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ADHD symptons and academic performance in adolescents

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ADHD symptoms and academic performance in adolescents

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Contents

List of Abbreviations

AARS = Adult ADHD Rating Scale

ADD = Attention Deficit Disorder

ADHD = Attention Deficit Hyperactivity Disorder

ANOVA = Analysis of Variance

APA = American Psychiatric Association

AQ = Aggression Questionnaire

BPS = British Psychological Society

CD = Conduct Disorder

CDT = Choice Delay Task

CFA = Confirmatory Factor Analysis

DA = Delay Aversion

DSM = Diagnostic and Statistical Manual

EF = Executive functioning

EFs = Executive functions

EFA = Exploratory Factor Analysis

GCSE = General Certificate of Secondary Education

HADS = Hospital Anxiety and Depression Scale

ISM = Inventory of School Motivation

MTA = The National Institute of Mental Health Collaborative Multisite Multimodal Treatment Study of Children with Attention-Deficit/Hyperactivity Disorder

ODD = Oppositional Defiant Disorder

RSPM = Raven's Standard Progressive Matrices

SES = Socio-Economic Status

SSP = Stop Signal Paradigm

SSRT = Stop signal Reaction Time

SST = Stop Signal Task

WURS = Wender Utah Rating Scale

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Summary

Most research into attention deficit hyperactivity disorder (ADHD) is concentrated on childhood cases, with less of a focus on adolescents and adults (Willoughby, 2003). Individuals with ADHD are likely to experience academic impairment (Barbaresi, Katusic, Colligan, Weaver, & Jacobsen, 2007; Biederman *et al.*, 1996; Loe & Feldman, 2007), however this has mostly been demonstrated in clinic-based childhood studies; non-clinical, adolescent-specific studies are few and far between (Loe & Feldman, 2007). This thesis examines community-based adolescent ADHD symptoms and their influence over academic attainment.

The introduction to the thesis provides a background to ADHD and an overview of the literature on influences over academic performance. Chapter 2 consists of a review of the literature on the relationship between ADHD and academic performance, and explores the reasons why ADHD individuals are at an academic disadvantage. Chapter 3 outlines methodological process of the empirical studies of the thesis, which can be found in chapters 4, 5, and 6. Chapter 4 is an investigation of the position of ADHD symptoms in the structure of adolescent mental health problems. In Chapter 5, the impact of ADHD symptoms over academic attainment is studied. Chapter 6 examines the role of underlying neuropsychological processes in the relationship between ADHD and academic attainment. Finally, the discussion summarises the findings of each paper and assesses the limitations and implications of the thesis.

The core findings are as follows. In Chapter 4, exploratory factor analysis demonstrated that community-based adolescent ADHD symptoms form a valid dimension that is distinct from symptoms of comorbid syndromes. This provides support for the notion that the adolescent ADHD construct is a continuum-based

psychopathology. In Chapter 5 regression analysis revealed that ADHD symptoms exerted almost as much influence over academic attainment as general cognitive ability and school-oriented motivation, and far more influence than other forms of psychopathology. Finally, in Chapter 6, it was found that delay aversion and deficient inhibitory control did not mediate the relationship between ADHD symptoms and academic performance. However it should be noted that in the final study, the sample was severely underpowered due to constraints placed on recruitment by the consent process.

Overall the results suggest that ADHD symptoms pose a significant academic risk for adolescents, even at sub-clinical levels. Efforts should be made to increase awareness among teachers and carers of the potential plight of adolescents who express symptoms of ADHD.

Chapter 1

General Introduction

The introduction focuses on the key aspects of this thesis: ADHD and academic performance. Firstly, an outline of ADHD is provided, which includes information on classification, prevalence, comorbidity, aetiology, theories, and associated impairments. Secondly, influences on academic performance are discussed; this includes factors that are investigated in this thesis (aside from ADHD), together with other factors that have also been shown to be influential. Finally, an introduction to the structure of the thesis is given, which includes a summary of the literature review chapter, and the objectives of the three empirical chapters.

Attention deficit hyperactivity disorder

Attention deficit hyperactivity disorder (ADHD) is a highly prevalent and heterogeneous developmental disorder. It is characterised by developmentally inappropriate levels of inattention and/or hyperactivity and impulsivity (DSM-IV; American Psychiatric Association [APA], 1994). ADHD is typically associated with impairment across several domains of functioning (Bauermeister *et al.*, 2007; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Nijmeijer *et al.*, 2008).

Classification and symptom structure

What is now known as ADHD has been conceptualised differently over the years; other conceptualisations have included *hyperkinetic syndrome*, *attention deficit disorder (ADD)*, *ADD with hyperactivity* (DSM-III-R; APA, 1987), and *hyperkinesis* (ICD-10; World Health Organisation, 1992); as research into the disorder advances, the working definitions subsequently change. The most widely used taxonomic tool is the American Psychiatric Association's Diagnostic and Statistical Manual (DSM),

whose most recent edition (4th edition) cites the syndrome named Attention Deficit/Hyperactivity Disorder (ADHD). DSM-IV criteria recognise three subtypes of the disorder: (a) predominantly inattentive type, (b) predominantly hyperactive type, and (c) combined type. A diagnosis of ADHD combined-type (DSM-IV; APA, 1994) (the most common diagnosis) requires a minimum of six out of nine inattentive symptoms, and a minimum of six out of nine hyperactive/impulsive symptoms. Symptoms must be present in two or more settings (for example at school and at home), and must significantly impair everyday functioning (APA, 1994). Symptoms of inattention include daydreaming, distractibility, and disorganisation; symptoms of hyperactivity/impulsivity include restlessness, fidgeting, and impatience (APA, 1994).

Prevalence and developmental span

The Worldwide Prevalence of ADHD study (Faraone, Sergeant, Gillberg, & Biederman, 2003) concludes that 5-10% of children are affected by ADHD, however this conceals a wide variation of findings. For example, studies by Costello and colleagues (2003) and Ford and colleagues (2003) report lower rates of between 1% and 2.5 %. It has been suggested that these discrepancies could be due to different criteria used to define a 'case' of ADHD; for example some studies will look only at symptom levels, while others will also take account of impairment (NICE ADHD Guideline: National Collaborating Centre for Mental Health, 2009). It is estimated that approximately 40% of childhood cases will continue to meet diagnostic criteria in adulthood (Fischer et al., 1993). Until recently, the commonly held viewpoint was that ADHD exists in school-age children only (Willoughby, 2003), however research has shown that ADHD has an early, pre-school onset (Daley, Jones, Hutchings & Thompson, 2009; Lavigne *et al.*, 1996), which persists through adolescence (Faigel, *et*

al., 1997; Wolraich *et al.,* 2005), into adulthood (Barkely, Fischer, Smallish, & Fletcher, 2002; Mannuzza *et al.,* 1993).

Comorbidity

A key clinical feature of ADHD is comorbidity. In childhood cases, comorbid diagnoses typically include conduct disorder, oppositional defiant disorder, depression, anxiety, and learning disorder (Bird, Gould, & Staghezza, 1993; Elia, Ambrosini, & Berrettini, 2008; Pliszka, 1998). In adult cases, comorbid conditions include mood disorders, antisocial behaviour disorders, and substance use disorders (Biederman *et al.,* 1993).

Aetiology

(a) Genetics

ADHD is a highly heritable disorder (Thapar, Holmes, Poulton, & Harrington, 1999). The genetic aetiology of ADHD has been demonstrated in many family, twin, and adoption studies. Parents and siblings of children with ADHD have been found to have a 2- to 8-fold increased risk for ADHD (Biederman, 2005). In a review of several twin studies, Biederman (2005) calculated a mean heritability rate of .77, suggesting that it is highly likely that the twin of an ADHD child will also have the disorder. Non-biological relatives of adopted ADHD children have been shown to have lower rates of ADHD than biological relatives of non-adopted ADHD children, and similar rates to the relatives of controls (Sprich *et al.,* 2000). Molecular genetic studies have established an association with a single dopamine transporter gene (Cook *et al.,* 1995), specifically implicating the 7-repeat allele of the human dopamine receptor D4 gene (Brookes *et al.,* 2006; Faraone, Doyle, Mick, & Biederman, 2001).

(b) Environmental and biological risk factors

Genetically predisposed individuals will not necessarily develop ADHD; it is thought that the disorder could result from a gene-environment interaction (Larsson, Larsson, & Lichtenstein, 2004), or perhaps a gene-environment correlation (Daley, Sonuga-Barke, Thompson, & Chen, 2008). For example, a key environmental risk factor is parenting; chaotic parenting is likely to bring about ADHD in genetically predisposed individuals (Johnston & Mash, 2001). It could be that this example is a gene-environment interaction, where individuals' genetic predispositions are expressed differently in different environments (Tsuang, Bar, Stone, & Faraone, 2004); here, the particular parenting environment accentuates the expression of ADHD. However, it could also be argued that the example above is actually a gene-environment correlation, where individuals experience environments that are correlated with their genotype (Jaffee & Price, 2007); the ADHD genome that is shared between parent and child helps to bring about the chaotic parenting style, which in turn stimulates the development of ADHD in the child. Further support of the importance of parenting comes from intervention studies, where improvements in ADHD symptoms have come about when parents are taught alternative parenting skills (Jones, Daley, Hutchings, Bywater, & Eames, 2008; Sonuga-Barke, Daley, Thompson, Weeks, & Laver-Bradbury, 2001). Other family-environment risk factors have been identified, including chronic family conflict, decreased family cohesion, and exposure to parental psychopathology, particularly ADHD (Biederman *et al.*, 1995b).

Biological risk factors include maternal smoking during pregnancy (Mick, Biederman, Faraone, Sayer, & Kleinman, 2002), obstetric complications (Sprich-Buckminster, Biederman, Milberger, Faraone, & Lehman, 1993), and diet (Schnoll, Burshteyn, & Cea-Aravena, 2003); however investigations into the impact of diet

have produced mixed results (Bateman *et al.*, 2004; McCann *et al.*, 2007; Schnoll, Burshteyn, & Cea-Aravena 2003).

Neuropsychological theories

Neuro-imaging studies have shown that ADHD individuals have a decreased size of the prefrontal cortex (Hill *et al.*, 2002; Mostofsky, Cooper, Kates, Denckla, & Kaufmann, 2002). Therefore there are expected deficits in certain prefrontal executive functions, such as response inhibition (Barkley, 1997) and working memory (Tannock, 1998). For many years it was accepted that ADHD symptoms arise from these prefrontal deficits, with impaired forethought, planning and control giving rise to the ADHD individuals' behaviour (Schachar, Mota, Logan, Tannock, & Klim, 2000). Subscribers to this theoretical perspective have identified a lack of inhibitory control as the key deficit (Barkley, 1997): they posit that ADHD is a disorder of dysregulation of thought and action associated with poor inhibitory control (Schachar *et al.*, 2000).

In contrast to the cognitive dysregulation theory, an alternative viewpoint has been proposed. The delay aversion hypothesis (Sonuga-Barke, Williams, Hall, & Saxton, 1996) suggests that ADHD is the manifestation of a motivational style associated with altered reward mechanisms, whereby ADHD symptoms are the functional expression of the child's attempts to avoid delay (Nigg, 2001; Sagvolden, Aase, Zeiner, & Berger, 1998; Sonuga-Barke *et al.*, 1996). When in control of their environment, the child will choose to minimize delay by acting impulsively; when not in control of their environment, the child will choose to distract themselves from the passing of time, by expressing inattentive and hyperactive behaviours (Daley, 2006).

It has since become apparent that it is not feasible to search for one unifying theory of ADHD; cognitive dysregulation and delay aversion can be merged to help explain ADHD. Solanto *et al.* (2001) examined the relative strength of the two theoretical camps as predictors of ADHD. It was found that cognitive dysregulation and delay aversion were not correlated with each other, however they were both highly diagnostic of ADHD cases. This finding led to the development of the dual pathway model of ADHD (Sonuga-Barke, 2002), which posits that there are two possible routes between biology and behaviour: cognitive dysregulation and motivational style.

Associated impairments

As well as being at risk for comorbid psychiatric conditions, ADHD individuals are at risk of impairment in other, age specific domains. Pre-schoolers who express ADHD symptoms are likely to experience difficulties with pre-academic skills (Mariani & Barkley, 1997) and social functioning (Spira & Fischel, 2005); ADHD children often struggle with schoolwork (Barry, Lyman, & Klinger, 2002) and social interaction (Nijmeijer *et al.*, 2008), experience physical injury (DuPaul, McGoey, Eckert, & Vanbrakle, 2001) and sleep disturbances (Corkum *et al.*, 1999); adolescents with ADHD are also likely to experience academic (Frazier, Youngstrom, Glutting, & Watkins, 2007), and social problems (Greene, Biederman, Faraone, Sienna, & Garcia-Jetton, 1997), and are at increased risk for substance abuse (Elkins, McGue, & Iacono, 2007); as adults, ADHD individuals have been shown to experience academic

(Heiligenstein, Guenther, & Levy, 1999), occupational (Mannuzza *et al.*, 1993), social (Sobanski *et al.*, 2008), and substance abuse (Wilens, 2004) problems.

Along with impaired social functioning, a common denominator at each developmental stage is impaired academic functioning. This impairment has been demonstrated in clinical (Barry, Lyman, & Klinger, 2002) and community (Merrel & Tymms, 2001) samples. Whilst the childhood literature is substantial, there has been less focus on older cases. The focus of this PhD thesis is to examine ADHD symptoms and academic performance in adolescents.

Academic performance

Research has shown that a great number of factors can be attributed to individual differences in academic performance, aside from ADHD symptomatology. These include cognitive ability, motivation, other forms of psychopathology, personality, and various social and societal factors.

Early beginnings

For over a century, researchers have been interested in predicting academic performance. The *raison d'etre* of the first intelligence tests was to predict academic outcomes (Binet, 1903). This early work sparked an interest in developing cognitive tests throughout the 20th Century (Raven, Raven, & Court, 2000; Wechsler, 1981), with educational outcomes being the primary outcome measure. Spearman (1904) suggested that the correlation between the latent trait from school examination

performance would be almost perfectly correlated with the latent trait from intelligence tests.

Cognitive ability

As predicted by early work (Binet, 1903; Spearman, 1904) the strongest indicator of academic success is cognitive ability (Neisser *et al.,* 1996), with many empirical studies demonstrating this relationship. For example, in a meta-analysis of over 3000 studies, Walberg (1984) found that the correlation between cognitive ability and academic performance in school children was .7. More recently, Deary, Strand, Smith and Fernandes (2007) studied the relationship between cognitive ability at age 11 and GCSE (General Certificate of Secondary Education) achievement at age 16, in a sample of 74,403 British children. A correlation coefficient of .81 was established between cognitive ability and academic performance, suggesting that general mental ability is an extremely strong predictor of educational achievement. This supported Petrides, Chamorro-Premuzic, Frederickson, and Furnham (2005), who, in a sample of 901 British adolescents, found that verbal ability (a good proxy for cognitive ability) was a powerful predictor of GCSE performance.

Although cognitive ability is the strongest predictor of academic performance, there are other, non-cognitive factors that contribute (Petrides *et al.,* 2005), such as motivation, psychopathology, personality, and social/societal factors.

Motivation

The relationship between motivation and achievement is well documented (Meece, Anderman, & Anderman, 2006; Walberg, 1984). For example, Gilman and Anderman (2006) compared adolescents who fell into high-, medium- and low-motivation groups on various outcome measures. It was found that the high-motivation group achieved significantly better academic grades than the other two groups, whilst also scoring high on measures of self-esteem, global satisfaction and family satisfaction, and low on measures of depression and anxiety. Boon (2007) compared high- and low-achieving groups of adolescents, finding that the high achieving group was more motivated, expressing a greater desire to learn. Interestingly, Gagne and St Pere (2001) found that perhaps motivation is not the most important academic predictor. They examined the predictive strength of motivation and cognitive ability over academic performance in high school pupils, and found that cognitive ability was by far the best predictor whilst self-rated motivation was not related to academic achievement; however, it should be noted that the results of Gagne and St Pere were established in an all-girl, private school sample.

