Using Childhood Activity and Sleep to Predict Emotional and Behavioural Difficulties at Age 11

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Abstract

This study utilised data from the Auckland Birthweight Collaborative (ABC) longitudinal study. The ABC study was established to compare the developmental trajectories of full-term children born small for their gestational age (SGA), with their average birthweight peers (appropriate for gestational age; AGA). Data has been collected at birth, and at the ages of 1, 3.5, 7, and 11. At each phase extensive amounts of physical, psychological, developmental, and familial data have been collected from the children and their families. Central to the current study, at the ages of 7 and 11 the children wore an Actigraph monitor, providing an objective measure of their daytime activity and sleep duration. At each age, two childhood behavioural questionnaires were also administered, one used commonly to screen for Attention Deficit Hyperactivity Disorder (ADHD), and the other for more general psychological difficulties; the Conners' Rating Scale (CRS) and the Strengths and Difficulties Questionnaire (SDQ) respectively. Data was collected successfully for 540 children at both ages across these measures.

Changes in activity and sleep are acknowledged to be indicators of over 30 psychological disorders. This study sought to examine whether objective measures of activity and sleep duration can be used to predict emotional and behavioural difficulties in 11 year old children, as measured by the CRS and the SDQ. This was with the overall aim of exploring whether activity and sleep duration measures may have diagnostic utility beyond more established risk factors. Given that accelerometers in smart phones now offer the opportunity to measure these variables in real world settings, such findings would suggest that these technologies could be used to inform clinical psychological assessment. It was also hoped to examine how accurately symptoms of motoric hyperactivity are identified by subjective behavioural measures.

The literature review explores the role of the two questionnaires in a New Zealand context, and the technical feasibility of using activity and sleep measures diagnostically. It first identifies the central and stable contribution of activity to a child's temperament, noting that positive associations with activity reverse as children get older. While concern is often focussed on the excessive activity associated with externalising disorders, it is found that activity is also a valuable predictor of internalising disorders. The evolving nature of the ADHD diagnosis is explored, noting the reduced emphasis placed on hyperactivity in recent years. Broader controversies around the diagnoses are then introduced, highlighting an arguably premature move away from the consideration of psychosocial aetiological factors. An overview of the research techniques used to measure activity and sleep is then presented, including how well these measures can distinguish subtypes of ADHD, conduct disorder, and internalising disorders. A more detailed consideration is given to why the role of genetics in ADHD may have been overestimated at the expense of environmental factors, before the environmental and psychosocial covariates utilised in this study are introduced.

Data screening highlighted that compared to the original ABC sample at birth, the children who provided data at age 11 were likely to have: had a higher birthweight; been born to older parents with a higher family income and who were married; and to mothers who reported less stress, and were non-smokers. In addition, due to participant attrition, the sample was restricted at an earlier stage of the study to European mothers. As such, some caution must be employed in generalising the results. Unsurprisingly, given that clinical screening questionnaires were being used on a general population, the outcome measures were also heavily skewed. It had been hoped to focus the study on predictors of ADHD, examining the predictive utility of the subscales of individual outcome measures, and contrasting Teacher and Parental ratings of hyperactivity. Due to normality restrictions,

however, the broader SDQ Total Difficulties score (continuous and categorical), and the CRS ADHD Index score (categorical), were selected as outcome measures.

This thesis is divided into five main stages of analysis. Firstly, in the absence of age specific norms, the appraisal of developmentally inappropriate activity and sleep is difficult. This research therefore aimed to provide such normative data in a New Zealand context, providing summaries of activity measures and sleep duration, as well as SDQ and CRS scores, at the age of 11. The data was also examined for main effects of gender and birthweight using 2x2 MANOVAs.

Secondly, the years between the ages of 7 and 11 represent a key developmental period for children, during which behavioural expectations increase greatly; raising the likelihood of developmental difficulties being exposed. As a central objective of the ABC study, all measures were examined between the ages of 7 and 11 using repeated-measures MANOVAs, to see if there had been differential changes across time between AGA and SGA children.

Thirdly, univariate correlational analyses were conducted to explore the relationship between activity and sleep duration, and the outcome measures. A variety of activity measures were employed to identify which has the most utility as a predictor. Analyses were also conducted with 48 more established risk predictors. Predictors showing significant correlations ($p<.01$; $p<.05$ for activity and sleep) were then examined for multicolinearity, before being taken forward for further analysis.

Fourthly, predictors that showed significant independent relationships as predictors were modelled using hierarchical regression analyses.

Finally, to explore the central hypotheses of this thesis, exploratory univariate analyse, using linear and logistical regression, were conducted on the Internalising and Externalising subscales of the SDQ and the CRS ADHD subscales.

The study therefore offers a valuable normative sample of 11 year old New Zealand European children's results on the CRS and SDQ, as well as their activity levels and sleep duration. For the first time in the ABC study's history the current research showed SGA children to perform more poorly on certain psychological measures. Examination of these measures between the ages of 7 and 11 indicated the possibility that previously latent difficulties are being uncovered as SGA children grow older. While the lack of normality of the subscale scores prevented definitive conclusions, internalising emotional difficulties were more evident among SGA females, but SGA males exhibited broader issues across both the internalising and externalising domains.

An examination is then presented of the relationship between Parental CRS and SDQ scores, activity and sleep duration, and established risk factors. These risk factors include: low-birthweight; prenatal alcohol, nicotine, and cannabis exposure; psychosocial adversity; maternal social support and perceived stress; child intellectual function; and child electronic media exposure. These analyses found theoretically consistent relationships between activity and the outcome variables, but activity measures were outperformed by the covariates as predictors. The most notable of these predictors were maternal stress, maternal cannabis use, and child IQ. Sleep duration was also a very poor predictor and neither variable appeared to have diagnostic utility based on the measures employed in this study. Environmental and psychosocial variables accounted for significant percentages of variance of the outcome measures, reemphasising the importance of these oft neglected factors. While normality restrictions again limited the conclusions that could be drawn, the strength of associations between activity and Parental CRS ADHD subscale scores suggest these scales capture motoric hyperactivity, but that teacher scales may capture the relationship between low activity, sleep duration, and inattention.

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List of Acronyms

A	Additive genetic effects
AACAP	American Academy of Child and Adolescent Psychiatry
ABC	Auckland Birthweight Collaborative study
	Phase 1 ABC Study sample at Birth
	Phase 2 ABC Study sample at Age 1
	Phase 3 ABC Study sample at Age 3.5
	Phase 4 ABC Study sample at Age 7
	Phase 5 ABC Study sample at Age 11
ADD	Attention Deficit Disorder
ADHD	Attention Deficit Hyperactivity Disorder
ADHD-C	Attention Deficit Hyperactivity Disorder - Combined type
ADHD-H	Attention Deficit Hyperactivity Disorder - Hyperactive type
ADHD-I	Attention Deficit Hyperactivity Disorder - Inattentive type
AGA	Appropriate for Gestational Age
AMSIS	Auckland Maternity Services Information System
ANOVA	Analysis Of Variance
APA	American Psychiatric Association
BASC	Behavioural Assessment System for Children
BMI	Body Mass Index
C	Shared environmental effects
CAMHS	Child and Adolescent Mental Health Services
CBCL	Child Behavioural Checklist
CD	Conduct Disorder
CDC	Center for Disease Control
CESD	Centre for Epidemiologic Studies Depression Scale
CFA	Confirmatory Factor Analysis
CRS	Conners' Ratings Scale
	CPRS-R:L Conners' Parental Rating Scale-Revised: Longform
	CTRS-R:L Conners' Parental Rating Scale-Revisied: Longform
D	Dominant genetic effects
DF	Degrees of Freedom
DISC	Diagnostic Interview Schedule for Children

DSM	Diagnostic and Statistical Manual of Mental Disorders
	II Second Edition
	III Third Edition
	III-R Third Edition Text Revision
	IV Fourth Edition
	IV-TR Fourth Edition Text Revision
	V Fifth Edition
DSMD	Deveraux Scales of Mental Disorders
DZ	Dyzygotic
E	Non-shared environmental effects
EEA	Equal Environment Assumption
EFA	Exploratory Factor Analysis
FAS	Foetal Alcohol Syndrome
FSIQ	Full-scale Intelligence Quotient
FSS	Family Support Scale
GxE	Gene by Environment interaction
ICD	International Classification of Diseases
IQ	Intelligence Quotient
K-S	Kolmogorov-Smirnova
LBW	Low Birth Weight
MANOVA	Multivariate Analysis Of Variance
MBD	Minimal Brain Dysfunction
MET	Metabolic Equivalents Units
MMFT	Matching Familiar Figure Test
MZ	Monozygotic
NCCMH	National Collaborating Centre for Mental Health
NICE	National Institute of Clinical Excellence
NOS	Not Otherwise Specified
NZ	New Zealand
NZMOH	New Zealand Ministry of Health
ODD	Oppositional Defiant Disorder
OR	Odds Ratio
PDD	Pervasive Developmental Disorder
PIQ	Performance Intelligence Quotient

PSS	Perceived Stress Scale
PTSD	Post-Traumatic Stress Disorder
ROC	Receiver Operating Characteristic
SD	Standard Deviation
SDQ	Strengths and Difficulties Questionnaire
SE	Standard Error
SES	Socio-Economic Status
SGA	Small for Gestational Age
Sig	Significance level
VIQ	Verbal Intelligence Quotient
WASI	Wechsler Abbreviated Scale of Intelligence
WHO	World Health Organisation
WISC	Wechsler Intelligence Scale for Children

1 Introduction

1.1 Background

When clinicians assess for childhood emotional and behavioural difficulties, by necessity they tend to rely on the reports of the parents, teachers, and the child themselves. To help ascertain whether such assessments are required, and to supplement such processes, subjective rating scales are often administered. These include the Strengths and Difficulties Questionnaire (SDQ) (Goodman, 1997) and the Conners' Rating Scale (CRS; Conners, 1997). The SDQ is a broad screening measure of childhood difficulties and is included within a nationwide pre-school assessment by the New Zealand Ministry of Health (NZMOH), named the 'B4 School Check' (NZMOH, 2008). The CRS is a more focussed instrument, designed primarily to identify the symptoms of Attention Deficit Hyperactivity Disorder (ADHD). ADHD is a psychiatric diagnosis in the Diagnostic and Statistical Manual of Mental Disorders- Fifth Edition (DSM-V; American Psychiatric Association [APA], 2013) characterized by developmentally inappropriate levels of over-activity, impulsivity and/or inattention. Perceptions of problematic behaviour utilising any subjective scale are, however, influenced by the experience and expectations of the rater, as well as by cultural and educational norms (Brewis, 2002).

The NZMOH recommends the administration of the CRS to parents and teachers as part of a comprehensive assessment for ADHD (NZMOH, 2001). This is because diagnosis of the disorder has historically required the demonstration of impairment across multiple settings. However, neither parents nor teachers are trained observers, and the appraisal of such ratings is often complicated by poor inter-rater agreement (Tryon, 2009). Such discrepancies do not necessarily suggest inaccurate observations, as childhood difficulties are known to manifest in different ways depending on the environment (Wolraich et al., 2004), but the validity of these ratings remains difficult to verify.

To further complicate childhood assessment, it is increasingly being recognised that the defining characteristics of problem behaviour do not remain stable as the child grows older, even within the context of discrete psychiatric diagnoses. For example, the symptoms of ADHD are now understood to represent an unfolding developmental pathway (Nigg, 2006), with greater displays of hyperactivity in younger children, which generally transition to more inattentive symptoms in later life (Lahey, Pelham, Loney, Lee & Willcutt, 2005). Despite this Barkley (1991; 2006) has rejected the utility of using objective measures of physical movement to support the diagnostic process, or to verify subjective reports of hyperactivity. This is of note as his published work is relied upon heavily by the NZ assessment guidelines for ADHD (NZMOH, 2001). Tryon (2009) believes that the dismissal of such technologies is premature and is based on poorly defined concerns about the ecological validity of such measures.

As classification systems for psychiatric diagnoses have developed, there has been an increasing focus on objective behavioural symptoms, with changes in activity levels and sleep patterns being recognised as reliable indicators of over 30 disorders (Teicher, McGreenery & Ohashi 2006). Both activity and sleep are difficult to appraise accurately using subjective rating scales, which generally employ Likert scales rather than meaningful units. Such scales also often conflate physical activity with broader behaviours such as impulsivity, conduct, and intellectual function (Rapport, Kofler, & Himmerlich, 2006). Historically, obtaining objective measures of activity and sleep has involved the use of expensive equipment in laboratory settings, resulting in questionable ecological validity. The advent of compact portable accelerometers (actigraphs; Actigraph™ denotes the common brand of sensor used in this study) has allowed these measures to be monitored in real-world settings for research purposes, but they remain relatively expensive. With the recent integration of accelerometers within modern smart-phones and Fitbits, it has now become

feasible for clinicians to collect activity data from the individuals they are assessing. This is especially true given that today's children appear to be surgically attached to such devices, so it is unlikely that compliance with such a recording protocol would be an issue.

The current research aimed to explore whether objective measurements of childhood activity and sleep duration predict subjective ratings of childhood emotional and behavioural difficulties at age 11. Using the SDQ and CRS as outcome measures, particular focus was placed on the relationship with subjectively identified symptoms of ADHD. Significant results would suggest that activity and sleep data may add valuable information to the clinical assessment process, as well as potentially assisting in the monitoring of treatment efficacy.

1.2 The Auckland Birthweight Collaborative (ABC) Study

The current research forms part of the Auckland Birthweight Collaborative (ABC) study. The ABC study was originally conceived as a longitudinal case-control study to examine for modifiable risk factors related to the growth and development of children born small for gestational age (SGA). As a result the sample population on which the current research is based is split approximately evenly between children who were appropriate (AGA) or small (SGA) for their gestational age. To date there have been five phases of data collection: at birth; 1 year; 3.5 years; 7 years and 11 years. At each phase extensive amounts of data have been collected to track each child's developmental trajectory, including physical, environmental, social, familial, and psychological variables.

1.2.1 Data relevant to the current study

Central to this thesis, when the children were 11 years of age (Phase 5; 2006-2008) data collection included a seven day period during which the children were fitted with an Actigraph monitor, which provided an objective measure of their daytime activity and their sleep patterns. In addition the data set includes two scales used commonly to identify symptoms of Attention Deficit Hyperactivity Disorder (ADHD); the long format versions of

the Conners' Parent Rating Scale Revised (CPRS-R:L) and the Conners' Teacher Rating Scale Revised (CTRS-R:L; Conners, 1997). The children were also administered the parental and self-report versions of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997). The SDQ includes a hyperactivity subscale, but also provides broader information about other potential emotional and behavioural difficulties. A measure of the children's intellectual function was obtained using the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999). In Phase 5, 620 of the original 871 European participants were successfully followed up, with data from these participants also being drawn from earlier phases of the study, most notably when the children were 7 years old (Phase 4; 2002-2004). When the children were 7 the study was restricted from the original birth cohort to the children of mothers who self-identified as European. This was due to attrition of non-European mothers from the study in earlier phases of data collection (Nixon et al., 2008). Thus, only data from children with European mothers are considered here.

The ABC data set includes actigraph data, Conners' Rating Scales (CRS), Strengths and Difficulties Questionnaires (SDQ), and IQ scores from children at both the ages of 7 and 11. This is a critical juncture when the behavioural expectations made of children change dramatically. Given that ADHD represents an unfolding developmental pathway longitudinal research is a particularly powerful tool for studying the disorder (Nigg, 2006). With the increasing evidence that the symptoms of ADHD exists on a spectrum of severity (Faraone et al., 2006; Faraone et al., 2009; Larsson et al., 2012), utilising a large non-clinical sample provides an exciting opportunity to examine the activity, sleep patterns, behavioural issues, lifestyle factors and cognitive performance of children across the entire range of this spectrum. Importantly this data comes from the perspective of multiple informants, including the children themselves.

1.3 Activity in Childhood

As Sarver, Rapport, Kofler, Raiker and Friedman (2015) identify, activity level is arguably the first enduring trait to develop in humans, with foetal movement recordings showing that individual differences are apparent from the 28th week of gestation. High levels of foetal activity during the last three months of pregnancy are significantly associated with positive behavioural attributes at 12 weeks, such as increased social interaction, motor and mental maturity, and inquisitiveness (Rapport et. al, 2006; Walters, 1965). The vigour of neonatal movement has been found to be positively correlated with daytime activity between the ages of 4 to 8, as well as the tendency to approach rather than withdraw from new experiences (Korner et al., 1985). Thomas and Chess (1977) developed a nine-factor model of child temperament, influenced by evidence that infant activity levels display greater individuality and less influence of the environment than predicted by learning theory alone. As the first factor in this model, activity level showed the most consistent correlation from the age of 1 through to 5. Activity remains remarkably stable during these preschool years, despite differences in context and environment (Rapport et al., 2006). Activity level is also one of the three core dimensions in Buss and Plomin's (1975) conception of child temperament, as well as carrying forward as a component of Extraversion in the Big Five model of adult personality (Costa & McCrae, 1988). While older, these models of temperament and personality are still considered to meet contemporary psychometric standards. It has been found that the constructs they measure contribute towards both an understanding of individual difference in preschool children, and the subsequent development of problem behaviours (De Pauw, Mervielde & Van Leeuwen, 2009).

As shown in Figure 1, across the first 16 years of life, a review of twelve studies shows that at a population level average activity levels demonstrate an inverted U-shaped developmental pattern, peaking at the age of 8 (Eaton, McKeen, & Cambell, 2001).

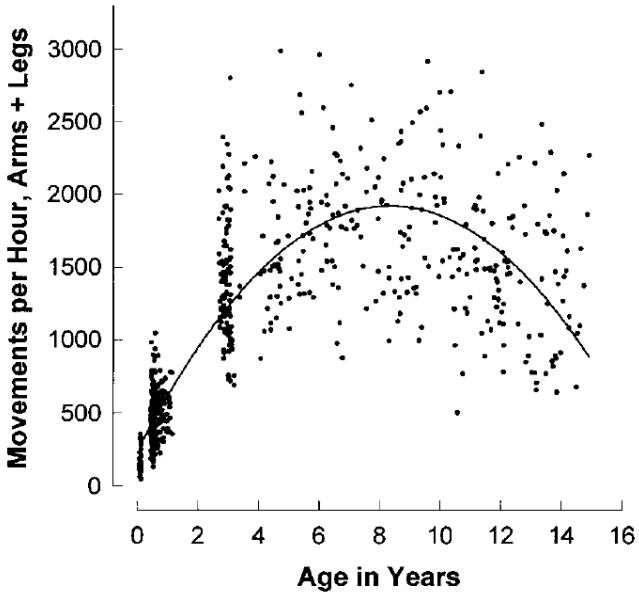

Figure 1: Mean arm-and-leg movements per hour by age in years (N =718; from Eaton et al., 2001).

Activity level declines with increasing age after the age of 8, with males and females showing a decline in total activity level of 69% and 36% respectively during school days from childhood through to adolescence (Gavarry, Giacomoni, Bernard, Seymat & Falgairette, 2003). A meta-analysis of 46 studies comparing boys' and girls' activity showed that male infants are more active than female infants when measured by parent ratings, direct observations, and objective measurements (Campbell & Eaton, 1999). Despite the enduring nature of activity traits, the favourable associations with high levels of activity observed during the neonatal period reverse once children enter school, especially for those less able to regulate their activity (Rapport et al., 2006). After this age above-average motor activity has been linked to later diagnoses of ADHD (Campbell & Ewing, 1990), externalising behaviour problems (Keown & Woodward, 2006), and poor educational and occupation performance (Barkley et al., 2006). Perhaps unsurprisingly given the data presented above from Gavarry et al. (2003), males are disproportionately represented within such diagnostic categories.

Epidemiological studies show the average male to female ratio for those diagnosed with ADHD is 3.4:1 among community samples and 6:1 in clinic-referred samples, with a higher proportion of males receiving clinical attention due to associated aggression (Barkley, 2006a). Thomas and Chess (1977) first proposed that the fit between a child's temperament and their environment might be important in understanding psychopathology. This certainly seems to be true in the case of ADHD, with evidence to suggest that certain alleles of the D4 dopamine receptor may confer a risk of externalising behavioural problems to those children receiving less responsive and stimulating maternal environments (Nikitopoulos et al., 2014). Anthropologists have also suggested that the substantial differences in the rate of diagnosis of ADHD between countries are influenced by cultural differences in how behaviour becomes classified by either parent or teacher, with cultural tolerances for specific behaviours varying markedly (Brewis, 2002).

1.4 Childhood Emotional and Behavioural Difficulties

A report by Perou et al. (2013) surveyed the percentage of children aged 3 to 17 years old in the United States who had been identified with mental health disorders between 2005 and 2011. They showed that ADHD (6.8%) and behavioural or conduct problems (3.5%) were the most common diagnoses, followed by anxiety (3.0%), and depression (2.1%). In New Zealand a similar survey of children aged 2 to 14 years (NZMOH, 2012) found that a lower percentage of children were diagnosed with emotional or behavioural problems (3.2%). Of these anxiety was the most common (2.0%), followed by ADHD (1.4%), and depression (0.3%). Despite these differing rates of diagnosis across countries, likely due to the variations in global diagnostic practices, the negative scholastic and behavioural outcomes associated with ADHD means that it receives a similarly high level of research attention in New Zealand as it does in other Western countries.

In a self-sustaining cycle, the global research focus on ADHD is likely to influence governmental policy, media coverage, and public awareness. An example of this is the rationale given in the 'B4 School Check practitioners handbook' (NZMOH, 2008) for the inclusion of the SDQ to assess New Zealand school children. The document cites the 'Church Report' (Church, 2003) to highlight the importance of the early identification of behavioural problems if treatment interventions are to be successful. Although few clinicians would argue with this principle, while the SDQ includes a broad range of indices (Hyperactivity, Conduct, Emotional, and Peer difficulties), the Church Report (Church, 2003) is focussed exclusively on pathways to anti-sociality, such as ADHD and Conduct Disorder. The use of this reference to justify the inclusion of the SDQ in the B4 School Check has led Miller (2013) to question whether this represents the influence of a political agenda to identify indications of criminality from an early age, as well as a focus on individual rather than societal explanations for anti-sociality. In contrast, while externalising problems of childhood have a more salient societal impact and are often associated with higher levels of physical activity, a recent British study involving 2500 adolescents has found that lower levels of physical activity were associated with more mental health concerns (Ussher, Owen, Cook & Whincup, 2007). The measurement of activity, combined with the exploration of the psychosocial factors that influence these outcomes, therefore has the potential to supplement the assessment of both externalising and internalising disorders in children.

The feasibility of using activity to help discriminate between mental health disorders is illustrated by Figure 2 below, taken from a study by Teicher et al. (2006). The same study also showed that by adding a measure of circadian-dysregulation (disturbances to sleep-wake cycles) this discrimination becomes clearer, highlighting the importance of also examining sleep patterns.

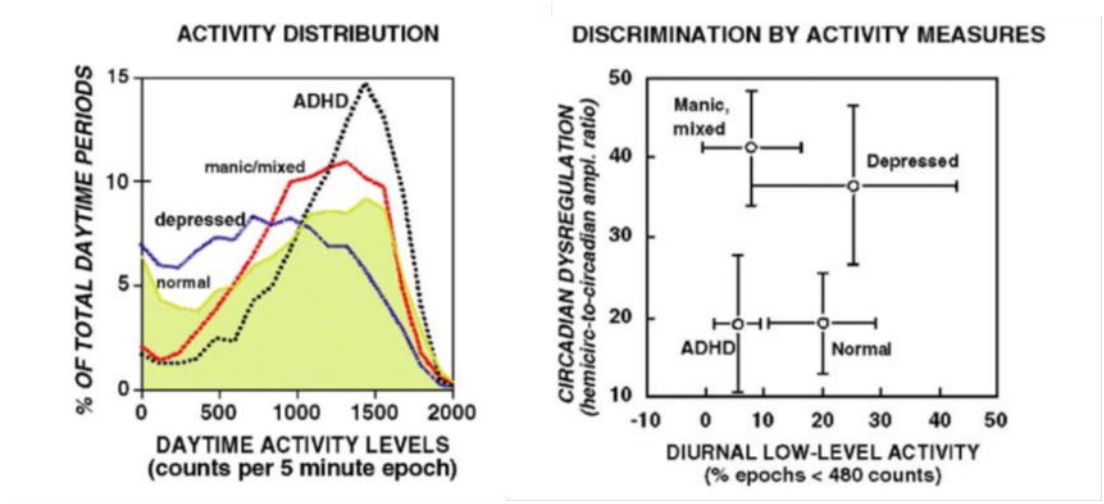

Figure 2: Illustration that the distribution of the percentage of daytime spent at different levels of activity can distinguish between psychiatric disorders; a discrimination made clearer by adding a measure of circadian dysregulation. Activity data were collected for 72 hours with five minute epochs (Figures from Teicher et al., 2006).

Table 1 below, summarises DSM-V disorders that include criteria that relate to activity/psychomotor changes or disturbances in sleep. Although it should be noted that many of these relationships may be bi-directional (Cousins et al., 2011), it further illustrates the potentially broad utility of monitoring such variables. Indeed sleep disorders are so prevalent among psychiatric problems, that researchers have long suspected that they share common biological roots (Harvard Mental Health Letter, 2009).

Table 1

Common DSM-V diagnoses/codings associated with changes in activity and sleep

	Increased Activity	Reduced Activity	Sleep Disturbance
ADHD (esp. ADHD-H and ADHD-C)	✓		✓
Major depressive episode		✓	✓
Bipolar disorder	✓ (manic phase)	✓ (depressive phase)	✓
Acute stress disorder/PTSD	✓		✓
Generalised anxiety disorder	✓		✓
Schizophrenia	✓ (positive symptoms)	✓ (negative symptoms)	✓
Premenstrual dysphoric disorder		✓	✓
Childhood abuse	✓ (with PTSD)	✓ (without PTSD)	✓
Autism spectrum disorders		✓	✓

Note. DSM-V = Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition; ADHD = Attention Deficit Hyperactivity Disorder; ADHD-H = Attention Deficit Hyperactivity Disorder – Hyperactive type; ADHD-I = Attention Deficit Hyperactivity Disorder – Inattentive type; PTSD = Post-Traumatic Stress Disorder.

1.5 Activity and Externalising Disorders

1.5.1 Attention Deficit Hyperactivity Disorder (ADHD)

As well as being an important dimension of a child's temperament, activity is a core symptom domain in the diagnosis of ADHD (Wood, Saudino, Rogers, Asherson, & Kuntsi, 2007). ADHD is a diagnostic category within the current Diagnostic and Statistical Manual of

Mental Disorders (DSM-V; APA, 2013). It is defined as a persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequently displayed and more severe than is typically observed in individuals at a comparable level of development. Yet despite the fact that symptoms of over-activity are often a key feature in the diagnosis of ADHD, little data exists to quantify the nature of this activity component (Wood, Asherson, Rijsdijk, & Kuntsi, 2009) and most contemporary models of the disorder view hyperactivity as being secondary to cognitive deficits associated with the disorder (Rapport et al., 2009). In New Zealand the most recent clinical guidelines for the assessment and treatment of ADHD were published almost 15 years ago in 2001 (NZMOH, 2001). The document recommends that a behavioural rating scale of good reliability and validity, such as the 1997 revision of the Conners' Rating Scale (Conners, 1997), should be administered to parents and teachers as part of a comprehensive assessment. Like other subjective measures of ADHD, the Conners' Rating Scale includes indices assessing hyperactivity, but does not use a standard or meaningful unit of activity due to its factor-analytic scale construction (Rapport et al., 2006). This makes it extremely difficult to separate activity level from the other factors that contribute to the assessment of whether a child has ADHD.

DSM-V is the first major revision of the diagnostic manual produced by the American Psychiatric Association since DSM-IV was published in 1994 (APA, 1994). The criteria for ADHD remain largely unchanged from DSM-IV-TR (APA, 2000), other than being placed within the section on neurodevelopmental rather than disruptive disorders. One notable revision is that while symptoms of ADHD must still be evident in more than one context, they no longer have to cause significant impairment across multiple settings. Also, whereas previously some symptoms must have presented before the age of 7, this has now been increased to 12.

For a DSM-V (APA, 2013) diagnosis of ADHD to be made (see Appendix A for full criteria), at least six of nine symptoms of inattention (Criteria 1), or six of nine symptoms of hyperactivity-impulsivity (Criteria 2) must be observed for at least six months. If both criteria are met the diagnosis of ADHD is coded as Combined Presentation (314.01). If one criterion is met it is coded as either Predominantly Inattentive Presentation (314.00) or Predominantly Hyperactive-Impulsive Presentation (314.01). A diagnosis of Other Specified Attention-Deficit/Hyperactive Disorder (314.01) is available for symptoms that cause significant impairment, but do not meet full criteria, with the reason for failing the criteria being specified. A classification of Unspecified Attention-Deficit/Hyperactive Disorder (314.01) can be used when there is impairment, but no reason is specified for why full criteria have not been met. As such, the new diagnostic criteria are more lenient and inclusive. This has fuelled existing and widely-publicised concerns about diagnostic inflation and the over-prescription of ADHD medications, including from respected commentators such as the Chairman of the DSM-IV task force Professor Allen Frances (Batstra & Frances, 2012). The relaxation of the requirement that impairment is shown across multiple settings also increases the possibility that a child who has particular problem with the parent child-relationship, or the child-teacher relationship, may risk being labelled as having ADHD.

The DSM-V diagnosis of ADHD Combined Type is broadly equivalent to Hyperkinetic Disorder defined in the current International Classification of Diseases (ICD-10; World Health Organisation [WHO], 2004). The ICD-10 only recognises a combined type of the disorder and requires that all symptoms are present in more than one context, so in effect describes a severe sub-group of people who meet the DSM-V criteria. Unlike DSM-V the ICD-10 criteria also exclude diagnosis if comorbid psychiatric disorders are present, and further distinguish between Hyperkinetic Disorder with and without Conduct Disorder. Despite these differences in classification and nomenclature the DSM and the ICD diagnostic

labels yield very similar search results from scientific literature databases such as PubMed. As ADHD is the more common and inclusive term, and because the CRS is based on the DSM definition of the disorder, it is this diagnostic label that will be used throughout this thesis.

1.5.2 History of the diagnosis of ADHD and the changing emphasis on hyperactivity.

ADHD is an acronym that is embedded in popular culture, but as Rafalovich (2001) identifies, the brief accounts of the history of the disorder offered in many scientific publications tend to be shaped by the theoretical orientation of the authors themselves. While heavily orientated towards the refinement of the biomedical conception of the disorder, one of the most complete histories of the development of the diagnosis of ADHD is offered by Barkley (2006a), whose work guided the writing of this section. Evident when reading such summaries, is that the significance placed on excessive motor activity or hyperactivity in the conceptualisation of the disorder has varied considerably during the last century.

As Barkley (2006a) identifies, a German physician named Heinrich Hoffman made perhaps the first reference to a child with ADHD in his 1865 poem entitled “Fidgety Phil”. Most researchers, however, point towards a series of published lectures by George Still in 1902 for bringing to attention to a behavioural condition in children that most closely resembles what is known today as ADHD (Still, 1902; Barkley, 2006b). Influenced by the theories of William James, Still proposed that a biological predisposition underlay related deficits in inhibitory volition, moral control, and sustained attention. Hyperactivity was also a central feature. The conception of the disorder in the period between 1920 and 1960 was dominated by theories that it represented evidence of brain damage. This was heavily influenced by the outbreak of an encephalitis epidemic in 1917-18 in North America, a condition that led to patterns of brain damage that induced symptoms of hyperactivity,

inattention, and impulsivity. This view would be reinforced by the observation of the similarity between hyperactivity in children and the behavioural sequelae of experimental frontal lobe lesions in primates, as well as observations of the often inverse relationship between a child's level of activity and their intelligence. The disorder later became known as minimal brain dysfunction (MBD), a term adopted because so few of the children had clear evidence of brain damage. However, by the mid-1960's the term MBD began to be broadly criticised by a number of independent researchers, most directly in a review article by Herbert (1964), for being vague and overly-inclusive (Barkley, 2006a).

In the absence of a clear neurological aetiology of MBD, many researchers began to focus on hyperactivity. This was the behavioural symptom thought to be most characteristic of the disorder and was reflected by the theoretical terminology of the time, which included 'hyperkinetic impulse disorder' (Denhoff, Laufer & Solomons 1957) and 'hyperactive child syndrome' (Chess, 1960). This shift to describing the condition at a behavioural level led to the inclusion of a single sentence description of 'Hyperkinetic Reaction of Childhood' in DSM-II (APA, 1968). It also marked the beginning of a theoretical divergence between America and Europe. Whereas in America the syndrome would be viewed as an extreme degree in the normal variation of temperament in children, influential researchers in Britain viewed the condition as a far less common state of excessive activity, often associated with symptoms of brain damage (Barkley, 2006a). While now more united in terms of aetiological conception, this divergence arguably remains evident when contrasting the American DSM-V (APA, 2013) diagnosis of ADHD, with the more selective European ICD-10 (WHO, 2004) definition of Hyperkinetic Disorder.

The specificity and utility of hyperactivity in diagnosing the disorder would later be questioned, given its association with mental retardation, organic brain damage, and other mental illnesses (Douglas, 1972). Influenced heavily by the work of Virginia Douglas

(Douglas, 1983), by the publication of DSM-III (APA, 1980) deficits in sustained attention and impulse control were conferred greater significance than hyperactivity; although Douglas felt the new diagnostic label exaggerated the importance of attentional problems (Douglas, 1984). Excessive movement was no longer considered a necessary criterion for diagnosing the disorder, and motor activity and impulsivity were grouped together based on factor analytic findings (Du Paul et al., 1998). As a result, the condition was categorised as Attention Deficit Disorder (ADD) with or without hyperactivity, although at the time little empirical evidence existed for a non-hyperactive subtype. In recognition of this concern, by the publication of DSM-III-R (APA, 1987), ADD without hyperactivity was no longer recognized as a subtype of the disorder, and ADD with hyperactivity was for the first time referred to as ADHD. By the end of the 1980's the condition would be conceived as a disabling condition with a strong hereditary predisposition. However, its severity, comorbidity, and outcome were viewed as being significantly affected by environmental factors, particularly familial influences (Barkley, 2006a).

The 1990's saw a huge increase in research on ADHD, with neuroimaging and genetic findings shifting the focus away from social and environmental factors. This conception has contributed to medication becoming the predominant treatment for ADHD, although the extent to which combined treatments are superior to medication alone remains a controversial issue (Parens & Johnston, 2009; Smith, Barkley & Shapiro, 2005). In 1994 DSM-IV reintroduced a purely inattentive form of ADHD, similar to the diagnosis of 'ADD without hyperactivity' in DSM-III. The criteria also required evidence of the pervasiveness of ADHD symptoms across settings, as well as impairment in a major domain of functioning. In the past decade a great deal of focus has been placed on the high levels of comorbidity of ADHD with other disorders as well as its neuropsychological profile. As reflected by recent revisions in DSM-V (APA, 2013), predominantly cognitive and inattentive manifestations of

ADHD are also believed to extend into adulthood. Yet as Rapport et al. (2009) highlight, given that hyperactivity remains a core symptom of ADHD, most contemporary models of ADHD place very little emphasis on it. Sergeant's (2005) cognitive-energetic model focuses exclusively on information processing and Sagvolden, Johansen, Aase and Russell. (2005) view hyperactivity as incidental behaviour that occurs due to rapid extinction of behaviour-consequence relationships in children with ADHD. Barkley (1997) sees hyperactivity as children's ongoing struggle to inhibit task irrelevant behaviour, whereas Halperin, Trampush, Miller, Marks and Newcorn (2008) posit that hyperactivity is a result of enduring subcortical impairments. The debate as to whether hyperactivity is a core feature of ADHD, or a secondary symptom, clearly has significant implications for the assessment and management of the disorder.

1.5.3 Limitations and controversies relating to the diagnosis of ADHD

As Barkley (2006a) identifies, those with ADHD represents a heterogeneous population displaying considerable variation in the: age of onset; severity of symptoms; pervasiveness of these symptoms across various situations; and in the level comorbidity with other conditions. Senior authors of DSM-IV-TR (APA, 2000) have conceded psychiatry remains in the position that most of medicine was in over two-hundred years ago, with most of its disorders being of unknown aetiology and subsequently having to be defined by their observable syndromes (Frances & Egger, 1999). ADHD therefore denotes a grouping of multi-factorially determined syndromes without an established causal aetiology (Nigg, 2006) and because it is defined at a behavioural level its presence does not imply neurological disease or dysfunction ('NICE Guidelines', National Collaborating Centre for Mental Health, 2009). However, while there remains extensive debate around the broader utility of psychiatric diagnosis, the current weight of evidence suggests that this grouping of symptoms is useful and valid in aiding communication between researchers and clinicians and in

identifying children who are impaired in meeting developmental milestones (Nigg, 2006). Importantly, DSM-IV criteria of ADHD also have been demonstrated to identify individuals with significant ongoing impairment (Wilcutt et al., 2012).

As is detailed in the previous section, the conception of ADHD has evolved over time. As a result of the varying methods and diagnostic criteria used to assess the disorder, historical prevalence estimates vary considerably. It is therefore unknown whether the incidence of ADHD is rising in terms of true population level, with a prevalence estimate of 3-7% stated in the majority of papers. Prevalence is often confused with treatment rates, which are certainly increasing, driven largely by the rapid increase in the rate of medication being prescribed for the condition (Nigg, 2006). This increase is one of number of factors often cited in public debate to support scepticism about the validity of the ADHD diagnosis. Others include the evolving nature of the diagnosis, variable prevalence and treatment rates, and more general concerns about the increasing trend towards medicating children.

Many who question the validity of ADHD as a diagnostic condition raise broader concerns about the increasing 'medicalisation' of behaviour in many Western countries. Social construct theories of ADHD argue that the diagnosis is used to describe behaviours that may well fail to meet societal norms, but that are not genuinely pathological (Parens & Johnston, 2009). The 'International Consensus Statement on ADHD' (Barkley, 2002) was written in response to such concerns. The consensus statement warns against the presentation of the condition in the media as a myth, fraud or benign condition. It asserts that ADHD is a valid medical disorder for which psychotropic medications are genuine correctives. Yet Russell Barkley, the lead author of the consensus statement, appears to be dismissive of psychological and environmental explanations of the disorder. In his own writings about the history of ADHD he states:

> Milder forms of hyperactivity, in contrast, were attributed in this era to psychological cause, such as spoiled child rearing practices or delinquent family environments. The idea that poor or disrupted parenting causes ADHD would also be resurrected in the 1970's and continues even today among many laypeople and critics of ADHD. (Barkley, 2006a, p. 6)

Such a statement may appear to be a well-intentioned attempt to prevent parents from being stigmatised unfairly. However, by grouping laypeople and the critics of the disorder together as the only proponents of such ideas, it seems to suggest that consideration of experiential explanations of the disorder is poorly informed. Allied with such a concern, others have criticised the consensus statement for attempting to forestall debate and future research (Timimi et al., 2004).

Consideration of Barkley's potential biological bias is important given how influential his publications have been. Indeed his research has been cited by leading clinical psychology textbook author Alan Carr to conclude that "vulnerability to attentional and over-activity problems…is largely constitutional." (Carr, 2006, p. 12). The current NZ Guidelines for the assessment and treatment of ADHD are also heavily influenced by Barkley's work (NZMOH, 2001). While presenting ADHD as a fundamentally biological disorder may be seen to remove stigma from parents, there are arguments to suggest that the same may not be true for the child. Biological explanations of mental health diagnoses have been found to be related to perceptions of dangerousness and unpredictability, and to fear and desire for social distance (Read & Harre, 2001; Read, Haslam, Sayce & Davies, 2006). Genetic causes may also lead to people being conceived of as "defective" or "physically distinct" which, contrary to Barkley's (2006a) apparent intentions, can also create an "associative stigma" for the whole family, who may be considered "at risk" or "carriers" (Phelan, 2002). While these arguments have no bearing on the aetiology of ADHD, they highlight credible concerns if biological

explanations of ADHD are overstated. When considering aetiology and treatment, such a bias may also lead to modifiable environmental factors being overlooked.

