no deterioration in model fit when factor structure was constrained to be equal for both zygosity groups in the younger cohort, χ^2(4, *N* = 310 twin pairs) = 12.63, p >.05, and the adolescent cohort χ^2(4, *N* = 1214 twin pairs) = 18.27, p >.05. This provides reasonable evidence that the equal environments assumption (EEA) has been met within the current sample using the key measure of interest.

4.3.1.5.3 Invariance of aggression subtypes as a function of sex

Like age effects, the presence of sex effects on the phenotypic expression of a trait undermines the basic assumptions of genetic model fitting where variances and covariances are not homogeneous (e.g., Neale & Cardon, 1992). The finding of sex differences in mean frequency of RA and PA prompted further analysis of the underlying covariance structure of the main variables of interest.

Preliminary analysis of male versus female group data involved evaluating correlations across zygosity groups (Table 4.5). Because these analyses were conducted prior to normalisation of data, Kendall's tau-b correlation statistic was used as it remains relatively robust when used with non-normal ordinal data and when transformed ranks cannot be assumed to be equidistant (*Statistical Package for the Social Sciences,* 2002). It is worth noting that the intraclass correlations displayed in Table 4.5, offer a first approximation of potential genetic relationships in the data. The matrices suggest the presence of small to moderate genetic effects that increase with age. More specifically, moderately higher intraclass RA and PA correlations for MZ twins relative to DZ twins suggests an additive genetic component is contributing to RA and PA behaviour—and this differential appears to increase for data derived from the adolescent cohort. However, comprehensive quantitative evaluation of the genetic architecture of RA and PA requires submitting the data to structural equation modeling techniques capable of providing maximum likelihood fit functions of model estimates (Brendgen et al., 2006). This process of model fitting will be the focus of subsequent chapters. Nonetheless, the Tables immediately below provide an initial rough indication concerning whether sex-related differences in RA and PA have some genetic basis.

A final note regarding the invariance tests for age and sex reported above. The current results are consistent with Polman et al.'s (2007) meta-analysis which found no gender differences in the statistical relations (including correlations and effects sizes across samples) between RA and PA.

Table 4.5

Cross-twin cross-trait correlations for aggression subtypes

			Twin 1		Twin 2	
			RA	PA	RA	PA
MZ twins						
	Twin 1	RA	—	0.37*	**0.82***	0.29*
		PA	0.34*	—	0.34*	**0.71***
	Twin 2	RA	**0.81***	0.35*	—	0.37*
		PA	0.36*	**0.80***	0.37*	—
DZ_{SS} twins						
	Twin 1	RA	—	0.38*	**0.65***	0.27*
		PA	0.54*	—	0.27*	**0.46***
	Twin 2	RA	**0.54***	0.29*	—	0.45*
		PA	0.26*	**0.50***	0.44*	—
DZ_{OS} twins						
	Twin 1	RA	—	0.38*	**0.60***	0.28*
		PA	0.39*	—	0.23*	**0.48***
	Twin 2	RA	**0.50***	0.13*	—	0.41*
		PA	0.14*	**0.30***	0.28*	—

RA= Reactive aggression, PA= Proactive aggression. MZ twins = Monozygotic twins, DZ_{ss} twins = Same sex dizygotic twins, DZ_{OS} twins = Opposite sex dizygotic twins. Correlations for younger age cohort in upper diagonal of each matrix. Correlations for older age cohort in lower diagonal of each matrix. Bold denotes intraclass correlations. Twin 1 represents males within Opposite sex dizygotic twins

Of particular interest is the differential magnitude of intraclass correlations across DZ same-sex pairs versus DZ opposite sex pairs. Where DZos twin correlations are substantially lower than their DZss counterparts, the presence of sex-specific factors are implied—a finding that generally cautions against the subsequent pooling of data across sex groups (Neale & Cardon, 1992). While an inspection of the above tables reveals that DZos intraclass correlations are generally lower than those found for

DZss twins, the magnitude of differentials is relatively small. This indicates that, to the extent sex-specific influences underpin differences in covariation, sex effects are likely to be small. For the older cohort, intraclass correlations for RA do not appear substantially different for DZss and DZos twins although small effects may be present. On the other hand, the intraclass correlations for PA in this same cohort are noticeably lower for DZos twins when compared to their DZss counterparts.

The correlational data above do not alone provide a comprehensive rationale for pooling sex group data for RA and PA. The anomalies observed in the correlational structure of data obtained from the older cohort—in which the differential between DZos twin intraclass correlation for PA in the older cohort ($r = 0.30$, $p<.05$) and their DZss counterparts ($r = 0.50$, $p<.05$) approached half the value of the latter correlation—warranted further detailed inspection of covariance structure in relation to male versus female RA-PA scores.

Again, LISREL version 8.2 (Joreskog & Sorbom, 1993) was used to fit an omnibus model of invariance to RA and PA using sex as a grouping factor with each age cohort considered separately. Within each age group, invariance tests were applied separately for MZ and DZ same-sex twins. The methodology necessarily precludes inclusion of DZ opposite sex twin pairs but nonetheless provides a statistical test of general sex effects on the covariance structure of the two aggression variables. The test involves comparing the fit of a model in which the covariance between RA and PA items is constrained to be equal across male and female groups with a model in which all parameters are allowed to vary freely. Consistent with Brendgen et al., (2006), no significant difference in model fit was obtained for the MZ twins, $\chi^2(4, N$

= 184 twin pairs,) = 5.19, $p < .05$ or DZ twins, $\chi^2(4, N = 126$ twin pairs) = 9.24, $p = .05$ in the younger cohort. However, it is worth noting that the chi-square for the DZ twins was marginal at $p = .05$. In the older cohort, an adequate model fit was obtained for the MZ twins, $\chi^2(4, N = 707$ twin pairs,) = 9.74, $p < .05$ with invariance constraints applied. However, imposing equality on the covariance structure for older DZss twins resulted in a significant reduction in fit, $\chi^2(4, N = 481$ twin pairs) = 24.99, $p < .05$. This suggested the pattern of item loadings for the RA and PA scales differed for males versus female DZss twins.

The equivocal results from the invariance analysis in the adolescent cohort, prompted the fitting of sex-limitation models separately to RA and PA data from this group. While these analyses constitute the focus of section 5.5 (p. 191), a brief summary of sex-limitation analyses is useful here. Small to moderate sex differences were found in relevant sex-limitation and sex moderation analyses. However, these effects appeared to be largely quantitative rather than qualitative in nature. As such data from both males and females were subsequently pooled for older cohort (as for the younger cohort). Prior to pooling, all scores were standardised separately for males and females to adjust for mean differences in RA and PA occurring between sex groups. All subsequent genetic modeling involved data derived from MZ and same-sex DZ twin pairs (e.g., Brendgen et al., 2006) unless otherwise specified.

4.3.2 DBD Scale Characteristics

The two ADHD sub-scales, hyperactivity and impulsivity, as well as the two aggressive DBD scales, ODD and CD, were all treated dimensionally rather than categorically in the present study—see Hay, McStephen and Levy (2001b) for

discussion of use of dimensional versus categorical data. Of the preliminary descriptives presented below that did not compare across zygosity groups, double-entered data were used resulting in an effective sample size of 4164 participants. Reliability statistics for the five DSM-IV scales can be found in Table 4.2, p. 138.

4.3.2.1 Evaluation of DBD Distributions

As expected, the distribution for both ADHD measures were positively skewed with a skew index ranging between 3 and 13 for hyperactivity and between 2 and 9 for impulsivity. The ODD scale and the CD scales were also positively skewed with index ranges of 7-30 and 15-92 respectively.

The skew index for CD was notably high. Intra-scale indices revealed the highest degree of skew for this scale (91.35) was associated with older DZ twins. In fact, relative to the younger cohort, the skew index was consistently higher across all scales associated with older participants. In the measurement of disruptive behaviour, greater skew is generally an indicator of lower prevalence. However, it is worth noting that because the current skew indices were determined by dividing the fundamental sample-dependent measure of skew by its own standard error of skew, the final estimate of skew is affected by sample size. Thus, the differences in range between younger and adolescent groups in the current research was exacerbated by the substantial differences in sample size between age cohorts. More specifically, the higher sample size of the older cohort resulted in relatively smaller standard errors of skew (and higher final estimates). In turn, the upper estimate of each range corresponded to older cohort estimates and likely provide a more accurate estimate of real skew. Taken together, this emphasises that the relatively more extreme

behaviours associated with CD are considerably less frequent than other DBDs and indeed than RA and PA.

4.3.2.2 Descriptives for DBD Scales in the Younger and Adolescent Cohort

Table 4.2 presents mean scores for hyperactivity, impulsivity, inattention, oppositional-defiant disorder (ODD), and conduct disorder (CD) scales by gender and zygosity group for both the younger and adolescent cohorts.

4.3.3 Correlation Matrices for All Behaviour Scales: Study 1

Wave One zero-order correlations were derived from the full Wave One data set which included 1823 families with all data double-entered. Notable is that most of the correlations between the aggression subtypes on the one hand and the DBD symptom dimensions on the other, were stronger than the correlation between RA and PA.

Consistent with Waschbusch et al. (2002), RA was strongly correlated with all DBD behaviour dimensions. These latter correlations appear to remain fairly consistent across the younger and older groups, with slightly higher estimates in the latter age cohort. The zero-order correlation between RA and CD is remarkably high. Follow-up analyses failed to identify any potentially spurious factors such as outliers, explaining this coefficient.

Table 4.6

Zero-order correlations for behaviour scales for younger (upper diagonal) and older (lower diagonal) participants in Study 1

	RA	PA	Hyp	Imp	Ina	ODD	CD
RA	-	**0.46**	**0.42**	**0.49**	**0.42**	**0.55**	**0.67**
PA	0.49	-	**0.28**	**0.35**	**0.24**	**0.6**	**0.49**
Hyp	0.46	0.35	-	**0.59**	**0.52**	**0.38**	**0.46**
Imp	0.56	0.41	0.58	-	**0.46**	**0.41**	**0.56**
Ina	0.46	0.34	0.48	0.48	-	**0.33**	**0.45**
ODD	0.58	0.69	0.40	0.46	0.43	-	**0.57**
CD	0.73	0.55	0.50	0.61	0.50	0.66	-

RA = Reactive aggression, PA = Proactive aggression, Hyp = Hyperactivity, Imp = Impulsivity, Ina = Inattention, ODD = Oppositional defiant disorder, CD = Conduct disorder. Younger cohort n = 768, Older cohort n = 2878. All correlations significant at $p = .05$.

PA also showed strong correlations with all DBDs, although the relevant magnitudes were somewhat attenuated when compared to the DBD associations with RA. Like RA, PA showed modestly stronger correlations with DBDs in the older cohort compared to the younger cohort.

Unsurprisingly, both RA and PA also showed a trend of stronger association with the antisocial DBDs versus the ADHD symptom dimensions. From amongst the ADHD symptom dimensions, both RA and PA showed a slightly high correlation with impulsivity than inattention or hyperactivity. A more suprising result was that PA was moderately more strongly associated with ODD than CD, while the inverse was true for RA. That is, RA was moderately more strongly associated with CD than ODD.

4.4 Sample Characteristics for Study 2

4.4.1 Sample Descriptives for Study 2

As discussed in Chapter 3, 2082 participants who contributed to the wave one of data collection were asked to provide consent to be approached for requests for participation in additional studies such as the wave two component of the current study. A total of 1217 families formally agreed to recieve such requests by our research team. A request for participation in the Wave Two component of this study was sent to all 1217 families. Of the families who received this request, 774 provided consent to participate in the second wave of data collection (i.e., the online component of the study). However, by the end of the three-month data collection phase associated with Wave Two, only 155 families (20% of consenting families and 12.75% of all families approached) had completed all aspects of the online component, including both the online parent questionnaire and two child-completed online neuropsychological tasks. A breakdown according to the various aspects of online assessment is provided immediately below.

Five hundred and eleven caregivers (representing 68.5% of the initial 744 consenting families) completed all items of the online (short-form) version of the ATBRS behaviour questionnaire. All caregivers who commenced the online questionnaire, completed it. Inspection of the web-based database responsible for storing online participant information revealed that four data sets included missing data. Due to the the way the questionnaire was designed and implemented, it was possible to determine that this missing data was due to transmission errors rather than the result of respondee error. A final 433 (84.7% of those submitted) of "completed"

questionnaires were used for longitudinal analyses. This final figure represents the omission of 77 families for the longitudinal analyses as a result of imposing the minimum 9-month follow-up period as discussed at the commencement of this chapter, further impacting on this truncated figure.

Compared to the proportion of completed data sets for the online questionnaire, proportion of incomplete data was higher again for the two child completed neuropsychological tasks. For the Multimorph (MM) task, 447 twins (representing 30% of the 744 consenting families) commenced the task. Four hundred and twenty eight of these twins (95.7%) completed the MM task. However, only 326 twins were ultimately associated with complete MM data sets. This latter figure represents 76% of those twins who completed the online study. Inspection of the data set again confirmed that the 24% of compromised data sets were the result of poor transmission of data across the internet. That is, random missing datum was scattered throughout compromised data sets. It was not possible for any user to deliberately transmit null or missing data during administration of the online neuropsychological tasks (or online questionnaire). Data integrity issues are discussed in greater detail in Chapter 7. For the Iowa Gambling Task, the following completion statistics were obtained; 463 twins commenced the task, 451 completed the tasks but only 348 (77.2%) complete data sets were obtained. Again the 22.8% of compromised data sets represented poor data integrity (i.e., poor data transmission).

The effect of non-participation and attrition on sample characteristics is the focus of a preceeding section. A breakdown of basic demographic information pertaining to Wave Two data collection is provided immediately below. Descriptives are derived

from uncompromised family data sets that were associated with completed online parent questionnaires. Inconsistencies between numbers of participants reported in text and numbers reported in tables generally reflects missing data for specific variables targeted in tables.

Table 4.7

Mean age by sex and zygosity for participants in Study 2

	Males		Females	
	Age (*SD*)	n	Age (SD)	n
Younger Cohort				
MZ	8.04 *(0.63)*	80	8.25 *(0.84)*	50
DZ_{SS}	8.23 *(0.98)*	50	8.14 *(0.70)*	30
DZ_{OS}	8.32 *(0.78)*	54	8.32 *(0.78)*	54
Older Cohort				
MZ	14.70 *(1.61)*	126	14.73 *(1.75)*	150
DZ_{SS}	15.17 *(1.60)*	70	14.89 *(1.62)*	60
DZ_{OS}	14.66 *(1.60)*	59	14.66 *(1.60)*	59

MZ = Monozygotic twins DZ_{SS} = Dizygotic same-sex twins, DZ_{OS} = Dizygotic opposite-sex twins,
NB Numbers in the above table indicate number of individual twins not twin pairs.

4.4.2 Factors Influencing Attrition and Non-participation in Study 2

Attrition associated with Wave Two data collection resulted in 341 (44%) of families who initially agreed to take part, informally withdrawing prior to successful completion. These families did not contribute any data to the final data set. Figure 4.2 provides the results of a short online survey of 78 initially consenting families who did not ultimately complete the study. The relevant data indicate that a majority of these families (51%) withdrew due to difficulties associated with the timeframe of the study. "Technical problems" was the next most highly rated reason for withdrawal accounting for 34% of the surveyed sample. Possible factors confounding the interpretation of this data are discussed in more detail in Chapter 7.

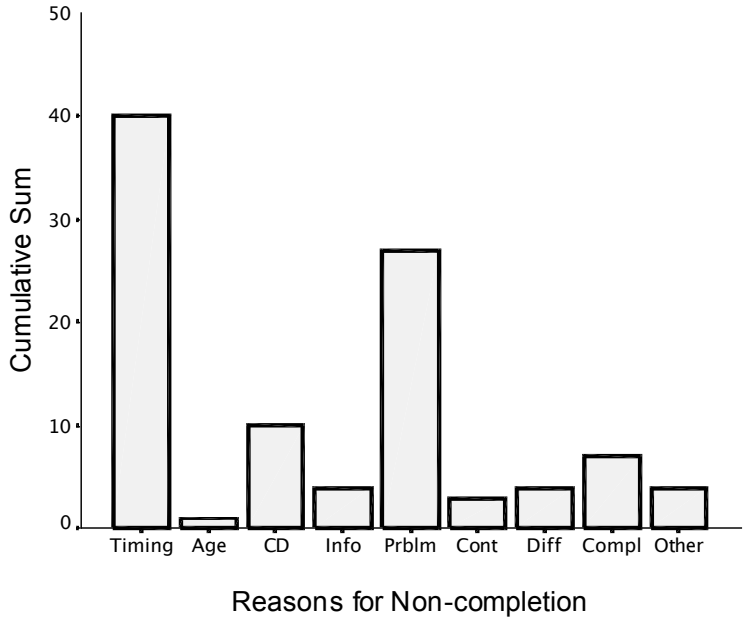


Figure 4.2 Responses to survey of reasons for non-completion of Study 2

Below is a table that provides a detailed description of each of the categories (as well as corresponding survey items) presented in the histogram in Figure 4.2 above.

Table 4.8

Survey Items for Online Non-completion Survey

Key	Label	Survey Item
Timing	Timing	"Our family was unable to find time to finish the study"
Age	Age	"The study did not seem appropriate because of the age of our twins"
CD	Compact Disc	"Our family did not receive the CD-ROM we requested"
Info	Information	"Our family was not sent all the details required to download or complete the tasks"
Prblm	Problem	"We obtained the necessary software but had technical problems getting it to work"
Cont	No Contact	"Tried to contact the person running the study but didn't receive a (timely) response"
Diff	Difficulties Completing	"Being a part of this kind of research was too complicated"
Comp	Compliance	"Our twins decided not to complete the study"
Other	Other	Allowed for qualitative responses

Given that this brief survey was conducted online, those who could not complete

Study 2 due to difficulties accessing the internet are likely to have been under-represented in this survey.

An attempt was made to determine the extent to which lack of access to the internet in the home contributed to non-participation. To this end, a response form (Appendix B) indicating reason for non-participation was included in the approach mail-out. Of the 365 families who returned this response form, 71 (19%) families indicated a lack of access to the internet as a primary reason for their decision not to take part.

Fifty eight (4.7% of the initial 1217 consenting families) approach packs were returned due to out-dated mailing addresses in the contact database. It was therefore assumed a total of 50 (4.1%) families initially agreed to be contacted regarding the online study and subsequently received the information pack, but then chose not to return the specific consent form for the study. The issue of participant response rate and patterns is explored in Chapter 3.

4.4.3 Influence of Participation Status on Sample Characteristics

The following section provides an evaluation of the degree to which attrition and non-participation affected sample characteristics in Wave Two of data collection. As in the analysis of descriptives provided in Wave One, participants were assigned to one of two age cohorts determined by an age cut-off of 10 years. For each age cohort, three groups were identified for comparison; 1) families who did not respond to the request for participation (Non-respondent Group) in Wave Two of data collection, 2) families who responded and consented to take part in Wave Two of data collection but withdrew prior to completion (Non-completion Group) and, 3)

families who consented and completed Wave Two of data collection (Completion Group). For the purpose of comparative analysis, the following descriptives are derived from the original Wave One of data collection sample to ensure comparisons of age were conducted with reference to a consistent time point.

Mean differences in age were considered as a function of participation groups (i.e., Non-respondent, Non-completion, Completion) and sex. These comparisons are plotted in Figures 4.3 and 4.4 for the Younger and adolescent cohorts respectively. Statistical comparisons did not include sex as a grouping variable but instead assessed mean differences amongst the three Participation Status groups. These analyses revealed a significant difference in mean age for participation status $F(2, 3261) = 12.53$, $p < .05$. Post-hoc Sheffe tests revealed that adolescent males in the non-respondent group were significantly older than both their non-completion and completion counterparts (mean difference, .48, $p<.05$ and .72, $p<.05$ respectively). This unsurprising result likely reflects that older adolescent male twins are less motivated to participate in social science studies such as this one than females or younger twins.

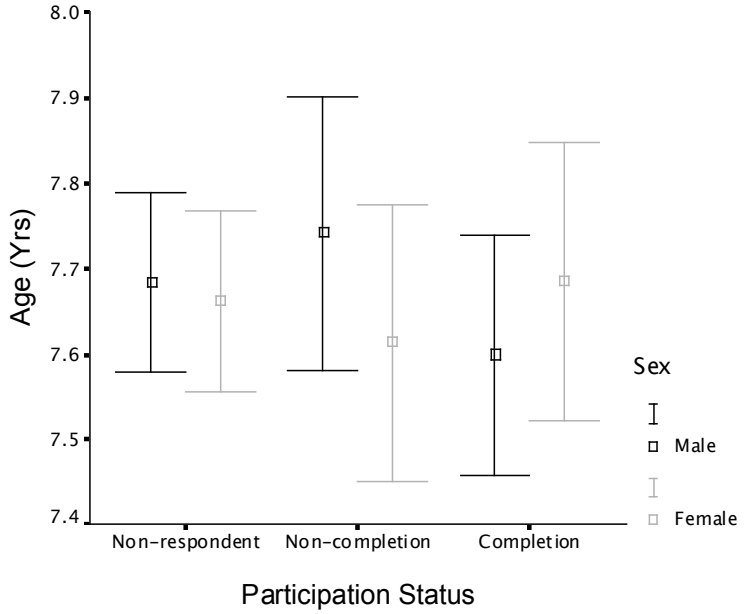


Figure 4.3 Differences in mean age in younger age cohort as a function of sex and participation status in Study 2

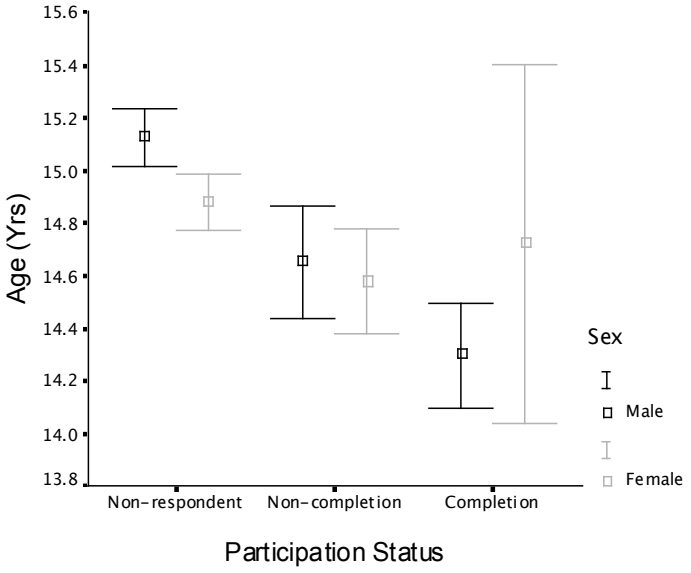


Figure 4.4 Differences in mean age in older cohort as a function of sex and participation status in Study 2

4.4.4 Participation Group Differences in Behaviour Scale Scores

4.4.4.1 Participation group differences in reactive and proactive aggression scale scores

For each age cohort, the three participation groups were further partitioned according to zygosity and sex. No significant differences in median scores were found for either RA or PA in the childhood cohort. However, in the older cohort, significant differences in median scores were found for MZ female twins, $\chi^2(2, N=363) = 6.86$, $p > .05$—significant group differences held for both Twin 1 and Twin 2 groups considered separately. As can be seen in Figure 4.5, this result reflected a lower median RA score for the Non-completion group compared with the Non-participation and Completion groups.

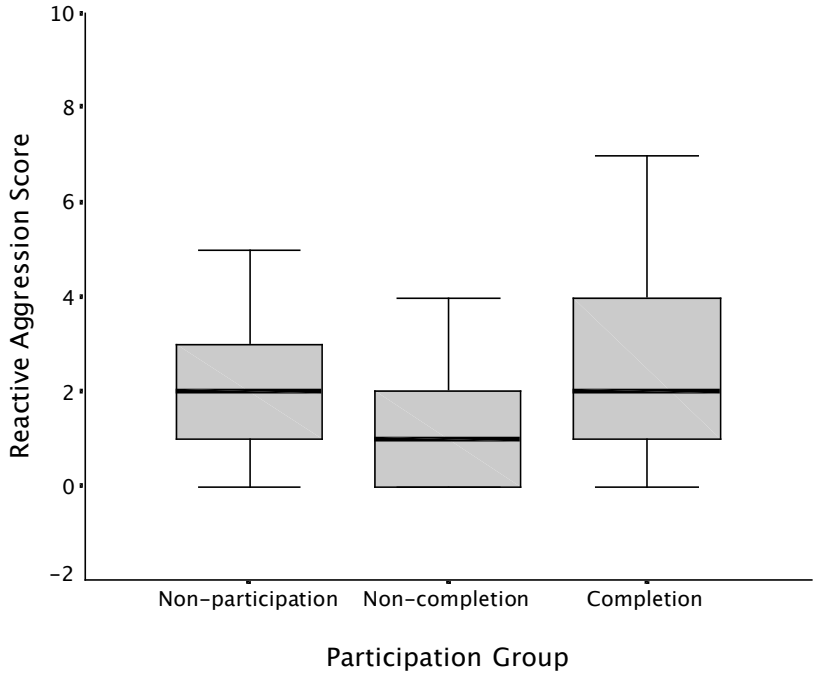


Figure 4.5 Differences in median reactive aggression for MZ female twins across participation groups in the older cohort

4.4.4.2 Participation group differences in disruptive behaviour scale scores

Kruskal-Wallis (K-W) tests were conducted for both younger or adolescent cohort to assess the extent to which non-participation in the second study affected representativeness of the sample in relation to the DBD scales measured at Wave One. Kruskal-Wallis (K-W) tests were used because this non-parametric statistic enables simultaneous comparison across three or more groups. Because, by definition, the Non-participation and Non-completion groups did not contribute data to the second wave, direct comparisons between all three groups were only possible using Wave One data.

Initial omnibus tests revealed significant participation group differences in Conduct Disorder symptoms for the younger cohort. These scores showed higher median level of CD symptoms for the Non-participation group, median = 1, relative to the Non-completion, median = 0, and Completion groups, median = 0. However, post-hoc Kolmogorov-Smirnov (K-S) comparisons conducted on each paired combination of the three groups failed to identify any significant group differences. The K-S test was preferred for pairwise group comparisons due to the fact it is less affected by violations of independence.

Significant group differences were found for Conduct Disorder symptoms in the older cohort. Post-hoc K-S tests indicated a significant difference between Non-participation (median=0, IQR=1, minimum score=0, maximum score=30) and Completion (median=0, IQR=1, minimum score=0, maximum score=10) groups for CD scores indicating a restricted range for the latter group, $Z(1, N=2594) = 1.38$, $p=.044$. The maximum score possible for CD was 45. Post-hoc tests were conducted

without any adjustment to the alpha level.

4.5 Scale Characteristics for Study 2 Behaviour Measures

4.5.1 Scale Characteristics for Aggression Subtypes and Disruptive Behaviour Symptoms

4.5.1.1 Descriptives for reactive and proactive aggression and disruptive behaviour scales

Reliability statistics for all behaviour scales can be found in Table 4.9. Descriptive statistics of the reactive and proactive aggression scale scores in the second wave of data collection are presented in parametric form in this same table. Raw scores were derived from a Likert scale indicating frequency of behaviours ranging from 0 ("Not at all") to 3 ("Very much/very Often"). After summing sub-scale items the highest possible score for either RA or PA was nine.

Descriptives are provided for submitted data sets ("All participants") that included missing data and uncompromised data sets ("Longitudinal cohorts") that excluded data sets with missing data. The decision to remove data sets with missing data reflects the finding that these compromised sets were subject to poor data transmission. To avoid exacerbating random noise in the data set due to cascading asyncronous or misallocation of data points to their respective fields within the database it was necessry to take a conservative approach and exclude any compromised data sets.

Table 4.9

Descriptive statistics for aggression subtypes and disruptive behaviour scale scores

	RA	PA	Hyp	Impulsivity	Inattention	ODD	CD	n
Scale characteristics								
No. items	3	3	6	3	9	7	15	
Scale range	0-9	0-9	0-18	0-9	0-27	0-21	0-45	
Cronbach's alpha: Wave one	.683	.678	.714	.706	.799	.861	.775	
Wave two	.749	.675	.764	.752	.804	-	-	
Younger cohort (Means)								
All participants								
Males	3.79 (*2.26*)	0.92 *(1.39)*	6.52 *(3.26)*	2.78 *(1.99)*	7.85 (4.27)	-	-	440
Females	3.13 *(2.11)*	0.65 *(1.04)*	5.38 *(2.97)*	2.39 *(1.96)*	6.49 (3.44)	-	-	377
Longitudinal cohort								
Males								179
Wave one	2.52 (*1.73*)	0.35 (*0.71*)	3.28 (*3.32*)	2.15 (*1.72*)	8.15 (4.63)	3.52 (*2.87*)	1.02 (*1.39*)	
Wave two	3.48 (*2.28*)	0.74 (*1.23*)	6.19 (*3.33*)	2.65 (*2.01*)	7.41 (3.28)	-	-	
Females								124
Wave one	2.12 (*1.55*)	0.07 (*3.80*)	2.57 (*2.13*)	1.74 (*1.92*)	6.44 (3.37)	2.59 (*2.01*)	0.64 (*1.20*)	
Wave two	2.96 (*2.01*)	0.63 (*1.04*)	5.17 (*2.96*)	2.19 (*1.96*)	6.60 (3.61)	-	-	
Older cohort (Means)								
All participants								
Males	3.03 (*1.98*)	0.69 *(1.19)*	4.87 *(3.50)*	1.89 *(1.91)*	7.63 (4.37)	-	-	1587
Females	2.85 *(2.01)*	0.44 *(0.95)*	4.25 *(2.95)*	1.50 *(1.61)*	6.10 (3.53)	-	-	1571
Longitudinal cohort								
Males								334
Wave one	2.37 (*1.92*)	0.36 (*0.83*)	2.35 (*1.77*)	1.66 (*1.70*)	7.65 (4.46)	3.57 (*3.41*)	1.07 (*1.69*)	
Wave two	3.03 (*1.99*)	0.70 (*1.20*)	4.79 (*3.42*)	1.91 (*1.94*)	7.54 (4.04)	-	-	
Females								349
Wave one	2.02 (*1.65*)	0.15 (*0.46*)	1.77 (*1.97*)	1.30 (*1.44*)	6.07 (3.60)	2.69 (*3.01*)	0.48 (*1.18*)	
Wave two	2.76 (*2.01*)	0.41 (*0.95*)	4.25 (*2.95*)	1.94 (*1.62*)	6.19 (3.27)	-	-	

RA = Reactive Aggression, PA = Proactive Aggression, Hyp = Hyperactivity symptoms, ODD = Oppositional defiant disorder, CD = Conduct disorder, SD = Standard deviation, n = number of participants in sample, Lng cohort = Longitudinal cohort, Standard deviations are presented in italics

Interestingly, Table 4.9 indicates generally increasing levels of behavioural pathology over the 9-month period between first and second measurement occasions. These general patterns in RA and PA are somewhat consistent with some (McAdams, 2002), but not (Barker et al., 2006). Given the inherently skewed nature of disruptive- and aggression-related behavioural scales (Levy et al., 1997), these

patterns, as detected in raw data alone, do not lend themselves to definitive preliminary interpretations. Nonetheless, as noted earlier, omnibus tests of the difference in prevalence of RA and PA by age group (with sex and zygosity both collapsed) suggested that mean levels of RA in adolescence were significantly lower than childhood levels, $Z(1, 2027) = 2.62$, $p = .00$, while no difference in mean levels of PA were found between older versus younger cohort mean scores, $Z(1, 2037) = .42$, $p = .99$.

4.5.2 Correlation Matrices for All Behaviour Scales: Study 2a

A matrix of zero-order correlations was derived from the 433 completed questionnaires used in the longitudinal analyses. Data were double-entered resulting in a total sample size of n=866. This included 671 in the older cohort (327 males and 344 females) 178 in the younger cohort (103 males and 75 females). Correlations for all behaviour measures used at each measurement occasion were collated and are presented in Table 4.10.

Of note amongst the cross-time point correlations is the finding that RA at time one predicts time-2 PA as well as it predicts time-2 RA in the younger cohort. This pattern is loosely consistent with the sequential hypothesis. It is interesting that this pattern does not hold for the older cohort. Certainly, PA does not appear to have the same prognostic value in predicting future RA.

The inverse relationship between RA and ODD on the one hand and PA and CD on the other, that was evident in the zero-order matrix for all Study 1 twins, is evident in this truncated sample. Interestingly, this inverse relationship was also evident over

time, with ODD predicting future RA to a greater magnitude than future PA, and CD predicting future PA to a greater magnitude than future RA. While the differentials in question were modest (particularly for the prognostic relationship between ODD and future RA in the older cohort), the findings indicate the link between RA and ODD versus PA and CD pattern is at least consistent. As noted immediately above, this pattern seems somewhat attenuated in the older cohort data.

Table 4.10

Zero-order correlations for Wave 1 and Wave 2 behaviour measures for the younger and older participants in study 2 (n=433)

	Wave One								Wave Two				
	RA	PA	Hyp	Imp	Ina	ODD	CD	UCF	RA	PA	Hyp	Imp	Ina
Wave One													
RA	-	**.45**	**.46**	**.53**	**.47**	**.63**	**.48**	**.39**	**.58**	**.43**	**.39**	**.39**	**.39**
PA	.50	-	**.27**	**.32**	**.18**	**.48**	**.73**	**.52**	**.28**	**.42**	**.21**	**.15**	**.14**
Hyp	.44	.36	-	**.65**	**.55**	**.34**	**.35**	**.23**	**.26**	**.30**	**.60**	**.40**	**.40**
Imp	.58	.43	.60	-	**.49**	**.53**	**.31**	**.30**	**.39**	**.27**	**.47**	**.50**	**.38**
Ina	.48	.34	.45	.52	-	**.42**	**.29**	**.25**	**.25**	**.16**	**.44**	**.30**	**.67**
ODD	.73	.63	.50	.66	.53	-	**.58**	**.49**	**.61**	**.51**	**.33**	**.42**	**.31**
CD	.55	.80	.43	.50	.46	.67	-	**.84**	**.32**	**.46**	**.31**	**.20**	**.19**
UCF	.49	.61	.31	.35	.36	.49	.79	-	**.31**	**.43**	**.26**	**.18**	**.20**
Wave Two													
RA	.56	.32	.34	.47	.34	.53	.37	.31	-	**.62**	**.34**	**.46**	**.27**
PA	.38	.46	.34	.42	.34	.48	.46	.34	.51	-	**.30**	**.40**	**.17**
Hyp	.28	.19	.52	.41	.25	.30	.25	.18	.40	.30	-	**.56**	**.45**
Imp	.42	.31	.48	.62	.35	.47	.36	.25	.55	.43	.57	-	**.46**
Ina	.24	.25	.29	.32	.55	.30	.30	.26	.39	.32	.30	.35	-

All correlations significant at $p < .05$. RA = Reactive aggression, PA = Proactive aggression, Hyp = Hyperactivity symptoms, Imp = Impulsivity symptoms, Ina = Inattention symptoms, ODD = Oppositional defiant disorder, CD = Conduct disorder, UCF=Underlying common form of aggression. Younger cohort (n = 178) presented in bold, Older cohort (n = 671).

The relationship between PA and CD is not unexpected given similarities in scale items (see Chapter 2). What is perhaps more revealing is the role of hyperactivity with relevant variables, as previously highlighted by Raine et al. (2006). Generally consistent with this latter study, hyperactivity at time-1 was modestly more strongly correlated with PA at time-2 than time-1 PA with time-2 hyperactivity. In regards to this longitudinal relationship, the differential prognostic value of hyperactivity (when compared with PA), this pattern appears to be even more pronounced in the older cohort. This is suggestive of some directionality in this relationship between hyperactivity and PA, and one that may well strengthen with age.

Impulsivity is identified as a variable of interest due to its potential unique

relationship with RA (e.g., Barratt & Slaughter, 1998; Raine et al., 2006). The zero-order correlations offer no indication of directionality or differential strength between these two behaviour dimension when compared over time, or with the other behaviour dimensions considered. However, while the above matrix offers useful preliminary insight into basic longitudinal relationships between the relevant behaviours, in order to ascertain etiological relationships it is necessary to evaluate genetically informative data using structural equation methods. This is the focus of the following chapter.

Chapter 5.

Phenotypic Relationships between Aggression Subtypes and the Disruptive Behaviour Disorders

5.1 Overview

This chapter presents results from preliminary genetic analyses of aggression subtypes and the DBDs. The first section revisits the model fitting process introduced in Chapter 3 providing more detail regarding the statistical methods and procedures employed. Findings from the basic univariate models of RA and PA are then presented and key aspects discussed. Next, a series of univariate statistical models are considered with the aim to elucidate sex differences in underlying etiology of aggression subtypes. While these latter models were performed prior to the univariate and multivariate analyses of aggression subtypes described in sections 5.3 and 5.4, they are reported in the current sequence because articulation of the relevant sex-limitation models benefit from earlier explanations relating to the univariate and bivariate models of aggression subtypes. Finally, basic multivariate analyses that include DBD symptom dimensions as covariates are provided.

5.2 Quantitative Genetic Model Fitting Revisited

Once behaviour ratings of MZ and DZ twins have been obtained, the basic univariate model fitting process involves decomposing the relevant covariance or correlation matrices (one matrix per zygosity group in the classic twin design) according to the structural specifications of the **ACE** model (as illustrated in Figure 5.1, p. 173).

To reiterate, the structural specification of the basic **ACE** model specifies that, for any given trait,

1. the additive genetic (**A)** component, where present, is asymmetrical such that the magnitude of **A** effects on DZ twin behaviour will be equal to half the magnitude of **A** effects on MZ twin behaviour,
2. the shared environmental (**C)** component, where present, is symmetrical such that it will exert an equal influence on both DZ and MZ twins' behaviour,
3. and the influence of the non-shared environmental (**E)** component, where present, constitute random effects that make the behaviour of both DZ and MZ twins less similar.

Decomposing the correlation matrices (as used in the current methodology) according to these specification derives a set of parameter estimates which, once standardised, represent the relative proportion of variance attributable to each of these three etioligical components. Standardisation of an estimate involves squaring the estimate and dividing it by the sum of all three squared estimates. For example, the parameter estimate for the **A** component is calculated as $a^2 / (a^2 + c^2 + e^2)$.

The inclusion of the "where present" qualifications in the list immediately above illustrates that alternative specifications of the **ACE** model are possible—namely, the presence of absence of any one or two of the above components. These alternative models are discussed in more detail further below.

A statistical software package commonly used for undertaking genetic analyses is the

mx program (Neale & Cardon, 1992). The program employs statistical algorithms that combine matrix algebra and numerical optimisation. These techniques represent a variant of the structural equation modeling capabilities found in LISREL (Joreskog & Sorbom, 1993), adapted to the relatively specific requirements of genetic modeling (Neale & Cardon, 1992). Like LISREL the producers of the software have designed a graphical user interface for ease of use. This allows the user to specify pathway models by representing them in graphical or schematic format (rather than as complex statistical equations). The program prepares and processes the final statistical formulation (i.e., structural equation) related to the graphic pathway model. It outputs parameter estimates associated with the pathways specified in the model, as well a series of fit statistic statistics for evaluating model fit. Despite the fact that model results are calculated by computer, the model fitting process nonetheless requires the researcher to be familiar with some basic concepts. Some of these concepts are introduced briefly below.

As shown in Figure 5.1, p. 173, the full univariate **ACE** model has three parameters (one each for **A**, **C** and **E**) and three degrees of freedom. Where covariances are used, degrees of freedom are calculated by subtracting the number of estimated parameters from the number of observed statistics—i.e., six in the univariate case. The six observed statistics represent the two twins scores and their correlation for both MZ and DZ groups. The fact that the number of parameters is equal to the number of degrees of freedoms in the full **ACE** model, illustrates that all parameters are allowed to vary. The full model, therefore, cannot be evaluated for fit because it imposes no restrictions on the parameter estimates themselves. In other words, the full model represents a "perfect" fit. Nonetheless, this model provides a valuable

reference to compare the fit of alternative solutions. That is, the basic **ACE** model allows researchers to begin asking questions about the relative importance of fundamental etiological influences.

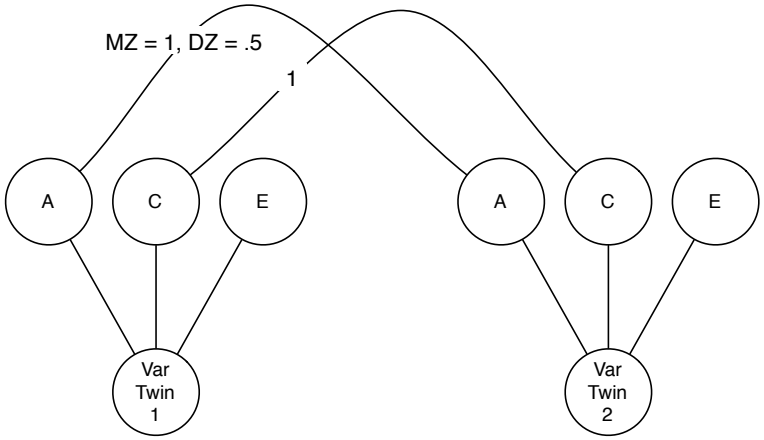


Figure 5.1 Basic path diagram for univariate behaviour genetic model. A denotes latent variable representing additive genetic variance component, C = shared environmental component and E = nonshared environmental component. MZ = monozygotic twins and DZ = dizygotic twins.

A note concerning degrees of freedom (*df*) and use of correlation matrices (as used in all current analyses). Neale and Mae (1992) recommend an adjustment to the *df* to account for the fact that the diagonals in any correlation matrix consist only of values equal to 1. Because these values cannot be considered unique, only one such value is sufficient to provide information in the analysis, i.e., to scale the covariance (Neale & Cardon, 1992) . Neale and Maes (1992) note that this value appears in the expected variances rather than the expected covariances. However, this recommendation has not been widely adopted in the literature and awaits further debate.

From a statistical perspective, a principal aim of the model fitting process is to derive

a model that best fits the data with the least amount of parameters. As noted, this approach is driven by the principal of parsimony. To this end, parameters are often systematically removed from the full **ACE** model and comparative fit indices are used to evaluate the most parsimonious solution.

So for example, the **A** parameter might be dropped from the ACE model (by constraining the A path to 0) and subsequent fit indices would then be used to evaluate any resultant significant deterioration in model fit. An **ACE** model with the **A** parameter removed is often referred to as a **CE** model. This model specifies no genetic effects on the behaviour under investigation. Additive genetic effects are assumed if the **CE** model results in (1) a significant reduction in model fit compared to other alternative specifications involving the **A** component, e.g., **ACE** or **AE** models. The **AE** model represents the dropping of the **C** parameter (indicating no shared-environmental effects). It is worth noting that model fitting invariably precludes dropping the **E** parameter for reasons explained further below.

As noted, *dropping* a single parameter is achieved by fixing the parameter on the relevant model pathway to 0. This precludes variance being attributable by the parameter in question and reduces the number of free parameters in the **ACE** model from three to two. Correspondingly, the degrees of freedom increases to four.

A brief comment regarding **E** effects is warranted. As mentioned earlier, the **E** component typically includes residual effects (i.e., error variance). Because error variance is unavoidable within the broader project of measuring behaviour, this component is usually required in most best fitting models. The fact that this error

variance is not modeled separately and instead is conflated with non-shared environmental effects may be considered a limitation of the modeling process. At the same time, because most behaviours that are of interest to psychologists invariably represents the end point of complex ontogenetic processes, it is rare for such behaviours to have no biological basis at all (Rutter, 2006). Thus it is rare to obtain a best fitting model that precludes the **A** parameter (i.e., the **CE** specification). This reflects a biological reality rather than a limitation of the modeling process. Nonetheless, it is not uncommon for researchers to report results from the **E** specification (i.e., the model with both **A** and **C** removed) to provide a test of the broader hypothesis that the modelled data predominantly consists of error variance rather than systematic variance.

The **C** component is typically the least reliable of the three parameter estimates for reasons discussed in earlier chapters. This, as noted previously, means it is especially susceptible to be being removed from models with a corresponding increase of fit, or at least no deterioration of fit.

Multivariate methods extend the univariate approach by assessing for familial influences that contribute to the *co-occurrence* of disorders or problem behaviours (Levy & Hay, 2001). More specifically, the addition of cross-twin correlations (i.e., correlations between a twin's score on one behaviour dimension and his/her co-twin's score on a second behaviour dimension) allow researchers to evaluate (1) the proportion of overlapping variance attributable to genetic versus shared and non-shared environmental determinants as well as (2) the proportion of non-overlapping, or syndrome-specific, variance attributable to familial determinants. Figure 5.2, p.

177, illustrates a typical bivariate genetic model.

The multivariate model has three variance components common to both variables of interest and these components resemble those associated with the univariate model; **A** = additive genetic variance component, **C** = shared environmental component and **E** = nonshared environmental component. It is these components that account for the shared variance or overlap between the two variables. Clearly, the model decomposes this variance according to the basic ACE model. In addition, each variable is also estimated for trait-specific ACE variance. Like the univariate model all A paths are constrained to .5 for MZ (monozygotic) twins and 1 for DZ (dizygotic) twins while all C paths are constrained to equality for both zygosity groups.

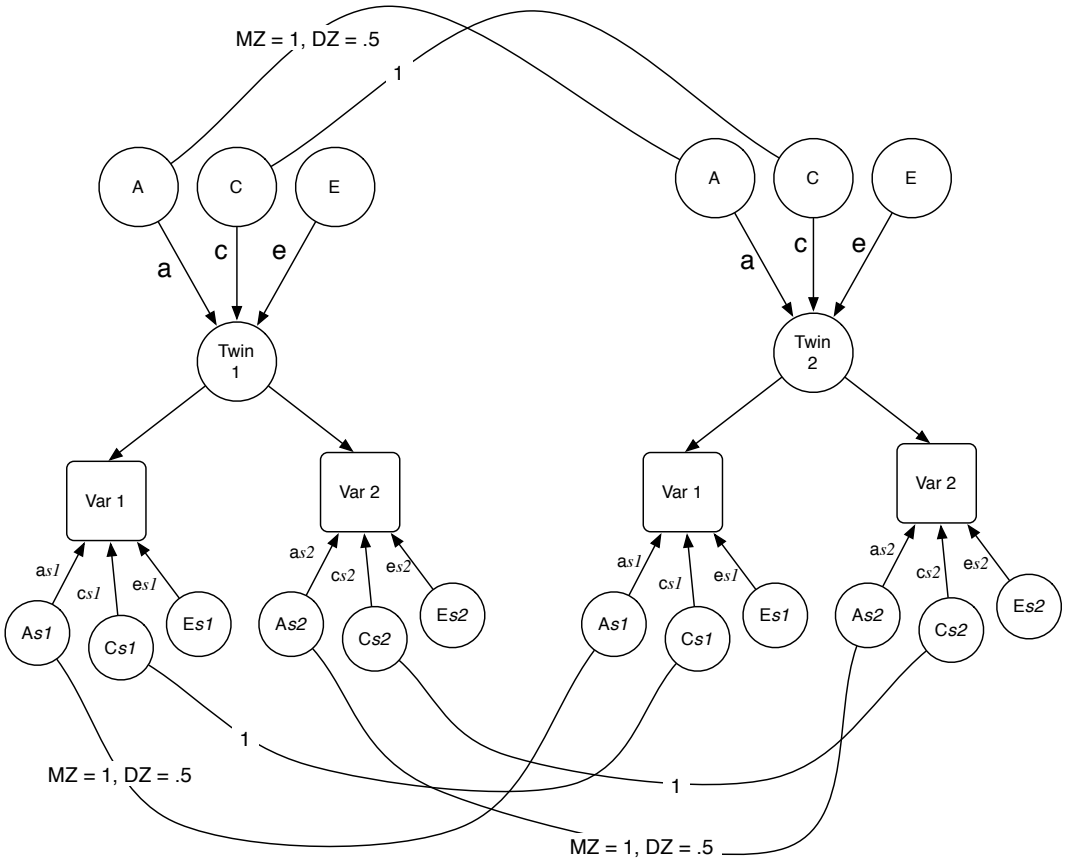


Figure 5.2 Basic path diagram for bivariate behaviour genetic model. A denotes latent variable representing additive genetic variance component, C = shared environmental component and E = nonshared environmental component. MZ = monozygotic twins and DZ = dizygotic twins. S denotes trait-specific variance component.

As with univariate modeling, the trimming of multivariate models involves systematically constraining specific paths to 0 as a means of removing, or dropping them from the model. This reduced the complexity of the structure without imposing any changes to the existing path specifications. Models reduced in this way are called *nested* models. As noted above, nested models can be tested for adequacy of fit using a range of complementary statistics. Clearly, in the multivariate model, there are many more possible paths that can potentially be constrained. It is typically best-practice to be guided by theoretical considerations when applying the model trimming process. Typically this will involved theoretical expectations regarding which nested model is likely to produce a better fit than an alternative or full

specification of the model.

With an understanding of the characteristics and limitations of the basic univariate and multivariate modeling processes, these structural equations represent an advanced technique for estimating the relative effects of genes and environment on a trait. The results derived from submitting RA and PA data from study one to the univariate model fitting process is described below.

5.3 Univariate Modeling of Aggression Subtypes

Table 5.1 presents the parameter estimates for the univariate models of RA and PA for both age cohorts. These estimates represent path coefficients linking the **ACE** variance components to the relevant trait. In order to derive standardised estimates, which reflect the proportion of trait variance accounted for by the relevant variance component, it is necessary to square the path coefficient. So for example, Table 5.1 shows that the path coefficient associated with the additive genetic component of RA in the younger cohort is .55 (with a confidence interval ranging between .41 and .69). Squaring this estimate gives $.55^2 = .302$. This results reveals that 30.2% of variance in RA (in the univariate model) is attributable to genetic factors.