Psychopathology

As well as ADHD, there are other psychopathological influences on academic performance. Frojd *et al.* (2008) studied depressive symptoms and school performance in 2,329 Finnish adolescents, and found that self-reported depression was associated with poor academic performance, and difficulties in concentration and social relationships. In a study of the effects of depression and anxiety on university

performance, Andrews and Wilding (2004) found that exam performance was aversely affected by the presence of depression, however symptoms of anxiety did not have an impact. The authors conclude that anxiety could induce worry, which could in-turn increase productivity. However, elsewhere, significant anxiety effects have been established. In one community-based study of 8-16 year olds (Mazzone *et al.,* 2007), significant relationships were established between self-reported anxiety symptoms and poor academic performance. It was found that high levels of anxiety predicted poor performance, however as a whole, poor performing participants were not more anxious than high performing participants, suggesting that anxiety only interferes with school functioning at extreme levels.

There has been a small amount of research focusing on the effects of aggression on academic performance. Loveland, Lounsbury, Welsh, and Buboltz (2007) found a negative relationship between adolescent aggression and academic attainment, with a correlation coefficient of -.4 established between self-reported aggression and grade-point averages. In another adolescent study, Schwartz, Gorman, Nakamoto, and McKay (2006) studied the role of popularity in the relationship between aggression and academic performance. It was found that at high levels of aggression, more popular individuals were more likely to experience declines in academic attainment, and at low levels of aggression, popularity was not linked to academic decline; i.e. popularity was related to poor academic performance only in aggressive individuals.

Personality

The role of personality in scholastic achievement has long aroused interest, especially since the conception of personality models, such as the Eysenckian model of personality (Eysenck, 1947), which proposed 3 dimensions of *psychoticism, extraversion,* and *neuroticism*, and the "Big Five" model (Digman, 1990; McCrae & Costa, 1997), which posits five dimensions of *extraversion*, *neuroticism*, *agreeableness*, *conscientiousness*, and *open-ness-to-experience.* Research into the nature of *extroversion (*and indeed *introversion)*, suggests that *introverts* are more adept at boring tasks, whilst *extroverts* perform better at interesting tasks (Eysenck & Eysenck, 1985). Building on this work, there has been a significant body of research into the relationship between personality types and academic outcomes. De Raad and Schouwenburg (1996) suggest that *conscientiousness* underlies academic performance as it encompasses elements such as organisation, drive, carefulness, concentration, and endurance. In a study of 115 Australian 14-16 year-olds, Heaven, Mak, Barry, and Ciarrochi (2002) found that low *psychoticism* and high *conscientiousness* were strongly linked with good attitudes to school, and high academic performance. In their study, Petrides *et al.* (2005) also found that *psychoticism* and *extraversion* had a negative impact on academic performance.

Social and societal factors

Aside from the psychological and cognitive factors discussed above, there are other factors that have been shown to play a significant role in academic performance, such as family environment. Parental monitoring, warmth, and fairness, are family

characteristics that are conducive to high academic achievement (Steinberg, 1996). Children with parents who are supportive and actively interested in their schoolwork are more likely to achieve higher at school, and possess greater beliefs in their academic ability (Juang & Silbereisen, 2002). However, children with parents who do not take an interest in their academic activities are less likely to perform well at school (Steinberg, 1996). Wentzel and Feldman (1993) suggest that educated parents who have a history of economic and career success can be role models of achievement for their children.

Research has also shown that peer relationships play an important role in academic outcomes. Children who lack peer-acceptance are at risk for poor academic outcomes (Flook, Repetti, & Ullman, 2005; O'Neil, Welsh, Parke, Wang, & Strand, 1997; Woodward & Fergusson, 2000). For example, O'Neil *et al.* (1997) found that pre-schoolers who were rejected by their peers were likely to under-perform in tests of mathematics and language at 2-year follow-up. Woodward and Fergusson (2000) found that peer relationship problems at age 9 were associated with poor academic achievement at age 16, and unemployment risk at age 18. Negative long-term effects of peer relationship problems were also demonstrated by Flook, Repetti, and Ullman (2005), who showed that a lack of peer acceptance in 4th Grade (age 9-10) predicted low academic confidence and internalizing symptoms (such as depression and anxiety) in 5th Grade (age 10-11), which in turn predicted low academic performance in 6th Grade (age 11-12).

In a recent UK-based study, Frederickson and Petrides (2008) investigated the impact of ethnicity and socio-economic status (SES) on academic performance in 517

adolescents. It was found that high-SES pupils consistently outperformed low-SES pupils, and *White UK* pupils outperformed *Black* and *Pakistani* pupils. However, for the ethnic group differences, when controlling for cognitive ability and SES, only the significant effect for *Pakistani* pupils remained. Interestingly it was also found that SES was less important a predictor of academic attainment than cognitive ability.

Discrepancies between ethnic groups were also established by Haque and Bell (2001), who found that *White UK* pupils outperformed all ethnic minority groups (*African*, *Bangladeshi*, *Indian*, *Pakistani*, and *Other*) at Key Stage 3 assessments (at age 14), but when controlling for SES the effect was attenuated; however, at age 16, there were no significant differences in academic performance (now, at age 16, GCSE performance) between ethnic groups, apart from *Indian* pupils outperforming *White UK* pupils.

School Factors

Research shows that variables related to the school environment have an impact on pupils' academic progress. Early work by Michael Rutter and colleagues suggested a causal effect of school qualities on pupil progress: they found that there were substantial variations among schools in pupil progress, and that these variations could not be fully attributed to variations in intake, but were systematically related to measured school qualities; they also found that the correlations between pupil achievement and school quality were greater at exit than at entry (Maughan, Mortimore, Ouston, & Rutter, 1980; Rutter, Maughan, Mortimore, Ouston, & Smith, 1979). The importance of the school environment was also demonstrated by

Scheerens and Bosker (1997), who conducted a meta-analysis of studies covering primary and secondary schools from Europe, North America, Australasia, and some Third World countries. They found that school-based variations accounted for 19% of variance in pupils' attainment. Rutter and Maughan (2002) suggest that "school qualities" or "school variations" encompass a wide range of factors, including teacher-pupil dyads, classroom management, pedagogic qualities, departmental features, and school-wide features (such as school ethos).

One school quality that has been extensively researched is class size. Bennett (1996) completed a survey of parents, teachers, head-teachers, and governors; each group believed that classes were too large, and that this had adverse effects on teaching and learning. There was also a consensus that large class sizes lead to reduced individual teacher attention, poorer assessment of work, and poorer behaviour, standards of work, and safety. A large-scale study in Tennessee, USA showed significant positive effects for lower class sizes on pupil achievement, which was more pronounced for younger children and children with special needs (Nye & Hedges, 2000). Ecalle, Magnan, and Gibert (2006) found that class size had a significant impact on reading and spelling skills in 6 to 8 year-olds, with those in smaller classes achieving higher than those in larger classes. More generally, research suggests that class size effects are reduced with age, and may be greatest for disadvantaged pupils (Goldstein *et al.*, 2000).

Another area of research has been the impact of school resources, which has been the subject of much debate (Greenwald, Hedges, & Laine, 1996; Hanushek, 1986; Hedges, Laine, & Greenwald, 1994). There have been mixed findings in studies

investigating the association between school expenditure and student performance: at any given level of resource, schools tend to differ in their ability to make use of what is available; also, schools with similar resources tend to vary in their level of effectiveness (Rutter & Maughan, 2002). However Rutter and Maughan (2002) suggest that it would not follow that a major increase or decrease in resources would make no difference.

Structure of thesis

This thesis is structured as a series of papers which follow a logical sequence: a literature review, followed by three empirical papers that investigate adolescent ADHD and its influence on academic performance. Prior to presenting the empirical studies in the thesis, Chapter 3 provides an overview of the methodology employed.

The aims of the thesis are to provide an examination of community-based adolescent ADHD symptoms, focusing on their (a) standing within the adolescent psychopathological structure, (b) relationship with academic performance, and (c) relative predictive power over academic performance compared to other variables.

Chapter 2: Literature Review

Chapter 2 provides a review of research into the relationship between ADHD and academic performance. Firstly cross-sectional and longitudinal studies which examine this relationship in pre-schoolers, children, adolescents, and adults are reviewed. Second, the chapter examines the reasons behind the relationship between ADHD and academic underperformance: can the impairment be attributed to ADHD symptoms, comorbidity, or underlying cognitive deficits? The chapter ends with an overview of

interventions that are directed towards addressing the academic impairment of ADHD individuals.

Chapter 3: Methodological overview

Chapter 3 describes overall the methodological, practical and ethical issues relating to the research. The chapter begins with a comparison of the characteristics of the four participating schools, focusing on nationally available socio-economic and academic statistics. Next, logistics of data collection are discussed, focusing on how data collection was arranged, at what time-point in the PhD period each school participated in the research, and what data were collected at these time-points. Ethical considerations are also outlined, specifically focusing on the rationale for the consent procedure. Finally, more specific aspects of data collection for *Study 1*, *Study 2*, and *Study 3* are discussed, including where exactly the testing took place, what participants were asked to do, and what information was fed back to schools.

Chapter 4: Empirical Study 1

The majority of ADHD research is concentrated on childhood cases, with less of a focus on adolescents and adults (Willoughby, 2003). High rates of co-morbidity in ADHD have been interpreted as evidence for and against the validity of the disorder in older individuals. It has been argued that ADHD symptoms in older cases are merely a facet of other disorders such as anxiety or depression (Wilens *et al.*, 2003), however others interpret the psychopathological similarities between younger and older cases as support for the validity of the disorder in older individuals (Spencer *et al.*, 1994). The aim of *Study 1* was to provide evidence that ADHD symptoms operate separately from other typically comorbid conditions in the adolescent

psychopathology spectrum, thus supporting the notion that adolescent ADHD is a valid, continuum-based syndrome. To achieve this aim, a community sample of 502 adolescents completed measures of ADHD (current and retrospective), depression, anxiety, and aggression; the item scores were then submitted for factor analysis. Five separate dimensions were identified, namely: current ADHD, retrospective ADHD, depression, anxiety, and aggression.

Chapter 5: Empirical Study 2

The relationship between ADHD and academic underperformance has been well documented (Barry, Lyman, & Klinger, 2002; Biederman *et al.*, 1996; McGee *et al.*, 1991), however most of this research has focused on child, clinical samples (Loe & Feldman, 2007). There is a lack of cross-sectional, community-based, purely adolescent studies investigating ADHD and academic performance. Loe and Feldman (2007) suggest that more emphasis should be placed on community/school samples as opposed to clinic samples to avoid selection bias, and a greater focus should be placed on secondary school settings. In light of these points, *Study 2* investigated the relationship between ADHD symptoms and academic performance in adolescents. The relative predictive weight of ADHD was compared to cognitive ability, school-oriented motivation, depression, anxiety and aggression. Four hundred and twenty-eight Year 11 pupils (aged 15 and 16), from 4 schools in England and Wales, participated in the study. The predictive power of ADHD symptoms over academic attainment was nearly on a par with school-oriented motivation and general cognitive ability.

Chapter 6: Empirical Study 3

The dual pathway model of ADHD (Sonuga-Barke, 2002) suggests that ADHD is underpinned by two distinct, but parallel pathways: (a) cognitive dysregulation, represented by impaired response inhibition (Barkley, 1997; Schachar *et al.*, 2000); and (b) altered motivational style, represented by delay aversion (Sonuga-Barke *et al.*, 1996). The respective roles that these pathways play in the relationship between ADHD and academic performance has received scant research attention, however one study found that cognitive dysregulation, not motivational style, underpins the relationship in preschoolers (Thorell, 2007). The aim of *Study 3* was to investigate the role of cognitive dysregulation and motivational style in the relationship between adolescent ADHD and academic attainment. Due to recruitment problems, unfortunately the sample size was severely constricted, with $n = 23$. Participants completed measures of ADHD, cognitive ability, response inhibition, and delay aversion. With the external validity under intense scrutiny, there was no support for the dual pathway model in the results.

The *Literature Review* (Chapter 2) has been published in a peer-reviewed journal, *Child: Care, Health and Development* (see Appendix 1). *Empirical Study 2* (Chapter 5) is currently submitted for publication in *Journal of Adolescence*.

Chapter 7: General discussion

The general discussion summarises the findings from each study in relation to the current literature. Methodological issues, including study limitations, are presented accordingly, along with future research directions. Theoretical and practical implications are also discussed.

Chapter 2

Literature Review

ADHD and academic performance: a review[1]

[1] This study formed the basis of an article accepted for publication in Child: Care, Health, and Development: Daley, D. and Birchwood, J. (2010). ADHD and academic performance: Why does ADHD impact on academic performance and what can be done to support ADHD children in the classroom?

Chapter 3

Methodological overview

Schools' characteristics

It was decided that the best method of accessing large numbers of adolescents for the research was through high schools; subsequently, approval was obtained from the Research Ethics and Research Governance Committee, School of Psychology, Bangor University, to invite schools to participate in the research. Six high schools (state and private) in the area surrounding Bangor University were contacted via email; one replied and agreed to participate in the research. Personal contacts were used to recruit three further schools: in Colwyn Bay, North Wales, and two in Sandwell, West Midlands. For more information on the participating schools' socio-demographic context and educational performance, see Table 1.

Table 1: Participating schools' characteristics.

School	LEA	School size (number of pupils	% pupils who achieved 5+ A*-C GCSEs; *national average for relevant year*	% pupils entitled to free School Meals	"ACORN" Postcode group
Perryfields High School	Sandwell, England	971 (2008)	60% (2008); *65.3%*	12.2% (2009)	Secure families
Oldbury College of Sport	Sandwell, England	1588 (2008)	58% (2008); *65.3%*	18% (2009)	Settled suburbia
Ysgol David Hughes	Gwynedd, Wales	1250 (2006)	64% (2005); *52%*	7.3% (2006)	Wealthy executives
Ysgol Bryn Elian	Conwy, Wales	823 (2008)	66% (2008); *56%*	17.7% (2008)	Struggling families

It should be noted that in contrast to England, GCSE results are not published annually in Wales. The figures obtained for Ysgol David Hughes and Ysgol Bryn Elian were taken from Estyn school inspection reports; the most recent inspections were in 2006 and 2008 respectively. Figures regarding the proportion of pupils who

were entitled to free school meals were also found in the Estyn reports, however this information was not available in the English schools' Ofstead reports; this information for the English schools was obtained directly from the schools – however, only data for 2009 was available. The GCSE A*-C grade percentage of the West Midlands schools seem to be slightly lower than the English national average for 2008, whereas the Welsh schools A*-C percentage seems to be higher than the Welsh national averages for the given years. The ACORN postcode group was obtained from ACORN (A Classification Of Residential Neighbourhoods; CACI, 2003). Using the percentage of free school meals and the schools' ACORN postcode group as a proxy for socioeconomic status, Table 1 shows that although the socioeconomic status of the schools' intake seems to differ somewhat between schools, there were similar percentages of children who achieved 5 or more A* to C grades at GCSE.

Study logistics and timeline

Once initial contact had been made with the schools and they had agreed to take part, meetings took place between school senior management staff and the principal investigator (James Birchwood) to discuss the logistics of data collection for *Study 1* and *Study 2* (Chapters 4 and 5). Data collection for *Study 1* and *Study 2* required parental *opt-out* consent (see below). In the first year of the PhD, the two Midlands-based schools took part in the research in the spring term. In the second year of the PhD, further data were collected for Studies 1 and 2: Perryfields High School again took part, along with the two North Wales schools; again, this occurred in the spring term. On this occasion however a number of participants were invited to complete neuropsychological testing, for empirical *Study 3* (*Chapter 6*). Data collection for *Study 3* required parental *opt-in* consent (see below). Unfortunately few parents

returned the *opt-in* consent forms, resulting in low participant numbers and it was decided to extend data collection into the third year.