Cortese, Faraone and Sergeant (2011) emphasise that it is a misunderstanding to believe that genetic research and high heritability figures favour the use of pharmacotherapy. Such research is however clearly the most commonly cited to support the biological models of the disorder then employed to justify medication use (Joseph, 2003). In recent years the influence of pharmaceutical companies on scientific research output, patient advocacy groups, and prescribing doctors has also come under closer scrutiny from high profile sources such as the former Editor-in-Chief of The New England Journal of Medicine (Kassirer, 2005). A detailed discussion of the various issues touched upon in this section is beyond the scope of this thesis, but these issues highlight the continued need to refine the diagnosis and treatment of ADHD, and to employ caution about narrowing the focus of research prematurely.

1.5.4 Criticisms of DSM diagnosis and subtypes

While ADHD is often treated as a static diagnosis, it actually represents an unfolding developmental pathway (Nigg, 2006). There tends to be a greater display of hyperactivity in younger children, but they rarely remain in this predominantly hyperactive classification over time, mostly being transitioned to a combined diagnosis if they continue to exhibit symptoms of ADHD (Lahey et al., 2005). Related to this, those people who then suffer symptoms into adulthood were not recognised in DSM-IV-TR (APA, 2000). While the number of criteria a person meets may fall as they get older, in part due to becoming less active and disruptive, there is evidence that this is not necessarily accompanied by a reduction in personal impairment (Barkley, Murphy, & Fisher, 2008). Polanczyk et al. (2010) suggested that the DSM-IV diagnostic requirement that people show symptoms of ADHD before the age of 7 was not based on empirical evidence. They found that adults who are able to report symptom onset by age 12 also had symptoms by age 7, even if they were not able to report them. They

also found that prevalence estimates, correlates, and risk factors of ADHD were not likely to be affected by extending the age-of-onset criteria (Polanczyk et al., 2010). These issues have subsequently been addressed in DSM-V (APA, 2013), which has increased the age by which impairment must first be demonstrated to 12, and has also recognised an adult manifestation of the disorder characterised by fewer symptoms. The decision to retain the ADHD subtypes was made despite an increasing endorsement of dimensional rather than categorical classifications of mental illness in DSM-V, and more specifically for ADHD (Wilcutt, 2012). It is of note that studies measuring activity seem to support a dimensional approach to the behavioural assessment and categorization of ADHD (Pinto & Tryon, 1996).

The existence of three subtype entities lends weight to their being real, even though the evidence to support their differentiation and stability over time is limited (Lahey & Wilcutt, 2010; Wilcutt et al., 2012). Predominantly inattentive ADHD is one of the most frequently used diagnoses with many of these children thought to show few, if any, manifestations of hyperactivity. The current subtype structure does not accurately allow for purely inattentive children, leading some researchers to propose a new classification of children with ADHD who have a sluggish cognitive tempo (SCT; Carlson & Mann, 2002). Yet available data does not support the evidence that SCT symptoms identify a primarily inattentive subgroup that is unrelated to ADHD-C (Willcutt et al., 2012). Interestingly, when actigraphs have been employed with children aged 8 to 12, measures of inattentive symptoms have been shown to be more correlated with motor activity than with subjective measures of attention (Konrad, Gunther, Heinzel-Gutenbrunner & Herpertz-Dahlmann, 2005).

The detailed meta-analysis by Wilcutt et al. (2012) highlights the important gaps that remain in the literature on ADHD subtypes, especially relating to ADHD-H in adolescents, making the validity of an ADHD-H subtype after preschool unclear. The data supports the concurrent, predictive, and discriminant validity of the distinction between inattention and

hyperactivity-impulsivity symptoms. The evidence is however more mixed with regards to the discriminant validity of the DSM subtypes, with the subtypes offering little unique information beyond that provided by the symptom dimensions (Wilcutt et al., 2012). One potential overarching issue with the current criteria is that they remain relatively ambiguous, despite efforts to provide case examples in DSM-V, which increases criterion variance. All of these issues highlight the utility of examining activity and other objective measures of symptomatology.

1.5.5 Prevalence and treatment rates of ADHD

The most recent meta-analysis of the worldwide prevalence of mental disorders in children and adolescents found a pooled-prevalence for ADHD of 3.4% (CI 95% 2.6-4.5; Polanczyk, Salum, Sugaya, Caye & Rohde, 2015) down from the 5.3% found in a similar study eight years previously (Polanczyk, de Lima, Horta, Biederman & Rohde, 2007). Yet the website for the Center for Disease Control (CDC) in the United States cites a study that shows in 2011 the diagnosis rate in America was as high as 11% (Akinbami, Liu, Pastor & Reuben, 2011). While numerous studies have relied on parent and teacher survey data alone, yielding prevalence estimates from 2.3% to 19.8%, such studies assess screening prevalence not true prevalence (Nigg, 2006). Nigg (2006) calculated unweighted median prevalence estimates across five international studies, which utilised either structured ratings or a combination of impairment criteria, teacher, and parental ratings. The results were 2.9% for ADHD-C, 3.2% for ADHD-I, and 0.6% for ADHD-H, yielding a total of 6.8%. In the UK, a survey of 10,438 children that combined information from multiple sources (parents, teachers, children, and a clinical appraisal of a structured interview with verbatim reports) found that 3.62% of boys and 0.85% of girls had ADHD (Ford, Goodman & Meltzer, 2003).

The only information that could be found on prevalence or treatment rates for ADHD in NZ was a single survey that relied on parental reports of a doctor's diagnosis (NZMOH,

2012). To explore this further, a request was made to the NZMOH's pharmaceutical claims data warehouse known as Pharmhouse for information about the prescription rates of the three most common ADHD medications. This showed that in 2010 prescriptions were given to 13,905 children under the age of 18. Projected census figures from the same year obtained from Statistics NZ estimated the number of school aged children (age 5-16) to be 707,450. Based on the number of individual children prescribed medication in this age group (13,180), divided by this population estimate, approximately 1.86% of children aged 5 to 16 are being prescribed ADHD medications in NZ. According to an information analyst at the National Health Board this is likely to be an underestimate of around 12%, as a result of the incidents where NHI numbers are not reported, so the actual figure is likely to be approximately 2%. In addition there are many children diagnosed with ADHD who are not on medication. This suggests that the NZMOH (2012) study results, which show 1.4% of children in their sample to be suffering from ADHD, represent an underestimation of the population total.

1.5.6 Sex distribution of children receiving medication in New Zealand

While medication use is not specifically examined in the current study, prescriptions rates highlight that ADHD diagnoses remain overrepresented among males. Across the period of 2006-2011, despite increases in the overall number of children receiving medication, the sex distribution of medicated children between the ages of 2-17 remained extremely consistent; see Figure 3. An average of 82.1% of the medicated population was male. See Appendix B for a more detailed breakdown of this information.

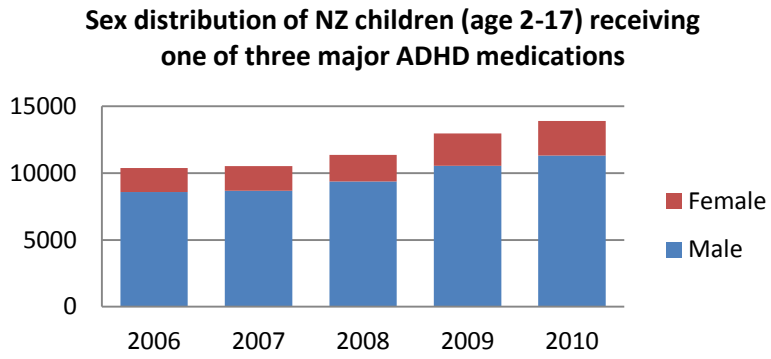

Figure 3: Graph showing sex distribution of medicated ADHD population

1.5.7 Trends of ADHD medication prescription rates in NZ over the past fifteen years

The total number of prescriptions for the three most commonly used ADHD medications in NZ increased by 294% between the years of 1996-2010; see Figure 4. For a more detailed breakdown of this information please see

Appendix C.

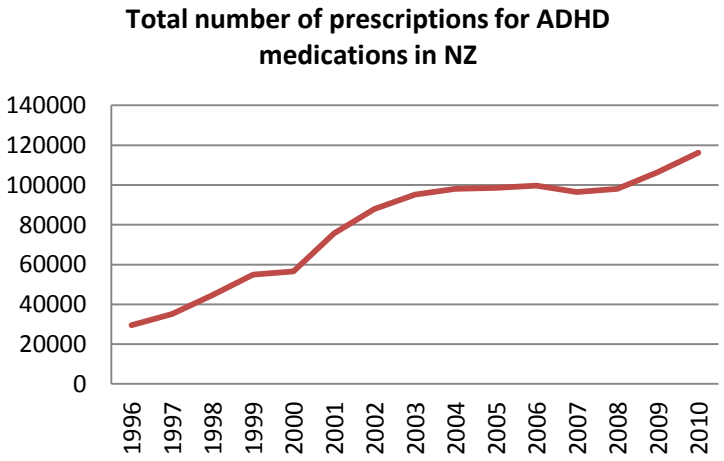

Figure 4: Graph showing increases in prescriptions for ADHD medications 1996-2010

1.5.8 Comorbidity with ADHD in the presence or absence of hyperactivity

The majority of children diagnosed with ADHD, are also identified to be suffering from at least one other comorbid or coexisting condition. These include oppositional defiant disorder (ODD) and conduct disorder (CD), depression, and anxiety disorders, tic disorders including Tourette's Syndrome, and a range of learning and motor coordination problems (Thapar & Cooper, 2015). As Gillberg et al. (2004) identifies, the word comorbidity is used variably within the research literature. It can be used to mean two or more disorders sharing a common underlying aetiology, one disorder leading to another, or two separate disorders occurring together. When considering DSM disorders it is particularly important to consider if comorbidity may just represent shared diagnostic criteria.

Barkley (2006a) does however point to evidence that suggests the presence or absence of Hyperactive-Impulsive traits may influence patterns of psychiatric comorbidity. Children with ADHD-C are more likely to be male, have significantly greater risk for other disruptive behaviour disorders, be oppositional and aggressive, and be rejected by peers. In contrast children with ADHD-I may show more internalising problems and tend to be more shy, passive, and withdrawn in peer relationships. The table below, adapted from Barkley (2006a) and drawn from US samples, cites prevalence estimates of the most well researched comorbid conditions among those diagnosed with ADHD.

Table 2

Common comorbidities with ADHD (adapted from Barkley, 2006a)

Comorbid condition	Average percentage of clinic-referred children with ADHD who have this condition	Average percentage of clinic-referred children with this condition who have ADHD
ODD/CD	45-55%/35-45%	NA
Anxiety Disorders	25-30%	15-30%
Major Depressive Disorder	25-30%	16-63%
Post-Traumatic Stress Disorder	1-6%	14-46%

Note. ADHD = Attention Deficit Hyperactivity Disorder; ODD = Oppositional Defiant Disorder, CD = Conduct Disorder. NA = Not applicable.

1.5.9 ADHD diagnostic practices in NZ

Remarkably, given the wealth of research funding dedicated to ADHD, the most recent NZMOH guidelines for the assessment and treatment of ADHD were published in 2001 (NZMOH, 2001). Clinical recommendations on assessment in this document are derived from the Barkley (1998) textbook and the ADHD 'Practice Parameters' of the American Academy of Child and Adolescent Psychiatry (AACAP Official Action 1997). Unlike the treatment guidelines contained within this document, the assessment guidelines were not subject to a critical evidence-based review. Instead the document states that "expert clinicians consider that [the assessment section] represents current best practice applicable to NZ conditions (NZMOH, 2001, pg. 14). As part of these guidelines it is recommended that comprehensive history of the child and the problem is taken, along with a detailed family and social history, an assessment of school functioning, and a one-on-one child evaluation. It is noted in this document that, unlike many other Western countries, general cognitive testing and neuropsychological testing are not carried out routinely in NZ when learning problems are present.

The NZ guidelines (NZMOH, 2001) recommend that a behavioural rating scale that has good reliability and validity should be administered as part of a comprehensive assessment. The information provided in the guidelines on ADHD rating scales is derived from a single review by Green, Wong, Atkins, Taylor and Feinleib (1999), which was considered "*the most recent and comprehensive available*" (NZMOH, 2001). Based on this review the NZ guidelines identify two indices of the 1997 Revision of the Conners' Rating Scale (Conners, 1997) as being highly effective for discriminating between children with ADHD and normal controls. These are the Parental and Teacher ADHD Index Scale and the DSM-IV symptoms scales. Based on the Green et al. (1999) findings other broad-band scales were not considered to be useful in distinguishing children with clinically significant symptoms of ADHD. These included the Total Problem Scale of the Parental and Teacher versions of Child Behavioural Checklist (CBCL; Achenbach, 1991), the Total Scale of the Deveraux Scales of Mental Disorders (DSMD; Naglieri, LeBuffe, & Pfeiffer, 1994), and the Global Problem Index of the Conners' Rating Scale (Conners, 1997). Unsurprisingly, given that it was only developed in 1997, no mention is made in the guidelines of the Strengths and Difficulty Questionnaire (SDQ). While broad-band scales such as the SDQ are unlikely to match the psychometric properties of a more focussed diagnostic instrument, they can provide useful information when considering a differential diagnoses.

1.5.10 Subjective measures of hyperactivity

1.5.10.1 Conners' Behavioural Rating Scale: Revised – Long Format (CRS-R:L)

The Conners' Rating Scale was originally developed to assess a wide variety of problem behaviour in children and as a measure of behavioural change for pharmacological studies (Conners, 1969). More recently it was expanded to include symptoms of ADHD, and for the past decade has been a commonly used measure of the behavioural problems

associated with this disorder (Conners, 1997; see Appendix D for scale criteria). The Parent and Teacher versions utilise Likert-scales to generate scores for six core factors: Oppositional Problems; Cognitive Problems; Hyperactivity; Anxious-Shy; Perfectionism; Social Problems. Specifically related to the DSM-IV diagnostic criteria for ADHD, three further subscales provide scores for: ADHD Hyperactive-Impulsive type; ADHD Inattentive type; ADHD Combined type. In addition to this the Conners' ADHD Index provides information about the probability of a diagnosis of ADHD.

1.5.10.2 Strengths and Difficulties Questionnaire (SDQ)

The SDQ is a screening measure for emotional and behavioural problems in children and adolescents (Goodman, 1999). Based on the diagnostic criteria of the DSM-IV and ICD-10, it is favoured for being short, and for examining competencies as well as problem behaviour (Goodman & Scott, 1999). While the Child Behavioural Check List (CBCL; Achenbach, 1991) has long been viewed as the "gold standard" in assessing childhood problems, the SDQ has quickly become one of the most utilised screening instruments by clinicians and researchers (Stone, Otten, Engels, Vermulst & Janssens, 2010). The SDQ comes in child, parent, and teacher versions and produces scores across five indices: Emotional symptoms; Hyperactivity; Conduct problems; Peer problems; and Prosocial behaviour. For broader population samples it has recently been proposed that emotional and peer items are combined into an 'internalising' subscale, and the behavioural and hyperactive items into an 'externalising' subscale (Goodman, Lamping, & Ploubidis, 2010). See Appendix E to H for parental and child subscale item definitions and cut points.

1.5.10.3 Reliability and validity of teachers' and parents' subjective ratings

As diagnostic definitions of ADHD changed and motor activity and impulsivity were grouped together on the basis of factor analytic findings (DuPaul, Barkley & Conners, 1998), rating scales based on these criteria became the principal tools to assist diagnosis. Yet as a

result, the assessment of hyperactivity using the CRS is derived from a scale that measures both hyperactivity and impulsivity. Whereas in the past diagnosis of ADHD was made mostly from the history provided from the parents, the publication of DSM-IV (APA, 1994) brought with it a requirement to demonstrate the pervasiveness of the behaviour across multiple settings, so both teacher and parent ratings were used. While DSM-V (APA, 2013) removes this requirement, symptoms still have to be present in multiple environments. Personal correspondence with a senior paediatrician suggests that in NZ information from multiple domains is sought during the assessment phase, in keeping with the 2001 NZMOH guidelines (NZMOH, 2001).

For research purposes the concurrent validity of teacher and parental reports is calculated by correlating one scale with the other, but the consistency between the measures does not validate either of them (Licht & Tryon, 2009). As Rapport et al. (2006) highlight, as well as the absence of a standard or meaningful unit of activity within or across scales and raters, both parental and teacher ratings are only weakly correlated with objective measurements of activity. Validation of the measures used to monitor children's hyperactivity is instead derived by referring to the same scales used to quantify the behaviour. Essentially, reliability is mistaken for validity and it remains to be demonstrated if either parental or teacher reports are accurate sources of data (Tryon, 2009). Such inadequate measurement units, the lack of age and sex norms available for activity, and potential rater inaccuracies, all limit the usefulness of such scales for assessing whether a child exceeds developmentally appropriate levels of activity (Rapport et al., 2006).

Parent and teacher ratings of ADHD behaviours taken from behavioural questionnaires tend to correlate at a modest 0.3 (Saudino, Ronald, & Plomin, 2005). Using the ABC dataset at age 7 Cornforth (2007) found that parent and teacher agreement for all Conners' behavioural subscales was poor to low, a finding consistent with other studies

(Amador-Campos, Forns-Santacana, Guardia-Olmos & Pero-Cebollero, 2006; Antrop, Roeyers, Oosterlaan & Van Oost, 2002; Mitsis, McKay, Schulz, Newcorn & Halperin, 2000; Wolraich et al. 2004). The presence of discrepancies between parents and teachers assessments does not however necessarily mean that either report is inaccurate. Such differences may represent the context dependent nature of the behaviour and the behavioural expectations of the situation in which the child is observed. As such each rater may be providing an insight in to the child's behaviour in a particular environment (Achenbach, McCoaughly, & Howell, 1987) with evidence that both parents and teachers interpret symptoms relative to the environment in which the child is observed (Gadow et al., 2004). As Barkley (2006a) identifies, while DSM-IV requires that ADHD symptoms must be evident in two domains, this does not mean that both parental and teacher ratings must surpass the diagnostic threshold of 6 out of 9 symptoms. Mitsis et al. (2000) demonstrate that such an agreement is unlikely to be found. Yet if certain symptoms of the disorder are less consistently noticeable across environments, there is a possibility that the historic requirement for such agreement could have led to these symptoms being discounted and certain ADHD subtypes being under-diagnosed. For example, hyperactivity that is clearly evident in the home environment, may manifest itself as fidgeting in a classroom setting, something that may be more easily missed by a teacher. A trunk mounted actigraph, however, is likely to detect both types of movement and provide a measure free from rater bias.

Consistent with other studies in the literature (Antrop, et al., 2002; Cornforth, 2007; Deng, Liu, & Roosa, 2004) found that when the children in the current data set were aged 7 years of age, parental ratings on the Conners' were generally higher than teacher ratings. Contrary to previous studies (Achenbach et al., 1987; Deng et al., 2004; Loeber, 1991), however, she found that agreement between the two ratings was not higher for externalising than internalising problems. Consistent with two other population based studies (Deng et al.,

2004; Kolko & Kazdin, 1993), but perhaps counter-intuitively, agreement also declined as behavioural problems increased. Like Deng et al. (2004), Cornforth (2007) found that parent and teacher agreement was highest for the Conners' Cognitive Problems subscale. Interestingly, given that observations of activity level at home have been found to correlate only modestly with observations at school (Fagot & O'Brien, 1994), Cornforth (2007) found a stronger association between parent and teacher reports of hyperactive-impulsive symptom, than inattentive symptoms (.45 vs .34). Yet Brewis (2002) found that parent and teachers ratings factored together across these symptom domains based on who was reporting the behaviour, rather than the behaviour being reported. When they report hyperactivity and inattention on rating scales, teachers and parents therefore appear not to be describing two distinct behavioural patterns.

Teachers' ratings may be affected by a child's academic performance or social abilities (Verhulst, Koot, & Van der Ende, 1994). Teachers have also been shown to rate children as hyperactive when they are: inattentive; oppositional; disruptive; aggressive; have a negative relationship with student; or think that the student needs extra services (Licht & Tryon, 2009). Children receiving higher teacher activity ratings than other children in the same classroom have been found to be less motorically active (Tryon & Pinto, 1994), again highlighting the problems with subjective observations. This is of particular note given that teacher ratings of problem behaviour on the Behaviour Assessment System for Children (BASC; Reynolds & Kamphaus, 1998) have been shown to both increase, and fall in inter-rater reliability, as the children are perceived to be more hyperactive (Kenney, Ninness, Rumph, Bradfield & Cost, 2004). Together this suggests the possibility of a negative halo effect associated with perceived activity, a judgement which itself may be inaccurate when gauged against objective measures.

Teacher ratings tend to show lower heritability estimates than parental ratings (Wood, Rijsdijk, Saudino, Asherson & Kuntsi, 2008); a statistic that estimates how much variation in a phenotypic is due to genetic variation. Yet when actigraphs have been used to monitor activity in the home, significant shared environmental effects have been found. Such patterns do not emerge when parental ratings are used (Saudino & Zapfe, 2008). Parents tend to exaggerate sibling differences on activity level (Saudino, Wertz, Gagne & Chawla, 2004) and even agreement between parents is not good. Parental stress was the only variable that predicted such discrepancies in ratings (Langberg et al., 2010).

In summary, subjective ratings of hyperactivity are conflated with impulsivity and do not correlate strongly with objective measures. They are also likely to vary across environments, both due to genuine differences, and due to inconsistencies between parental and teacher perceptions. In addition subjective judgements can lead to behavioural issues being mistaken for hyperactivity and perceived hyperactivity can lead to negative assessments across broader domains. Finally, differing measures can influence the assessment of the proportion of heritable versus environmental influences on activity level. Based on these factors, the potential appeal of utilising objective measures during assessment quickly becomes evident.

1.5.11 Objective measurement of activity

The shifting focus of ADHD research in the 1960's, to defining hyperactivity as the disorder's dominant feature, brought with it the development of a number of new methodologies for assessing activity. As Rapport et al. (2009) identify, empirical validation of over-activity was demonstrated using a range of techniques, including: rating scales (Werry, 1968); analogue measures (Barkley, 1991); direct observation (Whalen et al., 1978); pedometers (Plomin & Foch, 1981); stablimetric cushions (Conners & Kronsberg, 1985); and actigraphs (Porinno, Rapoport, Behar, Sceery et al. 1983).

An Actigraph is a unidirectional accelerometer that records acceleration in the vertical plane and most typically aggregates this data into 1 minute long epochs. The complexity of interpreting actigraph data is considerable. In addition to there being a number of potential sampling modes for determining when activity is recorded (see Rapport et al., 2006, for a technical discussion), there are a number of possible methodologies for discerning children with ADHD from controls. The majority of the studies discussed in the following sections use a mean measure of activity, however activity can be partitioned in to periods of sedentary, moderate, and vigorous activity. Despite the fact that average activity per minute is often used in research, a paper by Teicher (1995) provides a compelling argument for the advantages of using distributions of activity scores across these levels of activity intensity. This is because their hyperactivity appears to be more defined by the absence of settled periods, rather than the presence of extreme activity.

1.5.11.1 Are hyperactive children distinguishable from normal controls using objective measure of activity?

The methodology of attaching actigraph monitors to the waists of children across a week long period is considered the most appropriate for monitoring the over-activity symptoms associated ADHD (Licht & Tryon, 2009). In addition to reliability being increased by the aggregation of data across multiple days (Wood et al. 2008), this methodology detects the key diagnostic characteristics of the disorder. This includes squirming while seated, leaving the seat, and being on the go as if "driven by a motor" (DSM-V, APA, 2013; Licht & Tryon, 2009). Only two published studies could be found that utilise this methodology and they demonstrated largely conflicting results. Porrino, Rapoport, Behar, Sceery et al., (1983) compared 12 boys who met DSM-III criteria for ADHD and 12 controls. They reported that the ADHD group were significantly more active across all settings and that this activity was later reduced by stimulant medication (Porrino, Rapoport, Behar, Ismond & Bunney, 1983).

Tryon and Pinto (1994) found however that only 15 of 22 children rated as "clinically hyperactive" by teachers on the CRS were more active than children that they rated as "normal". Such a discrepancy may however have been explained by the apparently cruder measuring device used by Tryon and Pinto (1994), which is likely to have been less sensitive to seated movement.

More recently, using contemporary actigraphs, Licht and Tryon (2010) also found that only 1 out of the 9 children in their study diagnosed with ADHD-C was pervasively hyperactive across settings. The majority of other studies have only looked at activity in one setting, such as the testing environment or the classroom across a single day. These studies have supported Porrino, Rapoport, Behar, Sceery et al.'s (1983) findings that children with ADHD demonstrate significantly higher levels of physical activity than normal controls (Dane, Schachar & Tannock, 2000; Halperin, Matier, Gedi, Sharma & Newcorn, 1992; Halperin et al. 1993; Inoue et al, 1998; Porrino, Rapoport, Behar, Sceery et al.1983). McGrath, Handwerk, Armstrong, Lucas, and Friman (2004) examined classroom activity using a wrist mounted actigraph across three days. Of three objective measures of behaviour utilised, only the actigraph yielded statistically significance between children diagnosed with ADHD on the basis of the Diagnostic Interview Schedule for Children (DISC; Shaffer, Fisher, Lucas, Dulcan & Schwab-Stone, 2000). As such, while the evidence is mixed, there appears to be definite scope for ADHD children to be distinguished from normal controls using actigraphy.

1.5.11.2 Can actigraphy distinguish between ADHD and psychiatric controls?

Discrimination of ADHD children using activity measures is less clear when compared to non-ADHD psychiatric controls. Matier-Sharma, Perachio, Newcorn, Sharma and Halperin (1995) however suggest that actigraphs may provide information to aid the

differentiation of ADHD from other childhood psychiatric disorders, with a positive predictive power of .77 for activity level in the ADHD versus non-ADHD patient comparison. Despite high specificity when distinguishing between disorders in a clinical setting, Matier-Sharma et al. (1995) did however consider that actigraphy was not appropriate for routine screening of ADHD among a normal population because of low sensitivity. On the basis of this study's findings Barkley concluded that the use of activity measurement is not supported in distinguishing children with ADHD from either normal of psychiatric controls (Barkley, 2006a). However, the Matier-Sharma et al. (1995) study only examined activity for the duration of the psychometric test session and the children in the study were diagnosed using DSM-III-R, so a number may have met the contemporary criteria for ADHD-I. If this was the case, and a subset of these children was less active, the sensitivity of actigraphy in identifying children with ADHD-H and ADHD-C may have been underestimated.

Inoue et al. (1998) found that by combining actigraphy with the Continuous Performance Test (CPT) and the Matching Familiar Figure Test (MFFT), sensitivity and specificity could be increased to over 75%. This study was again only across the duration of a single psychometric test session and DSM-III-R diagnostic criteria were used, so the composition of the sample in terms of modern subtypes of the disorder is unknown. More recently actigraphs have been shown to be able to differentiate between children with ADHD and with Pervasive Developmental Disorder (PDD; Tsujii et al., 2009).

Teicher et al. (2006) found that compared to other psychiatric-controls that children with ADHD exhibited significantly higher diurnal activity levels, with a lower percentage of periods spent at lower activity levels. Wood et al. (2009) also found that they could use Receiver Operating Characteristic (ROC) analysis to distinguish ADHD-C from controls on basis of the magnitude of movements and intra-individual variability of activity, and that

these measures showed high-familial correlations. As such there is encouraging scope for actigraphy to be used to help discriminate ADHD from other comorbid psychiatric conditions.

1.5.11.3 Can actigraphy be used to distinguish between ADHD subtypes?

There is a remarkable paucity of literature that looks at the relationship between objective measures of children's activity and their scores on behavioural rating scales. This includes information about whether activity correlates with the ADHD subtype scores derived from these scales. Dane et al. (2000) looked at the activity of children between the age of 7 and 12 diagnosed with either ADHD-I or ADHD-C. They found that there were no significant group differences in activity level in the morning session, but that children with ADHD were more active than controls in the afternoon. Such a finding may support a differential impact of fatigue on children with ADHD, potentially linked to research that children with the disorder exhibit disrupted sleep (Silvestri et al., 2009). Despite the prediction that they should be more active, children with ADHD-I in this study could however not be distinguished from children with ADHD-H using actigraphy. This study did however use a relatively small sample size of 64 and again only measured movement for a single day.

Ohashi, Vitaliano,Polcari and Teicher (2010) found that children with ADHD have a reduced ability to inhibit activity to lower levels, as well as higher levels of seated hyperactivity, measured using infrared motion analysis of their head position. These issues showed differential responses to methylphenidate, suggesting that gross activity and distractibility may be mediated by differing neural circuits. As a result, activity alone may not be sufficient to distinguish subtypes. Alderson, Rapport, Kasper, Sarver and Kofler. (2011) found evidence that activity level is associated with basic attentional, rather than behavioural inhibitory processes. While children with ADHD showed higher activity across all experimental conditions, they were no more active when trying to inhibit activity in a

conventional stop-signal task. This goes against the prediction of theories that suggest over-activity is a by-product of a child's inability to prevent task irrelevant information from gaining access to their working memory (Barkley, 1997; Sonuga-Barke, 2003). Instead it supports theories that increased levels of motor activity functions to increase cortical arousal in ADHD children to help overcome working memory deficits, and is therefore reactive to demands on central executive function (Rapport et al., 2009). To the authors knowledge no studies have looked specifically at the relationship between scores on the Conners' Rating Scales and actigraphic measurement of activity.

1.5.12 Activity and conduct disorder

As previously discussed when, considering common comorbidities with ADHD, there is evidence that the hyperactive symptoms of ADHD are more linked to disruptive disorders than inattentive symptoms. The most commonly considered comorbidity with ADHD is Oppositional Defiant Disorder (ODD), a pattern of disobedient, hostile and defiant behaviour towards authority figures diagnosed on average in 45-55% of children with ADHD (Barkley, DuPaul & McMurray, 1990; Barkley, 2006a; Wilens et al., 2002). Conduct Disorder (CD) is also found in 35-45% of children with ADHD. It is defined a persistent pattern of violating others rights or age appropriate norms. The ICD-10 diagnosis of hyperkinetic disorder is classified as presenting with or without CD (WHO, 2004) and there is evidence that ADHD with CD marks a severe subtype of ADHD with poorer outcomes (Barkley, Fisher, Smallish, & Fletcher, 2004). While ODD indicates a clear risk for early onset CD (Burke Waldman & Lahey, 2010), there is a commonly held view that ADHD precedes ODD, which precedes CD, which precedes anti-social personality disorder. This is not clearly supported by the evidence (Gillbert et al., 2004) and this has led to the separation of ADHD from disruptive disorders in DSM V (APA, 2013). Also, a large percentage of children with ODD do not develop CD (Maughan, Rowe, Messer, Goodman & Meltzer, 2004), and only a minority of children with

CD have been diagnosed with ODD (Burke et al., 2010). While this could of course represent the pathways by which children enter diagnostic settings, Frick and Nigg (2012) suggest that ODD in children provides important additional information about problems with emotion regulation that may lead to the development of emotional disorders.

While no research has been found examining the relationship between ODD and CD with activity, it is possible that pervasively high activity levels may be used to discriminate when these conditions are more likely to present or be comorbid with ADHD. The challenge then becomes distinguishing task inappropriate activity suggested by pervasively high activity, and task appropriate activity that is unrelated to behavioural issues. Utilising a self-report questionnaire Wiles et al. (2008) found that children between that ages of 11-14 who engaged in more physical exercise scored higher on a hyperactivity index employed by the study, but that there was no relationship to their behavioural problems index. Another consideration is the age of the child. The links drawn between hyperactivity and CD/ODD (Barkley et al., 1990; Barkley, 2006a; Wilens et al., 2002) have been demonstrated largely in pre-adolescent populations. In adolescents, many risk-behaviours commonly associated with CD may also be considered early warning signs for internalising problems, such as depression. Heger et al. (2014) have found that along with a lack of physical activity and sleeping problems, alcohol abuse, smoking, media use, risky sexual behaviour, and school absenteeism all showed an association with depression. As such this adolescence may mark a turning point in the direction of the relationship between activity and conduct problems.

1.6 Activity and Internalising Disorders

Since the teachings of Plato, there has been a general belief that physical activity has positive effects on mood and anxiety. Although few studies have examined this association prospectively, the ones that have (Abu-Omar, Rutten & Lehtinen, 2004; Haarasilta, Marttunen, Kaprio & Aro, 2004; Motl, Birnbaum, Kubik & Dishman 2004) support this

hypothesis (Strohle, 2009). This included a dose-response relationship between physical activity and mental health in 15 nations (Abu-Omar et al, 2004). In a sample of 2,548 adolescents Motl et al. (2004) found that naturally occurring changes in physical activity, appraised using questionnaires, were inversely related to depressive symptoms during early adolescence. After four years, those participants who engaged in regular physical activity also had lower incidence of other comorbid disorders, including somatoform and some anxiety disorders (Strohle et al., 2007).

Utilising actigraphs, Vallance et al. (2011) found that lower odds of depression were associated with increasing physical activity and decreasing sedentary activity, a trend more apparent within overweight participants. Given the increasing rates of childhood obesity in NZ (NZMOH, 2015), such findings may be particularly significant. The benefits of physical activity for anxiety have been less well studied, in part because of the clinical diversity of such disorders (Strohle, 2009). The meta-analyses that have been conducted show beneficial effect of exercise in both the general and clinical populations (Guszkowska, 2004; Petruzzello, Landers, Hatfield, Kubitz & Salazar, 1991). Wiles et al. (2008) found that 11-14 year olds who self-reported higher levels of physical exercise were likely to score lower on the Emotional Problems index of the SDQ, although the magnitude of this difference was reduced when gender was controlled for.

1.7 Sleep and Externalising Disorders

1.7.1 Sleep and ADHD

Sleep problems have long been associated with ADHD and were part of the diagnostic criterion of the disorder in DSM-III (APA, 1980). Until the introduction of actigraphy, however, it has been difficult to get cost-effective measures of sleep across large research samples. Using actigraphs, children with ADHD have been shown to have reduced sleep quantity and more disturbed sleep (Owens et al., 2009). Sleep disorders are relatively

common in children, with up to 10% of children suffering disrupted sleep (Gottlieb, 2003), but an estimated 25-50% of children and adolescents with ADHD experience problems with sleep (Weiss & Salpekar, 2010). There is considerable overlap between the neurobehavioural and neurocognitive consequences of sleep disorders and the symptoms of ADHD (Owens et al., 2013). As Owens et al. (2009) identify, several studies suggest that those children predominantly characterised by increased hyperactivity are at greater risk of experiencing impairments relating to sleep. A number of studies have failed to find a clear increase in sleep disorders in children with ADHD, but did observe increased activity during their sleep (Corkum, Tannock & Moldofsky, 1998; Konofal, Lecendreaux, Bouvard & Mouren-Simeoni, 2001) which may explain such associations.

The relationships between sleep and ADHD are complex and may develop as a consequence of multidirectional and multifactorial pathways (Spruyt & Gozal, 2011). Sleep problems may be a side-effect of ADHD, a causal factor for the disorder, or a common underlying dysfunction may be causing both ADHD and the sleep disruption (Nigg, 2006). Landau et al. (2010) found that at the age of 7 weeks old, infants at familial risk for ADHD show differences in the stability of their sleep patterns. Parental ratings and actigraphic data however provide evidence that ADHD is often related to impairments in sleep quality due to sleep related behaviour problems (Corkum, Tannock, Moldofsky, Hog-Johnson & Humphries, 2001). Such problems include bed-time resistance, sleep onset difficulties, and morning waking. Aside from any underlying physiological aetiologies, these problems themselves may exacerbate daytime inattention and hyperactivity in children. While such issues appear independent of medication side effects, their interpretation can however be complicated by the use of psychostimulants, which increase sleep-onset latency (Nigg, 2013). Spruyt and Gozal (2011) argue that sleep problems are still too often perceived as part of the symptoms of ADHD and not a primary disorder that then contribute to the symptoms.

1.7.2 Sleep and conduct disorder

In a sample of 91 chidren aged 6 to 11 years old a study employing actigraphy showed that a children between the ages who sleeps for 1 hour less than the average child may be at risk of conduct problems as assessed using the parental version of the SDQ (Holley, Hill & Stevenson, 2011).

A more detailed study supported these findings showing that children with short sleep duration were found to be at increased risk of rule-breaking and externalising behaviours at the age of 8, based on paternal ratings (Pesonen et al., 2010). They found no associations between continuous measures of average sleep duration and behavioural problems. The did however find that for children who slept less than 7.7 hours per night, corresponding to the lower tenth-percentile threshold for sleep duration, showed considerably more behavioural issues. Importantly these associations were accounted for by weekday results and were largely related to problems with attentional control and externalising behaviours. Even once age, gender, and the use of medication has been contro lled for, sleep quality remains an important correlate of executive function in children with ADHD (Moreau, Fouleau & Morin, 2013) It has been suggested that sleep difficulties should be regularly screened for when assessing children with conduct problems (Holley, Hill & Stevenson, 2011), but to add value to this as a diagnostic tool, it will first be important to establish local populations norms.

1.8 Sleep and Internalising Disorders

Studies using different methodologies and populations have estimated that 65% to 90% of the adult patients with major depression, and about 90% of children with this disorder, experience some kind of sleep problem (Harvard Mental Health Letter, 2009). A recent twin study involving 300 twin pairs found that sleep problems at age 8 predicted depression at the age of 10, suggesting that identifying issues with sleep early may provide opportunities to intervene to prevent later mood disorders (Gregory, Rijsdijk, Lau, Dahl & Eley, 2009). This

is supported by a longitudinal study with 1, 014 youth aged 13 to 16, which found that anxiety disorders preceded insomnia 73% of the time, while insomnia occurred first in 69% of comorbid insomnia and depression cases (Johnson, Roth, & Breslau, 2006). This study suggests that insomnia may potentially have distinct directional associations with anxiety disorders versus depression. A further study by the same research group found that the lifetime prevalence of insomnia was 10.7%, the median age of onset was 11 years, and that 52.8% of those affected had comorbid psychiatric disorders (Johnson, Roth, Schultz, & Breslau, 2006).

The onset of menses in girls led to a 2.75 increase in their risk for insomnia (Johnson, Roth, Schultz, et al., 2006). Alongside this, an increasing body of literature has found the link between early pubertal maturation and internalising problems, especially among girls. A recent study suggested this is linked to cortisol reactivity to interpersonal challenge, which is not as clearly present in boys (Natsuaki, Klimes-Dougan, Shirtcliff, Hastings & Zahn-Waxler, 2009). Zeiders, Doane and Adam (2011) have shown that more hours of sleep lead to steeper decline in cortisol across the day with a bidirectional relationship between sleep and hypothalamic-pituitary-adrenal (HPA) axis activity. Pesonen et al. (2010) found that for 8 year old children, irregularity in sleep duration from weekdays to weekends was most significantly associated with an increased risk for internalizing symptoms based on parental ratings. Such associations do not however appear limited to adolescence and childhood. In study of 35 undergraduates, Vanderlind et al. (2014) found, using both actigraphic measures and self-report measures, that reduced sleep quality predicted greater difficulty disengaging attention from negative stimuli. Research with the children in the current data supports these findings, showing that short sleep duration is associated with higher emotional lability scores on the Parental Conners' Rating Scale (Nixon et al., 2008).