Table 5.1

Parameter estimates for univariate genetic and environmental effects on reactive and proactive aggression in younger and older cohorts

	Latent Factors										
	A		C		E		χ^2	*p*	AIC	RMSEA	*df*
	UPE	C.I.	UPE	C.I.	UPE	C.I.					
Younger Cohort											
RA											
ACE	**0.55**	**(.41, .69)**	**0.75**	**(.61, .88)**	**0.36**	**(.32, .40)**	**0.00**	**1.000**	**-6.00**	**0.00**	**3**
AE	0.9	(.83, .97)	-		0.35	(.32, .39)	25.19	0.000	17.20	0.15	4
E	-		-		1.00	(.94, 1.06)	338.78	0.000	328.78	0.64	5
PA											
ACE	0.75	(.55, .92)	0.41	(-.65, .65)	0.52	(.47, .58)	0.00	1.000	-6.00	0.00	3
AE	**0.70**	**(.49, .90)**	**-**		**0.71**	**(.57, .87)**	**1.38**	**0.847**	**-6.62**	**0.00**	**4**
E	-		-		1.00	(.90, 1.13)	162.23	0.000	152.26	0.42	5
Older Cohort											
RA											
ACE	**0.73**	**(.66, .81)**	**0.57**	**(.46, .66)**	**0.36**	**(.34, .38)**	**0.00**	**1.000**	**-6.00**	**0.00**	**3**
AE	0.91	(.89, .95)	-		0.35	(.34, .37)	26.49	0.000	18.49	0.08	4
E	-		-		1.00	(.97, 1.03)	>1000	0.000	>1000	0.59	5
PA											
ACE	0.85	(.77, .93)	0.35	(-.50, .50)	0.40	(.38, .42)	0.00	1.000	-6.00	0.00	3
AE	**0.91**	**(.87, .95)**	**-**		**0.40**	**(.38, .42)**	**2.96**	**0.564**	**-5.04**	**0.02**	**4**
E	-		-		1.00	(.97, 1.03)	971.81	0.000	961.81	0.51	5

RA = Reactive aggression, PA = Proactive aggression, UPE = Unstandardised parameter estimate, C.I. = Confidence interval, AIC = Akaike information criteria, RMSEA = Root-mean-square error of approximation. A = Genetic variance component, C = Shared environmental variance component, E = Non-shared environmental variance component. **NB** Bold text denotes best fitting models

5.3.1 Univariate Models of Reactive Aggression

As shown in Table 5.1, the full **ACE** model (Figure 5.3, p. 181) was the best fitting model for RA in both younger and older cohorts. That is, for both age cohorts, the alternative **AE** and **E** specifications resulted a significant deterioration in fit according to the relevant indices. A primary measure of fit in the current analyses is the change in chi square value that results from removing one or more parameters. The univariate fit indices provided in Table 5.1 show that the chi square value associated with the **AE** model of RA in the younger cohort increased (from 0 in the fully specified model) 25.19, and that the degrees of freedom increased from three to four.

Critical values associated with the chi square distribution (e.g.,Keppel, Saufley & Tokunaga, 1992) reveal that when alpha is set to $p = .05$ a chi square difference of 3.84 or greater is considered significant in comparisons involving a difference of one degree of freedom. In other words, for the **AE** solution to satisfy the conditions of parsimony, it would have needed to incur an increase in chi square of less than 3.84. The **E** solution, which involved dropping parameters **A** and **C**, resulted in an increase of $\chi^2_{difference} = 338.78$, $df_{difference} = 2$. The critical chi square value for this difference test is $\chi^2 = 5.99$. Clearly, the **E** model also deviated significantly from adequate model fit when compared to the benchmark of the full **ACE** model.

The complementary fit statistics also reflected this deterioration in fit. For example, the **AE** mode resulted in the Aikman's Information Criteria (AIC) statistic increasing from -6 to 17.20. As noted in Chapter 3, the model with the lowest AIC value is considered to have superior fit. Moreover, the RMSEA increased from 0 to .15. The RMSEA figure should range between 0 and .05 to indicate good fit and between .05 and .1 to indicate an adequate fitting model. Similar reductions in fit were found when the same set of parameter reductions were applied to the full **ACE** model using data from the older cohort.

Thus the fit statistics supported retaining the full **ACE** model for both younger and older cohorts. Regarding the younger group, standardised parameter estimates derived for the shared environmental effects (**C**) suggested this component contributed the single greatest source of variance (56.25%) in RA scores. As noted above, just less than a third of variance in RA (30.25%) was attributable to additive

genetic effects (**A**), with the remaining 12.96% associated with non-environmental (**E**) factors. Compared with their younger peers, data from the older cohort revealed a similar magnitude for **E** effects (30.25%) while the pattern of results for the **A** and **C** factors was reversed. That is, the genetic component accounted for 53.29%, and shared environment 32.49%, of variance.

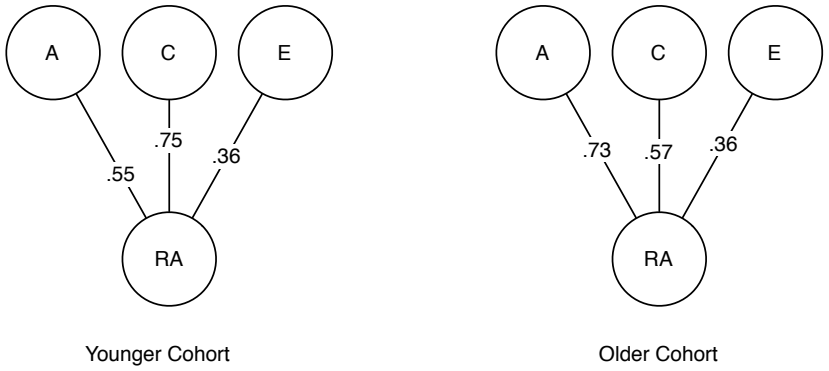


Figure 5.3 Best fitting univariate path model for reactive aggression in younger and older cohorts. A denotes latent variable representing additive genetic variance component, C = shared environmental component and E = nonshared environmental component.

5.3.2 Univariate Models of Proactive Aggression

Data obtained from both younger and older participants indicated that shared environment was not required to explain etiology in PA. By contrast, genes and non-shared environment were both important influences with genes accounting for 49% and 82.81% of variance in this aggression subtype for the younger and older cohorts respectively. The remainder of variance was attributable to **E** effects, being 50% and 15% for the two respective age groups. Again the trend for greater influence of genes in the older age group is evident. At the same time, environmental effects are clearly weaker in this same adolescent cohort.

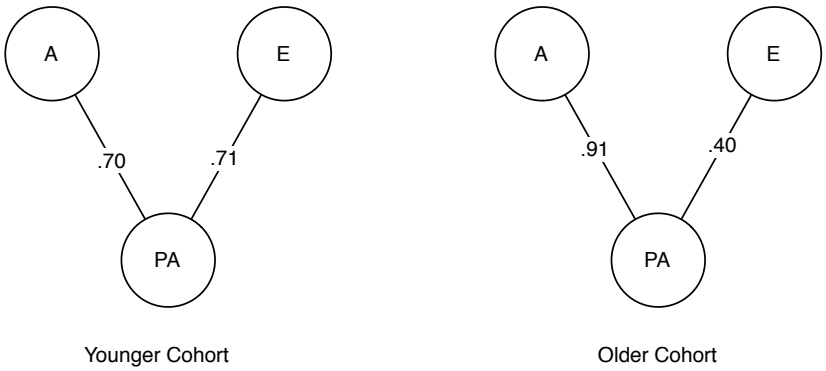


Figure 5.4 Best fitting univariate path model for proactive aggression for younger and older cohorts. A denotes latent variable representing additive genetic variance component, C = shared environmental component and E = nonshared environmental component.

Important to note here is that while both univariate models of PA for the younger and older groups consistently provided better fit when **C** effects were dropped, the wide confidence intervals around the estimates of shared environmental effects warrant some caution when interpreting parsimony. This is discussed in more detail below.

5.3.3 Discussion Concerning Univariate Models

The above analyses augment existing evidence that genes play an important role in the development of both RA and PA throughout development with estimates falling within the range of estimates detailed in Miles and Carey's meta-analysis. While this finding is consistent with other emerging research, it is also extends the RA and PA literature by including data that represent more comprehensive sampling of the developmental spectrum than has been reported to date. Consistent with Baker et al. (2008), the current findings suggest that PA is influenced by genes to a substantially greater extent than RA, and that this difference is larger in adolescent versus childhood samples. Moreover, the differences across aggression subtypes appear to be more than simply quantitative. Rather, results imply different architectures for RA

and PA. The results unequivocally demonstrate the importance of assessing subtypes of aggression.

The use of two distinct age groups, a younger and older cohort, in the present design allowed a preliminary evaluation of the smorgasbord hypothesis (Miles & Carey, 1997). This hypothesis suggests that through a process of increased autonomy in self-selection of environments, genes become increasingly important across development while, conversely, the effects of shared environment decline. The current data tentatively support this theoretical proposition suggesting the effect is particularly pronounced for RA for which genetic influences in the older cohort were almost double that of their younger counterparts. The tentative nature of these findings is underpinned by the fact that the comparison made here is not a longitudinal one, but is instead cross-sectional in nature. Nonetheless, the data are clear in showing a reversal of the gene-environment differential for both RA and PA as predicted by the smorgasbord hypothesis.

Divergent results were obtained in relation to shared environmental effects in RA and PA. Specifically, the current data suggest that the influences of shared environment are important for RA—and are particularly pervasive in childhood. Conversely, best fitting models for PA for both age groups involved dropping the **C** estimate from the covariance structure. Thus, these latter data indicate negligible impact of shared environment on the development of PA. It is noted the current results differ from Brendgen et al. (2006) who found no **C** effects for either aggression subtype. The present data also diverge with Baker et al. (2008) who found **C** effects for both RA and PA.

Noticeable in the univariate analyses is the relatively narrow confidence intervals around most parameter estimates reflecting the good fit of the models. This reflects the substantial sample size underpinning the univariate analyses. This high level of reliability of estimates held for all the univariate models for both RA and PA, with the notable exception of **C** effects for PA. With such a high degree of error inherent in these latter estimates it is not surprising that a better fit was obtained by removing them from the pathway model. The problem is that such a high degree of error precludes definitive conclusions regarding this component (whether retained or removed from the model). Statistical instability in the **C** term is not uncommon in the behaviour genetic literature and highlights the importance of extremely large samples for obtaining adequate statistical power for detecting **C** effects (Rutter, 2006).

At the same time, obtaining significant **C** effects does not necessarily preclude statistical ambiguity. Specifically, there are a number of confounds, including rater bias and sibling interaction effects, that can spuriously mimic **C** effects. Competitive SI effects in particular are considered below.

5.3.3.1 Sibling interaction effects and sample size in univariate models of aggression subtypes

In contrast to Brendgen et al. (2006), who reported an **AE** model as the best fitting specification for teacher rated RA and PA in a population cohort of 6 year olds, the current data derived from caregiver-rated RA suggested prominent **C** effects in both younger and older age cohorts—and particularly in the younger cohort. On the other hand, the current results also differ from findings reported by Baker et al. (2008) who

found **C** effects for both RA and PA in a sample of 9-10 year old twins.

The primary difference distinguishing the above results is the presence versus absence of **C** effects. Because aggression ratings have theoretically been associated with cooperative sibling interaction effects (SI) which are expected to spuriously inflate **C** effects (Rutter, 2006), it is informative to consider the possibility that the differences in reported estimates of **C** are in part the result of cooperative SI effects, or rater bias.

As noted previously, cooperative SI effects result in increased likelihood of a behaviour occurring in a co-twin due to its presence in the proband (Eaves et al., 1997a; Huizenga et al., 2007; Simonoff et al., 1998). Interestingly, RA has been shown to increase in the presence of provocative interactions (see Waschbusch et al., 2002) suggesting the possibility that RA is linked to cooperative SI effects (which increase likelihood of the same behaviour in the co-twin).

Levene's tests applied to the basic models reported here (and reported in Chapter 4) supported the assumption of homogeneity of variance across zygosity groups for both RA and PA in the younger cohort and RA in the older cohort (see Appendix F for table of variances). On this basis, it is reasonable to assume no SI effects operating on RA (Jinks & Fulker, 1970). However, it was not possible to assess whether **C** effects on RA included a rater bias component. Certainly, the current **C** estimates are notably higher than those obtained by Baker et al. (2008). Implications concerning the role of **C** effects in RA are discussed in more detail in Chapter 8.

Conversely, as noted above, the *lack* of **C** effects on PA potentially implicates competitive SI effects. Certainly, Levene's test of variance in adolescent PA revealed a significantly greater degree of variance in DZ twin scores (s^2=0.81) versus MZ variance (s^2=0.71) at alpha=.05 (see Appendix F). While this difference does not pose a serious threat to the assumption of homogeneity of variance, it raises the possibility of small competitive SI effects on adolescent PA. However, as discussed immediately below, lack of power due to insufficient sample size must also be considered as affecting the capacity of PA models to detect the **C** effects.

The smaller the **C** effect initially identified, the greater the statistical power required to adequately detect (i.e., retain) such effects. Generally in line with Baker et al. (2008), the initial estimates of **C** effects derived from the full models for PA constituted 17% of variance associated with childhood PA (n=310 twin pairs), and the 12.2% associated with the full PA model based on the older cohort data (n=1181 twin pairs). A basic power analysis (Visscher, 2004; Visscher, Gordon & Neale, 2008) of the initial full univariate model of PA suggested a sample size of 807 and 1284 twin pairs for the younger and older cohorts respectively would have been required to adequately detect (i.e., retain) these respective **C** estimates. Interestingly, Baker et al. (2008), with their sample size of n=1219 twin pairs who were similar in age to those in the current younger cohort, reported a robust estimate of PA of around 21%. While it is difficult to directly compare the current findings with Baker et al. (2008) who's results were derived from a bivariate Cholesky analysis (see Chapter 6 for description of Cholesky model), the current findings are nonetheless generally consistent with Baker et al.'s (2008) own conclusion that,

> "A salient finding is that genetic influences appeared to contribute more to proactive than reactive aggression...[suggesting that] reactive aggression in young children may arise more in response to environmental influences such as the aggravating behaviour of peer group members than proactive aggression, which may instead be more influenced by genetic processes".

It may well be that any true **C** effects on PA effects were substantially weaker than those for RA and hence more difficult to detect (e.g., more susceptible to the effects of sample size). Further clarification of the role of **C** effects as they relate to the current data set is reserved for subsequent analyses and chapters.

5.4 Results of Multivariate Modeling of Aggression Subtypes

Table 4.5, p. 149, displays cross-twin correlations for the aggression subtypes. Results from the best fitting multivariate models of RA and PA are presented in Table 5.2. Findings indicate that etiological overlap between subtypes, for both age groups, is explained predominantly by genetics (accounting for 37.2% and 44.9% of covariance in aggression subtypes in younger and older cohorts respectively), only marginally by non-shared environment (4.8% and 2.9%) and not at all by shared environment (see Figure 5.5).

There was also evidence of subtype specific genetic influences for RA (14.4%) in the older cohort and PA in both both younger and older age groups (36% and 38.4% respectively). Consistent with both Brendgen et al. (2006) and Baker et al. (2008) genetic effects were generally stronger on PA compared to RA. When considering

genetic influences on PA it is interesting that those effects contributing to overlap between subtypes are of equivalent magnitude to those operating exclusively on PA (and not RA). Of course, the qualification needs to be made that, like Baker et al., these multivariate analyses do not include a control for UCF.

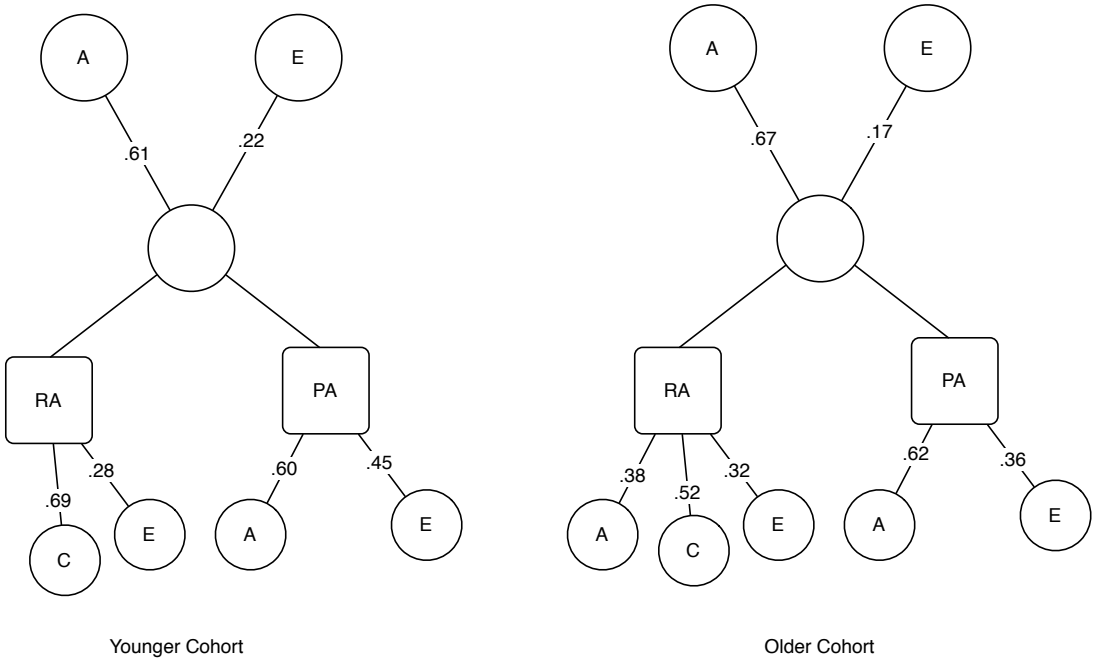


Figure 5.5 Best fitting multivariate path model for aggression subtypes in younger and older cohorts

Notably, while the univariate model of PA is generally consistent with the smorgasbord hypothesis, the multivariate model component of PA is more equivocal. In the latter analysis, estimates of both common and subtype-specific genetic and environmental effects on PA are similar across both age groups. This may reflect the limitations of using a single time point model in extrapolating to longitudinal hypotheses. Later chapters consider longitudinal data relevant to the current research.

Multivariate results for RA were somewhat more consistent and less ambiguous. The bivariate model indicates that any increased influence of genes on RA over the

course of development is likely to be underpinned by sub-type specific genes. It is noted that this result would be buttressed by verification from a longitudinal sample that includes some control for underlying common form. This analysis is presented in Chapter 6.

Table 5.2

Parameter estimates for bivariate genetic and environmental effects on reactive and proactive aggression in younger and older cohorts

	Latent Factors													
	Common Factors			RA-specific			PA-specific			$\chi 2$	*p*	AIC	RMSEA	*df*
	A	C	E	a	c	e	a	c	e					
Younger Cohort														
	.48 (.30, .63)	.41 (.00, .58)	.22 (.16, .28)	.22 (.00, .45)	.65 (.50, .76)	.28 (.23, .33)	.58 (.35, .68)	.10 (.00, .47)	.44 (.40, .50)	2.66	0.995	-19.34	0.00	11
	.61 (.53, .69)	-	.22 (.15, .27)	.00 (.00, .37)	.69 (.57, .78)	.28 (.23, .33)	.58 (.35, .69)	.16 (.00, .49)	.45 (.40, .50)	6.49	0.890	-17.51	0.00	12
	.61 (.53, .69)	-	.22 (.15, .27)	.00 (.00, .36)	.69 (.61, .78)	.28 (.24, .33)	.60 (.51, .69)	-	.45 (.40, .50)	6.54	0.920	-19.46	0.00	13
	.61 (.53, .69)	**-**	**.22 (.15, .27)**	**-**	**.69 (.61, .78)**	**.28 (.24, .33)**	**.60 (.51, .69)**	**-**	**.45 (.40, .50)**	**6.54**	**0.950**	**-21.46**	**0.00**	**14**
Older Cohort														
	.63 (.55, .70)	.24 (.00, .39)	.17 (.14, .20)	.40 (.28, .51)	.50 (.39, .59)	.32 (.29, .34)	.60 (.51, .66)	.14 (.00, .36)	.36 (.34, .38)	4.88	0.937	-17.12	0.00	11
	.67 (.55, .70)	-	.17 (.14, .20)	.39 (.26, .49)	.52 (.42, .60)	.32 (.29, .34)	.60 (.51, .66)	.17 (.00, .37)	.36 (.34, .39)	6.21	0.905	-17.78	0.00	12
	.67 (.63, .71)	**-**	**.17 (.14, .20)**	**.38 (.25, .48)**	**.52 (.43, .60)**	**.32 (.29, .34)**	**.62 (.58, .67)**	**-**	**.36 (.34, .38)**	**6.48**	**0.927**	**-19.52**	**0.00**	**13**

Confidence intervals presented in parentheses, AIC = Akaike information criteria, RMSEA = Root-mean-square error of approximation. A = Genetic variance component, C = Shared environmental variance component, E = Non-shared environmental variance component. **NB** Bold text denotes best fitting models.

Finally, as with the univariate models, results obtained fail to rule out the possibility of sample size effects. Specifically, it is possible a **C** effect is operating on PA, as found in Baker et al. (2008), but that a greater number of participants are required to achieve adequate statistical power to detect such an effect. This is reflected in the wide confidence intervals for the **C** component in PA, that ranged between .49 at the upper limit and 0 at the lower limit in the younger cohort, and .37 and 0 in the older cohort. For the younger cohort, this is a particular difficulty because it indicates the true proportion of variance attributable to this component ranges anywhere between negligible to influencing nearly a quarter of the variance. For the older cohort, it should be noted that the upper limit identified for the shared environment component suggests that the true proportion of variance attributable to this component (in relation to PA) is unlikely to exceed 13.7% at its upper limit. Whether statistically significant or not, the contribution of shared environment to PA in adolescence is thus likely to be modest at best. The question in this case concerns whether the effect of shared environment on PA is negligible, or alternatively, small but meaningful at this later developmental stage. The consistently wide confidence intervals derived from analyses of the older cohort suggest that any additional power gained by this comparatively larger sample (relative to the younger sample) is still not adequate to definitively determine an estimate of **C** effects on PA.

5.5 Etiology of Sex Differences in Aggression Subtypes

This section relates to the third aim of the first objective of Study 1; to assess for sex differences in aggression subtypes. The presentations of results is preceeded by a

brief synopsis of the current literature regarding sex differences in ASB and aggression.

5.5.1 Brief Literature Review of Sex Differences in Antisocial Behaviour and Aggression

Many key correlates of criminal behaviour have shown only negligible gender specificity (Messer, Maughan, Quinton & Taylor, 2004; Odgers et al., 2008). Relevant to the current context, Rhee & Waldman (2002) found that sex differences made no significant contribution to variation in heritability of ASB. However, where aggression is evaluated independently from non-aggressive ASB, the distinction between *quantitative* and *qualitative* differences in underlying etiology becomes an important consideration. Quantitative differences in the current context refer to aggressogenic influences that are shared by both males and females but vary in their relative magnitude as a function of sex. Qualitative differences are relevant where males are affected by an altogether different set of etiological mechanism compared to those that affect females.

Studies of caregiver- and teacher-rated aggression have consistently shown that while genes play an important aggressogenic role for both sexes, heritability estimates for males generally exceed those found for females (Baker et al., 2008; Miles & Carey, 1997)—although see Vierikko et al. (2004) for a notable exception. Crucially though, evidence is equally compelling that these sex differences in aggression are quantitative rather than qualitative (Baker et al., 2008; Brendgen et al., 2008; Eley, Lichtenstein & Stevenson, 1999; Vierikko et al., 2004). That is, analyses of caregiver- and teacher-rated aggression tend to favour statistical models

in which the genetic architecture of aggression (Eley et al., 1999), and RA and PA more specifically (Baker et al., 2008; Brendgen et al., 2006), are structurally identical for both sexes. In fact, both Brendgen et al. (2006), and Baker et al. (2008) reported no sex differences (not even quantitative differences) in either teacher- or caregiver-rated RA and PA. However, self-reported RA and PA in the Baker et al., study showed marked sex differences, with males showing high heritability for both RA (38%) and PA (50%), and females demonstrating zero heritability on both subtypes. The age range of participants in the Brendgen et al. study was 6 years, while participants in the Baker et al. study were 9-10 years.

Notable in the literature, however, is that existing reports on the etiology of sex effects in aggression have tended to treat the role of chronological age somewhat nominally. That is, age ranges are either relatively narrow or collapsed as a variable, with little reference to the (developmental) theoretical implications of doing so. In some cases, this is because neither sex- nor age-related differences in aggression are identified in initial omnibus tests (e.g., Eley et al., 1999). At the same time, longitudinal investigations that do include analysis of sex effects have tended to focus on the etiology of continuity versus discontinuity of aggression (Rushton, Fulker, Neale, Nias & Eysenck, 1986; Tuvblad et al., 2005; van Beijsterveldt et al., 2003; Vierikko, Pulkkinen, Kaprio & Rose, 2006). For clarity, the concept of *discontinuity* is different to the notion of *desistance*. The former refers to any change in the behaviour being measured whether that be an increase or decrease in the behaviour. Desistance refers to only a decrease in behaviour. While the value of examining continuity versus discontinuity in aggression is beyond question, the longitudinal literature remains silent regarding possible developmental changes in the

relative strength of etiological influences.

As such, the possibility that age differentially moderates the relative influence of etiological factors on aggressive behaviour as a function of sex has not been addressed in the genetic literature. This empirical question is nonetheless relevant given (1) extant evidence for quantitative sex differences, and (2) conclusions articulated in Miles and Carey's meta-analytic study which raised the possibility that genetic architecture of aggression potentially undergoes quantitative changes across development (i.e., the smorgasbord model). The current research provides genetic data on the etiology of sex differences in aggression subtypes that compares information from childhood versus adolescence cohorts.

There are a number of different ways to assess for sex differences in the genetic etiology of behaviour. The most common being the general sex-limitation (SL) model in which extends the classic twin model by stratifying male and female data within that common pathway model structure (described in Chapter 3). To the author's knowledge, this represents the only methdological approach to sex differences used in the RA and PA literature to date (e.g., (e.g., Baker et al., 2008).

As noted, the SL model is an extension of the basic twin model described in Chapter 3. Where the classical twin model represents a comparison of two pathway models (one for MZ twins and the other for DZ twins), the SL represents a manifold pathway model that is stratified on the basis of sex in addition to zygosity. As such, the overall structure includes five pathways models, one for each of the following groups; MZ males, MZ females, DZ males, DZ females, and opposite-sex DZ twins.

A path diagram illustrating the specification for opposite-sex DZ twin pairs is presented in Figure 5.6. The critical structural modification includes the addition of a primed pathway to each of the five pathway models. This additional path, labeled $\mathbf{a'_m}$ (see Figure 5.6 for an example), accounts for sex-specific genetic effects associated with male twins only, and thus which is not correlated with genetic effects on the female phenotype (Neale & Cardon, 1992).

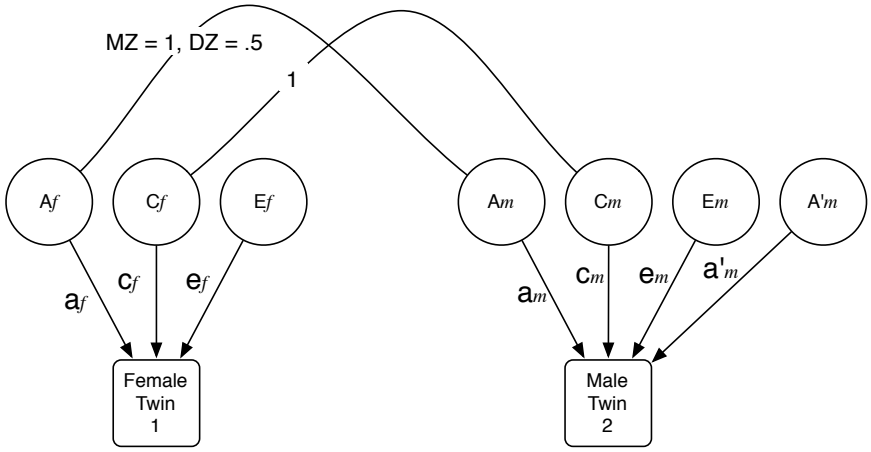


Figure 5.6 The general genotype x sex interaction model for twin data. Path diagram for opposite-sex DZ twin pairs. A denotes latent variable representing additive genetic variance component, C denotes shared environmental component and E denotes nonshared environmental component. f denotes female-specific variance component. m denotes male-specific variance component.

For MZ and same-sex DZ male twin models, the additional A'_m latent variable is essentially treated as a standard A standard additive genetic pathway. Specifically, the additional A'_m variables in the male MZ and same-sex male DZ models are fixed to 1 and .5 respectively (reflecting degree of genetic relatedness). The opposite-sex DZ pathway model (Figure 5.6) differs again from the other four pathway models in none of the a_f , c_f or e_f path coefficients are constrained to be equal with their respective a_m, c_m or e_m counterparts (Neale & Cardon, 1992).

The presence of sex effects is evaluated by comparing this *general* SL model with the *common effects* variant of the SL model (Neale & Cardon, 1992). The latter specification is a restricted version of the former in which all sex-specific primed pathways in the general SL model are set to 0 for both MZ and DZ male twins. If the nested (common effects) model shows no deterioration in fit when compared to the general specification, the hypothesis that qualitatively different etiological factors operate for males versus females can be rejected. An important point here is that this latter conclusion still allows for the *magnitude* of parameter estimates to vary across male and female groups.

At the same time, assessing for sex-differences can be achieved by comparing differences in underlying liability associated with the relevant behaviour. This is called the Polygenic Multiple Threshold (PMT) method and is relevant where (1) are identified sex differences in prevalence of the behaviour under investigation, and (2) etiology is assumed to be multifactorial (Rhee et al., 2001). RA and PA meet both criteria with females typically showing lower levels of both types of aggression (Connor, Steingard, Anderson & Melloni, 2003), and strong evidence suggesting these behavioural dimensions are multi-determined (Baker et al., 2008; Brendgen et al., 2006; Vitaro et al., 1998).

The PMT model views sex-related differences in prevalence as originating from differential sensitivity to a common set of etiological factors. In the current research then, PMT explains lower prevalence in females as reflecting a greater amount of liability required to manifest pathology. From this perspective, the female distribution of liability is, (1) similar in shape to that found for males, but (2)

negatively offset relative to the male distribution. This model leads to the testable prediction that, at any given level of pathology, females are more likely than males to have an affected relative (Rhee et al., 2001).

Thus, the PMT model defines sex-related etiological differences as essentially *quantitative* in nature. A useful complement to this analysis is the Constitutional Variability (CV) model which tests for qualitative differences in etiology as a function of sex (Rhee et al., 2001). The CV model tests for the inverse of the PMT in positing that the lower prevalence in females is the result of sex-specific atypical pathogens, while conversely, causal factors affecting male pathology are continuous in the normal population. It follows from these assumptions that, at any given level of pathology, males are significantly more likely than females to have affected relatives.

It is important to clarify that there is no a priori theoretical premise or empirical work that predicts RA or PA in females is caused by atypical pathogenic mechansims (i.e., influences that are not continuous in the normal population). The inclusion of the CV test nonetheless provides a worthwhile adjunct to the PMT analysis as it potentially affords more definitive conclusions.

In statistical terms, the CV model utilises DeFries-Fulker regression methodology (DeFries & Fulker, 1985; DeFries & Fulker, 1988) to evaluate whether the difference between heritability for deviant scores ($\mathbf{h^2_g}$) versus normally distributed scores ($\mathbf{h^2}$) significantly diverges for males and females. Deviant scores for RA and PA can be determined using Dodge's proposed cut-off of one standard deviation above the

estimated population mean. Support for the CV model constitutes findings for which $\mathbf{h^2_g - h^2} = 0$ for males while $\mathbf{h^2_g - h^2}$ for females is (1) positive where atypical causal factors represent rare genetic etiology, or (2) negative where atypical causal factors represent rare environmental influence (Rhee et al., 2001).

5.5.2 Results from the Sex-limitation Models of Aggression Subtypes

What is immediately apparent in current sex-limitation modeling for RA and PA is that common effects pathway models consistently fit better than the generalised model. This finding was consistent across both age groups. It should be noted that due to the extensive partitioning of data by both sex and zygosity, SL models place considerable burden on sample size to deliver the power required to detect effects. Partitioning for these analyses saw the *n* fall to between 70 and 100 participants for younger cohort subgroups, and around 280 and 320 for older cohort subgroups. Thus while the resulting models appeared to obtain adequate fit, parameter estimates should be interpreted with caution. A second point, is the negative parameter estimates that commonly occur in these models. This phenomenon can sometimes index competition effects (i.e., a form of sibling interaction in which behaviour in one twin decreases the likelihood of that behaviour in the co-twin) (Hay, 1985; Levy & Hay, 2001).

Table 5.3

Parameter estimates derived from general and common effects sex-limitation models of aggression subtypes in both age cohorts

	Males				Females			$\chi 2$	*p*	AIC	RMSEA	*df*
	a	c	e	a'	a	c	e					
RA												
Younger Cohort												
general SL model	.55 (-.25, .88)	.75 (.55, .91)	-.42 (-.49, .49)	.04 (-.64, .64)	.57 (.40, .74)	.77 (.59, .92)	-.30 (-.35, -.26)	0.00	1.00	-7.99	0.00	4
common effects SL	**.55 (.33, .71)**	**.75 (.56, .86)**	**-.42 (-,49, -.37)**	-	**.57 (.40, .73)**	**.76 (.59, .91)**	**-.30 (-.35, -.26)**	**0.02**	**1.00**	**-17.98**	**0.00**	**9**
Older Cohort												
general SL model	0.81 (.68, .99)	.75 (.29, .62)	-.33 (-.36, .36)	.11 (-.46, .46)	.69 (.59, .80)	.61 (.46, .72)	-.37 (-.40, -.35)	0.30	0.99	-7.68	0.00	4
common effects SL	**0.85 (.78, .91)**	**.75 (.26, .54)**	**0.33 (.31, .36)**	-	**.69 (.59, .81)**	**0.62 (.42, .73)**	**0.38 (.35, .40)**	**1.76**	**0.99**	**-16.24**	**0.00**	**9**
PA												
Younger Cohort												
general SL model	-.66 (-1.04, -.	.75 (-.20, .74)	-.53 (-.61, .61)	.11 (-.65, .65)	.34 (-.68, .62)	.79 (.59, .95)	-.50 (-.58, -.44)	0.14	0.99	-7.86	0.00	4
common effects SL	**.80 (.44, .92)**	**.75 (-.11, .72)**	**.52 (,46, .60)**	-	**.32 (-.63, .66)**	**.80 (.56, .96)**	**.51 (.44, .59)**	**0.99**	**0.99**	**-17.01**	**0.00**	**9**
Older Cohort												
general SL model	.90 (.82, .96)	.75 (-.10, .30)	-.39 (-.42, .28)	.14 (-.47, .47)	.77 (.66, .89)	.44 (-.61, .61)	-.45 (-.48, -.42)	6.62	0.16	-1.38	0.02	4
common effects SL	**.93 (.88, .97)**	**.75 (-.10, .26)**	**-.39 (-.43, -.37)**	-	**.78 (.66, .90)**	**.44 (-.60, .60)**	**0.45 (.41, .48)**	**9.08**	**0.43**	**-8.92**	**0.02**	**9**

RA = Reactive aggression, PA = Proactive aggression; SL = Sex-limitation; AIC = Akaike information criteria; RMSEA = Root-mean-square error of approximation. **NB** Bold text denotes best fitting models

5.5.3 Results from the Polygenic Multiple Threshold Model

The statistical model underpinning the PMT analysis is defined immediately below.

Equation 1 (from Rhee et al., 2001).

$$C = \beta_1 SP + \beta_2 P + \beta_3 SC + \beta_4 SPSC$$

C = co-twin/co-sibling, SP = sex of proband, $\beta_2 P$ = proband score, SC sex of co-twin/co-sibling, SPSC = product of sex of proband and sex of co-twin/co-sibling.

β_1 estimates the degree to which proband sex influences co-twin/co-sibling's score over and above that accounted for by (1) proband's score, (2) co-twin/co-sibling's sex, and (3) the interaction between proband's sex and co-twin/co-sibling's sex.

PMT analyses were conducted separately for each age cohort. In younger cohort, PA $t(228)=1.99$, $p = .048$, but not RA, $t(228)=.427$, $p = .670$, was found to be associated with a PMT effect in this group. For older cohort, both RA $t(922) = 2.42$, $p = .015$, and PA $t(926) = 2.11$, $p = .035$ were found to have different liability thresholds for males and females.

5.5.4 Results from the Constitutional Variability Model

A Constitutional Variability model was conducted for each age cohort separately to assess the possibility that sex differences indicate divergent underlying etiologies. Opposite-sex twins and siblings were included in this model (from families where

there are one or more siblings in addition to twins). All participants in the younger age cohort (including all siblings) were under 10 years old. Participants in the older cohort were 10 years or over.

The following modified DF regression equations were used to predict the co-twin/co-sibling score (C) from proband's score (P), the coefficient of genetic relationship (where R = 1 and .5 for monozygositic and dizygotic twins respectively), sex (coded as 0 and 1 for male and females respectively) and a series of interaction terms (where PS is the product of sex and proband's score, RS is the product of the genetic coefficient and sex and PRS is the product of the proband's score, genetic coefficient and sex).

Equation 2 (from Rhee et al., 2001).

$$C = \beta_1 P + \beta_2 R + \beta_3 S + \beta_4 PS + \beta_5 RS + A$$

Equation 3 (from Rhee et al., 2001).

$$C = \beta_6 P + \beta_7 R + \beta_8 S + \beta_9 PR + \beta_{10} PS + \beta_{11} RS + \beta_{12} PRS + A$$

Equation 2 provides an estimate of h^2_g via B_5 . B_{12} and B_{11} in Equation 3 are estimates of h^2 and $h^2 - h^2_g$ respectively.

Scores were represented as deviations from population means divided by proband

score - population mean in order for direct estimates of h^2_g and h^2 to be obtained. Population mean was estimated from the broader ATR sample. As in Rhee, standard error was corrected for double entry. Results yielded mixed support for the CV model. Table 5.4 displays the relevant results.

Table 5.4

Sex effects on h2g - h2 derived from the Constitutional Variability model

	RA			PA		
	t	*p*	n	*t*	*p*	n
Younger aged cohort						
h^2_g	-0.56	0.58	145	**-2.37**	**0.02**	**116**
h^2	1.20	0.23	145	0.68	0.50	116
$h^2_g - h^2$	-1.30	0.19	145	-1.34	0.18	116
Older aged cohort						
h^2_g	-0.51	0.61	342	0.40	0.69	236
h^2	**-2.90**	**<0.01**	**342**	-0.37	0.71	236
$h^2_g - h^2$	**2.73**	**<0.01**	**342**	0.71	0.48	236

RA= Reactive aggression, PA= Proactive aggression, h^2_g = heritability for deviant scores; h^2 = heritability for normally distributed scores; $h^2_g - h^2$ = difference between heritability for deviant scores and normally distributed scores.

Few sex differences were found in the younger cohort. The exception was for younger participants who scored above the cut-off of one standard deviation above the population mean for PA. For this selected "extreme" group, the CV model revealed higher levels of heritability for PA in males compared to females. In light of the fact that no sex differences in h^2_g-h^2 were found in the younger cohort, the finding of sex differences in h^2_g by itself does not necessarily pose a significant threat to the assumption that PA, on the whole, is distributed normally for children in the broader population. However, it is noted that results from the PMT model suggested quantitative sex differences in PA for the younger cohort. Larger samples are needed to determine the degree to which the relevant underlying distributions differ from

one another at the higher end of these distributions. The current results suggest that while heritability for PA in younger males scoring high on this measure is stronger than females in the upper end of the PA distribution, on balance, it is reasonable to suggest these differences are quantitative rather than qualitative.

More difficult to interpret are the results for the older cohort. Specifically, significant differences in h^2_g-h^2 were found between males and females for the RA aggression subtype. A subsequent post-hoc RA analysis of separate groups based on sex revealed that h^2_g-h^2 deviated from 0 for older males, h^2_g-h^2 = -.74 (t=-3.305, p=.001). Conversely, there was no significant difference between estimates of h^2_g and h^2 for RA in older females h^2_g-h^2 = -.127 (t=-5.12, p=.69). This result raises the possibility that extreme RA is discontinuous in the normal population for adolescent males. Specifically, the results further indicate that relative to more population-typical levels of RA in this group, extreme manifestations of RA are more strongly influenced by *environmental* pathogens—possibly those proposed by Dodge and colleagues. Put in another way, heritability of RA in adolescent males declines significantly at higher levels of this aggression subtype. The implications of this finding are discussed in Chapter 8.

5.5.5 Discussion

Generally consistent with previous research (Baker et al., 2008; Brendgen et al., 2006), results from basic sex-limitation (SL) modeling did not support the presence of qualitative sex-specific effects for RA or PA. The common effects model consistently demonstrated superior fit over the general SL structure for both RA and PA in both age cohorts. This suggests the main architecture for genetic and

environmental effects remains consistent for both sexes in the two age cohorts tested.

At the same time, small to modest differences were found in the magnitude of parameter estimates across sex groups. Results from the SL analyses suggests that these sex differences in prevalence for both RA and PA are due to quantitative rather than qualitative differences in underlying etiology. Consistent with this observation, the PMT analysis suggested quantitative differences in etiology for PA in the younger and older cohort, and for RA in the older cohort. Interestingly, the significant *t* value reflecting differential liability thresholds in PA for younger participants was less than that found in the older cohort. While this between-groups difference in *t* values ($t_{\text{younger cohort}} - t_{\text{older cohort}} = .43$, difference in $df = 240$) was well below the floor threshold required for significance in *t* value comparisons (i.e., 1), it is nonetheless consistent with the notion of a trend towards increased sex differences with age. Certainly, the absence of quantitative sex differences in RA in the younger cohort, compared to the presence of such effects in the older cohort supports this hypothesis. Taken together, these results provide preliminary support for the possibility that sex differences increase quantitatively as a function of age.

At the same time, the constitutional variability analyses provided some indication that sex differences in RA in the older cohort might be qualitative rather than merely quantitative. Specifically, it seems that environmental pathogens may play a significantly more substantive role in male RA in adolescence than in childhood. This pattern differs from the univariate and multivariate findings in which sex was collapsed as a variable. These latter results were generally aligned with expectations derived from the smorgasbord hypothesis. That is, increasing genetic influence (and

corresponding reduced environmental influence) with age.

It is noted however, that the results from the CV tests do not necessarily challenge the indications of SL models that structural consistency of the genetic architecture of RA and PA is maintained throughout development. However, it does raise the possibility that differences in the relative influence of genetic versus environmental influences may be of significant magnitude to be considered to represent qualitative differences between-, or (as in the present case) within-sex subgroups. The current CV analyses indicate any such qualitative differences are confined to male RA in the adolescent sample such that more extreme levels of RA in this subgroup are more strongly influenced by environmental factors than population-typical levels. Within-sex subgroup differences such as this necessarily preclude null between-sex subgroup effects on the relevant trait. As noted above, the theoretical implications of this finding are discussed in Chapter 8. While this finding certainly warrants further investigation, it does not, by itself, warrant sex-specific genetic analyses on all subsequent univariate and multivariate models involving adolescent RA. This is because the net effect of this relatively small subgroup of adolescent males (approximately around 7% of the participants in the current older cohort sample) is likely to be marginal on omnibus tests (i.e., models in which sex is collapsed as a variable and differences in means removed) with the potential only to slightly underestimate genetic effects (at most) on RA within the older cohort.

5.6 Etiology of Phenotypic Overlap Between Aggression Subtypes and Disruptive Behaviour Disorder Symptoms

5.6.1 Aggression Subtypes and ADHD Symptomatology

All three ADHD symptom dimensions including hyperactivity, impulsivity and inattention were each submitted to bivariate models that included either RA or PA as covariate. Univariate models for these ADHD symptom dimensions are presented in Appendix G. Table 5.5 presents parameter estimates for all full and best fitting bivariate models for both younger and older cohorts.

Table 5.5

Parameter estimates for reactive aggression and attention-deficit hyperactivity disorder symptoms for both age cohorts

	Latent Factors													
	Common Factors			Aggression subtype-specific			ADHD subtype-specific			$\chi 2$	p	AIC	RMSEA	df
	A	C	E	a	c	e	a	c	e					
RA and Hyperactivity														
Younger Cohort	.48 (.31, .62)	.37 (.00, .55)	.21 (.16, .26)	.32 (.00, .51)	.62 (.46, .75)	.29 (.25, .34)	.75 (.67, .83)	.00 (.00, .28)	.28 (.24, .33)	14.60	0.201	-7.34	0.05	11
	.57 (.49, .66)	**-**	**.20 (.15, .25)**	**-**	**.66 (.51, .77)**	**.29 (.25, .34)**	**.75 (.67, .84)**	**-**	**.28 (.24, .33)**	**18.14**	**0.201**	**-9.86**	**0.05**	**14**
Older Cohort	.48 (.39, .56)	.47 (.36, .55)	.16 (.12, .19)	.57 (.48, .66)	.30 (.00, .44)	.32 (.30, 35)	.67 (.61, .71)	.00 (.00, .24)	.31 (.29, .33)	5.35	0.913	-16.65	0.00	11
	.45 (.37, .54)	**.49 (.40, .57)**	**.16 (.13, .19)**	**.64 (.59, .68)**	**-**	**.32 (.30, 34)**	**.67 (.63, .71)**	**-**	**.31 (.29, .33)**	**8.33**	**0.821**	**-17.67**	**0.00**	**13**
RA and Impulsivity														
Younger Cohort	.51 (.35, .65)	.40 (.00, .57)	.21 (.16, .26)	.21 (.00, .43)	.64 (.50, .76)	.29 (.25, .34)	.65 (.67, .83)	.19 (.00, .48)	.27 (.23, .32)	7.44	0.762	-14.55	0.01	11
	.62 (.54, .70)	**-**	**.21 (.16, .25)**	**-**	**.68 (.60, .77)**	**.29 (.25, .33)**	**.69 (.61, .77)**	**-**	**.27 (.23, .32)**	**11.07**	**0.680**	**-16.93**	**0.03**	**14**
Older Cohort	.57 (.50, .64)	.44 (.32, .53)	.17 (.14, .20)	.48 (.33, .57)	.33 (.14, .45)	.32 (.30, .34)	.62 (.57, .66)	.00 (.00, .22)	.29 (.26, .31)	9.71	0.549	-12.21	0.02	11
	.57 (.50, .64)	**.44 (.32, .53)**	**.17 (.14, .20)**	**.48 (.33, .57)**	**-**	**.32 (.30, .34)**	**.62 (.58, .66)**	**-**	**.29 (.26, .31)**	**14.71**	**0.326**	**-11.29**	**0.02**	**13**
RA and Inattention														
Younger Cohort	.30 (.00, .48)	.50 (.31, .63)	.15 (.07, .21)	.48 (.28, .64)	.54 (.31, .69)	.31 (.27, .36)	.74 (.66, .83)	.00 (.00, .29)	.35 (.30, .40)	7.41	0.765	-14.59	0.02	11
	-	**.57 (.47, .66)**	**.17 (.10, .22)**	**.54 (.41, .58)**	**.48 (.22, .64)**	**.30 (.26, .35)**	**.76 (.68, .85)**	**-**	**.34 (.30, .39)**	**9.62**	**0.724**	**-16.38**	**0.02**	**13**
Older Cohort	.51 (.43, .58)	.43 (.31, .52)	.09 (.00, .14)	.53 (.43, .62)	.37 (.19, .49)	.35 (.33, .37)	.67 (.62, .71)	.00 (.00, .22)	.34 (.31, .36)	9.65	0.554	-12.25	0.01	11
	.52 (.45, .60)	**.42 (.30, .51)**	**-**	**.51 (.41, .60)**	**.39 (.22, .50)**	**.36 (.34, .38)**	**.66 (.62, .71)**	**-**	**.35 (.33, .37)**	**13.31**	**0.424**	**-12.85**	**0.01**	**13**

RA = Reactive aggression, AIC = Akaike information criteria; RMSEA = Root-mean-square error of approximation. NB Bold text denotes best fitting models.