In PhD year 3, Year 11 pupils in Perryfields High School were invited to participate in an initial screening phase; these data were added to the data set for *Study 1*. By this time, writing and data analysis for *Study 2* had been completed; as such there was a discrepancy in sample size between the two papers. All individuals (n=84) who participated in the third year screening phase were also invited to complete neuropsychological testing. However no parents returned the *opt-in* consent forms (despite receiving a reminder to do so, and being informed that for every consenting participant, the school would receive money to go towards an item of school equipment), which meant that participants for neuropsychological testing would have to be recruited from elsewhere. To complete the data set for *Study 3* (*Chapter 6*), a small number of participants were recruited from community-based ADHD support groups (in order to increase the number of high-ADHD participants in the sample), and the remainder were recruited using an opportunity sampling/snowballing method. These extra participants completed the study in their homes, and were contacted directly for their GCSE results.

Ethical considerations

The research was carefully designed in order to ensure that rigorous standards of ethical practice were adhered to. As minors were taking part in the research, it was ensured that there was ample opportunity for participants and parents to gain an understanding of the nature, purpose, and potential consequences of the research. Letters were sent to parents of every Year 11 pupil, outlining the research and stating that the principal investigator would be present at an upcoming parents' evening in

order to answer any queries. The letters also contained parental *opt-out* consent forms: here, parents were asked to return the form if they did not want their child to take part in the research. The use of opt-out parental consent in this study was reviewed and accepted by the Bangor University School of Psychology Research Ethics and Research Governance Committee. The parental *opt-out* approach was adopted for several reasons. Firstly, the data collection took place in school, under the supervision of the investigators and schoolteachers; there was to be no one-to-one testing (which would require full, opt-in, parental consent). Secondly, the opt-out procedure is less burdensome for busy parents, as it removes the requirement for the parent to take any action unless they do not wish for their child to take part. The consent process was in full accordance with British Psychological Society (BPS) guidelines: "...research with schoolchildren under the age of 18 also requires that parents or guardians be informed about the nature of the study and the option to withdraw their child from the study if they so wish." (British Psychological Society [BPS], 2004; p. 5). Parents are only required to return signed consent forms if the school requires it (BPS, 2004); in this research every school made a preference for the *opt-out* parental consent process which they had each used in previous research participation and was supported by the school governors.

On the day of testing all pupils taking part provided written consent to participate in the research and for the principal investigator to access their GCSE results from the school.

The neuropsychological testing for Study 3 had to be carried out one-to-one, therefore the participant would be in a room alone with the experimenter. This required full informed parental consent.

Data collection procedures

Data collection procedures for *Study 1* and *Study 2* (Chapters 4 and 5) followed a similar pattern. Participants had to first complete a consent form, which was followed by the questionnaire battery, and cognitive test (Raven's Standard Progressive Matrices). Depending on the school, participants either completed the study in the school hall (on exam tables), or in their form classroom; following consultation with senior management school staff, it was decided that data collection could take place during a PSHE (Personal, Social, and Health Education) class.

For school hall data collection, participants entered the hall along with their form teachers and were asked to take a seat at one of the exam tables. The principal investigator then addressed the participants and provided standard study instructions (see Chapters 4 and 5 for more details). At all times there were several form teachers, one senior management staff member, and the principal investigator present to oversee the session.

The process differed slightly for classroom data collection. All form teachers were briefed before the session; they were reminded about the nature of the study and were asked to remain in the classroom while the principal investigator supervised the students, dealt with any questions and ensured that the process ran smoothly.

The neuropsychological testing for *Study 3* (*Chapter 6*) required one-to-one testing, therefore before data collection, the principal investigator contacted senior management staff to reserve a suitable room in the school. On the day of testing, the principal investigator was given details of where the participants would be at the time of testing; the participants were then summoned to the room to complete the tests, one pupil at a time. Participants first completed a consent form, and then completed the computer-based neuropsychological tasks (for more information, see *Chapter 6*). The

participants who completed the study in their own homes (recruited via ADHD support groups and opportunity/snowball sampling) also completed the questionnaire battery and Raven's Matrices cognitive test. For these participants the study logistics were discussed with the parent of the participant; typically the data collection was carried out in one session (although the option was provided to split it into two sessions), with the participant completing the questionnaire battery, cognitive test, and neuropsychological tasks in approximately a two-hour period. At the start of the session the parent and child completed consent forms, and the testing would then begin.

For those who completed the study at school, GCSE results were collected from the school, once the new school term had begun, approximately one month after the results had been released. Each school was asked to provide a spreadsheet of the participants' results, subject-by-subject. This proved to be a lengthy process, with the time taken to return the spreadsheets varying from one week to several months. For those who completed the study in their own homes, GCSE results were collected directly from the participant via telephone, soon after they were released nationally.

Once the data had been analysed, the schools were provided with a feedback report, consisting of background to the research, core results, some discussion points, and educational implications. Perryfields High School requested a verbal presentation of the research, therefore in the spring term of 2007 the principal investigator visited the school and made a presentation to the senior management team and several heads of department; this presentation also contained a research proposal for the neuropsychological study (*Chapter 6* of this thesis).

Chapter 4

Study 1

Adolescent ADHD symptoms and the structure of psychopathology in a community sample

Abstract

Background: It is not known the extent to which the adolescent ADHD dimension remains valid when set alongside measures of adolescent mental health problems in non-clinical samples.

Aims: To investigate the factor structure of ADHD within the presence of other typically comorbid psychological problems in a community-based adolescent sample.

Methods: 502 participants aged 15 and 16, drawn from 4 schools, completed measures of ADHD, anxiety, depression, and aggression.

Results: Exploratory factor analysis revealed a five-factor solution including: current ADHD, past ADHD, aggression, anxiety, and depression.

Conclusions: Adolescent ADHD symptoms are a valid dimension within the spectrum of adolescent mental health characteristics.

Attention deficit hyperactivity disorder (ADHD) is one of the most common childhood developmental disorders, defined by the presence of symptoms of inattention, hyperactivity and impulsivity (APA, 1994). The disorder was traditionally viewed as affecting only children (Willoughby, 2003), and this is supported by DSM-IV criteria recognising only a childhood form (APA, 1994). However, despite the majority of ADHD literature focusing on childhood samples, there is significant evidence showing that the developmental span of the disorder is wider than originally thought, with a preschool onset, and persistence into adolescence and even adulthood (Barkley & Murphy, 1998; Stevenson, Stevenson, & Whitmore, 2003). Support for the concept and validity of an older disorder has arisen through a substantial increase in research in recent years (Willoughby, 2003).

Children with ADHD continue to exhibit symptoms in adolescence and adulthood (Fischer, Barkley, Fletcher, & Smallish, 1993; Mannuzza *et al.,* 1993), and both children and adolescents show levels of impairment within social, family, cognitive, and academic domains (Barbaresi *et al.*, 2007; Barkley, Anastopoulos, Guevremont, & Fletcher, 1991; Biederman *et al.*, 1998).

A key feature of ADHD is co-morbidity. Approximately 30-50% of children diagnosed with ADHD have oppositional defiant disorder (ODD)/conduct disorder (CD) (Biederman, Newcorn, & Sprich, 1991); there is a co-occurrence of anxiety in 20-30% of children with ADHD (Biederman, Newcorn, & Sprich, 1991; Pliszka, 1998); and bipolar disorder prevalence rates of 11-22% have been found (Biederman, 1998). Similar co-morbidity profiles have been found in adolescents. Bird, Gould, and Staghezza (1993) investigated co-morbidity in a group of 9- to 16-year olds diagnosed with ADD (attention-deficit disorder) with or without hyperactivity. They found that 48% had comorbid depression/dysthymic disorder, 36% had comorbid

oppositional defiant disorder/conduct disorder, and 36% had comorbid anxiety disorder. These high rates of co-morbidity have been interpreted as evidence for and against the validity of the disorder in older individuals (Spencer, Biederman, Wilens, & Faraone, 1994; Wilens *et al.*, 2003). Some clinicians argue that ADHD symptoms in older cases are merely a part of other disorders such as anxiety or depression (Wilens *et al.*, 2003), however others interpret the psychopathological similarities between young and old cases as support for the validity of the disorder in older individuals (Spencer *et al.*, 1994).

Much of the ADHD literature is based on clinic samples, which are at risk of a range of referral biases. Referred samples are usually more severely affected than non-referred samples, however a large number of affected children are not referred for specialist help (Lesesne, Visser, & White, 2003). A referral to specialist services can be the result of factors other than the severity of the disorder *per se*. Family cohesion has been shown to be a strong predictor of referral (Lavigne *et al.*, 1998), as has parents' perception of the seriousness of symptoms (Sayal *et al.*, 2002, 2003). Sawyer *et al.*, (2004) found that parents of non-referred children with symptoms of ADHD felt that they could effectively manage on their own, without outside help. In order to improve the overall generalisability of ADHD research, studies using clinically referred individuals with ADHD need to be complemented by studies using community-based samples (Diamantopoulou, Rydell, Thorell, & Bohlin, 2007; Smalley *et al.*, 2007). Thus while clinic-referred samples are normally composed of chronic, severe cases, there are a significant proportion of undiagnosed, but impaired individuals where specialist help is not sought (Angold, Costello, Farmer, Burns, & Erkanli, 1999; Smalley *et al.*, 2007). In other domains, children and adolescents who display significant symptoms of psychiatric disorder but do not necessarily seek help

or attract a formal diagnosis, have been found to be as psychosocially impaired as their clinically diagnosed counterparts (Angold *et al.*, 1999).

Research into the structure of ADHD has demonstrated a bi-dimensional factor structure, with inattention and hyperactivity/impulsivity loading separately (Bauermeister *et al.*, 1995; Healey *et al.*, 1993; Lahey *et al.*, 1988); these findings gave rise to the DSM-IV subtype differentiation (DSM-IV; APA, 1994). However, the DSM-IV structure was devised using largely childhood samples, therefore concerns were aired over the validity of diagnosing adolescents and adults using this child-based taxonomy (Span, Earleywine & Strybel, 2002). Subsequently efforts were made to identify the factor structure on older individuals. The bi-dimensional structure has been replicated in adult samples (Du Paul *et al.*, 2001; Spencer, Biederman, Wilens, & Faraone, 1998), however Span, Earlywine and Strybel (2002) concluded that a 3-factor solution of inattention, hyperactivity, and impulsivity best described adult ADHD symptoms. In a college-student study, Glutting, Youngstrom, and Watkins (2005) found that the factor structure changed according to where symptom reports came from: analysis using parent-report symptoms aligned well with the DSM-IV two-factor structure, however self-report symptoms elicited a three-factor solution, in line with the work of Span, Earlywine, and Strybel (2002). However, in a male adolescent study, Rasmussen *et al.* (2002) found that the bi-dimensional structure can be reproduced by self-report.

In a sample of 5-15 year-olds, Pillow *et al.* (1998) found that a 3-factor model was the best fit when examining ADHD in isolation. However, they also examined the factor structure of ADHD in relation to externalising behaviour problems (namely ODD and CD), and it was found that ADHD and ODD/CD formed two distinct factors. This supported the work of Hinshaw (1987) who, in a review of studies of

disruptive behaviour disorders, found that ADHD and ODD/CD were separate although related dimensions, suggesting that ADHD and aggressive disorders are correlated but ultimately distinct.

Similar to Pillow *et al.* (1998), in the present study it was decided to examine the factor structure of ADHD in respect of other typically co-morbid problems, both externalising (i.e. aggressive behaviours) and internalising (depression and anxiety). It was decided to carry out the study in a non-clinical adolescent sample, as much of the previous research has focused on either child or adult samples, and has seldom focused on a purely adolescent sample, in the non-clinical range. The ADHD symptom presentation tends to change with age, with symptoms – in particular hyperactivity and impulsivity – dissipating over time (Biederman, Mick, & Faraone, 2000). Therefore it seems pertinent to examine the make-up of the disorder at the adolescent stage.

ADHD is understood to be a developmental disorder that changes in presentation as the individual grows older (Schmidt & Petermann, 2009). As such, whether the adolescent and childhood (i.e. current and past) symptomatologies share latent properties, or not, was also examined: do childhood ADHD behaviours differ from adolescent ADHD behaviours enough to form separate constructs, or will they intertwine? Most studies examining the factor structure of ADHD do so using current-symptom checklists (Pillow *et al.*, 1998; Rasmussen *et al.*, 2002; Span, Earlywine, & Strybel 2002); however in the present study it was decided to capture the developmental nature of these symptoms by asking participants about the history of their ADHD-like symptoms. Retrospective ADHD symptoms were measured using the widely used Wender Utah Rating Scale (WURS; Ward, Wender, & Reimherr, 1993); although this is mainly aimed at adults, it was chosen because (a) the

participants had reached an age where retrospective ratings would be appropriate, (b) is the only retrospective ADHD symptoms checklist available, and (c) the participants were older adolescents, most only two years from adulthood. *Chapter 5* of this thesis aims to examine the impact of lifetime ADHD symptoms on academic performance and accordingly measures current and historical ADHD symptoms. As indicated, one might expect that in a developmentally based disorder like ADHD, the current and retrospective measures should be correlated but perhaps will load onto different factors; if this proves to be the case then it will support the measurement of current and historical symptoms in *Chapter 5*.

The aim of the present study was to examine the factor structure of ADHD within the presence of other typically co-occurring psychological problems (depression, anxiety, aggression), in a community-based adolescent sample. It was investigated whether adolescent ADHD symptoms form a dimension that is not polluted by symptoms of other typically comorbid forms of psychopathology. To the author's knowledge, a purely adolescent study of this exact nature has never been carried out before, therefore without any concrete *a priori* theoretical framework on which to structure a possible *confirmatory factor analysis* around, *exploratory factor analysis* was employed. Child (1990) suggests that exploratory factor analysis is used to explore the underlying factor structure of a set of measured variables without imposing a preconceived structure on the outcome.

Method

Participants

Ethical approval for the study was granted by the School of Psychology Research Ethics and Research Governance Committee, Bangor University. All Year 11 pupils

in four high schools in the West Midlands, and North Wales, UK, were invited to take part in the study. Consent was obtained via parents (using "opt-out" consent forms several weeks before the study) and pupils (immediately before the study began). There were 502 participants, aged 15 and 16, of which 47.8% were male and 45.2% were female (6.4% failed to state their gender).

Measures

All participants were asked to complete a questionnaire battery consisting of measures of ADHD, aggression, anxiety, and depression (Appendix 4). The measures included in the questionnaire were as follows:

The Adult ADHD Rating Scale (AARS; Barkley & Murphy, 1998): the AARS is a self-report scale consisting of 18 items based on the DSM-IV symptom list, including inattentiveness (9 items), impulsiveness (3 items) and hyperactivity (6 items). Participants are asked to rate their own behaviour over the past 6 months on a 4-point scale (0 = *rarely*; 1 = *sometimes*; 2 = *often*; 3 = *very often*). Edwards, Barkley, Laneri, Fletcher, and Metevia (2001) report Cronbach's alpha coefficients of .92 for the inattention items, and .91 for the hyperactive/impulsive items. Cronbach's alpha coefficients for the present sample are .87 for the inattention items, and .81 for the hyperactive/impulsive items. In the present sample, participants' total AARS scores ranged from 0 to 54. This measure has previously been used in studies employing older adolescent/young adult samples (DuPaul *et al*, 2001).