1.9 Aetiology of ADHD

Despite the categorical nature of the diagnostic criteria for ADHD, recent genetic studies (Frazier, Youngstrom & Naugle, 2007; Larsson, Anckarsater, Rastam, Chang & Lichtenstein, 2012), as well as comparisons of neuropsychological and personality measures (Faraone et al., 2006; Faraone, Kunwar, Adamson & Biederman, 2009), have suggested that ADHD symptoms exist on a continuum of severity. This makes this discovery of single aetiological explanation unlikely, with the view that ADHD is influenced by both biological and environmental factors appearing well consolidated in the scientific literature. This can be used to argue against creating a false dichotomy between the two aetiological focusses (Cortese et al., 2011) or overemphasising the contribution of one approach over the other. This is especially true given recent studies highlighting clear gene-environment interactions in ADHD (Nigg, Nikolas & Burt, 2010).

1.9.1 Genetic evidence

Heritability estimates for ADHD in the behavioural genetic literature tend to range from 75 to 90%, with Russell Barkley's widely distributed 'factsheet' on ADHD emphasising the strong biological contributions to the disorder (Barkley, 2015). Again it is important to stress the influence of Barkley's work on broader field of ADHD, and more particularly on the assessment guidelines for ADHD in NZ. In his factsheet Barkley's equates ADHD's approximately "80% heritability" to the role of genetics in human height (Barkley, 2015). Such high estimates of heritability, largely based on twin studies, are often used to justify the broad dismissal of potential environmental influences. Yet such calculations must arguably be treated with caution, with evidence presented below to suggest that they are significantly inflated.

Pooled data from twin studies do indeed show that the concordance of ADHD is considerably higher in monozygotic (MZ) than in dizygotic twins (DZ; Hudziak & Faraone,

2010). For this to be compelling evidence on which to calculate such high heritability estimates, does however depend on the validity of the "Equal Environment Assumption" (EEA). The EEA asserts that the environmental factors are not more similar for identical twins (MZ) than they are for fraternal twins (DZ). This assumption is important, as when the concordance between MZ twins is higher than DZ twins, behavioural genetic modelling proportions this entirely to genetic factors. Critics of the EEA highlight how few studies discuss this assumption and argue that such increased concordance could be in part because MZ twins share more environmental factors (Joseph, 2003). If the EEA is violated then this could lead to a false inflation of heritability. In a contemporary paper Cortese et al. (2011) argue that "to our knowledge there are no published studies showing that violation of the EEA significantly impacts on the results of twin studies in ADHD." Of course, however, absence of evidence does not equate to evidence of absence.

The field of behavioural genetics was shaped by Plomin and Daniels (1987) seminal paper that posited that environmental influences were critically important to psychological and behavioural outcomes. They argued that these influences resulted primarily in differences between twins (non-shared environment effects; E), with genetic influences being almost fully responsible for sibling similarities (additive and dominant genetic effects; A and D). While non-shared environmental effects play a role in most temperamental and personality traits, the influence of the environment (shared environmental factors; C) has been shown to have a less significant role in psychopathology, with the notable exception of conduct disorders (Eaves et al., 1997; Nigg, 2006). ADHD has been shown to have higher heritability, with small non-shared environment effects and no shared environmental effects (Bergen, Gardner & Kendler, 2007). A recent meta-analysis of shared environmental factors using structural equation modelling (a weighted meta-analysis) supported such conclusions, suggesting that 70% of the variance of ADHD phenotype was due to additive and non-

additive genetic factors (Burt, 2009). Unlike other childhood psychopathologies, ADHD was the only one which did not find an influence of shared environment. Yet a number of complex methodological issues with such studies can account for the overestimation of genetic parameters, at the expense of shared environmental factors.

As Wood, Buitelaar, Rijsdijk, Asherson and Kuntsi (2010) identify, among the factors that lead to the inflation of genetic estimates, is the role of contrast effects. This is a form of rater bias, which occurs when the raters use the behaviour of one twin as a benchmark to assess the other. This results in an over-emphasis of the similarities of MZ twins and the differences of DZ twins. Such effects may be evident in both Parental SDQ and CRS measures of ADHD, where raters are asked to rate presence of symptoms and their severity. Methods used to correct for such contrast effects and for the non-normal distribution of such scales, can then contribute to the underestimation of shared environmental factors. Adjusting for such factors correctly Wood et al. (2010) calculate a reduced genetic estimate of variance of 60 %. Therefore, a simple methodological correction can open up 10 % variance in this study (20% variance from Barkley's [2015] estimates of heritability), that may be accounted for by environmental factors. In exploring the influence of such environmental factors on the over-activity component of ADHD, actigraphic measures offer the benefit of being free from potential rater bias.

1.9.2 Gene by environment interactions (GxE)

As Nigg (2006) identifies, genetic contributions to ADHD are almost certainly made up of small additive effects of many genes. This is supported by the fact that many of the candidate genes in ADHD have been found to occur in over 50% of the population. This also raises the likelihood that such genes exert their influence along with environmental triggers. Gene-environment interactions can be conceived as a genetic sensitivity to a particular environment, or the triggering of genetic vulnerability by such an environment (Purcell,

2002). Yet parents tend to pass on both genes and environment to their children. As such the child's behaviour may evoke a particular response from the environment, and their genes may also influence the environments they seek out (Scarr & McCartney, 1983). Such genotype-environment correlations further complicate interpretation. GxE interactions have not been well studied in ADHD, although relatively consistent evidence points to the interaction of genotype with psychosocial factors in the disorder (Nigg et al., 2010). Yet if common shared environmental effects are involved in such interactions and correlations, and are not explicitly examined, they can make MZ twins look more alike than DZ twins and be mistaken for purely genetic effects. Less common experiential risk factors may also influence the development of ADHD, but tend to fall into the category of non-shared factors (Nigg, 2006). When conducting research on ADHD, this highlights the importance of factoring in potentially relevant environmental influences as covariates into the analysis. The current study offers data on an array of such factors, which are explored further below.

1.9.3 Environmental risk factors for ADHD: Prenatal and perinatal risk factors

1.9.3.1 Low-birthweight

According to ICD-10 (WHO, 2004) low-birthweight (LBW) is defined as a new-born infant of less than 2500 grams. While LBW can be as a result of premature birth despite a typical rate of growth, all of the children in the current study are full-term but small for gestational age (SGA). This means that they weigh less than the 10th percentile, which for NZ males at 37 weeks is 2440 grams (Thompson, Mitchell & Borman, 1994). Some, but not all of children SGA children in the current sample will have experienced intrauterine growth retardation (IUGR). IUGR responsible for a large proportion of premature births and is a particularly significant factor of interest in the study of ADHD. While children with low birthweight (LBW) make up a relatively small proportion of the children with ADHD, recent

research shows that LBW is an independent risk factor for the disorder. Even when IQ is controlled for, children with ADHD are three times more likely than controls to have been born LBW (Mick, Biederman, Prince, Fisher, & Faraone, 2002). Lower birthweights have been associated with higher rates of hyperactive behaviour, assessed using the Teachers CRS (Pinto-Martin et al., 2004). The authors found that among a sample of 463 very-low to moderately-low birthweight (500-2000g) 9 year old children , when compared to the general population, twice as many (5.2%) had scores of more than two standard deviations above the mean on the hyperactivity scale of the CRS. The prevalence of hyperactivity was 2.6 times greater among children in special education classes; who had the lowest birthweights, showed increased evidence of brain injuries, and were almost exclusively preterm. While these very low-weight prematureb infants are not representative of the current sample, the association between lower birthweight and increased hyperactivity was also evident among the heavier range of LBW infants (1500-2000g). Supporting this finding, in a recent study of 789 twins falling in to this heavier range, Groen-Blokhuis, Middeldorp, Beijsterveldt and Boomsma (2011) found that among birthweight discordant MZ and DZ twins, the child with the lower birthweight scored consistently higher on CBCL (Achenback, 1991) measures of hyperactivity and attentional problems. This raises the possibility that SGA infants may also be at risk of such difficulties.

While studies have shown that LBW is associated with a higher incidence of clinically significant attention problems, a study of 11 year old children found that this relationship was only evident in urban and not suburban children (Breslau & Chilcoat, 2000); a finding that was replicated when the children were 17 (Bohnert & Breslau, 2008). The potential influence of protective factors was also demonstrated by Tully, Arsenault, Caspi, Moffitt, and Morgan (2004) who found that maternal warmth moderated the association between LBW and ADHD. These findings highlight the potential complexity of the

relationship between that LBW and ADHD. LBW has multiple potential causes and is likely to reflect an accumulation of psychosocial and physical risk factors (Nigg, 2006), yet many of these risk factors may themselves be independently related to ADHD. Such factors include maternal alcohol or nicotine use during pregnancy, environmental exposure to toxins, maternal stress and psychosocial adversity (Banerjee, Middleton & Faraone, 2007; Biederman, Faraone, & Monuteaux, 2002; Chomitz, Cheung, & Lieberman, 1995). While LBW children have been shown to experience more emotional and behavioural problems (Dahl et al., 2006), it does however appear that the strength of the association between LBW and ADHD is particularly robust. This remains true even when parental ADHD, parental antisocial behavioural disorders, maternal substance abuse during pregnancy, IQ, and social class are controlled for (Mick et al., 2002).

1.9.3.2 Prenatal Alcohol Exposure

Alcohol is recognised as a teratogenic agent that can cause central nervous system dysfunction and foetal alcohol syndrome (FAS). While distinct from ADHD, FAS is a condition that incorporates many of the core symptoms of disorder (Coles et al., 1997). Linnet et al (2003) reviewed nine studies investigating the association between prenatal alcohol exposure and ADHD and found a significant relationship in four of the studies, including two which involved high levels of exposure. Mick et al. (2002) found that twice as many children with ADHD had mothers who drank daily or binged during pregnancy than controls. Hill, Lowers, Locke-Wellman & Shen (2000) did however find that univariate associations between prenatal alcohol exposure and ADHD disappeared when factors such as smoking and parental psychopathology were controlled for. As is discussed in greater detail in the methodology section, multicollinearity is a significant factor in such research, where two or more predictor variables are highly correlated in a multiple regression. Despite this, significant associations have been found between maternal alcohol abuse and ADHD

(Knopik et al., 2006). There are also credible mechanisms that explain how maternal alcohol use during pregnancy may be linked to symptoms of ADHD, increased externalizing behaviour, and decreases in general cognitive function (Huizink & Mulder, 2006). It is not clear however whether these effects would present themselves as hyperactivity.

1.9.3.3 Nicotine Exposure

The damaging effects of maternal smoking during pregnancy have been studied extensively. Kotimaa et al. (2003) found strong evidence for a dose-response relationship between maternal smoking and hyperactivity, even after SES and maternal alcohol use were controlled for. Linnet et al. (2003) reviewed 24 studies and found that while the strength of the association varied slightly across the studies, the children of smokers were approximately three times more likely to have a clinical diagnosis of ADHD. In a community twin sample, utilising both parental and teacher ratings of ADHD, maternal smoking accounted for a small but significant amount of the variance (Thapar et al., 2003). No studies could be found looking at the effects of maternal smoking on objective measures of activity, but utilising parental report there are indications in the literature that both pre and post-natal smoking may lead to increased childhood activity (Eskenazi and Trupin, 1995; Weitzman, Gortmaker, & Sobol, 1992). There is however evidence that ADHD is associated with a greater tendency to smoke (Burke, Loeber, & Lahey, 2001). As such, if a mother with ADHD has an increased likelihood to smoke during pregnancy, the effects of nicotine in causing ADHD could be acting via the mediation of genetic effects. This possibility has been supported by a recent study employing a sibling design, whose authors found no support for prenatal smoking as a strong causal factor for ADHD. Instead they concluded that the association found in previous studies appears to have been accounted for by maternal ADHD genetics or shared family environment (Obel et al., 2015).

1.9.4 Experiential risk factors for ADHD: Developmental factors

1.9.4.1 Psychosocial Adversity

As a result of the broad dismissal of shared environmental factors in ADHD research, familial factors have received very little research attention in recent years. Greenburg, Speltz, DeKlyen and Jones (2001) considered four domains of correlates when studying the development of early conduct problems, including vulnerable child characteristics, poor parenting practices, insecure attachment and adverse family ecology. When three or more of these factors were present a dramatic increase in clinical status occurred. Given the high levels of comorbidity seen between ADHD and conduct problems, especially in boys, it would seem surprising if some of these factors did not influence the diagnosis of ADHD. Biederman et al. (2002) found that psychosocial adversity, (including low social class, maternal psychopathology, and family conflict) all increased the risk for ADHD independent of gender, but that boys were more at risk for adverse cognitive and interpersonal outcomes. A study by Jester et al. (2005) found that the development trajectory of aggression was moderated by within-home conflict, but that the trajectory of inattention and over-activity was moderated by lower emotional support and lower intellectual stimulation. Interestingly, a more recent study by Larsson, Dilshad, Lichtenstein and Barker (2011) found that all of the subtypes of ADHD were associated with higher rates of family risk environments, but that prevalence rates of family risk factors was highest in the hyperactive and combined trajectory subtypes.

A common misconception of the diathesis-stress model is that the biological disposition must be genetically determined, but stressful life events in early childhood and in-utero can themselves create a biological vulnerability. Early trauma can disrupt neural development in brain regions that govern inhibitory control (Teicher et al., 2003) and affect (Teicher, Andersen, Polcari, Anderson & Navalta, 2002). Such regions also appear to

underlie many of the features of ADHD (Nigg & Casey, 2005). ADHD is the most frequently diagnosed disorder among sexually abused children (Mcleer, Callaghan, Henry & Wallen, 1994). When abuse leads to Post-Traumatic Stress Disorder (PTSD) it is more likely that the children will be hyperactive (Glod & Teicher, 1996), so clinical consideration of these factors is required to ensure that abused children are not misdiagnosed as having ADHD (Weinstein, Staffelbach, & Biaggio, 2000); or at least that the impact of the abuse is recognised as part of the aetiology of any presenting problems (Read & Bentall, 2012). At its extreme, severe early deprivation such as that as seen in many Romanian orphans, can lead to higher levels of inattention and hyperactivity (O'Connor & Rutter, 2000). Such psychosocial stressors can also influence a child before they are born. In a sample of 8 and 9 year old children, maternal anxiety has been linked to ADHD, externalising problems, and anxiety (Van den Bergh & Marcoen, 2004).

1.9.4.2 Diet and obesity

Interest in dietary management of ADHD was first popularised by the sugar-restricted, additive and salicylate-free 'Feingold diet' (Feingold, 1975). Despite popular interest and a positive public response, scientific evidence to support such theories has been lacking (Millichap & Lee, 2012).

While sugar does not usually affect the behaviour or cognitive performance of children, it may do so in a small subset of children (Wolraich, Wilson, & White, 1995). Schab and Trinh (2004) also observed a small but significant increase in the symptoms of children with ADHD when they consumed food colourings. Children are known to be particularly sensitive to the effects of caffeine and it is notable that despite a recent increase in the popularity of highly caffeinated beverages, the influence of such products on child behaviour are poorly understood (Temple, 2009). There is also emerging evidence of potential links between ADHD and obesity (Pulgaron, 2013), and evidence that healthier

dietary patterns are associated with lower odds of having ADHD (Woo et al., 2014). A recent animal study has even shown a possible link between a high sugar maternal diet and ADHD-like behavioural phenotypes in offspring (Choi et al., 2015).

1.9.4.3 Electronic Media Exposure

In recent decades children's exposure to electronic media, including television and computer use, has increased rapidly in Western populations to around 3 hours a day. ADHD may be particularly associated with the overuse of such devices (Weiss, Baer, Allan, Saran & Schibuk, 2011). As with sleep, the direction of this association is difficult to establish, with the majority of research examining the content of electronic media rather than the duration of exposure. Early television exposure appears associated with attentional problems at age 7 (Christakis, Zimmerman, Di Giuseppe & McCarty, 2004), but this may be mediated by a link between television and irregular sleep schedules in young children (Thompson & Christakis, 2005). A recent large study found no meaningful relationship between television exposure, or parental limits on television watching, and symptoms of ADHD (Stevens & Muslow, 2006). It does appear likely that children who watch more television will be less active. Therefore controlling for electronic media exposure becomes important when examining the relationship between activity and reported symptoms of ADHD.

1.10 Previous relevant research from the ABC Study

Cornforth (2007) examined the rate and determinants of both preschool (Age 3.5; Phase 3) and school age (Age 7; Phase 4) children's emotional and behavioural problems, using the SDQ Parental Total Difficulties Score as her principal outcome measure. She found no evidence that SGA children are at increased risk of such problems, but did find the following factors to be associated with increased SDQ scores at age 3.5: young maternal school age; maternal and paternal smoking prior to the child's birth; high maternal stress in the last month of pregnancy; not living with their biological father at 3.5 years; and child

assessed by their parents to have high activity levels compared to their peers. The relationship with activity was fairly weak and disappeared during multivariate analysis. It was also limited in that it was based on a subjective rating by the parent about whether there child was more, less, or similarly active when compared to their peers. The following factors were associated with emotional and behavioural problems at both 3.5 and 7 years: exposure to environmental tobacco smoke at age 1 year; maternal stress in the last month of pregnancy; young paternal school leaving age; and the child's current IQ.

Cornforth (2007) also examined the level of agreement between parental and teacher reports of child behaviour on the CRS at age 7. She found generally poor levels of agreement, decreasing as the child behavioural problems increased, although agreement was found to be higher on the DSM-IV ADHD subscales. Looking specifically at the three DSM-IV ADHD indices on the Conners' (Combined, Hyperactive-Impulsive; Inattentive) she found that SGA children were at no greater risk of developing ADHD symptoms at 7 than AGA children. Increased ADHD index scores at 7 were associated with: high maternal stress; young maternal age at the time of the child's birth. Parents also reported increased ADHD symptoms at age 7 if their child did not live with their biological father or if the child had emotional and behavioural problems (assessed using the SDQ) at the age of 3.5 years. Teachers reported increased ADHD symptom scores for children who had lower IQ scores.

Han (2010) looked at variables associated with physical activity in 7 year old children and found that the following variables were positively associated with physical activity: living with biological father; having younger siblings; parents with higher support from friends. Variables that showed a negative association with physical activity included: lower parental education and watching more than 3 hours of television a day. Interestingly children with higher verbal IQs and from higher socio-economic-status households spent less time in moderate and vigorous physical activity. One of the most significant contributions of this

study was the exploration of a number of technical aspects relating to the use of Actigraph monitors with the study population, which will be discussed in the Methodology section. A recent study by Thompson et al. (2014) has also shown that the use of paracetamol during pregnancy is associated with behavioural difficulties (Parental SDQ Total Score at 7; and Parental and Child SDQ Total Score at 11) and ADHD (Parental CRS Scale at 7 and 11).

1.11 The current study

Changes in activity and sleep patterns remain central to the identification of many psychological difficulties, but are rarely assessed accurately. Within the diagnosis of ADHD however, the conceptual importance given to hyperactivity has varied greatly within the past century. Despite a recent trend towards viewing over-activity as being secondary to cognitive difficulties, it remains a diagnostic criterion for both the ADHD-C and ADHD-H subtypes of the disorder within the new DSM-V (APA, 2013). A review of the relevant research raised a number of interesting questions and identified gaps in the literature that it was hoped could be addressed using the current data set.

Firstly, the conflation of over-activity and impulsivity within DSM IV and V definitions of ADHD and hyperactivity, evident in subjective scales such as the CRS, makes it difficult to assess the criteria of over-activity independently. This has resulted from the initial symptom criteria being selected by clinicians, rather than using a more tradition psychometric approach, which starts with much larger item pools (Rapport, 2012, personal communication). While there may be a failure to differentiate over-activity from impulsivity using factor analytic studies, the two may be separated using an objective measure of activity. Such objective measures have been previously dismissed for diagnostic purposes for lacking ecological validity, but this is arguably premature given developing technologies. However, for a measure of over-activity to have any diagnostic utility in identifying children who require additional support, normative samples of activity must first be derived. With the

exception of the current study these are not available in a NZ context. The data set at age 11 provides such measures for a large sample of children and for a recording period of a week. A week is considered the minimum period to make a reliable appraisal of activity. Given that the presenting symptoms of childhood behavioural disorders are known to change as the child grows older, and differ between the sexes, age and gender specific norms are also required. As such the first objective of this study was to:

1) **Examine the actigraph measures of activity at age 11 and provide summary data that offers a preliminary normative sample in a NZ context. To do this a distinction must be made between the times each child spends awake asleep, thus also providing a measure of sleep duration. Given that the literature remains divided as to which measure is the most helpful to identify children with ADHD, activity was examined as: average daytime activity per minute; average proportion of awake spent in each of the three intensities of physical activity (sedentary, moderate, vigorous); and time per day spent in each of the three intensities of activity.**

There remains a limited literature about whether objective measures of activity are predictive of emotional and behavioural disorders in childhood. This current data set offers both parental and child ratings on the SDQ and appeared detailed enough to allow both overall and subscale scores to be examined against activity. Cornforth (2007) previously only found a weak relationship between Parental SDQ Total Difficulties scores and subjective ratings of activity using the current data set. Given evidence that there that externalising problems may have a positive relationship with activity, and internalising problems a negative relationship, the two may be cancelling each other out. To explore this hypothesis with the greatest available power, the relationship between activity and the SDQ scores was analysed using an internalising/externalising factor structure. It was also unclear from the literature how closely objective measures of activity associate with subjective measures of

ADHD derived from the Conners' Rating Scales; the behavioural rating scale recommended by the NZ Guidelines for the Assessment of ADHD. A clear hypothesis was that high scores on ADHD-H and ADHD-C subscales of the CRS should be distinguishable from the ADHD-I subscale based on activity.

It was apparent that parental and teacher ratings of child behaviour on the CRS are unique and poorly correlated, but what was less clear was whether either observers' ratings are more closely associated with objective hyperactivity. It was also unclear whether symptoms of motoric hyperactivity are more indicative of emotional behavioural problems at a younger age, with questions about the validity of the ADHD-H as children grow older. While the dataset does not identify children with clinical diagnoses of ADHD, evidence suggests that ADHD symptoms exist on a spectrum of severity. The current sample allowed the association between the CRS and objectively measured activity to be examined across the entire range of this spectrum. With both the SDQ and the CRS, it remained to be explored whether categorical or continuous outcomes are preferable when exploring such potential associations. It was also unclear whether the study results provide sufficient power and suitable distribution for analyses to be run on the subscales. Therefore, the second objective of this study was to:

2) **Conduct univariate analysis to explore how strongly the each of the various measures of daytime physical activity at age 7 and 11 are associated with:**
    a. **Broader measures of emotional/behavioural function as measured by the Parental and Child versions of the Strengths and Difficulties Questionnaire at age 11. The scales that were intended to be examined included the Total Difficulties Score, each of the five SDQ subscale scores (hyperactivity; conduct; emotional; peer; prosocial), and the Internalising and Externalising Problem scores.**

b. Symptoms of ADHD as measured by the Teachers and Parental versions of the Conners' Rating scales at age 11. The scales that were intended to be examined were the Conners' Index, and the three DSM-IV subscales (ADHD-C; ADHD-H; ADHD-I).

c. **Based on the normality and distribution of the SDQ and Conners' data and this univariate analysis the most appropriate measures would be taken through to the multivariate regression analysis.**

This study includes a wealth of measures that have been identified as significant predictors of emotional and behavioural problems in children, both by previous studies using this data set, and in the current literature review. The third objective of this study was to:

3) Conduct univariate analyses to explore the relationship between activity and potential predictors available to the study (see Section 2.5 and Table 31). Given the likely interrelationship between these variables, multicolinearity analysis would be conducted before they were progressed to the stage of multivariate analyses. A model would be created for each of the outcome measures included within this set of analyses, exploring the proportion of variance that the available predictors can explain within the outcome measures.

A number of studies have shown that hyperactive children are more likely to have sleep problems. As identified it appears that sleep is often viewed as a symptom dimension of ADHD, rather than a driver of the symptoms themselves. As with activity, while the direction of the relationship may not be able to be determined, it was aimed to include measures of sleep duration within all relevant analyses. It was hypothesised that children with higher scores on both externalising and internalising SDQ indices, and with higher scores on the Conners', would show shorter sleep durations.

2 General Methodology

2.1 Introduction

This thesis utilises data collected as part of the Auckland Birthweight Collaborative (ABC) study. A brief history of this study is provided, followed by a more detailed consideration of the methodology of the current research.

2.2 Auckland Birthweight Collaborative (ABC) Study Data Collection

The ABC Study was originally conceived as a case-control study to identify modifiable risk factors related to the growth and development of children of low birthweight. The study therefore employed disproportionate sampling, with approximately half of the children born small for their gestational age (SGA). At present data collection has been completed at: birth (Phase 1); 1 year of age (Phase 2); 3.5 years of age (Phase3); 7 years of age (Phase 4); and 11 years of age (Phase 5).

2.2.1 Recruitment and eligibility

The participants in this study were born between 16th October 1995 and the 30th November 1997 in the Waitemata or Auckland Healthcare districts. During the recruitment period a computer programme generated a daily report of all infants born on the previous day, whose mothers were then approached to seek their consent. All were born at 37 weeks of gestation or more, with SGA infants defined as those who had a birthweight that was equal to or below the tenth percentile, corrected for gestational age and gender using NZ norms (Thompson et al. 1994). All SGA infants born in this time period were selected and a random sample of children born at a weight appropriate for their gestation age (AGA) was also selected. Infants were not eligible for inclusion if they were from a multiple birth, or suffered from any congenital abnormalities that were likely to affect their birthweight or subsequent growth and development. Infants delivered outside of a hospital were also not eligible, due to a lack of available obstetric data for these births. Of the 2308 mothers and infants that were

identified as potential participants, 126 were excluded due to the following reasons: adoption (n=3); congenital abnormalities (n=107); maternal psychiatric illness (n=6); and not being a resident in the study area (n=10).

2.2.2 Phases

2.2.2.1 Phase 1 data collection (Birth)

Obstetric information was available through the Auckland Maternity Services Information System (AMSIS). Additional, information was gathered from the mothers of the infants by interviewer administered questionnaire in the week following delivery. This included information about maternal health and nutrition during pregnancy, as well as family, sociodemographic, and lifestyle/environmental factors. Those measures utilised by this study are explained in more detailed in Section 2.5.

2.2.2.2 Phase 2 data collection (Age 1)

Mothers were sent a self-administered questionnaire which included measurements of their child's developmental progress using items adapted from the Revised Denver Pre-screening Developmental Questionnaire. In addition information about child health and nutrition (including respiratory health and allergies), socio-demographic, and lifestyle/environmental factors was obtained.

2.2.2.3 Phase 3 data collection (Age 3.5)

Mother and child attended an assessment at the Starship Children's Research Centre, in central Auckland. This involved the collection of data relating to a range of measures including child growth, allergies, iron status and serum fatty acid levels, Child IQ measured using the Stanford Binet Intelligence Scale 4th Edition (Thorndike, Hagen, & Sattler, 1986); and child behaviour using the Parental SDQ. Mothers were also interviewed about their child's diet, health, socio-demographic, lifestyle/environmental factors, and the mothers own perceived level of stress and social support.

2.2.2.4 Phase 4 data collection (Age 7)

Mother and child attended an assessment at the Starship Children's Research Centre. Data collected included child growth and development, physical activity (24 hour period of activity recorded using a waist mounted Actigraph), iron status and serum fatty acid levels, spirometry, child IQ (using the WISC-III; Wechsler, 1991), and child behaviour (Parental SDQ; Conners' Teacher and Parent Rating Scales). Parents were also interviewed about their child's diet, health, socio-demographic, lifestyle/environmental factors, and the mother's own perceived level of stress and social support.

2.2.2.5 Phase 5 data collection (Age 11)

Mother and child attended an assessment at the Starship Children's Research Centre. Data collected included child growth and development, physical activity (7 day period of activity recorded using a waist mounted Actigraph), iron status and serum fatty acid levels, spirometry, child IQ (using the WASI; Wechsler, 1999), and child behaviour (Parental and Self-Report SDQ; Conners' Teacher and Parent Rating Scales). Parents were also interviewed about their child's diet, health, socio-demographic, lifestyle/environmental factors, and the mother's own perceived level of stress and social support. To support the Actigraph recordings parents kept a diary of the times that they believed their children went to sleep and woke up, and both the child and their parents also logged when the monitor was taken off. The Actigraphs were not waterproof, so swimming is an example of an activity when it was necessary for the monitors to be removed.

2.2.3 Ethical approval

Each phase of the ABC Study has been approved by the local ethics committee, including Phase 5 data collection being approved by the Health and Disability Northern Y Regional Ethics Committee (NTY/06/11/112). Parents gave written informed consent for their own and their child's participation. Information is stored against participant numbers

rather than their names, with data protection procedures employed to ensure the security and confidential of all records. The relevant legislation governing the collection and storage of personal information is the NZ Privacy Act (1993).

2.2.4 Participant retention and sample restriction

Of the 2,182 potential participants that remained after eligibility criteria had been implemented during Phase 1, a further 498 declined to be involved, withdrew from the study, or could not be contacted. This left 1,714 participants, of whom 1,093 returned completed questionnaires at Phase 2, and 710 attended the assessment at Phase 3. Due to differential response rates at Phase 2 and 3 between mothers who identified as European or non-European at the time of their child's birth, a decision was made to restrict the study to European participants at Phase 3. Of the 1,714 participants who provided complete information at birth, 871 were the children of NZ European mothers. Of these participants 744 returned completed questionnaires at Phase 2, and 550 provided data at Phase 3.

A total of 591 children were assessed at Phase 4, including those who attended one or both of the Phase 2 and 3 assessments. At Phase 5 a total of 620 children were followed up at the age of 11 and this data formed the basis of the current study. All available data was used for each section of the SDQ and CRS normative analyses, but as return rates varied across measures this led to differing N's depending on the variables included. For the purposes of the main analyses, useable activity data was collected successfully from a total of 561 children. Of these children 540 also has data for both the CRS and SDQ indices explored, which was utilised alongside data collected from the same participants at earlier phases.

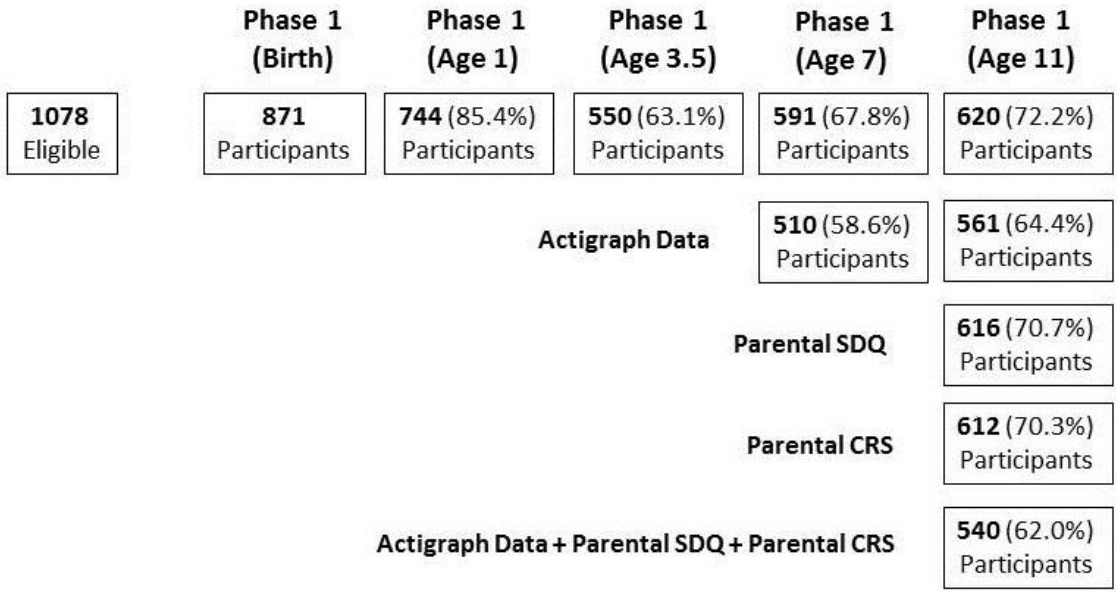

Figure 5: Number of European participants contributing data at each phase of the ABC Study

2.2.5 Screening for sample selection biases

The loss of participants in longitudinal studies can alter the composition of the sample over time, potentially introducing sample selection biases that can compromise validity. Previous analyses have found that compared to the sample at birth, respondents at 3.5 and 7 years were more likely to be older at the birth of their child, married, of high social economic status, have a tertiary education, and to be non-smokers (p<0.001; Cornforth, 2007). Table 3 presents comparative demographic information for those who were included in the present analysis at age 11 and those from the assessment at birth who either dropped out of the study or were excluded. As can be seen in Table 3, a similar pattern to previous phases was found at age 11, with participants more likely to been born: AGA; to an older mother; into a higher income family; to parents who were married or in a de facto relationship; to a mother with lower perceived stress at birth; and to a mother who did not smoke. Given also the exclusion

of children with non-European mothers at an earlier phase, some caution must therefore be employed about generalising the findings of this study to the broader NZ population.

Table 3

Analyses for selection biases between current sample and original study participants who did not provide data

Demographic	**Current sample** **N = 620**	**Those who did not provide data** **N=1135**	**Significance of between-group difference** **(ANOVA or Chi Square)**
Gender (N, %)			
Male	313 (***50.5***)	550 (***48.5***)	
Female	307 (***49.5***)	544 (***47.9***)	*Chi square* (1)= .934, P = .960
Birthweight (N, %)			
AGA	360 (***58.1***)	510 (***46.6***)	
SGA	260 (***41.9***)	584 (***54.4***)	Chi square (1)= 20.745**, P <0.001**
Maternal age at birth (N, %)			
<25	61 (***9.8***)	255 (***22.5***)	
25-34	371 (***59.8***)	655 (***57.7***)	
35+	180 (***29.0***)	169 (***14.9***)	*Chi square* (2)= 74.794, ***P*<.001**
Family income at birth (N, %)			
< $15,000	19 (***3.1***)	181 (***15.9***)	
$15,000 - $25,000	34 (***5.5***)	276 (***24.3***)	
$25,001 - $35,000	77 (***12.4***)	186 (***16.4***)	
> $35,000	479 (***77.3***)	356 (***31.4***)	*Chi square* (3)= 306.893, ***P*<.001**
Marital status at birth (N, %)			
Other	155 (***25.0***)	383 (***35.0***)	
Married/De facto	455 (***73.4***)	710 (***65.0***)	*Chi square* (1)= 17.907, ***P*<.001**
Stress at birth (Mean/SD)	**11.81** (6.63)	**14.76** (6.94)	*F*(91, 1698) = 73.161, ***P*<.001**
Maternal smoking at birth (N, %)			
Yes	137 (*22.4*)	305 (*28.1*)	
No	475 (*77.6*)	780 (*71.9*)	*Chi square* (1)= 6.658, ***P*=.011**

Note. N = Sample Size; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; SD = Standard deviation

2.3 Predictor Variables

2.3.1 Daytime activity

An objective measure of the child's activity was recorded for a period of one day at the age of 7 and for a period of one week at the age of 11 years. Activity was measured using a waist mounted Actigraph monitor.

2.3.2 Actigraphy

The actigraphs used in this study were unidirectional accelerometers that recorded acceleration in the vertical plane and aggregated this data into 1 minute long epochs; although the modern equivalents are now multi-axial/three-dimensional. Certain conditions should be met to ensure the validity of actigraphic measures of activity, including employing appropriate instruments and algorithms, recording over multiple days, and taking all necessary steps to limit artefact. The actigraphs (AM71256 Accelerometer, Actigraph Corporation) used in this study detect motion between 0.05 and 3.2 Gs, parameters designed to detect normal human motion, but exclude motion from other sources. The Actigraph monitor used in this study is of well-established reliability (Welk, Schaben, & Morrow, 2004) and validity, correlating well with both polysomnographic measures of sleep (Sadeh & Acebo, 2002) and a variety of measures of physical activity such as oxygen consumption and calometry (Wood, Kuntsi, Asherson & Saudino, 2008). The placement of the monitor around the waist was an established study protocol, but has been found by Licht and Tyron (2009) to be suitable for monitoring ADHD related movement, including squirming while seated.

2.3.3 Removal of potential sources of artefact and defining daytime activity

One of the major initial tasks of this research was to remove potential sources of artefact from the Actigraph data and to define periods of daytime activity. The first stage of this process involved transferring the data into SAS (Version 9.1), where the Sadeh algorithm

(Sadeh, Alster, Urbach, & Lavie, 1989) was run to define the period each night that the child was asleep. This algorithm identifies the first of three consecutive minutes of zero-activity after the parental reported bedtime and the first of five consecutive minutes of zero-activity prior to the parental reported wake time. All of these times were then verified manually, to prevent inaccurate parental reports from limiting the accuracy of the algorithm. This process was completed by two independent raters. Where there were discrepancies of greater than 15 minutes, the project's lead data analyst independently assessed each case. Where no satisfactory conclusion could be reached the data was excluded. In addition to providing a measure of sleep duration, defining the hours that each child was awake was required before their daytime activity could be analysed. The second stage of the process was to manually identify and exclude from the final data set any periods when the monitors were taken off. While participants were required to keep records of these periods, which were used to guide this work, these logs proved to be fairly inaccurate. In addition, experience from previous use of the monitors has shown that they are often knocked or moved during the period they are taken off, making it almost impossible to exclude this data using an algorithm. The positioning of the monitor around the waist was designed to minimise potential sources of artefact. Activity counts of above 20,000 were assumed to be spurious and excluded from the final daytime data set. Periods of 10 consecutive minutes of zero recordings or greater were also removed from analysis. This was an established project protocol as the sensitivity of the monitors would suggest that they had been taken off at these times, or that the children were asleep. In either circumstance, to derive an accurate measure of daytime activity, exclusion of these time periods was considered necessary.

2.3.4 Activity at age 11

Actigraph data was collected successfully from 561 children, who form the sample for this research. The mean age of the children in this sample was 11.4 years, 51.7% were male

and 41.9% were born small for gestational age (SGA). In addition to providing the basis for further analyses, this descriptive data provides a normative sample of the activity of eleven year old European children in New Zealand.

Evident in the preparation of the literature review of this thesis was the broad array of different measures of activity employed in the research literature, determined largely by the theoretical interests of the study. Much of the literature that looks at physical activity is linked to obesity and risk factors for cardio-vascular disease, as was the original rationale for the inclusion of activity data in the ABC study. Freedson, Pober, and Janz (2005) recommended that for such research, activity cut-offs are based on energy expenditure. As such the current study uses cut-off values defined by the Trost equation which are linked to Metabolic Equivalents Units (MET); METs=2.757 + (0.0015 x counts/min)-(0.08957 x age[yr])-(0.000038 x counts/min x age[yr]). The Trost equation takes into account the children's age and has been shown to exhibit the highest classification accuracy (Trost, Way, & Okely, 2006). The child's mean activity was calculated by dividing their total activity count during wake hours, by the number of hours they were awake. The proportion of awake hours spent in either sedentary (<3 METs = Actigraph count of 0-1135; equivalent to watching television), moderate (<6 METs = Actigraph count of 1136-3908; equivalent to walking), or vigorous (>6 METs = Actigraph count of 3909-20000; equivalent to running) activity were then calculated. The Trost equation was previously found by Han (2010) to be the most appropriate formula to use to analyse the current dataset when the children were 7 years old.

2.3.5 Sleep Duration

Utilising the Sadeh (Sadeh et al., 1989) algorithm (described on p. 62) to define sleep-wake times, the average duration of sleep was recorded at age 7 (one night) and 11 (one

week). This data was used both continuously and categorically (>10.5; 9.5-10.49; 8.5-9.49; <8.5 hours).

2.4 Outcome Measures

2.4.1 Strengths and Difficulties Questionnaire (SDQ)

The SDQ is a screening measure for emotional and behavioural problems in children and adolescents (Goodman, 1999). The questionnaire is available in three formats: parent (child age 3-16); teacher (child age 3-16); and self-report (child age 11-16). The SDQ asks about 25 attributes, both positive and negative, focussing on the child's behaviour over the past six months. Based on the nosological concepts of the DSM-IV (APA, 2000) and ICD-10 (WHO, 2004), it is favoured for being short and for examining competencies as well as problem behaviour (Goodman & Scott, 1999). The SDQ website (www.sdqinfo.com) provides all versions of the questionnaire as well as scoring information and normative data.