Table 5.6

Parameter estimates for proactive aggression and attention-deficit hyperactivity disorder symptoms for both age cohorts

	Latent Factors													
	Common Factors			Aggression subtype-specific			ADHD subtype-specific			$\chi 2$	p	AIC	RMSEA	df
	A	C	E	a	c	e	a	c	e					
PA and Hyperactivity														
Younger Cohort	.47 (.24, .57)	.00 (.00, .40)	.17 (.03, .24)	.59 (.31, .79)	.39 (.00, .63)	.49 (.44, .56)	.82 (.71, .91)	.00 (.00, .39)	.31 (.25, .36)	6.19	0.860	-15.81	0.00	11
	.50 (.39, .59)	-	-	**.69 (.59, .79)**	-	**.51 (.47, .57)**	**.80 (.72, 89)**	-	**.35 (.31, .39)**	**11.99**	**0.680**	**-18.81**	**0.00**	**15**
Older Cohort	.42 (.32, .52)	.36 (.20, .46)	.16 (.12, .19)	.73 (.64, .77)	.00 (.00, .33)	.37 (.34, .39)	.75 (.66, .80)	.07 (.00, .36)	.31 (.29, .33)	3.84	0.974	-18.15	0.00	11
	.42 (.32, .52)	**.36 (.20, .46)**	**.16 (.12, .19)**	**.73 (.68, .77)**	-	**.37 (.34, .39)**	**.75 (.66, .80)**	-	**.31 (.29, .33)**	**3.85**	**0.993**	**-22.15**	**0.00**	**13**
PA and Impulsivity														
Younger Cohort	.40 (.08, .59)	.35 (.00, .55)	.20 (.12, .26)	.64 (.40, .76)	.20 (.00, .54)	.47 (.42, .53)	.73 (.56, .85)	.24 (.00, .54)	.29 (.23, .34)	16.12	0.137	-5.87	0.05	11
	.53 (.43, .62)	-	**.19 (.11, .25)**	**.67 (.58, .77)**	-	**.47 (.42, .52)**	**.76 (.68, .85)**	-	**.29 (.23, .34)**	**19.25**	**0.155**	**-8.74**	**0.05**	**14**
Older Cohort	.62 (.54, .67)	.09 (.00, .32)	.14 (.10, .18)	.56 (.45, .66)	.35 (.06, .48)	.39 (.36, .41)	.58 (.49, .67)	.40 (.22, .52)	.30 (.28, .32)	0.74	1.000	-21.26	0.00	11
	.63 (.59, .67)	-	**.14 (.10, .17)**	**.65 (.61, .70)**	-	**.38 (.36, .41)**	**.56 (.47, .65)**	**.43 (.28, .53)**	**.30 (.28, .32)**	**5.15**	**0.972**	**-20.85**	**0.00**	**13**
PA and Inattention														
Younger Cohort	.13 (.00, .43)	.41 (.00, .53)	.04 (.00, .17)	.70 (.47, .83)	.24 (.00, .57)	.51 (.45, .57)	.83 (.72, .92)	.00 (.00, .40)	.38 (.32, .42)	4.88	0.937	-17.11	0.00	11
	-	**.43 (.31, .54)**	-	**.74 (.64, .84)**	-	**.51 (.46, .56)**	**.83 (.75, .92)**	-	**.38 (.34, .42)**	**5.09**	**0.991**	**-24.91**	**0.00**	**15**
Older Cohort	.45 (.35, .55)	.33 (.09, .44)	.07 (.00, .12)	.72 (.62, .77)	.13 (.00, .38)	.39 (.37, .42)	.76 (.67, .80)	.00 (.00, .35)	.34 (.32, .36)	1.54	1.000	-20.45	0.00	11
	.55 (.50, .59)	-	-	**.73 (.68, .77)**	-	**.40 (.38, .42)**	**.75 (.71, .80)**	-	**.35 (.33, .36)**	**8.33**	**0.910**	**-21.66**	**0.00**	**15**

PA = Proactive aggression; AIC = Akaike information criteria; RMSEA = Root-mean-square error of approximation. NB Bold text denotes best fitting models.

5.6.1.1 Bivariate modeling of reactive aggression and attention-deficit hyperactivity disorder symptom dimensions

Immediately apparent when observing the RA and ADHD model diagrams presented in Figure 5.7, is the striking similarity between the RA and hyperactivity models, on the one hand, and the RA and impulsivity models, on the other. This similarity extends to the structural differences between younger and older cohort models. Specifically, best fitting bivariate associations with childhood RA indicate that all genetic influences on this latter aggression subtype are shared with hyperactivity or impulsivity—and to a very similar extent, i.e., around a third of variance in the relevant two variables. Non-shared environmental effects common to both traits in this younger cohort are uniformly low, at just under 5% of variance. Both hyperactivity and impulsivity are impacted by a modestly greater magnitude of trait-specific (around 50%) when compared to the relative influence of common genetic effects. Other notable features include a substantial RA-specific shared environmental component in this younger cohort. This component accounted for around 45% of variance in RA (in both models).

As noted, the strong similarity between the RA-related bivariate results involving hyperactivity and impulsivity held for the older cohort models—although these models differed in important ways to those of the younger cohort. Namely, best fitting models for this older age group required the retention of a common shared-environmental component (accounting for around a quarter of variance in each trait). This was accompanied by a corresponding dropping of the RA-specific **C** component present in the younger cohort model. This suggests that in adolescence, shared environment impacts equally on RA and hyperactivity, as well as RA and

impulsivity. While there is increased generalisation of shared environmental effects when comparing younger to older cohorts, there is an inverse increase in the differentiation of genetic influences. This amounts to the presence of an RA-specific genetic component in the older cohort RA models involving hyperactivity and impulsivity, where there was none in the younger cohort. This trait-specific variance component accounted for around 40% of variance in RA in the hyperactivity model and 23% in the impulsivity model.

Interestingly, when inattention was included in a bivariate model with RA, the data suggest that genes do not contribute to covariance between these two symptom dimensions in childhood. This is curious given that some risk factor studies have found unique associations between RA and inattentiveness in amongst primary school aged participants (Dodge et al., 1997; Vitaro et al., 2002). Instead, the overlap between these two behaviour dimensions is accounted for predominantly by shared environment. This is not to say that genes do not play an important role in inattention in younger age groups. Indeed, the present results suggest that just over half of the variance in inattention was attributable to genes (specific to inattention). RA-specific genes (29%) were also present in the younger cohort along with RA-specific C effects (23%). The structure and magnitude of trait-specific variance components are clearly very similar for both RA and inattention when the relevant younger cohort bivariate model is compared to the older age group. The key difference in the older cohort model is the inclusion of a general additive genetic component accounting for around a quarter of the variance in both traits. Moreover, for this latter model, general **E** effects were dropped in the best fitting specification.

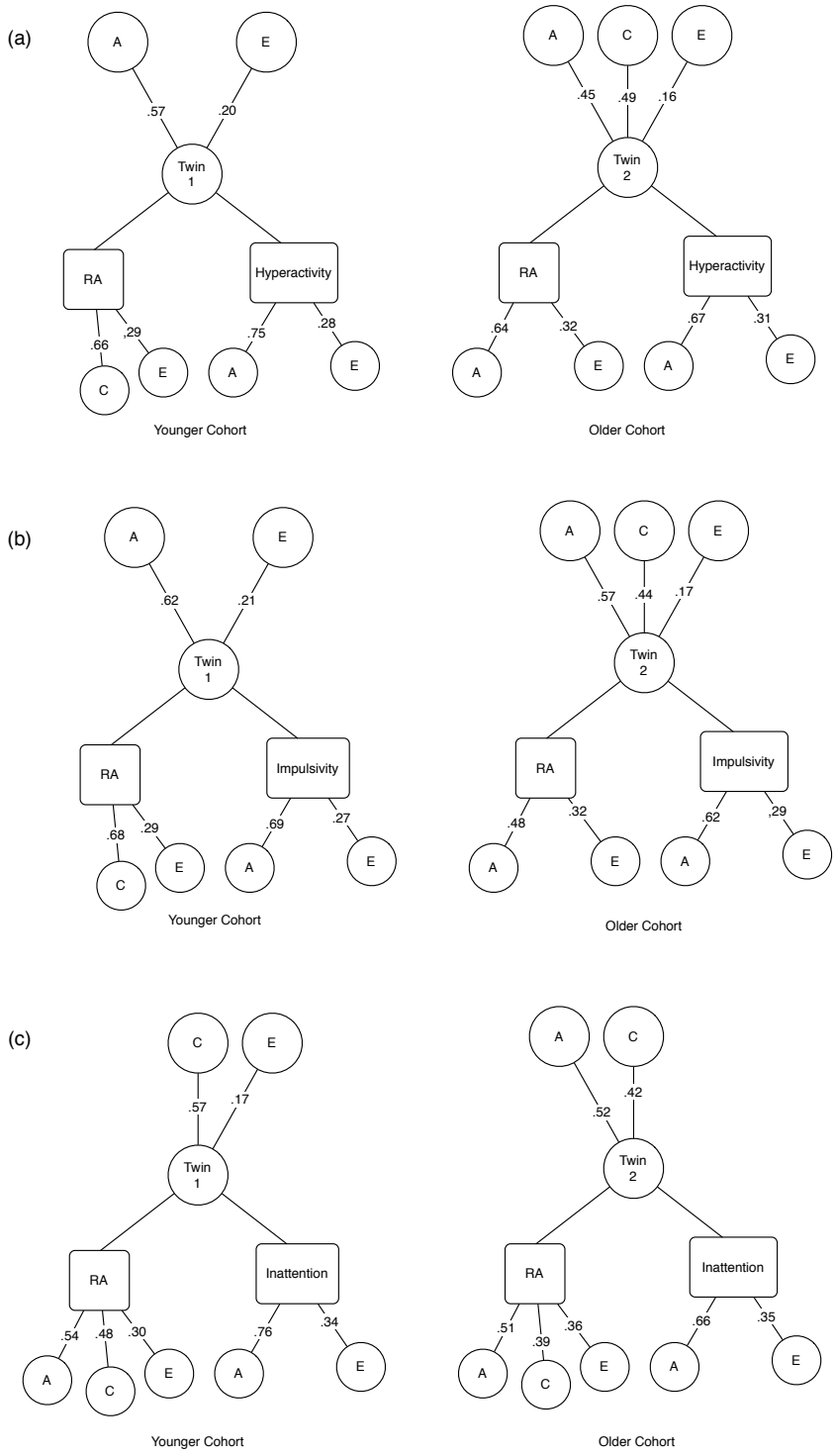


Figure 5.7 Best fitting multivariate path models for reactive aggression and attention-deficit hyperactivity disorder symptom dimensions.

When all models described in this section are considered together, trait-specific **E** effects were required for each variable evaluated. Moreover, the magnitude of these

estimates were remarkably similar across all variables accounting for around 9% of trait-specific variance.

The emergent cross-trait shared environmental component in the older cohort is curious and somewhat counter-intuitive for two reasons. Firstly, hyperactivity is generally associated with suppressed **C** estimates due to rater bias (Rutter, 2006). Secondly, late childhood to adolescence is generally associated with a reduction in the importance of shared environmental influences (Miles & Carey, 1997; Rutter, 2007). One possibility is that this shared **C** factor represents cooperative sibling interaction effects—a phenomenon which mimics the **C** effect (Vierikko et al., 2004), as detailed earlier. This possibility was assessed for both the relevant models (i.e., RA and hyperactivity, as well as RA and impulsivity) separately, and results contraindicate SI effects. For brevity, only the evaluation of the RA and hyperactivity model is described below.

Where cooperative sibling interactions are present, the following pattern of results are expected: (1) increasing levels of reactive aggression in adolescents as a function of increasing concomitant hyperactivity symptoms, and (2) decreasing intra-twin cross-trait covariance as a function of familial relatedness. A Kolmogorov-Smirnov (K-S) chi-square test revealed higher occurence of hyperactivity symptoms amongst those children who exceeded a cut-off of one standard deviation for RA $Z(1, N = 3174) = 7.79$, $p = .000$. A similar result was obtained for impulsivity, $Z(1, N = 3201) = 9.60$, $p = .000$. K-S tests were used because this method remains robust in the face of non-independence of scores within discrete rank groups. For RA and hyperactivity, cross-trait covariance in the MZ group was slightly lower (2.19) than

that found in the DZ group (2.47)—a result that is inconsistent with evidence for cross-trait cooperative sibling interaction effects in the older cohort. The covariances for RA and impulsivity were similar for MZ twins (1.66) and DZ twins (1.67), which contradicts the presence of any sibling interaction.

Thus, there was no evidence for sibling interaction effects on the overlap between RA and hyperactivity or impulsivity.

5.6.1.2 Bivariate modeling of proactive aggression and attention-deficit hyperactivity disorder symptom dimensions

A recurrent pattern in the series of analyses involving PA and the three ADHD symptom dimensions, was the consistent presence of trait-specific **A** and **E** effects (without accompanying **C** influences) for all variables. The only exception was for impulsivity in the older cohort which retained a impulsivity-specific **C** component along with the **A** and **E** estimates. This latter **C** component accounted for around 18% of variance in this ADHD symptom dimension. All other trait-specific components maintained reasonable consistency from model to model, with a range of 45%-55% of variance attributable to trait-specific genetic effects, and 9%-14% attributable to trait-specific non-shared environmental effects.

Somewhat more interesting patterns emerged for the general (i.e., cross-trait) variance components. For example, as in the RA bivariate model of inattention described immediately above, the overlap between PA and inattention in the younger cohort was predominantly the result of shared environmental influences. In fact, in this latter model, **C** effects were the only factor needed to explain phenotypic co-

occurrence (accounting for around 18% of total variance in each phenotype). There was also the expected corresponding greater role of genes in this trait overlap for the older cohort models. The pattern was more pronounced for the PA/inattention versus the RA/inattention models. Specifically, in the adolescent group, only genes were needed to account for the shared variance between PA and inattention (accounting for around 30% of variance).

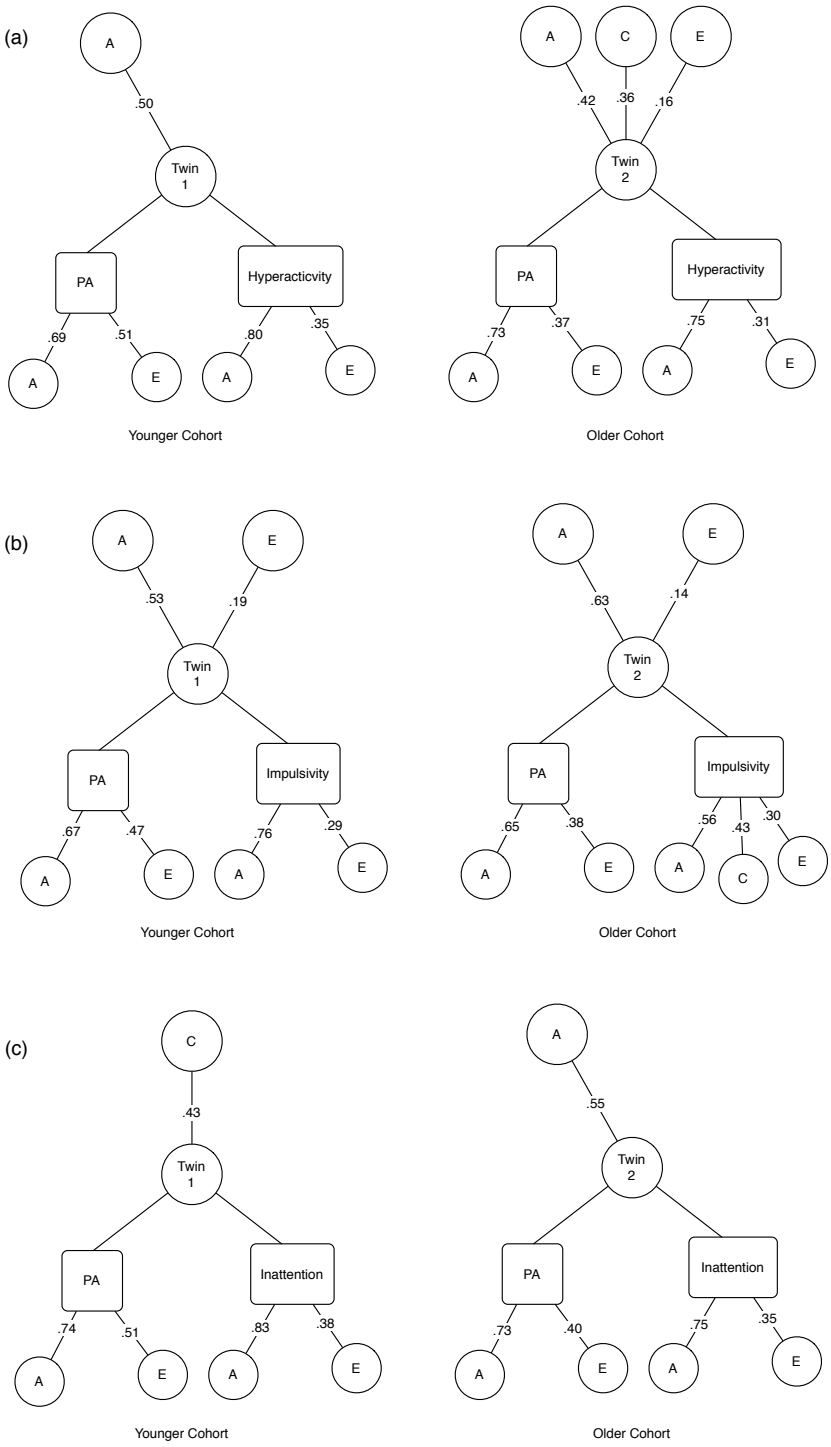


Figure 5.8 Best fitting multivariate path model for proactive aggression and attention-deficit hyperactivity disorder symptom dimensions

No general shared-environmental effects were present in the bivariate PA and impulsivity models. The cross-trait genetic factor was stronger in the older cohort (40%) compared to the younger cohort (28%). Non-shared **E** influences operating on

the trait overlap in these latter models remained relatively weak across both age cohorts (around 3%).

Again, resembling the RA models above, etiological influences on the overlap between PA and hyperactivity implicated an exclusive role for genes (25%) in the childhood sample on the one hand, and on the other, shared-environment (13%) as well as genes (18%) to explain this same overlap in the older cohort.

5.6.2 Aggression Subtypes and the Antisocial Disruptive Behaviour Disorders

Univariate models for ODD and CD symptom dimension are presented in Appendix G. Multivariate models which paired each of these DBD syndromes with each of the aggression subtypes are included in the proceeding sections.

Table 5.7

Parameter estimates for aggression subtypes, oppositional defiant and conduct disorder symptoms for both age cohorts

	Latent Factors													
	Common Factors			Aggression subtype-specific			Antisocial DBD subtype-specific			χ^2	*p*	AIC	RMSEA	*df*
	A	C	E	a	c	e	a	c	e					
RA and ODD														
Younger Cohort	.50 (.35, .64)	.59 (.42, .73)	.26 (.21, .31)	.18 (.00, .39)	.49 (.33, .68)	.25 (.20, .30)	.34 (.00, .51)	.31 (.00, .49)	.35 (.31, .48)	4.84	0.939	-17.16	0.00	11
	.50 (.34, .64)	.59 (.43, .73)	.26 (.21, .31)	.12 (.00, .37)	.51 (.37, .61)	.26 (.21, .30)	.44 (.34, .53)	-	.35 (.30, .39)	6.31	0.900	-17.69	0.00	12
	.51 (.38, .64)	**.59 (.42, .72)**	**.26 (.21, .30)**	-	**.53 (.44, .61)**	**.26 (.21, .30)**	**.44 (.34, .53)**	-	**.35 (.30, .39)**	**6.38**	**0.931**	**-19.61**	**0.00**	**13**
Older Cohort	.65 (.57, .72)	.31 (.00, .44)	.24 (.21, .26)	.37 (.24, .48)	.46 (.34, .54)	.29 (.27, .31)	.55 (.46, .61)	.16 (.00, .35)	.32 (.30, .35)	10.28	0.505	-11.72	0.00	11
	.65 (.57, .72)	**.31 (.00, .44)**	**.24 (.21, .26)**	**.37 (.24, .48)**	**.46 (.34, .54)**	**.29 (.27, .31)**	**.55 (.46, .61)**	-	**.32 (.30, .35)**	**12.87**	**0.378**	**-11.13**	**0.01**	**12**
RA and CD														
Younger Cohort	.42 (.24, .56)	.54 (.37, .67)	.23 (.18, .28)	.38 (.17, .53)	.52 (.33, .65)	.26 (.21, .31)	.25 (.00, .47)	.53 (.34, .66)	.37 (.32, .42)	1.43	1.000	-20.57	0.00	11
	.43 (.27, .57)	**.53 (.36, .67)**	**.23 (.18, .28)**	**.39 (.21, .54**	**.51 (.32, .64)**	**.26 (.20, .31)**	-	**.57 (.48, .67)**	**.38 (.34, .42)**	**2.28**	**0.999**	**-21.72**	**0.00**	**12**
Older Cohort	**.58 (.50, .65)**	**.45 (.33 .54)**	**.22 (.28, .25)**	**.36 (.20, .46)**	**.46 (.37, .54)**	**.28 (.26, .31)**	**.41 (.31, .50)**	**.40 (.27, .49)**	**.30 (.28, .32)**	**11.84**	**0.375**	**-10.16**	**0.01**	**11**
PA and ODD														
Younger Cohort	.41 (.00, .61)	.48 (.17, .64)	.35 (.29, .40)	.59 (.41, .68)	.00 (.00, .42)	.38 (.33, .43)	.42 (.15, .60)	.48 (.21, .63)	.28 (.22, .34)	1.33	1.000	-20.66	0.00	11
	.62 (.54, .70)	-	**.33 (.28, 38)**	**.61 (.52, .70)**	-	**.38 (.33, .44)**	-	**.61 (.53, 70)**	**.31 (.26, .36)**	**8.85**	**0.841**	**-19.15**	**0.00**	**14**
Older Cohort	.65 (.57, .72)	.31 (.00, .44)	.24 (.21, .26)	.55 (.46, .61)	.16 (.00, .35)	.32 (.30, .35)	.37 (.24, .48)	.46 (.34, .54)	.29 (.27, .31)	4.37	0.958	-17.63	0.00	11
	71 (.67, .75)	-	**.23 (.21, .26)**	**.57 (.53, .62)**	-	**.32 (.30, .35)**	**.34 (.20, .44)**	**.49 (.39, .56)**	**.29 (.27, .32)**	**7.87**	**0.852**	**-18.12**	**0.00**	**13**
PA and CD														
Younger Cohort	.49 (.28, .65)	.46 (.17, .63)	.39 (.34, .44)	.54 (.41, .62)	.00 (.00, .34)	.34 (.29, .39)	.02 (.00, .33)	.59 (.48, .68)	.17 (.08, .23)	5.03	0.930	-16.96	0.00	11
	.65 (.57, .73)	-	**.38 (.33, .43)**	**.54 (.42, .62)**	-	**.34 (.29, .39)**	-	**.60 (.52, .68)**	**.17 (.08, .22)**	**10.06**	**0.757**	**-17.93**	**0.00**	**14**
Older Cohort	.67 (.60, .74)	.40 (.23, .51)	.27 (.24, .29)	.49 (.43, .53)	.00 (.00, .22)	.30 (.28, .32)	.20 (.00, .33)	.44 (.36, .51)	.28 (.26, .30)	5.64	0.896	-16.36	0.00	11
	.68 (.62, .75)	**.38 (.20, .49)**	**.26 (.24, .29)**	**.49 (.45, .53)**	-	**.30 (.28, .32)**	-	**.48 (.44, .52)**	**.29 (.27, .31)**	**7.19**	**0.892**	**-18.81**	**0.00**	**13**

RA = Reactive aggression, PA = Proactive aggression, ODD = Oppositional defiant disorder symptoms, CD = Conduct disorder symptoms, AIC = Akaike information criteria, RMSEA = Root-mean-square error of approximation. **NB** Bold text denotes best fitting models.

5.6.2.1 Bivariate modeling of reactive aggression, oppositional defiant disorder and conduct disorder symptoms

Bivariate modeling of RA and ODD in the younger cohort revealed a somewhat familiar pattern suggesting RA shares all its genetic influences with this disruptive behaviour dimension. The emerging pattern is one that indicates low genetic specificity for RA in childhood. However, like previous RA related bivariate models, a RA-specific **C** component was retained in the childhood RA/ODD model accounting for around a quarter of variance in this aggression subtype. Furthermore, this recurrent pattern extends to what might be described as increased genetic specificity in older cohorts, with an RA-specific A component being required in the older cohort RA/ODD model (accounting for around 14% of variance in adolescent RA). ODD remained consistent across the two age cohorts in terms of trait-specific effects, requiring only **A** and **E** components and precluding trait-specific **C** effects for both younger and older age group models. All three general variance components were retained in the RA/ODD models for both age groups. In the younger age cohort, cross-trait **A** (25%) and **C** (35%) effects account for a similar proportion of variance with the latter marginally exerting a marginally stronger influence. In the older cohort, this **A** component strengthens to 42%, while the common **C** effects show a corresponding decrease in magnitude to 9%. As with the majority of bivariate models involving DBDs considered so far, non-shared environmental effects contributed only to a modest degree (ranging between 5%-12%).

An interesting pattern emerged for the RA and CD bivariate models. Virtually all variance components were retained in the best fitting models for both younger and older cohorts. The one exception involved dropping the CD-specific **A** component in

the bivariate model for the younger age group. This suggests that childhood CD shares all its genetic influences with RA. The remaining architectural features of these models suggest the relatively equivalent influence of variance components across both RA and CD and their phenotypic overlap.

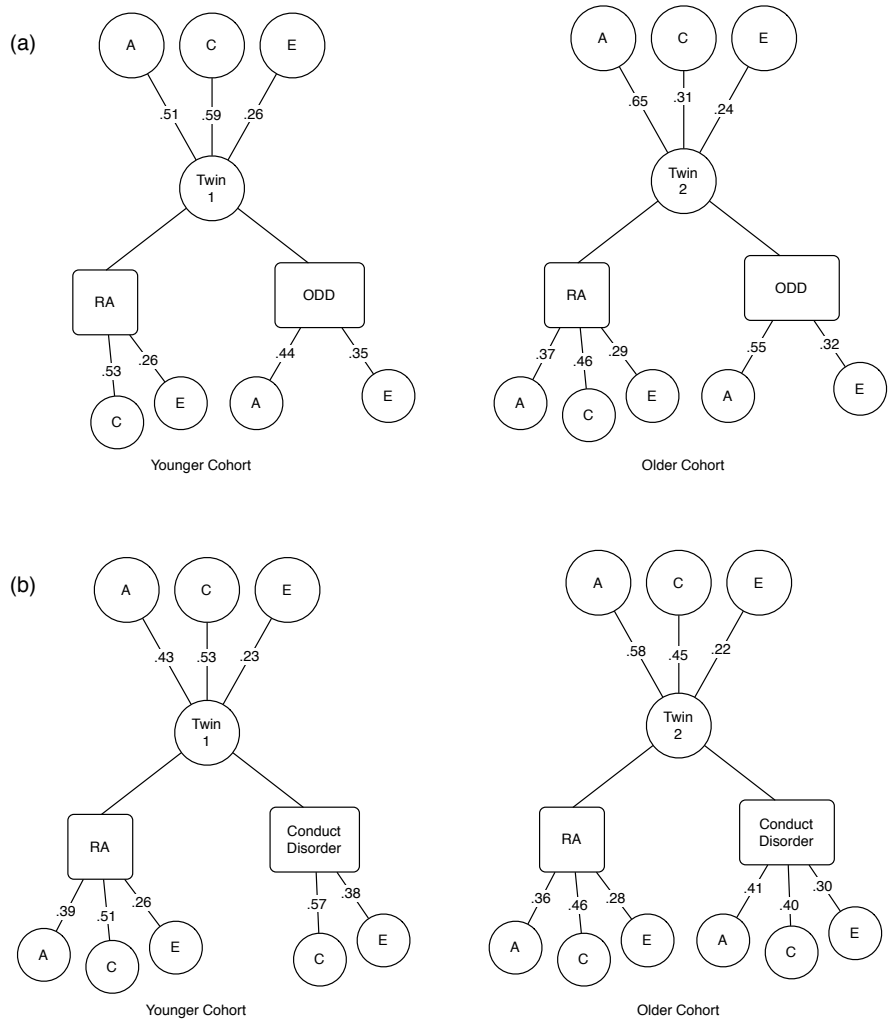


Figure 5.9 Best fitting multivariate path model for reactive aggression and antisocial disruptive behaviour disorder symptom dimensions.

5.6.2.2 Bivariate modeling of proactive aggression, oppositional defiant disorder and conduct disorder symptoms

Immediately apparent in the bivariate analyses involving PA and the two antisocial DBDs is the similarity of the childhood PA and ODD model, and the childhood PA

and CD model. Perhaps more interesting is the relative lack of genetic specificity for both ODD and CD in this younger cohort. That is, the current modeling suggests that all genes influencing ODD and CD in childhood, also influence PA. However, the reverse is not true. PA shows substantial trait-specific genetic influences (accounting for a round a quarter to a third of variance in PA) in this cohort. Conversely, both ODD and CD show greater environmental specificity when compared to PA in this age group. Indeed, while these latter influences account for around a third of variance in both ODD and CD, there are no shared environmental effects operating on childhood PA at all. Genes account for the majority of the overlap in PA+ODD (38%) as well as PA+CD (42%) with general **E** effects accounting for around 12% of trait variance in the younger age group.

The PA+ODD model in the adolescent cohort differed from its younger cohort counterpart only in the addition of an ODD-specific genetic component (12%). This indicates greater genetic specificity for ODD in the older (versus younger) cohort.

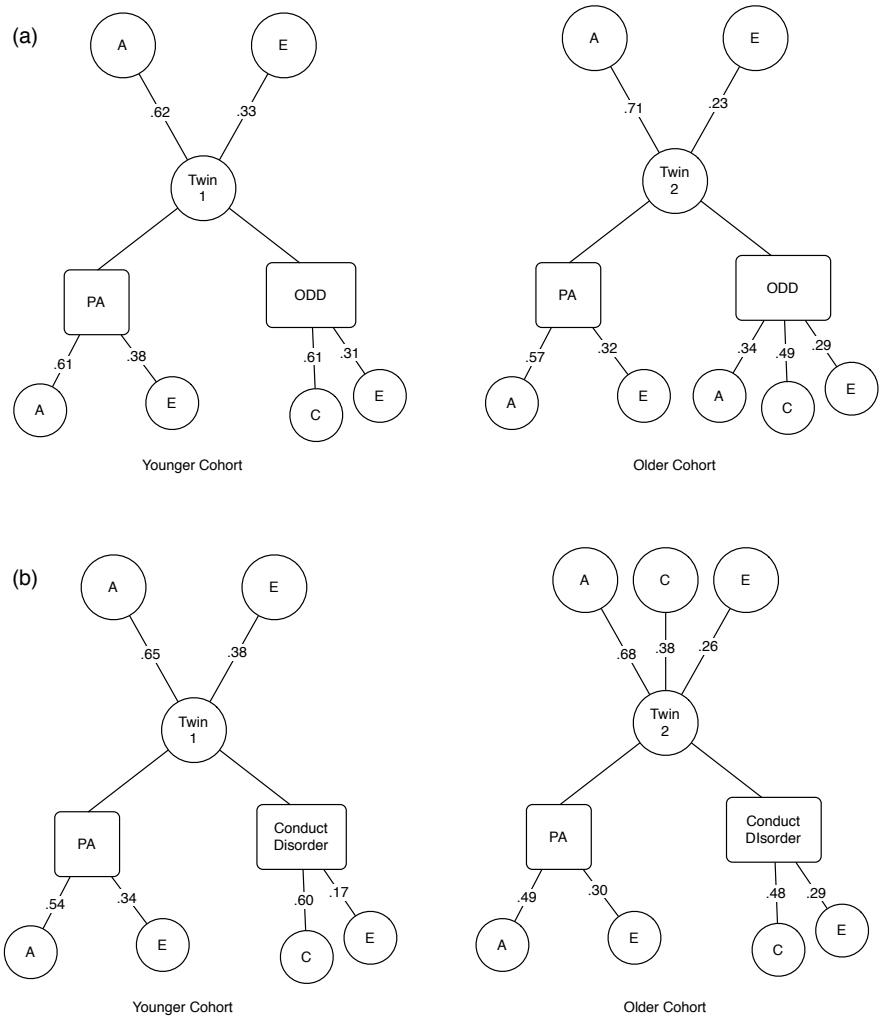


Figure 5.10 Best fitting multivariate path model for proactive aggression and antisocial disruptive behaviour disorder symptom dimensions.

Analysis of PA and CD in the older cohort revealed an emergent cross-trait **C** component (14%). Although only modest in magnitude, this clearly contrasts with previous models of PA which have suggested an absence of shared environmental effects. The possibility of cooperative sibling interaction was examined. A K-S chi-square test revealed higher occurence of CD symptoms amongst those older children who exceeded a cut-off of one standard deviation for PA $Z(1, N = 3174) = 6.06$, $p = .000$. K-S tests were used because this method remains robust in the face of non-independence of scores within discrete rank groups. Intra-twin cross-trait covariances

were pooled for MZ and DZ twins. The second necessary condition providing a preliminary indication of sibling interaction is the presence of decreasing intra-twin cross-trait covariance as a function of familial relatedness. Intra-twin cross-trait covariance was 1.66 for MZ twins and .68 for their DZ counterparts. This clearly contradicts an explanation of cooperative sibling interaction.

5.7 Summary of Cross-sectional Findings

Discussion of the main implications of the current results is reserved for Chapter 8. Nonetheless a few preliminary observations are offered here.

Firstly, the fit of models was generally very good with most models having an RMSEA between 0 and .05. This is due to the relatively robust sample size used in the present research and the resultant statistical power is reflected in the relatively small confidence intervals for most variance components. For example, robust estimates of the **A** variance component confirmed a strong genetic influence on both RA and PA. At the same time, the exception to the generally high reliability of estimates was the large error variance associated with **C** path coefficients—which as expected (e.g., Rutter, 2006), produced far less robust **C** components. Thus while post-hoc tests generally refuted the role of spurious factors underpinning the **C** components that were retained, it could not be ruled out that those best fitting models which precluded **C** effects lacked sufficient power to detect (i.e., retain) them.

Cohort differences in RA-related aspects of the bivariate RA-PA genetic architecture revealed patterns consistent with the smorgasbord hypothesis. That is, proportionally higher genetic loadings for genetic factors compared with shared environmental

effects in the older cohort, with the inverse pattern of results for the younger cohort. This pattern was accompanied by a trend suggesting low genetic specificity for RA in the childhood sample, and increased genetic specificity (i.e., increased differentiation from other disruptive/aggressive behaviours in regards to genes) in the older cohort. This indicates RA is a broader phenotype in childhood than adolescence. This issue is discussed in more detail in Chapter 8.

The smorgasbord hypothesis was clearly less relevant for PA given the overall lack of **C** effects found for this latter aggression subtype. Nonetheless, consistent with Baker et al. (2008), PA was found to be influenced by genes to substantially greater extent that RA. The current study suggests this is true for both younger and older cohorts. There was some evidence of slightly stronger genetic influences in the adolescent group compared with their younger counterparts. However, this differential was marginal (being compared, as it was, to an already high set of estimates).

The multivariate models of adolescent PA on the one hand, and hyperactivity and CD on the other, revealed a bivariate cross-trait **C** component. This result was anomalous in respect to the fact that no other models indicated **C** effects on PA. Because this shared **C** component was evident only in the adolescent cohort data, this pattern was, by definition, opposite to that expected by the smorgasbord hypothesis. Moreover, the magnitude of **A** effects on PA in these latter models seemed to show consistency across age cohorts (again, at odds with the smorgasbord hypothesis). These findings are considered in more detail in following chapters.

Chapter 6.

Developmental Relationships Between Aggression Subtypes and Disruptive Behaviour Disorders

6.1 Overview

The following chapter reports on the series of repeated-measures analyses relevant to Study 2a. This latter study involved the recruitment of a subset of 511 caregivers who completed the ATBRS as part of a larger Curtin research project (which encompassed the current Study 1) and who additionally consented to participate in the second study. Four hundred and thirty three of the 511 completed questionnaires were included in the longitudinal analyses (see Chapter 3).

All 24 items of the short-from ATBRS-ES questionnaire utilised in Study 2a derive directly from the 48 items used in Study 1 and both utilise the same four-point Likert scale. The quantitative genetic analyses of these longitudinal data are the focus of this chapter.

It is noted that unlike the initial measurement occasion, Study 2 data was obtained exclusively in electronic format via the internet. This involved adapting the ATRBRS into electronic format using a web-application software program called Flash—see Chapter 7. The rationale, technology and procedures underpinning this methodology are provided in Chapter 7.

The introduction section of this chapter extends the literature review provided in Chapter 2. The purpose of this elaboration is to relate identified issues of continuity

(also termed persistence) of aggressive and antisocial behaviour over time, to the specific hypotheses guiding the current analyses. The method section that follows briefly outlines the procedure, instruments and statistical methods used for the relevant analyses. The results section reports on longitudinal relationships revealed in the data with particular reference to the main objective and three aims of Study 2a.

6.2 Introduction

6.2.1 Developmental Continuity and Aggression Subtypes

6.2.1.1 Candidate etiological mechanisms for continuity

Despite extensive evidence highlighting the important role of genes in the stability of aggression over time (Eley, Lichtenstein & Moffitt, 2003; Haberstick, Schmitz, Young & Hewitt, 2006; van Beijsterveldt et al., 2003; Vierikko et al., 2006), there has been no longitudinal genetic analysis of RA and PA hitherto reported in the literature. The primary objective of this chapter is to provide a preliminary investigation of etiological factors effecting continuity in aggression subtypes.

As noted previously, research has consistently revealed substantial genetic effects on both RA and PA (Baker et al., 2008; Brendgen et al., 2006). However, these studies have indicated these effects contribute only marginally to the differential shaping of subtypes when either teacher- or caregiver rated reported are used (Brendgen et al., 2006)(Baker et al., 2008). Most notably, the work of Brendgen et al. raises the possibility that non-shared environmental (**E**) effects contribute to emergence, and persistence, in RA and PA to a substantially greater extent than genetic effects.

It is interestingly to note then that both peer affiliation and peer-rejection have been found to contribute to continuity in aggression (Ladd & Burges, 2001; Warman & Cohen, 2000, all cited in Vierikko et al., 2006) although this research has typically precluded identification of aggression subtypes thus failing to account for differential impact of peer relations on RA and PA. One exception is a recent risk factor study by Fite and Colder (2007) who reported on a 12-month longitudinal study of 77 children aged 9-12 years which revealed no longitudinal relationship between PA and peer delinquency on the one hand, and a strong relationship between RA and later peer delinquency on the other. It is noted that Fite and Colder's (2007) findings lead to a slightly different prediction than proposed by Brendgen et al. (2006) whose data derived from a somewhat younger age cohort. That is, this latter study predicts large peer effects on RA but not PA, while Brendgen et al's results predict large **E** effects for both RA and PA with PA showing relatively stronger effects.

To the author's knowledge, there are no reported longitudinal genetic studies on RA and PA. Nonetheless, extant, longitudinal research into the genetics of undifferentiated aggression offer some clues. Namely, Haberstick et al. (2006) identified non-shared environmental influences as an etiological source of stability in aggression measured in middle childhood and early adolescence. However, these **E** effects accounted for only a small proportion of variance associated with continuity (for both maternal and teacher rated aggressive behaviour) with genes ultimately accounting for the majority of longitudinal variance. Interestingly, the sample ascertained by Haberstick et al. (2006) was of similar age to those in Fite and Colder's (2007) study. This raises the speculative possibility of relatively larger **E**

estimates associated with RA versus PA in this late-childhood to early-adolescence age range, in contrast to the opposite pattern associated with mid-childhood (e.g., Brendgen et al., 2006).

Brendgen et al. (2006) acknowledged their identification of **E** effects are incompatible with original etiological formulations of RA and PA by Dodge and colleagues (Coie & Dodge, 1997). This latter theoretical model, which defines parenting practices as the primary proximal causal agent for both RA and PA, raises the prospect of differential **C** effects on aggression subtypes (Brendgen et al., 2006). To reiterate, parenting practices and parenting styles are generally considered to represent shared experiences to which twins raised together are equally exposed (Hay, 1985). As such, these practices are often associated with shared environmental effects (Rutter, 2006).

In technical terms, shared environmental effects are factors which increase behavioural similarity between twins, and non-shared environmental factors are those that decrease similarity. At the same time, the contention that all parenting practices *necessarily* increase similarity amongst siblings is clearly untenable. For example, different parenting behaviours directed at different siblings may operate to decrease similarity. Moreover, a large body of literature has confirmed the importance of child factors in affecting the outcome of dyadic interactions (Dadds & Salmon, 2003; Granic & Patterson, 2006; Greene & Doyle, 1999; Patterson, 1982; Patterson, DeBaryshe & Ramsey, 1989).

Thus, the broad concept of *parenting practices* does not map directly to either **C** or **E**

effects. Nonetheless, disciplinary practices underpinned by harsh and/or authoritarian parenting styles represent a particular risk factor in problem child behaviour (Coie & Lenox, 1994; Coie & Dodge, 1997; Weiss, Dodge, Bates & Pettit, 1992)—particularly where genetic risk for conduct problems is involved (Jaffee et al., 2005). Notwithstanding the issue of passive gene-environment correlations in which the apparent link between parenting and child aggression is mediated by parental genotype (Moffitt, 2005b), family-wide harsh parenting can reasonably be assumed to be indexed by **C** effects. It is noted that where gene-environment interactions occur (e.g., Jaffee et al., 2005), the synergistic contribution of **C** effects is typically indexed by the heritability coefficient (i.e., the **A** term) (Moffitt, 2005b; Rutter & Silberg, 2002). Nonetheless, the point here is that the harsh parenting interactions described above are consistent with those that Dodge and colleagues identified as a primary candidate etiological mechanism for RA. Thus, where other spurious causes that mimic **C** effects (i.e., sibling interaction effects and rater bias) can be ruled out, it is reasonable to postulate that **C** effects at least partly index harsh parenting (e.g., Brendgen et al., 2006). Equally, Dodge's theory suggests that generalised (positive) reinforcement of aggressive behaviour in the home encourages persistent instrumental use of aggression (PA). It is equally reasonable to assume, for the purpose of hypothesis testing, that such effects have an equivalent impact on sibling behaviour—thus leading to predictions of **C** effects for this latter subtype (e.g., Brendgen et al., 2006). It is important to emphasise that this formulation specifies that the **C** component for RA is independent of the **C** component of PA as they each theoretically represent effects that differentially shape aggression subtypes.

Using results obtained from a large community sample, Baker et al. (2008) reported

significant, albeit small, trait-specific shared environmental effects on maternally rated RA and PA. However, when teacher ratings were analysed, all genetic and shared environmental influences on RA and PA were found to be shared by both subtypes.

In any event, methodological limitations with both the Brendgen et al. (2006) and Baker et al. (2008) studies provide an important focus for the current longitudinal evaluation of genetic and environmental effects on RA and PA. Specifically, while Brendgen et al. (2006) utilised separate teacher ratings (which reflects regional public policy rather than deliberate research design) on a relatively small sample, the Baker study precluded any control for underlying form of aggression (an issue discussed in more detail below). Both studies reported data on a relatively narrow age range (i.e., participants ranging between 6 and 10 years).

The current study represents a relatively large scale population sample of twins reared together with behaviour ratings provided by caregivers. The inclusion of two age cohorts also provides a means to assess cross-sectional cohort differences alongside genuine longitudinal effects. Finally, the scope of behaviours measured in the current research allowed inclusion of a covariate control for underlying common form of aggression.

6.2.1.2 The role of underlying common form of aggression in reactive and proactive aggression subtypes

Brendgen et al. (2006) cite research conducted by Little at al. (2003) which argues that (bivariate) analyses that include only RA and PA, potentially confound the

effects of the common form of behaviour underlying both aggression subtypes which they refer to as overt physical aggression. In keeping with previous research (Little et al., 2003), the current paper refers to this as an underlying common form of aggression—abbreviated to UCF.

Little and colleagues (Card & Little, 2006; Little et al., 2003), argue that while there is strong evidence to validate the distinction between RA and PA, many researchers ignore the importance of UCF in disentangling heterogeneity in aggressive behaviour. This is of particular relevance to studies using scales such as the Aggression Scales (AS) for which every item indexes overt physical aggression.

In their study, Little et al. (2003) proposed two theoretically orthogonal dimensions; (1) an overt physical aggression—covert relational aggression continuum and (2) a reactive—proactive aggression dimension. According to these investigators, the first dimension indexes *form* of aggression while the second dimension is concerned with the *function* of aggression. Using this framework 36 self-report questionnaire items relating to aggressive behaviour were classified as being in one of four categories;

- *overt-reactive* (e.g., "When I'm hurt by someone, I often fight back"),
- *overt-proactive* (e.g., "I often threaten others to get what I want"),
- *relational-reactive* (e.g., "When I am upset with others, I often ignore or stop talking to them") and,
- *relational-proactive* (e.g., "I often keep others from being in my group of friends to get what I want").

Little et al. (2003) also identified a range of indices of *pure overt* aggression (e.g.,

"I'm the kind of person who often fights with others") and *pure relational* aggression (e.g., "I'm the kind of person who says mean things about others"). Crucially, neither scale included items that index the function of aggression.

Using an structural equation modeling approach, Little et al. (2003) demonstrated that a four factor solution representing the four subordinate categories provided superior fit when compared with (1) a six factor solution that included the four subtype factors as well as the two "pure" factors, and (2) a two factor solution in which items were only distinguished on the basis of one or the other of the identified orthogonal dimensions.

Brendgen et al. (2006) cite the above research as the basis for including an UCF index to control for overt physical aggression. While these latter authors did not detail the specific items that comprised their UCF measure, such scales necessarily include items that index overt physical aggression without differentiating on the basis of function (i.e., "pure" overt physical aggression items). This approach was adopted for relevant analyses reported below. The construction of the proxy UCF measure used in the present research is described in the following method section.

In sum, controlling for UCF when using the Aggression Scales is particularly important where the purpose is to disentangle discrete etiological factors that might, to some extent, be differentially associated with subtypes. The value of including such a covariate is further highlighted by theoretical suggestions of a RA-to-PA risk trajectory that proposes sequentially overlapping pathways (Vitaro & Brendgen, 2005). Certainly, the question of longitudinal effects needs to account for the

possibility that any longitudinal links between subtypes can distinguish whether putative sequential comorbidity is the result of subtype specific factors or simply underlying risk for overt aggressive behaviour.

6.2.2 The Sequential Hypothesis and Continuity in Aggression

The sequential hypothesis was introduced and discussed in Chapter 2. The model proposes that RA precedes PA in development and that PA simply represents positively reinforced RA (Vitaro & Brendgen, 2005).

There are currently no reported tests of this hypothesis and while Vitaro and Brendgen ((2005)) synthesise and present a range of empirical work consistent with their model, they qualify that their proposed risk pathway remains speculative.

The current research considers a number of testable predictions of the sequential hypothesis, two of which are relevant to the analyses reported in this chapter. Firstly, the main mechanism proposed to convert RA behaviour to PA behaviour constitutes simple operant conditioning—which is effectively homologous to the mechanisms proposed by Dodge and colleagues (for the development of PA). This leads to the prediction of shared environmental influences linking RA at time to PA at time 2.

It is worth highlighting that the kind of longitudinal **C** effects derived from the sequential model do not easily translate to cross-sectional data such as those reported by Baker et al. Indeed, the proposed transition from RA to PA is likely to implicate different mechanisms at different time points. Most notably, while the sequential model predicts **C** effects underpinning the linkage between RA and later PA, it also

suggests biogenic factors are important for (at least) the emergence of RA (Vitaro & Brendgen, 2005). Specifically, Vitaro and Brendgen emphasise both neuropsychological features underpinning poor emotional regulation and genetic vulnerability as contributing factors in the ontogenic emergence of RA in childhood.

At the same time, a number of related disruptive behaviour dimensions were also included in the present analyses to provide a meaningful clinical context within which to interpret results. Relationships between the aggression subtypes and DBDs are considered next.

6.2.3 Continuity, Aggression Subtypes and the Disruptive Behaviour Disorders

6.2.3.1 Do impulsivity and inattention symptoms represent etiologically distinct pathways to future reactive aggression?

6.2.3.1.1 The role of impulsivity in reactive aggression pathways

At around the time Dodge presented his seminal formulations of RA-PA, Barratt (1991) proposed a bimodal classification of aggression that distinguished between *impulsive* and *premeditated* types of aggression. For the purposes of the current thesis, this distinction is considered conceptually synonymous to the RA-PA distinction. In providing a theoretical formulation of impulsive aggression (IA), Barratt and Slaughter (1991) hypothesised that the purely aggressive features of IA behaviour are secondary to a deficit in impulse control. Although some commentators have noted the theoretical value of clarifying this proposed etiological relationship (Hinshaw, 2002), hitherto called the primacy hypothesis, the relationship between impulsivity and RA has received surprisingly little research attention

(Waschbusch et al., 1998).

It should be noted that, at least one attempt to elucidate links between IA on the one hand, and personality traits related to impulse control and/or anger/hostility on the other, failed to yield evidence for the primacy hypothesis (e.g., Barratt & Slaughter, 1998). Nonetheless, the strong phenotypic link between impulsivity and IA (herein referred to as RA) has been established (Dodge et al., 1997; Raine et al., 2006; Waschbusch et al., 1998). Recently, Raine et al., (2006) found a unique relationship between impulsivity and concurrent RA (after controlling for PA) in adolescent male participants using the Zuckerman Impulsivity Personality Scale. The report does not make clear whether impulsivity was included as a discrete measure in the first wave when participants were age 7 years, and so it is difficult to ascertain the potential prognostic value of impulsivity at age 7. A similar issue regarding the clarity of Wave One ADHD subtype measures used in this study is discussed in the following section.

In their genetic study, Seroczynski et al. (1999) reported that the phenotypic overlap between impulsivity and RA in adults is due to both genetic and environmental influences. However, it appears these authors derived their measure of reactive aggression from items that loaded onto the impulsivity factor. Furthermore, they report no specific parameter estimates associated with this particular bivariate modeling. Results from the current univariate analyses reported in Chapter 5 suggest the genetic architecture of this relationship is sensitive to age effects with genes playing a substantial role in the shared etiology between RA and impulsivity in both childhood and adolescence but with cross-trait **C** influences being important for the

latter age group only. Of course, these current cross-sectional data are clearly silent on the primacy hypothesis.