The Wender Utah Rating Scale (WURS; Ward, Wender, & Reimherr, 1993): this is a 25-item questionnaire that can be used to retrospectively yield estimated scores for individuals' ADHD symptoms during childhood. Participants are asked to rate their childhood behaviour on a 5-point scale (1 = *Not at all/slightly*; 2 = *Mildly*; 3 =

Moderately; 4 = *Quite a bit*; 5 = *Very much*). In a sample of adult outpatients with ADHD, psychiatric outpatients, and controls, Ward, Wender, and Reimherr (1993) found that scores on the WURS correlate significantly with retrospective reports of childhood symptoms by parents. The measure has excellent internal consistency and good test-retest stability (Ward, Wender, & Reimherr, 1993). Cronbach's alpha for the current sample is 0.94. In the present sample, participants' total scores on the WURS ranged from 25 to 115.

The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983): this is a self-report scale with 14 items; participants rate their behaviour by agreeing with one of four statements, which vary from question to question (for example: "I have lost interest in my appearance" – *'Definitely', 'I don't take as much care as I should', 'I may not take quite as much care', or 'I take just as much care as ever'*). In a review of the HADS, Bjelland, Dahl, Haug and Neckelmann (2002) report widespread agreement on its psychometric properties. For example, most factor analyses demonstrate a two-factor solution, HADS anxiety and HADS depression. The correlations between the two sub-scales vary from 0.40 to 0.74. The Cronbach's alpha for HADS anxiety vary from 0.68 to 0.93 (mean 0.83) and for HADS depression from 0.67 to 0.90 (mean 0.82). Cronbach's alpha coefficients for the current sample are 0.8 for the anxiety items, and 0.65 for the depression items. Correlations between the HADS and other commonly used measures range from 0.49 to 0.83. White, Leach, Sims, Atkinson, and Cottrell (1999) validated the HADS for use with adolescents: they gave the HADS to schoolchildren and adolescent psychiatric patients and found adequate test-retest reliability and factor structure, and found that it discriminates between adolescents diagnosed with depressive or anxiety disorders and those without a diagnosis. White *et*

al. (1999) concluded that the HADS could be used with adolescents as a diagnostic aid and for screening in non-clinical settings.

The Aggression Questionnaire, short form (AQ; Bryant & Smith, 2001): this is a 12 item self-report scale, based on the original 27-item scale from Buss and Perry (1992). It consists of 4 factors (each consisting of 3 items) measuring physical aggression, verbal aggression, anger and hostility. Participants rate their own behaviour on a scale of 1 – 6 (1 = *extremely uncharacteristic of me*; 6 = *extremely characteristic of me*). Bryant and Smith (2001) report Cronbach's alpha scores ranging from 0.70 to 0.83 for factors of the Aggression Questionnaire. Using structural equation modelling, Bryant and Smith (2001) also tested the comparability of the factors represented in the short and long forms of the questionnaire, and reported that shortening the questionnaire to 12 items did not appear to change the conceptual meaning of the underlying aggression sub-traits. The measure is a valid tool for use with adolescents: both the 12-item and 29-item versions have been used in several adolescent studies (Ang, 2007; Harty, Miller, Morren & Meesters, 2002; Newcorn, & Halperin, 2009), and indeed the original 29-item AQ was normed with a standardisation sample that included adolescents (Buss & Warren, 2000). In an examination of the 12-item AQ in a general population adolescent sample, Ang (2007) found Cronbach's alpha of 0.84 for the total aggression score. The total aggression score Cronbach's alpha for the present sample is 0.91. In the present sample, participants' total AQ scores ranged from 12 to 72.

Procedure

Parents of pupils were sent study information sheets (Appendix 2) and *opt-out* consent forms (Appendix 3); parents who did not wish for their child to take part were

asked to return the *opt-out* consent form to the research team. Immediately before the study, pupils were asked to read a study information sheet (Appendix 2) and complete a consent form (Appendix 3). Participants were then asked to complete the questionnaire battery; this took place either in the school hall or in their classrooms.

Results

Data preparation/analytical strategy

To explore the factor structure of adolescent ADHD, anxiety, depression, and aggression, exploratory factor analysis (EFA) was used. This method of factor analysis was used instead of confirmatory factor analysis (CFA), as there was no existing theoretical framework on which to base a CFA (Child, 1990).

Prevalence of psychopathological symptoms

(a) ADHD

A participant was classified as expressing significant symptoms of ADHD if he/she scored above the clinical cut offs on both the AARS (a measure of current ADHD symptoms) and WURS (a retrospective childhood measure of ADHD symptoms). Ward, Wender, and Reimherr (1993) cited a cut-off score for the WURS of 46, thus participants with a score of 46 or above would be classified as having ADHD as a younger child. For AARS scores, the DSM-IV ADHD criteria were employed in order to generate symptom counts. Therefore, a critical case would require six significant symptoms of inattention and/or six symptoms of hyperactivity/impulsivity; a symptom is classified as being significant if the participant endorses the "often" or "very often" response (as opposed to "rarely" or "sometimes"). Therefore, participants were classified

as expressing ADHD symptoms if they had a score of 46 or greater on the WURS and also fulfilled the DSM-IV criteria for current ADHD symptoms, as measured in the AARS.

According to the above criteria, 2.98% of the sample (15 participants: 9 male; 6 female) scored above the cut-off for ADHD combined type (significant level of inattention and hyperactivity/impulsivity), 3.38% of the sample (17 participants: 10 male; 7 female) scored above the cut-off for the inattentive sub-type, and 3.38% of the sample (17 participants: 10 male; 6 female; 1 individual did not state their gender) scored above the cut-off for the hyperactive/impulsive sub-type. This amounted to a total prevalence rate of 9.72%. This is slightly higher than in Smalley *et al.* (2007), who found an overall prevalence rate of 6.7% in a large sample of Finnish adolescents; similar to the present study, Smalley and colleagues used the stringent criteria of both prospective and retrospective ADHD symptomatology to identify critical cases.

(b) Anxiety and depression

A comparison between symptom levels of anxiety and depression in the current sample and the community sample (aged 12 to 16, $n = 248$) scores derived from White *et al.* (1999) is displayed in Table 2. Table 2 suggests that the distribution of symptom levels in the current sample is very similar to that of White *et al.*'s British adolescent general population sample, especially for anxiety scores. The general trend of the depression scores from the current sample is similar to White *et al.* but they are less severely skewed towards low scores, with slightly higher medium and high scores; perhaps the truncated age range of the present sample compared to White *et al.* could account for the slight discrepancy in scores.

Table 2: A comparison between the current study and White *et al.* (1999) of participants who fall within White *et al.*'s specified cut-off points for levels of anxiety and depression

Current sample			
Measure	Low score	Medium score	High score
HADS anxiety	63.3%	21%	15.8%
HADS depression	79.1%	15.2%	6%
White *et al.* (1999)			
Measure	Low score	Medium score	High score
HADS anxiety	67%	19%	14%
HADS depression	87%	11%	2%

(c) Symptoms of aggression

To identify elevated levels of aggression, the cut-off of at or above two standard deviations above the mean was applied (as used in Bryant & Smith, 2001). In the current sample, 4.8% of participants scored above this cut-off for total aggression score.

Gender differences

Analysis of variance (ANOVA) was conducted to examine the differences in psychopathology scores between males and females in the sample. There were no significant differences between males and females on scores derived from the AARS, WURS, HADS depression scale, and AQ. However on the HADS anxiety scale, females scored higher (mean = 8.26) than males (mean = 6.81), ($F = 15.79$; $p = .000$), a finding which has been established elsewhere (White *et al.*, 1999).

Associations between the variables

Correlations between the variables that were to be entered into factor analysis were examined. Results of a 1-sample Kolmogorov-Smirnov test indicated that the scores derived from the AARS, WURS, AQ, and HADS were non-parametric (with K-S *Z* values ranging from 1.911 to 2.803 and *p* from .000 to .001), therefore associations between the scores were examined using Spearman's correlation. The results are presented in Table 3. There were significant positive correlations between all of the variables. The strongest associations were within the ADHD scores (AARS total, AARS inattention, AARS hyperactivity/impulsivity, WURS) and between the ADHD scores and total AQ score. There were more moderate, but significant, correlations between both HADS scores and the rest of the variables. The results indicate that individuals with higher ADHD scores tend to report more problems with anxiety, depression and aggression.

Table 3: Spearman correlations between variables

Measure	AARS inattention	AARS hyp/imp	WURS	AQ score	HADS depression	HADS anxiety
AARS total	.94*	.93*	.70*	.61*	.38*	.55*
AARS inattention		.75*	.69*	.58*	.4*	.51*
AARS hyp/imp			.63*	.57*	.31*	.53*
WURS				.68*	.49*	.59*
AQ score					.36*	.46*
HADS depression						.42*

NB. AARS hyp/imp = AARS hyperactivity/impulsivity; * = $p < .01$

Factor analysis

Item scores for ADHD (current and retrospective), anxiety, depression, and aggression were submitted to exploratory factor analysis. Pearson's correlation analysis of the items revealed no coefficients of above 0.9, suggesting that no items should be removed from the analysis due to multicolinearity (in line with the recommendation of Field, 2000). The scree plot of eigenvalues (Appendix 5) from an unrotated factor analysis yielded an interpretable plot suggesting 5 factors should be extracted from the item-intercorrelation matrix. The Kaiser-Meyer-Olkin statistic yielded a value of 0.953 indicating a substantial level of common item variance. Bartlett's test of sphericity was significant (*Chi-square* = 17933.6; $p < .000$) indicating that there were potentially significant relationships between the variables and that factor analysis was appropriate. The varimax rotated five-factor solution is presented in Table 4; this accounted for 46.39% of the variance. Comrey and Lee (1992) suggest that only variables with loadings in excess of 0.32 should be interpreted, as loadings less than this value cannot be considered pure measures of the factor. Item loadings greater than 0.32 in bold type are intended to draw attention to items that primarily load on each factor; item loadings greater than 0.32 that cross-load onto other factors are presented in italic type.

Factor 1 was labelled *Retrospective ADHD* and included 21 of the 25 items from the WURS including *"Concentration problems, easily distracted"*, and *"Acting without thinking, impulsive"*. Factor 2 was labelled *Current ADHD* and contained 17 of the 18 AARS items: all 9 inattention items (such as *"Difficulty in sustaining my attention in class"* and *"Easily distracted"*), and 8 hyperactive/impulsive items (such as *"Feel restless"* and *"Talk excessively"*). The AARS item that did not primarily load in Factor 2 (*"Have difficulty taking part in leisure activities"*) did in fact cross

load onto this factor. Three items from the WURS also cross-loaded onto Factor 2 (*"Concentration problems, easily distracted"*, *"Trouble with not following things through"*, and *"Trouble with authorities, trouble with school"*).

Factor 3 was labelled *Aggression* and contained all items from the AQ such as *"Given enough provocation I may hit another person"* and *"I have trouble controlling my temper"*. In Factor 3 there was 1 cross loading item from the AARS (*"Have difficulty waiting my turn"*) and 5 from the WURS (including *"Hot or short temper, low boiling point"*).

Factor 4 was labelled *Anxiety* and included 6 HADS anxiety items (such as *"Worrying thoughts go through my mind"* and *"I get sudden feelings of panic"*), 1 HADS depression item (*"I feel as if I am slowed down"*), and 3 items from the WURS (*"Anxious, worrying"*, *"Nervous, fidgety"*, *"Low opinion of myself"*). Also there were several cross loading items from the WURS and AQ.

Factor 5 was labelled *Depression* and contained 6 items from the HADS, including 5 depression items (such as *"I can laugh and see the funny side of things"* and *"I look forward with enjoyment to things"*) and 1 anxiety item (*"I can sit at ease and feel relaxed"*). Factor 5 also included "*Difficulty taking part in leisure activities*" from the AARS, and *"Unpopular with other children, didn't keep friends for long, didn't get along with other children"* from the WURS.

Table 4: Varimax rotated 5-factor solution

Measure	R-ADHD	C-ADHD	Agg	Anx	Dep
AARS					
Fail to give close attention to details or make careless mistakes at work	.200	**.592**	.085	.159	.076
Fidget with hands, feet or squirm in my seat	.128	**.522**	.157	.248	.000
Difficulty sustaining my attention in tasks	*.321*	**.573**	.113	.119	.088
Leave my seat in situations in which seating is required	.254	**.544**	.198	-.042	.025
Don't listen when spoken to directly	.208	**.616**	.200	.020	.246
Feel restless	.210	**.467**	.219	.305	.115
Don't follow through on instructions and fail to finish work	.223	**.643**	.167	.091	.200
Have difficulty taking part in leisure activities	.086	*.325*	.004	.181	**.386**
Have difficulty organising myself.	.052	**.581**	.131	.189	.178
Feel "on the go" or driven by a motor	.167	**.444**	.060	.080	-.213
Avoid, dislike or reluctant to engage in work that requires sustained thinking	.194	**.585**	.174	.082	.055
Talk excessively	.153	**.548**	.217	.242	-.096
Lose things necessary for tasks or activities	.143	**.562**	.159	.196	.169
Blurt out answers before questions have been completed	.154	**.572**	.180	.157	.018
Easily distracted	.248	**.623**	.264	.122	.034
Have difficulty waiting my turn	.102	**.515**	*.368*	.060	.107
Forgetful in daily activities	.080	**.592**	.181	.246	.171
Interrupt or intrude on others	.217	**.566**	.310	.020	.139
WURS					
Concentration problems, easily distracted	**.528**	*.358*	.186	.165	.135
Anxious, worrying	*.400*	.021	.038	**.459**	.195
Nervous, fidgety	*.390*	.217	.158	**.406**	.148
Inattentive, daydreaming	**.490**	.306	.190	.288	.127
Hot or short temper, low boiling point	**.630**	.133	*.474*	.014	.079
Temper outbursts, tantrums	**.640**	.156	*.418*	.097	.091
Trouble with not following things through	**.591**	*.330*	.198	.164	.139
Stubborn, strong-willed	**.412**	.183	*.352*	.029	.016
Sad or blue, depressed, unhappy	**.420**	.121	.127	*.419*	*.403*
Disobedient with parents, rebellious	**.651**	.193	.294	.038	.102
Low opinion of myself	.432	.121	.078	**.498**	.222
Irritable	**.587**	.272	.256	.285	.146
Moody, have ups and downs	**.583**	.142	.176	*.349*	.236
Feel angry	**.670**	.129	*.356*	.193	.214
Acting without thinking, impulsive	**.649**	.213	.309	.143	.039
Tend to be immature	**.508**	.280	.221	.192	-.070
Feel guilty, regretful	**.461**	.081	.073	*.361*	.013
Lose control of myself	**.682**	.171	*.347*	.197	.043
Tend to be or act irrational	**.644**	.175	.317	.198	.121
Unpopular with other children, didn't keep friends for long, didn't get along with other children	*.395*	.122	-.007	.283	**.402**
Trouble seeing things from someone else's point of view	**.448**	.217	.246	.133	.203
Trouble with authorities, trouble with school	**.532**	*.327*	.193	-.051	.172

Measure	R-ADHD	C-ADHD	Agg	Anx	Dep
Overall a poor student, slow learner	**.461**	.296	-.009	.090	.297
Trouble with maths or numbers	**.387**	.156	-.114	.227	.104
Did not achieve up to my potential	**.578**	.305	-.056	.143	.133
AQ					
Given enough provocation I may hit another person	.267	.179	**.611**	-.079	-.048
There are people that push me so far that we come to blows	.259	.217	**.630**	.022	.048
I have threatened people I know	.248	.240	**.637**	-.051	.080
I often find myself disagreeing with people·	.109	.299	**.590**	.145	.095
I can't help getting into arguments when people disagree with me	.049	.238	**.685**	.094	.086
My friends say that I argue a great deal	.100	.244	**.663**	.113	.119
I flare up quickly but get over it quickly	.200	.163	**.615**	.227	-.077
Sometimes I fly off the handle for no good reason	.211	.209	**.668**	.149	.016
I have trouble controlling my temper	*.348*	.129	**.684**	.018	.082
At times I feel I have had a raw deal out of life	.121	.195	**.523**	*.342*	.278
Other people always seem to get lucky	.200	.203	**.445**	*.362*	.221
I wonder why sometimes I feel so bitter about things	.189	.198	**.467**	*.440*	.242
HADS					
I feel tense or 'wound up'.	.199	.207	*.336*	**.460**	.263
I still enjoy the things I used to enjoy	.057	.055	-.071	-.131	**.557**
I get a sort of frightened feeling as if something awful is about to happen	.074	.186	.047	**.686**	.030
I can laugh and see the funny side of things	.073	.020	-.001	.050	**.698**
Worrying thoughts go through my mind	.157	.139	.075	**.716**	.018
I feel cheerful	.169	.047	.168	.216	**.615**
I can sit at ease and feel relaxed	.208	.206	.149	.289	**.430**
I feel as if I am slowed down	.190	.175	.142	**.381**	.095
I get a sort of frightened feeling like butterflies in the stomach	.080	.094	.032	**.604**	.021
I have lost interest in my appearance	.111	.039	.069	.172	**.454**
I feel restless as if I have to be on the move	.251	.280	.180	**.373**	-.032
I look forward with enjoyment to things	.102	.024	.108	.007	**.667**
I get sudden feelings of panic	.131	.231	.008	**.691**	.116
I can enjoy a good book or radio or TV programme	.074	.153	.162	.055	.293

NB. R-ADHD = Retrospective ADHD, C-ADHD = Current ADHD, Agg = Aggression, Anx = Anxiety, Dep = Depression.