2.4.2 SDQ scoring and subscales

The SDQ contains 25 questions that each relate to a particular attribute. They are answered using a three-point Likert scale, depending on how much each attribute appears to apply to the target child (0=not true; 1=somewhat true; 2=certainly true). Scores from 0 to 10 are produced for five subscales, each derived from five questions: Emotional symptoms; Hyperactivity; Conduct problems; Peer problems; and Prosocial behaviour. The Total Difficulties score is calculated by summing all scores, with the exception of the Prosocial subscale, giving a number between 0 and 40. Clinical cuts offs were developed using cumulative frequencies (Goodman, 1997) the highest 10% of scores are labelled 'abnormal' (Total score 17-40), the next 10% 'borderline' (Total score 14-16) and the remainder 'normal (Total score 0-13). See Appendix E to H for parental and child subscale item definitions and cut points.

The five subscales were refined through exploratory factor analysis (EFA: Goodman, 1997) and are supported by other European EFA studies (Becker et al., 2006; Woerner et al., 2004) and by factor analysis of the SDQ data produced by the ABC study (Thompson & Waldie; in preparation). Yet as Goodman et al. (2010) highlight, EFAs are an exploratory technique, primarily useful in suggesting possible factor structures when these are not known. When a hypothesised factor structure exists it is more appropriate to use confirmatory factor analysis (CFA) and the CFA studies that have been carried out to date provide mixed support for a five-factor structure. One possible alternative that can be justified on theoretical grounds is to combine the emotional and peer items in to an 'internalising' and the behavioural and hyperactive items in to an 'externalising' subscale. This alternative two-factor structure was explored by Goodman et al. (2010). They concluded that there are advantages to its use for analyses in low-risk or general population samples, but that it remains preferable to retain all five subscales when screening for disorders. Given that the current study is interested in examining subgroups with clinically significant symptomatology, as well as more general trends across the entire sample, it was decided to use both the two and five factor scores in the analyses.

2.4.3 Psychometric properties of the SDQ

Goodman, Ford, Simmons, Gatward and Meltzer's (2000) original examination of the psychometric properties of the SDQ was based on a UK wide survey of 10,438 children and adolescents. Reliability was found to be generally satisfactory, with acceptable internal consistency (mean Cronbach alpha=0.73), a mean-cross informant correlation of 0.34, and a four to six month retest stability of 0.72. SDQ scores above the 90th percentile were found to predictive of a 15 fold increase of independently diagnosed psychiatric disorders. A recent meta-analysis of 48 studies by Stone et al. (2010) found that the internal consistency, test-retest reliability and inter-rater agreement are acceptable for the parent and teacher versions.

Correlations with other measures of psychopathology, as well of the screening ability of the SDQ, were found to be sufficient. Overall the study confirmed that the psychometric properties of the SDQ are strong, particularly for the teacher version.

2.4.4 SDQ in ABC Study

Although the SDQ is used extensively in child and adolescent mental health services (CAMHS) in NZ, only recently has research examined its psychometric properties within a NZ setting. This research was based on the current data set, finding the SDQ to be generally reliable and valid measure, and confirming the five factor structure (Thompson & Waldie; in preparation). For the Parental Scales used in the analyses of this thesis a total of 589 children at age 7 and 614 children at age 11 provided usable data on all five SDQ scales. A total of 542 children had complete information at both 7 and 11 years old.

2.4.5 Conners' Behavioural Rating Scale: Revised – Long Format (CRS-R:L)

The Conners' Rating Scale was originally developed to assess a wide variety of problem behaviour in children and as a measure of behavioural change for pharmacological studies (Conners, 1969). More recently it was expanded to include symptoms of ADHD and for the past decade has been a commonly used measure of the behavioural problems associated with this disorder (Conners, 1997). Available in both teacher and Parent versions it is the only behavioural rating scale that is recommended by 'NZ Guidelines for the Assessment and Treatment of ADHD' (NZMOH, 2001); in which the 'ADHD Index Scale' and 'DSM-IV Symptoms Scale' of both versions of the CRS-R:L are described as highly effective indices for discriminating between children with ADHD and normal controls. The scales were revised in 2008 and the most contemporary version is called the Conners' 3rd Edition (Conners, 2008).

2.4.6 CRS-R:L scoring and subscales

The parent and teacher versions of the CRS-R:L (Conners, 1997), used in this study, contain 80 and 59 items respectively. Each item is scored on a four point Likert scale depending on how much each statement applies to the child (0=not at all true; 1= just a little true; 2=pretty much true; 3=very much true). These scores are used to generate six core factors: Oppositional Problems; Cognitive Problems; Hyperactivity; Anxious-Shy; Perfectionism; Social Problems. The parent scale also contains a factor for Psychosomatic Problems. Three subscales referred to as the Conners' Global Indices (CGI) are also generated: CGI Restless-Impulsive subscale; CGI Emotional Lability; and CGI Total.

Specifically related to the DSM-IV diagnostic criteria for ADHD three further subscales provide scores for: ADHD Hyperactive-Impulsive type; ADHD Inattentive type; and ADHD Combined type. In addition to this the Conners' ADHD Index provides information about the probability of a diagnosis of ADHD. Subscale and index scores are converted to T-Scores, standardised by a child's age and sex, and compared to normative data. T-scores of 60-65 are considered mildly atypical, 66-70 are moderately atypical, and over 70 are markedly atypical (Robin, 1998). In this study all scores greater than or equal to 60 were classified in the borderline/abnormal range.

2.4.7 Psychometric properties of the CRS-R:L

Conners' (1996) original validation study of the CRS-R:L found the parental version (CPRS-R:L) to demonstrate excellent sensitivity and specificity, with the teachers version (CTRS-R:L) showing excellent specificity and moderate sensitivity. Internal consistency ranged from moderate to excellent, with test-retest reliability being more variable for parent's ratings than teachers (Conners, 1996). Agreement between parents and teachers is often modest (Collett, Ohan & Myers, 2003), but this may represent behavioural variability in different settings, rather than poor reliability.

2.4.8 CRS-R:L in the ABC Study

A total of 575 children at age 7 and 620 children at age 11 provided usable data on the Parental scales used in the analyses of this thesis. A total of 536 children had complete information at both 7 and 11 years old. For the purposes of this study the Conners' ADHD Index and the three DSM related ADHD subscales were examined.

2.5 Covariates

The following section details the variables that were examined as potential covariates. Those that showed a significant relationship with the outcome variables ($p \leq 0.01$) became candidate variables for multivariate analysis.

2.5.1 Biological variables

2.5.1.1 Sex of Child

The sex of each child was entered into the AMSIS database at the time of birth. It was also recorded at each phase of the study for validity purposes.

2.5.1.2 Birthweight

All infants in this study were born at full-term, which was defined as having a gestational age of 37 weeks or greater since the mother's last menstrual period. Infant birthweight, measured in grams, was recorded into the AMSIS database at birth. Children weighing the same or more than the sex specific tenth percentile, using New Zealand norms (Thompson et al., 1994), were classified as being AGA. Those whose birthweight fell below the tenth percentile were defined as AGA. The sample was approximately evenly split between AGA and SGA.

2.5.2 Socio-demographic variables

2.5.2.1 Socio-Economic Status (SES)

An indicator of socio-economic status was derived from information that mother's provided at birth about their most recent employment and the occupation of their partner. The

Elley Irving Index (Elley & Irving, 1985) was used to code parental occupations on a rating scale of 1 (professional) to 7 (government benefit). The highest code of the two parents was used as an indicator of family SES. The occupational rating codes were collapsed into three categories: *high* (1 and 2); *medium* (3 and 4); and *low (*5, 6, and 7*).* A measure of family income at birth was also recorded into one of four categories: less than $15,000; between $15,000 and $25,000; between $25,001 and $35,000; and greater than $35,000.

2.5.2.2 Parental education

As a measure of parental education, both maternal and paternal school leaving age was recorded. Historically these were coded into one of two categories, representing the boundary between compulsory and elective education: less than 16 years old; and 16 years or older.

2.5.2.3 Marital status

Marital status was recorded at the time of the child's birth, and also at 3.5 and 7 years. The data was coded into one of two categories: married and de facto; and other (single, never married, separated, divorced, and widowed). Recordings were also made as to whether the child lived with their biological father at each age.

2.5.3 Maternal and perinatal variables

2.5.3.1 Maternal age

Maternal age at the time of birth was drawn from the AMSIS database.

2.5.3.2 Maternal smoking

Information about smoking was derived from the maternal questionnaire as this was previously found to show excellent agreement with obstetric records. For the purpose of the current study the following variables are utilised: whether the mother smoked during pregnancy; and the number of cigarettes smoked per day in each trimester of pregnancy.

2.5.3.3 Paternal smoking

Mothers were asked at the times of birth if the biological father of their child was a smoker and if so how many cigarettes they smoked per day.

2.5.3.4 Maternal cannabis use

It was recorded whether or not the mother smoked cannabis during pregnancy.

2.5.3.5 Maternal paracetamol use

It was recorded whether or not the mother had taken paracetamol during pregnancy.

2.5.3.6 Maternal alcohol consumption

Maternal alcohol consumption was recorded during the maternal interview at birth. Previous analyses have coded this consumption into two variables, looking at the number of units of alcohol consumed per week in the first and last month of pregnancy: no alcohol; 7 or less units per week; more than 7 units per week. For the purpose of the current study this data was further recoded to include a variable that identified whether: no drinking occurred in either the first or last month of pregnancy; whether the mother drank 7 units or less during either the first or last month of pregnancy; or whether the mother drank more than 7 units during either the first or last month of pregnancy.

2.5.3.7 Maternal stress

Maternal perceived stress was assessed using the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983). The assessments took place at birth, 3.5, and 7 years of age and used the PSS-10, which is a 10 item abbreviated version of the 14 item full scale. Respondents are asked to rate the degree to they perceive that events over the previous month have exceeded their ability to cope, a measure that appears to be more associated with physical health outcomes than life event scales (Cohen et al., 1983). Abnormal stress reactivity is increasingly being identified as a vulnerability factor for both physical and psychological health problems and early environmental adversity has been linked to changes in physiological reactivity to stress for abused and neglected children (Evans et al., 2013;

Gunnar & Fisher, 2006). Individual, developmental, environmental, and substance use related factors have been shown to influence both perceived physiological stress and cortisol reactions in adolescents. Such a relationship is not evident in children, suggesting that the impact of early stressors may remain latent until a child becomes older (Evans et al., 2013).

For the purposes of the current study, raw PSS scores from birth, 3.5, and 7 years were recoded as dichotomous variables. Scores at or above the 75th percentile were coded as *high* stress, with all other scores being coded as *low* stress.

2.5.3.8 Maternal social support

Mothers were asked at birth, as well as at the 1 and 3.5 year follow ups, to appraise the social support that they had received over the previous six months. This was assessed using the Family Support Scale (FSS; Dunst, Jenkins, & Trivette, 1984), producing measures of both formal and informal support, as well as an overall index of total support. Formal support includes services from healthcare agencies and paid carers and informal support includes that provided by partners and family members. The FSS includes 18 questions scored on a 5-point Likert scale ranging from *not at all helpful* to *extremely helpful*.

2.5.4 Child variables

2.5.4.1 Anthropometry

The children's weight and height was recorded at each phase, and Body Mass Index (BMI) calculated and converted to BMI z-scores according to published norms at ages 3.5, 7, and 11. As well as being used as continuous variables, their BMI's were categorised into one of three classifications: underweight/normal; and overweight. This cut-off was calculated using Z-scores based on UK norms (Cole, Freeman, & Preece, 1995) and equated to a BMI cut-point of 20.55

2.5.4.2 Electronic media exposure

At the age of 7 the duration each child spent viewing television daily was recorded using parental questionnaires. The data was collected using the following intervals: less than 1; 1 to 3; 3 to 5; more than 5 hours. At the age of 11 recording was extended to both TV and computer exposure outside of school hours. This was recorded separately for weekdays and the weekend, into half hour intervals. Each interval was recoded so that the value represented the most exposure possible within that range (e.g. 0-0.5 hours =0.5). This meant that when the TV and computer values were combined the score represented the maximum exposure to electronic media the child could have received, to the nearest half an hour. Given the availability of this data, the decision was then made to categorise into equal 1.5 hour intervals to provide a more even distribution of children between each grouping: less than 1.5; 1.5-3; 3-4.5; more than 4.5 hours.

2.5.4.3 Reading age at 7

Reading ages were obtained from the children's Year 2 teachers' reports and were recorded as both continuous and categorical variables; by using a median split.

2.5.4.4 Intellectual function

The children were administered the Stanford Binet intelligence scale (4^{th} edition; Thorndike et al., 1986) at the age of 3.5 years, the Wechsler Intelligence Scale for Children Third Edition (WISC-III; Wechsler, 1991) at 7, and the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) at age 11. Both the WISC and the WASI yield three IQ indices, the Verbal IQ (VIQ); Performance IQ (PIQ); and Full-Scale/Overall IQ (FSIQ). Also examined was the WISC digit span subscale standard score at age 7, as a measure of working memory performance; there is evidence that hyperactivity may serve as a compensatory behaviour in children with impaired working memory (Sarver et al., 2015).

2.5.4.5 Depression

The Centre for Epidemiological Studies Depression Scale Revised (CESD-R self-report questionnaire was administered to the children at age 11 (Radloff, 1977; Eaton, Muntaner, Smith, Tien & Ybarra, 2004). This scale measures symptoms defined by contemporary DSM criteria for a major depressive episode. The scores were used continuously.

3 Data Screening

In this section the outcome of data screening is presented, details are provided about how missing data were handled, and the data is examined for violations of the assumptions of multivariate analysis. This includes the normality, distribution, linearity, and heteroscedasticity of the SDQ and Conners' scores (outcome variables), as well as of the activity data (predictor variable).

3.1 Missing Data

Data was analysed using SPSS Version 22.0 (IBM Corp., 2013). Attrition analysis is included in the methodology section and has shown a disproportionate loss of lower SES participants from the study. In addition to the specific methods used to ensure the accuracy of the Actigraph data (see Methodology section), the minimum and maximum values of each survey item were examined for plausibility. Tabachnick and Fidell (2012) state that as a general rule, variables containing missing data in 5% or fewer cases can be ignored. Subjects for the current study were selected on the basis that they had provided data on the major outcome variables. Participants had already shown significant commitment to a longitudinal study and as such, missing data fell well below this threshold. The SPSS protocols employed dealt with such missing values by excluding them from analysis. Where individual scale scores were missing the value defaulted to the lowest risk group. As such the remaining data was not lost and any effect was in a conservative direction. All available data was used to calculate normative values, as is apparent from the variable participant numbers listed. For the main analyses subjects were required to provide usable activity data, as well as Parental SDQ and CRS data. Of the 561 participants who provided activity data, 545 also provided the required questionnaire data. Activity data from 5 participants was excluded due to a failure to come to a consensus about their sleep or wake times. This left data from 540 participants to be included in the main modelling section of the results.

3.2 Normality

The following tables summarise the normality characteristics of the outcome measures (Parent and Child SDQ; Parent and Teacher CRS) and also of the predictor variables at age 11 (activity measures).

Table 4

Normality of SDQ scores at Age 11

	Variance	Skewness (SE)	Kurtosis (SE)	Kolmogorov-Smirnova (K-S, df, Sig)
Parental SDQ				
Total Difficulties	26.369	.860 (.099)	.532 (.197)	.112, 614, p<.001
Emotion	3.498	1.141 (.099)	1.065 (.197)	.214, 614, p<.001
Conduct	2.166	1.389 (.099)	2.234 (.197)	.228, 614, p<.001
Hyperactivity	5.115	.716 (.099)	.128 (.197)	.142, 614, p<.001
Peer	2.656	1.761 (.099)	2.982 (.197)	.262, 614, p<.001
Prosocial	3.094	-.968 (.099)	.585 (.197)	.194, 614, p<.001
Child SDQ				
Total Difficulties	25.316	.496 (.098)	.043 (.196)	.066, 618, p<.001
Emotion	3.853	.974 (.098)	.915 (.196)	.171, 618, p<.001
Conduct	2.518	.903 (.098)	1.043 (.196)	.173, 618, p<.001
Hyperactivity	4.299	.153 (.098)	-.329 (.196)	.110, 618, p<.001
Peer	2.534	1.036 (.098)	1.374 (.196)	.180, 618, p<.001
Prosocial	2.749	-.598 (.098)	-.180 (.196)	.171, 618, p<.001

Note. SDQ = Strengths and Difficulties Questionnaire; SE = Standard Error; df = degrees of freedom; K-S = Kolmogorov-Smirnova; Sig = Significance level.

As seen in Table 4, the distribution of scores for all subscales was positively skewed (prosocial index is reverse scored), with skewness and kurtosis values reflecting a non-normal distribution. This was true even when a less rigorous test than the Kolmogorov-Smirnova was employed. Tabachnick and Fidell (2006) suggest that skewness should be considered of note if the statistic is greater than twice the standard error of the skew, which it was across all of the SDQ Parental and Child scales at age 11.

Continuous SDQ scores were also grouped into an Internalising (Emotion, Peer) and Externalising (Conduct, Hyperactivity) score. As previously explained, internalising and externalising disorders are often associated with a respective increase and decrease in activity. It was hypothesised that potentially differential relationships between activity and these groupings may be reducing the strength of the relationship between activity and Total Difficulties scores. By combining subscales this two factor grouping was seen as the most statistically robust method available to permit later exploration of this hypothesis. Normality data for these two groupings on the Parental and Child SDQ date are presented in Table 5.

Table 5

Normality of Parental and Child SDQ Internalising and Externalising scores (two-factor)

	Variance	**Skewness (SE)**	**Kurtosis (SE)**	**Kolmogorov-Smirnova (K-S, df, Sig)**
		Parental SDQ		
Externalising	10.300	.891 (.099)	.732 (.197)	.126, 614, *p*<.001
Internalising	3.070	1.306 (.099)	1.780 (.197)	.167, 614, *p*<.001
		Child SDQ		
Externalising	10.074	.423 (.098)	.115 (.196)	.92, 618, *p*<.001
Internalising	8.356	.970 (.098)	1.349 (.196)	.136, 618, *p*<.001

Note. SDQ = Strengths and Difficulties Questionnaire; SE = Standard Error; K-S = Kolmogorov-Smirnova; df = degrees of freedom; Sig = Significance level.

Unsurprisingly, given the skewed SDQ Total Difficulties scores, the distributions of both internalising and externalising scores were found to be positively skewed, with skewness and kurtosis values reflecting non-normal distributions.

The SDQ Total Difficulties score was logged, but still remained outside of the more relaxed criteria of normality as described by Tabachnick and Fidell (2006; Skewness=.860 [SE=.099]; Kurtosis=.532 [SE=.197]). As a result of this, because a clinical screening scale is expected to be skewed towards low symptomatology, and to permit more easily interpretable

results, the decision was made not to transform the scale scores used later in the regression analyses.

Table 6

Normality of Conners' ADHD indices

	Variance	Skewness (SE)	Kurtosis (SE)	Kolmogorov-Smirnova (K-S, df, Sig)
		Parent		
Conners' ADHD Index	41.786	1.277 (.100)	1.850 (.200)	.129, 596, *p*<.001
DSM Hyp/Imp	15.913	1.619 (.100)	3.471 (.200)	.159, 596, *p*<.001
DSM Inattentive	78.630	1.274 (.098)	1.825 (.197)	.147, 616, *p*<.001
DSM Combined	69.049	1.465 (.100)	2.754 (.200)	.118, 596, *p*<.001
		Teacher		
Conners' ADHD Index	12.283	1.629 (.100)	2.855 (.200)	.185, 596, *p*<.001
DSM Hyp/Imp	13.814	1.521 (.100)	2.221 (.200)	.207, 596, *p*<.001
DSM Inattentive	30.741	1.164 (.100)	.578 (.200)	.187, 597, *p*<.001
DSM Combined	32.215	1.422 (.100)	2.233 (.200)	.158, 596, *p*<.001

Note. ADHD = Attention Deficit Hyperactivity Disorder; SE = Standard Error; K-S = Kolmogorov-Smirnova; df = degrees of freedom; Sig = Significance level; DSM = Diagnostic and Statistical Manual of Mental Disorders; Hyp/Imp = Hyperactive/Impulsive Type

As can be seen in Table 6, the distribution of scores for all scales on the Conners', for both parents and teachers, were positively skewed, with skewness and kurtosis values reflecting a non-normal distribution. This was also the case when values were transformed to T-scores and logged. As with the SDQ, the decision was made not to transform the scores for the Conners' as such abnormality was to be expected, as well as to maintain interpretability.

Table 7

Normality of the activity measures

	Variance	Skewness (SE)	Kurtosis (SE)	Kolmogorov-Smirnova (K-S, df, Sig)
		Age 11		
Mean Counts	35437	1.570 (.105)	5.788 (.210)	.092, 540, *p*<.001
% Sedentary	22.8	-.351 (.105)	.124 (.210)	.044, 540, *p*=.013
% Moderate	16.4	.264 (.105)	.135 (.210)	.410, 540, *p*=.029
% Vigorous	1.4	2.041 (.105)	5.819 (.210)	.127, 540, *p*<.001
Mins Sedentary	3120	-.025 (.105)	.689 (.210)	.035, 540, *p*=.156
Mins Moderate	1147	.307 (.105)	.180 (.210)	.038, 540, *p*=.054
Mins Vigorous	94	2.106 (.105)	6.224 (.210)	.135, 540, *p*<.001
		Age 7		
Mean Counts	67123	.794 (.108)	.991 (.216)	.069, 509, *p*<.001
% Sedentary	66.9	-.069 (.107)	-.151 (.214)	.033, 509, *p*=.200
% Moderate	44.0	.001 (.107)	-.191 (.214)	.024, 509, *p*=.200
% Vigorous	11.2	.994 (.107)	.945 (.214)	.092, 509, *p*<.001
Mins Sedentary	7287	.019 (.107)	1.068 (.214)	.033, 517, *p*=.200
Mins Moderate	3155	.061 (.107)	-.060 (.214)	.021, 517, *p*=.200
Mins Vigorous	718	.998 (.107)	.909 (.214)	.100, 517, *p*<.001

Note. SE = Standard Error; K-S = Kolmogorov-Smirnova; df = degrees of freedom; Sig = Significance; Mins = Minutes

As can be seen from Table 7 mean activity levels at both ages 7 and 11 are significantly skewed towards lower levels of activity. When activity is partitioned into time spent in each of the three intensities of activity, the sedentary and moderate categories both fall into a range considered to be normal, even by the more stringent of two indicators employed (Kolmogorov-Smirnova).

3.3 Linearity

A visual inspection was conducted of the relationship between the key predictor and outcome variables and no obvious curvilinear relationships were observed. An ANOVA examining the relationship between these variables at age 11, found that the only significant

deviation from linearity was in the relationship between mean activity at 11 and the Parental SDQ Total difficulties score. It was noted in examining this particular bivariate plot that there were outliers present. Unfortunately, when the analysis was re-run with outliers excluded the assumption of linearity continued to be violated. Such a non-linear relationship was not apparent on the individual subscale scores. Looking at the relationship with the times spent in various intensities of activity, the effect appeared to be largely influenced by time spent in moderate activity. This suggests that any relationship may be driven by high and low scores on the scale, consistent with the bimodal distribution often seen on clinical scales. This may potentially be exaggerated by less experienced parental raters, who often only have their other children against which to compare the child's behaviour. As such this particular variable may be more suited to categorical analysis based on established clinical cut-offs.

3.4 Heteroscedascticity

An examination of the variance of the outcome measures using Levene's test found them to be homoscedastic across grouping by both birthweight and gender.

3.5 Selection of Outcome Variables

While clinical questionnaire data is expected to be positively skewed towards low symptomatology, to use multivariate analysis the least skewed measures were selected at the primary outcome measures for the purposes of regression analysis. These were Parental SDQ Total Difficulties scores (continuous and categorically) and the Conners' Parental ADHD categorical scores. Categorical analysis permits an examination of clinical utility based on current diagnostic criteria, while the greater variability of a continuous measures may detect more subtle relationships and support the recent trend favouring dimensional interpretations of psychological difficulties.

3.6 Selection of Predictor Variables

While all activity measures will be utilised during the univariate analysis stage, due to their more normal distribution, time spent in sedentary and moderate activity were selected as predictor variables for regression analyses.

4 Results

The results of this study are presented in five sections. In Section 1 the SDQ and Conners' scores are summarised and examined for main effects of gender and birthweight (AGA, SGA). Also examined are actigraph measures of activity at age 11, offering a preliminary normative sample in a New Zealand context, as well as providing information about sleep duration. Given that the literature remains divided as to which activity measure is the most helpful measure to identify children with ADHD, activity is examined across a range of measures. These include: average daytime activity per minute; average proportion of awake spent in each of the three intensities of physical activity (sedentary, moderate, vigorous); and time per day spent in each of the three intensities of activity. In Section 2 changes in the outcome variables and in the activity and sleep recordings are examine across time between the ages of 7 to 11.

In Section 3 univariate analyses are presented which explore how strongly daytime physical activity and sleep duration at age 7 and 11 are associated with the outcome measures. As a result of normality analyses, the outcome measures selected in this section include: a broader measure of emotional/behavioural function, using both the continuous and categorical results from the Parental Total Difficulties score of the Strengths and Difficulties Questionnaire at age 11; and a measure of the likelihood of an ADHD diagnosis, using the categorical scoring of the Parental ADHD Index of the Conners' Rating scale at age 11. Also explored are the relationships between the outcome measures and variables within the dataset identified by the literature review as potential covariates. The interrelationships between the variables found to be significant predictors are then examined to screen for multicolinearity. In Section 4 predictor variables that show significant independent relationships with the outcome measures are included within a final stage of multivariate analysis. A model for each

of the three outcome measures identifies the proportion of variance that these variables explain.

Finally, in Section 5, due to the centrality of activity to many of the hypotheses in this thesis, further exploratory univariate analyses are conducted to inform future research. These analyses sought to examine whether the weak relationships found between activity and the outcome measures, may have been due to differential relationships between activity and outcome measures subscales. While normality restrictions prevented the inclusion of these subscales within formal modelling, their relationship with activity was tentatively examined. This included the Internalising and Externalising groupings of the continuous Parental SDQ scores, and the three continuous DSM ADHD scores of the Parental Conners' Rating Scale (Hyperactive/Impulsive, Inattentive, and Combined).

4.1 Section 1: Overall Performance

In this section, performance of the sample at age 11 is presented across measures. This begins with the behavioural data that serve as the outcome measures (SDQ and CRS). This includes analyses of the impact of gender and birthweight (AGA versus SGA) on these scores. This is followed by a summary of the actigraphic data, including the provision of sleep duration, that are used as predictor variables. Again the impact of gender and birthweight on these data are examined. These results can also be viewed as a preliminary normative data set for the activity and sleep patterns of 11 year old NZ European children

4.1.1 Outcome measures at 11 (SDQ and Conners')

4.1.1.1 Parental Strengths and Difficulties Questionnaire (SDQ) at 11

Parental SDQ data was collected for 616 children at the age of 11. There were approximately equal numbers of male (n=311, 50.5%) and female (n=305, 49.5%) children. Of these children just over half were AGA (n= 357, 58.0%) and the remainder were SGA

(n=259, 42%). Child SDQ data was collected for 619 children, with a near identical distribution. Summary data for the SDQ scale scores is presented in Tables 8 to 15.

Table 8

Parental SDQ mean scores and standard deviations (SD) for birthweight, sex, and the total sample at Age 11

	AGA N=357	SGA N=259	Male N=311	Female N=305	Total N=616
Total Difficulties	**6.8** (4.9)	**7.9** (5.4)	**8.1** (5.4)	**6.4** (4.7)	**7.2** (5.1)
Emotion	**1.7** (1.7)	**2.1** (2.0)	**1.8** (1.9)	**2.0** (1.8)	**1.9** (1.9)
Conduct	**1.2** (1.4)	**1.4** (1.6)	**1.4** (1.6)	**1.1** (1.3)	**1.3** (1.5)
Hyperactivity	**2.7** (2.2)	**3.1** (2.3)	**3.5** (2.4)	**2.3** (2.0)	**2.9** (2.3)
Peer	**1.2** (1.6)	**1.2** (1.7)	**1.4** (1.8)	**1.0** (1.5)	**1.2** (1.6)
Prosocial	**8.3** (1.7)	**8.2** (1.8)	**7.9** (1.8)	**8.6** (1.6)	**8.3** (1.8)

Note. SDQ = Strengths and Difficulties Questionnaire; SD = Standard Deviation, AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size.

As can be seen in Table 8, Total Difficulties scores are on average higher for both the SGA group and for the Male group, compared to the AGA and female groups. As shown in Table 9, differences between AGA and SGA become more apparent that when the results are presented separately across gender, especially among the males participants.

Table 9

Parental SDQ mean scores and standard deviations (SD) for sex by birthweight at Age 11

	Male AGA N=181/182	Male SGA N=129/130	Female AGA N=175	Female SGA N=130
Total Difficulties	**7.5** (5.1)	**8.9** (5.6)	**6.0** (4.5)	**6.9** (5.0)
Emotion	**1.5** (1.7)	**2.1** (2.1)	**1.9** (1.8)	**2.2** (1.9)
Conduct	**1.6** (1.7)	**1.6** (1.7)	**1.0** (1.3)	**1.2** (1.4)
Hyperactivity	**3.3** (2.3)	**3.8** (2.3)	**2.2** (1.9)	**2.5** (2.1)
Peer	**1.3** (1.8)	**1.4** (1.7)	**1.0** (1.4)	**1.1** (1.6)
Prosocial	**7.8** (1.8)	**7.8** (1.8)	**8.7** (1.5)	**8.5** (1.7)

Note. SDQ = Strengths and Difficulties Questionnaire; SD = Standard Deviation, AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size.

RESULTS

To determine if these patterns were statistically significant, a 2x2 MANOVA was conducted with gender (male, female) and birthweight (AGA, SGA) as grouping variables. As the principal outcome variable the Total Difficulties score was analysed first, along with the Prosocial subscale, before the four difficulty subscales were examined together separately. It was not possible to run a single analysis, as the Total Difficulties score is the sum of the four difficulty subscales, and thus not independent of these four measures.

The results of this first analysis indicated that for the Total Difficulties and Prosocial scales there was a significant main effect of gender [$F(2,609)=15.771$, $p<.001$], as well as a significant main effect of birthweight [$F(2,609)=4.413$, $p=.013$]. The interaction between birthweight and gender was not significant ($p>.05$). While the significant main effect of gender was evident across both scales ($p<.001$), the significant effect of birthweight was only evident on the Total Difficulty scale ($p=.006$) and not the Prosocial scale ($p=.885$).

A 2x2 MANOVA including the four difficulty subscales as outcome variables showed a significant main effect of both gender [$F(4, 607)=15.925$, $p<.001$] and birthweight [$F(4,607)=2.896$, $p=.022$], with no interaction between the two ($p<.05$). The following scales contributed significantly to the main effect of gender: Peer difficulties ($p=.007$), Hyperactivity ($p<.001$), and Conduct difficulties ($p=.009$). In each case, males produced significantly higher scores, indicating greater behavioural disturbance than females. The subscales that contributed to the main effect of birthweight were: Emotional difficulties ($p=.005$) and Hyperactivity ($p=.020$), with Conduct difficulties approaching significance ($p=.053$). These difficulties were all greater in SGA children. Caution must however be employed in interpreting these results due to the non-normal distribution of the scores. This remained evident even when the data was transformed, so the data was left in its original state for the sake of interpretability.

RESULTS

In addition to total scores, the SDQ data was examined in relation to established clinical cut-offs for normal, and abnormal/borderline behaviour. Table 10 presents this data for males and females by birthweight grouping.

Table 10

Percentage and frequency of Abnormal/Borderline and Normal scores across gender and by birthweight for all Parental SDQ scores at Age 11

	Male AGA N=181/182		**Male SGA N=129/130**		**Male Total N=310/312**		**Female AGA N=175**		**Female SGA N=130**		**Female Total N= 305**	
Total Difficulties		**%**		**%**		**%**		**%**		**%**		**%**
Abnormal/ Borderline	24	**13.3**	24	**18.8**	48	**15.5**	17	**9.7**	16	**12.3**	33	**10.8**
Normal	157	**86.7**	104	**81.3**	261	**84.5**	158	**90.3**	114	**87.7**	272	**89.2**
Emotion												
Abnormal/ Borderline	21	**11.5**	31	**24.0**	52	**16.7**	24	**13.7**	30	**23.1**	54	**17.7**
Normal	161	**88.5**	98	**76.0**	259	**83.3**	151	**86.3**	100	**76.9**	251	**82.3**
Conduct												
Abnormal/ Borderline	39	**21.4**	31	**24.0**	70	**22.5**	25	**14.3**	20	**15.4**	45	**14.8**
Normal	143	**78.6**	98	**76.0**	241	**77.5**	150	**85.7**	110	**84.6**	260	**85.2**
Hyperactivity												
Abnormal/ Borderline	27	**14.9**	25	**19.5**	52	**16.8**	8	**4.6**	11	**8.5**	19	**6.2**
Normal	154	**85.1**	103	**80.5**	257	**83.2**	167	**95.4**	119	**91.5**	286	**93.8**
Peer												
Abnormal/ Borderline	37	**20.3**	27	**20.9**	64	**20.6**	17	**9.7**	18	**13.8**	35	**11.5**
Normal	145	**79.7**	102	**79.1**	247	**79.4**	158	**90.3**	112	**86.2**	270	**88.5**
Prosocial												
Abnormal/ Borderline	23	**12.6**	16	**12.4**	39	**12.5**	8	**4.6**	9	**6.9**	17	**5.6**
Normal	159	**87.4**	113	**87.6**	272	**87.5**	167	**95.4**	121	**93.1**	288	**94.4**

Note. SDQ = Strengths and Difficulties Questionnaire; SD = Standard Deviation, AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size.

RESULTS

As can be seen in Table 10, when clinical cut-offs are used to categorise the Parental SDQ data at 11, SGA children show higher percentages of abnormal/borderline scales across all four of the difficulty subscales. These rates are higher among males on all of these scales, except for the Emotion subscale. A number of Chi-Square tests were run to determine whether these differences were significant. While a strong argument could be made to predict greater levels of problems among SGA children and males, these patterns had previously only been observed in males (Cornforth, 2007). In the absence of clear *a priori* hypotheses at age 11, the decision was made to report more conservative two-tailed significance values.

No overall effects of birthweight were seen on the Parental SDQ Total Difficulty categorisations [Chi-square(1)=2.076, p=.184]. However, a significant effect of birthweight was seen on the Emotion subscale [Chi-square(1)=12.626, p=.001; Males p=.005; Females p=.048] where SGA children were more likely than AGA children to fall in the borderline/abnormal category.

The effect of gender on the Parental SDQ Total Difficulty categorisations approached significance [Chi square (1)=2.979, p=.096]. Significant effects of gender were seen on the following subscales: Conduct [Chi-square(1)=6.097, p=.017; AGA p=.087; SGA p=.097]; Hyperactivity [Chi-square(1)=16.861, p<.001; AGA p=.001; SGA p=.012]; and Peer [Chi-square(1)=9.461, p=.002; AGA p=.007; SGA p=.143]. On all of these scales males were more likely than females to fall in the borderline/abnormal category. Of note, the Emotion subscale, which was the only one that did not show an effect of gender, was also the only scale to show an effect of birthweight.

Table 11

Two-factor Parental SDQ score means and standard deviations (SD) across gender and by birthweight

	Male AGA N=181	**Male SGA N=128**	**Male Total N=309**	**Female AGA N=175**	**Female SGA N=130**	**Female Total N=305**
Externalising	**4.6** (3.3)	**5.4** (3.5)	**4.9** (3.4)	**3.2** (2.8)	**3.7** (3.0)	**3.4** (2.9)
Internalising	**2.9** (2.8)	**3.5** (3.1)	**3.1** (3.0)	**2.9** (2.5)	**3.2** (3.0)	**2.0** (2.7)

Note. SDQ = Strengths and Difficulties Questionnaire; SD = Standard Deviation, AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size.

Multivariate Analysis of Variance (2x2 MANOVA) of the SDQ Parental Externalising and Internalising scores showed significant main effects of gender [$F(2, 609)=20.027$, $p<0.001$] and birthweight [$F(2,609)=3.900$, $p=.021$] but with no interaction between the two [$F(2,609)=.222$, $p=.801$]. For the Externalising scores males scored significantly higher than females ($p=.011$) and SGA higher than AGA ($p<.001$). For internalising scores there was only a main effect of birthweight with SGA children scoring significantly higher ($p=.034$) and thus exhibiting more symptoms of emotional and behavioural difficulties.

4.1.1.2 Child Strengths and Difficulties Questionnaire (SDQ) at 11

Child SDQ data was collected from 619 children at the age of 11. As with Parental SDQ data there were approximately equal numbers of male and female children, with slightly more AGA than SGA children represented. Means and standard deviations on the Child SDQ are presented by birthweight group and by gender in Table 12.

Table 12

Child SDQ mean scores and standard deviations (SD) for birthweight, gender, and the total sample at Age 11

	AGA N=359	**SGA N=260**	**Male N= 313**	**Female N=306**	**Total N=619**
Total Difficulties	**9.4** (4.9)	**10.6** (5.2)	**10.3** (4.9)	**9.5** (5.1)	**9.9** (5.0)
Emotion	**2.3** (1.9)	**2.6** (2.1)	**2.2** (1.9)	**2.6** (2.0)	**2.4** (2.0)
Conduct	**1.9** (1.5)	**2.2** (1.7)	**2.2** (1.6)	**1.8** (1.5)	**2.0** (1.6)
Hyperactivity	**3.5** (2.0)	**4.1** (2.1)	**4.0** (2.0)	**3.5** (2.1)	**3.7** (2.1)
Peer	**1.7** (1.6)	**1.9** (1.6)	**1.9** (1.6)	**1.6** (1.6)	**1.8** (1.6)
Prosocial	**7.7** (1.6)	**7.7** (1.7)	**7.2** (1.7)	**8.2** (1.5)	**7.7** (1.7)

Note. SDQ = Strengths and Difficulties Questionnaire; SD = Standard Deviation, AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size.

As shown in Table 12, while less pronounced than the trends shown by Parental SDQ data, self-reported Child SDQ scores highlight similarly consistent patterns. Illustrated by Table 13, Male and SGA children experience more emotional and behavioural difficulties, with the exception of the Emotion subscale where SGA females score the highest on average.

Table 13

Child SDQ mean scores and standard deviations (SD) for gender by birthweight at Age 11

	Male AGA N=183	**Male SGA N=130**	**Female AGA N=176**	**Female SGA N=130**
Total Difficulties	**9.7** (4.7)	**11.2** (5.1)	**9.0** (5.0)	**10.1** (5.2)
Emotion	**2.1** (1.8)	**2.4** (2.0)	**2.5** (1.9)	**2.8** (2.1)
Conduct	**2.0** (1.5)	**2.4** (1.8)	**1.8** (1.6)	**1.9** (1.5)
Hyperactivity	**3.8** (1.9)	**4.3** (2.2)	**3.2** (2.1)	**3.8** (2.0)
Peer	**1.8** (1.5)	**2.1** (1.6)	**1.5** (1.6)	**1.6** (1.5)
Prosocial	**7.2** (1.7)	**7.2** (1.7)	**8.2** (1.4)	**8.1** (1.5)

Note. SDQ = Strengths and Difficulties Questionnaire; SD = Standard Deviation, AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size.