Empirical work focusing on more proximal mechanisms (i.e., endophenotypes) underpinning this overlap between impulsivity and RA have implicated neuropsychological functioning (e.g., Barratt, 1991; Raine et al., 2006), with particular emphasis on the role of serotonin in impulse control (Apter et al., 1990; Krakowski, 2003; Stein, Hollander & Liebowitz, 1993). However, while it is likely that there exists some genetic basis for impaired serotonergic function as it relates to both ADHD and violent behaviour, it is also probable that the effects of any such biogenic pathogens involved are mediated by environmental conditions (Retz & Rösler, 2009). This suggests a better understanding of gene-environment interactions will be required for a comprehensive elucidation of these risk mechanisms. Nonetheless, evidence from patients with acquired frontal impairment offers compelling evidence for at least some directionality in the relationship between impulsivity and RA. Specifically, adult studies of neurological patients demonstrate a causal link between acquired frontal deficits after injury to the frontal lobe, and problem behaviours relating to impulse control, including RA (Anderson et al., 1999; Bechara et al., 1994; Blair et al., 2001; Damasio, 1994).

In their study of neurocognitive functioning in 198 10-12 year old boys, Giancola, Moss, Martin, Kirisci and Tarter (1996), found evidence that executive cognitive functioning (ECF), which according to the authors, links frontal functioning with impulsivity and cognitive flexibility, was a unique predictor of RA in boys with substance abusing (SA) parents—but, interestingly, not boys without SA parents. In

describing the putative mechanisms involved, the authors note that "impaired ECF facilitates aggressive behavior concomitant to decreased behavioral inhibition and deficiencies in generating alternative nonaggressive responses in provocative situations" (p.740). The neuropsychological literature on RA was discussed in more detail in Chapter 2.

The absence of research elucidating etiological relationships between impulsivity and RA in the developmental literature is curious in light of the fact that the former behaviour dimension is so commonly associated with aggression (Waschbusch et al., 1998). Clarifying possible directionality underpinning this relationship has particular relevance since it represents a notable alternative to psychosocial models of RA that preclude broader mechanisms of impulse control (e.g., Dodge, 1991; Dodge et al., 1997). The hypothesis is not inconsistent with Vitaro and Brendgen's (2005) sequential hypothesis—although the models provide different perspectives on broader etiological pathways.

In sum, due to methodological inconsistencies in the literature (including inconsistent separation or operationalisation of aggression subtypes and/or ADHD subtypes), it is difficult to derive definitive conclusions or predictions from extant research. Nonetheless, data assessing Barratt's primacy hypothesis—at least as far as it concerns RA—deserves some empirical consideration as it has significant implications for understanding the link between disruptive and aggressive pathways.

The series of longitudinal analyses undertaken for the present research therefore includes a model specifying a longitudinal relationship between impulsivity and later

RA. The primacy hypothesis effectively specifies impulsivity as a risk factor for RA that is underpinned by biogenic factors. These latter effects are theoretically distinct from an early propensity for "pure" overt physical aggression. This particular restatement of the primacy hypothesis represents the assumption that a propensity for physical aggression is not the primary causal agent in RA, but rather that poor impulse control leads to disinhibited aggression (Barratt & Slaughter, 1998).

It is well established that biogenic factors involved in the emergence of impulsivity are under partial genetic control (Levy & Hay, 2001; Seroczynski et al., 1999). The current research assesses whether genetic modeling of longitudinal links between impulsivity and RA will reveal a shared genetic component over and above genetic effects underpinning UCF related aggression.

6.2.3.1.2 The role of inattention in reactive aggression pathways

Waschbusch et al. (1998) note that along with a lack of empirical focus on impulsivity in aggression, there is also a paucity of research focusing on the role of attention in aggression. As these authors point out, this is somewhat surprising given that at least two key studies report clear links between inattention and RA (Dodge et al., 1997; Vitaro et al., 2002). For example, Vitaro et al. (2002) reported that reactively aggressive children were more inattentive than their peers, whether or not they also evidenced a proactively-aggressive style. Dodge et al. (1997) provided data demonstrating that highly reactively aggressive boys were rated by teachers as evidencing more attentional problems, even after controlling for impulsivity. As such, the current research includes an evaluation of the longitudinal relationship between inattention and RA with impulsivity included as a covariate control.

6.2.3.2 Do hyperactivity and oppositional defiant symptoms represent etiologically distinct pathways to future proactive aggression?

6.2.3.2.1 The etiological link between hyperactivity and proactive aggression

The frequent co-occurrence of aggression and ADHD symptoms implicates shared mechanisms underpinning continuity (Hinshaw, 2002; Retz & Rösler, 2009). By far the most frequently researched ADHD subtype in this area is the combined hyperactivity and impulsivity (HI) symptom dimension. Predictions of common pathways between HI and aggression have been borne out by both behavior genetic and risk factor studies (Retz & Rösler, 2009). Nonetheless, recent evidence has questioned the prognostic value of HI symptoms when concurrent aggression is accounted for (Broidy et al., 2003). This latter large scale multi-site research project analysed data from across six sites and confirmed the primary role of physical aggression in predicting long-term conduct problems.

Notably, however, this latter study did not differentiate aggression subtypes in their key meausures. A more recent longitudinal risk factor study involving a large community sample followed over 9 years, has reported on differential correlates of aggression subtypes (Raine et al., 2006). Data was derived from a sample of 334 boys and demonstrated that hyperactivity at age 7 years predicted PA at age 16 years after controlling for RA, but that this measure of early hyperactivity did not predict later RA (after accounting for the influence of PA). It should be noted that while these investigators refer to differential relationships between hyperactivity and PA on the hand, and impulsivity and RA on the other, they refer only to a combined HIA measure in their methodology (which combines all ADHD symptoms including hyperactivity, impulsivity and attention, into one scale). It therefore remains unclear

whether their reference to "hyperactivity" represents short-hand for the broader combined HIA scale used at the initial measurement occasion. Either way, in providing a theoretical context for their research, Raine et al. (2006) make reference to Lynam's construct of *fledgling psychopathy* which posits the co-occurrence of conduct disorder symptoms and *hyperactivity* as an early marker of severe life-course persistent ASB and aggression. It is possible that the separation of hyperactivity and impulsivity dimensions is important in clarifying prognostic relationships involving non-aggressive disruptive behaviour and aggression subtypes.

It should be further pointed out that the Raine et al. (2006) study revealed adolescent PA was simultaneously uniquely predicted by a range of additional childhood factors including delinquency, poor school motivation, poor peer relationships, single-parent status, psychosocial adversity, and substance-abusing parents. However, this latter constellation of risk factors represent psychosocial characteristics and socio-economic features which correlate with a range of generalised forms of persistent ASB (Broidy et al., 2003; Loeber et al., 1995). What makes hyperactivity a potentially valuable clinical marker is its potential to help refine our understanding of the high prevalence of comorbid pathways linking ADHD and conduct problems.

For example, given that behaviour genetic studies have consistently demonstrated a strong shared genetic component amongst DBDs (e.g., Dick et al., 2005; Silberg et al., 1996; Thapar, Langley, Owen & O'Donovan, 2007; Tuvblad, Zheng, Raine & Baker, 2008; Tuvblad et al., 2009; Waldman et al., 2001) and particularly ADHD subtypes (Hay, Bennett, McStephen, Rooney & Levy, 2004; Hay et al., 2001; Levy & Hay, 2001; Rasmussen et al., 2002; Rasmussen et al., 2004), it is reasonable to

postulate a genetically mediated pathway linking hyperactivity and PA. However, the empirical question of a unique pathway in which hyperactivity predicts later PA necessitates controlling for concurrent RA—as undertaken in the Raine et al. (2006) study. This basic specification is the focus of a series of longitudinal genetic analyses reported in the method section.

6.2.3.2.2 Proactive aggression and the ODD-to-CD risk pathway: Is hyperactivity involved?

Longitudinal risk factor studies have implicated PA in the ODD-to-CD risk pathway proposed by Loeber and colleagues (Pulkkinen, 1996; Raine et al., 2006; Vitaro et al., 2002; Vitaro et al., 1998). Specifically, PA at age 12 was found to predict both ODD and CD in mid-adolescence (Vitaro et al., 2002). Pulkinnen et al. (1996) reported that PA, but not RA, measured at age 8 years, was longitudinally associated with a range of adjustment problems in adolescence and criminality in adulthood. More recently, Raine et al. (2006) reported that PA at age 16 was uniquely associated a range of concurrent behaviours associated with both CD and psychopathy including, blunted affect, delinquency and serious violent offending.

The overlap between PA on the one hand, and CD behaviours on the other, is not unexpected considering the significant operational overlap between the clinical constructs of PA and CD (Vitaro et al., 2002). While some have argued this overlap renders the RA-PA distinction redundant in within clinical taxonomy (Bushman & Anderson, 2001), others have argued that distinguishing RA and PA may provide means to clarify the ODD-CD risk pathway (Vitaro et al. 2002; Kempse et al., 2005). For example, Vitaro et al. (2002) suggests the possibility that children with ODD and PA may be at increased risk of going on to manifest CD later in development. This

proposition implicates shared etiologies between PA and ODD and there is certainly value in investigating this link.

Taken together, recent findings support both a hyperactivity-to-PA risk pathway, and a role for concomitant PA and ODD in the widely cited ODD-to-CD pathway. It is useful then to investigate etiological associations between hyperactivity, ODD and PA within a common longitudinal model. A series of partial correlational analyses guided the specification of this model. Briefly, these analyses revealed both that hyperactivity predicts future PA after controlling for concurrent ODD (younger cohort, r=.12, p<.05; older cohort; r=.11, p<.05; n=301), and that ODD also showed a significant longitudinal relationship with later PA after concurrent hyperactivity was accounted for (younger cohort, r=.36, p<.05; older cohort; r=.41, p<.05; n=672). Given the substantially greater magnitude of the unique predictive relationship between time-1 ODD and time-2 PA, hyperactivity was assigned to the covariate control. This approach allows for a more detailed elucidation of pathways involving ODD and later PA than would be possible if ODD was placed as the covariate control.

It is noted that due to the constraints of the data collected at the second wave, a longitudinal model including CD at the second wave was not possible. As such, the current evaluation involved modeling only the "front end" of this pathway (i.e., hyperactivity, ODD, and PA).

6.3 Method

6.3.1 Sample Characteristics and Instruments

As noted above, 510 caregivers provided twin ratings on ADHD, RA and PA behaviour dimensions constituting the 24-item ATBRS-ES at the Wave Two data collection which occurred in the three months between March and June of 2008. The 47 items derived from the earlier Wave One ATBRS questionnaire were combined with the 24 items from the ATBRS-ES, affording evaluation of longitudinal etiological relationships between the main variables of interest. As noted in Chapter 2, the series of analyses pertaining to these longitudinal data constitutes **Study 2a**.

It is useful to point out a number of qualifications regarding the longitudinal data. Firstly the number of participants contributing longitudinal data is noticeably less than the combined total of Wave One participants and this has implications regarding the power of the relevant models. Secondly, no measures of the antisocial DBDs (i.e., ODD and CD) were included in the Wave Two **ATBRS-ES** questionniare. As mentioned earlier, this reflects a decision to minimise the length of the online questionnaire as many creagivers had completed the full 289-item version between 18 and 24 months previously and many had participated in other associated research projects in the intervening time period.

6.3.2 Proxy Index of Underlying Common Form (UCF)

In order to derive a proxy measure of underlying common form (UCF) of aggression, i.e., physical aggression (Brendgen et al., 2006; Little et al., 2003), three items were combined from the Conduct Disorder scale of the ATBRS. These items were chosen

due to their explicit reference to physical aggression and included;

1. Initiates physical fights,
2. Has been physically cruel to people and
3. Has used a weapon that can cause serious harm to others (e.g., bat, brick, broken bottle, knife or gun).

As noted above, the defining characteristic of items contributing to a UCF scale requires that they identify only the *form* (i.e., the 'what') and not the *function* (i.e., the 'why') of aggression. The three above items fulfill this criteria on the basis of face validity.

An estimate of Chronbach's alpha was obtained as a measure of scale reliability. The alpha for the UCF index was $\alpha = .59$. This reliability coefficient was considered low but adequate given it was derived from three items only.

6.3.3 Statistical Methods and Pathway Models

6.3.3.1 Use of the Cross-lagged panel Analysis

One of the most structurally simple multivariate longitudinal pathway models is the cross-lagged panel (CLP) analysis (Kline, 2005; Neale & Maes, 2002). The basic CLP model involves evaluating two or more variables each measured across two or more time points. For the current analyses, this results in four variables being entered into the basic cross-lagged panel model (i.e., RA time 1, PA time 1, RA time 2 and PA time 2).

Structurally, the model describes the following relationships; intra-trait longitudinal correlations, cross-trait time-specific correlations, and cross-lagged panel effects. LISREL (Joreskog & Sorbom, 1993) was used to submit longitudinal data to the CLP model so that estimates of independent correlations could be derived while simultaneously accounting for the expected auto-correlations between variables. This method enabled a robust estimate of the degree of independence associated with specific pathway estimates and provided a statistical method of controlling for unmeasured effects. For example, longitudinal path models are generally specified to allow the error terms of observed variables to correlate. This specification is useful for complex multi-determined variables because it accounts for the possibility that unmeasured factors—e.g., rater bias—may be contributing to the covariance of variables within and across Waves.

An important assumption of this model is that causal processes impacting on the variable are stable (and remain stable) across all occasions of measurement (Kline, 2005). This assumption of *stationarity* underscores the fact that the model does not account for dynamic changes associated with causal inputs across measurement occasions. Stationarity can be assumed when the correlation between the two variables are similar at each time point (Hall, Milburn & Epstein, 1993). This relationship is referred to as *synchronous correlations.*

The CLP analyses are included to butress the rationale for the more complex statistical modeling procedures utilised in this chapter.

6.3.3.2 Logitudinal Bivariate Genetic Modeling

The basic bivariate genetic model introduced in Chapter 3 has been utilised in this chapter to provide a first indication of longitudinal relationships between key variables. More specifically, two overall sets of models have been included; one set for RA measured at each of the two time points, and another set for PA. The longitudinal bivariate model provides an evaluation of etiological sources of variance across time points and accounts for both longitudinal and time-specific variance.

Following these bivariate analyses, a series of Cholesky Decomposition Models was undertaken to assess the effect of covariate phenotypes on model results. This approach is described in more detail immediately below.

6.3.3.3 The Cholesky Decomposition Model

To assist disentangling shared from discrete disruptive behaviour pathways, a series of trivariate analyses involving RA, PA as well as the DBDs have been included. The rationale for this approach extends the logic presented in previous chapters. That is, firstly it recognises the high degree of phenotypic overlap between aggression subtypes (and DBDs), and secondly, it allows examination of the degree that RA and PA provide additional information over and above that offered by existing clinical constructs. Because limitations in sample size preclude the ability to model all variables at once, modeling is restricted to a complementary series of key triavriate longitudinal Cholesky Decomposition Models (CDM).

Figure 6.1 illustrates the basic CDM design.

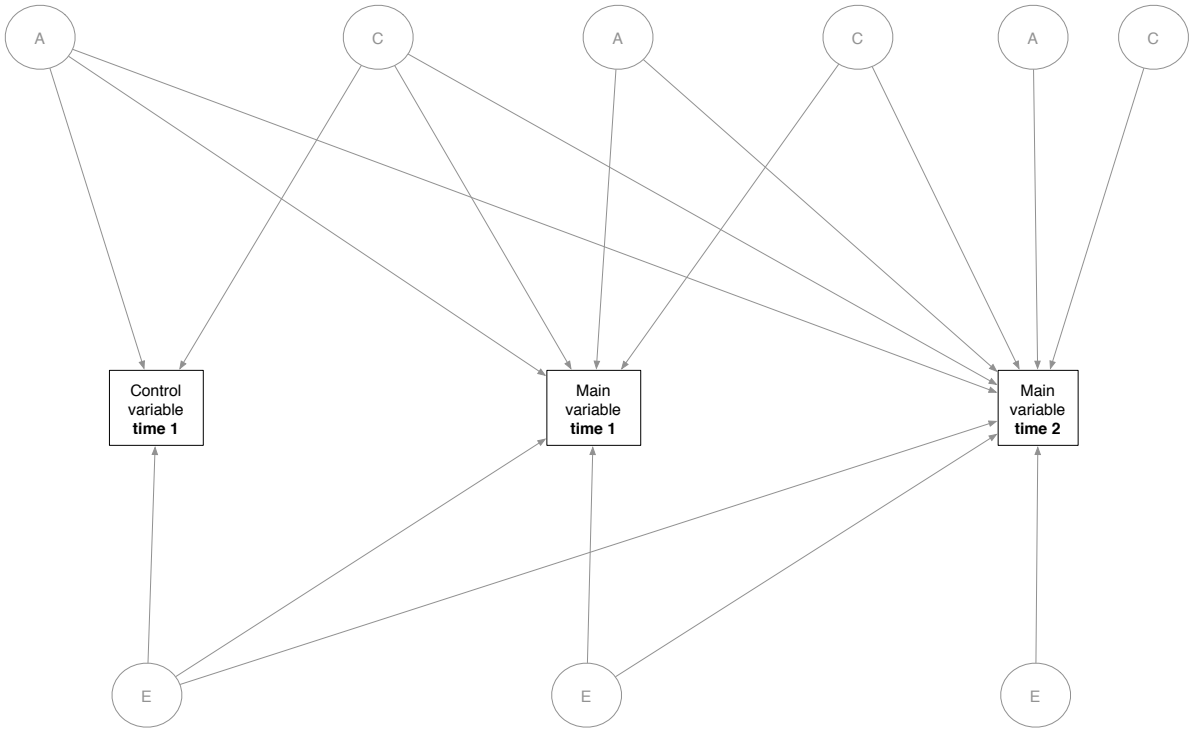


Figure 6.1 The basic Cholesky Decomposition Model pathway diagram. A denotes latent variables associated with genetic effects. C denotes latent variables associated with shared environmental effects. E denotes latent variables associated with non-shared environmental effects.

The CDM is reasonably well suited to temporal (i.e., longitudinal) analysis (Loehlin, 1996) because its hierarchical arrangement allows variance at follow-up Waves to be partitioned into two components; (1) covariance associated with "upstream" effects, and (2) variance not associated with previous Waves (i.e., time-specific, or "new", variance). In this way, the CDM addresses both sources of continuity and change over time.

6.3.3.3.1 Adapting the Cholesky model to control for overlap with concurrent Wave One behaviour dimensions

The trivariate analyses reported in this chapter represent a novel adaption of the CDM longitudinal design. The first variable entered into each model is always a time-1 trait that is chosen as a control variable. The main time-1 trait of interest is

placed in the secondary position of the model. The time-2 trait is specified in the final position. As such, the covariate entered at the first position of the model, along with the trait entered at the second position, represent data obtained from the first measurement occasion. The variable entered into the third position represents the only data in the model that is measured at the second time point.

As alluded to above, in the fully specified CDM, the most "upstream" latent variables specified in the model (i.e., the most far-left **A**, **C** and **E** etiological variance components in Figure 6.1) have effects on all three observed variables (Loehlin, 1996; Waldman et al., 2001). The second set of etiological variance components are uncorrelated with the first set and have effects on the subsequent two observed variables. Finally, the last set of etiological variables represent residual effects indexing variance only in the last observed variable, and only variance in that variable that has not already been accounted for by the "upstream" etiological variance components (Loehlin, 1996; Waldman et al., 2001).

The current CDM design as specified in Figure 6.1 represents a novel adaption of the conventional longitudinal CDM. In conventional longitudinal specifications of the CDM, all observed variables correspond to consecutive measurement occasions. At the same time, the CDM has also been used for multivariate analysis of concurrently measured variables (e.g., Waldman et al., 2001). Due to the hierarchical nature of the CDM structural design, the order that variables are placed in will affect parameter estimates derived. For this reason, it is important to establish a clear rationale for the particular order chosen for concurrently measured variables entered consecutively into the model (Loehlin, 1996).

The current adaption of the CDM represents a mix of the concurrent multivariate and longitudinal designs. Because of its priority positioning in the model, the first observed variable operates as a powerful control for the relationship between then second and third variables. As noted above, in the hybrid CDM design proposed for the current analyses, the longitudinal relationship is specified between the second and third observed variables. With the control variable included in the first position, parameter estimates derived from this longitudinal path represents only residual variance between the time-1 and time-2 variables over and above trivariate variance accounted for by the time-1 control variable placed in the first position of the model. In one sense, the choice of using a time-1 or time-2 variable in the first position, as a control variable, is arbitrary. A time-1 variable was chosen for every model to maintain consistency of design across all longitudinal CDM models, as well as to maintina some temporal consistency between variables within the model specification.

Given the high degree of overlap between both subtypes as well DBDs, trivariate analyses have been included for a range of theoretically important behavioural phenotypes. This is clearly an analysis-intensive approach which is necessarily exploratory in nature. The ideal would be to include all "control" variables into the same model. Unfortunately, the complexity of the genetic design makes this prohibitive due to the exponential loss of power associated with adding additional variables into the model.

In sum, the aim of the trivariate CDM analyses in the present chapter is to provide a preliminary and conservative evaluation of unique versus overlapping longitudinal

relationships between time and 1 and time 2 of the key variables of interest (i.e., RA, PA and DBDs) to complement the bivariate longitudinal analyses.

6.3.3.3.2 Guiding Hypotheses

The process of fitting data to the CDM models was initially informed by a number of guiding hypotheses. This allowed the main model trimming process to be guided by theoretically relevant expectations about the relative impact of different etiological factors.

Presented in Table 6.1 are eight basic hypotheses outlining key alternative structural underpinnings of the main etiological sources of variance. These hypotheses all represent "nested" structures within a full Cholesky Decomposition Model (CDM).

Table 6.1

Guiding hypotheses to inform longitudinal model trimming

	Guiding Hypothesis	Description
1	No A effects operating on continuity	This model represents the hypothesis that genetic effects do not influence continuity
2	A effects are exclusively trait-specific	Model constrained by dropping all cross-trait genetic effects
3	All A effects shared across traits	Model constrained such that all genetic effects are shared across traits
4	No C effects present in the model	This model represents the strong hypothesis that there is a complete absence of shared-environmental effects operating on variables in the model
5	No C effects operating on continuity	Model constrained by dropping all shared-environmental effects influencing continuity
6	C effects are exclusively trait-specific	Model constrained by dropping all cross-trait shared-environmental effects
7	All C effects shared across traits	Model constrained such that all shared-environmental effects are shared across traits
8	No trait-specific A or C effects present	This model is constrained so that there are no trait-specific genetic or shared-environmental effects present in the model

The first three hypotheses refer directly to the influence of genetic (**A**) effects. Hypotheses four through to seven focusing on various possibilities regarding shared environmental (**C**) effects. Hypothesis eight accounts for the possibility of no genetic or shared environmental effects operating exclusively on specific traits. Hypotheses that have no theoretical or empirical basis (e.g., no genetic effects on aggression) have been omitted from this list of guiding hypotheses (and hence from the analyses). Not all hypotheses are mutually exclusive. So for example, Hypotheses 1 and 2 (as well as 5 and 6), conceptually speaking, are potentially mutually inclusive, but not necessarily so. To reiterate, the main purpose of the above hypotheses is to provide a starting point for model fitting that is maximally congruent with existing theoretical frameworks.

To illustrate, Hypothesis 4 articulates a basic prediction based on the replication of Brendgen et al. (2006), i.e., no **C** effects present in the model—with the additional qualification that all **A** effects are predominantly shared across traits. As this latter qualification highlights, the hypotheses listed are not designed to test specific model conditions, but rather provide a range of alternative starting conditions for the model trimming process. To maintain simplicity and focus throughout this process, predictions concerning **E** effects were precluded from the list of hypotheses.

Nonetheless, there is a key prediction concerning this class of effects that stems from Brendgen et al. (2006). At the level of replication, this amounts to the prediction that that non-shared environmental effects will underpin the differentiation of RA and PA. As noted above, these authors speculate that such effects may well be mediated

by peer relationships. Given previous studies showing that key aspects of peer relationships contribute to continuity in aggression, the current analysis also proposes to evaluate the specific hypothesis that **E** effects contribute to continuity in both RA and PA, at least in childhood.

6.3.3.3.3 Hypothesis Testing and the Cholesky

All nested models were tested against the full CDM to determine which provided the most parsimonious description of the data. The most parsimonious model was then subjected to further model trimming in order to derive the best fitting model (again based on the principal of parsimony).

6.3.3.3.4 Issues Relating to the Cholesky

A distinct characteristic of the CDM approach is that the full model—against which subsequent nested specifications are compared—is structurally heirachcal. Specifically, the order in which variables are placed within the model impacts on the nature of the results (Loehlin, 1996). This is particularly problematic when there is no a priori theoretical basis to assign variables to a specific order within the model. However, in the case of longitudinal data, the ordering of variables is non-arbitrary and this limitation can in fact represent a useful property. Nonetheless, there remains significant limitations associated with the longitudinal CDM. Notably, the full CDM does not include trait-specific variance for any but the last variable entered into a the model.

6.4 Results

6.4.1 Descriptives

6.4.1.1 Longitudinal descriptives

Please see Table 4.9, p. 165, for descriptives associated with longitudinal scales.

6.4.1.2 Correlations

Zero-order correlations for RA and PA between time one and time two are presented in Table 4.10, p. 168. It is worth noting that the intraclass cross time-point correlations for both RA and PA remained significant (r=.38, p=.000. and r=.34, p=.000, respectively) after simultaneously controlling for; (1) their respective counter-part aggressive subtype at time one and two, (2) age, (3) sex, (4) zygosity, (5) impulsivity at both time one and two, (6) hyperactivity at both time one and two, and (7) impulsivity at both time one and two. This finding reveals considerable independence and stability for both subtypes over at least short-to-medium time periods. Please note that ODD and CD were not included as control variables due to the fact they were not measured at the second wave.

6.4.2 Initial Examination of Longitudinal Relationships Between Aggression Subtypes

6.4.2.1 Cross-lagged panel analysis of aggression subtypes

Data for RA, PA obtained consecutively over the two measurement occasions were submitted to LISREL, version 8.2 (Joreskog & Sorbom, 1993) for cross-lag panel modeling of longitudinal effects. Results are presented in Figures 6.1 and 6.3.

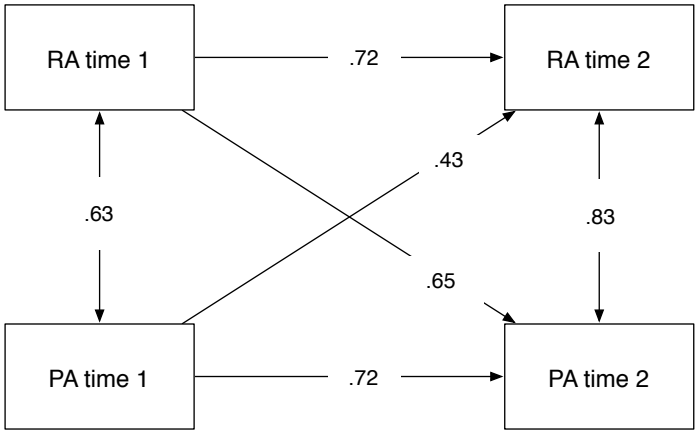


Figure 6.2 Cross-lagged panel model of reactive and proactive aggression for younger participants.

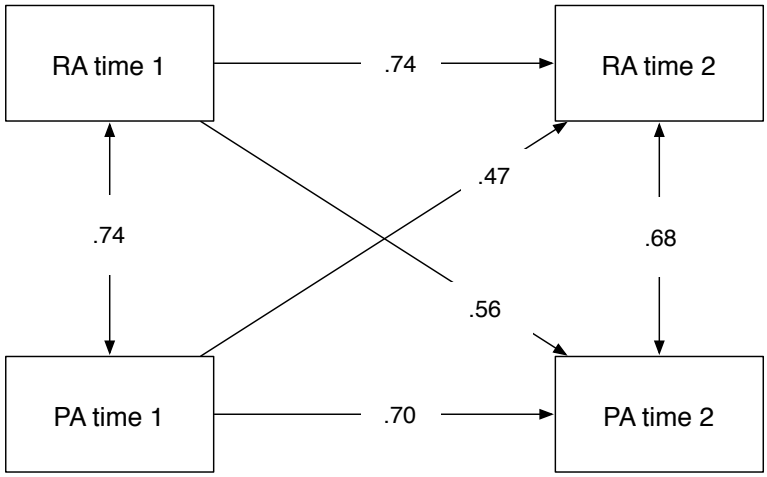


Figure 6.3 Cross-lagged panel model of reactive and proactive aggression for older participants.

For the CLP models, data were double entered (i.e., zygosity collapsed). The n was adjusted according to previously reported procedures. The assumption of stationarity was largely met in the CLP models, particularly in the older cohort, where the two synchronous correlations were r=.74 for RA and PA at time 1 and r=.68 for RA and PA at time 2. Unsurprisingly, there was more variability in the younger cohort with

synchronous correlations of r=.63 and r=.83.

Fit statistics for baseline models were χ^2=66.38, df=42, p=.001, RMSEA=.043 for the younger and χ^2=117.17, df=42, p=.00, RMSEA=.051 for the older cohort. Noticeable, in the younger cohort cross-lagged panel model is the greater magnitude of the $RA_{time1}PA_{time2}$ path coefficient relative to the $PA_{time1}RA_{time2}$ path correlation. This differential which favours the cross-trait predictive capacity of RA over PA, albeit to a small degree only, is broadly consistent with the sequential hypothesis. While this differential was not evident in the older cohort cross-lagged panel model, the $PA_{time1}RA_{time2}$ cross-lag path could nonetheless be fixed to 0 for both cohort models without a significant deterioration in fit (younger cohort, χ^2=69.08, df=43, p=.007, RMSEA=.044; older cohort, χ^2=120.62, df=43, p=.000, RMSEA=.051).

These preliminary results are consistent with the sequential hypothesis. However, it is noted that the most parsimonious fit was ascertained when, for each model, both cross-lagged panel paths were fixed to 0 (younger cohort, χ^2=65.28, df=44, p=.001, RMSEA=.042; older cohort, χ^2=116.47, df=44, p=.000, RMSEA=.050). Along with the consistently low p values for both models, this likely reflects a lack of statistical power due to the relatively small sample size (i.e., relative to the numbers typically required for adequate statistical power in pathway modeling).

6.4.2.2 Bivariate longitudinal modeling of aggression subtypes

For the multivariate longitudinal models, all scores were regressed on age and standardised for sex to account for mean differences across variables.

6.4.2.2.1 Bivariate longitudinal models of reactive aggression

The bivariate model for RA revealed a somewhat complex pattern (see Figure 6.4 and Table 6.2). For the younger cohort, the best fitting model revealed trait-specific effects at time 1 for **C** effects only— accounting for a substantial 47.6% of time-1 variance. The only trait-specific influences operating at time 2 were non-shared environmental (**E**) effects. These latter effects accounted for 41% of overall variance in time-2 RA. Both **A** and **E** effects were found to be shared across time points with the former accounting for a substantial 50% of the variance associated with continuity versus 5% attributable to **E** effects. This loosely resembles a pattern of declining **C** effects and increasing **E** effects at a developmental period marked by a significant increase in peer contact.

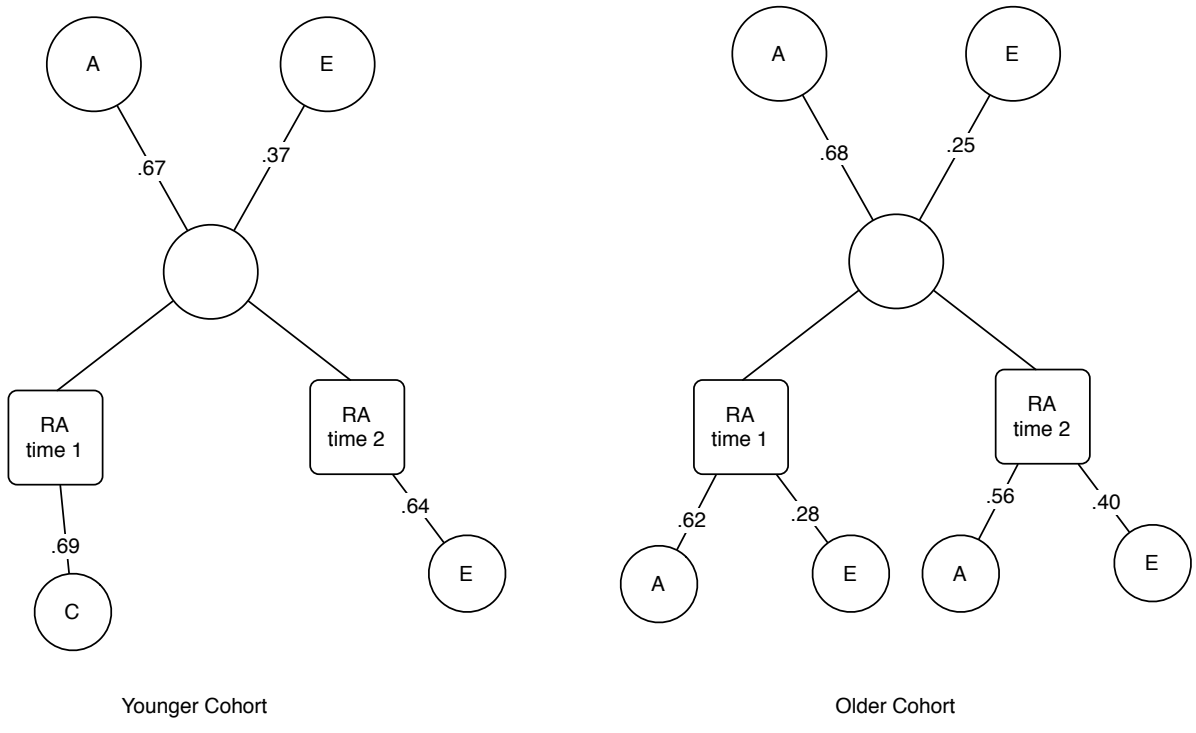


Figure 6.4 Best fitting bivariate path model for time-1 reactive aggression and time-2 reactive aggression for younger and older cohorts

The best fitting bivariate longitudinal model for RA associated with the older cohort

precluded all **C** effects. Again, genetic influences were substantially more dominant (46%) than non-shared environmental effects (6.2%) in accounting for variance associated with continuity across time points. In contrast to the younger cohort, time-specific genetic effects were found at both first (38.4%) and second (31%) measurement occasions. While results indicate that time-specific **E** effects were less influential than their genetic counterparts, they showed a doubling from 7.8% at time 1 to 16% at time 2.

Table 6.2

Parameter estimates for longitudinal bivariate models of time-1 and time-2 reactive aggression for both age cohorts

	Latent Factors													
	Common Factors			RA-specific at time-1			RA-specific at time-2			χ^2	*p*	AIC	RMSEA	*df*
	A	**C**	**E**	**a**	**c**	**e**	**a**	**c**	**e**					
All ages														
	.70 (.59, .76)	.00 (.00, .37)	.24 (.18, .29)	.28 (.00, .46)	.55 (.41, .66)	.29 (.24, .34)	.44 (.20, .60)	.30 (.00, .49)	.42 (.37, .47)	2.72	0.994	-19.27	0.00	11
	.70 (.63, .76)	-	.24 (.18, .29)	.28 (.00, .46)	.55 (.41, .66)	.29 (.24, .34)	.44 (.20, .60)	.30 (.00, .49)	.42 (.37, .47)	2.72	0.997	-21.27	0.00	12
	.70 (.64, .77)	-	.24 (.18, .29)	.24 (.00, .43)	.57 (.44, .67)	.29 (.24, .34)	.53 (.45, .61)	-	.41 (.37, .46)	3.98	0.991	-22.02	0.00	13
	.71 (.65, .77)	**-**	**.23 (.18, .28)**	**-**	**.61 (.54, .68)**	**.30 (.26, .34)**	**.53 (.45, .61)**	**-**	**.41 (.37, .46)**	**5.05**	**0.985**	**-22.95**	**0.00**	**14**
Younger cohort														
	.60 (.25, .81)	.31 (.00, .67)	.27 (.13, .40)	.00 (.00, .52)	.63 (.34, .81)	.25 (.09, .36)	.45 (.00, .67)	.00 (.00, .54)	.51 (.41, .65)	1.41	1.000	-20.59	0.00	11
	.66 (.51, .82)	-	.27 (.12, .39)	.00 (.00, .50)	.63 (.47, .81)	.25 (.08, .36)	.46 (.00, .67)	.00 (.00, .57)	.51 (.41, .65)	1.69	1.000	-22.31	0.00	12
	.66 (.51, .82)	-	.27 (.12, .39)	.00 (.00, .50)	.63 (.47, .81)	.25 (.08, .36)	.46 (.00, .67)	-	.51 (.41, .65)	1.69	1.000	-24.31	0.00	13
	.66 (.51, .82)	-	.27 (.12, .39)	-	.63 (.47, .81)	.25 (.08, .36)	.46 (.10, .67)	-	.51 (.41, .65)	1.69	1.000	-26.31	0.00	14
	.63 (.47, .80)	-	.37 (.31, .46)	-	.66 (.51, .83)	-	.41 (.00, .65)	-	.58 (.48, .71)	6.56	0.969	-23.44	0.00	15
	.67 (.52, .82)	**-**	**.37 (.31, .46)**	**-**	**.69 (.55, .86)**	**-**	**-**	**-**	**.64 (.55, .75)**	**9.164**	**0.907**	**-22.84**	**0.00**	**16**
Older cohort														
	.69 (.50, .78)	.00 (.00, .48)	.25 (.19, .30)	.46 (.15, .46)	.42 (.00, .62)	.28 (.23, .34)	.51 (.16, .65)	.22 (.00, .54)	.40 (.35, .45)	6.86	0.810	-15.14	0.00	11
	.69 (.60, .78)	-	.25 (.19, .30)	.46 (.15, .68)	.42 (.00, .62)	.28 (.23, .34)	.51 (.16, .65)	.22 (.00, .54)	.40 (.35, .45)	6.86	0.867	-17.18	0.00	12
	.69 (.60, .78)	-	.25 (.19, .30)	.61 (.52, .70)	-	.28 (.23, .33)	.47 (.00, .65)	.31 (.00, .57)	.40 (.35, .45)	8.86	0.784	-17.14	0.01	13
	.68 (.60, .77)	**-**	**.25 (.19, .30)**	**.62 (.53, .71)**	**-**	**.28 (.23, .33)**	**.56 (.46, .66)**	**-**	**.40 (.35, .45)**	**9.36**	**0.810**	**-18.64**	**0.01**	**14**

RA = Reactive aggression, AIC = Akaike's information criteria, RMSEA = Root-mean-square error of approximation. A = Cross-trait genetic variance component, C = Cross-trait shared environmental variance component, E = Cross-trait non-shared environmental variance component. a = Subtype-specific genetic component, c = Subtype-specific shared environment component, e = Subtype-specific non-shared environment component **NB** Bold text denotes best fitting models

6.4.2.2.2 Bivariate longitudinal modeling of proactive aggression

Like RA in the older cohort, the best fitting model for PA in the younger cohort precluded **C** effects (see Figure 6.5 and Table 6.3). At the same time, compared with RA in the younger cohort, genetic effects on continuity appeared to be of slightly lesser magnitude, accounting for 29.1% of the variance in stability. Conversely, **E** effects were relatively higher at 17.6%. Time-specific effects played an important role with genetic influences accounting for 46% of overall variance in PA at time 1—and 10.9% of this same time 1 variance attributable to **E** effects. Like RA, **E** effects accounted for all time 2-specific variance in PA, representing 34.8% of the overall variance associated with this second measurement occasion.

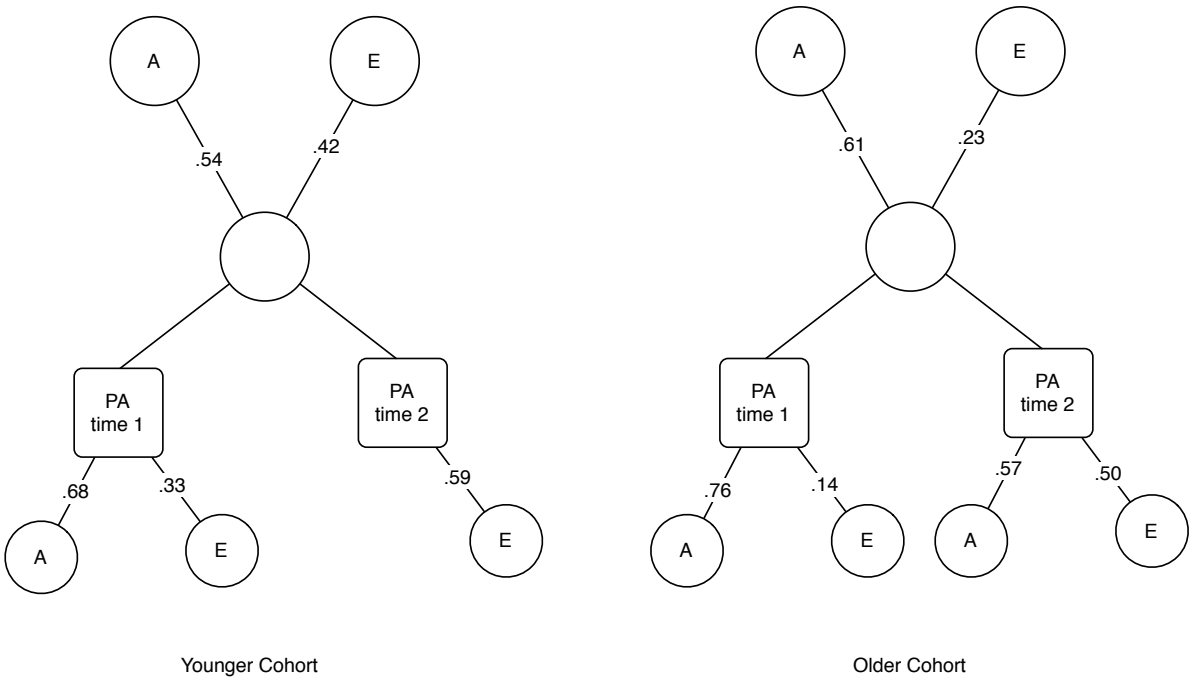


Figure 6.5 Best fitting bivariate path model for time-1 proactive aggression and time-2 proactive aggression for younger and older cohorts

Results from the older cohort were consistent with earlier cross-sectional models in suggesting **C** effects do not play a significant role in the etiology of PA. The effects

of genetic factors influencing continuity in PA appeared marginally stronger (37.2%) for the older cohort (relative to the younger cohort), with a smaller proportion of variance common to both measurement occasions attributable to **E** effects (5.3%) compared with younger cohort. The effect of genes on time-specific variance declined longitudinally from a total of 57.7% of overall time-1 variance to 32.5% of time-2 variance. These figures increased substantially from 2% to 25% across time points for **E** effects.

Table 6.3

Parameter estimates for longitudinal bivariate models of time-1 and time-2 proactive aggression for both age cohorts

	Latent Factors													
	Common Factors			PA-specific at time-1			PA-specific at time-2			χ_2	*p*	AIC	RMSEA	*df*
	A	C	E	a	c	e	a	c	e					
All ages														
	.59 (.50, .66)	.00 (.00, .27)	.26 (.21, .31)	.76 (.70, .83)	.00 (.00, .28)	.21 (.14, .27)	.57 (.32, .66)	.00 (.00, .45)	.52 (.47, .57)	19.14	0.059	-2.86	0.05	11
	.59 (.51, .66)	**-**	**.26 (.21, .31)**	**.76 (.70, .83)**	**-**	**.21 (.14, .27)**	**.57 (.48, .66)**	**-**	**.52 (.47, .57)**	**19.14**	**0.160**	**-8.86**	**0.03**	**14**
Younger cohort														
	.54 (.00, .74)	.00 (.00, .48)	.42 (.26, .57)	.68 (.42, .87)	.00 (.00, .49)	.33 (.09, .50)	.05 (.00, .67)	.43 (.00, .66)	.59 (.46, .73)	6.42	0.840	-15.58	0.00	11
	.54 (.28, .74)	-	.42 (.26, .57)	.68 (.42, .87)	.00 (.00, .49)	.33 (.09, .50)	.05 (.00, .67)	.43 (.00, .66)	.59 (.46, .73)	6.42	0.893	-17.58	0.00	12
	.54 (.28, .74)	-	.42 (.26, .57)	.68 (.42, .87)	-	.33 (.09, .50)	.05 (.00, .67)	.43 (.00, .66)	.59 (.46, .73)	6.42	0.930	-19.58	0.00	13
	.54 (.28, .74)	-	.42 (.26, .57)	.68 (.49, .87)	-	.33 (.09, .50)	-	.43 (.00, .66)	.59 (.47, .73)	6.42	0.960	-21.58	0.00	14
	.54 (.28, .74)	**-**	**.42 (.26, .57)**	**.68 (.49, .87)**	**-**	**.33 (.09, .50)**	**-**	**-**	**.59 (.47, .73)**	**10.36**	**0.797**	**-19.64**	**0.02**	**15**
Older cohort														
	.61 (.48, .84)	.00 (.00, .37)	.26 (.17, .28)	.76 (.60, .84)	.00 (.00, .48)	.14 (.00, .20)	.31 (.00, .65)	.48 (.00, .67)	.51 (.45, .57)	20.89	0.034	-1.10	0.09	11
	.61 (.51, .71)	-	.23 (.17, .28)	.76 (.60, .84)	.00 (.00, .48)	.14 (.00, .20)	.31 (.00, .65)	.48 (.00, .67)	.51 (.45, .57)	20.89	0.052	-3.10	0.08	12
	.61 (.51, .71)	-	.23 (.17, .28)	.76 (.60, .84)	-	.14 (.00, .20)	.31 (.00, .65)	.48 (.00, .67)	.51 (.45, .57)	20.89	0.080	-5.10	0.07	13
	.61 (.52, .71)	**-**	**.23 (.17, .28)**	**.76 (.67, .84)**	**-**	**.14 (.00, .20)**	**.57 (.46, .68)**	**-**	**.50 (.45, .56)**	**22.92**	**0.062**	**-5.10**	**0.07**	**14**
	.61 (.52, .71)	-	.23 (.18, .28)	.76 (.68, .845	-	.13 (.00, .20)	-	.57 (.45, .68)	.51 (.46, .57)	21.35	0.090	-6.65	0.06	14
	.61 (.52, .71)	-	.23 (.18, .28)	.76 (.68, .845	-	-	-	.57 (.45, .68)	.51 (.46, .57)	24.2	0.060	-5.79	0.06	15

PA = Proactive aggression, AIC = Akaike's information criteria, RMSEA = Root-mean-square error of approximation. A = Cross-trait genetic variance component, C = Cross-trait shared environmental variance component, E = Cross-trait non-shared environmental variance component. a = Subtype-specific genetic component, c = Subtype-specific shared environment component, e = Subtype-specific non-shared environment component **NB** Bold text denotes best fitting models.

6.4.2.3 Trivariate longitudinal modeling of aggression subtypes

6.4.2.3.1 Longitudinal model of reactive aggression with underlying common form aggression as covariate

A series of Cholesky decomposition models were undertaken which assessed each aggression subtype across measurement occasions with the inclusion of UCF as a covariate control. This reflects an attempt to account for the fundamental effects of an underlying common form of aggression, which can theoretically confound discrete etiological features of the subtypes. The best fitting models associated with these analyses are presented in Figure 6.6, with associated parameter estimates can be found in Table 6.4, p. 270.

These figures show the models for both the younger and older cohort data superimposed. Results reveal that, for both RA and PA in both age cohorts, genes are the single greatest contributor to variance underpinnning continuity in both subtypes. The degree to which these pathways represent subtype specific genetic influences varies across subtypes and age groups.

In regards to continuity in RA in the younger cohort, 41% of variance in time-2 RA was attributable to trait-specific genes that influence RA over and above influences attributable to UCF. Trivariate genetic effects that influenced time-1 UCF as well as the RA over time accounted for around 10.2% of variance in time-2 RA. In the older cohort, this shared genetic component represented the only genetic effects on continuity in RA. In other words, subtype-specific genes did not appear to contribute to continuity in RA the adolescent sample but instead could be said only to underpin the persistence of undifferentiated overt physical aggression. This shared component

accounted for 28% of variance in time-2 RA.

Trivariate **E** effects appear to be important in continuity for both age cohorts (4.8% and 1.4% of variance in time-2 RA respectively). However, as above, the shared nature of these influences means that they are unrelated to mechanisms that differentiate subtypes.

RA-specific **E** effects were found for the older cohort, however they explained only around 1.9% of variance in time-2 RA.