Although there was no statistical item overlap, there are several items from the WURS that appear to directly tap into other constructs, namely aggression, anxiety,

and depression. It was decided to remove these conceptually overlapping items, and re-run the analysis. Indeed there is also conceptual overlap between the AARS and WURS, however these items were not removed, as the intention of including the WURS in this analysis was to examine how separate or intertwined the AARS and WURS items were: would there be separate factors for current and retrospective ADHD symptoms? These findings could help form conclusions about the developmental progression of ADHD symptoms between childhood and adolescence.

The WURS items that were deemed to overlap with anxiety, depression, and aggression were removed and the analysis was re-run. The removed items were as follows: *"Anxious, worrying"*, *"Hot or short temper, low boiling point"*, *"Temper outbursts, tantrums"*, *"Sad or blue, depressed, unhappy"*, and *"Feel angry"*. The scree plot of eigenvalues (Appendix 6) from an unrotated factor analysis yielded an interpretable plot suggesting 5 factors should be extracted from the item-intercorrelation matrix. The Kaiser-Meyer-Olkin statistic yielded a value of 0.950 indicating a substantial level of common item variance. Bartlett's test of sphericity was significant (*Chi-square* = 15579.1; $p < .000$) suggesting that factor analysis was appropriate. The varimax rotated five-factor solution is presented in Table 5, and accounted for 45.81% of the variance. Again, only variables with loadings in excess of 0.32 were interpreted as measures of the factor (Comrey & Lee, 1992); these items are presented in bold type. Cross-loading items are presented in italic type.

Table 5: Varimax rotated 5-factor solution with overlapping items removed

Measure	C-ADHD	R-ADHD	Agg	Anx	Dep
AARS					
Fail to give close attention to details or make careless mistakes at work	**.597**	.197	.083	.165	.076
Fidget with hands, feet or squirm in my seat	**.506**	.158	.158	.247	.005
Difficulty sustaining my attention in tasks	**.566**	*.328*	.126	.115	.086
Leave my seat in situations in which seating is required	**.548**	.244	.215	-.054	.016
Don't listen when spoken to directly	**.633**	.189	.205	.013	.236
Feel restless	**.470**	.208	.218	.309	.120
Don't follow through on instructions and fail to finish work	**.639**	.232	.173	.088	.195
Have difficulty taking part in leisure activities	*.339*	.088	-.004	.177	**.384**
Have difficulty organising myself.	**.565**	.087	.127	.191	.176
Feel "on the go" or driven by a motor	**.441**	.166	.065	.086	-.209
Avoid, dislike or reluctant to engage in work that requires sustained thinking	**.591**	.183	.178	.087	.052
Talk excessively	**.547**	.158	.220	.236	-.099
Lose things necessary for tasks or activities	**.561**	.151	.154	.201	.170
Blurt out answers before questions have been completed	**.575**	.154	.179	.160	.014
Easily distracted	**.620**	.248	.275	.117	.032
Have difficulty waiting my turn	**.526**	.084	*.368*	.060	.102
Forgetful in daily activities	**.590**	.097	.175	.245	.172
Interrupt or intrude on others	**.565**	.213	.318	.017	.129
WURS					
Concentration problems, easily distracted	*.354*	**.528**	.219	.135	.125
Nervous, fidgety	.210	**.415**	.186	.360	.140
Inattentive, daydreaming	.291	**.498**	.208	.283	.136
Trouble with not following things through	*.320*	**.591**	.230	.142	.134
Stubborn, strong-willed	.183	**.381**	*.371*	.032	.012
Disobedient with parents, rebellious	.188	**.627**	*.332*	.021	.094
Low opinion of myself	.117	**.453**	.100	*.464*	.212
Irritable	.261	**.586**	.283	.272	.146
Moody, have ups and downs	.154	**.557**	.199	*.332*	.231
Acting without thinking, impulsive	.196	**.636**	*.344*	.136	.043
Tend to be immature	.251	**.527**	.253	.182	-.072
Feel guilty, regretful	.063	**.484**	.102	*.336*	.010
Lose control of myself	.163	**.656**	*.378*	.194	.041
Tend to be or act irrational	.156	**.641**	*.352*	.185	.120
Unpopular with other children, didn't keep friends for long, didn't get along with other children	.098	**.452**	.022	.236	*.380*
Trouble seeing things from someone else's point of view	.198	**.472**	.285	.091	.188
Trouble with authorities, trouble with school	.302	**.552**	.232	-.078	.155
Overall a poor student, slow learner	.269	**.506**	.022	.055	.282
Trouble with maths or numbers	.121	**.440**	-.091	.207	.108
Did not achieve up to my potential	.256	**.643**	-.015	.114	.124
AQ					
Given enough provocation I may hit	.170	.241	**.635**	-.085	-.050

another person

Measure	C-ADHD	R-ADHD	Agg	Anx	Dep
There are people that push me so far that we come to blows	.220	.223	**.646**	.018	.049
I have threatened people I know	.238	.219	**.658**	-.061	.074
I often find myself disagreeing with people	.290	.101	**.594**	.147	.096
I can't help getting into arguments when people disagree with me	.227	.048	**.695**	.084	.076
My friends say that I argue a great deal	.233	.096	**.672**	.107	.114
I flare up quickly but get over it quickly	.159	.176	**.623**	.232	-.071
Sometimes I fly off the handle for no good reason	.212	.172	**.675**	.160	.026
I have trouble controlling my temper	.133	.290	**.695**	.037	.095
At times I feel I have had a raw deal out of life	.189	.116	**.518**	*.352*	.287
Other people always seem to get lucky	.186	.212	**.450**	*.363*	.228
I wonder why sometimes I feel so bitter about things	.194	.188	**.465**	*.445*	.247
HADS					
I feel tense or 'wound up'.	.207	.192	.327	**.475**	.274
I still enjoy the things I used to enjoy	.046	.078	-.066	-.145	**.563**
I get a sort of frightened feeling as if something awful is about to happen	.183	.100	.032	**.688**	.039
I can laugh and see the funny side of things	.028	.076	-.005	.041	**.696**
Worrying thoughts go through my mind	.132	.179	.063	**.726**	.031
I feel cheerful	.044	.175	.167	.213	**.623**
I can sit at ease and feel relaxed	.195	.224	.148	.293	**.445**
I feel as if I am slowed down	.161	.211	.142	**.385**	.099
I get a sort of frightened feeling like butterflies in the stomach	.086	.114	.027	**.595**	.025
I have lost interest in my appearance	.035	.121	.062	.176	**.464**
I feel restless as if I have to be on the move	.273	.257	.183	**.376**	-.026
I look forward with enjoyment to things	.025	.102	.105	.007	**.675**
I get sudden feelings of panic	.225	.166	-.001	**.685**	.122
I can enjoy a good book or radio or TV programme	.169	.051	.157	.057	.299

NB. C-ADHD = Current ADHD, R-ADHD = Retrospective ADHD, Agg = Aggression, Anx = Anxiety, Dep = Depression.

The results reveal a strikingly similar pattern to the initial analysis, with the same five factors emerging: *Current ADHD*, *Retrospective ADHD*, *Aggression*, *Anxiety*, and *Depression*. Factor 1, *Current ADHD*, contained 17 of the 18 AARS items; Factor 2, *Retrospective ADHD*, consisted of all 20 items from the WURS; Factor 3, *Aggression*, contained all items from the AQ; Factor 4, *Anxiety*, contained 6 HADS anxiety items

and 1 HADS depression item; finally, Factor 5, *Depression*, included 5 HADS depression items, 1 HADS anxiety item, and 1 AARS item. As can be seen in Table 5, there were also several cross-loadings.

Post-hoc factor analysis models

The models above consisted of several current symptomatology measures and one retrospective symptomatology measure. To test the possibility that the retrospective ADHD measure was biasing the factor structure of the current symptomatology measures, the analysis was repeated without the items from the WURS.

The removal of all WURS items from the analysis did not affect the factor structure. The results revealed four factors, explaining 45.4% of the variance. Factor 1, *Current ADHD*, consisted of 17 out of the 18 AARS items; although AARS item 8, "*Have difficulty taking part in leisure activities*", did not primarily load onto this factor, it did indeed cross-load. Factor 2, named *Aggression*, contained all items from the AQ. Factor 3 was labelled *Anxiety*, and contained 6 items from the HADS anxiety scale, and 1 from the HADS depression scale. Factor 4, *Depression*, consisted of 5 HADS depression items, 1 HADS anxiety item, and 1 AARS item, "*Have difficulty taking part in leisure activities*". See Appendix 7 for full results.

A second order model was also created using scale scores. Total scores for AARS inattention, AARS hyperactivity/impulsivity, WURS, HADS depression, HADS anxiety, and AQ were entered for factor analysis. This produced a six-factor solution with each scale loading onto its own factor, mimicking the findings of the original model. Interestingly however, AARS inattention and AARS hyperactivity/impulsivity cross-loaded onto each other's factor, and the WURS scale score cross-loaded onto the AQ factor. For full results see Appendix 8.

Discussion

The results demonstrate that ADHD symptoms are a valid dimension in non-clinical adolescent populations. The overwhelming majority of ADHD symptoms (both current and retrospective) appeared in separate clusters to other forms of psychopathology: i.e. there was a current ADHD factor, a retrospective ADHD factor, an aggressive behaviours factor, an anxiety-dominated factor, and a depression-dominated factor. The results of the second order model confirm the existence of separate psychopathological dimensions, but also highlight the close link between inattention and hyperactivity/impulsivity, and – in line with the literature – suggests that ADHD and aggressive behaviours are co-existing dimensions (Biederman, Newcorn, & Sprich, 1991; Hinshaw, 1987).

The findings give support to the hypothesis that adolescent ADHD is a valid dimension in adolescence and not merely an expression (or "phenocopy") of symptoms of other co-occurring disorders (Spencer, Biederman, Wilens, & Faraone, 1994; Wilens *et al.*, 2003). While current ADHD symptom ratings were correlated with anxiety, depression and aggression, they loaded on a separate factor, suggesting that these symptoms are linked but ultimately separate constructs, i.e. comorbid (Bird, Gould, & Staghezza, 1993; Jarrett & Ollendick, 2008; Pliszka, 1998). There are several possible reasons why individuals with higher ADHD scores reported more symptoms in other domains. Firstly, as mentioned above, comorbidity: in the literature, ADHD has repeatedly been shown to be closely linked to these other symptoms; in this study those who rated highly for ADHD also rated highly for anxiety, depression, and aggression, however the factor analysis showed that these were conceptually independent dimensions. Secondly, risk: the impact of ADHD poses a high risk for developing other problems and symptoms (Dolpheide & Pliszka,

2009). Participants who reported high levels of childhood ADHD symptoms will have been at risk for developing anxiety, depression, and aggression later in life. Finally, acquiescence bias: it cannot be discounted that the high correlations arose because of acquiescence bias, which refers to the "propensity for respondents to agree (or disagree) with questionnaire items independent of their content" (Podsakoff, MacKenzie, Lee & Podsakoff, 2003, p. 882). However, it is likely that such bias would have led to a failure to find separate dimensions of psychopathology as participants would in effect be treating all items and all scales with the same acquiescence tendency: this was not found.

The finding of a single factor for current ADHD symptoms established in this study (and not two separate factors for inattention and hyperactivity/impulsivity) is at variance with previous research into the factor structure of clinical ADHD; admittedly, the second-order model showed inattention and hyperactivity/impulsivity loading onto separate factors, but there was mutual cross loading. Child studies have typically unearthed a bi-dimensional ADHD structure; indeed this is why DSM-IV states that ADHD can be separated into subtypes of inattention and hyperactivity/impulsivity. Attempts to test this structure in adult samples have produced mixed results, with some replicating the bi-dimensionality (Du Paul *et al.*, 2001; Spencer, Biederman, Wilens, & Faraone, 1998), and others finding a three-factor structure of inattention, hyperactivity, and impulsivity (Span, Earlywine, & Strybel, 2002). Other research has found that parent-reports of symptoms more reliably produce a two-factor solution than self-report (Glutting, Youngstrom, & Watkins, 2005).

Studies of this type in non-clinical, purely adolescent, samples are rare; however Rasmussen *et al.* (2002) compared the latent class models of parent-reported

and adolescent-reported ADHD symptoms, and found that the bi-dimensional structure in parent-report symptoms can be reproduced in adolescent-report symptoms. However they also found that parents tend to report different information about their child's ADHD symptoms compared to their offspring's self-report, with the adolescents being more likely to report combined-type symptoms. Perhaps the uni-dimensionality of current ADHD symptoms found in the present study could have roots in the participant's reporting style, as demonstrated by Rasmussen *et al.* (2002), with participants reporting inattentive and hyperactive/impulsive symptoms in equal measure.

The separate factors of ADHD and aggressive behaviours supports previous work which suggests that ADHD is correlated with externalising behaviour disorders, but with the constructs, however, forming unique latent clusters (Hinshaw, 1987; Pillow *et al.,* 1998). The present results also suggest that these findings can be applied to internalising behaviour disorders such as depression and anxiety.

Another interesting finding was the derivation by factor analysis of two (separate) ADHD factors, current and retrospective ADHD symptoms, and that these two measures were highly inter-correlated ($r = .7$). This could be due to a number of reasons. As the AARS and WURS are both measuring ADHD symptoms, it could be expected that they load on the same factor, and one concern could be that their factorial independence is due to an artefact of the measures' response format. However it was found that there were several cross loadings between the factors, which suggests there was some relationship, but ultimately the latent structure of the factors was intact. The formation of two separate factors could be an indication of the developmental nature of the disorder: as previously discussed, the ADHD symptom presentation changes with age, therefore it seems likely that participants rated

childhood ADHD experiences on the WURS, and adolescent ADHD-related behaviours on the AARS with genuine differentiation ("I used to be hyperactive, but less so now"). Hence in this study two different factors emerged. The stability of the factor structure once the retrospective items had been removed supports the notion that adolescent and childhood ADHD symptoms are distinguishable (though correlated) dimensions.