To further examine this pattern a 2x2 MANOVA was conducted with Child SDQ scores as the dependent variables. For the Total Difficulty and Prosocial scores the results

showed significant main effects of both gender [$F(2,613)=27.233$, $p=.<.001$] and birthweight [$F(2,613)=5.446$, $p=.005$], but no interaction between the two ($p>.05$). As with the Parental SDQ Scores at 11, while both scales showed significant effects of gender (<.05), the effect of birthweight was only seen in the Total Difficulties score ($p=0.001$) and not the Prosocial score ($p>.05$).

A 2x2 MANOVA containing the four Child SDQ difficulty subscales also showed main effects of gender [$F(4, 611)=9.581$, $p=.001$], and birthweight [$F(4,611)=3.163$, $p=.014$], with no interaction between the two ($p>.05$). For gender, the scales that contributed to this effect were: Peer ($p=.005$); Hyperactivity ($p=.002$); Emotion ($p=.010$); and Conduct ($p=.002$). Males performed worse across each of these indices, with the exception of Emotion. For birthweight, the contributing scales were Hyperactivity ($p=.001$) and Emotion ($p=.039$), with Conduct ($p=.056$) approaching significance. SGA children again performed worse than AGA children on each of these measures. These results must again be interpreted with caution due to the non-normal distribution of these scores.

As with parental SDQ, it is possible to examine child SDQ scores both in terms of clinical cut-offs for normal, borderline and abnormal behaviour, and also through collapsing the scales into those reflecting internalizing and externalizing behaviours. These are shown in Tables 14 and 15 below.

Looking at Table 14, it is apparent that categorising the Child SDQ data using clinical cut-offs also highlights a general trend for male and SGA children to show higher levels of clinically significant self-reported symptoms of behavioural and emotional difficulties. A number of Chi-Square test were run to explore the significance of these differences, again reporting two-tail significances in the absence of clear directional hypotheses.

RESULTS

An overall effect of birthweight was seen on the Child SDQ Total Difficulty categorisations, [Chi-square(1)=5.079, *p*=.030; Males *p*=.064; Females *p*=.237], but not on any of the subscales, with males showing greater levels of difficulties.

The effect of gender on the Child SDQ Total Difficulty categorisations was not significant. A significant effects of gender was however seen on the Conduct subscale [Chi-square(1)=4.385, *p*=.041; AGA *p*=.260; SGA *p*=.092], with a greater incidence of conduct problems among males.

Table 14

Percentage and frequency of Abnormal/Borderline and Normal SDQ Child scores across gender and by birthweight at Age 11

	Male AGA N=183		**Male SGA N=130**		**Male Total N=313**		**Female AGA N=176**		**Female SGA N=129**		**Female Total N= 305**	
Total Difficulties		**%**		**%**		**%**		**%**		**%**		**%**
Abnormal/ Borderline	38	**20.8**	39	**30.0**	77	**24.6**	29	**16.5**	29	**22.5**	58	**19.0**
Normal	145	**79.2**	91	**70.0**	236	**75.4**	147	**83.5**	100	**77.5**	247	**81.0**
Emotion												
Abnormal/ Borderline	38	**20.8**	35	**26.9**	73	**23.3**	46	**26.1**	44	**33.8**	90	**29.4**
Normal	145	**79.2**	95	**73.1**	240	**76.7**	130	**73.9**	86	**66.2**	216	**70.6**
Conduct												
Abnormal/ Borderline	65	**35.5**	53	**40.8**	118	**37.7**	52	**29.5**	39	**30.0**	91	**29.7**
Normal	118	**64.5**	77	**59.2**	195	**62.3**	124	**70.5**	91	**70.0**	215	**70.3**
Hyperactivity												
Abnormal/ Borderline	34	**18.6**	32	**24.6**	66	**21.1**	28	**15.9**	22	**17.1**	50	**16.4**
Normal	149	**81.4**	98	**75.4**	247	**78.9**	148	**84.1**	107	**82.9**	255	**83.6**
Peer												
Abnormal/ Borderline	55	**30.1**	47	**36.2**	102	**32.6**	43	**24.4**	35	**26.9**	78	**25.5**
Normal	128	**69.9**	83	**63.8**	211	**67.4**	133	**75.6**	95	**73.1**	228	**74.5**
Prosocial												
Abnormal/ Borderline	28	**15.3**	24	**18.5**	52	**16.6**	10	**5.7**	12	**9.2**	22	**7.2**
Normal	155	**84.7**	106	**81.5**	261	**83.4**	166	**94.3**	118	**90.8**	284	**92.8**

Note. SDQ = Strengths and Difficulties Questionnaire; SD = Standard Deviation, AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size.

To explore the potential utility of a two-factor structure into which to categorise SDQ Child scores at 11, continuous scores were grouped into an Internalising (Emotion, Peer) and Externalising (Conduct, Hyperactivity) score. Table 15 shows measures of central tendency for this data.

Table 15

Two-factor Child SDQ score means and standard deviations (SD)

	Male AGA N=183	**Male SGA N=130**	**Male Total N=313**	**Female AGA N=176**	**Female SGA N=129**	**Female Total N=305**
Externalising	**5.8** (2.9)	**6.7** (3.5)	**6.2** (3.2)	**5.0** (3.2)	**5.7** (3.0)	**5.3** (3.1)
Internalising	**3.9** (2.7)	**4.4** (2.9)	**4.1** (2.8)	**4.0** (2.9)	**4.4** (3.1)	**4.2** (3.0)

Note. SDQ = Strengths and Difficulties Questionnaire; SD = Standard Deviation, AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size.

A 2x2 MANOVA of the SDQ Child Externalising and Internalising scores showed significant main effects of sex [$F(2, 613)=7.853$, $p<.001$] and of birthweight [$F(2,613)=10.0$, $p=.004$], with no interaction between the two. Males ($p<.001$) and SGA ($p=.002$) children showed significantly higher scores on the Externalising index. For the Internalising index SGA scored showed great difficulties ($p=.035$), but there was no significant difference between genders. This mirrors findings from Parental SDQ Scores, suggesting that children are able to self-report behavioural symptoms also observed by their parents.

4.1.1.3 Parental Conners' Rating Scale (CRS) at age 11

Parental CRS data was collected for 612 children at the age of 11. There were approximately equal numbers of male (n=311, 50.8%) and female (n=301, 49.2%) children. Of these children just over half were AGA (n=357, 58.3%) and the remainder were SGA (n=259, 41.7%). The CRS has many available indices, but for the purposes of this study the Conners' ADHD Index and each of the three DSM scales were selected. Summary data for the CRS scores is presented in Tables 16 to 20.

Table 16

Parental CRS indices mean scores and standard deviations (SD) across gender and by birthweight at Age 11

	Male AGA N=181	**Male SGA N=130**	**Male Total N=311**	**Female AGA N=176**	**Female SGA N=129**	**Female Total N=301**
Conners' ADHD Index	**8.9** (6.9)	**9.9** (7.9)	**9.3** (7.3)	**5.5** (5.1)	**5.7** (5.6)	**5.6** (5.3)
DSM Hyperactive	**4.9** (4.6)	**5.1** (4.3)	**5.0** (4.4)	**3.4** (3.3)	**3.7** (4.0)	**3.5** (3.6)
DSM Inattentive	**7.2** (5.7)	**7.7** (6.1)	**7.4** (5.9)	**4.1** (3.9)	**4.4** (4.5)	**4.2** (4.2)
DSM Combined	**12.1** (9.4)	**12.8** (9.4)	**12.4** (9.4)	**7.5** (6.3)	**8.0** (7.7)	**7.8** (7.0)

Note. CRS = Conners' Ratings Scale; SD = Standard Deviation, AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size; ADHD = Attention Deficit Hyperactivity Disorder; DSM = Diagnostic and Statistical Manual of Mental Disorders

As shown in Table 16, males had higher scores than females on the CRS indices that were examined. While the differences were slight, it was also apparent that SGA children again showed higher average scores across genders and indices.

To test whether these differences were significant, a 2x2 MANOVA was run, including the CRS ADHD Index, the DSM Hyperactive Index, and the DSM Inattentive index as dependent variables with gender and birthweight as grouping variables. It was not possible to include the DSM Combined index, as it is the sum of the Hyperactive and Inattentive index, and thus not independent. Significant main effects of sex [$F(3,610)=20.326$, $p<.001$], but not of birthweight [$F(3,610)=.839$, $p=.473$. On all three scales males scored significantly higher on measures of ADHD ($p<.001$). Caution should be employed interpreting these results due to the non-normal distribution of these scores.

To further explore these scores on the Parental CRS ADHD indices, they were catagorised based on established clinical cut-offs. For the purposes of analysis the abnormal and borderline categories were collapsed together (>60).

Table 17

Percentage and frequency of Abnormal/Borderline and Normal Parental CRS scores across gender and by birthweight

	Male AGA N=183		**Male SGA N=130**		**Male Total N=313**		**Female AGA N=177**		**Female SGA N=130**		**Female Total N=307**	
Conners' ADHD Index		**%**		**%**		**%**		**%**		**%**		**%**
Abnormal/ Borderline	26	**14.2**	22	**16.9**	48	**15.3**	12	**6.8**	16	**12.3**	28	**9.1**
Normal	157	**85.8**	108	**83.1**	265	**84.7**	165	**93.2**	114	**87.7**	279	**90.9**
DSM Hyperactive												
Abnormal/ Borderline	21	**11.5**	13	**10.0**	34	**10.9**	14	**7.9**	16	**12.3**	30	**9.8**
Normal	162	**88.5**	117	**90.0**	279	**89.1**	163	**92.1**	114	**87.7**	177	**90.2**
DSM Inattentive												
Abnormal/ Borderline	39	**21.3**	29	**22.3**	68	**21.9**	17	**9.7**	16	**12.3**	33	**10.7**
Normal	142	**78.7**	101	**77.7**	243	**78.1**	159	**90.3**	114	**87.7**	273	**90.3**
DSM Combined												
Abnormal/ Borderline	30	**16.4**	20	**15.4**	50	**16.0**	13	**7.3**	12	**9.2**	25	**8.1**
Normal	153	**83.6**	110	**84.6**	263	**84.0**	164	**92.7**	118	**90.8**	282	**91.9**

Note. CRS = Conners' Ratings Scale; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size; ADHD = Attention Deficit Hyperactivity Disorder; DSM = Diagnostic and Statistical Manual of Mental Disorders

As is presented in Table 17 male children show consistently higher scores across all four indices, with no clear effect of birthweight. To test the significance of these differences, a number of Chi-square tests were run, with two-tailed values recorded.

Significant effects of gender were seen on the CRS ADHD Index [Chi-square(1)=5.566, p=.021; AGA p=.026; SGA p=.380], and the DSM Inattentive index [Chi-square(1)=14.647, p<.001; AGA p=.002; SGA p=.031]; with males performing worse on both. No significant overall effects of birthweight were seen on any of the four subscales.

4.1.1.4 Teachers Conners' Rating Scale (CRS) at age 11

Teacher CRS data was collected for 597 children at age 11 (male n=299; female n=298; AGA n=343; SGA n=254). Performance on the Teachers CRS by gender and birthweight is presented in Table 18.

Table 18

Teachers Conners' indices mean scores and standard deviations (SD) across gender and by birthweight at Age 11

	Male AGA N=173	**Male SGA N=126**	**Male Total N=299**	**Female AGA N=170**	**Female SGA N=128**	**Female Total N=298**
Conners' ADHD Index	3.8 (3.6)	4.4 (3.9)	4.1 (3.7)	2.5 (2.9)	2.8 (3.3)	2.6 (3.1)
DSM Hyperactive	4.0 (3.9)	4.9 (4.4)	4.4 (4.1)	1.8 (2.5)	2.3 (3.2)	2.0 (2.8)
DSM Inattentive	6.1 (5.6)	7.5 (6.4)	6.7 (6.0)	3.0 (4.2)	3.5 (4.8)	3.2 (4.5)
DSM Combined	10.1 (9.6)	12.7 (11.2)	11.2 (10.3)	5.1 (7.2)	5.7 (7.6)	5.4 (7.4)

Note. SD = Standard Deviation, AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size; ADHD = Attention Deficit Hyperactivity Disorder; DSM = Diagnostic and Statistical Manual of Mental Disorders

Examining Table 18, shows that male children are given higher hyperactivity scores by their teachers across all four of the indices. Again, SGA children scored more poorly on average.

A 2x2 MANOVA was run to examine for any significant differences, and included the Teachers CRS ADHD Index, DSM Hyperactive index, and DSM Inattentive index as dependent variables and both gender and birthweight as grouping variables. A significant main effect of gender [$F(3,591)=26.859$, $p<.001$] was found, but not for birthweight [$F(3,591)=1.915$, $p=.126$]. The interaction between the two was also not significant ($p<.05$). On all three scales males scored significantly higher than females on measures of ADHD

(p<.001). While there was no main effect of birthweight both of the DSM subscales showed significant (p<.05) between-subject effects of birthweight.

The data was also categorised to see if these patterns became more apparent when clinical cut-offs were imposed.

Table 19

Percentage and frequency of Abnormal/Borderline (>60) and Normal Teacher CRS scores across gender and by birthweight

	Male AGA N=173		**Male SGA N=130**		**Male Total N=303**		**Female AGA N=177**		**Female SGA N=130**		**Female Total N=307**	
Conners' ADHD Index		**%**		**%**		**%**		**%**		**%**		**%**
Abnormal/ Borderline	0	**0.0**	22	**16.9**	22	**7.3**	12	**6.8**	16	**12.3**	28	**9.1**
Normal	173	**100.0**	108	**83.1**	281	**92.7**	165	**93.2**	114	**87.7**	279	**90.9**
DSM Hyperactive												
Abnormal/ Borderline	6	**3.5**	13	**10.0**	19	**6.3**	14	**7.9**	16	**12.3**	30	**9.8**
Normal	167	**96.5**	117	**90.0**	284	**93.7**	163	**92.1**	114	**87.7**	277	**90.2**
DSM Inattentive												
Abnormal/ Borderline	17	**9.8**	29	**22.3**	46	**15.2**	17	**9.6**	15	**11.5**	32	**10.5**
Normal	156	**90.2**	101	**77.6**	257	**84.8**	159	**90.4**	114	**88.5**	273	**89.5**
DSM Combined												
Abnormal/ Borderline	0	**0.0**	20	**15.4**	20	**6.6**	13	**7.3**	18	**13.8**	31	**10.1**
Normal	173	**100.0**	110	**84.6**	283	**93.4**	164	**92.7**	112	**86.2**	276	**89.9**

Note. CRS = Conners' Ratings Scale; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size; ADHD = Attention Deficit Hyperactivity Disorder; DSM = Diagnostic and Statistical Manual of Mental Disorders

As Table 19 highlights, by examining the CRS data using clinical cuts-offs, SGA children showing higher rates of clinically significant symptoms across both genders. This was examined using a Chi-square tests, with two-tailed significances reported.

Significant effects of gender were seen on the Teachers CRS ADHD Index [Chi-square(1)=10.204, *p*=.001; AGA *p*=.029; SGA *p*.=060] and the DSM ADHD Combined [Chi-square(1)=4.582, *p*=.038; AGA *p* =.245; SGA *p*=.213). A significant effect of birthweight was only seen on the DSM Inattentive index [Chi-square(1)=4.576, *p*=.044; Male *p*=.058; Female *p*=.364] that was carried by SGA males showing greater difficulties.

4.1.2 Predictor variables at 11 (Activity and Sleep Duration)

4.1.2.1 Activity data

Activity data was successfully collected for 540 children at the age of 11. Data was collected for 281 males (52.0%) and 259 females (48.0%), 312 of whom were AGA (57.8%) and 228 who were SGA (42.2%). Recordings were taken for a period of a week and all values represent daily averages. While the Actigraph counts are largely an arbitrary unit, there are twelve algorithms cited by the manufacturers of the monitor that can be used to convert them into MET rates. A MET value of 1 represents the amount of energy the human body expends at rest. MET units were used to inform the cuts-off values employed to partition the data into sedentary, moderate, and vigorous activity levels (Han, 2010). The time spent in each of these intensity categories of activity is examined, as well as the proportion of awake hours spent at each intensity, as hyperactivity may be disguised by proportion values if the child is also awake for a longer period.

Table 20 below provides a summary of the activity data collected. It provides a preliminary normative sample, in a NZ European context, across gender and birthweight.

Table 20

Means and standard deviations (SD) of mean daily activity measures at Age 11

Age 11 Years	Male AGA N=165	Male SGA N=116	Male Total N=281	Female AGA N=147	Female SGA N=112	Female Total N=259
Mean Counts	**593** (207)	**583** (187)	**589** (199)	**493** (148)	**502** (183)	**497** (164)
% Sedentary	**84.1** (4.6)	**84.2** (4.6)	**84.1** (4.8)	**87.3** (3.9)	**86.7** (4.6)	**87.1** (4.2)
% Moderate	**14.1** (3.8)	**14.1** (4.5)	**14.1** (4.1)	**11.4** (3.4)	**12.0** (3.9)	**11.6** (3.6)
% Vigorous	**1.7** (1.4)	**1.5** (1.3)	**1.6** (1.3)	**1.2** (1.1)	**1.2** (1.1)	**1.2** (1.0)
Mins Sedentary	**672** (57)	**671** (57)	**672** (57)	**686** (54)	**686** (54)	**686** (54)
Mins Moderate	**113** (32)	**113** (38)	**113** (34)	**90** (28)	**95** (33)	**92** (30)
Mins Vigorous	**13** (11)	**12** (11)	**13** (11)	**9** (7)	**9** (9)	**9** (8)

Note. SD = Standard Deviation, AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample size; Mins = Minutes

As can be seen in Table 20, there appears to be no effect of birthweight on activity, but there is a clear effect of gender, with boys being more active than girls.

A 2x2 MANOVA examined whether gender and birthweight should be factors to consider if utilising the data provided in Table 20 as a normative sample. To ensure the assumption of independence this analysis was limited to the activity count and the total time spent in sedentary and moderate activity; as if vigorous was included, the three values would add up to the child's total activity and would not be independent. The results showed a clear main effect of gender [$F(3, 534)=18.097$, $p<.001$] and no main effect of birthweight [$F(3, 534)=.768$, $p=.512$]. This suggests that gender specific activity norms should be used.

4.1.2.2 Sleep Duration

At the age of 11 sleep duration was successfully collected for 545 children, of whom 284 (52.1%) were male, and 261 (47.9%) were female, of who 314 (57.6%) were AGA and 231(42.4%) were SGA. As described in the methodology section the Sadeh algorithm (Sadeh et al., 1989) was employed to determine sleep-wake times. The normality of this measure and

average sleep durations are presented in Tables 21 and 22. The data are also displayed categorically in Table 23.

Table 21

Normality of Average Sleep Duration at Age 11

Age 11 Years	Variance	Skewness (SE)	Kurtosis (SE)	Kolmogorov-Smirnova (K-S, df, Sig)
Sleep Duration (Hours)	.525	.837 (.117)	4.032 (.233)	.083, 439, p<.001

Note.; SE = Standard Error; K-S = Kolmogorov-Smirnova; df = degrees of freedom; Sig = Significance level.

Table 22

Average Sleep Duration (Hours) and standard deviations (SD) across sex and by birthweight at age 11

Age 11 Years	Male AGA N=167	Male SGA N=117	Male Total N=284	Female AGA N=147	Female SGA N=114	Female Total N=261	Total N=545
Sleep Duration (Hours)	**9.46** (.77)	**9.37** (.78)	**9.42** (.77)	**9.53** (.72)	**9.62** (.66)	**9.57** (.69)	**9.49** (.74)

Note.; SD = Standard Deviation; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample Size

Table 23

Average Sleep Duration (categorical) across gender and by birthweight at Age 11

Age 11 Years	Male AGA N=167		Male SGA N=117		Male Total N=284		Female AGA N=147		Female SGA N=114		Female Total N=261		Tota1 N=545	
Sleep Duration		**%**		**%**		**%**		**%**		**%**		**%**		**%**
>10.5 hrs	6	**3.6**	7	**6.0**	13	**4.6**	10	**6.8**	9	**7.9**	19	**7.3**	32	**5.9**
9.5-10.5 hrs	76	**45.5**	49	**41.9**	125	**44.0**	67	**45.6**	57	**50.0**	124	**47.5**	249	**45.7**
8.5-9.49 hrs	72	**43.1**	47	**40.2**	119	**41.9**	62	**42.2**	43	**37.7**	105	**40.2**	224	**41.1**
<8.5 hrs	13	**7.8**	14	**12.0**	27	**9.5**	8	**5.4**	5	**4.4**	13	**11.5**	40	**7.3**

Note.; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample Size

RESULTS

Examining Tables 22 and 23, there is no obvious effect of either gender or birthweight. However, a 2x2 MANOVA showed a significant main effect of gender [$F(1, 541)=6.680$, $p=.010$], with males sleeping for less time than females, but not of birthweight [$F(1, 541)=.003$, $p=.956$]. The interaction between the two was not significant ($p>.05$). Based on this measure, with only a 9 minute difference between males and females, it appears that gender specific norms are not informative when examining normative samples of sleep duration.

4.2 Section 2: Changes Over Time in Outcome Variables, Activity Levels, and Sleep Duration

This section examines the changes over time, from ages 7 to 11, for the selected outcome (Parental SDQ and Parental CRS) and predictor variables (activity and sleep duration).

4.2.1 SDQ over time

Data is presented to show the changes in Parental SDQ scores between the ages of 7 and 11. As can be seen in Figure 5 and Table 24, when the data is examined categorically, the percentage of children with abnormal and borderline Parental SDQ scores increases with age. SGA children show a higher rate of abnormal/borderline scores at 11 and appear to exhibit a steeper gradient of increasing problems over times. Perhaps the most notable trend is the apparent divergence between AGA and SGA boys between the ages of 7 and 11.

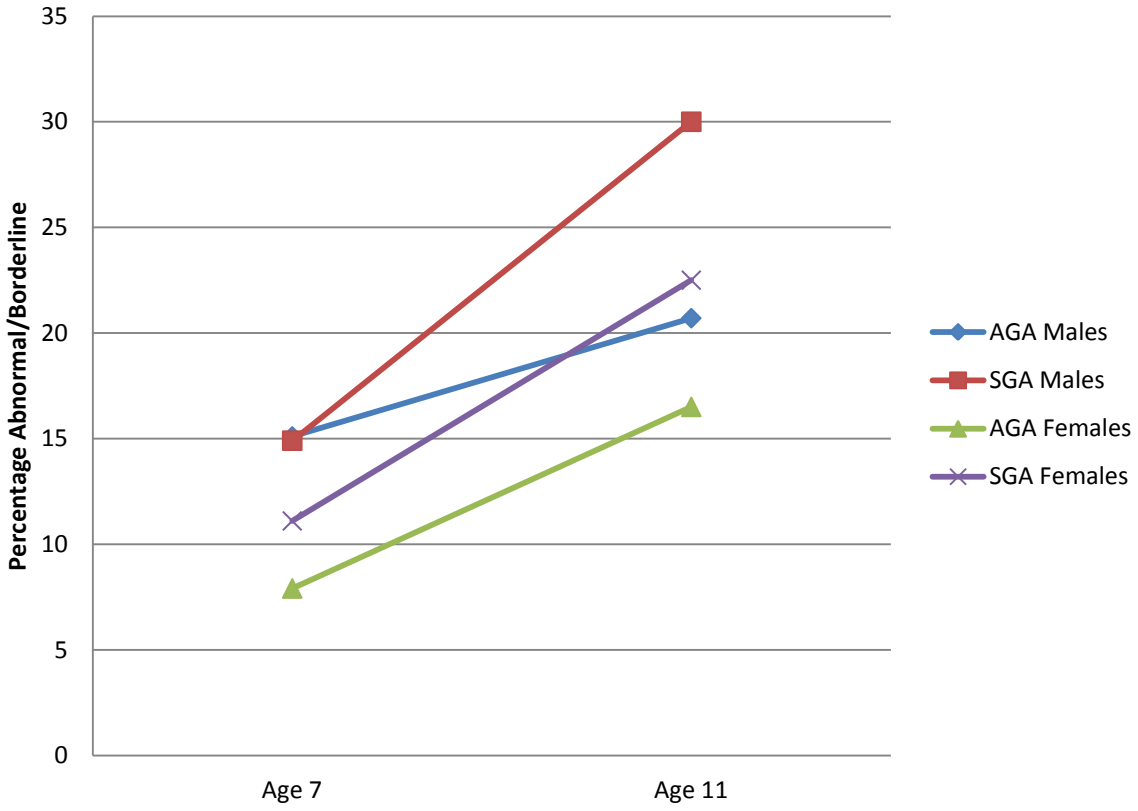

Figure 6: Parental SDQ Total Difficulty scores (categorical) for sex by gender at Age 7 and 11. AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age.

Table 24

Percentage of Abnormal/Borderline and Normal Scores for all Parental SDQ subscales across gender and by birthweight at Age 7 and 11

	7 Years				11 Years			
	Male		Female		Male		Female	
	AGA	SGA	AGA	SGA	AGA	SGA	AGA	SGA
	N=172	N=114	N=177	N=126	N=182	N=129	N=175	N=130
Total Difficulties	%	%	%	%	%	%	%	%
Abnormal/Bord.	**15.1**	**14.9**	**7.9**	**11.1**	**20.7**	**30.0**	**16.5**	**22.5**
Normal	**84.9**	**85.1**	**92.1**	**88.9**	**79.2**	**70.0**	**83.5**	**77.5**
Emotion								
Abnormal/Bord.	**11.6**	**14.0**	**10.7**	**11.9**	**20.7**	**26.9**	**26.1**	**34.1**
Normal	**88.4**	**86.0**	**90.3**	**88.1**	**79.2**	**73.1**	**73.9**	**66.7**
Conduct								
Abnormal/Bord.	**29.1**	**29.8**	**19.2**	**17.5**	**35.5**	**40.8**	**29.6**	**30.3**
Normal	**78.2**	**70.2**	**80.8**	**82.5**	**64.5**	**59.2**	**70.5**	**70.5**
Hyperactivity								
Abnormal/Bord.	**18.8**	**22.6**	**6.8**	**11.1**	**18.6**	**24.6**	**15.9**	**17.1**
Normal	**81.2**	**77.4**	**93.2**	**88.9**	**81.4**	**75.4**	**84.1**	**82.9**
Peer								
Abnormal/Bord.	**28.8**	**14.8**	**13.0**	**15.9**	**30.1**	**36.2**	**24.4**	**27.2**
Normal	**82.4**	**85.2**	**87.0**	**84.1**	**69.9**	**63.8**	**75.6**	**73.6**
Prosocial								
Abnormal/Bord	**17.6**	**12.2**	**6.8**	**4.0**	**15.3**	**18.5**	**5.7**	**9.3**
Normal	**82.4**	**87.8**	**93.2**	**96.0**	**84.7**	**81.5**	**94.3**	**91.5**

Note.; SDQ = Strengths and Difficulties Questionnaire; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample Size; Bord = Borderline

These differences were examined in more detail using the continuous scores presented in Table 25 and Figure 6.

Table 25

Parental SDQ mean scores and standard deviations (SD) for gender by birthweight at Age 7 and 11

	7 Years				11 Years			
	Male		Female		Male		Female	
	AGA N=172	SGA N=114	AGA N=177	SGA N=126	AGA N=182	SGA N=129	AGA N=175	SGA N=130
Total Difficulties	**8.0** (5.3)	**8.3** (5.3)	**6.6** (4.4)	**7.2** (4.5)	**7.5** (5.1)	**8.9** (5.6)	**6.0** (4.5)	**6.9** (5.0)
Emotion	**1.4** (1.7)	**1.4** (2.0)	**1.5** (1.7)	**1.7** (1.7)	**1.5** (1.7)	**2.1** (2.1)	**1.9** (1.8)	**2.2** (1.9)
Conduct	**1.7** (1.7)	**1.7** (1.7)	**1.5** (1.6)	**1.3** (1.2)	**1.6** (1.7)	**1.6** (1.7)	**1.0** (1.3)	**1.2** (1.4)
Hyperactivity	**3.7** (2.6)	**3.8** (2.5)	**2.5** (2.1)	**2.9** (2.2)	**3.3** (2.3)	**3.8** (2.3)	**2.2** (1.9)	**2.5** (2.1)
Peer	**1.2** (1.6)	**1.2** (1.4)	**1.1** (1.5)	**1.2** (1.4)	**1.3** (1.8)	**1.4** (1.7)	**1.0** (1.4)	**1.1** (1.6)
Prosocial	**7.5** (1.9)	**8.0** (1.9)	**8.3** (1.8)	**8.3** (1.6)	**7.8** (1.8)	**7.8** (1.8)	**8.7** (1.5)	**8.5** (1.7)

Note.; SDQ = Strengths and Difficulties Questionnaire; SD = Standard Deviation; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample Size

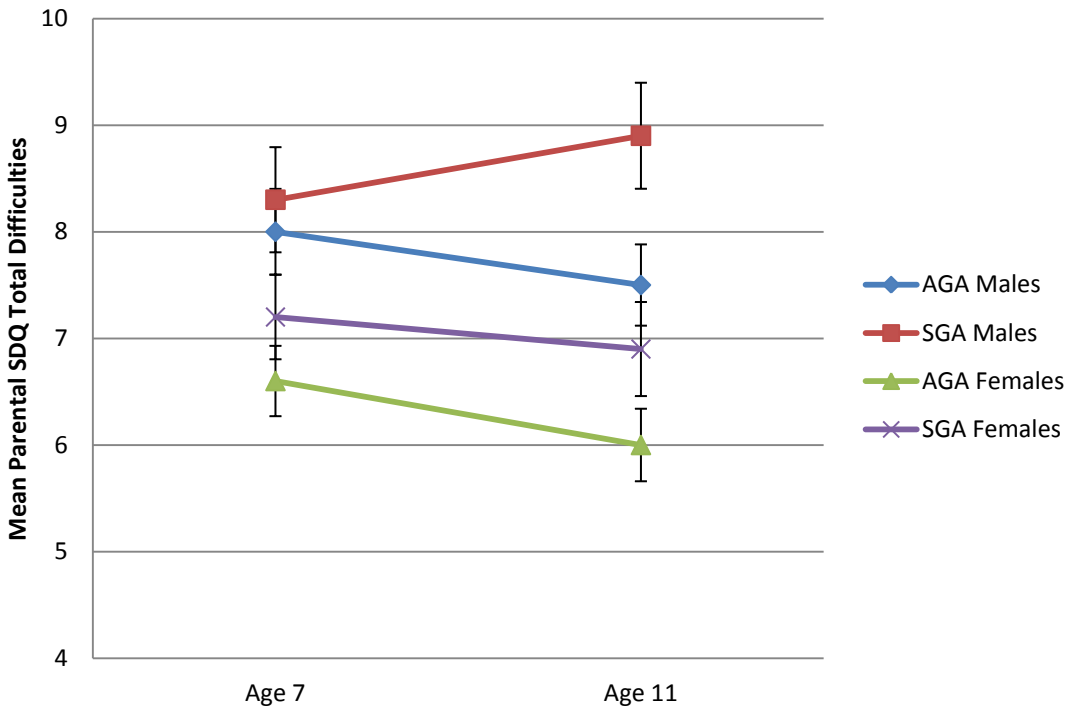

Figure 7: Parental SDQ mean Total Difficulties scores for gender by birthweight at Age 7 and 11. AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age.

To examine the significance of change over time from 7 to 11 years, a repeated-measures MANOVA was conducted on the Parental SDQ Total Difficulty scores, with birthweight and gender as between subject factors. While there was no significant change in Parental SDQ Total Difficulty score over time [$F(1, 538)=2.203$, $p=.138$], there was a significant interaction between this score and birthweight across time [$F(1, 538)=3.885$, $p=.049$]. Looking at Figure 6, it can be seen that by the age of 11 both SGA girls and boys had higher scores than their normal birthweight gender-matched peers. Together with the categorical data in Figure 5, this suggests that despite the reduction in SDQ scores over time for all but the SGA boys, based on clinical cut-offs, emotional and behavioural difficulties become more prevalent in children as they grow older. This effect is significantly greater among SGA children. To explore what appears to be the discrete trajectory of male SGA children, who are the only group to show an increase in difficulty scores between the ages of 7 and 11, the data file was split by gender. Once split, with diminished power, neither gender showed an interaction of birthweight across time: Male [$F(1,261)=2.521$, $p=.114$; Female $F(1,277)=1.328$, $p=.250$).

4.2.2 CRS over time

Data is presented to show the changes in Parental CRS scores between the ages of 7 and 11.

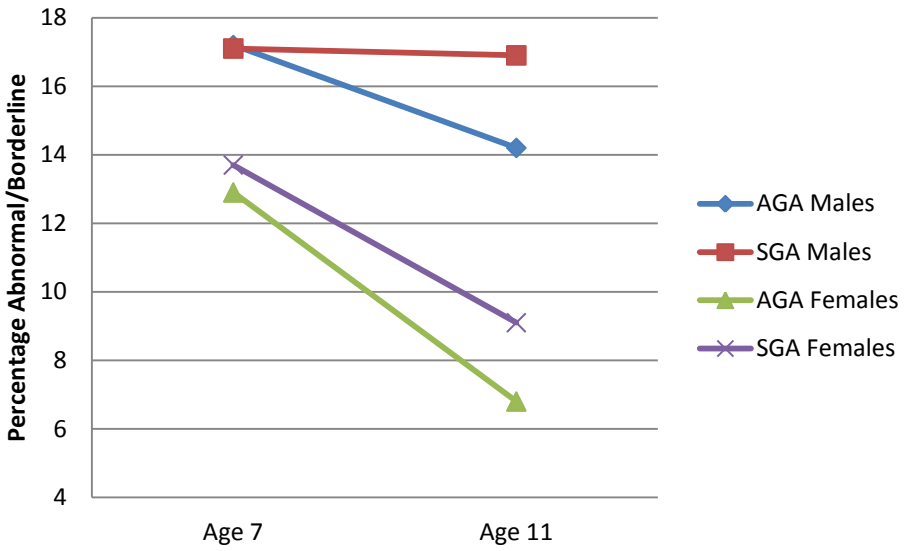

Figure 8: Parental CRS ADHD Index scores for gender by birthweight at Age 7 and 11

Table 26

Percentage of Abnormal/Borderline and Normal Parental CRS subscale scores for gender by birthweight at Age 7 and 11

	7 Years				11 Years			
	Male		Female		Male		Female	
	AGA	SGA	AGA	SGA	AGA	SGA	AGA	SGA
	N=169	N=111	N=171	N=124	N=183	N=130	N=177	N=130
Conners' ADHD Index	%	%	%	%	%	%	%	%
Abnormal/Borderline	**17.2**	**17.1**	**12.9**	**13.7**	**14.2**	**16.9**	**6.8**	**9.1**
Normal	**82.8**	**82.9**	**87.1**	**86.3**	**85.8**	**83.1**	**93.2**	**90.9**
DSM Hyperactive								
Abnormal/Borderline	**20.1**	**22.5**	**19.3**	**29.0**	**11.5**	**10.0**	**7.9**	**9.8**
Normal	**79.9**	**77.5**	**80.7**	**71.0**	**88.5**	**90.0**	**92.1**	**90.2**
DSM Inattentive								
Abnormal/Borderline	**19.5**	**16.2**	**11.1**	**9.7**	**21.3**	**22.3**	**9.7**	**10.7**
Normal	**80.5**	**83.8**	**88.9**	**90.3**	**78.7**	**77.7**	**90.3**	**90.3**
DSM Combined								
Abnormal/Borderline	**20.7**	**23.4**	**14.6**	**17.7**	**16.4**	**15.4**	**7.3**	**8.1**
Normal	**79.3**	**76.6**	**85.4**	**82.3**	**83.6**	**84.6**	**92.7**	**91.9**

Note.; CRS = Conners' Rating Scale; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample Size; ADHD = Attention Deficit Hyperactivity Disorder; DSM = Diagnostic and Statistical Manual of Mental Disorders

RESULTS

Examining the categorical classifications of the four Parental CRS scores in Table 26, and of the Parental CRS ADHD Index scores (Figure 7), boys show more difficulties than girls. Unlike the Parental SDQ Total Difficulty scores, however, there does appear to be an overall reduction over time of children falling into the abnormal or borderline classifications of the Parental CRS ADHD Index. As Figure 7 shows the SGA males were again an exception to this overall pattern, demonstrating broadly consistent percentages of difficulties between ages. As can be seen in Table 27 when the continuous scorings of the Parental CRS indices were examined there were again clear differences of gender.

Table 27

Parental CRS mean scores and standard deviations (SD) by gender and birthweight at Age 7 and 11

	7 Years				**11 Years**			
	Male		**Female**		**Male**		**Female**	
	AGA N=169	**SGA N=111**	**AGA N=171**	**SGA N=124**	**AGA N=181**	**SGA N=130**	**AGA N=176**	**SGA N=129**
Conners' ADHD	**9.9** (7.4)	**10.1** (7.0)	**6.7** (5.0)	**7.1** (5.5)	**8.9** (6.9)	**9.9** (7.9)	**5.5** (5.1)	**5.7** (5.6)
DSM Hyperactive	**7.1** (5.1)	**7.6** (5.1)	**5.5** (4.0)	**5.7** (4.6)	**4.9** (4.6)	**5.1** (4.3)	**3.4** (3.3)	**3.7** (4.0)
DSM Inattentive	**7.3** (5.5)	**7.0** (5.3)	**4.6** (3.8)	**4.8** (3.7)	**7.2** (5.7)	**7.7** (6.1)	**4.1** (3.9)	**4.4** (4.5)
DSM Combined	**14.3** (9.9)	**14.6** (9.2)	**10.1** (6.9)	**10.4** (7.5)	**12.1** (9.4)	**12.8** (9.4)	**7.5** (6.3)	**8.0** (7.7)

Note. CRS = Conners' Rating Scale; SD = Standard Deviation; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample Size; ADHD = Attention Deficit Hyperactivity Disorder; DSM = Diagnostic and Statistical Manual of Mental Disorders

To examine change over time a repeated measures MANOVA was conducted on the Parental CRS ADHD Index scores, with gender and birthweight as between subjects factors. As evident in Figure 8, this confirmed a clear main effect of gender [$F(1,532)=3079.858$,

p<.001]. It also demonstrated the significant reduction in scores as children get older [F(1,532)=20.537, p<.001], but no differential effects of gender or birthweight across the two ages (Gender p=.570; Birthweight p=.465).

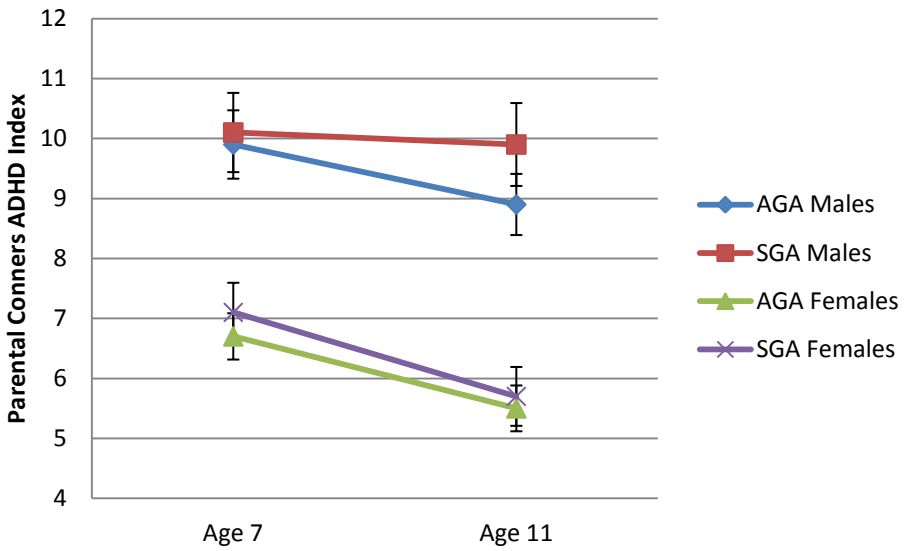

Figure 9 Parental CRS Index mean scores by sex by gender and birthweight at Age 7 and 1. AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age

4.2.3 Activity over time

Data is presented to display the changes in activity level of the children between the age of 7 and 11. It is important to note that at the age of 7, activity was only sampled over a 24 hour period, whereas a week-long sample was taken at age 11.