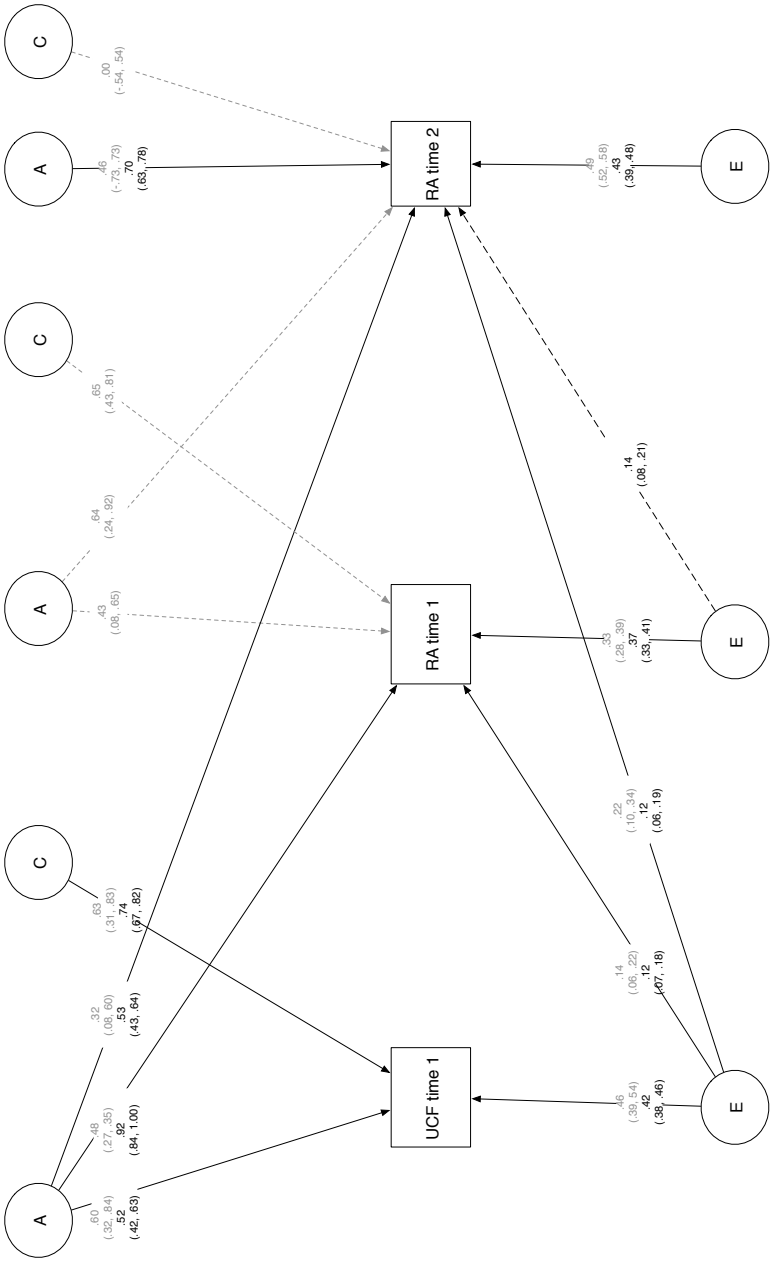


Figure 6.6 Best fitting Cholesky Decomposition Model of time-1 and time-2 reactive aggression with time-1 UCF as covariate control. A, C and E denotes latent variables representing etiological effects. UCF denotes underlying common form aggression. Grey estimates and paths derive from younger cohort data. Black estimates and dashed black paths derived from the older cohort data. Unbroken black paths represent paths common to both younger and older cohort n

6.4.2.3.2 Longitudinal model of proactive aggression with underlying common form aggression as covariate

The CDM models involving PA at both time points and UCF as covariate control revealed a very similar pattern for younger and older cohorts (see Figure 6.7 and

Table 6.4, p. 270). Unambiguously the single most important contributor to continuity in PA constitutes trait-specific genetic effects (accounting for 42% and 49% of variance in time-2 PA respectively). At the same time, a trivariate shared **C** component explained a significant 16.8% and 24% in time-2 PA in the younger and older cohort data respectively. Trivariate shared **E** influences were found to account for 5.3% and 4.4% of variance in time-2 PA in the younger and older cohorts respectively. An trait-specific **E** effect explained a very small 1.4% of variance in time-2 PA in the older cohort. No such trait-specific **E** effects were found for the younger cohort.

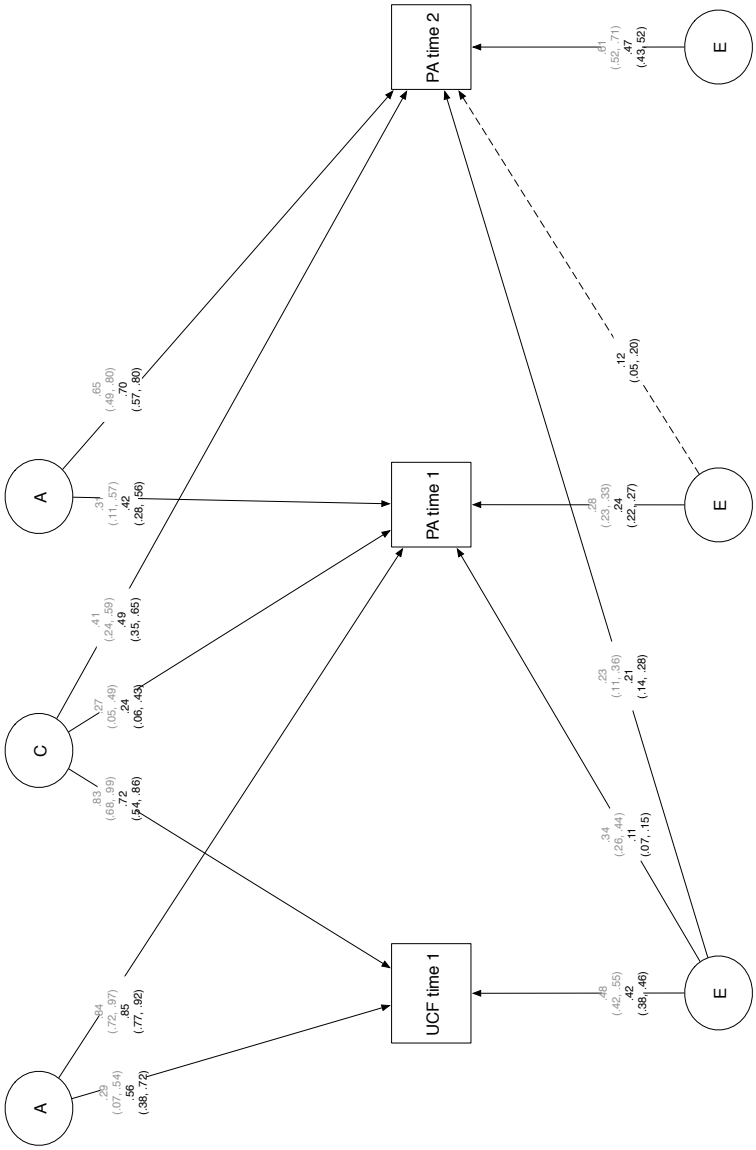


Figure 6.7 Best fitting Cholesky Decomposition Model of time-1 and time-2 proactive aggression with time-1 UCF as covariate control. A, C and E denotes latent variables representing etiological effects. UCF denotes underlying common form aggression. Grey estimates and paths derive from younger cohort data. Black estimates and dashed black paths derived from the older cohort data. Unbroken black paths represent paths common to both younger and older cohort n

6.4.2.4 Etiological factors linking reactive aggression with future proactive aggression

A Cholesky decomposition model was used to assess etiological factors affecting the putative sequential relationship between time-1 RA and time-2 PA. This analysis represents a direct examination of the sequential hypothesis. UCF was included in

both the younger and older cohort models as a concurrent time-1 covariate control.

Comparative fit indices for full and best fitting CDM models are presented in Table 6.4, p. 270. Results from the younger cohort (Figure 6.8) revealed two notable features. Firstly, a strong shared **C** component accounting for up to 75.7%, 15.2% and 15.2% in UCF, RA and PA respectively. Secondly, there was some evidence for genetic influences on this proposed pathway that do not implicate UCF related mechanisms. Although these latter effects accounted for a modest 3.2% of variance in later PA, this is interesting pattern of results. This suggests that the aggression subtypes share biogenic mechanisms that are not shared with an underlying common form of aggression. This finding is discussed in more detail in the discussion section at the end of this chapter.

While the strong trivariate **C** component found in the younger cohort data likely reflects substantial overlap measured between time-1 RA and time-1 PA—the latter of which was not included in this model—it is certainly consistent with the sequential hypothesis which posits very general operant learning principles compatible with shared environmental effects occurring in a home environment.

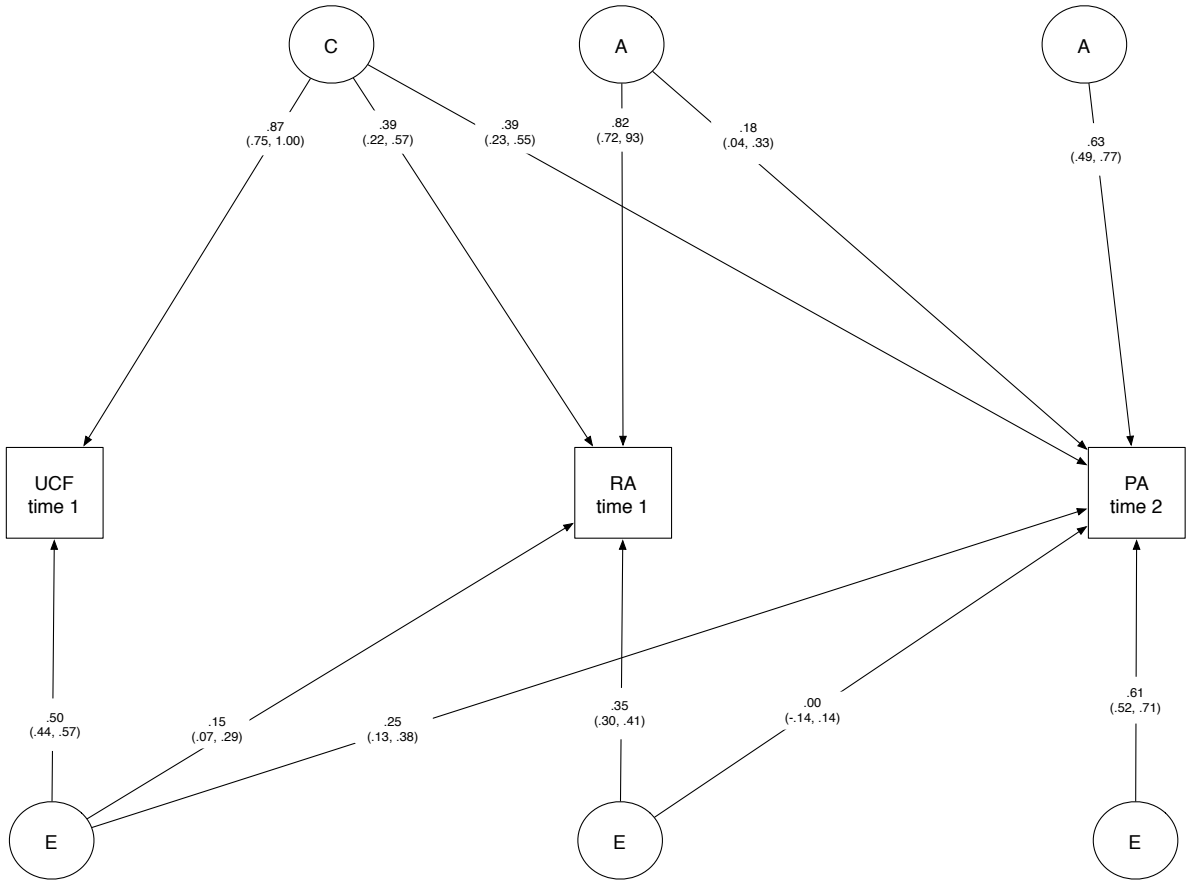


Figure 6.8 Best fitting Cholesky Decomposition Model of time-1 reactive aggression and time-2 proactive aggression with time-1 UCF as covariate control for younger cohort. A, C and E denote latent variables representing etiological effects. UCF denotes underlying common form aggression.

The older cohort CDM (Figure 6.9) revealed a somewhat different etiological picture with the total absence of **C** effects. Instead, the etiological link between time-1 RA and time-2 PA is predominantly attributable to genes, (up to 14.3% in PA) with a small contribution from non-shared environmental effects (4.5% in PA). In this best fitting model, genetic contributions to sequential transition could be partitioned into genes associated with overt physical aggression (9%) and those that were not (5.3%).

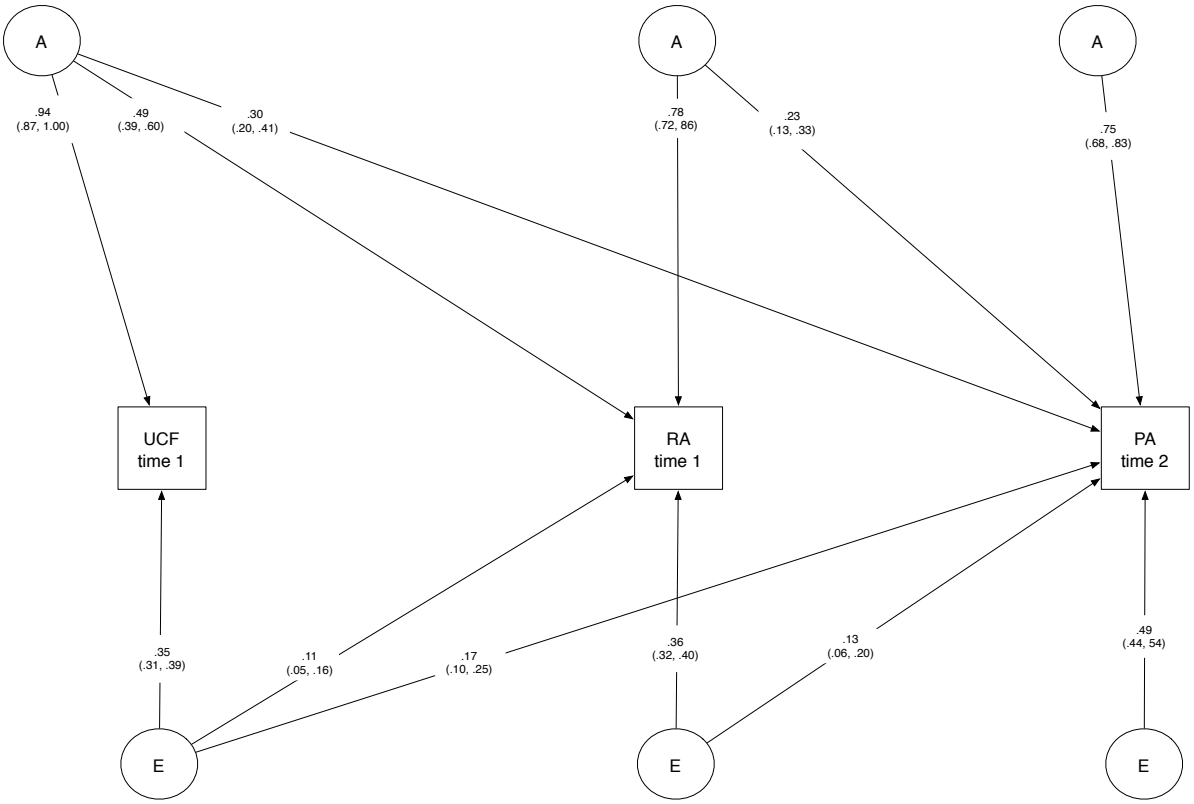


Figure 6.9 Best fitting Cholesky Decomposition Model of time-1 reactive aggression and time-2 proactive aggression with time-1 UCF as covariate control for older cohort. A and E denote latent variables representing etiological effects. UCF denotes underlying common form aggression.

6.4.3 Trivariate Modeling of Longitudinal Relationships Between Aggression Subtypes and Disruptive Behaviour Symptoms

This section presents a series of Cholesky decomposition models (CDM) that evaluate the relative independence of longitudinal pathways involved in RA, PA and DBDS with particular reference to recent proposed risk pathways (e.g., Raine et al., 2006).

The first step in the modeling process was to consecutively test each CDM against each of the guiding hypotheses. The guiding hypothesis that lead to the best fit (for each of the CDMs tested) provided a starting point for further model trimming. This

approach aimed to strike a balance between theoretically- and empirically-driven model testing.

The first model presented in this section represents a specification that is designed to examine aspects of Barratt's primacy hypothesis relating to impulsivity. The second model provides an etiological test of the hyperactivity-to-PA risk pathway proposed by Raine et al. (2006). Finally, the last model is an attempt to integrate key aspects of both the hyperactivity-to-PA and the ODD-to-PA risk pathways.

Two omissions are worth highlighting. Firstly, the intended trivariate model involving inattention symptoms could not be fit to the younger cohort data as a result of a number of anomalous cells in the relevant correlation matrix. An attempt to determine the source of deviation from expected values failed to elucidate the problem. Unfortunately, due to the low sample sizes, this problem rendered the model untenable. Without the capacity to compare the younger and older cohort data, it was decided to provide the modeled data for the adolescent cohort in the Appendices only (see Appendix H). Secondly, a decision not to include CD in any of the longitudinal analyses was made because this scale is rarely used as a predictor of other DBDs or aggression subtypes, being that it represents the most severe constellation of ASB behaviours from amongst those included. Moreover, it typically has a low prevalence in childhood populations (e.g., Caspi et al., 2008). As mentioned previously, it is was not possible to model either ODD or CD as follow-up variables as they were not included in the second measurement occasion.

See Table 6.4 below for all CDM model fitting results.

Table 6.4

Fit statistics for longitudinal trivariate Cholesky Decomposition Models

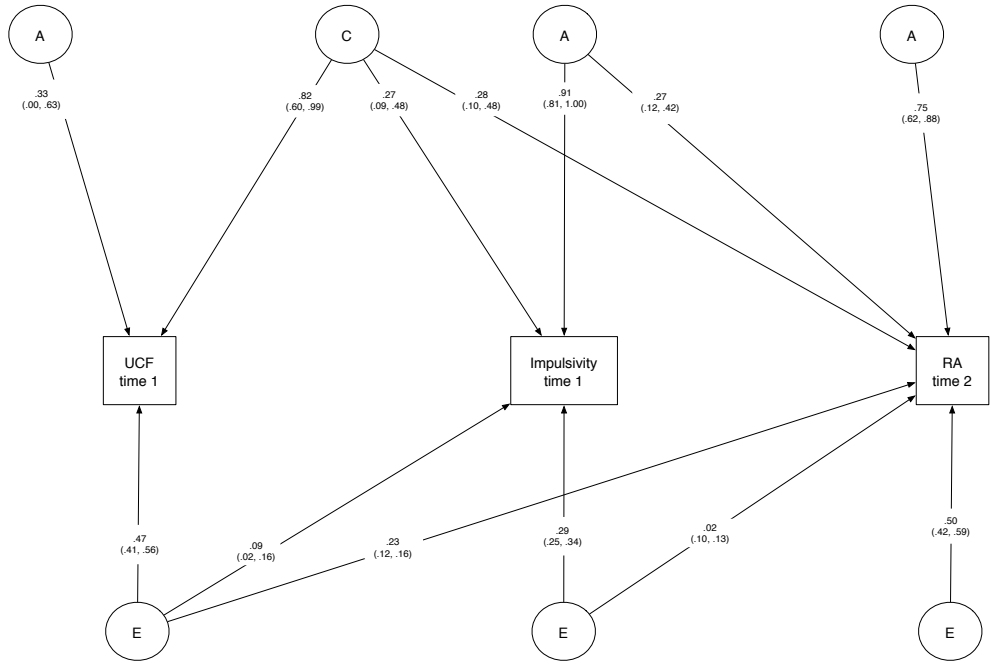
Cholesky Decomposition Models	GH	$\chi 2$	*df*	*p*	AIC	RMSEA	Model comparisons[a] Δ $\chi 2$	Δ *df*	*p*	n pairs
Younger Cohort										
*UCF_{time1} > RA_{time1} > PA_{time2}		29.65	24	0.20	-18.35	0.06				
	7	**40.71**	**30**	**0.09**	**-19.29**	**.07**	**11.06**	**6**	**>.05**	122
*UCF_{time1} > Imp_{time1} > RA_{time2}		17.85	24	0.81	-30.15	0.00				
	na	**18.12**	**29**	**0.94**	**-39.88**	**0.00**	**0.27**	**5**	**>.05**	121
*RA_{time1} > Hyp_{time1} > PA_{time2}		24.2	24	0.45	-23.79	0.06				
	7	**37.23**	**31**	**0.21**	**-24.76**	**0.07**	**13.03**	**7**	**>.05**	119
*Hyp_{time1} > RA_{time1} > PA_{time2}		10.05	24	0.99	-37.95	0.00				
	7	**20.88**	**32**	**0.93**	**-43.12**	**0.00**	**10.83**	**8**	**>.05**	119
*Hyp_{time1} > ODD_{time1} > PA_{time2}		18.61	24	0.77	-29.38	0.02				
	4	**28.45**	**33**	**0.69**	**-37.55**	**0.01**	**9.84**	**9**	**>.05**	121
Older Cohort										
*UCF_{time1} > RA_{time1} > PA_{time2}		16.77	24	0.86	-31.23	0.00				
	4	**24.47**	**30**	**0.75**	**-35.53**	**0.02**	**7.7**	**6**	**>.05**	265
*UCF_{time1} > Imp_{time1} > RA_{time2}		24.03	24	0.46	-23.97	0.03				
	5	**24.03**	**29**	**0.73**	**-33.97**	**0.01**	**0**	**5**	**>.05**	259
*RA_{time1} > Hyp_{time1} > PA_{time2}		24.17	24	0.45	-23.82	0.01				
	4	**29.37**	**30**	**0.49**	**-30.63**	**0.02**	**5.3**	**6**	**>.05**	253
*Hyp_{time1} > RA_{time1} > PA_{time2}		25.21	24	0.39	-22.8	0.02				
	4	**28.14**	**31**	**0.62**	**-33.86**	**0.00**	**3.0**	**7**	**>.05**	253
*Hyp_{time1} > ODD_{time1} > PA_{time2}		26.30	24	0.34	-21.7	0.02				
	4	**31.19**	**31**	**0.45**	**-30.81**	**0.02**	**4.89**	**7**	**>.05**	260

RA = Reactive aggression; PA = Proactive aggression; Hyp = Hyperactivity; Imp = Impulsivity; Ina = Inattention; ODD = Oppositional defiant symptoms; * denotes full Cholesky model; bf = best fitting model; Bold denotes best fitting model; GH = Guiding Hypotheses

6.4.3.0.1 Evaluating the longitudinal relationship between impulsivity and future reactive aggression while controlling for underlying common form aggression

The prediction that, after controlling for the presence of UCF at time-1, impulsivity would retain strong biogenic links with future RA received only modest support. Best fitting models (see Figure 6.10) revealed three longitudinal sources of variance (**A**, **C**, and **E**) impacting on RA in the younger cohort, and two for the older cohort (**A** and **E**). For both samples, only genes were exclusively associated with any substantial shared variance involving time-1impulsivity and time-2 RA. However, this longitudinal relationship accounted for a modest 7.3% and 11.6% of total variance in RA for younger and older cohorts respectively. While **E** effects showed some longitudinal effects over and above that accounted for by UCF for both cohorts, this partitioned component contributed only negligibly to total variance in time-2 RA (0.4% and 1.4% for younger and older cohorts respectively). A **C** component was identified in the younger cohort that contributed to shared variance involving all three variables—accounting for 7.8% of time-2 RA variance. In both models the single most influential source of variance in time-2 RA was attributable to trait specific **A** effects (56.2% and 40.9%).

(a)



(b)

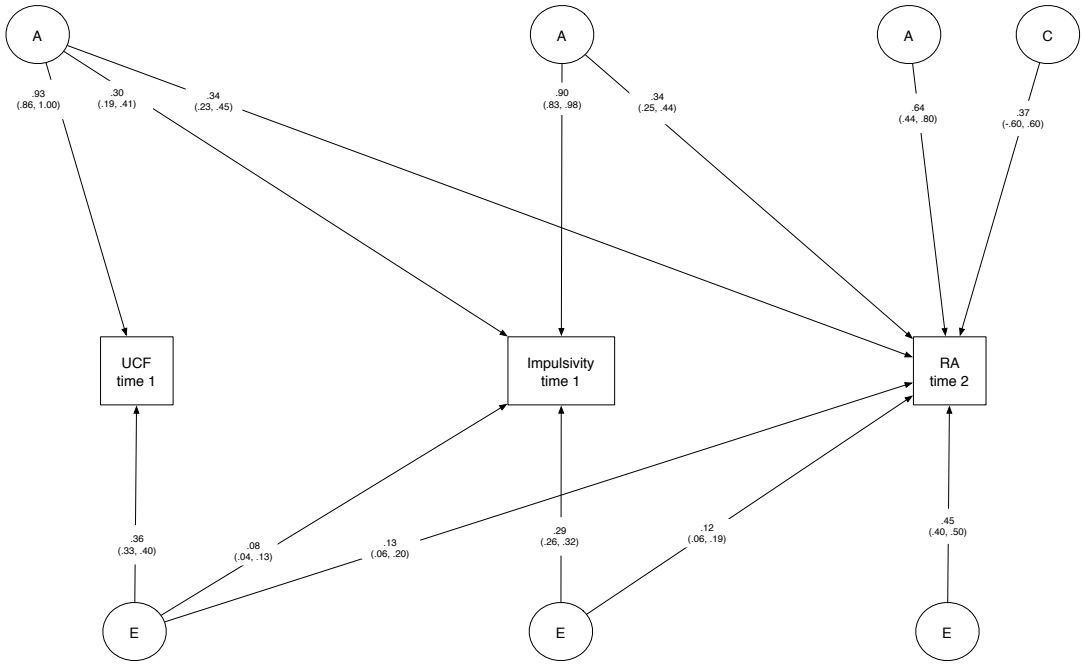


Figure 6.10 Best fitting Cholesky Decomposition Models of impulsivity and future reactive aggression with concurrent underlying common form aggression as covariate control. (a) denotes younger cohort model, (b) denotes older cohort model.

Implications that these results have for the primacy hypothesis are discussed in the

summary section at the end of this chapter.

6.4.3.0.2 Evaluating the longitudinal relationship between hyperactivity, reactive aggression and future proactive aggression

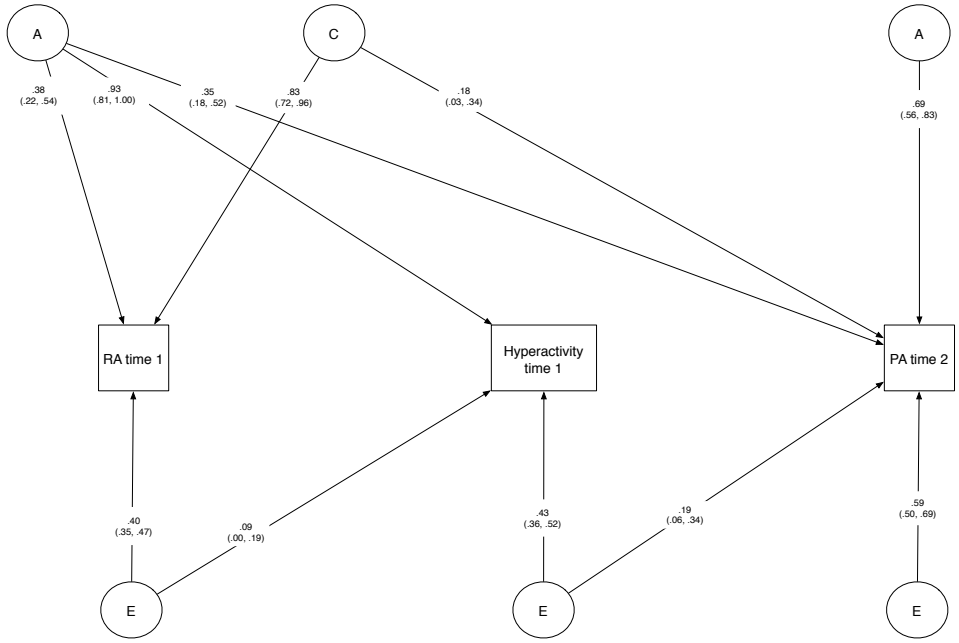
The possibility of an independent hyperactivity-to-PA risk pathway was recently raised by Raine et al. (2006) who demonstrated a unique prognostic association between early hyperactivity and later PA. The present aim effectively represents a test of this pathway using the current trivariate genetic models. The basic specification of this trivariate model involves RA placed in the covariate control position (i.e., first position) of the Cholesky model, with hyperactivity placed in the second position and PA in the third (see Figure 6.11). This specification estimates the longitudinal relationship between hyperactivity and future PA over and above concurrent RA. A second model was included which placed hyperactivity in the covariate control position (see Figure 6.12) was undertaken to assess the complementary question of whether the sequential association between RA and PA accounts for unique etiological variance over and above hyperactivity. Based on earlier tests of the sequential hypothesis reported in section 6.4 (p. 265), this latter specification was constrained, throughout the model fitting process, to maintain a **C** pathway between RA and PA that was independent of hyperactivity. Likewise, the basic specification was constrained such that a bivariate **C** path linking RA and PA (but not hyperactivity) was maintained in each of the subsequent nested models tested for best fit. It is noted that because the model trimming process related to these two models was a priori constrained on the basis of extant theoretical specification, there was no need to utilise the guiding hypotheses.

6.4.3.0.2.1 The basic specification

For the younger cohort, current results indicate the only non-RA related source of variance influencing the etiological pathway between hyperactivity and PA associated involved **E** effects which accounted for only a modest 3.6% of variance in time-2 PA (see Figure 6.11). Interestingly, although the model was constrained to have a bivariate path from RA to future PA, this specification led to the most parsimonious model—even when compared to best fitting nested alternatives with no such constraint (not reported here). However, as in previous sequential specifications, the impact of the this sequential path on PA was small (accounting for 3.2% of variance). A trivariate **A** component accounted for 14.4% in RA, 86% in hyperactivity and 12.2% in PA. Almost half the variance (47%) in PA was attributable to time-specific genetic effects.

Results from the older cohort suggested two unique pathways between hyperactivity and PA associated with **A** and **E** effects which respectively accounted for 8.4% and 1.9% of variance in time-2 PA. The childhood cohort model data revealed a common genetic factor underpinning shared variance in all three variables (RA, PA and hyperactivity) and accounting for a similar proportion of variance in each of the behaviour scales included. This shared genetic component contributed around 29.1% of variance in time-2 PA. A similar shared genetic component was found in the older cohort accounting for 11.5% variance in PA. The single greatest source of variance in PA, in both younger and older cohort models, was attributable to the trait-specific **A** component (33.5% and 51.8% respectively).

(a)



(b)

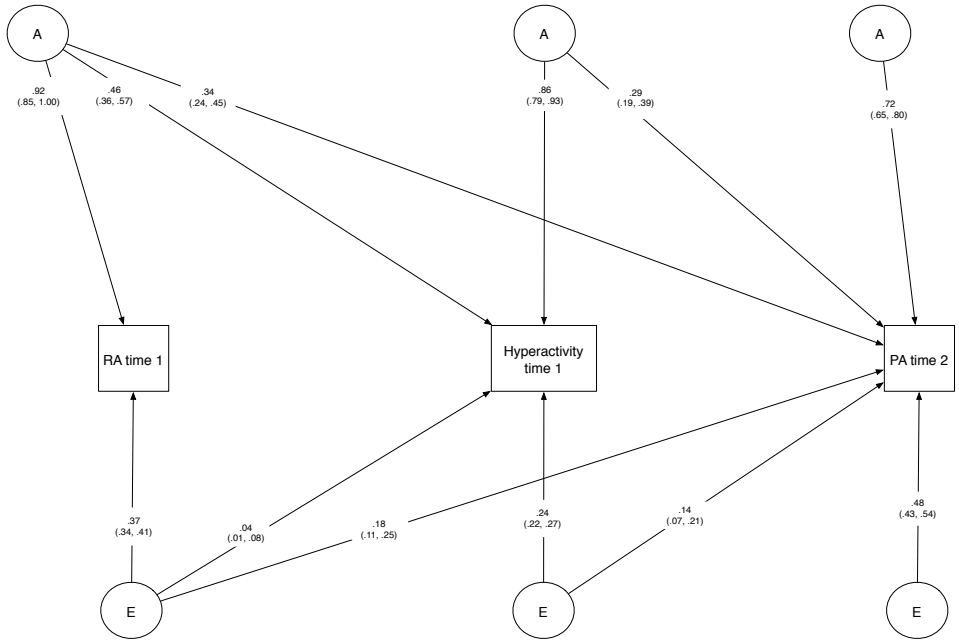


Figure 6.11 Best fitting Cholesky Decomposition Model of hyperactivity at time 1 and proactive aggression at time 2 with concurrent reactive aggression as covariate control. (a) denotes the younger cohort model and (b) denotes the older cohort model.

6.4.3.0.2.2 The sequential specification

For the younger cohort, current results indicate two key pathways to PA. The first pathway supports the sequential hypothesis, indicating a cross-trait (cross-time point) shared environmental pathway between RA and PA that is relatively independent of hyperactivity (see Figure 6.12). However, it is noted that this pathway accounted for only 2% of variance in PA. The second pathway represents a trivariate additive genetic component which explained around 9% of variance in PA. In other words, this second genetic pathway represented a set of genetic influences that simultaneously impacts on hyperactivity and RA at time one and PA at time two. The majority of variance in PA was attributable to trait-specific (time-specific) genes (45%) and non-shared environment (42.2%). A small degree of variance (2%) in the longitudinal relationship between RA and PA was accounted for by non-shared environmental effects.

No **C** effects were present in the older cohort model. Instead genes accounted for the majority of longitudinal variance. Specifically a shared bivariate genetic pathway between RA and PA accounted for around 6.2% of variance in PA, while a shared trivariate genetic pathway accounted for 10.2% of variance in this latter subtype. Like the younger cohort model the greatest single source of variance in PA were trait-specific genetic influences (56.2%). Trait-specific non-shared environmental influences were associated with 25% of this same variance.

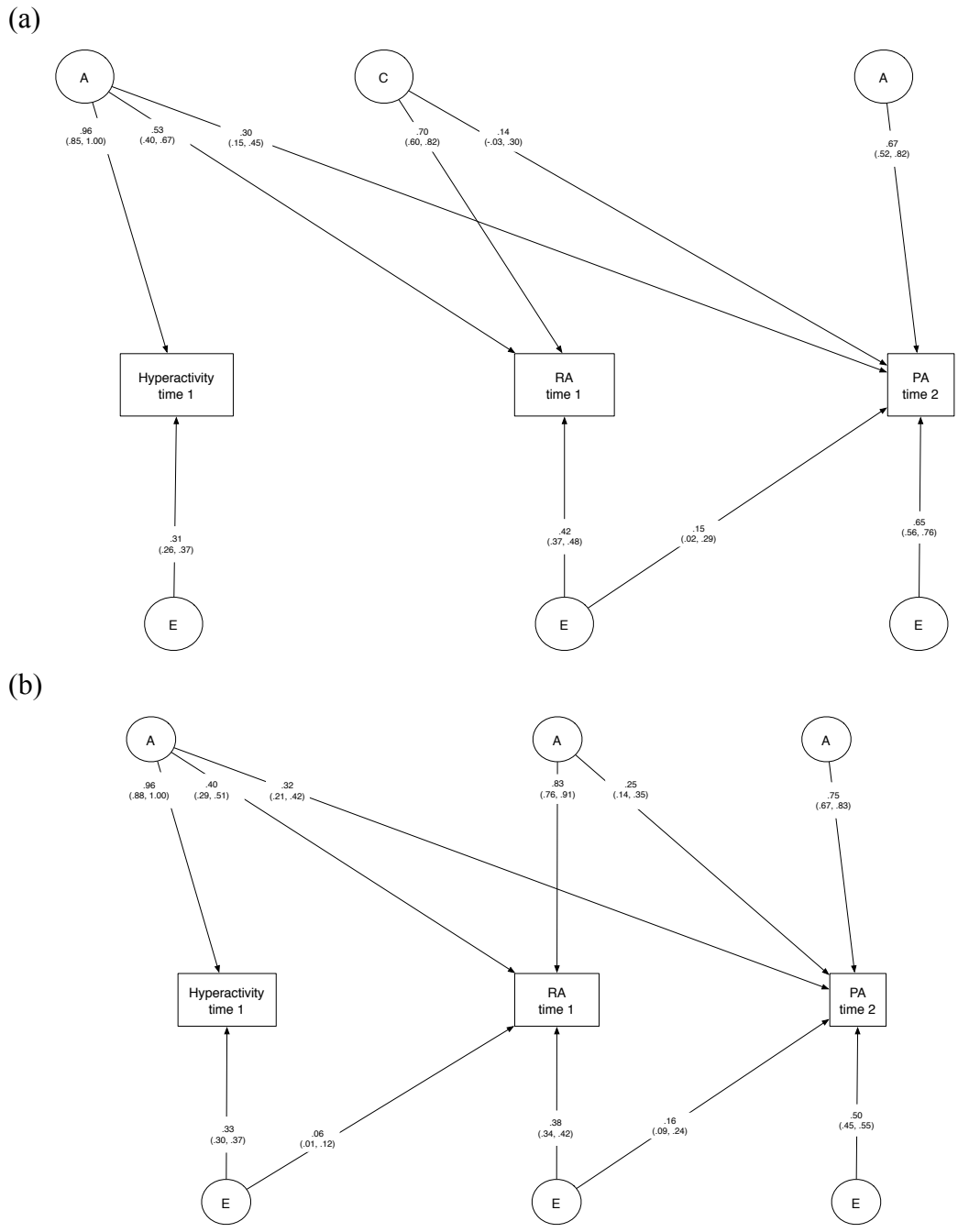


Figure 6.12 Best fitting Cholesky Decomposition Model of time-1 reactive aggression and time-2 proactive aggression with concurrent time-1 hyperactivity as covariate control. (a) denotes the younger cohort model and (b) denotes the older cohort model.

6.4.3.0.3 Evaluating the longitudinal relationship between oppositional behaviour and future proactive aggression with concurrent hyperactivity as covariate

The ODD-to-PA Cholesky model with hyperactivity as control revealed a strong trivariate **A** component for both age cohorts (see Figure 6.13). Data from the younger cohort suggested this shared genetic influence accounted for around 9% in time-2 PA for both age groups. For the older cohort, this trivariate component was accompanied by a bivariate **A** component involving ODD and PA and accounted for an equivalent 9.6% of variance in the latter variable. Bivariate **E** effects were also required in explaining around 10% longitudinal variance shared between ODD and PA in the childhood cohort. The same path accounted for a small 2% of variance in adolescent PA measured at time-2. As with a majority of the CDM models presented in this section, the single most important source of variance in time-2 PA was trait specific A effects accounting for 40.9% and 32.5% of variance in younger and older cohorts respectively.

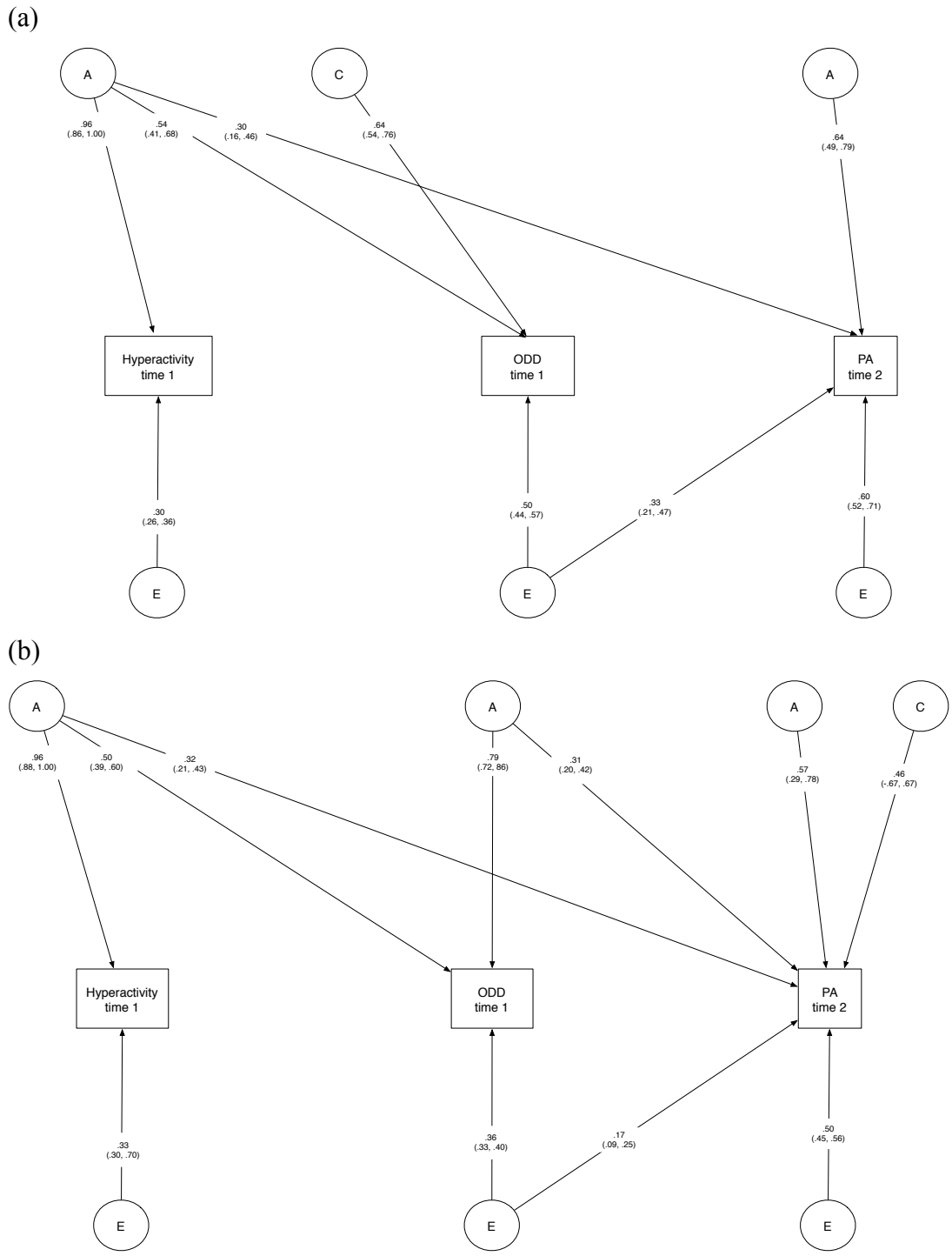


Figure 6.13 Best fitting Cholesky Decomposition Model of time-1 oppositional defiant behaviours and time-2 proactive aggression with concurrent time-1 hyperactivity as covariate control. (a) denotes the younger cohort model and (b) denotes the older cohort model.

6.5 Summary of Findings from Analyses Involving Repeated-measures

The aim of the current research was to examine longitudinal relationships between aggression subtypes and core DBD symptomatology. Discussion of results is divided into sections associated with each of the main analytic methods utilised.

6.5.1 Cross-lagged Panel Models of Reactive and Proactive Aggression

Unsurprisingly, the cross-lagged panel models revealed significant phenotypic overlap, both cross-sectionally and longitudinally, for RA and PA in the younger and older cohorts. From a purely structural perspective, it is interesting to note that, in childhood, RA appears to be a moderately stronger predictor of future aggression than PA in both age groups.

These data are broadly consistent with the speculative suggestion that there exits a sequential pathway from RA to PA (Vitaro & Brendgen, 2005). Polman et al. (2007) have suggested that sequential models predict increasing correlations between RA and PA with age. The cross-lagged models continue to support data reported in earlier chapters which contradict this prediction. At the same time, zero-order correlations provided no evidence that PA possesses greater generalised prognostic value in adolescence compared to childhood as speculated earlier in the literature review.

It is clear that the intra-class longitudinal correlations for both RA and PA are equally strong across both age cohorts. While this does not pose any direct challenge

to the sequential model, the overall results suggest that sequential transition is but one of multiple pathways in the development of aggression subtypes.

Considerable caution must be taken in interpreting the best fitting cross-lagged panel models as there are strong indications that a lack statistical power is affecting results. Nonetheless, despite the ambiguity inherent in the current cross-lagged panel model results, the data are relatively clear in establishing the need to consider multivariate pathway models, in addition to single phenotype pathways models, when addressing longitudinal relations between RA and PA. Before covariate models are considered, however, single phenotype bivariate genetic models of RA and PA are presented.

6.5.2 Bivariate Repeated-measures Modeling

Findings reported by Brendgen et al. (2006) suggested the possibility that **E** effects contribute substantially to the differential shaping aggression subtypes. The degree to which **E** effects operate on continuity of RA and PA was therefore of particular interest. Current longitudinal bivariate results indicate only modest **E** effects on continuity with effects ranging between around 13.7% and 6.2% for RA in younger and older cohort respectively, and 17.6% and 5.3% for PA in younger and older cohort respectively.

However, two qualifications pertaining to the current data on **E** effects are necessary. Firstly, the bivariate longitudinal models did not include any control for UCF and so are silent in regards to the differential impact of **E** effects on RA and PA. Discussion concerning this somewhat more complex analysis is reserved for the relevant

trivariate longitudinal models described in the following sections. Secondly, current bivariate longitudinal results were at odds with Brendgen et al.'s speculation that **E** effects on aggression subtypes are (at least partly) indexing peer influence. As noted previously, **E** effects are generally associated with experiences that accrue outside of the home environment (Moffitt, 2005b), and peer effects certainly represent an obvious candidate influence in this category Brendgen et al. (2006). It is thus interesting to observe that, when compared to the younger cohort estimates, the adolescent cohort longitudinal **E** effects were of substantially less magnitude. Adolescence is generally viewed as a time in development when peer effects are increased rather than attenuated—relative to childhood influences (Coie & Dodge, 1997), and certainly this time of development is associated with increased autonomy and activity outside the home environment. Moreover, this pattern of lower magnitude non-shared environmental effects in older versus younger cohort data was also evident in the substantial time-specific **E** effects found on both RA and PA. In other words, at first blush, the current data on **E** effects appears at odds with some basic expectations of developmental dynamics.

The most likely explanation for this apparently anomalous result is the unexpected differential in magnitude of **E** effects reflects differing proportions of error present in the younger versus older cohort modeled variance. Given the substantially lower sample size of the younger cohort models, one would expect these models to be far more sensitive to error variance than the older cohort models that involved around three times the number of contributing participants. Given that error variance is indexed by the **E** term (Levy & Hay, 2001; Moffitt, 2005b; Rutter, 2007), it is in fact unsurprising that the younger cohort showed proportionally higher **E** estimates

relative the older cohort. Indeed, this observation is in line with earlier cautions concerning over-interpretation of **E** effects evidenced in Brendgen's models based on a sample that was around a third of the size of the current younger cohort sample.

Nonetheless, despite possible differential impact of error variance on younger versus older cohort models, the data unambiguously reveal that **E** effects are more prominent on discontinuity than they are on continuity in both RA and PA in both age groups. To the extent that **E** effects (partially) represent peer effects, the data indicate pervasive **E** effects on discontinuity. This suggests that stage-like changes in social affiliations and peer networks are more important in understanding the role of peer effects on RA and PA, than are continuities in peer relations. This is an interesting find because, on face value at least, it is contrary to intuitive notions of (negative) peer effects operating primarily through stable and continuous antisocial affiliations (e.g., Loeber & Hay, 1997).

Neither the bivariate longitudinal model of RA, or PA, retained variance components associated with longitudinal **C** effects. In fact, the only **C** component retained in any of these models represented RA-specific **C** effects at Wave One for younger cohort data. This component accounted for almost half the variance in RA at this first measurement occasion. Given the somewhat volatile nature of **C** effects, interpretations concerning their impact on current data are reserved for later discussions that include consideration of the results of the present trivariate modeling.

Genetic effects unambiguously evidenced the greatest impact on continuity overall

on the bivariate models, with around 45% of variance in RA shared across time points for both age cohorts, (compared with 13.7% and 6.2% attributable to **E** effects in the younger and older cohorts respectively).

Somewhat surprisingly, relative to the childhood RA bivariate model, the genetic effects associated with PA in the younger cohort were moderately less influential regarding their effects on continuity (accounting for just under 30% of variance in PA at either time point). However, both RA and PA showed remarkably similar patterns of results for the older cohort data.

The bivariate results provided mixed support for the smorgasbord hypothesis. The overall influence of genes on both RA and PA was clearly stronger in the older cohort compared to the younger age group. For example, averaging results across measurement occasions, the overall proportion of genetic (versus environmental) influence is 45% for the younger cohort and 80% for the older cohort for RA, and 37% (younger cohort) and 82% (older cohort) respectively for PA. Taken alone, this suggests tentative support for the smorgasbord model. However, the picture within each cohort shows somewhat of a different pattern. Specifically, the overall magnitude of genetic influences declined across measurement occasions for all bivariate models—if only moderately in some cases. Inversely, the effects of non-shared environmental effects tended to increase.

Remembering that the behaviour genetic model provides estimates of relative influence rather than absolute magnitude of etiological sources, the above results might tentatively be interpreted as reflecting the more dynamic influence of **E** effects

relative to the more stable influence of genes. Unfortunately, the smorgasbord hypothesis is only broadly specified and it is not clear what pattern of results is predicted in the absence of (declining) **C** effects. Certainly **E** effects are often considered to be more sensitive than **C** effects to factors operating outside the family environment (Rutter, 2006). As such, it might reasonably be expected that **E** effects will increase over time as the influence of family environment (i.e., **C** effects) decreases. This effectively pits **E** effects against **A** effects as sources of increasing variance within the smorgasbord model. The present data suggests the possibility that **E** effects typically prevail in the short-term while **A** effects prevail in the longer-term. This tentative comparison is of course confounded by the use of a dual cohort longitudinal model. Moreover, such a pattern is necessarily artifactual in the sense that both patterns cannot be ultimately be true. However, it is plausible that **E** effects are susceptible to a more dynamic (or oscillating) pattern of relative influence compared to that of genetic effects. For example, albeit a highly speculative one, **E** effects might consistently strengthen during periods of relative stability in peer group affiliation but, at times of transition between peer groups, there may be a correction downwards relative to the genotype which theoretically consistently strengthens in its role as the driving force behind the selection of social (and other) environments (Miles and Carey, 1997). This is not to propose that transitioning between peer groups represents a discrete or directly measurable developmental change. The point is that the changing influence of **E** effects may occur on a different time scale and/or follow a different (perhaps more volatile) pattern, when compared to putative gradual accumulating effects of genotype.

Certainly, results suggest that to the extent **E** effects are associated with peer

influence it appears this influence occurs earlier than for RA. However, the current bivariate models provided no means to control for the substantial phenotypic overlap between aggression subtypes. A series of trivariate Cholesky models were used to provide a means to include a covariate control in subsequent longitudinal analyses.