The study has several limitations of note. All scores in this study were based on self-report measures; no secondary symptom reports were acquired. Due to the differing ADHD structures established by parent- and self-report in previous studies (Glutting, Youngstrom, & Watkins, 2005; Rasmussen *et al.*, 2002), perhaps the results could have differed when looking at secondary symptom ratings. It is a moot point however whether a parental report is any more 'valid' than a self report; one could argue that both perspectives are complementary. Another possible limitation could be that the factor structure was due to the questionnaires' response format; for example, all AARS ADHD items loaded onto one factor, and all AQ aggression items loaded onto another. This could have been because they were from different measures, and not because of some underlying latent structure. However, given the existence of several cross loadings between the factors, this conclusion seems unlikely. Perhaps in future research, the use of multiple measures (and not only in ADHD) could test this assertion. It should be noted that, in the literature, most factor analytical studies of human characteristics use measures which employ different response formats. To have changed and standardised the response formats of the measures employed in this study would have exposed it to the reasonable criticism that these individual measures were no longer valid.

The findings have a number of implications for ADHD theory and treatment. Firstly, the finding that adolescent ADHD symptoms seem to be distinguishable from the overall structure of mental health symptoms, in a similar way to children, suggests adolescent ADHD symptomatology is a valid dimension worthy of attention. Secondly, the finding that the psychopathological profile usually associated with clinical cases of ADHD also exists in a non-clinical community sample suggests that ADHD symptoms lie on a continuum, and are not a dichotomous entity. Teachers and parents could be made aware that there are individuals who lie near to the diagnostic threshold and may experience an array of problems similar to those who are diagnosed, and that these individuals are likely to need support. Finally, the continuum finding can be utilised in treatment procedures, where sub-clinical individuals with typical ADHD-related functional impairments can be helped using adaptations of interventions normally reserved for clinical cases, such as peer tutoring for those with academic difficulties (DuPaul, Ervin, Hook, & McGoey 1998).

Chapter 5

Study 2

ADHD symptoms and academic performance in an adolescent community sample[2]

[2] This study formed the basis of an empirical paper submitted for publication in Journal of Adolescence: Birchwood, J. and Daley, D. (submitted). ADHD symptoms and academic performance in an adolescent community sample.

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Chapter 6

Study 3

Do response inhibition and delay aversion underpin the relationship between adolescent ADHD symptoms and academic attainment?

Abstract

Background: The role of the dual pathway model of ADHD in the relationship between ADHD and academic performance is unknown.

Aims: To investigate the role of response inhibition and delay aversion in the relationship between ADHD symptoms and academic performance.

Methods: 23 participants aged 15 and 16, of which two were diagnosed with ADHD, completed measures of ADHD, general cognitive ability, response inhibition, and delay aversion.

Results: Correlation analysis revealed that ADHD symptomatology was not correlated with response inhibition or delay aversion; response inhibition and not delay aversion was correlated with academic performance.

Conclusions: The underlying assumptions of the dual pathway model were not met. Sampling problems hindered the statistical power of the study, pointing toward the need of further research in the area.

Attention deficit hyperactivity disorder (ADHD) is characterised by elevated levels of inattention, hyperactivity, and impulsivity (APA, 1994). Neuropsychological research has identified several key features of the disorder, including impaired response inhibition (Barkely, 1997), deficits in working memory (Tannock, 1998), poor time perception (Smith, Taylor, Rogers, Newman, & Rubia, 2002), and increased intra-subject variability in neuropsychological test performance (Klein, Wendling, Huettner, Ruder, & Peper, 2006).

Over the past 15 years attempts have been made to identify a single core underlying process that underpins ADHD. Research has given rise to two theories: (a) ADHD is a disorder of cognitive dysregulation (Schachar *et al.*, 2000); and (b) ADHD is an altered motivational style (Sonuga-Barke *et al.*, 1996).

Cognitive dysregulation, motivational style, and the dual pathway model

The cognitive dysregulation theory of ADHD (Schachar *et al.*, 2000) suggests that the ADHD individual experiences problems with executive functions such as planning, working memory, and response inhibition (Barkley, 1997; Pennington & Ozonoff, 1996). The core deficit is impaired inhibitory control, which is said to underlie symptomatology (Barkley, 1997). Most support for this theory comes from research employing the stop signal paradigm (SSP) (Schachar, Mota, Logan, Tannock, & Klim 2000), which tests the ADHD individual's ability to inhibit a pre-programmed response. In computerised versions of the stop signal task (SST), the participant is asked to respond to an on-screen stimulus (the "go signal"), by pressing a button, which corresponds to the stimulus. On certain random trials, the participant hears a "beep" (the "stop signal"), which is the signal to *not* respond to the on-screen stimulus. The "stop signal" is presented *after* the "go signal" at varying intervals prior

to the participant's expected response to the "go signal". The stop signal reaction time (SSRT) – the speed of the participant's stopping process – provides a measure of the efficiency of inhibition; ADHD individuals typically have a longer mean SSRT (Schachar, Tannock, Marriott, & Logan, 1995), i.e. they take longer to stop upon hearing the "stop signal" thus making more mistakes.

Research has shown that not all ADHD individuals display EF deficits (Solanto *et al.*, 2001). An alternative theory has been proposed that characterises ADHD behaviours as a manifestation of an underlying motivational style, where the individual is motivated to actively avoid delay (Sagvolden, *et al.*, 1998; Sonuga-Barke *et al.*, 1996). Support for this hypothesis typically comes from studies adopting the delay aversion paradigm, which is tested using the choice-delay task (CDT) (Sonuga-Barke, Taylor, Sembi, & Smith, 1992). Here the participant is typically asked to choose between an immediate small reward, and a delayed large reward, and is given a limited number of trials in which to choose; ADHD children will tend to opt for the small reward, suggesting a preference for less delay and an overall shorter session length (Sonuga-Barke *et al.*, 1992).

In a "head-to-head" study, Solanto *et al.* (2001) brought the two opposing theories together and compared the ecological validity of the SST and CDT as effective utilities in discriminating ADHD and non-ADHD individuals. It was found that (a) performance on the two tasks was not correlated, (b) there was an effect of ADHD on task performance, and (c) the two measures were highly diagnostic of ADHD cases. These findings led to the proposal of a "dual pathway" model of ADHD (Sonuga-Barke, 2002), in which ADHD is *either* the result of cognitive dysregulation (resulting from impaired inhibitory control), *or* is the manifestation of an altered motivational style (characterised by delay aversion).

ADHD, underlying processes, and academic performance

The link between ADHD and impaired academic attainment has been demonstrated across the developmental spectrum, in preschoolers (DuPaul *et al.,* 2001; Lahey *et al.,* 1998; Mariani & Barkley, 1997), children (Barry, Lyman, & Klinger, 2002; Loe & Feldman, 2007), adolescents (Barkley *et al.,* 1991; Biederman *et al.,* 1998; Frazier *et al.,* 2007), and young adults (Heiligenstein *et al.,* 1999; Reaser *et al.,* 2007). The relationship between ADHD and academic impairment has been established in both clinical (Barry, Lyman, & Klinger, 2002) and community-based samples (Rodriguez *et al.,* 2007). Many investigations into the relationship between ADHD and academic underperformance have focused on the impact of the core behavioural symptoms of inattention, hyperactivity and impulsivity (Merrell & Tymms, 2001; Rodriguez *et al.,* 2007) and have discounted any possible role played by comorbid problems such as conduct disorder (Frick *et al.,* 1991; Rapport, Scanlan, & Denney, 1999).

Research into the link between the dual pathway model and academic performance is sparse, however there have been investigations into the role played by EF deficits in the relationship between ADHD and academic performance. Biederman *et al.* (2004) found that children with ADHD and impaired EFs performed worse on tests of academic achievement than children with ADHD and adequate executive functioning. Biederman *et al.* (2004) also showed that impaired executive functioning in controls was unrelated to academic achievement, suggesting an interaction between ADHD, executive functioning, and academic performance. Diamantopoulou *et al.* (2007) found that ADHD symptoms and poor executive functioning independently predicted poor school performance, however an interaction was only evident when ADHD was broken down into its subtypes: high levels of inattention along with deficits in executive functioning predicted higher levels of special educational needs.

To-date, only one study has been published on the relative impact of impaired EFs and delay aversion on academic performance. Thorell (2007) tested 145 preschoolers on age-appropriate measures of EF (tests of inhibition and working memory), delay aversion and early academic skills (specifically mathematics and language abilities). The author found that there was a link between delay aversion and symptoms of hyperactivity/impulsivity, but neither was linked with early academic skills. However, there were significant correlations between EFs and symptoms of inattention, and *both* were correlated with mathematics and language ability. Furthermore, mediation analysis revealed that EFs were shown to mediate the relationship between inattention and early academic skills.

Although research suggests that EFs could be at the heart of the link between ADHD and academic performance, the role played by delay aversion is yet to be studied in-depth. Whilst delay aversion has been discounted in a preschool sample (Thorell, 2007), the role of the two processes of the dual pathway model in academic attainment needs to be studied in child and adolescent populations.

The present study

In light of the above, the present study aimed to investigate the role played by the dual pathway model (Sonuga-Barke, 2002) in the relationship between ADHD symptoms and academic performance, in a community-based adolescent sample.

Based on the "head-to-head" study by Solanto *et al.* (2001), the stop signal task (SST) and choice delay task (CDT) were chosen as key candidate measures of underlying EF deficits (deficient inhibitory control) and altered motivational style. As a measure of academic performance, it was decided to use participants' GCSE (General Certificate of Secondary Education) grades, in order to gain a representation

of the participants' real-life academic attainment. Most previous studies into ADHD and academic achievement employ standardised tests of achievement, such as the Wide Range Achievement Test (WRAT; Jastak & Jastak, 1985), as used by Biederman *et al.* (1998). Whilst such measures are statistically proven to reflect a child's academic ability, it was decided to take a measure of the child's *actual* academic attainment, and not their performance in a one-off test. Also it was decided that ADHD symptomatology would be represented by a composite score consisting of an aggregation of current and retrospective symptoms, capturing the developmental nature of the disorder.

Method

Participants

Ethical approval for the study was granted by the Ethics and Research Governance Committee, School of Psychology Bangor University. Participants were recruited by two methods: (a) through two high schools in the West Midlands and North Wales (n = 11), and (b) via an opportunity sampling method (n = 12). Consent was obtained via parents (using "opt-in" consent forms several weeks before the study) and pupils (immediately before the study began). Out of 84 high school pupils approached, 11 agreed to participate; all 12 adolescents approached via opportunity sampling agreed to take part. Therefore out of a total of 96 approached, 23 individuals agreed to participate in the study. The participants were aged 15 and 16, of which 6 were female and 17 male. Two participants recruited via opportunity sampling were diagnosed with ADHD; one was on a course of ADHD treatment medication, one was not.

Measures

Current symptomatology: The Adult ADHD Rating Scale (AARS; Barkley & Murphy, 1998): the AARS is a self-report scale consisting of 18 items based on the DSM-IV symptom list, including inattentiveness (9 items), impulsiveness (3 items) and hyperactivity (6 items). Participants are asked to rate their own behaviour over the past 6 months on a 4-point scale (0 = *rarely*; 1 = *sometimes*; 2 = *often*; 3 = *very often*). Edwards *et al.* (2001) report Cronbach's alpha coefficients of 0.92 for the inattention items, and 0.91 for the hyperactive/impulsive items. Cronbach's alpha coefficients for the present sample are 0.81 for the inattention items, and 0.84 for the hyperactive/impulsive items. In the present sample, participants' total AARS scores ranged from 3 to 37. This measure has previously been used in studies employing older adolescent/young adult samples (DuPaul *et al.*, 2001).

Retrospective symptomatology: The Wender Utah Rating Scale (WURS; Ward, Wender, & Reimherr, 1993): this is a 25-item questionnaire that can be used to retrospectively yield estimated scores for individuals' ADHD symptoms during childhood. Participants are asked to rate their childhood behaviour on a 5-point scale (1 = *Not at all/slightly*; 2 = *Mildly*; 3 = *Moderately*; 4 = *Quite a bit*; 5 = *Very much*). Ward *et al.* (1993) found that scores on the WURS correlate significantly with retrospective reports of childhood symptoms by parents. The measure has excellent internal consistency and good test-retest stability (Ward, Wender, & Reimherr, 1993). Cronbach's alpha for the current sample is 0.96. In the present sample, participants' total WURS scores ranged from 29 to 115.

Cognitive ability. This was measured using the Raven's Standard Progressive Matrices (RSPM) cognitive test (Raven, Raven, & Court, 1998; Raven, Raven, & Court, 2000). This is one of the most well-known, and widely used intelligence tests, and was designed to measure Spearman's "g", the general factor of intelligence. The measure has excellent reliability and validity (Raven, Raven, & Court, 2000) and associations with full-scale IQ scores are high (Raven, Raven, & Court, 2000). In the present sample, participants' RSPM scores ranged from 25 to 54.

Inhibitory control: Stop Signal Task (SST; Logan, 1994). To measure inhibitory control, a computerised version of the SST, similar to that used in Solanto *et al.,* (2001) was used. The task was programmed using E-Prime software. The task required approximately 20 minutes to complete, and consisted of 6 blocks of 32 trials, with block 1 considered a practice trial. In each block, either an "O" or an "X" appeared briefly in the middle of the computer screen. The participant was instructed to press the correspondingly labelled mouse button as quickly as possible when each letter appeared. In 8 of the trials in each block, an auditory stop signal was presented through the computer speakers; this was the cue for the participant to refrain from responding to the primary ("O" or "X") stimulus (the "go signal"). The stop signal was presented 250ms after the go signal; this delay was adjusted by 50ms increments with every successful (delay shortened) and unsuccessful (delay lengthened) inhibition. Block 1 was considered a practice trial as the delay was reset to 250ms at the end of the block. Subsequently, Blocks 2-6 were entered into the analysis.

To measure inhibition, participants' stop signal reaction time (SSRT) was taken. ADHD individuals tend to record longer SSRTs than non-ADHD individuals (Schachar, *et al.,* 1995). In the present sample, participants' SSRTs ranged from

162.65ms to 577.24ms. The stop signal reaction time test is one of the most widely used neuropsychological tests of inhibitory control, and has been widely used in research studies with young and older children (Soreni, Crosbie, Ickowicz, & Schachar, 2009) and young adults (MacLaren, Taukulis, & Best 2007).

Delay aversion: Choice-Delay Task (CDT; Sonuga-Barke *et al.,* 1992): A computerised CDT was used, which was similar to the task used in Solanto *et al.* (2001). The task required approximately 20 minutes to complete. The participant used the mouse to choose between two on-screen squares, which were presented side-by-side; one was labelled "1 Point" and the other labelled "2 Points". Participants were instructed that they were about to play a game in which they could earn points that could be exchanged for 5 pence each (i.e. 1 point = 5 pence). Participants were told that after clicking "1 Point" there would be a small delay, and after clicking "2 Points" there would be a large delay, and that they would be permitted 20 trials (i.e. 20 clicks) with no time limit.

After selecting 1 point, there was a delay of 2 seconds; after selecting 2 points there was a delay of 60 seconds. The 2-point delay was set at 60 seconds in order to avoid ceiling effects with an adolescent sample; previous research using childhood samples has used a 30 second 2-point delay (Sonuga-Barke *et al.,* 1992; Solanto *et al.,* 2001).

In the analysis, delay aversion was represented by the percentage of small, immediate choices made by the participant, with a high score representing greater delay aversion tendencies. This was termed delay aversion (DA) score. In the present sample, participants' DA scores ranged from 0% to 95%. The choice delay task requires a simple choice between small immediate or larger later rewards. It is usually

considered to be free from developmental constraints, and is widely used with preschool children, and school aged children (Spronk, Jonkman, & Kemner, 2008). A recent study by Marco *et al.* (2009) examined the role of delay aversion as measured by the Choice Delay task in 360 ADHD probands, 349 siblings and 112 controls; here the participants were aged 6 to 17 years.