As can be seen in Table 28 and Figures 9 and 10 there is a reduction in physical activity at 11 when compared to 7, with reductions in vigorous and moderate activity, and a corresponding increase in sedentary activity. There are no clear effects of gender or birthweight.

A repeated-measures MANOVA, with gender and birthweight as between-subject factors, was conducted. This confirmed that the reduction in activity (using mean counts) as children get older was significant [F(1,425)=391.26, p<.001], but that there were no significant effects of gender or birthweight across the two ages (p>.05).

Table 28

Mean activity measures and standard deviations (SD) for gender by birthweight at Age 7 and 11

	7 Years				11 Years			
	Male		Female		Male		Female	
	AGA N=149	SGA N=103	AGA N=153	SGA N=104	AGA N=165	SGA N=116	AGA N=147	SGA N=112
Mean Counts	**837** (277)	**820** (275)	**778** (243)	**754** (231)	**593** (207)	**583** (187)	**493** (148)	**502** (183)
% Sedentary	**66.4** (8.3)	**66.7** (8.8)	**67.5** (8.4)	**69.5** (7.7)	**84.1** (4.6)	**84.2** (4.6)	**87.3** (3.9)	**86.7** (4.6)
% Moderate	**27.9** (6.2)	**27.8** (7.0)	**27.9** (6.7)	**26.0** (6.6)	**14.1** (3.8)	**14.1** (4.5)	**11.4** (3.4)	**12.0** (3.9)
% Vigorous	**5.7** (3.6)	**5.5** (3.5)	**4.7** (3.0)	**4.5** (3.1)	**1.7** (1.4)	**1.5** (1.3)	**1.2** (1.1)	**1.2** (1.1)
Mins Sedentary	**529** (89)	**534** (88)	**541** (84)	**549** (79)	**672** (57)	**671** (57)	**686** (54)	**686** (54)
Mins Moderate	**222** (54)	**222** (58)	**223** (55)	**206** (58)	**113** (32)	**113** (38)	90 (28)	95 (33)
Mins Vigourous	46 (28)	44 (29)	37 (24)	36 (25)	13 (11)	12 (11)	9 (7)	9 (9)

Note. SD = Standard Deviation; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample Size; Mins = Minutes

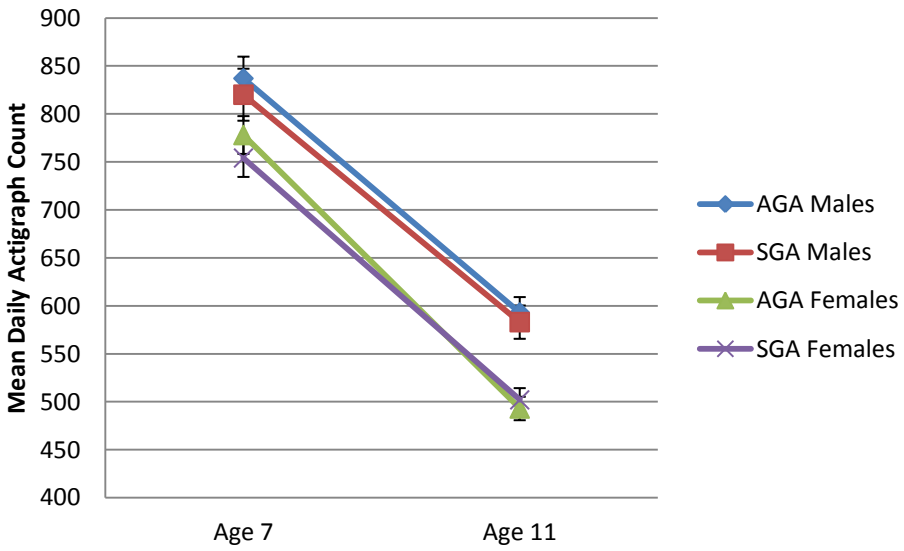

Figure 10: Average daily activity counts for gender by birthweight at Age 7 and 1. AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age

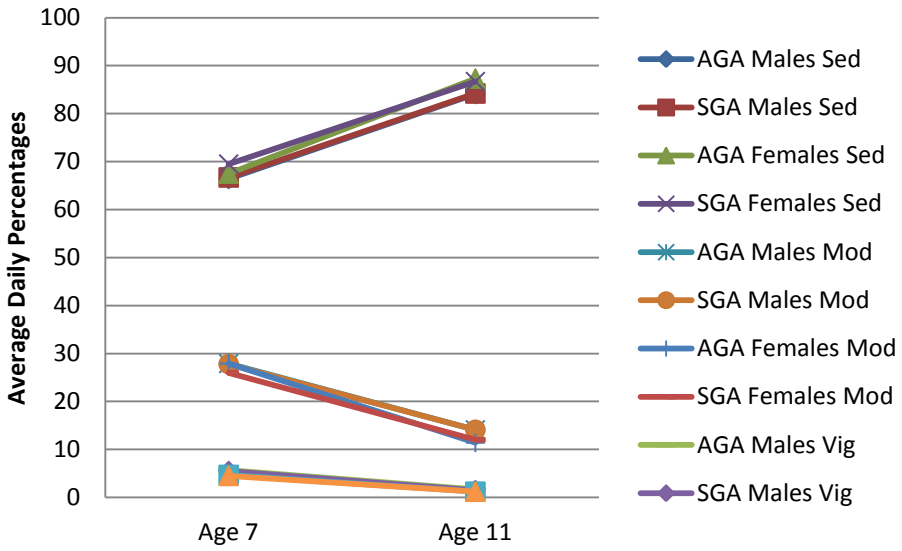

Figure 11: Average daily percentage of time spent in each intensity of activity for gender by birthweight at Age 7 and 11. AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; Sed = Sedentary; Mod = Moderate Activity; Vig = Vigorous Activity.

4.2.4 Sleep Duration over time

Data is presented to display the changes in the average sleep duration of the children between the ages of 7 and 11. Unlike at age 11, where data was aggregated across a seven day period, it is again of note that the data from 7 was only recorded over a single night.

Table 29

Mean Sleep Duration and standard deviations (SD) for gender by birthweight at Age 7 and 11

	7 Years				**11 Years**			
	Male		**Female**		**Male**		**Female**	
	AGA N=	**SGA N=103**	**AGA N=153**	**SGA N=104**	**AGA N=167**	**SGA N=117**	**AGA N=147**	**SGA N=114**
Sleep Duration (Hours)	10.01 (.89)	9.98 (.83)	10.01 (.82)	10.07 (.78)	**9.46** (.77)	**9.37** (.78)	**9.53** (.72)	**9.62** (.66)

Note. SD = Standard Deviation; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample Size.

Table 30

Sleep Duration (categorical) for gender by birthweight at Age 7 and 11

	7 Years				11 Years			
	Male		Female		Male		Female	
	AGA	SGA	AGA	SGA	AGA	SGA	AGA	SGA
	N=143	N=101	N=145	N=102	N=183	N=130	N=177	N=130
Sleep Duration	%	%	%	%	%	%	%	%
>10.5 hours	**30.1**	**26.7**	**25.5**	**26.5**	**3.6**	**6.0**	**6.8**	**7.9**
9.5-10.5 hours	**41.3**	**45.5**	**46.9**	**57.8**	**45.5**	**41.9**	**45.6**	**50.0**
8.5-9.49 hours	**25.9**	**24.8**	**24.1**	**10.8**	**43.1**	**40.2**	**42.2**	**37.7**
<8.5 hours	**2.8**	**3.0**	**3.4**	**4.9**	**7.8**	**12.0**	**5.4**	**4.4**

Note.AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; N = Sample Size;

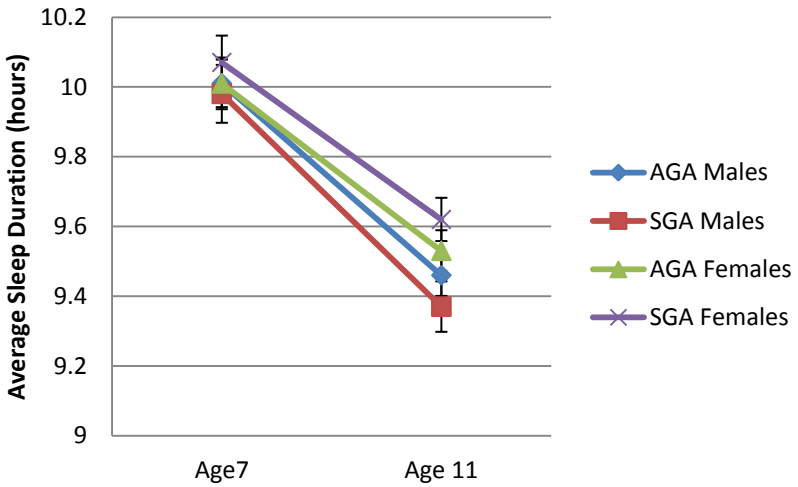

Figure 12: Average Sleep Duration for gender by birthweight at Age 7 and 11. AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age

Looking at Tables 29 and 30, and Figure 11, it is apparent that the children in this study are sleeping for a shorter duration at the age of 11 than at the age of 7. A repeated-measures MANOVA was conducted with gender and birthweight as between subject factors. This confirmed a significant reduction in the hours children are sleeping as they get older

[$F(1,435)=111.733$, $p<.001$] , but no significant effects of gender or birthweight across the two ages.

4.3 Section 3: Relationships Between Variables

In this section correlations were used to assess which factors were likely to be most predictive of the outcome measures. As previously explained, based on normality analyses, the outcome measures selected for further analysis were Parental SDQ Total Difficulties Score (continuous and categorical) and Parental CRS ADHD Index (categorical) at age 11. First the relationship with the outcome measures was examined for activity, sleep duration, and other available predictors of interest. Following this, the inter-relationships of any significant potential predictors were explored to examine for multicolinearity. Correlations were chosen as the clearest way to present the data at this exploratory phase, with linear regression and logistic regression employed in the next section to explore significant predictors in more detail. Continuous predictor and outcome pairings were therefore examined using Pearson's Correlation Coefficients. Categorical data was treated as if it were ranked by the risk of emotional and behavioural difficulties it conferred, and Spearman's Rho was used. Due to the large number of correlations, and the increased risk of Type 1 errors, only those correlations significant at the stringent level of $p< .01$ were considered as candidates for further analysis.

4.3.1 Correlation of potential predictors to behavioural outcomes

The degree and direction of the relationship between potential predictors and the outcome measures were explored, with potential predictors including: activity and sleep duration at 7 and 11; gender, birthweight; and a range of factors identified by the literature (maternal smoking, paracetamol, cannabis and alcohol use during pregnancy; parental school leaving age; marital status; maternal perceived stress; maternal social support; body mass index; electronic media exposure; reading age; intellectual function; child depression). This

identified predictive factors of emotional and behavioural issues at 11, which may merit further investigation if a child is being assessed clinically for such problems at this age. It also served as a preliminary to the next section on regression analysis, which helped to determine if activity and sleep duration measures contributed independently to the prediction of the emotional and behavioural outcomes; see Table 31.

Table 31

Correlation between potential predictors and parental outcome measures

Potential Continuous and Categorical Predictors	**SDQ Total Difficulties at 11** (Continuous)	**SDQ Total Difficulties at 11** (Categorical)	**Conners' ADHD Index at 11** (Categorical)
Birthweight (AGA; SGA)	.103*	.058	.061
Gender (male; female)	**-.162****	-.070	-.095*
Mean activity (counts) at 7	.021	-.017	.070
Average minutes of sedentary activity at 7	-.022	-.044	-.075
Average minutes of moderate activity at 7	.096*	.058	.098*
Average minutes of vigorous activity at 7	-.002	-.051	.028
Percentage of sedentary activity at 7	-.067	-.039	-.102*
Percentage of moderate activity at 7	.090*	.074	.098*
Percentage of vigorous activity at 7	-.009	-.049	.027
Mean activity (counts) at 11	.062	-.031	-.010
Average minutes of sedentary activity at 11	.045	.061	.018
Average minutes of moderate activity at 11	.011	-.076	-.010
Average minutes of vigorous activity at 11	.005	-.065	-.057
Percentage of sedentary activity at 11	.000	.083	.019
Percentage of moderate activity at 11	-.008	-.080	-.016
Percentage of vigorous activity at 11	-.001	-.063	-.058
Sleep duration (hours) age 7	-.075	.024	-.022
Sleep duration (hours) age 11	-.058	-.014	-.005
Sleep duration at 7 (<8.5,<9.5,<10.5,>10.5)	.068	-.006	.040
Sleep duration at 11 (<8.5,<9.5,<10.5,>10.5)	.060	.008	.001
Maternal age at birth (<25; 25-34; 35+)	-.104*	-.**140****	-.058
Maternal smoking during pregnancy? (Yes; No)	**-.158****	-.085*	-.036

Potential Continuous and Categorical Predictors (continued)	**SDQ Total Difficulties at 11** (Continuous)	**SDQ Total Difficulties at 11** (Categorical)	**Conners' ADHD Index at 11** (Categorical)
Maternal cigarettes (per day) second trimester	**.147****	.104*	.033
Maternal cigarettes (per day) third trimester	**.122****	.096*	.032
Paternal smoking during pregnancy? (Yes; No)	**-.156****	-.079	.016
Use of paracetamol during pregnancy (Yes; No)	-.077	-.067	-.046
Maternal cannabis use during pregnancy (Yes; No)	**-.125****	-.092*	-.050
Maternal standard drinks in first month of pregnancy	.043	.018	-.010
Maternal standard drinks in last months of pregnancy	-.077	-.019	-.014
Alcohol use during pregnancy (no; light; heavy)	-.009	-.023	-.061
Age mother left school (<16; 16; >16)	-.093*	-.037	-.041
Age father left school (<16; 16; >16)	**-.129****	-.075	-.013
Occupational Status (high; medium; low)	.096*	.002	.005
Family income at birth (<15K; 15-25K; 25-35K; >35K)	**-.217****	**-.174****	-.025
Marital status at birth (married/de facto; other)	**-.173****	**-.188****	**-.106****
Marital status at age 3.5 (married/de facto; other)	-.126	-.090*	-.029
Living with biological father at age 3.5	-.112*	-.080	-.045
Marital status at age 7 (married/de facto; other)	-.085*	-.074	-.039
Living with biological father at 7	**-.113****	-.111*	-.083
Marital status of at age 11 (married/de facto; other)	-.064	-.075	-.006
Living with biological father at age 11	**-.127****	**-.139****	-.074
Perceived Stress Scale at birth	**.146****	.100*	.035
Perceived Stress Scale at 3.5	**.209****	**.187****	.099*
Perceived Stress Scale at 7	**.191****	**.132****	.073
Informal social support at birth	-.056	-.047	-.059
Formal social support at birth	.038	-.032	-.008
Total social support at birth	-.032	-.053	-.064
Informal social support at 1	-.009	.001	.035
Formal social support at 1	.038	.005	.051
Total social support at 1	.021	.021	.040
Informal social support at 3.5	-.076	-.022	.003
Formal social support at 3.5	.066	.014	-.018

Potential Continuous and Categorical Predictors (continued)	SDQ Total Difficulties at 11 (Continuous)	SDQ Total Difficulties at 11 (Categorical)	Conners' ADHD Index at 11 (Categorical)
BMI at 7	.072	.089*	.045
BMI at 11	**.184****	**.136****	.051
BMI age 3.5 (underweight/normal; overweight/obese)	.043	.115*	.084
BMI age 7 (underweight/normal; overweight/obese)	.050	.083	.030
BMI age 11 (underweight/normal; overweight/obese)	**.169****	**.148****	.065
TV watching at age 7 (<1; 1-3; 3-5; >5 hours)	.104*	.082	.005
Weekday home electronic media at 11 (<1.5; 1.5-3; 3- 4.5; >4.5)	**.172****	.104*	.047
Weekend home electronic media at 11 (<1.5; 1.5-3; 3-4.5; >4.5)	**.197****	**.143****	**.120****
Reading age at 7	**-.172****	-.116*	**-.187****
Stanford Binet Total IQ score at 3.5	**-.147****	-.111*	-.087
WISC at 7 VIQ	**-.202****	-.109*	**-.126****
WISC at 7 PIQ	**-.166****	**-.125****	**-.157****
WISC at 7 FSIQ	**-.213****	**-.140****	**-.157****
WISC Digit Span (Working Memory) at 7	**-.165****	-.110*	**-.126****
WASI at 11 VIQ	**-.210****	**-.128****	**-.112****
WASI at 11 PIQ	**-.204****	**-.123****	**-.115****
WASI at 11 FSIQ	**-.235****	**-.142****	**-.132****
Depression variable age 11	**.318****	**.218****	**.210****

Note: Pearson's Correlation Coefficient values are listed for continuous predictor/outcome pairs, and Spearman's Rho values if either predictor or outcome were categorical; *p<.05, **p<.01. SDQ = Strengths and Difficulties Questionnaire; ADHD = Attentional Deficit Hyperactivity Disorder; SD = Standard Deviation; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; BMI = Body Mass Index; IQ = Intelligence Quotient; WISC = Wechsler Intelligence Scale for Children; VIQ = Verbal Intelligence Quotient; PIQ = Performance Intelligence Quotient; FSIQ = Full-scale Intelligence Quotient.

As can be seen in Table 31, increased scores on Parental SDQ Total Difficulties at 11 (continuous) was significantly related to (p<.01): male gender; maternal smoking (and cigarettes used during each trimester) and maternal cannabis use during pregnancy; reduced age when father left school; reduced family income at time of birth; single marital status of mother at birth; not living with father at age 7 and 11; perceived maternal stress at birth, 3.5

and 7 years; higher BMI at 11; lower reading age at age 7; lower levels of cognitive functioning (IQ) at age 3.5, at age 7 (overall IQ, VIQ, PIQ, working memory), and at age 11 (VIQ, PIQ, FSIQ); and increased levels of depression at 11.

Similarly, Parental SDQ Total Difficulties at 11 (categorical) was related to lower maternal age at time of birth; reduced family income at birth; single marital status at birth; perceived maternal stress at 3.5 and 7 years of age; higher BMI at 11; increased weekend home electronic media at 11; lower PIQ and FSIQ at age 7 years, and lower VIQ, PIQ, and FSIQ at 11 years; and increased levels of depression at 11.

Finally, poorer performance on the Parental Conners' ADHD Index at 11 (categorical) was related to single maternal marital status at birth; lower reading age at 7 years; lower VIQ, PIQ, FSIQ, and working memory at 7, and VIQ, PIQ, FSIQ at 11; and increased levels of depression at 11.

4.3.2 Inter-relationships of potential predictor variables (multicolinearity)

The correlations between the significant potential predictors for each outcome variable were examined to appraise for multicolinearity and were found to be extensively inter-related. Where predictors were measuring similar constructs, the predictor with the strongest correlation with the outcome variable, the earliest predictor in time, or the most theoretically compelling measure was selected. For example the Perceived Stress Scale closest to birth was used as this represented a measure of maternal stress least influenced by the child's behaviour, and the WISC at 7 was selected as the most clinically robust measure of IQ. Tables 33 to 35 show the relationships between these selected predictors, for each of the three outcome variables

Table 32:

Interrelationships of significant predictors of Parental SDQ Total Difficulties (continuous)

Spearmans Rho /*Pearsons (grey)*	**Gender**	**Mins of mod activity at 7**	**Maternal age at birth**	**Maternal smoking during pregnancy**	**Maternal cannabis use during pregnancy**	**Family income at birth**	**Marital status at birth**	**Living with biological father at age 11**	**Perceived Stress Scale at birth**	**BMI at 11**	**Weekend home electronic media at 11**	**WISC at 7 FSIQ**	**Depression variable age 11**
Gender (male; female)	1.000												
Average minutes of moderate activity at 7	-.042	1.000											
Maternal age at birth (<25; 25-34; 35+)	-.010	-.141**	1.000										
Maternal smoking during pregnancy? (Yes; No)	.012	-.078	.232**	1.000									
Maternal cannabis use during pregnancy	.003	-.027	.180**	.372**	1.000								
Family income at birth (<15K; 15-25K; 25-35K; >35K)	-.004	-.081	**.241****	**.196****	**.155****	1.000							
Marital status at birth (married/de facto; other)	-.006	-.070	**.231****	**.364****	**.254****	**.398****	1.000						
Living with biological father at age 11	-.024	-.031	**.172****	**.263****	**.185****	.257**	**.336****	1.000					
Perceived Stress Scale at birth	.017	*.044*	**-.154****	**-.201****	**-.125****	**-.265****	**-.158****	**-.180****	1.000				
BMI at 11	-.003	*0.089*	-.005	**-.168****	-.078	**-.122****	-.045	**-.104***	***.092****	1.000			
Weekend home electronic media at 11 (<1.5; 1.5-3; 3-4.5; >4.5)	**-.169****	.025	-.008	**-.161****	-.076	**-.125****	**-.136****	**-.112****	.096*	.166**	1.000		
WISC at 7 FSIQ	-.054	***-.090****	**.091***	**.113****	.009	**.191****	**.120****	**.151****	***-.090****	***-.093****	**-.093***	1.000	
Depression variable age 11	-.047	*.003*	-.046	-.064	-.012	**-.167****	-.066	**-.132****	***.165****	***.161****	**.090***	***-.246****	1.000

Note. SDQ = Strengths and Difficulties Questionnaire; BMI = Body Mass Index; WISC = Wechsler Intelligence Scale for Children; FSIQ = Full-scale Intelligence Quotient; Mins = Minutes

RESULTS

As can be seen from Table 32, there is a cluster of higher inter-correlations linking maternal age, family income, relationship at status, smoking, and perceived stress at birth. While selection from these variables merited careful consideration at later stages of analysis to prevent redundancy, none of these relationships exceeded 0.4. This suggested that multicollinearity was not an issue, representing less than 16% of variance shared.

Table 33

Interrelationship of significant predictors of Parental SDQ Total Difficulties (categorical)

Spearmans Rho / *Pearsons (grey)*	**Maternal age at birth**	**Family income at birth**	**Marital status at birth**	**Perceived Stress Scale at 3.5**	**BMI at 11**	**Weekend home electronic media at 11**	**WISC at 7 FSIQ**	**Depression variable age 11**
Maternal age at birth (<25; 25-34; 35+)	1.000							
Family income at birth (<15K; 15-25K; 25-35K; >35K)	**.241****	1.000						
Marital status at birth (married/de facto; other)	**.231****	**.398****	1.000					
Perceived Stress Scale at 3.5	**-.075***	**-.115****	-.049	1.000				
BMI at 11	-.005	**-.122****	-.045	*.033*	1.000			
Weekend home electronic media at 11 (<1.5; 1.5-3; 3-4.5; >4.5)	-.008	**-.125****	**-.136****	**.115***	**.166****	1.000		
WISC at 7 FSIQ	**.091***	**.191****	**.120****	***-.116*****	***-.093****	**-.093***	1.000	
Depression variable age 11	-.046	**-.167****	-.066	***.094****	***.161*****	.090*	***-.246*****	1.000

Note. SDQ = Strengths and Difficulties Questionnaire; BMI = Body Mass Index; WISC = Wechsler Intelligence Scale for Children; FSIQ = Full-scale Intelligence Quotient; Mins = Minutes

RESULTS

As seen in Table 33, other than an unsurprisingly stronger correlation between marital status at birth and family income, none of the other interrelationships between potential predictors exceed 0.25 (<6.25% variance shared). Multicolinearity was therefore not considered a concern.

Table 34

Interrelationship of significant predictors of Parental Conners' ADHD Index at Age 11 (categorical)

Spearmans Rho / *Pearsons (grey)*	**Percentage of sedentary activity at 7**	**Marital status at birth**	**Perceived Stress Scale at 3.5**	**Weekend home electronic media at 11**	**WISC at 7 FSIQ**	**WISC Digit Span (Working Memory) at 7**	**Depression variable age 11**
Percentage of sedentary activity at 7	1.000						
Marital status at birth (married/de facto; other)	**.161****	1.000					
Perceived Stress Scale at 3.5	*-.045*	**-.075***	1.000				
Weekend home electronic media at 11 (<1.5; 1.5-3; 3-4.5; >4.5)	.019	-.008	.115*	1.000			
WISC at 7 FSIQ	*.083*	**.091***	***-.116****	***-.101****	1.000		
WISC Digit Span (Working Memory) at 7	*.013*	.069	*-.065*	***-.151****	***.486****	1.000	
Depression variable age 11	*-.014*	-.046	***.094****	***.085****	***-.246****	***-.195****	1.000

Note. ADHD = Attention Deficit Hyperactivity Disorder; WISC = Wechsler Intelligence Scale for Children; FSIQ = Full-scale Intelligence Quotient; Mins = Minutes

As can be seen from the above Table 34, other than the higher relationship between WISC FSIQ at 7 and the working memory subscale of this measure (included as it has theoretical significance to ADHD), none of the other relationships exceed 0.25 (<6.25% variance shared). Multicolinearity was therefore not considered a concern.

4.4 Section 4: Regression Analysis

In this section analyses are presented which explore in more detail the relationship between the potentially independent predictor variables identified in Section 3, and the three selected outcome measures of emotional and behavioural difficulties at age 11. This was with the ultimate aim of modelling and exploring the proportion of variance of the outcome measures accounted for by these variables.

4.4.1 Univariate regression analysis

Due to the normality restrictions detailed in the Data Screening section, and the weak correlations shown between activity and sleep duration and the outcome variables, exploration of many of the hypothesised predictors and intended outcome measures were not included in the multivariate statistical modelling section of this thesis. To inform future research, more detailed univariate regression analyses were however conducted for all permutations and combinations of the intended predictors and outcomes.

4.4.2 Multivariate regression analysis

Due to the focus of the ABC study, all analyses included birthweight. The results detailed in Sections 4 determined which predictors were entered into each model. While a stringent inclusion criteria of $p<.01$ was set for the covariates, inclusion of the activity and sleep data was permitted at $p<.05$ level of correlation with the outcome variables due to their centrality to the study's hypotheses. Hierarchical regression (if outcome was continuous) or logistical regression (if outcome was dichotomous) was used in a block design. Each block consisted of variables measured at particular phases of the study (e.g., birth, age 7, age 11).

4.4.2.1 Parental SDQ Total Difficulties Score at 11 (Continuous outcome measure)

The following variables were considered in regression analyses to predict Parental SDQ Total Difficulties score at age 11: birthweight; gender; maternal age at birth; income at birth; marital status at birth; maternal smoking during pregnancy; maternal cannabis use during pregnancy; maternal stress at birth; WISC full scale IQ at age 7; living with father at 11; BMI at 11; weekend home electronic media at 11; and depression at age 11.

In step 1 the following variables were entered into the equation: birthweight; gender; maternal age at birth; income at birth; marital status at birth; maternal smoking during pregnancy; maternal cannabis use during pregnancy; and maternal stress at birth. The equation significantly predicted the outcome, R^2=.081 [F(8, 399)=4.380, p<.001]. Gender (p<.001) was the only variable to make a significant independent contribution to prediction, with boys having more Total Difficulties than girls.

In step 2 the following variables were added: WISC FSIQ at 7 and percentage of moderate activity at 7. The addition of these variables to the equation significantly improved prediction of the outcome, R^2_{change}=.035 [F_{change}(2, 397)=7.898, p<.001]. In this step, gender (p<.001), maternal cannabis use (p=.048), and FSIQ at age 7 (p<.001) were all significant independent contributors to prediction.

In step 3 the following variables were added: living with father at 11; BMI; weekend use of electronics; and depression at 11. The addition of these variables to the equation significantly improved prediction of the outcome, R^2_{change}=.064 [F_{change}(4, 393)=7.647, p<.001]. In this final step, gender (p=.002), maternal cannabis use (p=.025), FSIQ at 7, weekend home electronic media (p=.032), and depression at 11 (p<.001) all made a significant contribution to prediction. Data including all predictors entered into the model is

presented in Table 35. As shown in the Table, males, cannabis non-use, electronic media use, lower IQ, and depression at are associated with Total Difficulty increases.

Table 35

Combined multivariate regression for significant predictors of Parental SDQ Total Difficulties score at Age 11 (continuous outcome measure)

Predictor	B (SE)	β	Lower	Upper	t	p
Birthweight (AGA; SGA)	.619 (.498)	.061	-.360	1.598	1.242	.215
Gender (male; female)	-1.481 (.464)	-.150	-2.392	-.570	-3.195	**.002****
Maternal age at birth (<25; 25-34; 35+)	-.186 (.415)	-.023	-1.003	.630	-.449	.654
Marital status at birth (married/de facto; other)	-1.054 (.610)	-.091	-2.254	.146	-1.726	.085
Family income at birth (<15K; 15-25K; 25-35K; >35K)	-.148 (.376)	-.021	-.887	.590	-.395	.693
Maternal smoking during pregnancy? (Yes; No)	.658 (.660)	.053	-.640	1.956	.997	.319
Maternal cannabis use during pregnancy? (Yes; No)	2.424 (1.080)	.111	.301	4.547	2.244	**.025***
Perceived Stress Scale at birth	.013 (.036)	.017	-.057	.083	.369	.712
WISC at 7 FSIQ	-.046 (.018)	-.123	-.081	-.010	-2.541	**.011***
Average minutes of moderate activity at 7	.020 (.036)	.026	-.050	.090	.553	.581
Living with biological father at age 11	-.155 (.569)	-.013	-1.274	.964	-.272	.786
BMI at 11	.063 (.075)	.041	-.086	.211	.830	.407
Weekend home electronic media at 11 (<1.5; 1.5-3; 3-4.5; >4.5)	.591 (.275)	.104	.051	1.131	2.152	**.032***
Depression at age 11	.143 (.031)	.224	.082	.204	4.620	**.000***

Note. SDQ = Strengths and Difficulties Questionnaire; B = Unstandardised Beta Coefficient; SE = Standard Error; β = Standardised Beta Coefficient; t = Test statistic; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; WISC = Wechsler Intelligence Scale for Children; FSIQ = Full-scale Intelligence Quotient; BMI = Body Mass Index.

4.4.2.2 Parental SDQ Total Difficulties Score at 11 (categorical outcome measure)

The following predictors were considered in logistic regression analyses to predict Parental SDQ Total Difficulties category at age 11: birthweight; maternal age at birth; marital status at birth; maternal stress at 3.5; WISC FSIQ at 7; BMI at 11; weekend home electronic media at 11; and depression at 11. The proportion of variance accounted for at each step was appraised using the Nagelkerke R Square statistic.

In step 1 the following variables were entered: birthweight; maternal age at birth; marital status at birth. This equation significantly predicted the outcome [Chi square(4)=16.936, p=.002], accounting for 7.5% of the variance.

In step 2 maternal perceived stress at age 3 was added to the equation, significantly improving prediction of the outcome [Chi Square(1)=12.144, p<.001] and accounting for a further 5.3% of the variance (Step 2 cumulative total=12.8%).

In step 3, WISC FSIQ was added, falling just short of being a significant independent predictor [Chi Square(1)=2.994, p=.084]. This third block did however add a further 1.2% to the variance explained by the model (Step 3 cumulative total=14%).

In step 4, weekend electronic media, depression at 11, and BMI at 11 were added to the model. They significantly improved prediction of the outcome [Chi Square(4)=19.421, p=.001], accounting for a further 8.0% of variance. In total the model accounted for 22.0% of the variance of Parental SDQ Total Difficulties scores at 11(categorical). The overall model is presented in Table 36.

Table 36

Combined multivariate logistical regression for significant predictors of Parental SDQ Total Difficulties Score (categorical outcome measure)

Predictor	OR	Lower	Upper	p
Birthweight				
AGA	REF			
SGA	1.107	.576	2.129	.760
Maternal age at birth				
<25	1.834	.677	4.968	.233
25-34	REF			.363
35+	.819	.387	1.736	.603
Marital Status at Birth				
Married/Defacto	REF			
Other	2.079	1.002	4.315	**.049***
Perceived Stress Scale at 3.5	1.085	1.029	1.145	**.003****
WISC at 7 FSIQ	.993	.970	1.017	.570
Weekday home electronic media at 11				
<3	REF			.440
3-4.5	1.375	.589	3.212	.461
>4.5	1.731	.747	4.010	.200
Depression at age 11	1.076	1.036	1.118	**<.001****
BMI at age 11	1.051	.956	1.155	.302

Note: -2LL=261.979; R^2=.116 (Cox & Snell), .220 (Nagelkerke); Model $X^2_{(10)}$=51.494, p<.001. SDQ = Strengths and Difficulties Questionnaire; OR = Odds Ratio; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; WISC = Wechsler Intelligence Scale for Children; FSIQ = Full-scale Intelligence Quotient; BMI = Body Mass Index

4.4.2.3 Parental CRS ADHD Index Score at 11 (Categorical outcome measure)

The following predictors were considered in logistic regression analyses to predict Parental CRS ADHD Index Score category at age 11: birthweight; marital status at birth; maternal stress at 3.5; WISC FSIQ at 7; WISC digit span at 7; percentage of sedentary activity at 7 (chosen over other significant measures of moderate activity at 7 as the strongest

predictor); weekend home electronic media at 11; and depression at 11. The proportion of variance accounted for at each step was appraised using the Nagelkerke R Square statistic.

In step 1, birthweight and marital status at birth were entered into the equation, but did not significantly predict the outcome [Chi Square(2)=1.252, *p*=.535], accounting for just .6% of the variance of the outcome measure.

In step 2, maternal stress at 3.5 was added into the model. This significantly improved prediction of the outcome [Chi-Square(1)=4.805, *p*=.028], accounting for an additional 2.5% of the variance (Step 2 cumulative total=3.1%).

In step 3, the following variables were added: WISC FSIQ at 7; WISC digit span at 7; and percentage of sedentary activity at 7. Together these variables significantly improved prediction of the outcome [Chi-Square(3)=13.493, *p*=.004], accounting for 6.6% of the variance (Step 3 cumulative total=9.7%).

In step 4, weekend home electronic media at 11 and depression at 11 were added, and did not significantly improve prediction of the outcome [Chi-Square(3)=2.636, *p*=.451]. Together they accounted for a further 1.3% of the variance, with the total model accounting for 11% of the variance of the Parental CRS ADHD Index scores at 11 (categorical). The overall model is presented in Table 37.

Table 37

Combined multivariate logistical regression for significant predictors of Parental CRS ADHD Index Score (categorical oucome measure)

Predictor	OR	Lower	Upper	p
Birthweight				
AGA	REF			
SGA	.967	.494	1.891	.921
Marital Status at Birth				
Married/Defacto	REF			
Other	1.147	.544	2.419	.719
Perceived Stress Scale at 3.5	1.037	.984	1.094	.177
WISC at 7 FSIQ	.965	.938	.993	.015*
WISC Digit Span at 7	1.006	.892	1.134	.922
Percent Sedentary at 7	.963	.925	1.003	.069
Weekday home electronic media at 11				
<3	REF			.501
3-4.5	1.357	.579	3.180	.482
>4.5	1.646	.717	3.781	.240
Depression at age 11	1.023	.981	1.066	.286

Note: -2LL=253.771; R^2=.057 (Cox & Snell), .110 (Nagelkerke); Model $X^2_{(9)}$=22.187, p<.008. CRS = Conners' Rating Scale; ADHD = Attention Deficit Hyperactivity Disorder; OR = Odds Ratio; AGA = Appropriate for Gestational Age; SGA = Small for Gestational Age; WISC = Wechsler Intelligence Scale for Children; FSIQ = Full-scale Intelligence Quotient;

4.5 Section 5: Exploratory univariate regression analysis

As activity and sleep duration were central to the hypotheses this thesis sought to explore, detailed in the tables below are the univariate results for the Parental SDQ Total Difficulties Score (continuous and categorical) and the Parental CRS ADHD Index (categorical). Linear regression is used for the continuous outcome measures and for categorical outcomes logistical regression identifies the change in risk of belonging to the abnormal group associated with each unit change in activity or sleep duration. Also included in this section are the analyses of the externalising and internalising breakdowns of the SDQ Difficulties scores, and the three DSM ADHD subscales. These results must be treated with

caution due to normality limitations, although the externalising scale was within acceptable limits. With the increased risk of Type 1 errors when so many independent analyses are conducted, additional caution must be employed in interpretation.

4.5.1 Parental SDQ at 11

Table 38

Relationship between activity and Sleep Duration and Parental SDQ Total Difficulties score at 11 (continuous outcome measure)

Predictor	B (SE)	β	Lower	Upper	t	p
Average Counts at 7	.000 (.001)	.021	-.001	.002	.461	.645
Percent Sed at 7	-.040 (.027)	-.067	-.094	.013	-1.480	.140
Percent Mod at 7	.068 (.034)	.090	.000	.135	1.971	**.049***
Percent Vig at 7	-.013 (.069)	-.009	-.149	.122	-.193	.847
Av Time Sed at 7	-.001 (.003)	-.022	-.007	.004	-.471	.638
Av Time Mod at 7	.009 (.004)	.096	.001	.017	2.117	**.035***
Av Time Vig at 7	.000 (.009)	-.002	-.017	0.16	-.046	.964
Sleep duration (hrs) at 7	-.449 (.275)	-.075	-.990	.092	-1.630	.104
Predictor	**B (SE)**	**β**	**Lower**	**Upper**	**t**	**p**
Percent Sed at 11	-7.929^{-5} (.046)	.000	-.090	.090	-.002	.999
Percent Mod at 11	-.010 (.054)	-.008	-.116	.096	-.180	.857
Percent Vig at 11	-.004 (.182)	-.001	-.361	.353	-.022	.982
Av Time Sed at 11	.004 (.004)	.045	-.004	.012	1.048	.295
Av Time Mod at 11	.002 (.006)	.011	-.011	.014	.246	.805
Av Time Vig at 11	.003 (.022)	.005	-.041	.047	.117	.907
Sleep duration (hrs) at 11	-.392 (.293)	-.058	-.967	.183	-1.339	.181

Note. SDQ = Strengths and Difficulties Questionnaire; B = Unstandardised Beta Coefficient; SE = Standard Error; β = Standardised Beta Coefficient; t = Test statistic; Sed = Sedentary; Mod = Moderate activity; Vig = Vigorous activity; Av = Average; hrs = Hours.

Table 39

Relationship between activity and Sleep Duration and Parental SDQ Total Difficulties score at 11 (categorical outcome measure)

Predictor	OR	Lower	Upper	p
Average Counts at 7	1.000	.999	1.001	.772
Percent Sed at 7	.985	.954	1.017	.355
Percent Mod at 7	1.034	.992	1.077	.113
Percent Vig at 7	.965	.886	1.050	.405
Av Time Sed at 7	.999	.995	1.002	.374
Av Time Mod at 7	1.003	.998	1.008	.247
Av Time Vig at 7	.995	.984	1.005	.323
Sleep duration (hrs) at 7	1.112	.800	1.546	.529
Average Counts at 11	1.000	.998	1.001	.840
Percent Sed at 11	1.055	.998	1.115	.058
Percent Mod at 11	.937	.878	1.000	**.050***
Percent Vig at 11	.843	.659	1.080	.176
Av Time Sed at 11	1.003	.999	1.008	.164
Av Time Mod at 11	.993	.985	1.001	.068
Av Time Vig at 11	.980	.950	1.010	.186
Sleep duration (hrs) at 11	.915	.654	1.280	.603

Note. SDQ = Strengths and Difficulties Questionnaire; OR = Odds Ratio; Sed = Sedentary; Mod = Moderate activity; Vig = Vigorous activity; Av = Average; hrs = Hours.

As can be seen in Tables 38 and 39 the only significant relationship found between activity and continuous Parental SDQ Total Difficulties scores at 11, was the time (and percentage of time) spent in moderate activity at the age of 7. When the categorical outcomes were examined the percentage of moderate activity was found to be significantly related, with children who fell in the borderline/abnormal group spending a lower percentage of time exhibiting this intensity of movement.