6.5.3 Trivariate Repeated-measures Modeling

6.5.3.1 Trivariate repeated-measures modeling of reactive and proactive aggression controlling for underlying common form aggression

The trivariate Cholesky Decomposition model (CDM) afforded the additional inclusion of a UCF variable in basic longitudinal models of aggression subtypes. This provided a means to assess subtype-specific versus common longitudinal pathways associated with RA and PA.

Unsurprisingly, these latter trivariate CDM models (i.e., the UCF>RA>RA and UCF>PA>PA models) generally concurred with the relevant bivariate longitudinal specifications presented above as well as the cross-sectional genetic modeling reported in earlier chapters. Specifically, best fitting CDMs confirmed that genetic mechanisms constituted the single most pervasive class of etiological influence on continuity of aggression subtypes. However, there were some notable exceptions and these are discussed in the following section.

Of the guiding hypotheses, one to three (all of which articulated restricted effects of genes on aggression subtypes) could be ruled out for all models. Failure in attempts to significantly constrain genetic effects underscores the complex and pervasive

influence of genes on aggression. In particular, genetic effects were found to consistently effect both continuity and discontinuity in the longitudinal data.

Unsurprisingly, conclusions regarding **C** effects also revealed complex interrelationships between variables. Nonetheless, all models complied with at least one hypothesis that specified the restriction of **C** effects (i.e., hypotheses four to seven). Broadly speaking, this indicates a generally less pervasive influence of **C** effects on aggression subtypes compared to **A** effects.

As in the bivariate longitudinal models, **E** effects on continuity in the UCF>RA>RA and UCF>PA>PA CDM models were generally modest to small, while their influence on discontinuity appeared to be somewhat stronger in comparison. This might be interpreted as inconsistent with Brendgen et al.'s (2006) speculative hypothesis regarding the primary role of **E** effects in differentially shaping aggression subtypes—although see the following section for a more thorough examination of alternate conceptions of continuity versus discontinuity of effects over time.

An additional word of caution regarding the interpretation of Cholesky models is appropriate here. The frequent occurrence in the literature of interpreting bivariated or trivariated pathways (that link an individual variance component to two or more variables) as indexing cross-trait variance is somewhat misleading (Loehlin, 1996). Specifically, these linking pathways do not entirely disentangle trait-specific versus cross-trait variance. Instead, the most "upstream" path (indicated by the most vertically aligned path in the current models presented in this text) actually partially

indexes both trait-specific variance associated with the trait it is directly connected to, in addition to cross-trait variance.

This ambiguity emphasises the value of corroborative evidence from divergent classes of models. Notably, the findings from bivariate longitudinal models of RA and PA go a considerable way to help clarify some of the structural ambiguities inherent in CDM models—and vice versa. So for example, the finding that **E** effects operate more strongly on discontinuity than continuity in bivariate models of RA and PA, supports the general finding that the forward projecting **E** pathways in the CDM are generally associated with weaker estimates than their vertically projecting counterparts. It is these vertically projecting paths that confound trait-specific variance associated (with the trait this latter path is directly connected to) and cross-trait variance. While some argue this is a weakness of the CDM (on the grounds that the above structural feature is often misunderstood leading to misinterpretations of data (Loehlin, 1996)), another way of thinking about this model is that the forward projecting pathways provide an unambiguous and *conservative* (i.e., underestimated) coefficient of longitudinal effects.

6.5.3.2 Preliminary theoretical implications relating to trivariate repeated-measures modeling of aggression subtypes

In relation to the proposition that **E** effects are the primary source of differentiation between RA and PA (Brendgen et al., 2006), the CDM models of RA and PA measured across time points and including time-1 UCF as a covariate control, suggest that subtype-specific non-shared environmental effects contributed only to change (and not continuity) for both RA and PA in the younger cohort. That is, their

effects were invariably time-point specific and not longitudinal.

Attempts to interpret discontinuous versus continuous effects raise the empirical question of how best to understand Dodge and colleague's broader etiological formulation of RA and PA within the relatively complex structural specification of the proposed hybrid CDM design.

While Dodge's (1991) framework is frequently described as a psychosocial model, it encompasses multiple levels of explanation. Specifically, while parenting practices are posited as primary causal agents responsible for the *emergence* or acquisition of RA and PA in childhood, the broader model suggests that once RA and PA become a part of the child's typical response set, these behaviours are in fact maintained by subtype-specific socio-cognitive biases.

Specifically, although requiring social input to operate, Dodge and colleagues ultimately attribute primary maintaining mechanisms to cognitive distortions that represent (1) *hostile attribution bias* in the case of RA, and (2) *positive outcome expectancies* for aggression for PA. Cognitive factors are often conceptualised as child factors (Fontaine, 2008; Giancola et al., 1996; e.g., Guerra & Slaby, 1989; Séguin, Pihl, Harden, Tremblay & Boulerice, 1995) and in light of the pervasive influence of genes on general cognitive functioning (Dickens & Flynn, 2001; Haworth et al., 2009), as well as the robust empirical link between cognitive functioning and aggression (Giancola & Zeichner, 1994), it is reasonable to postulate that Dodge's cognitive distortions are at least partly under the influence of genetic factors.

Moreover, if the acquisition of cognitive distortions posited by Dodge and colleagues is influenced by genotype to a non-trivial degree, then any act of child aggression that (1) is influenced by biased socio-cognitive processing and, (2) subsequently elicits behaviour change in others (within the child's social environment) compatible with that aggression (e.g., increased hostility in the case of RA, or social submission in the case of PA), will produce a gene-environment correlation indexed by the genetic component of the behaviour genetic twin model.

In short, this somewhat post-hoc re-formulation of Dodge's model represents one way to understand the presence of subtype-specific genetic effects on continuity in RA and PA. Of course, empirical validation of this elaborated hypothesis would, at least, require targeted measures of socio-cognitive processing ascertained within a genetically sensitive design.

Interestingly, defining the likely role of **C** effects in this post-hoc specification prompts further examination of potential ambiguities in Dodge and colleagues' formulation of RA and PA. For example, it is reasonable to define the concept of *emergence* as representing etiologically significant change (i.e., an increase) in a child's RA or PA behaviour. Depending on the actual time frames involved in this acquisition, *emergence* might be indexed to as either discontinuous change (i.e., time-specific effects), or continuous change (i.e., continued reinforcement or strengthening of the aggression subtype-relevant behaviour). Although speculative, from this perspective, the longitudinal genetic model may act as a proxy measure of the timeframe associated with the effects of more voltiale putative etiological

mechanisms such as those capture in **C** effects.

So for example, if it is assumed that **C** effects (at least partially) index maladaptive parenting practices (Brendgen et al., 2006), then the current longitudinal models suggest the possibility that these influences operate on different timescales (over the 9-month testing period) for RA and PA. Certainly, it might be expected that the basic social learning processes proposed to underpin the emergence of PA (Dodge, 1991; Vitaro & Brendgen, 2005) are associated with *continuous change* (i.e., incremental strengthening of behaviour via reinforcement mechanisms) as described immediately above. That is, Dodge and colleagues' formulation of PA describes maladaptive positive reinforcement of aggression (in the home) which ultimately elicits PA. It is plausible that these reinforcement processes persist within the home environment, and possibly combine synergistically with a genetic vulnerability for PA, to strengthen PA behaviour over time. These observations highlight the profound challenge of disentangling complex synergistic risk mechanisms. Nonetheless, from this perspective, reinforcement of aggression is both an eliciting (i.e., causal) agent, as well as a "maintaining" mechanism. In other words, it can be assumed relevant reinforcement mechanisms continue to elicit/reinforce even after PA becomes a characteristic response.

Certainly, substantial **C** effects were found on continuity in PA in both younger and older cohort trivariate longitudinal models. To the degree the sensitivity of the longitudinal model is effected by the length of testing period, this suggests that the ultimate effects of such mechanisms are relatively continuous over a 9-month period. This (post-hoc) formulation might go some way to help resolve the paradoxical

finding of no **C** effects in current cross-sectional models of PA versus substantial **C** effects in the longitudinal models. Specifically, if **C** effects are incremental over time, then they are more likely to be picked up in longitudinal versus cross-sectional data (Rutter, 2006). However, this speculation does not explain the absence of **C** effects in the bivariate longitudinal models of PA—this inconsistency is discussed shortly below.

On the other hand, a somewhat different pattern of **C** effects was found for RA. While substantial **C** effects were found in cross-sectional models of RA, both bivariate and trivariate longitudinal models were clear in suggesting these **C** effects do not operate on continuity of RA in either age cohort. In fact, longitudinal models indicate RA-specific **C** effects only operated on discontinuity at the first measurement occasion in the younger age group. To the extent this reflects psychosocial mechanisms responsible for the emergence of RA in childhood (as per the post-hoc formulation presented above), these results indicate such mechanisms have relatively time-specific or at least sporadic effects—i.e., effects that are not continuous over a 9-month period.

It should also be remembered that where the assumption of homoscedacity amongst variables is adequately met (as in the current project), all etiological effects identified by the behaviour genetic twin model are bidirectional in the sense that resultant estimates derived from such models do not distinguish between elicititive versus inhibitory effects on the trait in question. As such, while there is much attention given to the elicititive effects of psychosocial (and child) factors, the current results equally suggest the patterns in the data might also apply in the inhibitive direction. It

is thus possible to interpret the above data as indicating that psychosocial interventions (whether formal or informal) might have a somewhat more immediate impact on RA, while any amelioration or extinction of PA behaviour is likely to represent a more gradual or incremental process. While bidirectionality represents a potential limitation in the interpretability of modeled data, this predicted pattern of inhibitory effects is in fact roughly in line with current understanding in regards to the amenability of RA versus PA to intervention—i.e., with the RA being considered as more amenable to treatment than PA-related behaviours (Connor et al., 2004; Dadds & Rhodes, 2008; Hawes & Dadds, 2005; Merk et al., 2005; Raine et al., 2006; Vitiello & Stoff, 1997).

Finally, it is noted that the trivariate **C** component in the younger and older PA models (which by definition covaried with UCF), challenges the notion that this set of shared **C** effects reflect environmental mechanisms that operate to make PA different to RA. While this latter observation represents a significant deviation from Dodge's original etiological specification, it is not necessarily inconsistent with a recent re-formulation of Dodge's developmental model of RA and PA proposed by Vitaro and Brendgen (2005). Below, interpretations of the current longitudinal data linking RA with later PA are considered in the context of the sequential hypothesis proposed by Vitaro and Brendgen (2005).

6.5.3.3 Developmental relationships between reactive aggression and future proactive aggression: Assessing the sequential hypothesis

Utilisation of the longitudinal Cholesky model allowed for a clear etiological specification of the sequential hypothesis. Specifically, the relevant CDM specified

RA at time one and PA at time two and included UCF as a covariate control. This latter model provided considerable support for the sequential hypothesis when applied to data from the younger cohort. That is, the main source of shared variance longitudinally associated with this pathway was attributable to **C** effects as predicted by the sequential model. These latter effects were also shared with the UCF variable suggesting the mechanisms involved are not subtype specific. Indeed, the simple operant conditioning mechanisms related to the sequential pathway, as proposed by Vitaro and Brendgen (2005), would be expected to operate indiscriminately on the full spectrum of overt physical aggressive behaviours. This represents an interesting reconfiguration of Dodge's original theory of RA and PA and potentially helps explain the cross-trait C effects between PA and UCF found in the UCF>PA>PA models described in the previous section. This reconfiguration is discussed in more detail in the final conclusions chapter.

Contrary to the younger cohort, there was an absence of **C** effects in the sequential CDM modeling of data from adolescent group. Rather, the longitudinal relationship between RA and PA appeared to be predominantly underpinned by genes specific to RA and PA, as well as genes shared by all three variables, i.e., RA, PA and UCF. **E** effects contributed to this pathway but these were of relatively smaller magnitude compared with the influence of genes.

Taken together, the current results tentatively suggest the putative sequential pathway does not operate continuously across the entire developmental spectrum but instead operates in childhood only. Indeed, it seems that by adolescence the mechanisms proposed by the authors are unlikely to be operating to the same extent.

Instead, as predicted by the smorgasbord hypothesis, genotype appears to become a predominant determinant of any transition from RA to PA.

It is important to state that this last observation is not an appeal to determinism. As noted earlier, quantitative genetic modeling is unable to disentangle gene-environment correlations and interactions—both of which are typically pervasive in the areas of ASB and aggression (Rutter, 2006). Gene-environment correlations are of particular relevance to the smorgasbord hypothesis. What this latter theory suggests is that the impact genotype has on environments selected increases with age at the *population level of analysis*. This says nothing about choices made by individuals that lead to deviations from this overall pattern of effects. Due to the limitations of the quantitative genetic model, any such correlational effects (which invariably implicate specific environmental conditions) are captured as an increase in the additive genetic (**A**) term.

6.5.3.4 Developmental relationships between aggression subtypes and disruptive behaviours

A series of CDM models were undertaken to evaluate developmental relationships between RA, PA and DBDs. A summary of findings is provided below.

6.5.3.4.1 Evaluating the primacy hypothesis

Expectations associated with the primacy hypothesis predicted that the developmental relationship between impulsivity and RA would implicate a set of genes that were independent of "pure" overt aggression (i.e., UCF). This prediction received modest support. As predicted, non-UCF related genes constituted the only

significant source of variance contributing to the developmental relationship between impulsivity and later RA (for both age cohorts). However, the degree to which this gene set influenced later RA was modest at best. For the younger cohort, **C** effects shared by UCF, impulsivity and RA accounted for an equal degree of variance when compared to the bivariate gene set. For the older cohort, **A** effects shared by UCF, impulsivity and RA accounted for an equal degree of variance as the specific gene set. These results suggest some plausibility in the notion of an impulsivity-to-RA risk pathway, however, this pathway is unlikely the dominant mode of transmission of risk for RA. Indeed, it is likely there are multiple pathways involved in RA (Raine et al., 2006).

Moreover, given the marked similarities in bivariate genetic architecture between RA and hyperactivity on the one hand, and RA and impulsivity on the other, it would need to be established that this primacy pathway retains some independence when the effects of hyperactivity are simultaneously considered in longitudinal modeling. At the same time, given the relatively high inter-class phenotypic correlations between impulsivity and RA at both Waves of the current series of studies (see Table 4.10, p. 168), additional analyses might focus on assessing whether RA independently increases risk of later impulsivity over and above other relevant disruptive behaviours (i.e., inattention, hyperactivity and PA) and PA.

6.5.3.4.2 Evaluating the role if proactive aggression and hyperactivity in the ODD-to-CD risk pathway

Current findings suggest that any risk hyperactivity confers on future PA in childhood is largely accounted for by etiological factors that also underpin RA. One

etiological source of variance, an **E** component, was uniquely associated with hyperactivity and later PA in childhood, however, these effects accounted for only a relatively small amount of variance in PA. Nonetheless, in the adolescent sample data, a more substantial factor (i.e., representing genetic influences) unique to the hyperactivity-to-PA pathway was present. This may represent a shift to greater genetic specification for hyperactivity in adolescence (i.e., greater differentiation between RA and hyperactivity). This may help explain why the prognostic association between hyperactivity at age 7, and PA at age 16, reported by Raine et al. (2006), showed unique predictive value after controlling for RA *at age 16*. The current results augment this developmental picture by suggesting that if the Raine et al. (2006) analysis was replicated with the inclusion of time-1 aggression subtype measures, controlling for RA at age 7 (instead of age 16) would reduce the probability of identifying a unique prognostic relationship between childhood hyperactivity and adolescent PA.

At the same time, the relative independence of the sequential pathway was evident and robust when hyperactivity was included as a covariate in trivariate longitudinal models. However, the hyperactivity, ODD and PA (i.e., denoted herein as hyperactivity>ODD>PA) trivariate model effectively replicated the sequential pathway model (both in terms of fit and pathway estimates). The hyperactivity, ODD and PA trivariate model too revealed greater genetic differentiation in the older cohort. Specifically, while both age cohorts showed a strong trivariate **A** component, only in the adolescent cohort data were substantial unique genetic effects identified. Again, a unique **E** component was identified in the childhood cohort data—possibly partly reflecting peer influence.

From this perspective, both the sequentially specified hyperactivity models as well as the ODD related models potentially implicate two potentially complementary risk pathways underpinning the transition from RA (and/or ODD) to PA (and/or possibly CD); a pathway associated with peer influence that precludes hyperactivity symptomatology, and another more biologically based pathway that does implicate this latter ADHD subtype dimension. Obviously, because it was not possible to model CD as an outcome variable, interpretations that include this clinical construct remain speculative and await verification from future research.

Chapter 7.
Online Technologies and Genetically Informative Data Collection

7.1 Overview

This chapter reports on the online component of the current research, i.e., Study 2. Specifically, the chapter focuses on attempts to ascertain reliability and validity of the online measures. Related technical and methodological issues unique to collecting psychological data online are also considered.

As noted in earlier chapters, caregiver ratings derived from the online questionnaire were used in longitudinal analyses reported in Chapter 6. The current chapter first examines the data derived from the two neuropsychological tasks completed by twins. As explained herein, this neuropsychological data failed to meet basic assumptions regarding expected associations with key behavioural indices measured by the online questionnaire. A brief synopsis of the task data is provided and possible reasons for the null results are explored.

The penultimate section briefly presents the rationale underpinning the adaption of the ATBRS into a web-ready electronic short-form format (ATBRS-ES). This section includes an examination of the reliability of the adapted ATBRS-ES.

7.2 Introduction

Online technology was utilised for Study 2 data capture and included two distinct methodologies; an online questionnaire and a series of computer-based neuropsychological tasks. Both the online questionnaire and tasks were delivered to families on a single CD-ROM. The software on the CD-ROM represented a

computer program that allowed families to login to an integrated testing environment and access both questionnaire and neuropsychological tasks. Although this software was run on the participants' home computer, the software required families to be connected to the internet so that security-related information (i.e., username and passwords) and participant data could be transmitted across the internet and into a secure database.

Both methods of online data collection were achieved by developing internet applications written using the object-oriented Flash (MX Professional 2004) platform (Underdahl, 2004; Vogeleer & Pizzi, 2004; Wilder, Wilson & Volion, 2003). Flash software is available commercially and is used extensively by web developers for a wide range of purposes.

7.3 Evaluating the Data Derived from the Neuropsychological Tasks

7.3.1 Validity of the Two Neuropsychological Tasks

For the purposes of establishing preliminary validity of the online neuropsychological tasks, a number of ATBRS items were selected a priori from the first wave of data collection on the basis that they provided some degree of face-validity to each respective neuropsychological task. These item are presented below.

7.3.1.1 Face Validity of the Online IGT Task

Items chosen from the ATBRS for establishing preliminary face-validity of the online IGT task included the following:

1. Actively defies or refuses to comply with adult's requests or rules
2. Has been suspended from school
3. Is able to delay being rewarded (reverse scored)

These items were chosen because (1) they reflect some failure of operant learning (e.g., insensitivity to aversive conditioning) that broadly requires a degree of response reversal, and (2) the behaviours indexed were not reliant on the need to discern covert socio-emotional cues such as facial expressions. The second point relates to the fact that, as much as possible, items were chosen that would offer a differential comparison between the IGT and Multimorph tasks. Note there is an inevitable overlap between proposed OCF function and ADHD symptomatology as evidenced by item three.

Combining and aggregating these items produced a Cronbach's alpha of .22 which is notably low. However, this collection of items was chosen solely on the basis of face validity, i.e., for the purpose of creating a set of behavioural indicators that individually share phenotypic characteristics with the external scale being evaluated. As such, internal coherence of the face validity indices was not considered a priority.

The current validity index did not correlate with the IGT index for either the younger (r=-0.06, p>.05) or older cohort (r=-0.04, p>.05).

7.3.1.2 Face Validity of the Online Multimorph Task

Items chosen to provide a preliminary assessment of face validity for the Multimorph task included:

1. Fails to understand the facial expression of others
2. Comprehends the emotions of others (reverse scored)
3. Feels bad for other people his/her age who are sad or upset (reverse scored)
4. Cares about other people's feelings (reverse scored)

It is noted that two of these items refer to a general rather than specific deficit in emotion recognition. The last two items provide a more targeted index of the phenotype of interest given their reference to empathic responsivity. A Cronbach's alpha of .61 was achieved for this rough index.

The main measure of interest derived from the Multimorph task was named the Selective Sensitivity Index (SSI). The creation of this variable is described in more detail in section 3.1 (p. 119). Briefly, the SSI reflects a combined index of perceptual sensitivity to sad and fearful facial expressions of emotion divided by the difference between this combined index and a second composite sensitivity score for happy and angry facial expressions of emotion. Sensitivity here generally refers to accuracy (i.e., number of emotional cues correctly identified) divided by the level of intensity required for accurate recognition. The resulting SSI had a skew value of 4.48 and was subsequently log transformed.

For the younger cohort, the transformed SSI index did not correlate significantly with the validity index (r=-.04, p>.05). Analysis of data from the older cohort also failed to obtain any significant correlation between the SSI and validity index (r=-.04, p>.05).

7.3.1.3 Reliability of the Multimorph Task

It was not possible to undertake an evaluation of reliability for the IGT given that results from this neuropychological task provided a single score only for each participant. By contrast, it was possible to undertake a preliminary assessment of the internal reliability of the Multimorph (MM) task. Table 7.1 below presents intercorrelations between the four indices derived for each of the emotions (happy, sad, angry and scared) from the MM data. The table suggests low-to-moderate inter-item reliability although unexpectedly there was no significant correlation between performance on identifying happy and sad facial expressions of emotion in the younger cohort.

Table 7.1

Correlation matrix for accuracy scores for recognition of facial expressions of emotion

	Happy	**Sad**	**Angry**	**Fear**
Correlated raw scores				
Happy	—	-0.13	0.25*	0.21*
Sad	0.18*	—	0.15*	0.23*
Angry	0.17*	0.19*	—	0.34*
Fear	0.16*	0.10*	0.18*	—
Sensitivity Index				
Happy	—	0.06	0.32*	0.25*
Sad	0.19*	—	0.16*	0.19*
Angry	0.30*	0.24*	—	0.31*
Fear	0.27*	0.19*	0.37*	—

Younger cohort (n=146) correlations are presented in the upper matrix; Older cohort (n=460) correlations presented in the lower matrix; Correlated raw scores indicates correlations between scores for the total number of facial expression identified correctly; Sensitivity Index presents correlations between index scores derived from dividing raw scores by response time; * significant at p<.05; Kendall's tau-b used due to non-normal nature of raw data anlaysed

7.3.2 Results Obtained from the Two Online Tasks

The first thing to note is that the online neuropsyhological study (i.e., Study 2b) failed to secure the number of participant families originally projected and this precluded behavioural genetic analysis. There were a number of reasons for this outcome and these are discussed further below. However, there was enough participant data to provide a number of tests relating to the double dissociation hypothesised in Chapter 2. Briefly, the literature review in this latter chapter identified the possibility that scores from the Multimorph task would correlate with PA not RA, while task performance on the IGT task would correlate with RA but not PA .

The correlation between SSI and PA was not significant for either the younger (r=-0.04, p>.05) of the older cohort (r=0.04, p>.05). Similarly, the correlation between the IGT index and RA failed to reach significance for both younger (r=-0.06, p>.05) and older cohort (r=-0.04, p>.05) data. In addition, for each analysis, residualised aggression subtype scores were used to assess the possibility that it was suppressing any underlying correlation. While this latter analysis was theoretically consistent with the double dissociation hypothesis, it failed to yield any significant results.

Information was collected to indicate if caregivers had scrolled to the end of the initial study overview presented on the front page of the assessment website and clicked a button to confirm having read the overview. The database contained only 318 families who had completed this introductory task. Of these families, 196 were

associated with complete data sets. While this variable was not able to definitively ascertain if all introductory instructions had been read, it provided a proxy measure indicating families who were perhaps more engaged in following task instructions than others. Conducting an analysis on this subset of families provided no additional information.

7.4 ATBRS Short-form Questionnaire for Online Administration

Many researchers have recognised the considerable advantages of administering a range of psychometric and behavioural questionnaires online (e.g., Carlbring et al., 2007)(Coles, Cook & Blake, 2007)(Lucia, Herrmann & Killias, 2007). A growing body of evidence suggests that this method demonstrates both reliability and validity equivalent to that of its pencil-and-paper counterpart in clinical research (Denscombe, 2006; Evans & Mathur, 2005).

The validity of web-based questionnaires has been confirmed in a number of areas of clinical study including Obssessive Compulsive Disorder (Coles et al., 2007), symptoms of panic (Austin, Carlbring, Richards & Andersson, 2006; Carlbring et al., 2007) and the self-report of delinquent behaviour (Lucia et al., 2007). To the author's knowledge, there are no reports assessing the reliability of online caregiver-rated data collection methods in research concerning aggression subtypes and/or the Disruptive Behaviour Disorders (DBD).

The remainder of this section provides an overview of the reliability of the online version of the Australian Twin Behaviour Rating Scale's (ATBRS) ADHD and aggression subtype indices. Validity of the online version of these two scales is

assumed given that the online behavioural measures used replicate established measures of the constructs they represent.

7.4.1 Reliability of the Online ATBRS-ES

In testing the reliability of the online questionnaire, invariance in the underlying factor structure for the Aggression Scales was assessed using two age bands; a cohort of children aged between 8 and 10 years and another cohort aged between 14 and 16 years. Each age range was analysed using a two-group between-subjects design. The first group represent participants whose parents completed the pencil-and-paper version of the ATBRS and whose twins fell into the relevant age range before July 2005. The second group represent participants whose parents submitted their ratings using the online ATBRS short-form questionnaire between May and August 2007 and whose twins fell into the relevant age range from July 2005 onwards. The two year age range (for both younger and older cohorts) was chosen to maximise the number of particpants able to be included while minimising the potential for overlap between comparison groups.

Parental ratings derived from the online questionnaire represented the second measurement occasion for raters (i.e., these parents had all previously completed a pencil-and-paper ATBRS). All families who fell into both groups were removed to maintain a between groups design. As with estimates of invariance reported in previous chapters the *n* of each sample was adjusted (i.e., halved) to accomodate the fact that data were double entered. Table 7.2 shows means for aggression subtype scores for both age cohorts.

Table 7.2

Mean raw scores for aggression subtypes by measurement occasion

	pre July 2005 Group			**Post July 2005 Group**		
	n	Mean RA (SD)	Mean PA (SD)	*n*	Mean RA (SD)	Mean PA (SD)
age 8-10 Cohort	172	2.27 (*1.92*)	0.35 (*0.76*)	92	3.57 (*2.22*)	0.81 (*1.27*)
age 14-16 Cohort	1072	2.42 (*1.89*)	.35 (*0.94*)	258	3.09 (*2.03*)	0.63 (*1.06*)

RA = Reactive Aggression, PA = Proactive aggression. **NB** Numbers in the above table indicate number of individual twins not twin pairs.

In the unconstrained model, data from the two younger cohorts resulted in a chi squaure of $\chi^2(16)=4.526$, p=.99, RMSEA =.00. Constraining the model so that the factor structure of the 'online questionnnaire' group conformed to that of the 'pencil-and-paper' group did not result in a significant reduction in fit, $\chi^2(20)=7.20$, p=.99, RMSEA =.00 with the chi square difference test yielding a chi square of $\chi^2(4, N=86, N=96) =2.67$, p>.05, RMSEA=.00.

The older cohort, the outcome was somewhat more marginal. The constrained model, $\chi^2(20)=23.95$, p=.24, RMSEA=.02, resulted in a marginally worse fit than the unconstrained model, x2(16)=10.06, p=.22, RMSEA=.02 with a difference of $\chi^2(4, N=536, N=129) = 13.89$, p<.05. The cut-off value for a chi square difference test involving 4 degrees of freedom is 13.28 (Keppel et al., 1992). It is clear that the difference obtained for the older cohort approximated this value. However, it is also noteworthy that, relative to the younger cohort, there were considerably more participants contributing data in the two older age bands.

While it was possible to analyse the ADHD items in a similar manner, the number of items involved (18 compared to the six for RA-PA) resulted in inadequate power, making reliability analysis untenable.

Finally, as noted in Chapter 4, the method of using a value for *n* that reflects the number of twin pairs rather than the number of individuals rated (Brendgen et al., 2006) has the paradoxical effect of increasing the capacity to determine a null effect by reducing the theoretical power of the model. This therefore, may not constitute the optimum method for determining null effects.

7.5 Discussion

This chapter evaluates methodology for obtaining genetically informative data using online technologies. Preliminary evaluation supports the value of using online questionnaires for research into aggression. Specifically, the ATBRS, an established pencil-and-paper parent questionnaire used for rating externalising behaviours in twins (Levy et al., 1996) was adapted for administration over the internet using (Dillman & Smyth, 2007) guidelines. A basic between-groups test of invariance of factor structure comparing pencil-and-paper versus internet administered questionnaire data provided preliminary support for equivalence across delivery methods. While replication with greater numbers is required for defintive conslusions this methodology appears to hold promise.

Efficiency is a key advantage in online administration of genetically informative behaviour questionnaires (Kaplowitz, Hadlock & Levine, n.d.). Notably, mail out procedures can potentially be significantly simplified through the use of online

technologies. This is particularly pertinent for large questionnaires involving multiple pages that require extensive collating. Printing and mailing costs can be reduced substantially and once initial consent has been obtained, convenient and cost-free correspondence can be maintained electronically via email or other online methods (Balter, Balter, Fondell & Lagerros, n.d.).

Perhaps one of the most significant advantages of online methods of data collection is the capacity for the automated entry of participant data. No data entry was required in the online component of the current study and, if good data integrity and security methods are established, this approach can substantially reduce research hours, resources and the potential for human error in the data entry process.

Unfortunately, examination of data integrity for the current study revealed that 85% of caregivers who submitted online questionnaires provided complete data sets. This is comparable to the 89% of complete data sets for the pencil-and-paper ATBRS. In fact, the figure of 89% was derived from a missing values analysis that included only the 47 variables of interest to the current study. A missing values analysis that included all 289 variables would have likely reduced this figure further. In other words, it is likely that the data integrity achieved from the short-form online questionnaire at least equals that of the larger pencil-and-paper questionnaire. Of course, length of questionnaire must also be considered in understanding the difference between completion rates. Interestingly, Table 4.9, p. 165, indicates small but consistent improvements in scale reliability (i.e., increased Cronbach's alpha) across three of the four longitudinal behavioural measures (i.e., RA, impulsivity and hyperactivity) when the online questionnaire is compared with its pencil-and-paper

counterpart. While this increased reliability was not evaluated for significance, the trend across the majority of relevant measures is interesting. Security of data transmission and storage was ensured using an industry standard security protocol called a Secure Socket Layer (SSL).

As the data integrity issue identified immediately above illustrates, efficiencies associated with utilisation of internet technologies are accompanied by additional challenges unique to online delivery of questionnaire content (Dillman & Smyth, 2007). This issue is examined further below. The next section considers factors affecting participation rates, attrition and completion associated with the online component of the current study.

7.5.1 Participation Rates and Attrition in the Online Study

The current study achieved a participation rate of 58% (i.e., only 1217 of 2082 families who were originally approached to participate consented and completed the study) which was reasonable but lower than would be expected given that all contacted families had, a priori, voluntarily provided consent to be approached to participate in genetically-informative studies (for example see, Dillman et al., 2000). However, it should be remembered that indications from the current study suggest that as many as 19% of the families initially approached to participate in Study 2 may not have had access to the internet. Adjusting for this estimate of access results in a higher 73% participation rate. Regardless of adjustment, it is likely that a broader range of factors impacted on response rates for both participation and attrition.

For example, it is possible that circumstantial factors such as recent or ongoing participation in other research projects may have impacted on ATR families' decisions to provide consent to participate in the current study—helping to explain the high level of non-respondants. At the same time, the genuinely proactive support of ATR families for twin research may have led to an overcommitment of resources for some participating families—potentially helping to explain those families who consented but subsequently informally withdrew. Indeed, in the small feedback survey conducted with families who did not complete the online component of the study (see section 4.2, p. 157), the most common reason provided for informal withdrawal was difficulties with the time-frame of the study. This survey (n = 77) was adequately large enough to be considered moderately representative. Moreover, the general sentiment expressed via this survey was generally consistent with qualitative feedback received from parents via emails and telephone.

Familiarity with (online) technologies may have been a factor influencing risk of participation or attrition. A suggestion for future research utilising internet technologies is to include a feedback question in initial approach and consent information packs that allows families to indicate that they are chosing not to participate because they do not feel comfortable using the internet in the way requested for the specific research project being proposed. Certainly, the second most common reason for withdrawal from the study according to the feedback survey was "technical problems" which may further indicate a certain lack of comfort with relevant technologies. At the same time, the issue of technical problems was indeed a real one and a valid reason for informal withdrawal—discussed in more detail below.

The prerequisite of home access to the internet impacted on the number of families who were able to participate. Indications from the feedback questionnaire provided in the initial approach pack suggested that around 19% of families could not take part for this reason. While this is a substantial number, it is likely that both access to the internet and people's comfort levels with using it will continue to increase significantly over the next decade. For example Australian national census information demonstrates that homes with access to the internet have increased over the last decade and continue to do so.

This statistic provides an interesting comparison to the most recent statistics offered by the Organisation for Economic Co-operation & Development (OECD, http://www.oecd.org/document/54/0,3343,en_2649_34225_38690102_1_1_1_1,00.html, 26/10/2008) pertaining to broadband usage in Australia. The recent OECD report suggests that only around 23.6% of Australians have access to broadband connection. This suggests that possibility that many participant families were utilising older dial-up internet technology which is slower and less reliable than its broadband counterpart. This has direct implications for the issue of data integrity as discussed below.

7.5.2 The Role of Data Integrity in Completion Rates

Perhaps the most disappointing trend was the poor completion rate in the present research due to technical problems. Specifically, an average of only 78.9% of participant data was received from parents and children who genuinely completed the online questionnaires and tasks. As there was no means for users to deliberately submit null or missing data during administration of the online questionnaire or neuropsychological tasks, any evidence of random (i.e., non-contiguous) missing

data within an individual data set represented compromised data transmission. This led to the statistically costly exercise of removing cases wherever missing data was identified. While most contemporary statistical software packages provide relatively robust methods for minimising the effects of missing data, a potential problem with online data collection is that unless the data capture system used includes a sophisticated capacity for monitoring errors in data transmission, it is extremely difficult to ascertain if faulty transmission has caused cascading errors, i.e., errors that compound to compromise all subsequent data received from that participant. It was thus necessary to be conservative and remove any case where there was any evidence of missing data.

7.5.2.1 Possible Causes of Data Transmission Errors

While an exploration of all possible technical reasons for errors in data transmission is beyond the scope of this text, it is useful to identify two classes of errors that can occur. The first category represents problems with transmission that result from temporary failures of the network supporting data transmission. The second pertains to possible problems with the design and/or implementation of client software protocols (i.e., the specific web-based applications that the user interacts with to collect and send the data). Each of these is considered in turn.

7.5.2.1.1 Network Integrity and Failure

Compared with other developed nations Australia has been slow to adopt and implement a state-of-the-art national-wide digital network and supporting infrastructure to enable fast and ready access to the internet. This lag in infrastructure development and service provision is due to a confluence of geographical, political

and commercial reasons. In providing its constituents with information on broadband technology and infrastructure in Australia, one Australian municipality suggests,

> Australia is currently behind most other developed nations sitting at 17th spot in terms of Broadband Penetration per 100 inhabitants on the International Broadband Table compiled by the Organisation for Economic Co-operation & Development...The problem exists due to Australia's large land mass and comparably small population. This makes for a very expensive infrastructure roll-out. The other hindrance to progress has been Australia's traditionally tight communications legislation meaning investment is restricted by government regulations (http://www.whittlesea.vic.gov.au/content/content.asp?cnid=3030, 26/10/2008).

The OECD (http://www.oecd.org/document/54/0,3343,en_2649_34225_38690102_1_1_1_1,00.html, 26/10/2008) report statistics show that in June 2008 Australia increased its rank to 16th in the international Broadband Penetration table mentioned above, with 23.6% of inhabitants receiving broadband services. This is compared to 36.7% and 35.5% penetration in the two top ranked countries; Denmark and the Netherlands respectively. According to figures from the same set of reports, Australia's broadband penetration grew by 133% in the year 2006-2007 (and was ranked 27th amongst developed nations in regards to growth of services). Although information on types of technology used by households is currently limited in these OECD reports, it appears from current statistics that around 3.5% of broadband subscribers rely on mobile (i.e., satellite) technology.

The direct consequence of the slow adoption of new technologies is poor quality infrastructure that lacks the bandwidth to provide full coverage and full capacity for all users. This has the dual effect of limiting internet access to households as well as

adverse effects on the quality of data transmission. Most users of the internet are familiar with the net "dropping out" or "going down". These colloquial terms describe when the internet becomes periodically unavailable due to a temporary failure of the connection that links the users computer and the internet. These times constitute periods in which data transmission is highly compromised. The most common cause of this problem is "overcrowding" (i.e.,) which occurs when the broader network is subject to levels of data transmission that exceed network capacity—ultimately causing significant interruptions to data flow and, frequently, data loss (Wu & Irwin, 1998). These interruptions have many technical causes and a myriad of effects. In regards to online collection of psychological data, the net effect of current network limitations can mean relatively unreliable means to transmit data where complex and dynamic assessment protocols are required (as in the current research).

This problem of an unreliable connection is particularly pronounced for users who receive internet services via satellite technologies or dial-up modem based services. These technologies are well-known to be subject to frequent dropping out. Utilising older technologies such as dial-up modem based services is accompanied by an additional slate of data transmission difficulties such as significantly slower speeds and significantly restricted bandwidth.

Unfortunately, information concerning the type of internet service (i.e., broadband versus dial-up etc) that participant families used during the online component of the current study was not collected. While this would have provided useful information to help explain which internet delivery methods proved most problematic, frequent

advances in technology mean that the kinds of technology available to everyday consumers changes rapidly. Indeed, the positive news for future researchers hoping to take advantage of internet technologies for data collection is that the current limitations on data transmission are inevitably a temporary problem. This is an area receiving increasing political attention as the role of internet technologies in everyday commerce and finance continues to grow.

7.5.2.1.2 Software Design

Another important factor in ensuring data integrity is software design. For social science researchers wanting to utilise technologies that employ high-level programming languages, such as the one used in the current study (explained below), there are three facets to software design to consider; (1) choice of programming language and the specific limitations of that language (and its supporting framework), (2) implementation of data transfer protocols, and (3) presentational and design features that allow the user/participant to interact with the software.

Before these facets are examined, it is useful to briefly introduce the concept of "high-level" programming languages. The diverse range of computer programming languages available to programmers vary in regards to the extent they have been simplified for ease of learning and use. Generally speaking, the term high-level refers to programmatic protocols designed to make computer code more closely resemble human language (including syntactic structure). This attempt to make computer programming are more accessible to a broader range of non-professional programmers is typically achieved by pre-packaging many of the functional elements that comprise the broader framework in order to reduce the amount and complexity

of coding required. Such accessibility usually comes at the expense of flexibility. That is, the programmer is often limited in options when trying to operationalise procedural elements of the software.

The web-ready software authoring package used for all online data collection protocols in the current study (based on the Adobe Flash platform, Wilder et al., 2003) incorporates a high-level object oriented programming language (ActionScript; Moock, 2003; Sanders, 2002) that allows users to develop interactive content for the internet. The compromised flexibility often inherent in high-level languages speaks to the first facet of software design important to consider. Object oriented programming languages are organised around the concept of "classes" that represent aggregations of variable properties that can be programmed to interact with one another. This is in contrast to the more traditional "procedural" languages that are based on the concept of "methods" representing a hierachy of computational operations triggered on conditional bases.

That is, high-level languages are often limited in their capacity to implement sophisticated computations that involve, for example, precision in time-based processing. Many neuropsychological tasks require precise measurement of reaction time and other chronological features. While tasks that rely on these functions were deliberately avoided in the current study, there was still a need to standardise exposure time of stimuli for the MM task in a consistent manner. At the time the computer task was adapted for delivery on the internet, the version of the Flash software used was 7.2. This version utilised ActionScript 2 (Lott & Reinhardt, 2006; Moock, 2003) which is relatively limited in its ability to use time-based functions—

including the standardisation of exposure time. A stable work around was ultimately achieved but earlier versions of the software appeared to cause some problems for some participant families using certain computer configurations. That is, around 50 families reported that the MM task froze during operation. This number likely reflects only a proportion of all families experiencing this specific difficulty. At the same time, it was clear that a majority of families who commenced the task, completed it without any problem.

When the above problem did occur, this technical fault did not seem to relate to a specific operating system (i.e., Windows XP, Mac OS X) installed on the home personal computer used by participant families. Providing timely support around this issue was a significant challenge as the fault was not replicable on either the Apple Macintosh computer (using OS X) or Windows PC (XP) used for testing prior to or after the fault was reported by families. Unfortunately, no matter how well a cross-platform computer language translates across multiple platforms and operating systems, it is common for conflicts to occur between the implementation of a specific protocol (i.e., in this case, time-based functionality within Flash) and system software and idiosyncratic hardware configurations upon which it is installed. There are literally countless configurations and, as such, many possible reasons why some systems will not implement a specific protocol well (Dillman et al., 2000).

At around the time the fault was being reported by families, the author was able to obtain an updated version of the Flash authoring software (version 8; Lott & Reinhardt, 2006). This included improved implementation of time-based processing. The task was (re)compiled with this enhanced protocol and sent out to families who

had reported experiencing trouble. This appeared to substantially reduce the number of faults being reported. This underscores the fact that user-friendly software authoring packages are becoming increasingly sophisticated with each updated version. This software is highly recommended to social scientists who wish to deliver assessment protocols that involve visuo-spatial content or content that relies on other multimedia features (e.g, audio, video, etc).

A second issue concerns the implementation of data transfer protocols. Although there is an increasing convergence of online technologies, the internet consists of a diverse and often divergent range of information technology protocols and frameworks that differ on the basis of functionality and commercial implementation. The Adobe Flash platform has evolved from a simple web-ready application designed to deliver scalable and interactive graphics within web-browsers, to a highly complex "rich media" platform which can perform many sophisticated functions including the transfer of data from the user to a database on a remote server. It is the combination of these various features that make Flash attractive to the prospective social science researcher. However, the task of getting the data from the Flash application (typically downloaded to the users computer) to the remote database requires implementing an appropriate data transfer protocol.

The Practical Extraction and Report Language (Perl; see, Fraley, 2005), was chosen as the intermediary protocol to assist in transferring participant data from the Flash application installed on users' home computer to the secure online mySQL database (Dyer, 2005; Kroenke, 2002). This choice was based on the ubiquity and reliability of the Perl framework. While technical details of how this protocol was implemented

are beyond the scope of this text, an issue relevant to its functional implementation (one that may have affected data integrity) is worth mentioning. That is, the choice of whether to send smaller but higher volume "packets" of participant data across the internet versus larger packets less frequently. Essentially, this refers to whether data from a single user response on a specific trial is sent individually to the database at the time of the response, or, alternatively, individual trial data is stored cumulatively on the user's computer and sent as one package at the completion of the task. The risk associated with the latter option relates to the fact that when data transmission fails, all data is lost for the entire task. Conversely, the risk of transmission failure increases with the number of packets sent. Ultimately, a decision was made to send data at each trial because the former risk (i.e., losing the participant's entire data set for a specific task) was deemed too great. However, as explained above, it was not ultimately possible to maintain a data set with missing data, so in retrospect, the better option would have been to transmit larger packets of data less frequently.

Finally, the graphical user interface (GUI) and other presentational design features used to deliver web-based psychological assessment protocols is also crucial (Dillman et al., 2000). If there are multiple tests to be delivered to the same participant or user, as in the current study, then designing a secure, easy-to-use integrated testing environment is important. In the current study, users logged in to an integrated assessment environment. To describe this interactive assessment environment (IAE) as a web site is not strictly correct as the IAE was in fact a Flash application delivered to families on CD-ROM and requiring manual installation on the home computers of participants. Nonetheless, a stable connection to the internet was required for the duration of interaction with this IAE because the applications

within this assessment environment were designed to send information from the user's computer to the remote database. For example, to gain access to the assessment environment, users were required to provide a username and password that was checked against a unique ID entry for the family within the remote database.

On successful login users were taken to an introduction page where caregivers were asked to read through an introduction to the study that included basic instructions for completing the study. Delivering clear easy-to-access instructions online is crucial (Dillman et al., 2000). As mentioned above, a "button" was placed at the end of these instructions requesting parents click it once they had read the introductory material.

One design feature that required resolving in the current study is worth noting. Because all assessment protocols were integrated into one online testing environment, this meant that parents and twins would be navigating within the same online testing environment. A such, it became necessary to decide between providing (1) secondary security within this environment to restrict child access to the parent questionnaire section, versus (2) providing instructions that recommended parents either maintain the initial password provided and closely monitor and guide their twins navigation through the testing environment, or simply complete the parent questionnaire after the twins had completed the neuropsychological tests. This "instructions" option was chosen over providing the family with an additional security measures (i.e., an additional password) due to the undesirable consequence of increasing the complexity of study demands on participant families associated with the latter option.

The assessment environment is but one aspect of the overall testing environment. The issue of parental monitoring and guidance extends to the broader question of controlling overall environmental testing conditions. This is the topic of the next section.

7.5.2.1.3 Environmental testing conditions

Arguably, one of the most fundamental factors affecting any assessment procedure is the environmental conditions under which testing takes place. A defining feature of the current online methodology, particularly as it pertains to the neuropsychological tasks completed by participating twins, is that the testing environment constituted a relatively uncontrolled, or under-controlled, variable. In short, despite providing families with clear instructions regarding what constitutes adequate testing conditions, it was not possible to precisely ascertain whether (1) these instructions were followed, or (2) other unforeseen factors were impacting on the testing environment that were not directly addressed in the instructions. Because testing occurred in the family home rather than a controlled laboratory environment it is difficult to predict and manage the diversity of distractions that might occur at any given moment in the family home. As mentioned previously, a simple check was included in the study which asked whether a parent was present while the twin completed the neuropsychological tasks. Again, it was not possible to determine the accuracy with which respondents answered this question. Moreover, although caregivers were instructed not assist or interfere while their child completed their tasks, it was not possible to determine adherence to this request.

The caregiver monitoring variable provided no additional clarification when entered

into the analyses. It therefore remains the subject of future investigations whether assessment protocols can be developed that ensure adequate integrity of testing environments that involve "remote" self-directed assessment using online neuropsychological tasks.

An alternative explanation for the current lack of results is of course that the actual neuropsychological tasks themselves failed to provide an adequate index of the behavioural dimensions under investigation. It is clear that any empirical progress requires a set of direct comparisons of neuropsychological tasks administered remotely online versus under conventional controlled settings. This would allow some level of reliability to be established for use of this kind of novel methodology.

7.5.3 Provision of Support Services to Families Contrinuting Online Data

In regards to the provision of support to families, the time and resources potentially saved by eliminating mail out and data entry requirements, may be offset by the need to provide timely support services to families who are having trouble using the technologies employed in the online study. For the duration of the three to four month period in which most families who engaged in the online component of the study were actively contributing data, maintenance of web site and database consumed approximately 15-20 hours a per week for one person. A vast majority of this time was dedicated to support services including responding to email requests for assistance.

Three families expressed expressed a mild degree of frustration for not receiving

timely response to their enquiries. Although these families were clearly in the minority, it is likely that the number of instances of expressed frustration under-represents the degree to which frustration was experienced by families. Frustration pertained to both problems with technology and occasionally a sense that support was not timely and/or adequate. Despite using all available technologies (i.e., sophisticated electronic mail merge technologies) to reach as many families, experiencing similar difficulties, this did not aid in attending to specific enquires that were uniquely expereinced by individual participants. It is the latter circumstance that consumed a disproportionate amount of support-related resources.

Chapter 8.
General Discussion

8.1 Overview

To the author's knowledge, the current thesis is the first to report on shared genetic architecture between aggression subtypes and DBDs. Specifically, caregiver-rated data pertaining to RA, PA, ADHD, ODD and CD behaviour dimensions were derived from a large community sample of families of twins (aged 6-18 years) and were submitted to quantitative genetic modeling to assess core etiological relationships between aggressive subtypes and disruptive behaviour disorders. These analyses were guided by a number of core aims including to;

1. elucidate the role of shared environmental effects in RA and PA,
2. assess the relevance of the smorgasbord hypothesis to RA and PA, and disruptive behaviours,
3. evaluate a recent sequential model of RA and PA,
4. provide the first investigation of etiological relations between DBDs,
5. evaluate a number of putative risk pathways involving aggression subtypes and specific DBD related behaviour dimensions, and finally,
6. evaluate the use of online technologies for endophenotypic research into potential differential neuro-biogenic mechanisms underpinning RA and PA, and psychological research more broadly.

A summary of findings and implications for clinical models of ASB pathways that derive from these analyses are considered. A brief synopsis of methodological considerations is also included pertaining to the use of online protocols and

procedures in this project. Recommendations for psychological research that utilises online technologies are discussed. Finally, a number of limitations associated with the current research are presented.

8.2 Etiological Relationships Between Aggression Subtypes and Disruptive Behaviours

Results from genetic modeling of the current cross-sectional data were generally consistent with previous studies suggesting a strong influence of genes on reactive and proactive aggression (Baker et al., 2007; e.g., Brendgen et al., 2006). Best fitting univariate models of RA produced ACE estimates accounting for **A**=30%, **C**=56% and **E**=14% of variance in RA in the younger cohort, and **A**=53%, **C**=32% and **E**=15% in the older cohort. Best fitting models of PA produced AE estimates of **A**=50% and **E**=50% for PA in the younger cohort and **A**=83% **E**=17% in the older cohort.