Academic performance. This was measured using pupils' GCSE (General Certificate of Secondary Education) results. GCSEs reflect two years of an adolescent's schoolwork, assessed by coursework and examinations (both modular and final).

For participants recruited via schools, this measure was not collected from participants, but directly from the school concerned, with the participant's permission.

Participants recruited via opportunity sampling were contacted directly by the research team to collect their results.

The participants' grades were transformed into corresponding points scores (using the official GCSE points system) (Appendix 9), which were added up to produce an overall academic performance score. In the present study, participants' GCSE points scores ranged from 0 to 778.

Procedure

Recruited through schools: Parents of pupils were sent study information sheets (Appendix 2) and consent forms (Appendix 3); parents were asked to respond stating whether or not they wished for their child to participate. Immediately before the study, pupils were asked to read a study information sheet (Appendix 2) and complete a consent form (Appendix 3), where they were asked for permission for their GCSE grades to be released to the research team. Participants were then asked to complete

the measures of ADHD and the RSPM. Afterwards, they were invited to a second testing session, held at a later date, where they completed the SST and the CDT. GCSE results were obtained from the schools several weeks after they had been released to individual pupils.

Recruited via opportunity sampling: Participants were recruited through a snowballing technique and through community-based ADHD support groups (to increase the numbers of high-ADHD scorers in the sample). The testing procedure was as above, except these participants completed the study in their own homes, which was arranged verbally with parents. At the beginning of the testing session, parents and participants read the information sheet (Appendix 2) and completed the consent form (Appendix 3). The participants then completed the ADHD measures, RSPM, SST, and CDT in one session. Participants were later contacted by telephone in order to collect their GCSE results.

Results

Data preparation/analytical strategy

Results of a 1-sample Kolmogorov-Smirnov test indicated that the data were parametric (with K-S Z values ranging from .467 to 1.053 and p values ranging from .217 to .981) therefore a Pearson's correlation was used to examine relationships between the variables. Kruskal-Wallis tests were also used to examine the data; this test was used because several of the assumptions for analysis of variance (ANOVA) were violated.

ADHD versus non-ADHD

Not all of the assumptions of analysis of variance were met therefore Kruskal-Wallis tests were carried out to explore the difference in scores on the key variables between those scoring above (n = 6) and below (n = 17) the DSM-IV ADHD clinical cut-off (the above cut-off group included combined-type, inattentive sub-type, and hyperactive/impulsive sub-type scorers). There were no significant differences between the groups in GCSE score (*Chi square* = 2.593; *df* = 1; *p* = .107), stop signal reaction time (SSRT) (*Chi square* = .176; *df* = 1; *p* = .674), or delay aversion (DA) score (*Chi square* = .061; *df* = 1; *p* = .805).

Effect size calculations were also carried out to examine differences between the two groups; the means and standard deviations are displayed in Table 5. Table 5 shows that the difference in GCSE score between the groups was meaningful, with Cohen's *d* = 0.69. A power calculation revealed that a sample size of n = 45 per group would be needed to test for a significant difference between the groups at the .05 level with 90% power.

Table 8: Descriptive statistics and effect sizes (Cohen's *d*) comparing those scoring above (n = 6) and below (n = 17) the ADHD cut-off on GCSE score, SSRT, and DA score

Measure	Above/below ADHD cut-off	Mean	SD	Cohen's *d*
GCSE points score	Above	248.00	149.76	**0.69**
	Below	360.82	176.22	
SSRT (ms)	Above	262.00	82.24	0.11
	Below	252.53	95.32	
DA score (%)	Above	52.50	22.74	0.08
	Below	54.71	30.28	

Correlations between variables

A Pearson correlation analysis was administered to explore the relationships between the variables. The results are displayed in Table 6. As would be expected, there were strong positive correlations between the scores derived from the ADHD self-report scales – AARS total score, AARS inattention score, AARS hyperactivity score, and WURS. Of these measures, AARS inattention score and WURS score were correlated with GCSE performance, with moderate negative associations in both cases. Participants' SSRT was not correlated with any of the ADHD measures, however moderate negative correlations were present between SSRT and GCSE performance, and SSRT and cognitive ability. Cognitive ability was also correlated with total AARS score, inattentive AARS score, and GCSE score. DA score did not correlate with any other variable.

Table 9: Pearson correlations between variables

Measure	AARS inattention	AARS hyp/imp	WURS	Raven's score	SSRT	DA score	GCSE score
AARS total	**.90****	**.92****	**.81****	**-.46***	.14	.08	-.34
AARS inattention		**.65****	**.78****	**-.44***	.17	.17	**-.42***
AARS hyp/imp			**.70****	-.39	.09	-.02	-.20
WURS	-.40	.24		.20			**-.49***
Raven's score			**-.42***	-.40			**.51***
SSRT				.41			**-.47***
DA score							-.25

NB. AARS hyp/imp = AARS hyperactivity/impulsivity; * = $p < .05$; ** = $p < .01$

Mediation Model

To explore whether SSRT or DA score mediated the relationship between ADHD symptomatology and academic performance, the rules of Baron and Kenny's (1986) mediation model were applied to the data. However, the lack of a significant correlation between either SSRT or DA score and ADHD symptomatology (either total AARS, inattentive AARS, hyperactive/impulsive AARS, or WURS) renders the proposed mediational model moot.

Discussion

The present study aimed to investigate the role played by the two processes of the dual pathway model of ADHD (Sonuga-Barke, 2002) in the relationship between ADHD symptoms and academic performance. It was found that ADHD-inattentive symptomatology and SSRT (but not DA score) were negatively correlated with

academic achievement, however neither SSRT nor DA score was correlated with ADHD symptomatology, which violated Baron and Kenny's (1986) terms for successful mediation. Therefore, in the present study, neither deficient inhibitory control nor delay aversion mediated the relationship between ADHD symptoms and academic performance.

However it should be noted that the sample was greatly under-powered, therefore the validity of the results could be called into question. These sampling issues will be addressed later in this section.

The results of the present study do not support previous research into neuropsychological underpinnings of ADHD. The dual pathway model of ADHD (Sonuga-Barke, 2002) suggests that delay aversion and cognitive dysregulation (EF deficits) are parallel, but distinct, pathways to ADHD; research has shown that ADHD individuals are likely to either experience EF impairments or be delay averse (Solanto *et al.*, 2001; Sonuga-Barke 2002). In the present study it was found that (a) SST and DA score were moderately correlated (however this was not significant), and (b) SST and DA score were not correlated with ADHD symptomatology. These findings do not support the dual pathway model.

There could be several possible reasons for the discrepancy between the present findings and previous research. One explanation could simply be that as this was not a purely clinical sample, the sample size was not large enough to enable a full variance in scores, to in-turn produce the expected results. Sample size issues are discussed below. Also it should be noted that there were two ADHD-diagnosed individuals who participated in the study: one had not taken medication for several months and the other had not taken medication on the day of testing. As such it is unlikely that their performance will have been "normalised" because of medication.

Another reason could be that the present study employed a community-based sample, but used the SST and CDT, which are tools designed to be used with clinical cases (where there are a narrow range of scores), to examine underlying processes of individuals with a diagnosis of ADHD (such as in Solanto *et al.*, 2001). It could be argued that SST and CDT performance may not be linked to sub-clinical ADHD symptomatology. However this notion is countered by the results of Sonuga-Barke *et al.* (2003) and Thorell (2007) who found that cognitive dysregulation and motivational style were independently linked to ADHD symptoms in community-based preschool samples. They found a linear relationship between underlying psychological processes and ADHD symptomatology, suggesting that the CDT and SST underlie ADHD symptoms across the continuum. In a recent study, Paloyelis, Asherson and Kuntsi (2009) provided evidence of a link between performance on the choice delay task and ADHD symptoms in a community sample of 1,062 children. They found that symptoms of inattention uniquely predicted a preference for small, immediate rewards, suggesting that performance on the CDT is linked to sub-clinical symptomatology. However as Sonuga-Barke *et al.* (2003), Thorell (2007), and Paloyelis, Asherson and Kuntsi (2009) studied preschool and child samples, these results cannot necessarily be applied to adolescents.

The choice of computer tasks is also cause for discussion. Perhaps the CDT was not stimulating enough for this particular age group. Whilst carrying out this task the participants seemed bored and disengaged; this raises the possibility that participants were likely to select small, immediate rewards to end the task faster, to alleviate boredom. Performance may not have been a function of their ADHD symptomatology, but more their relative boredom. A more relevant and stimulating task could have been similar to the Delay Frustration Task (another common index of

delay aversion), as used in Bitzakou, Psychogiou, Thompson, and Sonuga-Barke (2008). Here, participants were asked to complete a computer-based mathematics task. This involved pressing a button to go onto the next question, however the program had an in-built delay between certain questions, and would measure how many button presses the participant would make during this delay. Such problems with developmental appropriateness should not have been a problem with the SSRT, which more directly taps into a temporal process, which is independent of developmental level.

Whatever the reason for the discrepancy between the present results and previous research, more work needs to be carried out in order to fully examine the role of underlying psychological processes across the developmental spectrum.

Sample size issues

The intention of the present study was to examine the role of the dual pathway model in the relationship between ADHD and academic performance in a large community-based adolescent sample. However due to consent-process constraints, the sample was severely under-sized. As the experimenter would be alone with participants to administer the SST and CDT, ethical guidelines dictated that parents' consent had to be obtained via an *opt-in*, rather than *opt-out* method; i.e. full informed parental consent was required. Unfortunately this resulted in an extremely low number of parents returning consent forms. Previous studies employing an *opt-out* method, where parents were asked to return consent forms only if they did not wish for their child to take part (for example in Chapter 5 of this thesis), resulted in large sample sizes.

The results showed that those scoring above the DSM-IV cut-off did not differ significantly with those who scored below the cut off on GCSE performance, SSRT, and CDT performance. However descriptive statistics and effect size calculations revealed that the difference in GCSE score was meaningful. With a much larger sample size perhaps the differences between the groups would be significant. Similarly, it is likely that the small sample size may have been at the root of the failure to meet mediation criteria. As the sample was small in size there may not have been enough opportunity for full variance in SSRT and DA score, thus decreasing the likelihood of a true reflection of the role that these factors play in the relationship between ADHD and academic performance across the ADHD continuum. Perhaps a large increase in sample size would increase the likelihood of significant correlations between ADHD and the two pathways of the dual pathway model.

Implications and future research

The low statistical power of the present study scrutinises the external validity of the findings. However, despite the disappointing results with regard to the dual pathway model, one finding does stand out: even in a severely underpowered community-based study, the correlation between ADHD symptoms and academic performance held true, and effect size calculations revealed meaningful GCSE score differences between high- and low-ADHD scorers. Therefore even at sub-clinical levels, adolescent ADHD symptoms are strongly inversely associated with academic success. This promotes the need for a heightened awareness of adolescent ADHD symptoms in the classroom and at home, which will enable undiagnosed individuals who express symptoms to receive necessary academic support.

In terms of the underpinnings of the relationship between ADHD symptoms and academic performance, few inferences can be made based on the present study. However, this study and indeed Thorell (2007) have opened the door to a body of research into the dual pathway model and academic performance. If cognitive dysregulation impacts more severely on academic attainment than an altered motivational style does, then why? Sonuga-Barke (2002) suggested that the delay averse have intact cognitive control systems, therefore perhaps they are able to develop effective cognitive strategies to help cope with their condition, which in-turn leads to unimpaired academic performance. Both clinical and community-based research should be carried out to investigate the role of the two processes of the dual pathway model in academic attainment, across the developmental spectrum, to unearth the reasons why ADHD is associated with impaired academic performance.

Chapter 7

General Discussion

Summary of main thesis findings

Overview of main objectives

The aim of this thesis was to examine the dimensional structure of adolescent ADHD symptoms and the impact of this symptomatology on academic attainment. Within this general aim, there were several specific objectives. Due to the lack of previous research using community-based, adolescent samples, it was decided to examine the relationship between ADHD symptoms and academic performance in a cross-sectional sample of UK adolescents who were near to undertaking crucial state examinations. In light of this, a further aim was to test the validity of the adolescent dimension of ADHD, and to examine whether ADHD symptoms operate in a community sample in a similar way to clinical cases, in terms of being distinguishable from, and not the result of, symptoms of co-morbid syndromes. The final aim was to explore the role played by delay aversion and deficient inhibitory control in the relationship between ADHD symptoms and academic performance.

Chapter 2: Literature review

In Chapter 2 a review of research into the relationship between ADHD and academic performance was presented. This began with an examination of the relationship in pre-school, childhood, adolescence, and adulthood; cross-sectional and longitudinal studies were discussed. Next, there was an examination of the reasons behind the relationship between ADHD and academic underperformance, which unearthed a possible *inattention–EF impairment* pathway to academic problems in ADHD individuals. The chapter ended with an overview of interventions that are directed towards addressing the academic impairment of ADHD individuals. A revised version

of this chapter has been accepted for publication in *Child: Care, Health and Development* and is appended (Appendix 1).

Chapter 4: Empirical Study 1

The validity of an adolescent ADHD dimension in a community sample was examined using a large cross sectional sample of adolescents taken from four schools in Wales and England. Each participant completed measures of current ADHD, retrospective ADHD, anxiety, depression, and aggression. A simple correlation analysis revealed that both current and retrospective ADHD symptom ratings were correlated with anxiety, depression and aggression. Next, the item scores from the measures were entered for factor analysis. Results revealed a five-factor solution, with the following factors: (a) *Retrospective ADHD*, (b) *Current ADHD*, (c) *Aggression*, (d) *Depression*, and (e) *Anxiety*. Several conceptually overlapping items were identified and removed, and the analysis repeated, but the five-factor solution remained intact. The analysis was repeated again but without the items from the retrospective measure of ADHD; results revealed a four-factor solution of *Current ADHD*, *Aggression*, *Depression*, and *Anxiety*.

The finding that ADHD symptoms loaded independently to anxiety, depression, and aggression, and that ADHD is nevertheless correlated with these other conditions, suggests that they are linked, but ultimately distinct, constructs. This provided support for the validity of the adolescent ADHD dimension. The results were established in a community sample, supporting the notion that ADHD is not a dichotomous entity, but a continuous characteristic in young people.

Chapter 5: Empirical Study 2

The relationship between adolescent ADHD symptoms and academic performance has rarely been studied in a community sample; therefore this was the aim of *Study 2*. This was carried out using a large cross-sectional sample of UK adolescents recruited from four schools in England and Wales who were followed up to ascertain GCSE performance. The predictive power of ADHD symptoms over GCSE (General Certificate of Secondary Education) performance was examined, alongside the respective predictive power of anxiety, depression, aggression, school-oriented motivation, and general cognitive ability. The separate dimensions of ADHD, anxiety, depression, and aggression established in *Study 1* supported the feasibility of examining their relative impact on academic performance.

The results of multiple linear regression analyses showed that lifetime ADHD symptoms (current symptoms and retrospective symptoms) were a strong predictor of GCSE performance indices (GCSE points score, number of GCSE entries, and weighted GCSE score). Of the ADHD subtypes, only inattention exerted a significant impact on GCSE performance (specifically, GCSE points score). Lifetime ADHD symptomatology and inattention was behind only general cognitive ability and motivation in predictive power. This suggests that individuals who express ADHD symptoms are likely to experience poorer academic outcomes, and that ADHD symptoms are almost as important a factor in understanding individual variations in academic performance as general cognitive ability and motivation, which are well validated predictors themselves. The results were established in a non-clinical sample, supporting the continuum theory of ADHD, and supporting the proposition that there are impaired individuals who would not attract a traditional diagnosis (Angold *et al.*, 1999). Of particular note was the prediction by lifetime ADHD symptoms of a "gold

standard" measure of academic performance, total GCSE points score – which is a gateway to further and higher education.