Table 40

Relationship between activity and Sleep Duration and Parental SDQ Externalising score at 11 (continuous)

Predictor	B (SE)	β	Lower	Upper	t	p
Average Counts at 7	.001 (.001)	.082	.000	.002	1.786	.075
Percent Sed at 7	-.047 (.017)	-.124	-.080	-.013	-2.736	**.006****
Percent Mod at 7	.061 (.022)	.129	.019	.104	2.842	**.005****
Percent Vig at 7	.053 (.043)	.056	-.032	.138	1.223	.222
Av Time Sed at 7	-.002 (.002)	-.066	-.006	.001	-1.439	.151
Av Time Mod at 7	.008 (.003)	.133	.002	.013	2.930	**.004****
Av Time Vig at 7	.007 (.005)	.060	-.003	.018	1.324	.186
Sleep duration (hrs) at 7	-.249 (.174)	-.066	-.592	.094	-1.428	.154
Average Counts at 11	.002 (.001)	.099	.000	.003	2.294	**.022***
Percent Sed at 11	-.053 (.029)	-.079	-.110	.004	-1.821	.069
Percent Mod at 11	.058 (.034)	.073	-.009	.125	1.695	.091
Percent Vig at 11	.123 (.115)	.046	-.103	.349	1.066	.287
Av Time Sed at 11	9.591^{-5} (.002)	.002	-.005	.005	.039	.969
Av Time Mod at 11	.008 (.004)	.087	.000	.016	2.009	**.045***
Av Time Vig at 11	.017 (.014)	.053	-.010	.045	1.228	.220
Sleep duration (hrs) at 11	-.289 (.186)	-.067	-.653	.075	-1.558	.120

Note. SDQ = Strengths and Difficulties Questionnaire; B = Unstandardised Beta Coefficient; SE = Standard Error; β = Standardised Beta Coefficient; t = Test statistic; Sed = Sedentary; Mod = Moderate activity; Vig = Vigorous activity; Av = Average; hrs = Hours.

Table 41

Relationship between activity and Sleep Duration and Parental SDQ Internalising score at 11 (Continuous)

Predictor	B (SE)	β	Lower	Upper	t	p
Average Counts at 7	-.001 (.001)	-.059	-.002	.000	-1.284	.200
Percent Sed at 7	.007 (.015)	.022	-.022	.037	.490	.624
Percent Mod at 7	.006 (.019)	.013	-.032	.043	.290	.772
Percent Vig at 7	-.069 (.038)	-.083	-.144	.006	-1.817	.070
Av Time Sed at 7	.001 (.001)	.038	-.002	.004	.835	.404
Av Time Mod at 7	.001 (.002)	.021	-.003	.006	.453	.651
Av Time Vig at 7	-.008 (.005)	-.076	-.017	.001	-1.666	.096
Sleep duration at 7	-.204 (.153)	-.061	-.504	.097	-1.333	.183
Average Counts at 11	-2.750^{-5} (.001)	-.002	-.001	.001	-.043	.966
Percent Sed at 11	.053 (.025)	.089	.003	.103	2.074	**.039***
Percent Mod at 11	-.068 (.030)	-.097	-.126	-.009	-2.260	**.024***
Percent Vig at 11	-.127 (.101)	-.054	-.325	.072	-1.254	.210
Av Time Sed at 11	.004 (.002)	.079	.000	.008	1.842	.066
Av Time Mod at 11	-.007 (.004)	-.079	-.014	.000	-1.842	.066
Av Time Vig at 11	-.015 (.012)	-.051	-.039	.010	-1.187	.236
Sleep duration at 11	-.289 (.186)	-.067	-.653	.075	-1.558	.120

Note. SDQ = Strengths and Difficulties Questionnaire; B = Unstandardised Beta Coefficient; SE = Standard Error; β = Standardised Beta Coefficient; t = Test statistic; Sed = Sedentary; Mod = Moderate activity; Vig = Vigorous activity; Av = Average; hrs = Hours.

Examining the Externalising and Internalising scorings of the Parental SDQ scores in Tables 40 and 41 a somewhat clearer pattern emerges than when looking at the SDQ Total Difficulties score. It appears that higher levels of physical activity at 7 (reduction in percentage of time spent in sedentary activity and increase in time spent in moderate activity), as well as at 11 (average counts and increased percentage moderate activity) are related to higher externalising scores at 11; with activity at 7 being a more robust predictor than activity at 11. Conversely, an increased percentage of time spent in sedentary activity and a reduced percentage of time spent in moderate activity at 11, but not 7, is significantly related to higher internalising scores at 11. As seen in Appendix I this pattern is replicated by

the Child SDQ Internalising and Externalising scores, suggesting that this more than just a chance finding. No significant relationships were found with sleep duration at either age.

4.5.2 Parental CRS at 11

Table 42

Relationship between activity and Sleep Duration at the Parental CRS ADHD Index at 11 (categorical outcome measure)

Predictor	OR	Lower	Upper	p
Average Counts at 7	1.001	1.000	1.002	.096
Percent Sed at 7	.966	.936	.998	.038
Percent Mod at 7	1.046	1.004	1.090	**.033***
Percent Vig at 7	1.039	.962	1.122	.329
Av Time Sed at 7	.998	.994	1.001	.158
Av Time Mod at 7	1.005	1.000	1.010	**.042***
Av Time Vig at 7	1.005	.995	1.015	.307
Sleep duration (hrs) at 7	.988	.713	1.370	.943
Average Counts at 11	1.000	.999	1.002	.687
Percent Sed at 11	1.015	.960	1.072	.605
Percent Mod at 11	.981	.919	1.047	.561
Percent Vig at 11	.951	7.57	1.195	.665
Av Time Sed at 11	1.001	.997	1.006	.595
Av Time Mod at 11	.998	.991	1.006	.694
Av Time Vig at 11	.996	.969	1.024	.800
Sleep duration (hrs) at 11	.961	.677	1.364	.823

Note. CRS = Conners' Rating Scale; ADHD = Attention Deficit Hyperactivity Disorder; OR = Odds Ratio; Sed = Sedentary; Mod = Moderate activity; Vig = Vigorous activity; Av = Average; hrs = Hours.

As can be seen in Table 42 a higher percentage of time spent in moderate activity at the age of 7 is significantly related to the categorical rating on the Parental CRS ADHD Index at the age of 11, with those in the borderline/abnormal group spending more time in moderate activity. Sleep duration is not predictive of the Parental CRS ADHD Index classification at age 11.

Table 43

Relationship between activity and Sleep Duration and the Parental CRS DSM Subscale T-Scores (continuous outcome measure)

Hyperactive/Impulsive	**B (SE)**	**β**	**Lower**	**Upper**	**t**	**p**
Average Counts at 7	.003 (.001)	.098	.000	.006	2.148	**.032***
Percent Sed at 7	-.128 (.041)	-.141	-.208	-.048	-3.138	**.002****
Percent Mod at 7	.163 (.052)	.142	.061	.264	3.148	**.002****
Percent Vig at 7	.169 (.104)	.074	-.034	.373	1.634	.103
Av Time Sed at 7	-.009 (.004)	-.097	-.017	-.001	-2.141	**.033***
Av Time Mod at 7	.019 (.006)	.137	.007	.031	3.053	**.002****
Av Time Vig at 7	.022 (.013)	.075	-.004	.047	1.666	.096
Sleep duration at 7	-.335 (.418)	-.037	-1.156	.487	-.801	.424
Average Counts at 11	.005 (.002)	.118	.001	.008	2.751	**.006****
Percent Sed at 11	-.071 (.068)	-.045	-.204	.062	-1.044	.297
Percent Mod at 11	.064 (.080)	.035	-.092	.221	.808	.420
Percent Vig at 11	.142 (.270)	.023	-.388	.672	.528	.598
Av Time Sed at 11	.000 (.006)	.003	-.011	.012	.073	.942
Av Time Mod at 11	.008 (.010)	.038	-.010	.027	.876	.381
Av Time Vig at 11	.021 (.033)	.027	-.045	.086	.617	.538
Sleep duration at 11	-.497 (.435)	-.049	-1.352	.358	-1.142	.254
Inattentive						
Average Counts at 7	.001 (.002)	.038	-.002	.004	.836	.403
Percent Sed at 7	-.056 (.047)	-.054	-.149	.037	-1.180	.239
Percent Mod at 7	.086 (.060)	.065	-.032	.203	1.430	.153
Percent Vig at 7	.015 (.119)	.006	-.219	.250	.130	.897
Av Time Sed at 7	-.003 (.005)	-.027	-.012	.006	-.582	.561
Av Time Mod at 7	.010 (.007)	.065	-.004	.024	1.420	.156
Av Time Vig at 7	.003 (.015)	.009	-.026	.032	.205	.838
Sleep duration at 7	-.229 (.478)	-.022	-1.169	.710	-.479	.632
Average Counts at 11	.003 (.002)	.065	-.001	.007	1.502	.134
Percent Sed at 11	-.022 (.079)	-.012	-.177	.134	-.271	.786
Percent Mod at 11	.015 (.093)	.007	-.168	.199	.166	.868
Percent Vig at 11	-.013 (.315)	-.002	-.632	.607	-.040	.968
Av Time Sed at 11	.002 (.007)	.015	-.011	.016	.349	.727
Av Time Mod at 11	.002 (.011)	.009	-.019	.024	.220	.826
Av Time Vig at 11	.003 (.039)	.003	-.074	.079	.070	.944
Sleep duration at 11	-.270 (.511)	-.023	-1.273	.733	-.529	.597

Combined	B (SE)	β	Lower	Upper	t	p
Percent Sed at 7	-.099 (.043)	-.105	-.183	-.015	-2.323	**.021***
Percent Mod at 7	.135 (.054)	.113	.029	.241	2.504	**.013***
Percent Vig at 7	.094 (.108)	.040	-.118	.306	.872	.383
Av Time Sed at 7	-.006 (.004)	-.065	-.014	.002	-1.431	.153
Av Time Mod at 7	.016 (.006)	.111	.003	.028	2.462	**.014***
Av Time Vig at 7	.013 (.013)	.042	-.014	.039	.933	.351
Sleep duration (hrs) at 7	-.311 (.434)	-.033	-1.164	.542	-.717	.474
Average Counts at 11	.004 (.002)	.101	.001	.008	2.354	**.019***
Percent Sed at 11	-.054 (.072)	-.033	-.194	.086	-.755	.450
Percent Mod at 11	.050 (.084)	.025	-.116	.215	.590	.555
Percent Vig at 11	.058 (.285)	.009	-.501	.617	.203	.839
Av Time Sed at 11	.001 (.006)	.010	-.011	.013	.225	.822
Av Time Mod at 11	.007 (.010)	.028	-.013	.026	.658	.511
Av Time Vig at 11	.011 (.035)	.014	-.058	.080	.314	.753
Sleep duration (hrs) at 11	.382 (.459)	-.036	-1.285	.520	-.832	.406

Note: Interpret with caution due to non-normal distribution of Conners' DSM Subscale scores. B = Unstandarised Beta Coefficient; SE = Standard Error; β = Standardised Beta Coefficient; t = Test statistic; Sed = Sedentary; Mod = Moderate activity; Vig = Vigorous activity; Av = Average; hrs = Hours.

As evident in Table 43, when analysed continuously, activity measures do appear to predict scores on the three CRS DSM Subscale Scores as would be anticipated theoretically. The strongest correlations with activity are on the Hyperactive scale, with reduced but significant relationships on the Combined Scale, and no significant relationships on the Inattentive subscale. Again there appears to be no significant relationship with sleep duration on any of these subscales.

5 Discussion

The primary aims of this thesis were to: provide a normative data set of the activity and sleep duration of New Zealand children at the age of 11; compare these findings to similar data at the age of 7; and to examine whether such measurements could act as predictors of emotional and behavioural problems at the age of 11. It was also hoped to examine whether such measures had diagnostic utility beyond more established risk factors for ADHD, and whether they were associated with subjective ratings of motoric hyperactivity. Due to normality restrictions this investigation was largely limited to Parental outcome measures, on both the Strengths and Difficulties Questionnaire (SDQ) and the Conners' Rating Scale (CRS). To explore specific hypotheses of this study, the subscale scores of these measures were also examined tentatively.

5.1 Normative Data

5.1.1 Normative data: Activity

As described in the methodology section, much of the literature that looks at physical activity is linked to obesity and risk factors for cardio-vascular disease; and this was the original rationale for the inclusion of activity data in the ABC study. With a very small established literature using activity data as a predictor of psychological outcomes, physical health studies were used to inform the cut-off values employed in the current research. These cut-offs classify activity into sedentary, moderate, or vigorous intensity. Many health studies cite the time spent in moderate and vigorous physical activity (MVPA) as a principal predictor variable, but even today these values can be influenced by a researchers' choice of one of the 13 available published sets of cut-points. As Brazendale et al. (2015) highlight, in the absence of universally accepted cut-points, disparities can occur even among ostensibly similar studies, considerably impeding the synthesis of any literature on child and adolescent activity. To prevent such ambiguities, the following comparisons between this study's

activity recordings and the existing literature, are made using mean Actigraph counts rather than measures of intensity.

The closest comparison to the current study that cites mean activity counts was a UK study involving 5595 children who were measured over a 7 day period (Riddoch et al., 2007). Riddoch et al. (2007) found that the median physical activity level of 11 year old boys was 644 [IQR 528-772] counts/min, and of 11 year girls was 529 [IQR 444-638] counts/min. In the current study the median Actigraph count for 11 year old boys was 552 [IQR 460-671] counts/min, and 497 [IQR 385-577] counts/min for 11 year old girls (see Table 20, page 98). Based on median values, the children in the Riddoch et al. (2007) sample were 12% more active overall than those in the current study (boys 17%; girls 6%), with boys accounting for much of this difference. They also found boys to be 22% more active than girls, which is larger than the 11% difference found in the current study. Like the Riddoch et al. (2007) study, boys in the ABC sample at age 11 were significantly more active than girls (see Table 20, page 98), as was found at age 7 (Cornforth, 2007). No differences between birthweight groups were observed.

Considering that the ABC sample is known to contain an overrepresentation of more highly educated upper SES families (see Table 3, page 61), part of the reason why the ABC sample was slightly less active may be explained by the fact that Riddoch et al. (2007) also found an inverse relationship between level of parental education and the child's physical activity. A systematic review did, however, find that the relationship between SES and physical activity was far from uniform (Stalsberg & Pederson, 2010) and correlational analysis on the current data set found no consistent or significant relationship between mean activity and indicators of SES. Perhaps a more parsimonious explanation is that the Apple iPhone was released in 2007, essentially popularising the smart-phone. As identified by Arif et al. (2014), the very technology that now makes activity monitoring feasible in the general

population, may also contribute to reductions in activity and the rising rates of obesity seen in NZ (NZMOH, 2015; 23.2% of the current sample fell in the 'overweight' range).

While the activity levels found by the Riddoch et al. (2007) study and the current sample are broadly equivalent, they both illustrate a well-documented international trend that today's children are not active enough according to current recommended levels of activity for physical health. Only 2.9% of the children in the Riddoch et al. (2007) sample met current internationally recognised recommendations of at least 60 minutes of physical activity a day. This is of particular concern given that the children in the ABC study were even less active. As shown recently by Mangerud, Bjerkeset, Lydersen and Indredavik (2014), the lower levels of physical activity typically demonstrated by adults suffering psychiatric disorders, can also be observed in the adolescent population. While they found variations in the direction of the associations between activity levels and different disorders, as illustrated in Table 1 (page 8), their broader findings support the well-established bidirectional relationship between the most common mental health difficulties and inactivity (Elfrey & Ziegelstein, 2009). If studies on adolescent clinical populations are generalisable to the ABC sample, in addition to the impact on their physical health, such low levels of activity may also become of concern to their mental wellbeing as they reach their teenage years (Vallance et al., 2011).

5.1.2 Normative data: Sleep Duration

The current study found that on average 11 year old boys slept for 9.42 (SD= .077) hours and girls for 9.57 (SD=.69) hours (Table 22, page 99). The closest comparison study that used Actigraphs and that employed the Sadeh algorithm (Sadeh et al., 1989) to distinguish between sleep and wake times, involved 679 Danish children (Hjorth et al., 2014). The participants had a mean age of 10 and the study found that boys slept for 9.22 (SD=.45) hours and the girls for 9.26 (SD=.47) hours. These findings were very similar to the current results, with girls in both studies sleeping slightly longer, and on average all children sleeping

for over 9 hours. In a recent review of the available evidence for optimal sleep durations for children, Martricciani et al. (2013) concluded that there is insufficient data to support sleep recommendations for children. They did, however, provide a composite figure of various international guidelines for sleep duration, which is shown in Figure 12. As can be seen from the figure, the data in the current study fell between the European and USA recommendations.

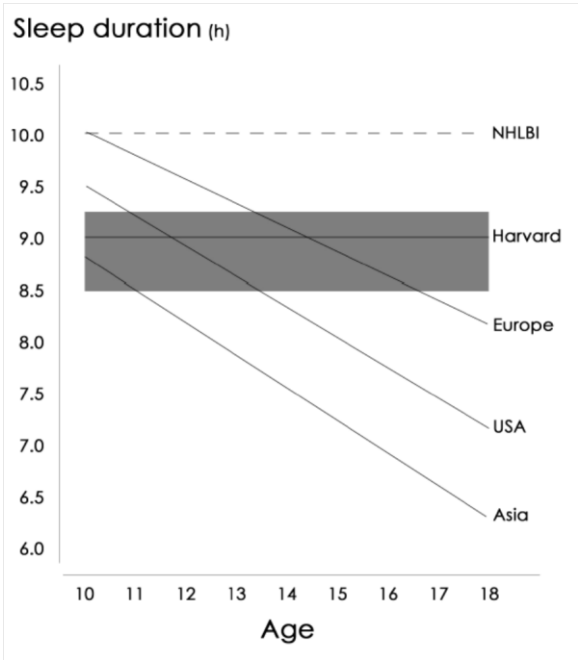

Figure 13: International recommendations for mean childhood sleep durations (from Matricciani, Blunden, Rigney, Williams & Olds, 2013). National Heart, Lung and Blood Institute (NHLBI) guidelines; Harvard guidelines; European, USA, and Asian averages from Old, Maher and Matricciani, (2011)

5.2 Outcome Measures at Age 11

5.2.1 Strengths and Difficulties Questionnaires

Preliminary analysis of the parental scores on the SDQ highlighted a number of significant findings, which while incidental to main focus of the thesis, are notable. For the first time in the history of the ABC study, SGA children could be distinguished from AGA controls on a psychological measure. Cornforth (2007) looked at the Parental SDQ Total Difficulties scores categorically from the ABC sample at ages 3.5 and 7. She found differences between genders, with males showing more difficulties, but not between

birthweight groups. In the current study at age 11, while there was no interaction between the two, significant main effects of both gender and birthweight were observed on the Parental SDQ Total Difficulties score. The differences between birthweight groups became more apparent when analysed separately by gender.

On the SDQ Parental Total Difficulties measure, and on all of the difficulty subscales, males and SGA children showed consistently higher problem scores (see Table 9, page 83). When the subscales that make up the Total score were examined, it was discovered that the significant effect of gender by was driven by Peer difficulties, Hyperactivity, and Conduct difficulties. The significant effect of birthweight was driven by Emotional difficulties and Hyperactivity, with Conduct difficulties approaching significance.

An apparent trend of elevated difficulties for males and SGA children was also seen when Parental SDQ subscale scores were examined categorically using standard clinical cuts-offs (see Table 10, page 85). The only significant categorical difference of birthweight was however on the Emotion subscale, with SGA children showing more problems. When examined categorically, males showed greater difficulties on the Conduct, Hyperactivity, and Peer subscales. The fact that Emotion subscale showed an effect of birthweight, but not of gender, suggests that these emotional difficulties are seen more broadly in SGA children. Unlike SGA females, SGA males however appear to show more salient difficulties across a number of additional domains, including externalising issues likely to be viewed negatively by others. While this is the first time such differences have been shown using the current data set, this is consistent with findings from NZ (Silva, McGee & Williams, 1984), and more recent research internationally (Lundgren & Tuvemo, 2008), showing increased behavioural difficulties in SGA children.

The same pattern of effects observed for Parental SDQ scores was also seen for Child SDQ scores at age 11, with main effects of both gender and birthweight on the Total

Difficulties score (see Table 13, page 88). Males scored more poorly on the Peer, Hyperactivity, Emotion, and Conduct scales. SGA children scored worse on the Hyperactivity and Emotion subscales, with the Conduct subscale approaching significance. Again these trends were evident when the data was examined categorically (see Table 14, page 91), although there was only a significant effect of birthweight on the Total Difficulties categorisation, and a significant effect of gender on the Conduct subscale.

Caution must be employed interpreting all of the SDQ subscale scores due to normality violations, however there is a consistency of results across both Parental and Child scales, and the continuous and categorical outcome measures. This supports the interpretation that the results illustrate a genuine trend of SGA children showing greater difficulties than AGA children. Again, while no significant interactions were found between gender and birthweight, the highest average difficulty scores were consistently exhibited by SGA males.

5.2.2 Conners' Rating Scales

On the Parental CRS indices a main effect of gender was observed across all four scales that were examined (CRS ADHD Index; CRS DSM Hyperactive, Inattentive, Combined), but no effect of birthweight was seen (see Table 16, page 93). Similarly to the SDQ scores, males exhibited greater difficulties. While not significant, SGA children also showed a trend towards higher difficulties scores across all scales, when compared to their gender-matched AGA peers. When examined categorically (see Table 17, page 94) these trends were also evident, but the only significant differences were for males on the Parental CRS ADHD Index and CRS DSM Inattentive scales.

A similar pattern of results was also observed across the Teachers CRS scores (see Table 18, page 95). While again there were no main effects of birthweight, between-subject effects for birthweight were observed across all three of the Teachers CRS DSM subscales. This was most notable the two subscales that included symptoms of hyperactivity

(Hyperactive and Combined). This discrimination became more apparent when the Teacher results were examined categorically, with elevated rates of abnormal/borderline scores among the SGA children (Table 19, page 96). The only significant categorical effect of birthweight was on the DSM Inattentive index. Such a differential level of sensitivity between Parental and Teacher categorical scores is likely due to the fact that far fewer of the Teacher scores fell within the abnormal/borderline range. Unfortunately, the smaller numbers of children identified by teachers as having problems, led to the statistical limitations that prevented Teacher scores from being taken forward to the multivariate analysis stage.

5.2.3 Comparing the outcome measures

In linking the previous two sections to the literature, consistent with previous studies, males showed statistically higher emotional and behavioural problems across the majority of the outcome measures (Cornforth, 2007). The outcome measures at age 11 seem to confirm an entirely consistent trend towards SGA children, and especially SGA males, showing a greater level of subjectively reported behavioural and emotional difficulties when compared to their AGA peers. For the first time in the history of the current study a difference between birthweight groups was found to be statistically significant (Cornforth et al., 2012); on the Parental SDQ Total Difficulties scale.

While the lack of normality of the individual SDQ subscale scores prevented definitive conclusions, when a two-factor breakdown of the subscales scores was examined, higher internalising scores were the most evident difficulties presented by SGA females. In contrast, SGA males exhibited broader issues across both the internalising and externalising domains (Table 11, page 87). Such trends were also identifiable among the Child SDQ data, suggesting that self-report measures at age 11 identify similar issues to parental report (see Table 15, page 92). To the author's knowledge this is the first time that evidence has been presented to suggest that there may be a distinction between the nature of the emotional and

behavioural difficulties suffered by SGA males and females. This may of course represent a more general pattern of how males and females manifest psychological difficulties at this age, rather than being specifically linked to birthweight (Herting et al., 2015). A recent study by Johnson et al. (2015), albeit a study of late and moderately late premature babies, may however account for the current results, as they found male gender to be an independent predictor of poorer neurodevelopmental outcomes.

Despite SGA children showing consistently higher scores across all of the CRS indices examined, given that the CRS scale focuses on a smaller range of problem behaviours, it is perhaps unsurprising that any differences across birthweight groups were not as statistically robust as on the SDQ. Consistent with Cornforth (2007) at age 7, teachers tended to give lower scores on the CRS. When compared to parents, teachers appear to be identifying a smaller subset of children at the more problematic end of the spectrum, among which SGA children were overrepresented. While the only significant effect of birthweight was found on the categorical Teachers CRS DSM Inattentive subscale, this may support the finding of Gadow et al. (2004), which suggests that observers interpret symptoms most relevant to the domain in which the child is observed.

5.3 Changes Over Time

5.3.1 Outcome measures across time

The higher difficulties scores demonstrated by SGA children at age 11, were not apparent at age 7 (Cornforth, 2007). This suggests the possibility that SGA children may be carrying latent psychological difficulties, or structural neurological differences that make them more vulnerable to such difficulties, which become more apparent as they grow older. There is clear evidence from infants who have suffered traumatic brain injuries (Barlow, Thompson, Johnson & Minns, 2005) and those born at a very low birthweight (Wolfe et al., 2015) that social and behavioural difficulties may manifest later in adolescence. This is true

especially if the affected brain region is the frontal lobes, which develop later, coinciding with a developmental stage where psychosocial expectations increase (Thompson et al., 2005).

While there was no interaction between age and gender on the Parental SDQ continuous scores, all groups other than SGA boys showed a reduction in SDQ scores between the ages of 7 and 11 (see Figures 5 & 6, pages 101 & 103). When examined categorically and visually, it became apparent that SGA boys appear to be demonstrating quite a distinct trajectory, beginning to show greater difficulties between the ages of 7 and 11. This trend also appeared evident on the Parental CRS scores, although it did not reach significance (see Figures 7 & 8, pages 105 & 107). This was also supported by the children's own appraisal of their difficulties on the Child SDQ scores at 11 (see Table 14, page 91).

If the trend shown in this study towards male SGA children showing even greater difficulties than SGA females could be reproduced, this may fit with evidence for the relative biological fragility of the male foetus; vulnerabilities further compounded by social attitudes that expect boys to be more resilient (Kraemer, 2000). In addition, pubertal hormone changes that have gender specific effects on brain development, may contribute to the emergence of sex differences in internalizing and externalizing behaviour during adolescence (Herting et al., 2015). The externalising behaviour problems, shown by this study to be disproportionately exhibited by males, tend to be more disruptive and conferred greater social stigma than other difficulties (Miller, 2013). Such difficulties may therefore be more salient to untrained observers, such as parents and teachers. This is likely to be especially apparent at the age of 11, around the onset of puberty, where greater social and academic expectations are placed on children, but where girls tend to have started to mature 1 to 2 years earlier than boys (Bramen et al., 2012). It also supports the link between early pubertal maturation and the development of internalising disorders in girls (Natsuaki et al., 2009).

5.3.2 Predictor variables across time

Based on mean activity counts, a significant 32% reduction in activity was observed from the age of 7 to 11 across the whole sample (see Table 28, page 108). This was reflective of an 18.1% increase in time spent in sedentary activity, a 14.5% reduction in moderate activity, and a 3.7% reduction in vigorous activity. Such a reduction in activity was consistent with the findings of Gavarry et al. (2003) and Eaton, Mckeen, and Cambell (2001; illustrated in Figure 1, page 4). In terms of group differences in the level of reduction in activity between the two ages, no main effects of gender or birthweight were observed.

Between these ages of 7 and 11 average sleep duration fell from 10 to 9.5 hours (see Tables 29 & 30, pages 109 & 110). There were no main effects of gender or birthweight at age 11, or in the level of reduction between the two ages. While the average sleep duration at age 11 is similar to the Hjorth et al. (2014) study, the average duration at age 7 is 30 minutes less than that found in a sample of 6-7 year old Australian children (Price, Quach, Wake, Bittman & Hiscock, 2015). The gradient of reduction is also less steep than can be extrapolated from the composite figure of recommended sleep times presented by Matricciani et al. (2013; see Figure 12, page 136). Together this suggests that while sleep duration of the ABC sample lies between US and European recommendations at age 11, the children may have been sleeping for shorter periods than recommended at the age of 7.

5.4 Activity and Sleep Duration as Predictors of the Outcome Measures

5.4.1 Activity and Sleep Duration as Predictors of the Overall Outcome Measures

5.4.1.1 Parental SDQ at 11

Of the activity and sleep duration measures, the only significant predictor of the Parental SDQ Total Difficulty score (continuous) at age 11, was the time spent in moderate activity at age 7 (see Table 38, page 126). Those children who showed a higher percentage of

moderate activity at age 7, scored higher on the SDQ Total Difficulties score at age 11. The only predictor of Parental Total Difficulty score (categorical) was the percentage of time spent in moderate activity at 11, with those falling in the borderline/abnormal range spending 1% less [$mean_{normal}$(SD)=13.07(4.04)%; $mean_{borderline/abnormal}$(SD)=12.04(3.97)%] of their awake hours in moderate activity (see Table 39, page 127).

While the differences are small, the findings of this study therefore suggest that children who showed greater moderate activity at age 7, and less moderate activity at age 11, were more likely to exhibit higher Parental SDQ Total Difficulties scores at 11. As such the direction of the relationship between SDQ scores and activity may be changing as the children age. Increased activity at age 7, but reduced activity at 11, appears to be the predominant indicator of subjectively rated behavioural and emotional problems at 11. The relationship with activity at age 11 is consistent with the findings of Ussher et al., (2007) and Heger et al. (2014), who found lower levels of activity to be a predictor of diminished psychological wellbeing among adolescents. No relationships were found with sleep duration.

5.4.1.2 Parental CRS ADHD Index at 11

Significant predictors of the Parental CRS ADHD Index categorisations at 11 included both the percentage and time spent in moderate activity at age 11 (Table 42, page 130). Unlike the inverse relationship between activity and the SDQ scores at age 11, the relationship between activity and CRS scores at age 11 was in the opposite direction (see Table 42, page 130). Those children in the borderline/abnormal group of the Parental CRS ADHD index spent an average of 2% [$mean_{normal}$(SD)=27.27(6.52)%; $mean_{borderline/abnormal}$(SD)=29.22(3.97)%] and 16 minutes [$mean_{normal}$(SD)=217.39(55.38) mins; $mean_{borderline/abnormal}$(SD)=233.02(57.10) mins] more time per day engaged in moderate activity. The reason that moderate activity seems to be the most sensitive predictor is likely to

be because it is the most normally distributed, capturing a broader range of activities. No relationships were found with sleep duration.

5.4.1.3 Challenges of using activity and Sleep Duration to predict the outcome measures employed by this study

Greater activity at age 7 appears to predict higher Parental SDQ Total Difficulties scores at age 11, and lower activity at age 11 appears to predict higher Parental SDQ Total Difficulties scores at age 11. Due to the current analysis being conducted at a group level, it is unclear whether these contrasting relationships are driven by the same children. If this could be demonstrated, it may provide further evidence for the hypothesis that ADHD represents an evolving condition, supporting concerns about the validity of hyperactivity being a key feature of the disorder in adolescence (Willcutt et al., 2012). Contrary to this hypothesis, however, is the finding that children showing greater levels of moderate activity at 11 were more likely to exhibit difficulties on the CRS ADHD Index at age 11. This relationship was not evident between activity at age 7 and the CRS Index score at age 11. As such, the results may instead represent that hyperactivity at age 7 is predictive of a broad range of emotional and behavioural disorders at age 11, but that hyperactivity at age 11 may be a more specific indicator of ADHD at age 11. While for this subset of children at age 11, hyperactivity may still be a predictor of emotional and behavioural problems, the predominant relationship of concern appears to be with low levels of activity. This potentially fits with the finding that, on average, internalizing problems increase and externalizing problems decrease with age (Bongers et al. 2003). Such ambiguity, introduced when looking at total scale scores, confirms the utility of examining the relationship between the individual subscales of the outcome measures and activity; or at least examining the relationship between activity and the internalising and externalising subscales separately.

Sleep duration showed no predictive relationship with any of the outcome measures. While surprising, given the strong theoretical relationships between sleep and mental health conditions, it is consistent with the recent findings of a similar study conducted in Australia (Price et al., 2015). Price et al. (2015) found weak and inconsistent relationships between sleep duration and measures of child mental health using the SDQ, leading them to suggest that measures of sleep quality and scheduling may be more important.

5.4.2 Activity and Sleep Duration as predictors of the outcome measure subscales

A summary is offered of the relationship between activity and sleep duration and the subscales of the Parental SDQ and CRS. Any conclusions in this section are however tentative, due to the non-normal distributions of the scores on these subscales. To limit the potential impact of such issues, these relationships were examined categorically.

5.4.2.1 SDQ subscales at age 11

Activity was shown to be related to the Hyperactivity, Conduct, and Emotional difficulty subscale categorisations on the Parental SDQ at age 11 (normal vs. borderline/abnormal; see Appendix J). No relationships were found with sleep duration.

Activity at age 7 showed no significant relationships with categorical Hyperactivity scores at age 11, however a number of measures of activity at age 11 were related to the Hyperactivity scores at age 11. These included, average activity counts at age 11, and the percentage of time and average time spent in vigorous activity at age11. This is consistent with the hypothesis posed in the previous section (page 146) to explain the relationship of activity the two ages with the three selected outcome measures; that over-activity at age 11 is a more reliable predictor than activity at age 7, of ADHD-like behaviours at age 11. This would make intuitive sense given that an ADHD diagnoses is based primarily on current rather than past behaviours (DSM-V; APA, 2013). While there was a trend for there to be

greater activity within the borderline/abnormal groups on the Hyperactivity scale, these differences were not shown to be significant by subsequent independent samples T-tests (Appendix K).

The Conduct subscale classifications at age 11 were significantly related to the percentage of time spent in sedentary activity at age 7, as well as the percentage of time and average time spent in moderate activity at age 7 (Appendix J). Those in the borderline/abnormal group spent less time engaged in sedentary activity, and more time engaged in moderate activity, so were more active at age 7. T-tests showed all of these relationships to be significant (Appendix K). As detailed in the literature review (pages 34-35) the direction of the relationship between activity and conduct problems is not clear, but this finding would support the trend of higher levels of activity being associated with externalising problems. It also suggests that increased activity at age 7 may be a better predictor of the Parental Conduct difficulty subscale at age 11, than it is of the Hyperactivity subscale at age 11.

Classifications on the Parental SDQ Emotional difficulties subscale at 11 were significantly related to the percentage of sedentary activity at age 11, and the percentage and time spent in m oderate activity at age 11 (Appendix J). Those in the borderline/emotional group spent greater time in sedentary activity and less time in moderate activity, so were less active at 11. T-tests found these relationships to be significant, consistent with Ussher et al. (2007).

In summary: higher levels of activity at age 7 appear to be associated with greater difficulties on the Parental SDQ Conduct scale at age 11; higher levels of activity at age 11 appear linked to increased Hyperactivity subscale scores at age 11; and lower levels of activity at age 11 appear to predict higher scores on the Emotional difficulties subscale at age 11. While statistically weak, all of these relationships are theoretically consistent with the

literature reviewed, which supports the possibility that internalising and externalising disorders can be distinguished using activity measures (see pages 6-8).

When the Parental SDQ results were scored using the Externalising (Hyperactivity and Conduct) and Internalising (Emotion and Peer) subscales, the relationship with activity was even more apparent. The following activity measures were significantly related to the Externalising score at age 11: percentage of sedentary activity at age 7; percentage of moderate activity at age 7; average time in moderate activity at age 7; average counts at age 11; and average time spent in moderate activity at 11 (see Table 40, page 128). The direction of all relationships showed that higher Externalising scores were associated with increased activity. The only predictors of the Internalising scores at age 11, were an increase in sedentary activity at age 11, and a decrease in moderate activity at age 11 (see Table 41, page 129). This further supports the hypothesis that increased activity is associated with externalising problems and decreased activity with internalising problems. It also demonstrates that such relationships may feasibly be disguised if the Internalising and Externalising subscales are combined to form the SDQ Total Difficulties score. This may explain why previous studies have only observed weak relationships between this measure and ratings of activity (Cornforth, 2007), suggesting that re-analysis using subscales might be warranted.

5.4.2.2 CRS subscales at age 11

The relationship between activity and the Parental CRS also became clearer when the DSM subscale scores were examined. Significant associations were seen between CRS ADHD Hyperactive/Impulsive subscales scores at age 11 and the following predictors: average counts at 7; percentage of time and average time spent in sedentary activity at 7; percentage of time and average time spent in moderate activity at 7; and average activity counts at age 11 (see Table 43, page 131). All of these relationships were in the same

direction, with higher measures of activity predicting higher Hyperactive/Impulsive scores. As would be expected, an almost identical pattern of results was seen for the Combined subscale, but with a weaker level of association; as the scale includes a broader array of behaviours. Interestingly no associations were seen with Inattentive scale for either activity or sleep duration. Such a pattern of relationship is theoretically consistent and supports the hypothesis that high scores on Hyperactive/Impulsive and Combined subscales of the CRS should be distinguishable from the Inattentive subscale based on activity. Unlike the findings derived from the Parental CRS ADHD Index scores, which suggested activity at age 11 was a stronger predictor of ADHD at age 11, activity at 7 was the only predictor of higher scores on the hyperactive subscales (Hyperactive and Combined) of the Parental CRS at age 11.

An interesting distinction was observed when the Teacher CRS DSM subscale scores at 11 were examined continuously (Appendix L). In an opposite pattern to the Parental scores, neither the Teacher CRS Hyperactive/Impulsive nor the Combined subscales showed any relationship with activity, but the Inattentive subscale did. Measures of activity associated with the Teacher CRS Inattentive subscale scores at 11 included: the percentage of time spent in sedentary activity at 11; the percentage of time and average time spent in moderate activity at 11. Again these relationships were all consistent, with lower levels of activity predicting higher scores on the Teachers CRS Inattentive subscale. While Konrad et al. (2005) found a relationship between greater levels of inattention and higher levels activity, the current finding was in the opposite direction, with children rated as being more inattentive by teachers moving less.

Scores on the Teachers CRS was the only time in the current study that sleep duration was predictive, with reduced sleep duration at age 11 being associated with increased scores on the Teachers CRS Inattentive scale (Appendix L). As suggested by Brewis (2002), these findings support the hypothesis that Parents and Teachers capture different aspects of

behaviour. Parental ratings on the CRS appear to provide a clearer idea of motoric hyperactivity and Teacher ratings capture reduced levels of activity and sleep duration, which are associated with inattention.

5.5 Relationship Between Activity and Sleep Duration, Common Risk Factors for ADHD, and the Outcome Measures

The current study highlighted a large array of environmental, physical, and psychosocial variables that are significantly correlated with the outcome variables (see Table 32, page 116). Many were highly inter-correlated, or measuring similar underlying constructs. However, once multicollinearity had been accounted for, a number of broadly independent predictors remained.

Hierarchical regression analysis, including the following significant independent predictors, accounted for 18% of the variance of Parental SDQ Total Difficulty Scores at 11: male gender; maternal cannabis use during pregnancy; lower WISC FSIQ at age 7; higher weekend home electronic media at 11; and greater depression at age 11.

Hierarchical logistic regression, including the following factors, accounted for 22% of the variance when the Parental SDQ Total Difficulties scores at 11 were examined categorically: birthweight; gender; maternal age at birth; income at birth; marital status at birth; maternal smoking during pregnancy; maternal cannabis use during pregnancy; maternal stress at 3.5; WISC FSIQ at age 7; living with father, BMI, weekend home electronic media, and depression at age 11. The only significant independent predictors were however a single marital status at birth, higher maternal perceived stress at age 3.5, and higher child depression at age 11.

Hierarchical logistic regression, including the following factors, accounted for the 9.7% of the variance of the categorical Parental CRS ADHD Index results at age 11: birthweight; marital status at birth; maternal stress at 3.5; WISC FSIQ at 7; WISC digit span at 7;

percentage of sedentary activity at 7; and BMI, weekend home electronic media, and depression at 11. The only significant independent predictor was lower WISC FSIQ at age 7.