By comparison, using maternal rated data derived from a large community sample of 9-10 year olds, Baker et al. (2008) reported ACE estimates representing 26%, 27% and 47% of variance in RA, and 32%, 21% and 47% of variance in PA respectively. Brendgen et al. (2006) reported best fitting AE models for both RA (**A**=40% and **E**=60%) and PA (**A**=41% and **E**=59%) as ascertained from teacher ratings of RA and PA in a sample of 6-year-old children.

While the current estimates of genetic effects for childhood RA are consistent with those of Baker et al. and Brendgen et al., the corresponding estimates of **A** for childhood PA were of higher magnitude than previous research, and nearly twice that

reported by Baker et al. The addition of an adolescent cohort in the current study produced estimates of additive genetic influence of significantly higher magnitude than childhood estimates—and this was especially true for PA.

Establishing definitive patterns of **C** effects was more elusive in cross-sectional analyses. These latter class of etiological influences are typically more difficult to ascertain due to their statistical volatility (Rutter, 2006). Certainly, the typical pattern of wider confidence intervals associated with **C** effects was evident in most models. However, when issues of sample size and possible informant bias are taken into account for current and previous genetic studies, the overall evidence for **C** effects on RA is compelling—with current estimates suggesting moderate to large magnitude effects. These results buttress previous reports of moderate **C** effects on RA (Baker et al., 2008). The evidence for **C** effects on PA remains somewhat more tenuous. However, when all three extant genetic studies are considered together, there is a clear trend for shared environmental influences of low to moderate magnitude, with increasing sample size.

Multivariate cross-sectional modeling of RA and PA revealed that despite strong effects of genes on childhood RA, these influences entirely overlapped with those of PA, suggesting that genes do not differentially contribute to RA at this formative time in development. By contrast, cross-trait genetic influences for PA in the younger cohort, as well as RA and PA in the older cohort, accounted for around half the overall genetic influence. Baker et al. (2008) produced estimates of total overlap constituting around 75% of all genetic influence on RA and PA. These estimates are considered preliminary because neither the bivariate models reported here, or in

Baker et al., controlled for etiological effects on physical aggression which were common to both RA and PA. trivariate models which did include this control are discussed shortly. In line with univariate models, **C** effects operated on RA only in both younger and older cohorts.

The results of Brendgen et al. (2006) versus Baker et al. (2008) contradict the smorgasbord hypothesis which predicts that (1) the magnitude of **C** effects will decrease with age, and (2) that this decrease will be accompanied by a proportional increase in the magnitude of **A** effects. However, the current project has a number of advantages over across-studies comparison in evaluating this broad hypothesis. Firstly, the age range of the current sample encompassed a wider developmental window than captured by the previous studies. Importantly, this included both childhood and adolescent cohorts. Compared to childhood, adolescence is generally considered to represent a time in development when family influences significantly decline and autonomy substantially increases (Miles & Carey, 1997). Secondly, the current project was able to assess age cohort differences using consistent measures and consistent overarching methodology. Conversely, the Brendgen and Baker studies utilised different measures of RA and PA and submitted resultant data to contrasting analytical procedures.

Consistent with the smorgasbord hypothesis, current cross-sectional modeling of RA demonstrated that shared environmental effects tend to be stronger than genetic influences in childhood RA with the inverse being true in adolescent RA. In regards to PA, there was a substantially greater magnitude of genetic effects in the adolescent versus the childhood cohort. Given the possibility that PA-related **C** effects were

dropped from the initial fully specified univariate model due to lack of power, it is interesting to note that estimates for these dropped parameters were indeed less for the older age group compared to the younger age group.

The substantial genetic estimates for PA in univariate models, along with the correspondingly absent (or possibly low) **C** effects raise the possibility of spurious effects. The small but statistically significant higher estimate of variance in adolescent PA in DZ versus MZ twins (see Appendix F) potentially implicates competitive sibling interaction effects (i.e., Neale & Cardon, 1992). However, higher variance in DZ versus MZ twins as a result of these latter effects is usually accompanied by DZ correlations that are less than half those for MZ twins (i.e., mimicking non-additive genetic effects). This pattern was not found in the current data.

The possibility remains that genetic effects on PA are inflated by gene-environment interplay. Gene-environment interplay might include either GxE interactions or GE correlations, both of which represent a synergistic combination of **A** and **C** influence which is mediated primarily through genetic effects. Standard genetic models are not able to differentiate this interplay from main effects. Instead, interplay effects are captured (i.e., confounded) within the **A** variance component (Moffitt, 2005b; Rutter & Silberg, 2002). It was not possible to test for these effects in the current series of studies, however, the issue is considered in more detail when longitudinal data are discussed below.

At the same time, current modeling revealed exceptions to the smorgasbord

hypothesis. Most notably, bivariate models that included PA on the one hand, and hyperactivity and CD on the other, showed a pattern opposite to that predicted by this latter formulation. This finding is discussed in more detail below.

Longitudinal analyses revealed strong genetic effects operating on both continuity and discontinuity for RA and PA. Results highlight the importance of subtype-specific genes for persistence in aggression subtypes by revealing that genes influenced continuity over and above UCF for both RA and PA in the younger cohort, and PA in the older cohort. The exception was adolescent RA which was affected by two separate sets of genes; one simultaneously operating on continuity in RA as well as UCF, and another operating at the second wave only. Moreover, while continuity in childhood RA was substantially impacted by subtype specific genes, the relevant model also revealed a trivariate **C** component which affected both continuity and UCF. On the other hand, both older and younger cohort models of PA revealed that all genes operating on continuity were subtype specific. It is noted, that no analyses were included in which RA, PA and UCF were all included at the same time point, which places some limits on current interpretations about the influence of subtype-specific genes on continuity.

When compared to genetic effects on continuity, the longitudinal effects of non-shared environmental influences were small to insubstantial. Specifically, there were no subtype-specific **E** effects found on continuity in either RA or PA in the younger cohort, and only small effects in the older cohort. Interestingly, the structure and magnitude of effects across both age cohorts were very similar for both RA and PA, indicating no differential strength of impact of peer influence on RA and PA found in

previous research (Fite & Colder, 2007). As such, the current results generally concur with previous longitudinal genetic research into undifferentiated aggression which indicates only small **E** effects on continuity (i.e., Haberstick et al., 2006). Thus, the current results appear to challenge Brendgen et al.'s (2006) hypothesised primary role for **E** effects in the shaping of aggression subtypes.

However, non-shared environmental effects did rival the substantial impact of genes on trait-specific Wave-2 estimates of aggression subtypes. This implicates an important role for **E** effects in discontinuity of aggression subtypes. To the extent these **E** influences are indexing non-error related variance, they may reflect a degree of volatility in social circumstances outside of the home. For example, this might include significant shifts occurring in peer affiliations and associated peer networks. In this way, current results might be considered to support Brendgen if a broader definition of shaping is used. Certainly, consistent with the pattern of **E** effects reported by Brendgen et al., the current estimates of **E** effects associated with discontinuity were moderately higher for PA than RA in the younger cohort.

Whatever specific mechanisms are being indexed by genetic and non-shared environmental effects on discontinuity in the current longitudinal data, the above results highlight a pervasive dynamism associated with the development of RA and PA across the developmental spectrum.

There was limited evidence for shared environmental effects on persistence in RA. Again, depending on how the social learning mechanisms proposed by Dodge and colleagues are conceptually mapped within a behaviour genetic framework, this

result might be considered contrary to the seminal psychosocial model of RA and PA (Crick & Dodge, 1996). More clearly at odds with this latter etiological model, the only **C** effects on continuity in PA were shared with UCF. This result was consistent across both age cohorts.

It is interesting to examine in more detail this apparent emergent cross-trait **C** component, given that basic univariate and bivariate models (including a bivariate longitudinal specification) of PA precluded **C** effects. The observation provided in parentheses is important because it undermines the explanation that longitudinal designs are more sensitive to **C** effects than cross-sectional modeling. From this perspective, the key difference in PA specifications that included, versus precluded, **C** effects, was the presence of the additional UCF variable in the relevant trivariate models.

The UCF factor consisted of a subset of aggression-related CD symptoms. Certainly, some studies have shown at least moderate **C** effects on CD (Burt et al., 2001; Waldman et al., 2001). Still other studies have suggested a more specific link between *non-aggressive* CD symptoms and shared environmental effects (Simonoff et al., 1998). At the same time, Simonoff et al. (1995) demonstrated that at least a portion of **C** estimates on caregiver rated CD were attributable to rater bias. It was not possible to formally evaluate the possible presence of rater bias in the current data. Nonetheless, the issue is considered in more detail shortly below.

Notwithstanding the important issues raised above, the presence of **C** effects in the current trivariate longitudinal model involving UCF and PA suggests that, to an

important degree, what is common to both UCF and PA (presumably overt physical aggression) is mediated through shared environmental mechanisms. In short, the above set of observations constitute a mediation based explanation of **C** effects on PA. That is, the covariance between UCF and PA may work to amplify **C** effects present in the PA data. Given that cross-trait **C** effects were found for PA and CD in the relevant model for the adolescent and not the younger cohort (which was of significantly large sample size than the younger cohort), it might be surmised that the three factors of (1) sample size, (2) increased sensitivity of the longitudinal design to **C** effects, and (3) the presence of a primary mediating factor, might all combine to increase the statistical power to a level capable of detecting **C** effects on PA.

The expected high interclass phenotypic correlations between aggression subtypes and DBD behaviour dimensions were almost all under at least some genetic control. Genetic estimates for the relevant cross-trait overlap ranged from 17.6% (in adolescent PA and hyperactivity) through to around 50% (in adolescent PA and ODD) of overall variance in the paired traits. The only model in which genes did not account for any bivariate covariance was in the model for childhood PA and inattention. In this latter model, all overlap was explained exhaustively by shared environmental effects (accounting for 18.5% of variance in each of these traits). Mentioned earlier, the older cohort models for PA and hyperactivity, and PA and CD both showed emergent **C** effects on trait overlap explaining around 10% and 15% of variance in paired traits respectively). Otherwise, no other best fitting models involving PA were associated with cross-trait C effects.

Non-shared environmental effects on shared variance were small to negligible for the

bivariate PA and DBD models. In fact, three of the six bivariate models pairing PA with the three ADHD-related behaviour dimensions precluded **E** effects altogether. These models included hyperactivity and inattention in the younger cohort, and inattention in the older cohort. The remaining ADHD-related PA models revealed **E** estimates that ranged from 2% to 3.5% as a proportion of trait variance. **E** estimates in the models in which PA was paired with the antisocial DBDs, ODD and CD, evidenced consistent but small effects ranging from 5.3% to 14.4% of variance (in PA and ODD in the older cohort, and PA and CD in the younger cohort respectively).

In regards to RA, the only bivariate DBD-related model that did not include E effects was for RA and inattention in the older cohort. Otherwise, consistent, albeit small, **E** estimates were found for all other DBD behaviour dimensions when paired with RA. These estimates ranged from 2.5% to 6.8% of relevant trait variance.

As noted above, where **C** effects were present in best fitting models of RA and DBD behaviour dimensions, these effects tended to be substantial in the younger cohort, ranging from 28.1% (for variance associated with RA and CD in the younger cohort) and around 35% (for variance associated with RA and ODD). **C** effects were present in all older cohort bivariate models involving DBD behaviours and RA. These latter estimates ranged from 9.6% (RA and ODD) to 24% (RA and hyperactivity). Younger cohort models describing the relationship between childhood RA, on the one hand, and hyperactivity and impulsivity on the other, precluded all **C** effects. It is worth noting that these latter two models were structurally identical. Conversely, the older cohort models that included these two ADHD behaviour dimensions, showed

substantial cross-trait **C** influences—accounting for 24% and 19.4% of trait variance in the hyperactivity and impulsivity models respectively.

The finding of cross-trait **C** effects in the older cohort bivariate models that included RA on the one hand, and hyperactivity or impulsivity on the other warrants further comment. As indicated above, there was no evidence for cooperative SI effects in cross-trait models involving RA and hyperactivity (or RA and impulsivity) in the older cohort. Although speculative, it may be that these latter effects reflect results from the CV regression model in the sex-differences analyses reported in Chapter 5. Findings from the CV analysis suggest the possibility of a discontinuous form of male RA in adolescence due to environmental influences. It is at least theoretically plausible that discontinuous adolescent male RA resulting from shared environmental influences may be associated with concurrent hyperactivity and impulsivity. Determining the exact nature of these effects may warrant focus for future study.

A somewhat broader observation concerning the range of apparent emergent cross-trait **C** effects in a number of older cohort models, is the general finding that these effects tended to be less segregated in the older versus younger cohorts. Specifically, in many of these models (the bivariate RA and hyperactivity and RA and impulsivity models being good example, see Figure 5.7, p. 211), shared environmental effects were more strongly represented in trait-specific **C** terms in the younger cohort models, with the older cohort specifications showing a stronger (or at least increased) representation of **C** effects at the cross-trait level.

So for example, in the bivariate model including RA and hyperactivity, the best fitting configuration in the younger cohort involved only one **C** component; a trait-specific **C** effect on RA (accounting for 43% of variance in this aggression subtype). In the older cohort, there was again only one **C** component, but in this model, the relevant effects were entirely cross-trait in nature—accounting for 24% of variance in RA (and hyperactivity).

Conversely, there was an opposite trend for genetic effects. Extending the above example, as predicted by the smorgasbord hypothesis (SH), genetic effects on RA were of greater magnitude in the older versus younger cohort. However, in addition, there was also increased segregation of genetic effects. That is, these latter effects manifested at the trait-specific level in both RA and hyperactivity in the older whereas only hyperactivity showed trait-specific effects in the younger cohort.

Although speculative, this potentially represents an elaboration of the SH suggesting that **C** effects decrease in magnitude with age *and become more diffuse* (i.e., less likely to operate in different ways on different disruptive behaviours) with the inverse pattern being true for genetic effects, i.e., more diffuse and diluted genetic effects in childhood that increasingly strengthen *and segregate across different disruptive behaviours* with age.

This pattern of age effects on the diffuse versus segregated nature of etiological effects was at least partially present in bivariate RA models with the following covariates; hyperactivity, impulsivity, and ODD, and in PA bivariate models with following covariates; ODD and CD.

A number of exceptions of this pattern are worth mentioning. Firstly, inattention showed a somewhat unusual bivariate genetic architecture in both RA and PA models. The fact that it was not possible to fit the inattention data to trivariate models suggested anomalies with this particular data set. Inspection of the data revealed a number of somewhat unexpected deviations from expected patterns in MZ and DZ correlations. Given the dearth of theories that articulate a central role for inattention in aggression (Dadds & Rhodes, 2008; although see Dadds et al., 2006), elucidation of these patterns awaits further enquiry. It is noted however, that zero-order correlations derived from the current scales (see Table 4.10, p. 168) certainly indicated inattention is at least moderately correlated with both RA and PA and this relationship has been confirmed in previous studies (Dodge & Coie, 1987; Vitaro et al., 2002).

Secondly, given the inconclusive results concerning **C** effects on PA, it was more difficult to assess for the above pattern in models involving this latter aggression subtype. It may be worthwhile to undertake a series of more focused analyses aimed at identifying the reliability, validity and scope of this pattern in a larger cohort. Certainly, as mentioned above, there was some evidence of this general pattern in the PA bivariate models involving ODD and CD.

Returning to the broader issue of **C** effects (and as noted previously), the possibility must be considered that rater bias is spuriously impacting on **C** effects retained in the current series of best fitting models. The possibility of rater bias impacting on the cross-trait **C** effects involving PA (and CD as well as hyperactivity) are of particular

interest given the apparent lack of **C** effects on univariate PA. Certainly, the likelihood that caregivers rate twins more similarly *across* phenotypically similar behaviour dimensions (i.e., dimensions that have similar operational criteria), may lead to increased likelihood of cross-trait **C** estimates. Most notably, PA is generally considered to be phenotypically similar to CD and indeed shares one similar symptom criterion. The cross-trait **C** effects in the adolescent models that included CD and PA may reflect this overlap. The absence of a similar cross-trait **C** effect in the younger cohort may be due to the significantly lower sample size (i.e,., reduced power) associated with that age group. However, it is noted that cross-trait **C** effects were found for PA in longitudinal models involving the UCF measure in both age cohorts—see Figure 6.2 (p. 263).

The UCF represents a subset of items derived from CD symptom criteria. At first blush, this appears to undermine the use of the UCF as a control for overt aggression. However, there was no overlap in symptom criteria between UCF and PA. This raises the intriguing possibility that PA is indexing an etiologically distinguishable component of the polytypic (or heterotypic) syndrome of CD. Previous studies have suggested subclasses of CD that are distinguishable on the basis of heritability (Eaves et al., 1993; Simonoff et al., 1995). Interestingly, in the current bivariate models involving both PA and CD, all genes associated with CD were shared by PA, while PA showed additional subtype-specific genetic influences. This pattern was true for both younger and older cohorts. Thus, the current results suggest that PA encompasses the latent class (or classes) of CD that show high heritability. Given the finding that genes play an important role in life-course persistent ASB (Moffitt, 2005a), this characteristic renders PA a strong candidate as an important early

marker of persistent CD. Thus, although speculative, this may reflect the parsing of an important clinical phenotypes in a potentially etiologically meaningful way.

It is noted that this explanation of phenotypic similarity does not help explain the cross-trait **C** effects found between PA and hyperactivity in the older cohort, as these two behaviour dimensions are somewhat phenotypically dissimilar. The longitudinal relationships between aggression subtypes and DBDs may shed more light on these complex set of results. These relationships are examined more closely shortly below.

Evidence ascertained from the present analyses provide robust support for the sequential hypothesis, and suggests this theory is a useful reconfiguration of Dodge and colleague's original etiological formulation of RA and PA. Notably, modeling this sequential pathway within two alternative specifications of the putative hyperactivtiy-to-PA risk pathway model generally led to best fitting configurations. That is, with one important exception. Dropping the crucial path between the bivariate **C** component and PA—effectively eliminating the sequential path—improved the model fit by a small amount. However, this most likely reflects the very modest magnitude of the bivariate **C** effects on PA. The large trait-specific genetic component operating on time-2 PA in these pathways models, along with the apparently weak, but robust, **C** effects conferred by time-1 RA (and ODD) raise the possibility of gene-environment interplay suppressing the magnitude of **C** effects on PA and inflating the PA-specific **A** component. It turns out, the sequential pathway implies such interplay effects as discussed in more detail below.

Certainly, consistent patterns within both the RA>hyperactivity>PA and

hyperactivity>RA>PA longitudinal Cholesky models suggest the possibility of two somewhat complementary pathways to childhood PA. The fact that both these pathways were retainable in each of the Cholesky configurations described immediately above is significant, given the expectation that reconfiguration will lead to alternative structural explanations which cannot be resolved via the normal procedures of model fitting and parsimony (Loehlin, 1996).

The first proposed pathway is represented by the unique **C** component linking time-1 RA to time-2 PA. It is possible this trajectory reflects an operant conditioning (i.e., positively reinforced aggression) pathway as specified by the sequential hypothesis. The second putative trajectory is represented by the trivariate genetic component involving all three variables. Specifically, this latter trajectory may reflect a biogenic pathway that implicates a gene set responsible for both RA and hyperactivity at time one, as well as PA at time two.

These separate risk pathways need not necessarily operate independently of one another. In fact, as suggested by the sequential hypothesis, it is likely that the child at high risk of RA is one with an initial biological vulnerability for behavioural disinhibition. This predisposition may give rise to more frequent aggressing and hence more operant learning opportunities. In turn, the more disinhibited child whose needs are (inadvertently) met through aggressing may thus be at higher risk of longer-term conduct problems (Vitaro & Brendgen, 2005). What is being described here is an interaction between biological and environmental risk. As noted earlier, in the classical twin model, GxE interactions and GE correlations are captured in the **A** variance component in the standard genetic model, and are accompanied by a

corresponding under-estimate of **C** effects (Rutter, 2006). Indeed, it was noted that the genetic component for time-2 PA in the relevant sequential pathway models was invariably substantial. It is possible, albeit speculative, that this phenomenon helps explain why bivariate **C** effects, which putatively represent the sequential pathway, demonstrated considerable robustness in the younger cohort data despite exhibiting only modest to marginal impact on time-2 PA.

The above speculation of a dual pathway may also help elucidate findings from the UCF>RA>PA Cholesky model which revealed a bivariate genetic component common to both RA and PA *but not UCF*. Using the logic of Little et al.'s (2003) appeal to distinguish form versus function, this result appears to suggest a unique genetic component underpinning use of aggression as a functional tool. While this may, or may not, be an interesting proposition—a more parsimonious explanation is that a third unmeasured variable may be present that partially underpins both RA and PA but not UCF. The strong and consistent trivariate genetic component linking hyperactivity to both RA and PA would seem to be a reasonable candidate. Testing this hypothesis would require including UCF, hyperactivity, RA and PA within the same model. This would further necessitate a substantially greater sample size than available in the current project. However this constitute an interesting future research project. Certainly, this interpretation of the current findings is broadly supported by an investigation reported by Bennett, Pitale, Vora and Rheingold (2004) who found that while RA-related ASB was more strongly related to with ADHD behaviours than was PA-related ASB (in a childhood clinical sample), the association between PA-related ASB and ADHD behaviours nonetheless increased from middle-childhood to adolescence.

Given the high degree of phenotypic similarity between RA and ODD on the one hand, and on the other, the fact that ODD showed consistent **C** effects across most models (all of which were entirely shared with RA in relevant bivariate models), it was perhaps not surprising to find that the best fitting hyperactivity>ODD>PA model structurally replicated the best fitting hyperactivity>RA>PA (sequential) model. Enforcing a sequential ODD to PA path in the former model resulted in a comparable fit (χ^2=25.74, *df*=32, *p*=.775, AIC=-38.26, RMSEA=.00) although not as strong a set of fit indices as that derived from the sequential specification, i.e., the hyperactivity>RA>PA model, (χ^2=20.88, *df*=32, *p*=.930, AIC=-43.12, RMSEA=.00). Nonetheless, the ODD comparison model had very similar set of path coefficients to the sequential specification.

The fact that ODD appears to replicate similar pathway structures when used to substitute for RA, raises the issue of whether RA is a redundant clinical construct. Firstly, Waschbusch et al. (1997) provided compelling evidence in confirming the clinical utility of RA on the basis that it accounted for a unique proportion of overall impairment and childhood behaviour problems even after simultaneously controlling for *all* other disruptive and aggressive behaviours (including PA, ODD, ADHD and CD). Secondly, and perhaps related, is fact that the bivariate models of RA and ODD (see Figure 5.7, p. 211) show that RA encompasses more extensive **C** influences than ODD which, by contrast, shares all its **C** effects with RA. This suggests RA is more sensitive to a wider range of **C** related influences. If **C** influences are indeed indexing fundamental environmental pathogens responsible for perpetuating high risk ASB pathways, then this may potentially render RA a more effective risk indicator than

ODD. Finally, as demonstrated above, even though ODD replicated the basic pathway structure, it did not demonstrate quite as good a fit as when RA was placed in this model.

Current research also considered the suggestion that impulsive types of aggression, such as RA, result from biogenic mechanisms underpinning impulsivity over and above mechanisms responsible for overt physical aggression (e.g., Barratt & Slaughter, 1998)—as measured by UCF. Findings provide preliminary, albeit modest, support for this latter model with genes representing the only substantial etiological influence linking impulsivity and later RA which is independent of influences operating on overt physical aggression. This indicates impulsivity may be an independent risk factor for RA. This is generally consistent with recent findings that impulsivity and aggression are empirically distinct constructs (García-Forero, Gallardo-Pujol, Maydeu-Olivares & Andrés-Pueyo, 2009). Future research would need to extend this finding by confirming that impulsivity contributes to RA over and above contributions made by hyperactivity.

8.3 Implications for Future Research

A prominent issue currently facing the field of aggression and disruptive behaviour research is how best to refine current clinical phenotypes to better reflect the role of etiological factors. Behaviour genetic research represents a useful empirical tool in attempts to disentangle the myriad of influences impacting on risk pathways.

In broad terms, the current research confirms the importance of considering multiple pathways and etiological influences in the development of aggression subtypes. More

specifically, results reported in this text strengthen previous findings which suggest extant psychosocial models of RA/PA may need to be reconsidered to accommodate the role of biogenic factors such as genes. Certainly, current results are encouraging for a recent sequential model of RA and PA which integrates both biogenic and environmental mechanisms within a single pathway specification (i.e., Vitaro & Brendgen, 2005). However, to the author's knowledge, the current series of studies represents the first to provide empirical data directly relating to this this etiological hypothesis.

The path analyses considered herein not only provide preliminary support for the sequential pathway model, but they also contribute a set of recommendations for future research aimed at consolidating this theoretical specification. In particular, future genetic research might consider the possibility of a GE correlation operating on the putative RA to PA pathway (and PA more generally). Generally speaking, the twin design is poor at disambiguating the effects of gene-environment interplay. Despite their own set of limitations, the adoption design is more adept at disentangling such effects.

The current results further indicate that the sequential pathway is most relevant in childhood, and as such, research efforts might focus particularly on an elementary school age range. At the same time, because the age range of participants was limited to six years (and above), it may prove useful to assess for the evidence of the sequential pathway in a yet younger age range. This is suggested in recognition that early detection represents a core principal driving the evolution of current clinical models of aggression.

In sum, the sequential hypothesis represents a promising developmental model that appears able to integrate a number of core facets of RA and PA research, as well as risk factor research based on clinical phenotypes such as ADHD, ODD and PA. Most notably, the current findings support the proposition that hyperactivity represents a marker for genetic vulnerability within the context of the sequential pathway.

More broadly, the present thesis argues for increased efforts in refining clinical phenotypes via assessing individual symptom dimensions (relating most clearly to ADHD) separately. The current data provide some rationale for doing this by showing distinct genetic architectures differentiating ADHD symptom dimensions (particularly in bivariate models with PA).

At the same time, the current research raised as many questions as it attempted to answer. In particular, results from the genetic modeling undertaken to elucidate the role of sex differences in the etiology of RA and PA suggest more attention is warranted in regards to the possibly non-normative etiological processes underpinning adolescent male RA. The current research suggests these factors may have their origins in shared environmental effects. It is possible this finding points to non-normative (i.e., extreme) environmental conditions impacting on adolescent male RA. However, the relevant effects were relatively small and the result would need to be replicated before additional research is warranted. More broadly, the literature would certainly benefit from focused research evaluating the applicability of categorical approaches to RA and PA versus the benefit of treating these constructs as dimensional. To reiterate (as above), the current research suggest the

possibility that the extreme end of adolescent male RA may be etiologically distinct from adolescent female RA.

8.4 Implications of the Current Findings for Clinical Practice

Increasingly, risk factor research suggests while children with ADHD are at higher risk for long-term antisocial outcomes compared to their non-affected peers, this risk is mediated via physical aggression rather than component ADHD behaviours such as hyperactivity or impulsivity (Broidy et al., 2003). However, much of this research has proceeded without reference to the importance of accounting for heterogeneity in aggression subtypes (Raine et al., 2006).

Although the current longitudinal analyses precluded an independent clinical outcome variable (e.g., over all behavioural impairment), there was strong evidence for etiologically distinct contributions of early RA and hyperactivity to later PA. While future studies may evaluate the independent contribution of these variables to risk for later impairment, enough is known about the role of PA in clinical risk pathways (Pulkkinen, 1996; Vitaro et al., 2002) to suggest that late-childhood PA is a significant risk factor in conduct disordered pathways.

While highly speculative it may be that the clinical value of hyperactivity as a risk marker is obscured by broad antisocial phenotypes (such as CD) that might represent primary predictors of future ASB, but do so by providing a chronicle of the relevant behaviour set across development (Simonoff et al., 1998). From this perspective, using behavioural descriptors to predict future similar behaviour may undermine the task of elucidating interplay between constituent etiological factors that manifest

across divergent phenotypes. In other words, using risk factor approaches alone does little to advance the scientific agenda of establishing valid etiological models to shed light on relevant risk pathways (Hinshaw, 2002).

From this perspective, the current research suggests that the combination of RA and hyperactivity may significantly increase risk for later PA, particularly where there are frequent opportunities in the child's social environment for the inadvertent reinforcement of impulsive reactive aggressive behaviour. Together these factors, may constitute a core set of risk factors that might be assessed for clinical settings. Indeed, this tentative suggestion is unlikely to represent a radical departure from typical clinical practices, and indeed, these features will all typically be identified where relevant in a thorough clinical assessment. Nonetheless, the use of aggression subtypes has not yet permeated the prevailing clinical taxonomies, and the current research generally supports positive expectations that formal recognition of the RA-PA subtypology will provide clinicians with an efficacious tool for disentangling risk-related phenotypes.

From a treatment perspective, it has been suggested that treatments addressing aggression may benefit from cognitive interventions targeting the selective social information processing deficits described by Dodge and colleagues (Dodge, 1991; Merk et al., 2005). For RA, this might include anger management programs that attempt to challenge and ameliorate the hostile attribution bias proposed to maintain this aggression subtype behaviour (Kempes et al., 2005). Conversely, operant techniques (i.e., contingency management procedures) and social skills training may be more effective for PA following the same psychosocial model (Kempes et al.,

2005; Vitiello & Stoff, 1997). In regards to this latter proposition, a recent report emphasises the likelihood that PA-related phenoypes (ie, C/U) may be somewhat resistant to psychosocial treatments (Dadds & Rhodes, 2008). Specifically, a sample of 3 to 8 year old boys with ODD and what the author's describe as a 'cold' temperamental form of aggression (i.e., involving callous-unemotional traits) were less responsive to a standardised parent training intervention (at 6-month followup) than their 'hot' (i.e, assumedly RA) peers. This psychopathy spectrum view of PA has led to suggestions that psychopharmacological interventions may complement psychosocial treatments (Soller, Karnik & Steiner, 2006). In deed such suggestions typically include different pharmacological options for both RA and PA (Miczek, Fish, De Bold & De Almeida, 2002; Soller et al., 2006). For RA, this includes a range of medications that selectively target disinhibition/impulsivity have been suggested in the treatment for RA (Kempes et al., 2005).

There are few studies reporting on the differential treatment efficacy associated with these alternative approaches (Kempes et al., 2005) reflecting the fact that the field of differential treatments for RA and PA is in its infancy. Nonetheless, implications from etiological studies, and genetic studies in particular, are valuable in setting the broad parameters within clinical research can best proceed (Hinshaw, 2002).

The large subtype-specific **C** effects on continuity of PA in the current study would seem to be at odds with suggestions that psychosocial treatments of PA may lack efficacy. At the same time, very strong genetic effects were found for PA. It is probable that current high estimates for genetic effects include some degree of variance associated with GxE interactions or GE correlations. This highlights the

complex interplay between genes and environment. From a clinical perspective, these kind of non-linear patterns are typically consistent with more complex and treatment resistant presentations. On the other hand, the RA models showed that **C** effects were associated with more immediate, rather than long-term cumulative effects. This perhaps raises the prospect that psychosocial interventions on RA may have more immediate short-term benefit relative to PA.

Again, the literature awaits more definitive clinical research. However, progress in clinical research is likely remain slow until the RA-PA subtypology is formally adopted within prevailing nomenclature. As noted throughout this thesis, effective clinical models typically rely on a good understanding of the fundamental etiological architecture of a trait (or traits). This work is ongoing and it is hoped that the current project contributes in some way to broader clinical knowledge regarding the etiology of RA and PA.

8.5 Implications for the Use of Online Technologies in Task-based Psychological Research

The availability of advanced and highly flexible rich media applications has increased dramatically over the past decade and this has seen a corresponding increase in the range of sophisticated data collection techniques available to social scientists. The specific rationale for delivering two online computer-based neuropsychological protocols within a basic endophenotype framework was twofold. Firstly, investigating endophenotypes for elucidating ontogentic processes is becoming increasingly recognised as a useful tool in attempts to integrate multiple levels of explanation for psychopathology (Doyle et al., 2005; Gottesman & Gould,

2003). Secondly, this agenda is defined by the challenge of obtaining task-based data in the quantities necessary for genetic analysis (Haworth et al., 2007). At the time of writing this text, the adaption of laboratory-based neuropsychological tasks for online delivery constitutes a relatively new methodological paradigm. As such, there is currently no established consensus to assist in guiding design and implementation.

The inconclusive set of results derived from the current methodology highlighted a number of issues that have implications for future research in this area. These issue are considered below.

8.5.1 Methodological Issues in Online Delivery of Neuropscyhological Tasks

There are a number of methodological issues that necessitate caution in designing online research. These issues fall into four broad categories;

1. **Access to technologies:** Differential access to technology amongst the wider population impacts on the representativeness of samples and the generalisaibility of findings.
2. **Data integrity / fidelity**: This includes issues such as speed and capacity constraints on data transfer across the internet, as well as the suitability of various data transfer protocols available,
3. **Data security**: This issue pertains to both transfer and storage (with associated ethical implications).
4. **'Client-side' task implementation**: Most notably, remote data collection by definition means the researcher is absent from the testing environment. This

has obvious impications in regards to achieving/ensuring suitable testing conditions.

It is expected that as technology develops and the empirical literature expands, so to will the nature of the various challenges. Nonetheless, the issues identified above sketch out the beginnings of a possible framework for addressing the technical issues involved in designing and implementing psychological tasks online.

8.6 Limitations Associated with the Current Project

There were a number of limitations associated with the current project that warrant caution when interpreting the current findings. Each of these issues is considered in turn.

Firstly, sample size is frequently an issue in genetic research due to the large numbers of participants required to achieve adequate statistical power (Rutter, 2006). The current project was no exception. Indeed the issue of sample size was exemplified by the basic power analyses undertaken for the univariate models of PA. This analysis revealed that even the adolescent cohort of n=1181 did not confer adequate statistical power to retain the **C** effects identified. The current project was the beneficiary of a larger research project undertaken at Curtin University and initial Wave One data collection involved ascertaining behaviour ratings from pencil-and-paper ATBRS questionnaires returned by parents registered with the Australian Twin Registry. Since this initial data collection occasion, additional questionnaires have continued to be mailed out and collected. This augmented data set will potentially afford a means to validate the current analyses with a larger sample size.

A second and somewhat related issue concerns the impact of participants' self-selection into, or out of, the second study. Most notably, there was some evidence to suggest that of the families who agreed to join the second study, those that completed their initial ATBRS prior to June 2006 (i.e., families assigned to the longitudinal analyses) were significantly less likely to have children that showed symptoms of CD. This suggested a certain lack of representativeness in regards to the longitudinal sample. However, because CD is typically conceptualised as an outcome variable and was not measured at the second time point, this scale was not used in any of the longitudinal analyses. More generally, there was a substantial degree of attrition in the second study. At least some attrition is inevitable in a large scale longitudinal study and it appears that this had limited adverse impact on the representativeness of the final sample.

The point raised above regarding CD alludes to a further limitation. That is, neither of the antisocial DBDs (i.e., ODD and CD) were measured in the online data collection Wave. Without this information it was difficult to provide comprehensive evaluations of risk pathways associated with DBDs. Moreover, the addition of a third measure (e.g., child psychopathy screening device) to provide construct validity would have been useful given that both PA and RA shared some items with DBD symptomatology. This latter overlap in symptom criteria represents the challenge of refining clinical constructs and was a necessary feature of the current investigations.

At the same time, the length of time between measurement occasions varied considerably. Specifically, the 9-month window in longitudinal study represented

only the minimum "window" associated with the total range of Study 2a families, some of whom contributed longitudinal data at measurement occasions of up to 12 months apart. This represents substantial variation in such a small overall longitudinal window. In large part, this highlights the fact that this minimum length of time between measurement occasions represented less elapsed time than the typical longitudinal study.

The UCF measure used in the current study was derived from the CD measure putting further restrictions on how CD was able to implemented in the current modeling. To the author's knowledge there is no official measure of UCF and its use as a control measure in RA and PA research has not been well investigated. Given the high phenotypic correlations between PA and CD, the use of UCF in PA models may be less than ideal. Nonetheless, as long as the requirements of devising a UCF measure are met, as they were in the current study, the measure should be able to fulfill its purpose.

To the author's knowledge, the present adaption of the Cholesky Decomposition Model (CDM) is an entirely novel implementation of this design. Due to the absence of literature on this approach, it is difficult to establish any clear indication of the reliability or validity of this method. There has certainly been criticism of the conventional CDM when used without a comprehensive knowledge of its unique structural characteristics and statistical ambiguities (e.g., Loehlin, 1996). Nonetheless, interpretations of results derived from the current hybrid CDM specifications was informed by corroborative evidence from other more conventional classes of genetic models reported in this thesis. Generally speaking, results from the

CDM models were consistent with non-CDM models.

As noted earlier in this chapter, a significant limitation of the classical twin design is its inability to disambiguate gene-environment interplay. There is reasonable theoretical basis for expecting that GE correlations may be operating on the sequential pathway as specified in the trivariate longitudinal Cholesky models reported in Chapter 6. The inability to test for these effects left a number of anomalous results unaddressed. A related limitation is the fact that the twin model of caregiver rated data precludes the capacity to test for the possibility of rater bias. Although rater bias is relatively rare in aggression research, there were a number of unexpected cross-trait **C** estimates that were difficult to interpret and may have been candidates for spurious rater bias.

A common problem with the study of disruptive behavior is the high degree of skew typically associated with the measurement scales. This problem was particularly pronounced for PA which showed very high levels of skew. Normalisation procedures were effective in reducing this skew, but these methods were unable to reduce the skew to the same extent as other scales. At the same time, it was noted that the reliabilities for the RA and PA scales were modest. While the reliabilities obtained for both RA and PA are reasonable for any 3-item scale, previous literature from risk factor research has typically reported much higher estimates than obtained in the current analyses. It is difficult to understand why this might be the case because although the current sample was only moderately sized within the context of genetic research, by comparison to other risk factor studies, the current sample size is considered moderate to large.

A more general limitation of the behaviour genetic approach in the study of aggression is the lack of data on the issue of assortive mating. Assortive mating occurs when two parents show some degree of complementarity for psychopathology (Rutter, 2006). Some have questioned the validity of the assumption of assortive mating in ASB research and there is some data to support this skepticism (Rutter, 2006). What is needed is a better understanding of the impact of assortive mating on ASB and aggression more particularly, in order to better guide genetic research in ways of limiting this impact.

8.7 Final Summary

Historically, the study of aggression has encompassed a broad range of research and theoretical paradigms. Certainly, there is much progress required before a true synthesis can occur across these varied domains. Nonetheless, aggression represents a unique topic of empirical enquiry in so far as there have been few other clinically relevant behaviours that have produced more clearly specified models across more divergent paradigms over such a long time period. To a large extent, this reflects the somewhat unique diversity of the feature set associated with aggression including its likely evolutionary roots (Medina, 2000), and subsequent paradoxical adaptiveness versus non-adaptive consequences when viewed from an ontogenetic perspective.

Behaviour genetic perspectives on aggression offer an important viewpoint in understanding the complexities of this multifarious phenotype. As stated throughout this thesis, one of the most significant challenges for clinical scientists is to find a meaningful way to reduce (that is, classify) heterogeneity in aggression. The research

reported in the current thesis contributes to an expansive and expanding evidence base confirming the relevance of this distinction in the clinical and developmental literatures.

However, the current research further suggests that the notion of differential etiologies for RA and PA alone may not suffice in capturing aspects of the interplay and multifinality that characterise the overlap between these subtypes. Most specifically, the current results support a recent sequential model suggesting early RA may be a precursor to PA (Vitaro & Brendgen, 2005). While this is unlikely to represent an entirely discrete RA/PA pathway, the current research suggests it may provide a clinically useful conceptualisation regarding the high prevalence of co-occurrence.

Most notably, the present findings suggest the possibility that etiological overlap between RA and hyperactivity represents a potential clinical risk indicator associated with the sequential broadening of a constellation of problematic aggressive features associated with PA (for example, callous-unemotional traits). In other words, this research supports speculation that points to RA and hyperactivity as at least partially etiologically distinct but complimentary syndromes that may combine to increase risk for developmental psychopathy. While there is more data needed to confirm the validity of this developmental specification, this avenue of enquiry offers a potential refinement of clinical methods that may eventually prove useful in early detection of problematic antisocial behaviour.

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Every reasonable effort has been made to acknowledge the owners of copyright material. I would be pleased to hear from any copyright owner who has been omitted or incorrectly acknowledged.

Appendix A

Operational criteria for the Psychopathy Screening Device (Frick et al., 2000, p. 454)

Psychopathy Screening Device

Callous-Unemotional Items

1. Is unconcerned about others' feelings
2. Does not feel bad or guilty
3. Is unconcerned about schoolwork
4. Does not keep promises
5. Does not show emotions
6. Does not keep the same friends

Narcissism Items

1. Thinks more important (than others)
2. Brags excessively
3. Uses or "cons" others
4. Can be charming
5. Teases others
6. Becomes angry when corrected
7. Emotions seem shallow

Impulsivity Items

1. Acts without thinking
2. Does not plan ahead
3. Engages in risky activities
4. Blames others for mistakes
5. Gets bored easily

Appendix B

Information Pack: Introduction letter to parents

Page 1

Faces and Cards Study
Request for participation in a Curtin PhD research project

Dear Parents of twins

Thank you for taking the time to fill in the questionnaire about twins' behaviour sent to you by my supervisor, Professor David Hay, National Patron of AMBA. Over the last 3-4 years over 3000 families have taken part and your contributions have provided invaluable information about multiple birth families. I am currently undertaking a PhD at Curtin University (Western Australia) under David's supervision and my particular interest is in how we can obtain novel information about twins using the Internet.

While many families have access to the Internet at home, lots do not. If that applies to you, you do not have to read further but please tick the box (at the end of this letter) to indicate no you do not have access to the web and send back the form so we know it has reached you. Thanks.

Page 2

My research aims to investigate how children perceive emotions on computer-based tasks done at home. This is an exciting new approach to twin studies which has never been tried before. This project has been approved by Curtin University Human Research Ethics Committee and by the Australian Twin Registry. Given all the current concern about computer privacy, we have done everything possible to ensure your family's input is protected.

If you want to take part, what does the research involve?
We will be providing a CD-ROM that allows the families, who are connected to the net, to log in and complete three online activities. Yes, we know this seems funny, but the files are large and difficult to download directly in a form easily accessible on all computers. The first activity, or task, is a simple 24-item electronic questionnaire that parents are asked to fill out and takes approximately 10 minutes to complete. Secondly, each twin is asked to complete two computer tasks. Twins attempt the tasks individually with each task taking about 10 minutes to finish. The tasks require no special computer skills. Both the tasks and the questionnaire will run on any home computer that has an internet connection. Please see the accompanying information sheet for more details about individual tasks.

What do you get out of it?
This is the first trial of getting Internet data from multiple birth families in Australia. The UK has some large studies of twin children but there they can visit each family as distances are so much less. Will the Internet approach work in Australia where so many more families have access, irrespective of distance? As some recompense for your time and especially that of your twins, we are offering family packs of six movie tickets but not to every family! If the data are completed, you will go in a draw where about one family in 20 will get the movie pack

How do I sign up?
To sign up to the study, you simply go to following website…

www.facesandcards.com

…where you will find an online consent form. Completing this short electronic form automatically registers the family and they will be sent out a CD-ROM in the mail. You will also receive a login name and password by email. Once the CD-ROM arrives, simply connect to the internet, put the CD-ROM in your computer and follow the instructions. As the family completes the questionnaire and tasks, the data is sent automatically to our secure database at Curtin University via the web.

What happens to the information that you put in the online consent form?
As you can see when you go to **www.facesandcards.com**, the online consent form asks you to provide the following information: Your first name and the first names of your two twins as well as an email address that I can send your username and password to. When you submit this online consent form, your information is sent to a secure database and a corresponding email is automatically sent to me. I will check that the information submitted to the database matches the email and then finally I will cross check this information with our existing database records from your previous involvement with the Curtin research team. Once this check is completed the email is discarded. After this initial sign-up process, your first name will be omitted from the database. The twins' first names are maintained in the secure database for the duration of your family's involvement in the study so that we are able to keep track of who has completed what tasks.

Page 3

Finally, it is important to emphasise that you and your children may withdraw from the study at any stage. The research data gathered from the results of this study will be published but will be based on the hundreds of families involved, so that no child or family can ever be identified. If any aspect of the study concerns your family before or during involvement in the research, please contact the Ethics Office at Curtin on 08 9266 2784 or by email for human ethics hrec@curtin.edu.au

There is also an information page on the **www.facesandcards.com** website that answers common questions that families may have about the study. Please feel free to visit this site to get more information.

Thank you for your time,

James Dent
Ph: (08) 9266 3575
james.dent@postgrad.curtin.edu.au

Please tick the relevant check boxes and send back this letter if your family does not have access to the internet or if you do not wish to take part in the study.

- ☐ **Our family does not have access to the internet**
- ☐ **Our family does not wish to take part in the Faces and Cards Study**
- ☐ ***(Optional)* Our family does not wish to be contacted about involvement in future studies undertaken by David Hay and the research team at Curtin university**

Appendix B continued

Information Pack: Letter explaining the study

Page 1

About the Study:
What is the Faces and Cards Study?

Introduction
Thank you for showing interest in the Faces and Cards Study. This study is being undertaken at the School of Psychology, Curtin University in Western Australia as part of a broader research project made possible by the very generous support of Aussie twins and their families. In this study we are hoping to find out what twins can tell us about the way behaviour is related to recognising emotions and decision making.

What happens in the study?
In this study, twins are asked to complete two tasks which take around 10 minutes each to finish. Tasks are completed by each twin one at a time. A quiet and distraction-free environment is very important. So, as much as possible, parents or guardians are encouraged to ensure that only they and the twin completing tasks are in the room when tasks are being attempted

There is also a short questionnaire for parents or guardians to fill out. This 24-item survey can be completed at any time before or after the twins have finished the two tasks

If a family starts the study do they have to finish?
This is a voluntary study so any family member can choose to cease involvement in the study at any time. However, to show appreciation for the time taken to finish the study, families who complete all tasks along with the questionnaire will automatically go into the running to win one of 20 family movie packs (movie tickets for 2 adults and 4 kids).

Do all tasks have to be completed at once?
Tasks can be attempted one at a time, whenever it is most convenient for the family. A twin can finish one task, shut the program down and then return later to log in again to start the other task. However, each task can only be attempted once. If your child starts a task but then chooses to stop the task **before it is completed**, unfortunately they cannot return to continue with that task later. However, they are welcome to return to complete the other task later if they have not already started this second task. If for some reason your internet connection is lost in the middle of completing a task, or something else goes wrong with your computer while doing one of the tasks. Don't

Page 2

worry, please email me using the links within the task interface (or below) and I will contact you ASAP to reset the task.

Can I help my children complete the tasks?
Depending on the age of the twins, parents or guardians may need to assist their children to log in and prepare for the tasks. Parents may also need to read through the task instructions with their child so they can help answer any questions that arise. However, once the child has started a task, it is important that mum, dad or guardian does not offer the twin any help in choosing responses to the task questions. By the same token, although every effort have been made to make these tasks as engaging as possible for the twins, they probably will benefit from a little encouragement to stick through to the end of each task

Is the Faces and Cards website secure?
We have installed industry standard encryption software on the Faces and Cards website. This software is called a Secure Socket Layer or **SSL** for short. This means, when you send us personal information using the online consent form, and also when you send data to our database during the study, all of the information is encrypted. This brings peace of mind because if the information is intercepted by another computer when in transmission between your computer and our server, it will not be decipherable or usable.

Please email me at james.dent@postgrad.curtin.edu.au if you have any further queries about the study or the processes involved

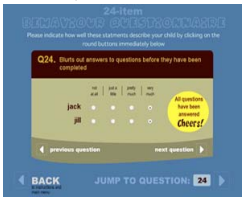
Appendix B continued

Information Pack: Letter explaining the online questionnaire and tasks

The faces and Cards Study

Explanations of the questionnaire and 2 tasks

THE QUESTIONNAIRE



Time: 10 minutes

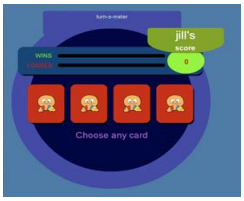
The 24-item behaviour questionnaire which parents or caregivers fill out online is similar in style to the original Australian Twin Behaviour Questionnaire except it's a shorter version. It asks parents to rate 24 different behaviours for both their twins and takes about 10 minutes to complete.

THE FACES TASK

Time: 10 minutes per child

The Faces Task is called ***Multi Morph***. The child doing the task is shown a series of faces, one at a time. When the START button is pressed, the face displayed begins to change to show one of six emotions. The child's job is to guess what emotional expression the face is changing into before it stops morphing!

THE CARDS TASK



Time: 10 minutes per child

The Cards Task is called ***Hit the Decks***. The child doing this task gets to choose cards, one at a time, from any one of four card decks (see picture on the right). Each card is worth a certain amount of points. However, some cards also result in points being taken away. The aim is to score as many points as possible in 100 turns while trying to avoid the penalty cards!

Step-by-step instructions are included in the CD-ROM that you will receive.