In relation to the principal questions explored by this study, only two measures of activity were incorporated in the hierarchical modelling: average minutes of moderate activity at age 7 in the Parental SDQ (Continuous) model; and percentage of sedentary activity at age 7 in the Parental CRS (Categorical) model. As a whole the three models contributed towards the explanation of a reasonable proportion of the variance for each of the outcome measures, but as individual predictors the activity variables made only modest contributions, and were not significant predictors on their own. Sleep duration showed no predictive relationship with the selected outcome measures. As such, based on the outcome measures employed in this study, activity and sleep duration are outperformed as predictors by the selected covariates.

5.6 Conclusions and Clinical Implications

This study has provided normative data for a sample of 11 year old NZ European children, against which objective measures of physical movement and sleep duration can be compared. In exploring the impacts of birthweight and gender on these results, it is apparent that gender specific norms should be used for activity, but are not needed for sleep duration.

This study has also provided normative data for the SDQ and CRS, with ratings provided by multiple observers. For the first time in this history of the ABC study, there is evidence that SGA children may be experiencing more emotional and behavioural difficulties as they grow up when compared to their AGA peers. While such difficulties appear more notable among boys, this may in part be because SGA males show a broader array of raised problem scores when compared to SGA females. This included externalising traits that are not as present in SGA females and which are likely to be more salient to external observers. In both genders, however, SGA children appear to show elevated levels of emotional

difficulties. This suggests that it may be a prudent public health measure to screen low-birthweight adolescents for emotional and behavioural difficulties. For low-birthweight boys in particular, it may also be important conduct a more thorough mental health and cognitive screen if they demonstrate conduct problems, before such behaviours become stigmatising.

Previously research on this data set showed only very weak relationships between subjective measures of activity and measures of childhood emotional and behavioural difficulties (Cornforth 2007). By employing objective measures of activity, a number of significant associations were observed. The most promising measure of activity to predict such difficulties appears to be to partition activity recordings into the proportion of time spent in sedentary, moderate, and vigorous activity. Moderate activity appears to be the most useful of these measures, capturing a greater range of activities, and therefore being more statistically robust. Supporting the findings of Matier-Sharma et al. (1995), based on the current outcome measures, the strength of these associations does not however appear sufficient to offer clear diagnostic utility as a screening measure.

Caution must be employed not to over-interpret the univariate relationships between activity and the subscales of the outcome measures. It had always been hoped to model these subscales independently, but this was not permitted due to normality restrictions. Their examination did however appear to illuminate a possible reason why only weak relationships were seen between activity and the outcome measures employed. As hypothesised, it appears that the internalising and externalising dimensions of the SDQ, and the Hyperactive and Innattentive subscales of the CRS, have differential relationships with activity. This supports the construct validity of these scales, and their ability to offer an appraisal of motoric hyperactivity. It does however also demonstrate that that the relationships of these subscales and activity may be cancelling each other out when combined to calculate the outcome measures utilised by this study. Therefore, while activity remains unlikely to have clinical

utility as a screening measure within the general population, it may have diagnostic utility when honing the diagnosis of a child already within a clinical setting; supporting the findings of Teicher et al. (2006).

Higher levels of activity at age 7 appear predictive of Parental CRS hyperactivity scores (DSM Hyperactive/Impulsive & Combined) and SDQ Conduct scores at age 11. In contrast, higher levels of activity at age 11 appear linked to higher scores on the overall Parental CRS ADHD Index, but not specifically the hyperactivity subscales. As such, there appears to be a transition, with activity at age 7 being predictive of hyperactive ADHD symptoms at age 11, and activity at age 11 being predictive of a broader array of both hyperactive and inattentive symptoms at age 11. This may explain the inconsistent relationships observed between measures of ADHD and measures of activity (see page 31), supporting the evolution of the ADHD diagnosis from predominantly hyperactive symptoms, to more inattentive symptoms as children grow older (Lahey et al., 2005).

Subscale results also support the hypothesis that parental and teacher ratings are capturing different aspects of child behaviour (Gadow et al., 2004). This is likely due to environmental expectations and restrictions, which limit the range of behaviours that can be expressed and observed. It does however potentially inform the weighting that is given to parental and teacher reports when considering differential diagnoses. Based on the current findings, parental ratings appear more sensitive at identifying motoric hyperactivity and teacher ratings appear more attuned to the relationship between inattention and low levels of activity and reduced sleep duration. Based on the findings from the SDQ it is possible that this reduced activity may however be linked to internalising problems. An exploration of a potential link between these findings merits further investigations, so that children are not categorised as being inattentive, when they are in fact experiencing emotional difficulties such as depression.

DISCUSSION

Given how intimately sleep is related to so many emotional and behavioural disorders (Harvard Mental Health Letter, 2009), it was surprising that it proved to be such a poor predictor. Other than one possible relationship with Teacher CRS DSM Inattention at 11, which would need to be confirmed due to the non-normal distribution of this measure and the possibility of Type 1 errors, no associations were seen with sleep duration. One interesting incidental finding did however become apparent when preparing the sleep data for analyses. For the Sadeh algorithm (Sadeh et al., 1989) to accurately calculate the point at which a child falls to sleep, the parents must provide an approximately accurate estimate of this time. As became apparent, due the large percentage of children for whom this calculation had to be made manually, many parents provided very inaccurate estimates; with children falling asleep later than assumed. Looking at the average hours spent engaged with electronic media in the current study, and existing the literature (Shochat, Flint-Bretler & Tzischinsky, 2010; Van den Bulck, 2010) it is hypothesised that technology use may play a role in this discrepancy.

A principal argument in this thesis is that psychosocial variables are often minimised or overlooked when considering the aetiology of disorders such as ADHD, in favour of genetic explanations. While activity was examined as a predictor of emotional and behavioural problems, this was to explore how robustly activity is assessed by subjective measures, and whether it may contribute towards screening for children who may merit additional support. There was no underlying assumption that activity necessarily represents a causal factor in the development of such disorders. There was, however, a contention that psychosocial variables may influence the scales used in the assessment of ADHD and contribute towards Genetic x Environmental interactions. This study demonstrated that a number of such psychosocial factors proved to be robust predictors of emotional and behavioural problems at the age of 11. On the SDQ these included: male gender; maternal cannabis use during pregnancy; single marital status at birth, higher maternal perceived stress

at age 3.5; lower WISC FSIQ at age 7; higher weekend home electronic media at 11; and greater depression at age 11. The only significant independent predictor of the CRS was lower WISC FSIQ at age 7.

While gender is not strictly a psychosocial variable, findings from this study suggest that males are more vulnerable to emotional and behavioural difficulties. While not significant within multivariate analysis, SGA males appear to show a consistent trend of higher problem scores. Johnson et al. (2015) found that male gender and low SES were both independent predictors of poor neuropsychological outcomes in low-birthweight children. Combined with the current results that single marital status and higher maternal stress are also predictors of increased problems scores, males are likely to be particularly vulnerable to interactions between constitutional and psychosocial risk factors, consistent with Kraemer's (2000) conception of the "fragile male."

There are a number of explanations of how single maternal status and higher levels of maternal perceived stress at the age of 3.5 may influence results on a parental scale. Further investigation is merited to whether higher maternal stress leads to children experiencing more problems, or whether the children are being perceived to have more problems as a result of their mother's being under greater pressure. It is notable that parental stress is known to impact ratings between parents of the same child on the same measure (Langberg et al., 2010). Yet, a cursory analysis of the relationship between Child SDQ Total Difficulties at 11 and maternal perceived stress at age 3.5, showed a significant positive relationship (r^2=.091, *p*=.043). This supports the contention that the children of mothers' recorded as being under greater stress when the child was 3.5 years old, are themselves experiencing greater personal difficulties at age11. Regardless of whether this relationship is mediated by birthweight, parental stress may contribute towards raised male difficulty scores, as Biederman et al.

(2002) found that boys are more at risk for adverse cognitive and interpersonal outcomes when exposed to psychosocial adversity.

While the relationship between depression at 11 and the SDQ seems less interesting, as it is an issue captured by the SDQ, it does highlight that child-rated depression is a central feature of the emotional and behavioural difficulties perceived by parents when their children are aged 11. The findings from this study suggest that across other indices, parental and child measures at age 11 capture similar patterns of difficulties.

Finally, it was found that IQ at age 7 is the strongest predictor of the CRS ADHD Index at 11. As noted in the NZ Guidelines for the Assessment and Treatment of ADHD (NZMOH, 2001), while "tests of general cognitive functioning and neuropsychological tests are not routinely carried out in the public education system in New Zealand, in some countries such testing appears to be routine when learning problems are present". Both at age 7 (Cornforth, 2007), and now at age 11, a significant relationship has been found between lower IQ scores and higher difficulty scores on both the CRS and SDQ. This suggests that within a NZ context, when ADHD or other emotional and behavioural problems are suspected, an assessment of the child's neuropsychological performance would be a valuable addition to the clinical assessment process.

5.7 Strengths of the Current Study

The ABC study offers a rich data set. It has followed a large sample of children from birth, offering longitudinal data that incorporates an extensive array of physical, environmental, and psychosocial variables. It also provides the first large sample of SDQ and CRS scores in a NZ context. The current author is unaware of any sample of this size that has monitored activity for a week in a real world setting, and certainly not alongside the psychological questionnaires utilised in the current study. The fact that these questionnaire scores came from multiple informants was extremely valuable. The combination of objective

data and subjective data also permitted and examination of the validity of parental and self-report measures against objective data on sleep duration and activity. The study highlights a number of key environmental and socio-demographic factors that are predictive of later problems, which could be used to inform clinical interviewing. Perhaps the most notable of these are maternal cannabis use, higher levels of maternal stress, and intellectual function.

5.8 Limitations of the Current Study

As was demonstrated by the sensitivity analysis, due to participant attrition, the ABC study has disproportionately lost children who would likely fall in the higher risk groups. As is sadly represented by numerous NZ statistics, this included many children from Maori and Pacific backgrounds. This led to an earlier decision to restrict the study to the children of self-identified European mothers, limiting the generalizability of the findings.

The activity and sleep measures at the ages of 7 and 11 were recorded over different durations (one day/night versus one week), meaning that some caution must be taken in interpreting any of the analyses that make a direct comparison between the two measures. While an activity measure across a week is likely to be robust, a measurement across a day is unlikely to capture the true variability of a child's movement. These concerns are greater for the sleep measures, which is one of the reasons that this thesis was focussed more on activity.

Another issue with this study was the inability to distinguish task-appropriate physical activity, from task-inappropriate activity such as fidgeting. There are of course children who are highly active, but who can moderate their activity when required. It has also been shown that physical exercise can be a protective factor against ADHD (Kamp, Sperlich, & Holmberg, 2014). The inability of the current study to reference activity against environmental expectations may, therefore, be masking the relationship between activity and problematic behaviour. As discovered in the current study, replicating the recent findings of Price et al. (2015), sleep duration appears to be a very weak predictor.

Finally, as is always the case when research questions are formed retrospectively from an existing dataset, the relationships demonstrated do not necessarily imply causation. As acknowledged, the large number of analyses in this thesis increases the chances of Type 1 errors so the correlational analyses in particular should be interpreted with caution; their purpose was however primarily to determine which variables should be taken forward to the modelling stage. While many of the significant relationships in this thesis do only account for a small amount of variance in the outcome measures, they do however highlight a number of interesting future research questions.

5.9 Future Directions

Within the current study it would be very interesting to try and find a way to analyse the data, or collect future data, in a manner that allows periods of task-appropriate and task-inappropriate activity to be distinguished. Perhaps the simplest way of doing this would be to contrast school and home hours, and also to delineate sporting and play activities. This was made difficult in current study, as activity monitoring occurred across different dates for each child. Recording periods also often spanned holiday periods, which themselves varied between schools.

While cell phones and activity bands offer a potential opportunity to assist with such measurements, one important issue is that cell phone accelerometers are multi-axial, whereas that the current accelerometers were unidirectional. While technologically cruder, accelerometers that measure vertical acceleration are a less affected by sources of artefact (with the notable exception of trampolines), such as the forces imparted within a motor vehicle. Thus, a technological exploration of the utility of cell phones and Fitbits as research tools would be useful.

In the absence of significant findings using sleeps duration, it would also be interesting to examine measures of sleep quality, such a latency to fall to sleep, and measures of restlessness. These can be derived from the current data set.

Beyond the current data set, it would be interesting to replicate aspects of this study by contrasting a clinical and a non-clinical population. Increasing the sample size of children with abnormal scores would likely reduce the asymptomatic skew observed when screening measures are employed on a general population. This may provide the statically power to examine whether teacher and parents ratings identify discrete relationships between activity and problem behaviour, as suggested by the current study. It may also permit a more complete examination of the clinical utility of using activity to help discriminate between ADHD subtypes, or indeed whether such discriminations are valid based on activity.

6 References

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Appendices

Appendix A: DSM-V Definition of ADHD

Attention Deficit Hyperactivity Disorder

A. A persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development, as characterized by (1) and/or (2):

1. Inattention: Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities: Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.
    a. Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks or misses details, work is inaccurate).
    b. Often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading).
    c. Often does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction)
    d. Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked).
    e. Often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks; difficulty keeping materials and

belongings in order; messy, disorganized work; has poor time management; fails to meet deadlines).

f. Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers).

g. Often loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones).

h. Is often easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts).

i. Is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults, returning calls, paying bills, keeping appointments).

2. Hyperactivity and impulsivity: Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

   Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or a failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

   a. Often fidgets with or taps hands or feet or squirms in seat.

   b. Often leaves seat in situations when remaining seated is expected (e.g., leaves his or her place in the classroom, in the office or other workplace, or in other situations that require remaining in place).

c. Often runs about or climbs in situations where it is inappropriate. (Note: In adolescents or adults, may be limited to feeling restless.)

d. Often unable to play or engage in leisure activities quietly.

e. Is often "on the go," acting as if "driven by a motor" (e.g., is unable to be or uncomfortable being still for extended time, as in restaurants, meetings; may be experienced by others as being restless or difficult to keep up with).

f. Often talks excessively.

g. Often blurts out an answer before a question has been completed (e.g., completes people's sentences; cannot wait for turn in conversation).

h. Often has difficulty waiting his or her turn (e.g., while waiting in line).

i. Often interrupts or intrudes on others (e.g., butts into conversations, games, or activities; may start using other people's things without asking or receiving permission; for adolescents and adults, may intrude into or take over what others are doing).

B. Several inattentive or hyperactive-impulsive symptoms were present prior to age 12 years.

C. Several inattentive or hyperactive-impulsive symptoms are present in two or more settings (e.g., at home, school, or work; with friends or relatives; in other activities).

D. There is clear evidence that the symptoms interfere with, or reduce the quality of, social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of schizophrenia or another psychotic disorder and are not better explained by another mental disorder

(e.g., mood disorder, anxiety disorder, dissociative disorder, personality disorder, substance intoxication or withdrawal).

Specify whether:

- **314.01 (F90.2) Combined presentation:** If both Criterion A1 (inattention) and Criterion A2 (hyperactivity-impulsivity) are met for the past 6 months.
- **314.00 (F90.0) Predominantly inattentive presentation:** If Criterion A1 (inattention) is met but Criterion A2 (hyperactivity-impulsivity) is not met for the past 6 months.
- **314.01 (F90.1) Predominantly hyperactive/impulsive presentation:** If Criterion A2 (hyperactivity-impulsivity) is met and Criterion A1 (inattention) is not met for the past 6 months.

Specify if:

- In partial remission: When full criteria were previously met, fewer than the full criteria have been met for the past 6 months, and the symptoms still result in impairment in social, academic, or occupational functioning.

Specify current severity:

- Mild: Few, if any, symptoms in excess of those required to make the diagnosis are present, and symptoms result in no more than minor impairments in social or occupational functioning.
- Moderate: Symptoms or functional impairment between “mild” and “severe” are present.

- Severe: Many symptoms in excess of those required to make the diagnosis, or several symptoms that are particularly severe, are present, or the symptoms result in marked impairment in social or occupational functioning.

Other Specified Attention-Deficit/Hyperactivity Disorder 314.01 (F90.8)

This category applies to presentations in which symptoms characteristic of attention-deficit/hyperactivity disorder that cause clinically significant distress or impairment in social, occupational or other important areas of functioning predominate but do not meet the full criteria for attention-deficit/hyperactivity disorder or any of the disorders in the neurodevelopmental disorders diagnostic class. The other specified attention-deficit/hyperactivity disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for attention-deficit/hyperactivity disorder or any specific neurodevelopmental disorder. This is done by recording "other specified attention-deficit/hyperactivity disorder" followed by the specific reason (e.g., "with insufficient inattention symptoms").

Unspecified Attention-Deficit/ Hyperactivity Disorder 314.01 (F90.9)

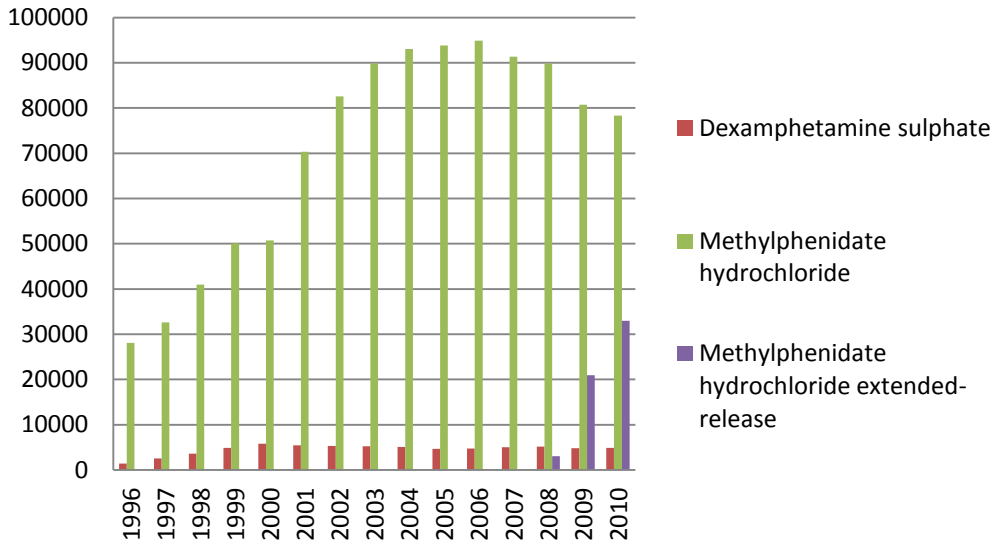
This category applies to presentations in which symptoms characteristic of attention-deficit/hyperactivity disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for attention-deficit/hyperactivity disorder or any of the disorders in the neurodevelopmental disorders diagnostic class. The unspecified attention-deficit/hyperactivity disorder category is used in situations in which the clinician chooses not to specify the reason that the criteria are not met for attention-deficit/hyperactivity disorder or

for a specific neurodevelopmental disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Appendix B: Sex distribution of children in NZ receiving ADHD medication

	Male	Female	Total	% Male	% Female
2006	8590	1790	10380	82.8	17.2
2007	8680	1853	10533	82.4	17.6
2008	9373	2004	11377	82.4	17.6
2009	10558	2417	12975	81.4	18.6
2010	11316	2589	13905	81.4	18.6
Mean				82.1	17.9

Appendix C: Number of individual scripts for three most commonly prescribed ADHD medications in NZ across the period of 1996 to 2010



Appendix D: Conners' subscale scoring criteria

Conners' ADHD Index	Parental Scoring Criteria	Teacher Scoring Criteria
Identifies children at risk for ADHD	• Inattentive, easily distracted • Distractibility or attention span a problem • Gets distracted when given instructions to do something • Avoids, expresses reluctance about, has difficulties engaging in tasks that require sustained mental effort • Fidgets with hands or feet or squirms in seat • Short attention span • Has trouble concentrating in class • Messy or disorganized at home or at school • Only attends if it is something they are very interested in • Does not follow through on instructions and fails to finish schoolwork, chores or duties in the workplace (not due to oppositional behaviour or an inability to understand) • Leaves seat in classroom or in other situations in which remaining seated is expected • Easily frustrated in efforts	• Disturbs other children • Cannot remain still • Excitable or impulsive • Fidgets with hands or feet or squirms in seat • Short attention span • Only attends if it is something they are very interested in • Distractibility or attention span a problem • Fails to finish things they start • Interrupts or intrudes on others • Does not follow through on instructions and fails to finish schoolwork, chores or duties in the workplace (not due to oppositional behaviour or an inability to understand) • Restless, always up and on the go • Inattentive, easily distracted

Conners' DSM Hyperactive/Impulsive		
High scores reflect an above average presentation of DSM-IV diagnostic criteria for Hyperactive/ Impulsive Type ADHD.	• Talks excessively • Has difficulty waiting in lines or awaiting turn in games or group situations • Is always on the go and acts as if driven by a motor • Interrupts or intrudes on others (e.g. butts into others' conversations) • Fidgets with hands or feet or squirms in seat • Has difficulty playing or engaging in leisure activities quietly • Runs about or climbs excessively in situations where it is inappropriate • Leaves seat in classroom or in other situations in which remaining seated is expected • Blurts out answers to questions before the questions have been explained	• Talks excessively • Runs about or climbs excessively in situations where it is inappropriate • Has difficulty playing or engaging in leisure activities quietly • Fidgets with hands or feet or squirms in seat • Blurts out answers to questions before the questions have been explained • Is always on the go and acts as if driven by a motor • Interrupts or intrudes on others' (e.g. butts into • others conversations) • Leaves seat in classroom or in other situations in which remaining seated is expected • Has difficulty waiting turn

Conners' DSM Inattentive		
High scores reflect an above average presentation of DSM-IV diagnostic criteria for Inattentive Type ADHD	• Fails to give close attention to details and makes careless mistakes • Forgetful in daily activities • Avoids, expresses reluctance about or has difficulties engaging in tasks that require mental effort • Has difficulty sustaining attention in tasks or play activities • Does not seem to listen to what is being said to them • Loses things necessary for tasks or activities • Does not follow through on instructions and fails to finish schoolwork, chores or duties in the workplace (not due to oppositional behaviour or failure to understand instructions) • Has difficulty organising tasks and activities • Easily distracted by extraneous stimuli	• Forgets things they have already learned • Fails to give close attention to details and makes careless mistakes • Avoids, expresses reluctance about or has difficulties engaging in tasks that require mental effort • Loses things necessary for tasks or activities • Does not seem to listen to what is being said to them • Does not follow through on instructions and fails to finish schoolwork, chores or duties in the workplace (not due to oppositional behaviour or failure to understand instructions) • Easily distracted by extraneous stimuli • Has difficulty organising tasks and activities • Has difficulty sustaining attention in tasks or play activities
Conners' DSM Combined		
High scores reflect an above average presentation of DSM-IV diagnostic criteria for Combined Type ADHD	• A combination of the DSM Hyperactive/Impulsive and Inattentive Parental criteria above.	• A combination of DSM Hyperactive/Impulsive and Inattentive Teacher criteria above

Appendix E: Parental SDQ Subscales and Item Definitions

Subscale	Items
Emotional Problems	Often complains of headaches, stomach-aches or sickness
	Many fears, easily scared
	Nervous or clingy in new situations, easily loses confidence
	Often unhappy, downhearted or tearful
	Many worries, often seems worried
Conduct Problems	Can be spiteful to others
	Often argumentative with adults
	Often fights with other children or bullies them
	Often has temper tantrums, or hot tempers
	Generally obedient, usually does what adults request
Hyperactivity	Sees tasks through to the end, good attention span
	Can stop and think things out before acting
	Easily distracted, concentration wanders
	Constantly fidgeting or squirming
	Restless, overactive, cannot sit still for long
Peer Problems	Gets on better with adults than with other children
	Has at least one good friend
	Rather solitary, tends to play alone
	Picked on or bullied by other children
	Generally liked by other children
Prosocial Behaviour	Helpful if someone is hurt, upset or feeling ill
	Considerate of other peoples' feelings
	Shares readily with other children
	Kind with younger children
	Often volunteers to help others

Appendix F: Child SDQ Subscales and Item Definitions

Subscale	Items
Emotional Problems	I get a lot of headaches, stomach-aches or sickness
	I worry a lot
	I am often unhappy, downhearted or tearful
	I am nervous in new situations
	I have many fears, I am easily scared
Conduct Problems	I get very angry and often lose my temper
	I usually do as I am told
	I fight a lot
	I am often accused of lying or cheating
	I take things that are not mine
Hyperactivity	I am restless, I cannot stat still for long
	I am constantly fidgeting or squirming
	I am easily distractedI think before I do things
	I finish the work I am doing
Peer Problems	I am usually on my own
	I have one good friend or more
	Other people my age generally like me
	Other children or young people pick on me
	I get on better with adults than with people my age
Prosocial Behaviour	I usually share with others
	I try to be nice to other people
	I am helpful if someone is hurt, upset or feeling ill
	I am kind to younger children
	I often volunteer to help others.

Appendix G: Defining 'Caseness' From Parental SDQ Symptom Scores

Subscale	Normal	Borderline	Abnormal
Total Difficulties Score	0 – 13	14 – 16	17 - 40
Emotional Symptoms Score	0 – 3	4	5 - 10
Conduct Problems Score	0 – 2	3	4 – 10
Hyperactivity Score	0 – 5	6	7 – 10
Peer Problems Score	0 – 2	3	4 – 10
Prosocial Score	6 – 10	5	0 – 4

Appendix H: Defining 'Caseness' From Child SDQ Symptom Scores

Subscale	Normal	Borderline	Abnormal
Total Difficulties Score	0 – 15	16 – 19	20 - 40
Emotional Symptoms Score	0 – 5	6	7 - 10
Conduct Problems Score	0 – 3	4	5 – 10
Hyperactivity Score	0 – 5	6	7 – 10
Peer Problems Score	0 – 3	4-5	6 – 10
Prosocial Score	6 – 10	5	0 – 4

Appendix I: Relationship between activity and sleep and Externalising/Internalising coding of Child SDQ Difficulties scores (Continuous)

Externalising	**B (SE)**	**β**	**Lower**	**Upper**	**t**	**p**
Average Counts at 7	.001 (.001)	.090	.000	.002	1.972	**.049***
Percent Sed at 7	-.030 (.017)	-.080	-.064	.003	-1.770	.077
Percent Mod at 7	.029 (.022)	.061	-.013	.072	1.353	.177
Percent Vig at 7	.076 (.043)	.080	-.009	.161	1.765	.078
Av Time Sed at 7	-.002 (.002)	-.051	-.005	.001	-1.113	.266
Av Time Mod at 7	.003 (.003)	.059	-.002	.008	1.290	.198
Av Time Vig at 7	.010 (.005)	.082	-.001	.020	1.799	.073
Sleep duration at 7	-.250 (.174)	-.065	-.592	.093	-1.432	.153
Average Counts at 11	.000 (.001)	.021	-.001	.002	.493	.622
Percent Sed at 11	-.016 (.029)	-.024	-.073	.041	-.550	.582
Percent Mod at 11	.029 (.034)	.037	-.038	.096	.859	.391
Percent Vig at 11	-.109 (.117)	-.040	-.339	.121	-.930	.353
Av Time Sed at 11	.000 (.002)	-.004	-.005	.005	-.099	.921
Av Time Mod at 11	.003 (.004)	.032	-.005	.011	.744	.457
Av Time Vig at 11	-.015 (.015)	-.043	-.043	.014	-1.003	.316
Sleep duration at 11	-.321 (.185)	-.074	-.684	.042	-1.739	.083
Internalising						
Average Counts at 7	.000 (.001)	-.012	-.001	.001	-2.68	.789
Percent Sed at 7	-.005 (.015)	-.013	-.035	.026	-.294	.769
Percent Mod at 7	.011 (.019)	.026	-.027	.049	.566	.572
Percent Vig at 7	-.015 (.039)	-.017	-.091	.061	-.385	.700
Av Time Sed at 7	1.785^{-5} (.002)	.001	-.003	.003	.012	.991
Av Time Mod at 7	.001 (.002)	.014	-.004	.005	.307	.759
Av Time Vig at 7	-.002 (.005)	-.020	-.012	.007	-.447	.655
Sleep duration at 7	-.094 (.156)	-.028	-.400	.212	-.601	.548
Average Counts at 11	-.001 (.001)	-.036	-.002	.001	-.827	.409
Percent Sed at 11	.037 (.025)	.063	-.013	.087	1.459	.145
Percent Mod at 11	-.037 (.030)	-.053	-.096	.022	-1.225	.221
Percent Vig at 11	-.177 (.101)	-.075	-.376	.022	-1.745	.082
Av Time Sed at 11	.005 (.002)	.091	.000	.009	2.112	**.035***
Av Time Mod at 11	-.003 (.004)	-.032	-.010	.004	-.751	.453
Av Time Vig at 11	-.021 (.012)	-.072	-.045	.004	-1.674	.095
Sleep duration at 11	-.381 (.164)	-.099	-.70 2	-.059	-2.327	**.020***

Appendix J: Relationship between activity and sleep and the Parental SDQ Subscale Scores (Categorical)

Hyperactivity	OR	Lower	Upper	p
Average Counts at 7	1.001	1.000	1.002	.252
Percent Sed at 7	.973	.941	1.007	.120
Percent Mod at 7	1.034	.990	1.080	.132
Percent Vig at 7	1.039	.957	1.128	.361
Av Time Sed at 7	.998	.995	1.001	.261
Av Time Mod at 7	1.003	.998	1.008	.223
Av Time Vig at 7	1.004	.994	1.015	.423
Sleep Duration at 7	.865	.617	1.213	.400
Average Counts at 11	1.001	1.000	1.003	.019*
Percent Sed at 11	.957	.907	1.011	.116
Percent Mod at 11	1.033	.968	1.102	.327
Percent Vig at 11	1.273	1.059	1.529	.010**
Av Time Sed at 11	.999	.994	1.004	.609
Av Time Mod at 11	1.005	.997	1.012	.254
Av Time Vig at 11	1.032	1.009	1.055	.007**
Sleep Duration at 11	.839	.594	1.184	.317
Conduct				
Average Counts at 7	1.000	1.000	1.000	.410
Percent Sed at 7	.968	.941	.995	.021*
Percent Mod at 7	1.055	1.018	1.093	.004**
Percent Vig at 7	1.000	.933	1.071	.989
Av Time Sed at 7	.999	.996	1.001	.296
Av Time Mod at 7	1.007	1.002	1.011	.003**
Av Time Vig at 7	1.000	.992	1.009	.949
Sleep Duration at 7	.800	.608	1.054	.113
Average Counts at 11	1.001	1.000	1.002	.207
Percent Sed at 11	.971	.928	1.016	.207
Percent Mod at 11	1.036	.982	1.093	.195
Percent Vig at 11	1.033	.865	1.234	.718
Av Time Sed at 11	.999	.995	1.003	.732
Av Time Mod at 11	1.004	.998	1.011	.182
Av Time Vig at 11	1.003	.982	1.026	.763
Sleep Duration at 11	.939	.700	1.258	.672
Emotion				
Average Counts at 7	1.000	.999	1.001	.532
Percent Sed at 7	1.012	.984	1.041	.411
Percent Mod at 7	.989	.954	1.025	.553
Percent Vig at 7	.967	.897	.1042	.374
Av Time Sed at 7	1.002	.999	1.005	.158
Av Time Mod at 7	1.000	.995	1.004	.866
Av Time Vig at 7	.996	.987	1.006	.446
Sleep Duration at 7	.813	.613	1.077	.149
Average Counts at 11	.999	.998	1.000	.187
Percent Sed at 11	1.071	1.018	1.127	.008**
Percent Mod at 11	.925	.871	.981	.010*
Percent Vig at 11	.799	.634	1.007	.057
Av Time Sed at 11	1.004	1.000	1.008	.051
Av Time Mod at 11	.992	.985	.999	.028*
Av Time Vig at 11	.974	.947	1.002	.070
Sleep Duration at 11	.848	.629	1.144	.280

Peer	**OR**	**Lower**	**Upper**	**p**
Average Counts at 7	1.000	.999	1.001	.681
Percent Sed at 7	.987	.958	1.017	.379
Percent Mod at 7	1.019	.981	1.058	.334
Percent Vig at 7	1.011	.939	1.089	.768
Av Time Sed at 7	.999	.996	1.002	.390
Av Time Mod at 7	1.002	.997	1.006	.415
Av Time Vig at 7	1.002	.992	1.011	.747
Sleep Duration at 7	1.010	.746	1.367	.948
Average Counts at 11	1.001	1.000	1.002	.229
Percent Sed at 11	.986	.940	1.035	.568
Percent Mod at 11	1.017	.961	1.076	.568
Percent Vig at 11	.994	.820	1.206	.954
Av Time Sed at 11	.999	.994	1.003	.483
Av Time Mod at 11	1.002	.995	1.009	.532
Av Time Vig at 11	1.000	.976	1.024	.974
Sleep Duration at 11	.994	.729	1.355	.969
Prosocial				
Average Counts at 7	1.001	.999	1.002	.406
Percent Sed at 7	.988	.951	1.026	.521
Percent Mod at 7	1.006	.959	1.055	.818
Percent Vig at 7	1.054	.964	1.152	.247
Av Time Sed at 7	1.000	.996	1.004	.930
Av Time Mod at 7	1.001	.996	1.007	.663
Av Time Vig at 7	1.007	.996	1.018	.194
Sleep Duration at 7	.806	.554	1.173	.259
Average Counts at 11	1.001	1.000	1.003	.112
Percent Sed at 11	.950	.894	1.009	.096
Percent Mod at 11	1.058	.985	1.138	.123
Percent Vig at 11	1.137	.914	1.414	.250
Av Time Sed at 11	.996	.991	1.001	.127
Av Time Mod at 11	1.005	.997	1.014	.243
Av Time Vig at 11	1.014	.987	1.042	.300
Sleep Duration at 11	1.461	.956	2.235	.080

Appendix K: Independent Samples T-Tests of significant relationships between activity and sleep and the Parental SDQ Difficulties Subscale Scores (Categorical)

	Normal Mean (SD)	Bord/Abnor Mean (SD)	Mean Diff	Lower	Upper	t	p
Hyperactivity							
Av Counts at 11	537.82 (176.50)	598.96 (259.18)	-61.13	-128.76	-6.50	-1.803	.076 [1]
Percent Vig at 11	1.35 (1.10)	1.78 (1.78)	-.43	-.89	.04	-1.842	.070 [1]
Av Time Vig at 11	10.77 (8.78)	14.43 (14.88)	-3.66	-7.52	.20	-1.895	.062 [1]
Conduct							
Percent Sed at 7	67.82 (8.30)	65.53 (8.59)	2.29	.35	4.22	2.325	**.021***
Percent Mod at 7	27.09 (6.57)	29.39 (6.63)	-2.29	-3.82	-.77	-2.959	**.003****
Av Time Mod at 7	215.71 (55.67)	235.59 (53.01)	-19.88	-32.66	-7.10	-3.55	**.002****
Emotion							
Percent Sed at 11	85.29 (4.73)	86.76 (4.78)	-1.47	-2.56	-.39	-2.667	**.008****
Percent Mod at 11	13.13 (3.96)	11.90 (4.30)	1.23	.31	2.14	2.620	**.009****
Av Time Mod at 11	104.36 (32.81)	95.67 (37.60)	8.69	.99	16.39	2.218	**.027***

Note: Equal variances assumed, unless Levene's is significant [1]

Appendix L Relationship between activity and sleep and the Conners' Teachers DSM Subscale T-Scores (Continuous)

Hyperactive/Impulsive	**B (SE)**	**β**	**Lower**	**Upper**	**t**	**p**
Average Counts at 7	.000 (.001)	.012	-.002	.002	.254	.800
Percent Sed at 7	-.032 (.032)	-.047	-.096	.031	-1.007	.314
Percent Mod at 7	.060 (.040)	.069	-.019	.139	1.493	.136
Percent Vig at 7	-.039 (.083)	-.022	-.201	.123	-.474	.636
Av Time Sed at 7	-.003 (.003)	-.044	-.009	.003	-.954	.341
Av Time Mod at 7	.005 (.005)	.050	-.004	.015	1.092	.276
Av Time Vig at 7	-.004 (.010)	-.020	-.025	.016	-.430	.668
Sleep duration at 7	-.274 (.324)	-.039	-.911	.363	-.845	.399
Average Counts at 11	.000 (.002)	.014	-.002	.003	.311	.756
Percent Sed at 11	.024 (.056)	.019	-.086	.135	.434	.665
Percent Mod at 11	-.041 (.066)	-.027	-.172	.089	-.626	.532
Percent Vig at 11	.011 (.226)	.002	-.432	.454	.049	.961
Av Time Sed at 11	.002 (.005)	.018	-.007	.011	.423	.673
Av Time Mod at 11	-.004 (.008)	-.024	-.020	.011	-.547	.585
Av Time Vig at 11	.006 (.028)	.009	-.049	.060	.198	.843
Sleep duration at 11	-.608 (.365)	-.072	-1.326	.110	-1.664	.097
Inattentive						
Average Counts at 7	-4.007^{-7} (.000)	-.019	.000	.000	-.398	.691
Percent Sed at 7	-.012 (.025)	-.023	-.061	.036	-.501	.617
Percent Mod at 7	.041 (.031)	.061	-.020	.101	1.321	.187
Percent Vig at 7	-.090 (.063)	-.066	-.214	.034	-1.425	.155
Av Time Sed at 7	-.001 (.002)	-.018	-.006	.004	-.377	.707
Av Time Mod at 7	.004 (.004)	.052	-.003	.011	1.110	.268
Av Time Vig at 7	-.011 (.008)	-.064	-.026	.005	-1.382	.168
Sleep duration at 7						
Average Counts at 11	-.002 (.001)	-.064	-.004	.001	-1.451	.147
Percent Sed at 11	.090 (.042)	.094	.008	.173	2.147	**.032***
Percent Mod at 11	-.106 (.050)	-.094	-.204	-.009	-2.147	**.032***
Percent Vig at 11	-.244 (.168)	-.064	-.575	.086	-1.451	.147
Av Time Sed at 11	.004 (.004)	.053	-.003	.011	1.210	.227
Av Time Mod at 11	-.013 (.006)	-.092	-.024	-.001	-2.116	**.035***
Av Time Vig at 11	-.028 (.021)	-.060	-.069	.013	-1.359	.175
Sleep duration at 11	-.931 (.454)	-.089	-1.823	-.039	-2.051	**.041***
Combined						
Average Counts at 7	.000 (.001)	-.015	-.002	.001	-.330	.742
Percent Sed at 7	-.017 (.026)	-.031	-.068	.034	-.669	.504
Percent Mod at 7	.046 (.032)	.066	-.017	.110	1.439	.151
Percent Vig at 7	-.081 (.066)	-.057	-.211	.049	-1.229	.220
Av Time Sed at 7	-.001 (.003)	-.025	-.006	.004	-.550	.583
Av Time Mod at 7	.004 (.004)	.053	-.003	.012	1.145	.253
Av Time Vig at 7	-.010 (.008)	-.054	-.026	.007	-1.163	.246
Sleep duration at 7	-.364 (.260)	-.065	-.875	.147	-1.400	.162
Average Counts at 11	-.001 (.001)	-.034	-.003	.001	-.790	.430
Percent Sed at 11	.072 (.045)	.070	-.016	.160	1.606	.109
Percent Mod at 11	-.090 (.053)	-.075	-.194	.013	-1.719	.086
Percent Vig at 11	-.151 (.179)	-.037	-.503	.201	-.843	.400
Av Time Sed at 11	.003 (.004)	.035	-.004	.010	.801	.424
Av Time Mod at 11	-.011 (.006)	-.073	-.023	.002	-1.688	.092
Av Time Vig at 11	-.016 (.022)	-.032	-.060	.028	-.723	.470
Sleep duration at 11	-.204 (.291)	-.030	-.776	.368	-.701	.484

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