For more information please visit the website **www.facesandcards.com** or contact me at …

james.dent@postgrad.curtin.edu.au
Ph: 9266 3575

Appendix B continued

Information Pack: Download instructions

Page 1

Download Instructions:
Getting started with the Faces and Cards Ta

Instructions for downloading the software
If you chose to download the Faces and Cards software you will receive an email that includes a username, a password and link to download the software. Follow the instruction below to complete the download:

1. Double click or right-click (ctrl-click for macs) on the link inside the email to download.
2. If you are prompted to save the file onto your hard drive, choose a place that is easy to find like your desktop. If you cannot find the file once it has been downloaded, try using your computer's search program to find it. For Windows users, the search program can be found by clicking on the **Start** (menu) > **Search** > **For Files or Folders…** > **All files and folders** option and then entering the name **tasks.zip** into the top search field
3. Once you have downloaded and located the **tasks.zip** on you hard drive you need to make sure it is unzipped. In most cases the computer will automatically unzip the file once it has finished downloading. If it did not do this, you can do it manually by simply double clicking the **tasks.zip** file to unzip it.
4. When the file is unzipping, Windows users may be prompted with a **compressed (zipped) folders warning**. If so, you should choose the **extract all files** option and follow the prompts.
5. You will know when the file is unzipped because you will see a folder called **tasks** on your hard drive
6. You are now ready to follow the instructions on the next page which explain how to run the tasks

Page 2

Instructions for starting up the tasks
Once you have downloaded the tasks you are ready to go. Follow these instructions when you want to log on and start completing the tasks:

1. Make sure you are connected to the web
2. Now double click on the file that is named **startup** which is found in the downloaded **tasks** folder
3. This should start up the task interface. Some Windows users may get a warning saying the **publisher could not be verified**. If this happens, simply choose the **run** option to start the task interface.
4. Use the username and password I have emailed you to log into the interface

NB If you have security software or a firewall operating on your system, you may be asked to **allow** or **deny** the software access. Please choose **allow**. If you are unable to connect you may want to check your firewall settings. Also the task has not yet been tested in Vista the new Windows operating system.

Please report any issues relating to these or any other problems at
james.dent@postgrad.curtin.edu.au

Appendix C

Online parental consent form

The Faces and Cards Study

Sign Up

Submitting this form confirms consent to take part in the faces and cards study. Please send us any questions concerning this online registration process using the link below.

Online Consent Form

Parent/caregiver's FIRST name ONLY:

I have read the information explaining the study entitled **The Faces and Cards Study**. I have understood the information given to me. Any questions I have asked have been answered to my satisfaction.

I agree to allow my children,

First name ONLY of the first twin sibling:

First name ONLY of the second twin sibling:

to participate in the study. I understand my children may withdraw from the study at any stage and withdrawal will not interfere with routine care. I agree that research data gathered from the results of this study may be published, provided that names are not used.

Please email the necessary log-in information to me using the following email address

Your e-mail address:

Please indicate whether you are happy to be contacted regarding future studies undertaken by David Hay and his research team at Curtin:

Agree Disagree

Sign Up!

NB You must be registered with the ATR to take part in this study

Appendix D

Zygosity questionnaire included in the Australian Twin Behaviour rating Scale (Levy et al., 2001)

ZYGOSITY

The purpose of these questions is to determine whether your twins are genetically identical (formed from the splitting of one fertilised egg) or genetically non-identical (formed from the fertilisation of two eggs). If your twins are **not** the same sex as each other, please go to the medication questions (**Question 25**).

22. I believe the twins to be:
- ☐ Genetically identical (one egg, monozygotic)
- ☐ Genetically non-identical (two eggs, dizygotic)
- ☐ Not sure

If your twins are of the same sex, and you have had their zygosity determined by blood or DNA test, please answer the following questions: (if they have not been tested, please go to Question 24)

23 A. The test found the twins to be: genetically identical ☐
genetically non-identical ☐

B. What was the test used?

24. To what extent are the twins similar at this time for the following features	**Not at all similar**	**Somewhat similar**	**Exactly similar**
A. Height	☐	☐	☐
B. Weight	☐	☐	☐
C. Facial Appearance	☐	☐	☐
D. Natural Hair Colour	☐	☐	☐
E. Eye Colour	☐	☐	☐
F. Complexion	☐	☐	☐

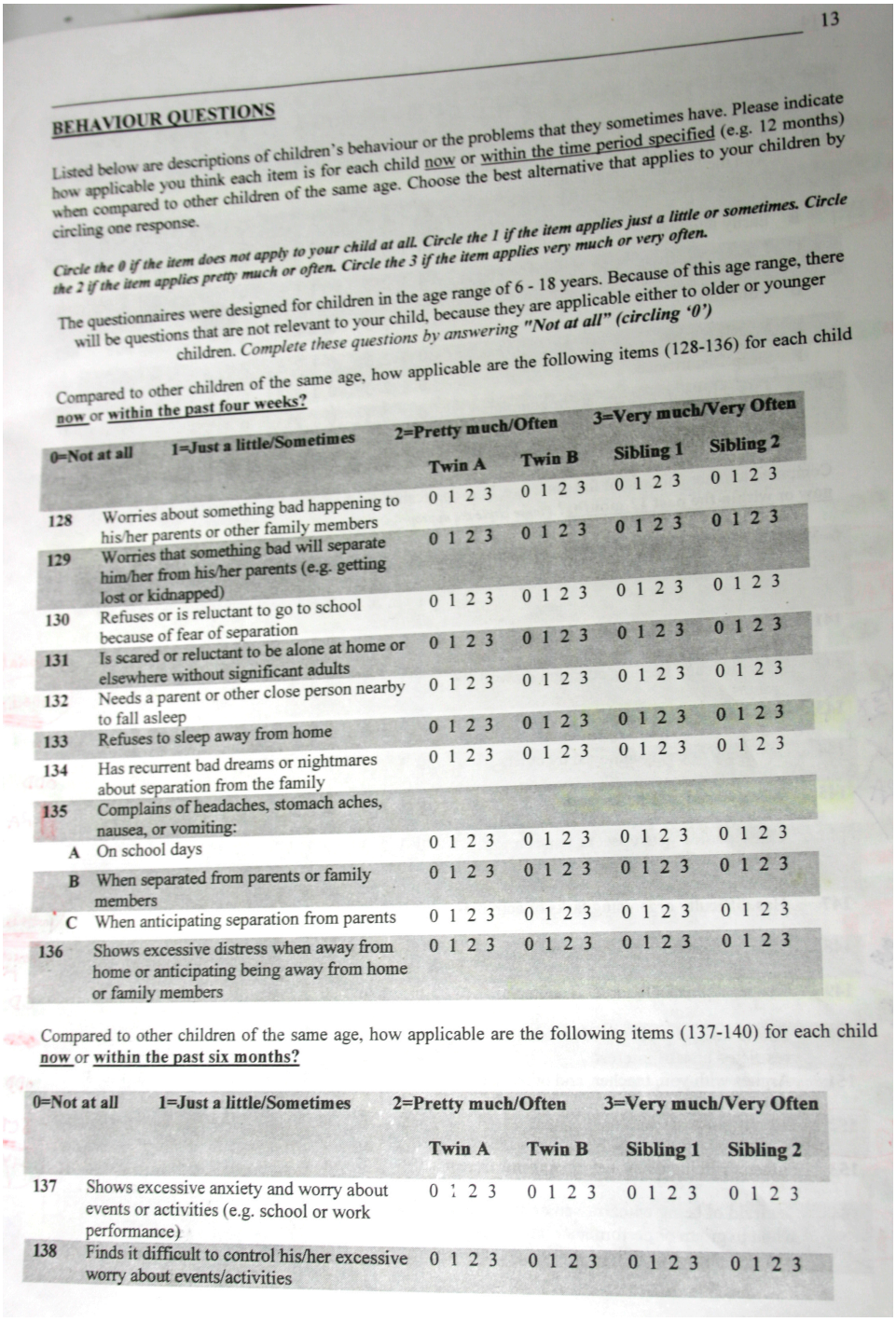
	Yes	**No**
G. Do they look as alike as two peas in a pod?	☐	☐
H. Does their mother ever confuse them in appearance?	☐	☐
I. Does their father ever confuse them in appearance?	☐	☐
J. Are they sometimes confused in appearance by other people in the family?	☐	☐
K. Is it hard for strangers to tell them apart?	☐	☐
L. Do they have very similar personalities?	☐	☐

6

	Yes	**No**	**Don't Know**
M. Did they have the same placenta?	☐	☐	☐
N. Do they have the same blood group?	☐	☐	☐

Appendix E

Example page of the Australian Twin Behaviour Rating Scale (Levy et al., 2001)



13

BEHAVIOUR QUESTIONS

Listed below are descriptions of children's behaviour or the problems that they sometimes have. Please indicate how applicable you think each item is for each child now or within the time period specified (e.g. 12 months) when compared to other children of the same age. Choose the best alternative that applies to your children by circling one response.

Circle the 0 if the item does not apply to your child at all. Circle the 1 if the item applies just a little or sometimes. Circle the 2 if the item applies pretty much or often. Circle the 3 if the item applies very much or very often.

The questionnaires were designed for children in the age range of 6 - 18 years. Because of this age range, there will be questions that are not relevant to your child, because they are applicable either to older or younger children. *Complete these questions by answering* ***"Not at all" (circling '0')***

Compared to other children of the same age, how applicable are the following items (128-136) for each child **now** or **within the past four weeks?**

0=Not at all	1=Just a little/Sometimes	2=Pretty much/Often		3=Very much/Very Often	
		Twin A	Twin B	Sibling 1	Sibling 2
128	Worries about something bad happening to his/her parents or other family members	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3
129	Worries that something bad will separate him/her from his/her parents (e.g. getting lost or kidnapped)	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3
130	Refuses or is reluctant to go to school because of fear of separation	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3
131	Is scared or reluctant to be alone at home or elsewhere without significant adults	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3
132	Needs a parent or other close person nearby to fall asleep	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3
133	Refuses to sleep away from home	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3
134	Has recurrent bad dreams or nightmares about separation from the family	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3
135	Complains of headaches, stomach aches, nausea, or vomiting:				
A	On school days	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3
B	When separated from parents or family members	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3
C	When anticipating separation from parents	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3
136	Shows excessive distress when away from home or anticipating being away from home or family members	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3

Compared to other children of the same age, how applicable are the following items (137-140) for each child **now** or **within the past six months?**

0=Not at all	1=Just a little/Sometimes	2=Pretty much/Often		3=Very much/Very Often	
		Twin A	Twin B	Sibling 1	Sibling 2
137	Shows excessive anxiety and worry about events or activities (e.g. school or work performance)	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3
138	Finds it difficult to control his/her excessive worry about events/activities	0 1 2 3	0 1 2 3	0 1 2 3	0 1 2 3

Appendix F

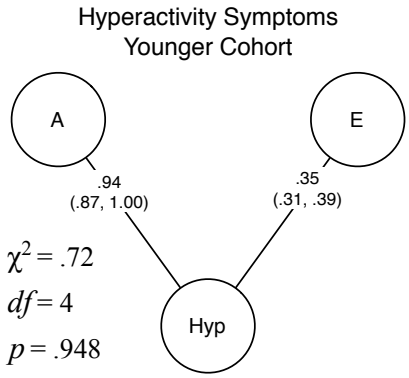
Table of variances for all scales by zygosity

		RA	PA	Hyp	Imp	Ina	ODD	CD	n range
Raw data									
Younger Cohort	MZ	2.63	0.45	10.07	2.63	17.16	**6.82**	2.04	359-374
	DZ	2.99	0.54	8.51	2.96	13.31	**10.21**	1.98	436-458
Older Cohort	MZ	3.44	**0.72**	**5.03**	2.48	15.05	**11.12**	**4.52**	1342-1442
	DZ	3.58	**0.86**	**7.01**	2.72	17.44	**12.69**	**6.81**	1624-1760
Normalised data									
Younger Cohort	MZ	2.73	0.69	7.02	**2.34**	15.69	**8.22**	4.42	346
	DZ	2.99	0.69	7.09	**2.69**	14.53	**10.43**	4.33	436
Older Cohort	MZ	3.51	**0.71**	**5.98**	2.63	15.99	11.38	**4.58**	1340
	DZ	3.53	**0.81**	**7.35**	2.75	16.47	11.64	**5.43**	1618

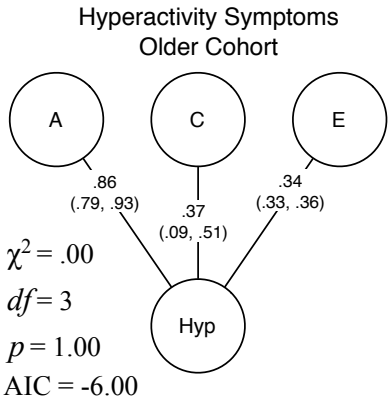
MZ=Monozygotic twins, DZ=Dizygotic twins, RA=reactive aggression, PA=proactive aggression, Hyp=hyperactivity, Imp=Impulsivity, Ina=Inattention, ODD=oppositional defiant disorder, CD=conduct disorder, n range=range for number of participants in each MZ/DZ comparison per scale. Embolded statistics denote MZ/DZ variances determined to be significantly different using Levene's test at p=.05

Appendix G

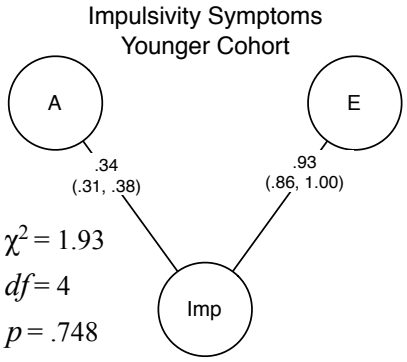
Univariate twin models for ADHD Symptom Dimensions



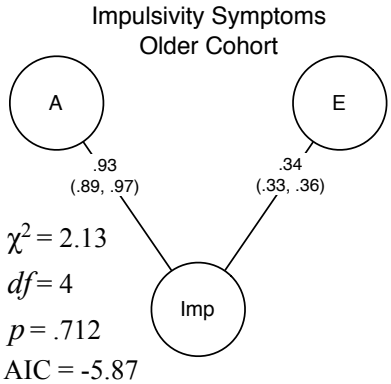
$\chi^2 = .72$
$df = 4$
$p = .948$
AIC = -7.27
RMSEA = .00



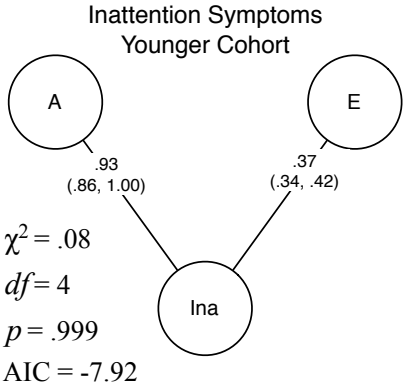
$\chi^2 = .00$
$df = 3$
$p = 1.00$
AIC = -6.00
RMSEA = .00



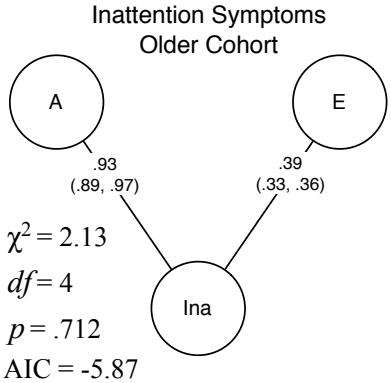
$\chi^2 = 1.93$
$df = 4$
$p = .748$
AIC = -6.07
RMSEA = .00



$\chi^2 = 2.13$
$df = 4$
$p = .712$
AIC = -5.87
RMSEA = .00



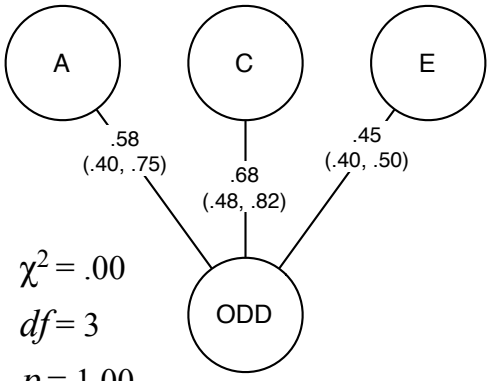
$\chi^2 = .08$
$df = 4$
$p = .999$
AIC = -7.92
RMSEA = .00



$\chi^2 = 2.13$
$df = 4$
$p = .712$
AIC = -5.87
RMSEA = .00

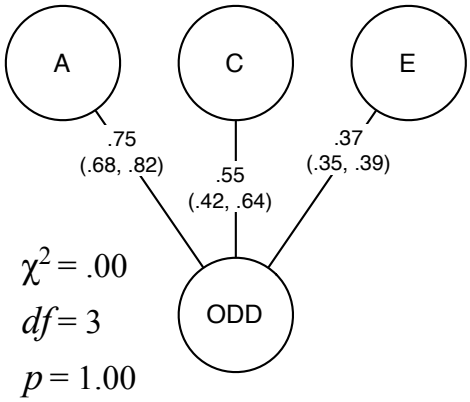
Univariate twin models for ODD and CD Symptom Dimensions

Oppositional Defiant Disorder Symptoms
Younger Cohort



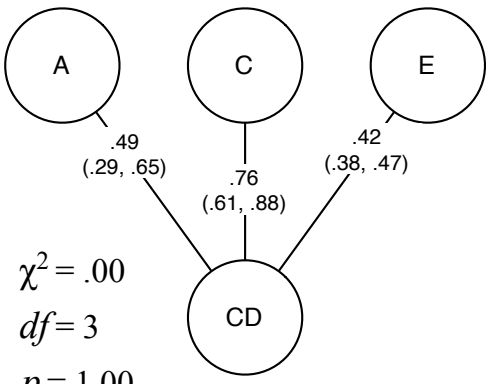
$\chi^2 = .00$
$df = 3$
$p = 1.00$
AIC = -6.00
RMSEA = .00

Oppositional Defiant Disorder Symptoms
Older Cohort



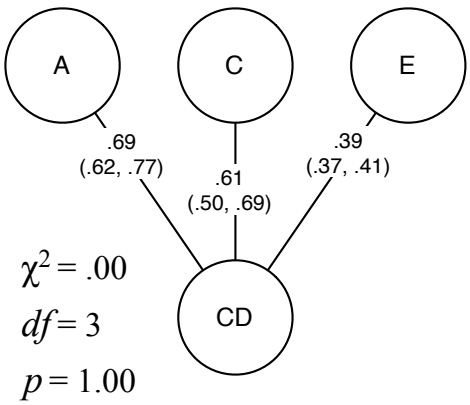
$\chi^2 = .00$
$df = 3$
$p = 1.00$
AIC = -6.00
RMSEA = .00

Conduct Disorder Symptoms
Younger Cohort



$\chi^2 = .00$
$df = 3$
$p = 1.00$
AIC = -6.00
RMSEA = .00

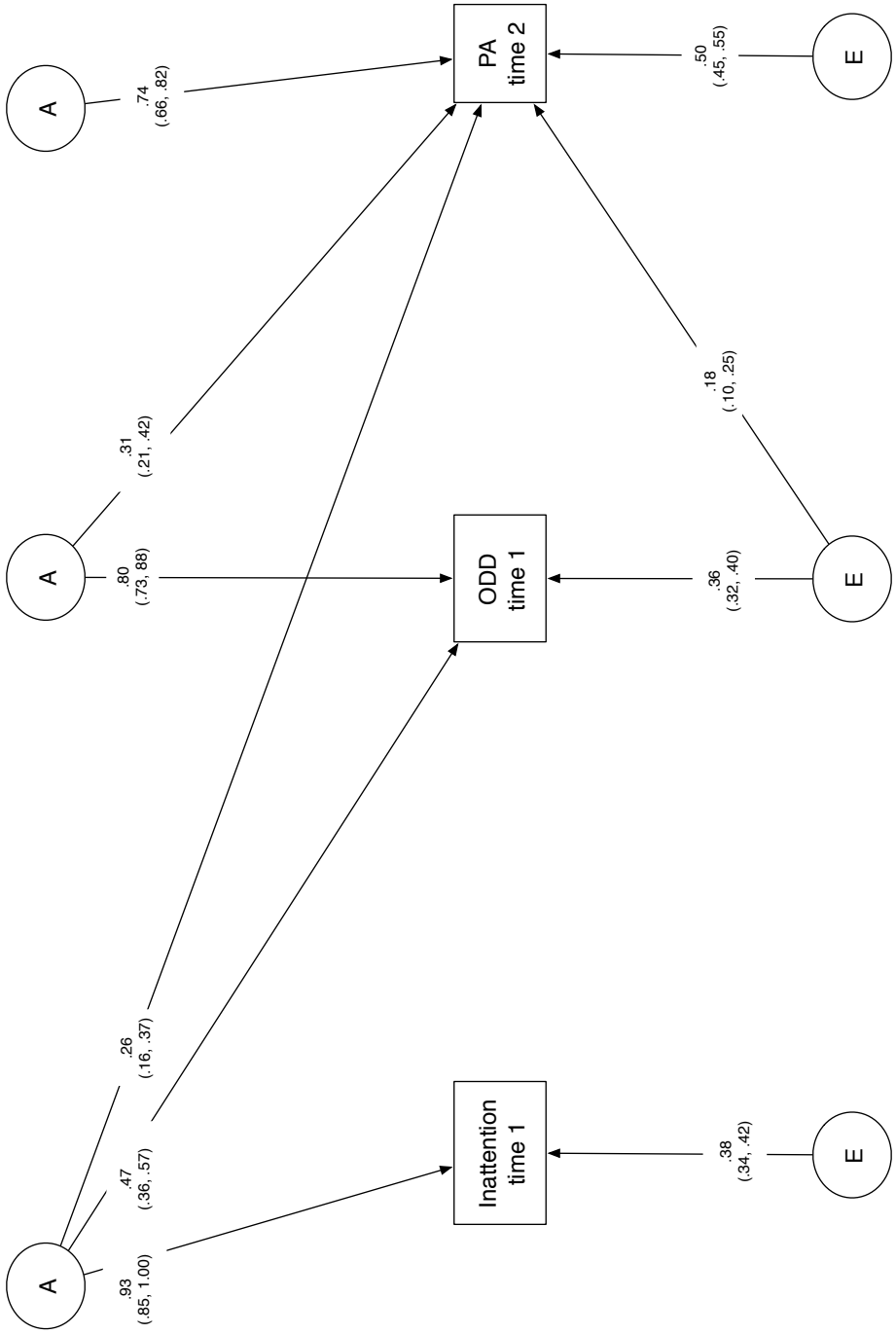
Conduct Disorder Symptoms
Older Cohort



$\chi^2 = .00$
$df = 3$
$p = 1.00$
AIC = -6.00
RMSEA = .00

Appendix H

Longitudinal trivariate Cholesky model involving inattention, ODD and PA symptoms for the older cohort



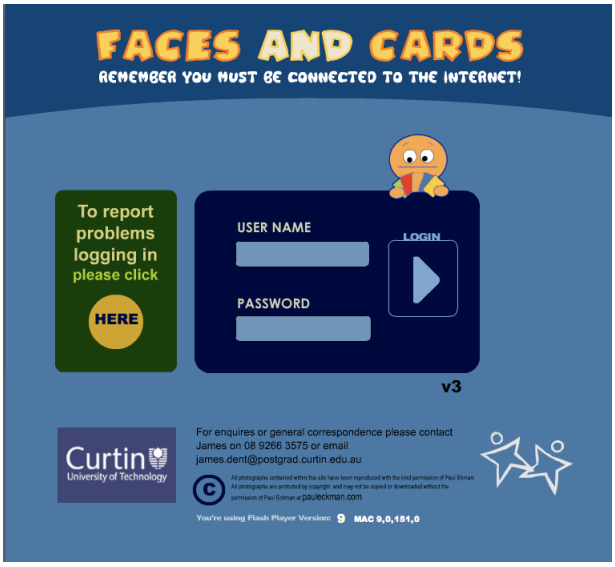
$\chi^2 = 29.25 \quad df = 32 \quad p = .606 \quad \text{AIC} = -34.75 \quad \text{RMSEA} = .02$

Appendix I

Screen shots of the user interface software developed for delivery of online questionnaire and neuropsychological tasks

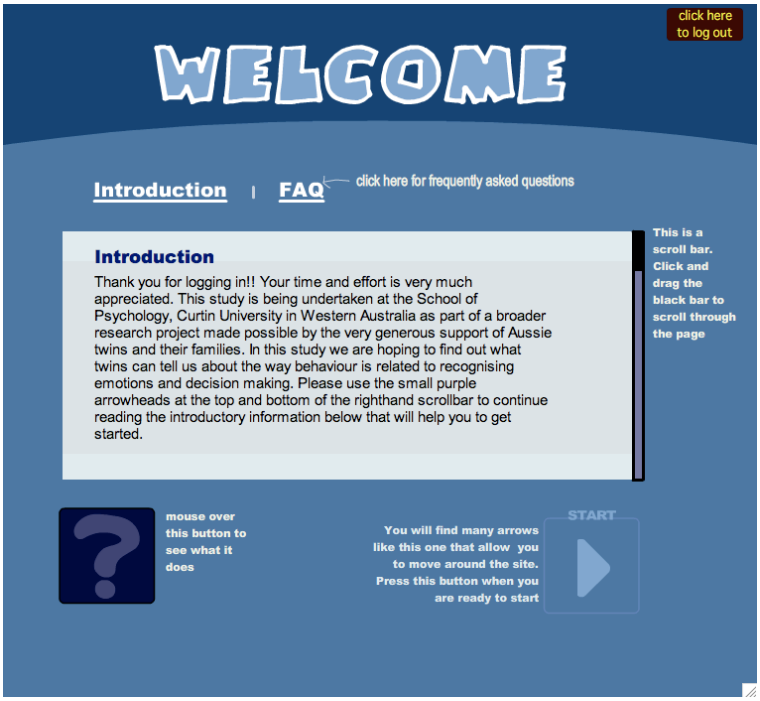
Login screen

This is the first screen families saw when starting the online user interface software. Care-givers were supplied with a username and password by email which was stored in an on-line secure (MySQL) database protected by a Secure Socket Layer.

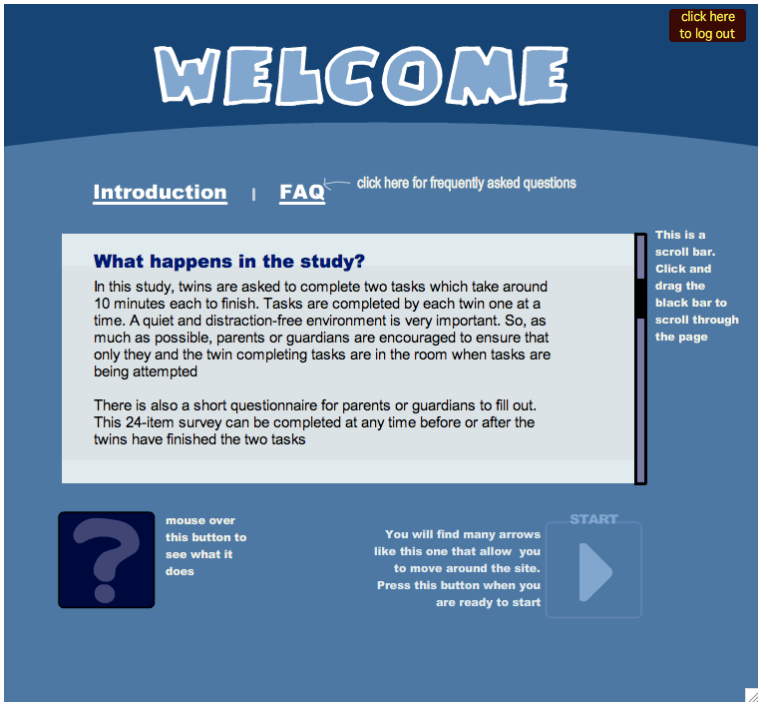


Welcome screen 1

Instructions for the study were always available once logged in. Users could scroll down to (re)familiarise themselves with an overview of the study. These instructions are displayed over the next couple of screen shots.

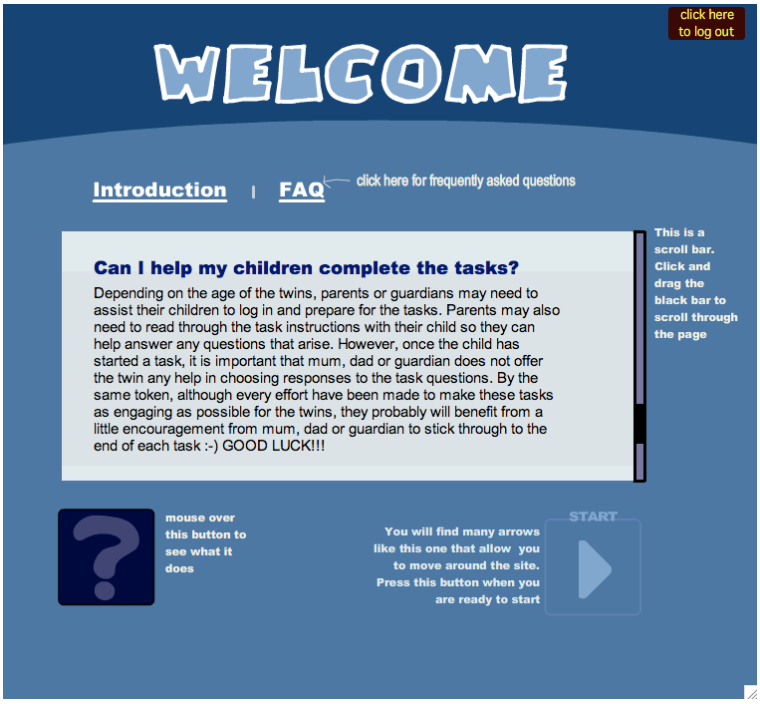


Welcome screen 2

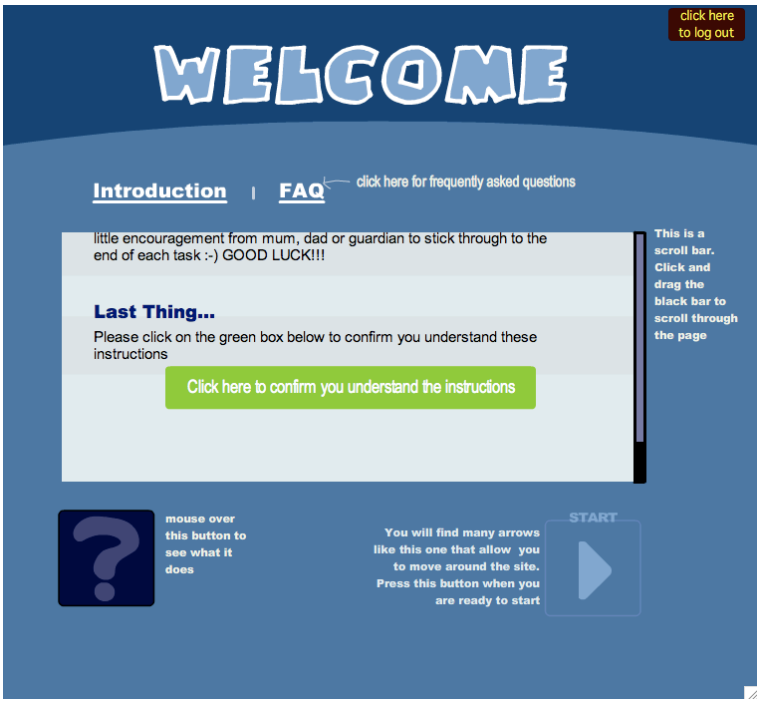


Welcome screen 3

Welcome screen 4



Welcome screen 5

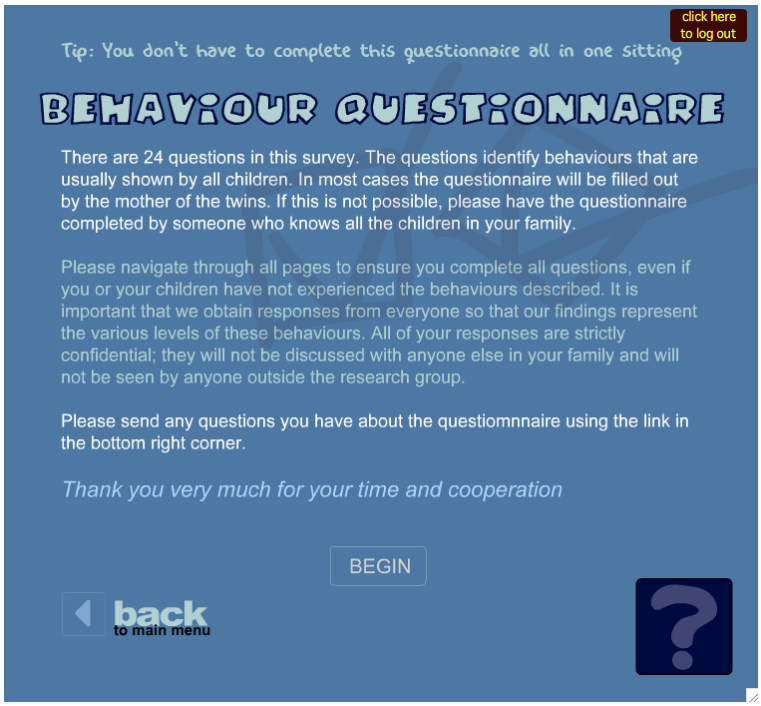


Main menu screen

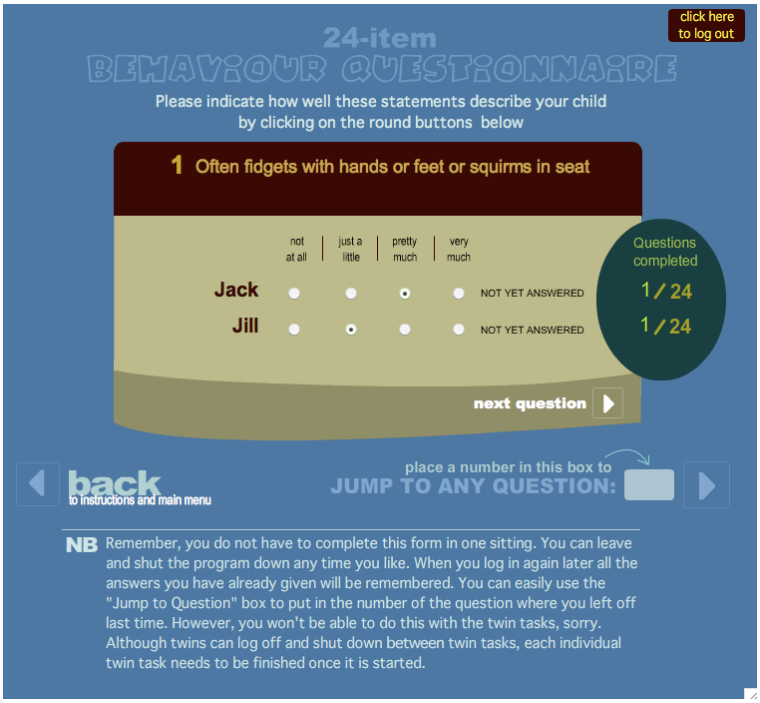
AT any point after logging in to the user interface software, the user can link straight to the main menu where caregivers can access the behaviour questionnaire, and/or the twins can access the two neuropsychological tasks.

Introduction to behaviour questionnaire (AT-BRS-ES)

Linking to the caregiver behaviour questionnaire presents the user with instructions for using the questionnaire.



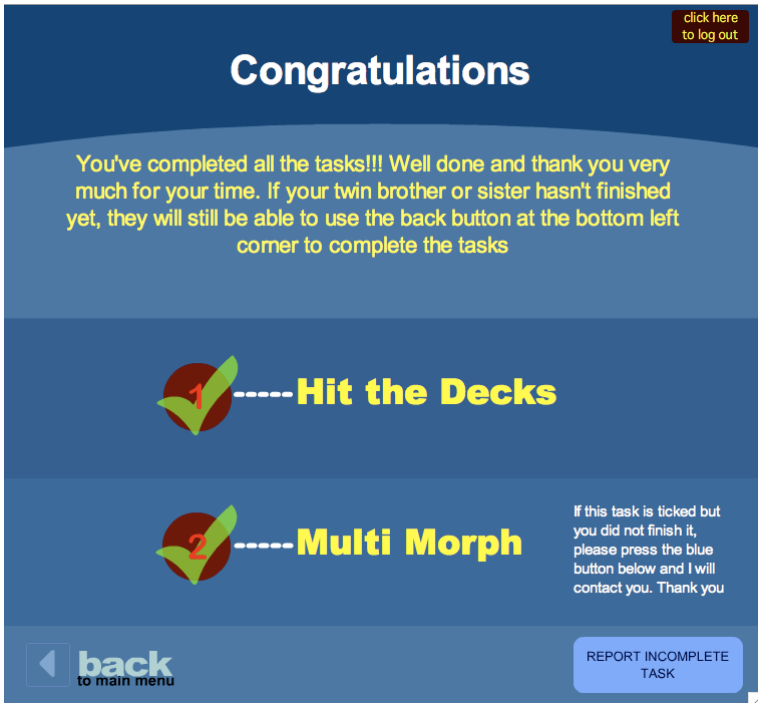
Example page from AT-BRS-ES



Neuropsychological tasks page

Linking to the neuropsychological tasks page presents the user with the task manager page. Prior to linking to this page the user is asked to identify which twin they are and is also asked (1) whether they are doing the task with or without their caregiver present and (2) if the room they are in is quiet. Once these questions are answered they link to the page to the right. The order of tasks is randomised for each twin and the user is directed to which task to complete first (as seen in the picture to the right).

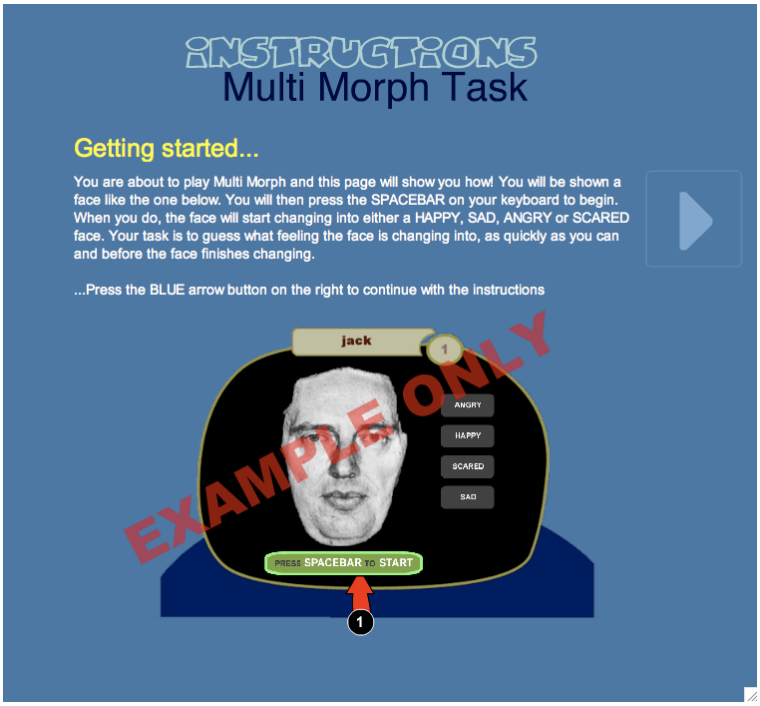
Congratulations screen: Neuropsychological tasks



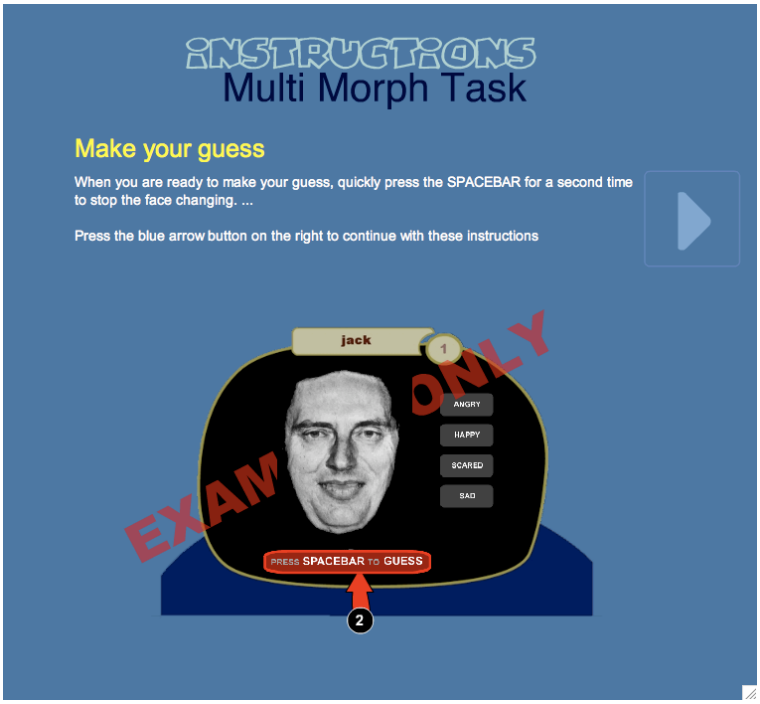
Certificate of completion screen

Multimorph instructions 1

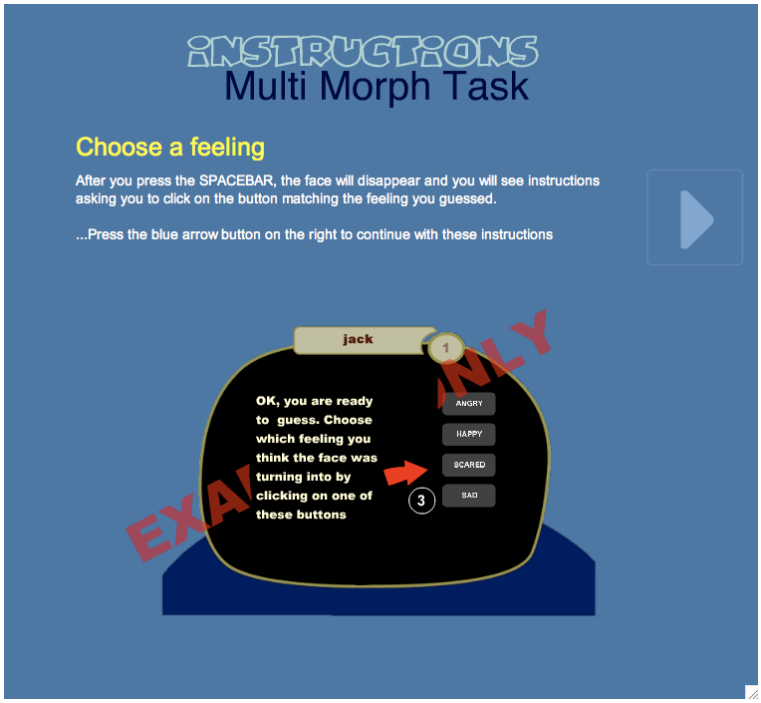
Instructions for Multimorph as presented to the user are shown here in the following series of screenshots.



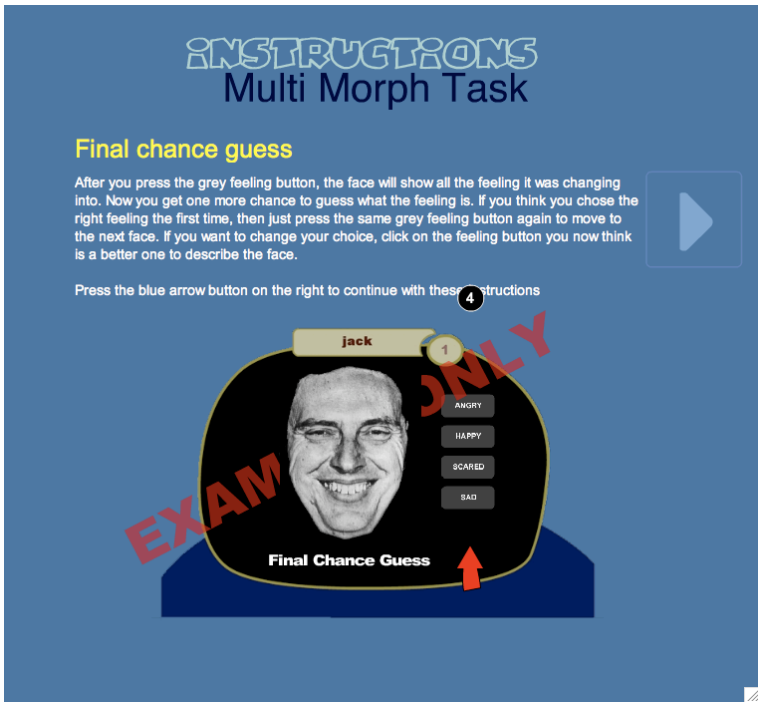
Multimorph instructions 2



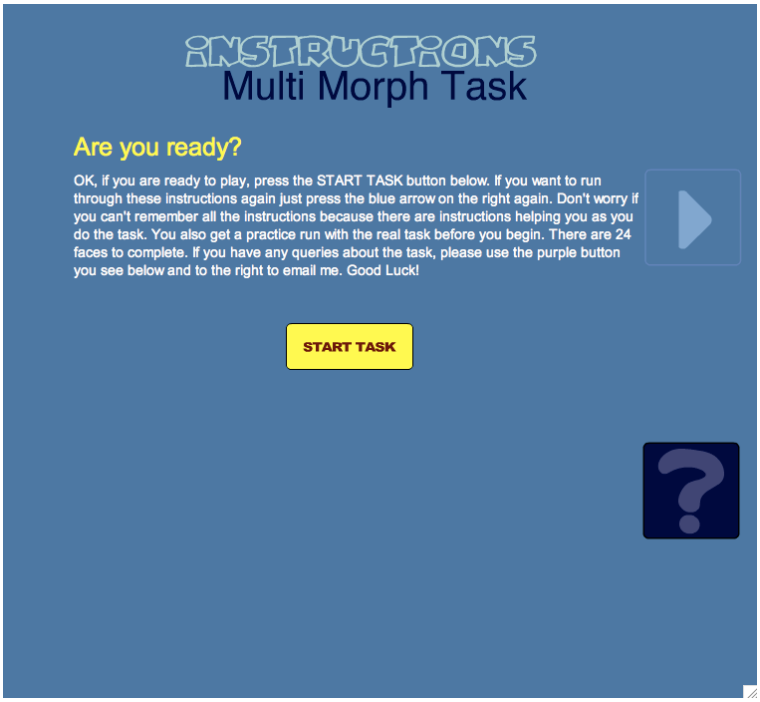
Multimorph instructions 3



Multimorph instructions 4

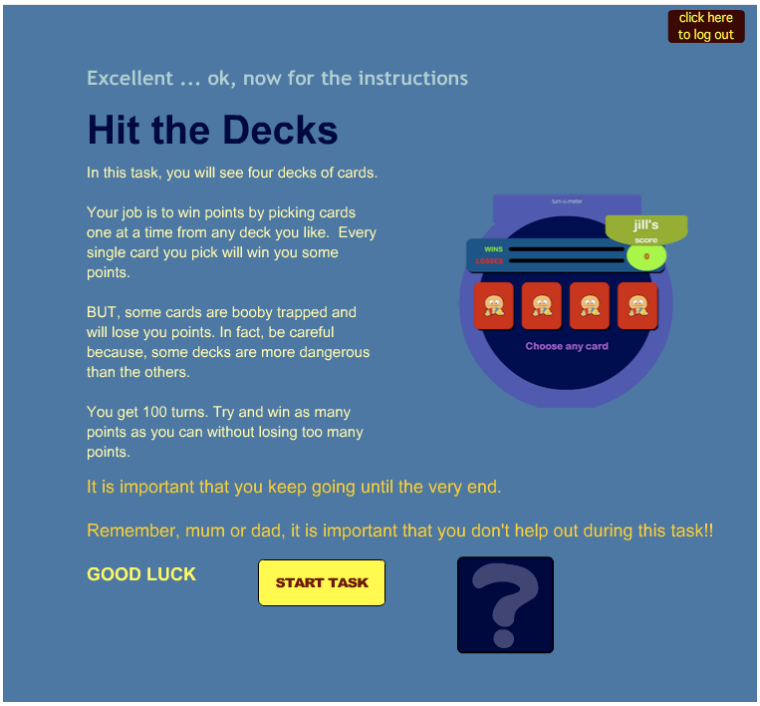


Multimorph instructions 5



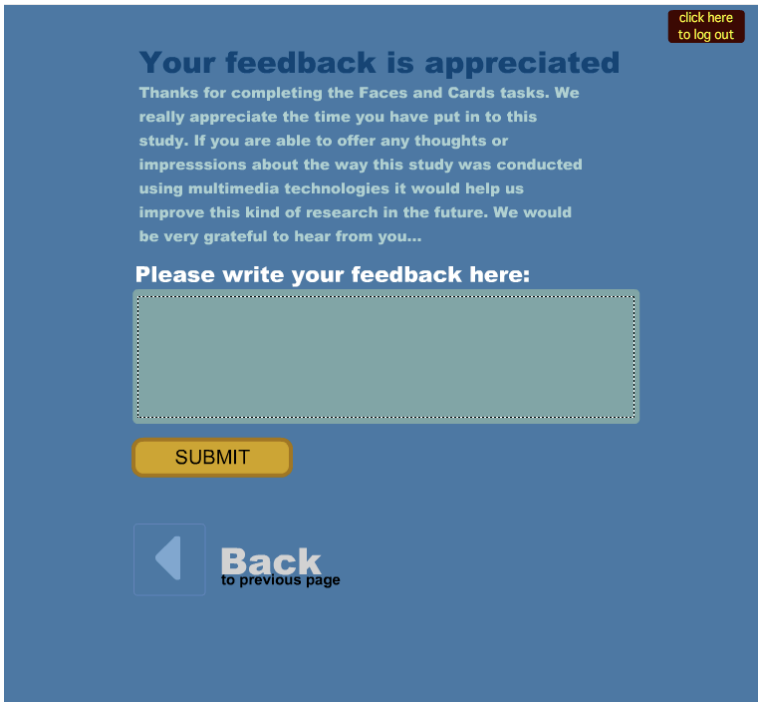
Iowa Gambling Task instructions

Instructions for Iowa Gambling Task, which was named Hit the Decks" in the current study, is shown to the right.



Congratulations for completing study screen

Feedback screen



Example error screen