Chapter 6: Empirical Study 3

Research into ADHD has unearthed two key underlying mutually exclusive features of the disorder: deficient inhibitory control (Barkley, 1997; Schachar *et al.,* 2000), and delay aversion (Sonuga-Barke *et al.,* 1996). The role of these processes in the relationship between ADHD and academic performance has only been studied once before, in preschoolers (Thorell, 2007). A small sample of 23 adolescents was asked to complete measures of ADHD, inhibition, delay aversion, and general cognitive ability. Analysis revealed that neither delay aversion nor inhibition was correlated with ADHD symptomatology, providing no basis for mediation analysis (Baron & Kenny, 1986); however, ADHD symptomatology and inhibition, along with cognitive ability, were correlated with GCSE performance.

On face value, the results suggest that delay aversion and response inhibition play no role in the relationship between ADHD and academic performance. However, the external validity of the results was significantly impaired by the small sample size. No relationship was found between ADHD symptomatology and either delay aversion or response inhibition – a finding that is directly in opposition to the literature (Solanto *et al.,* 2001; Thorell, 2007). Therefore the key underlying premise for the study was not established, calling into question the results of the analysis. With a significant increase in sample size there would be more scope for concrete inferences to be made from the results.

Although the results of *Study 3* are handicapped by the sample size, collectively the three empirical studies provide evidence to suggest that (a) non-clinical adolescent ADHD symptoms are a valid dimension, (b) adolescents who report ADHD symptoms (but do not necessarily have an ADHD diagnosis) are likely to encounter significant academic impairment, and (c) ADHD symptoms are almost as important as general cognitive ability and motivation in explaining variance in academic performance.

Methodological Considerations

There are several methodological issues that should be considered when interpreting the results of the papers in this thesis. Firstly, in each study psychopathology ratings were based on self-report measure only. Research has shown that individuals are likely to show themselves in a favourable light when completing self-report measures (King & Bruner, 2000; Van de Mortel, 2008) and there may therefore have been under-reporting. There is an argument that secondary psychopathology ratings made by teachers, parents, or peers may have helped. However in relation to anxiety, depression and some aspects of ADHD, these are quintessentially subjective characteristics that are not always revealed in behaviour. Using observer ratings would have equally been subject to the criticism that these have just as many if not more problems of validity than self-report. Nevertheless secondary rating of ADHD symptoms would have been interesting. However, it should be noted that the AARS is merely a re-wording of the DSM-IV checklist; oriented toward individuals in late adolescence and over. Although the concept of using secondary informant ratings would be potentially advantageous to studies of this nature, in reality the logistics of asking parents or teachers to complete the measures would have compromised the

achievement of a sufficient sample size, unless they were collected on a randomly selected sub-sample of participants.

As well as a favourable ratings bias, the questionnaire used in this study was at risk of acquiescence bias; this refers to participants' propensity to agree or disagree with questionnaire items regardless of their content (Podsakoff, MacKenzie, Lee & Podsakoff, 2003). As such it is possible that the results could be due to this bias, and not to actual relationships between the constructs. For example this could have been the case when strong correlations were found between ADHD, anxiety, depression, and aggression. However there are several reasons to suggest that acquiescence bias can be ruled out. Firstly, participants do not respond to the items in the HADS on a likert scale: participants are asked to endorse one out of four statements, where the "high-anxiety" or "high-depression" answers can appear either first or last in the list of statements; if participants slipped into a rhythm of always selecting the first or last statement (as would be in acquiescent responding), then it is unlikely that the results of the research (comparable prevalence rates and correlations) will have been established. Another reason is that the questionnaire also contained a measure of motivation, in which the items were worded positively and negatively; if participants constantly selected the same rating for each statement then it is unlikely that positive, expected results will have been established. However, it was found that motivation was highly correlated with academic performance; this expected result is not likely to have materialised if acquiescent responding were widespread. Furthermore, referring back to the factor analytic analyses in Chapter 4, if acquiescent responding accounted for the majority of variance across measures, then the factor analysis would not have differentiated *between* the various scales. In other words, the finding that dimensions of current ADHD and aggression were differentiated in the factor analysis would not

have been observed had participants been responding mainly or solely on an acquiescent basis; clearly participants were thinking carefully when responding to the items in each scale.

Although it is likely that rater biases such as acquiescence did not affect the findings, there are procedures which could have been adopted to help protect the validity of the findings. For example, some items in the AARS and AQ could have been reversed; however this would have created a non-standard measure and raised concerns about reliability and validity. Alternatively, the items in the questionnaire could have been counterbalanced: there could have been several versions of the questionnaire, with the items appearing in a different order in each one, thereby counteracting possible order effects.

Overall, then, the ratings on each scale are valid because: (a) they are predominantly and quintessentially subjective characteristics for which there is no definitive final arbiter of validity, and (b) the differentiation achieved on the factor analysis between the scales demonstrates that participants were responding according to these 'internal dimensions of experience' and not, for example, a generalised response bias.

Another issue is the validity of the academic ability measure (GCSE); an alternative could have used teacher report, for example, rather than GCSEs. Teachers have regular contact with pupils, and would be able to provide first-hand indications of their ability. However, GCSEs are the principal academic focus of 16 year olds, and all class-work completed in Years 10 and 11 is directed towards GCSE achievement. They are independent and 'blind' to individual educational or special needs status. By using GCSE performance as the dependent variable, the results of

this study have relevant, externally valid, implications and will be taken more seriously as a result.

This study was conducted in community, not clinical samples to test hypotheses about continuum aspects and their link with continuum of performance ('impairment'). Whilst there are clear benefits to employing community samples (Angold *et al.*, 1999), the results cannot necessarily be extrapolated to individuals with a diagnosis of ADHD. For example, perhaps at extreme levels of ADHD symptomatology, cognitive ability and motivation might "play second fiddle" to ADHD in terms of accounting for variance in academic attainment. In addition, an ADHD diagnosis is typically accompanied by several other comorbid diagnoses (Biederman, 1998; Bird, Gould, & Staghezza, 1993; Pliszka, 1998), therefore the impact of these additional problems on school adjustment could exacerbate the academic problems experienced by these individuals. However, the link between clinical diagnosis of ADHD and attainment is a robust finding. The use of a community sample in *Chapter 6* was impaired because of the small sample size. Here, perhaps a clinical sample of this size, or a much larger community sample may have produced more positive results.

There are other limitations of note that are specific to each study. In the case of *Study 1* the reader should be made aware that the factor structure could be attributed to the differing response formats in each measure, and not to underlying latent psychopathological structures. However the cross loadings of several items between factors suggests that this is not likely to be the case, and thus the factor structure was indeed a representation of the psychopathological latent structure. Also, *exploratory* – not *confirmatory* – factor analysis was used. Confirmatory factor analysis would allow a more direct hypothesis examination, i.e. an examination of a

possible relationship between the observed variables and their underlying latent constructs. However, as this was the first study of its kind using an adolescent sample, there was no concrete *a priori* theoretical framework on which to structure a possible confirmatory factor analysis, therefore exploratory factor analysis was employed.

Also, in *Study 1* the possibility of item overlap may have led to spurious correlations and factor analysis results. However as indicated in *Chapter 4*, preliminary analysis revealed that there was no statistical overlap between the items entered for factor analysis. Field (2000) suggests that items should be removed if they correlate with other items very highly, with a coefficient of above 0.9; this was not the case in the present data set and therefore the analysis was carried out with all items included. The results revealed several cross-loadings of items between the *Retrospective ADHD, Anxiety*, and *Depression* factors; under close inspection it seemed that the items from the WURS were conceptually very similar to some items from the HADS anxiety, HADS depression, and AQ scales. It seemed that perhaps the WURS did not only tap into retrospective ADHD symptoms, but also dysfunctional personality traits – observation has been supported elsewhere (Hill, Pella, Singh, Jones, & Gouvier, 2009). It was decided therefore to test this by removing several WURS items from the factor analysis. After re-analysis, the five-factor structure remained intact, suggesting that item overlap did not account for the underlying structure of the data. Perhaps in future research an alternative method of identifying overlapping items would be to gather a panel of experts and ask them to assess whether there are overlapping items (see Lemery, Essex, & Smider, 2002); this would provide comprehensive, objective opinion, and is an approach adopted in other areas examining item overlap. Lemery, Essex, and Smider (2002) suggested that the advantage of using expert opinions was their ability to differentiate behaviour.

In *Study* 2 the regression analyses examined only main effects of predictor variables; interactions between variables were not examined. Perhaps interactions between the variables could have explained more of the variation in academic performance: it was found that aggression, anxiety, and depression did not predict GCSE performance, however perhaps interactive effects between the variables would significantly impact GCSE performance; for example depression in the context of ADHD might have been a toxic mix. However this was not the hypothesis under test, and would require a secondary interrogation of the database. To do this would be particularly demanding of statistical power as there are at least five interaction terms and many more third order interactions, in addition to the main effect variables. The analytical approach used here was based on *a priori* research questions whereas second and third order interactions would be post-hoc and would require the equivalent of a bonferonni correction. Also, there were several other variables that could have been controlled for in the analysis as well as the participants' school, such as socio-economic status, ethnicity, and even parental psychopathology. Further research may plausibly examine the impact of confounding variables; however, the aim of this study was first to examine whether there *is* a link between adolescent ADHD symptoms and academic performance, which was found. The next stage would need then to test for plausible confounders. This is an approach common in much psychological and psychiatric research. A clear rationale needs to be put together as to why socioeconomic status, parental psychopathology or ethnicity could act as a confounder. Perhaps it could be examined whether the effect is more (or less) pronounced in certain socioeconomic or ethnic groups. Parental psychopathology might be argued to be the common cause of ADHD symptoms and academic under-

performance, thus explaining the observed effect; a further study examining genetic and psychosocial mediators and moderators would be of great interest.

It should also be noted that in *Study 2* there was a large number of cases that had to be removed from the analysis due to missing GCSE scores; however comparisons of "completers" and "refusers" did not suggest any systematic bias within the data.

Finally, there were significant limitations to *Study 3* that may have led to the unexpected results. Due to problems with the opt-in consent process (see Chapter 3 for explanation of consent procedures), the sample size was severely underpowered. As such, the external validity of the negative results comes into question.

Theoretical Implications

The findings of this thesis have implications for the theoretical understanding of ADHD. The results of *Study 1* suggest that adolescent ADHD symptoms are a valid dimension, and exist on a continuum. It has been argued that ADHD symptoms are merely an expression of symptoms of other co-morbid syndromes (Spencer, Biederman, Wilens, & Faraone, 1994; Wilens *et al.*, 2003), however the results of *Study 1* suggest that this is not the case. Current ADHD symptom ratings were correlated with anxiety, depression and aggression, but importantly they loaded onto a separate factor. This suggests that ADHD is linked to these other constructs, but ultimately is separate (i.e. co-morbid); this link between ADHD and associated problems has also been established in clinical cases (Bird, Gould, & Staghezza, 1993; Jarrett & Ollendick, 2008; Pliszka, 1998). The results of *Study 1* were found in a community sample, suggesting that adolescent ADHD can be considered as a

dimension, not just a clinical category. As a whole, the findings of *Study 1* provide support for the validity of the adolescent ADHD dimension, and as such provide reassurance that the ADHD aetiology literature is valid – i.e. there is a specific set of factors that put the individual at risk for expressing a specific set of symptoms; factors such as genetics have been shown to define ADHD symptoms across the whole population, not just in clinical cases (NICE ADHD Guideline: National Collaborating Centre for Mental Health, 2009). If the factor analysis did not differentiate between ADHD, anxiety, depression, and aggression, then the specificity of the risk factors that have been identified in the literature could be called into question.

The findings also raise questions regarding the factor structure of ADHD in adolescent communities. The ADHD structure established in *Study 1* did not differentiate between the subtypes of ADHD, however when the scale scores were entered for second-order factor analysis, AARS inattention and AARS hyperactivity/impulsivity loaded onto their own factors, but with mutual cross-loading. This raises the possibility that the ADHD subtypes merge in the adolescent community. This is the first study of its kind using an adolescent sample, and so there is no opportunity for direct comparison with the literature, however child studies have typically unearthed a bi-dimensional ADHD structure (DSM-IV; APA, 1994) and adult studies have found both two factors (Du Paul *et al.*, 2001; Spencer, Biederman, Wilens, & Faraone, 1998), and three-factors (Span, Earlywine & Strybel, 2002). These findings were from both clinical and community samples. The mixed results suggest there is a need to further investigate the symptom structure of ADHD in the community.

The findings of the thesis support observations about ADHD in the classroom made in the NICE ADHD guideline (National Collaborating Centre for Mental

Health, 2009). Firstly, the NICE guideline raises issues regarding the prevalence of ADHD in schools. The worldwide prevalence of ADHD is estimated at 5-10% (Faraone, Sergeant, Gillberg, & Biederman, 2003); however elsewhere, the *UK* prevalence has been found at 0.85% for girls and 3.62% for boys (Ford, Goodman, & Meltzer, 2003). In this thesis, using clinical cut-offs, the prevalence of combined ADHD in *Study 1* was 9.72%; somewhat higher than has been reported in the literature. However, interestingly the NICE guidelines report that the proportion of schoolchildren observed by their class teachers to be inattentive, hyperactive, and/or impulsive is estimated to be between 8.1% and 17%; figures more in-line with the current findings. Prevalence rates based *only* on teacher- and self-report measures of symptoms do not take account for impairment: when defining ADHD it is not only the presence of sufficient numbers of ADHD symptoms that should be considered, but also their association with clinical and social impairments at home, school and elsewhere (National Collaborating Centre for Mental Health, 2009). Canino *et al.* (2004) found that prevalence rates measuring symptom count alongside impairment are approximately half that of prevalence rates where there is no evidence of impairment. As such it should be emphasised that prevalence rates taken from screening in the community merely show the proportion of high levels of symptoms; in order to gain an accurate picture of the true prevalence rate, measures of impairment should also be taken.

Secondly, the NICE guideline suggests that both ADHD diagnoses *and* ratings of ADHD behaviours are negatively linked to academic outcomes, with symptoms of inattention being a key ingredient; this was supported by the findings of *Study 2* of this thesis. Thirdly, the NICE guidelines state that early interventions can be successful in reducing ADHD behaviours and negative outcomes. The present results

show that those in Year 11 who report a history of ADHD symptoms are likely to struggle with their GCSEs, suggesting that early detection of those with high levels of symptoms would be crucial in ensuring satisfactory long-term academic outcomes.

The results of *Study 3* have implications for the understanding of the Dual Pathway model of ADHD (Sonuga-Barke, 2002). The model suggests that delay aversion and cognitive dysregulation are parallel, but distinct, pathways to ADHD; research has shown that ADHD individuals are likely to either experience cognitive dysregulation or be delay averse (Solanto *et al.*, 2001; Sonuga-Barke, 2002). This was not found in the community sample in *Study 3*. However perhaps with a larger sample size and more age-appropriate tasks (see Chapter 6 for discussion of these issues), the expected results will have been produced. Previous research has shown that the Dual Pathway Model can be applied successfully to preschool and child community samples (Paloyelis, Asherson & Kuntsi, 2009; Sonuga-Barke *et al.*, 2003; Thorell, 2007), as such there is a need to fully examine the model in older samples. This could provide evidence that all aspects of ADHD – not merely symptoms – are continuum-based.

Practical implications

The finding that ADHD symptomatology is associated with academic impairment in a non-clinical sample suggests a need for increased awareness of symptomatic individuals. Teachers and carers should be made aware that ADHD symptoms persist into adolescence and that they raise the possibility of significant impairment; by increasing awareness, high-risk individuals could be identified and given necessary remedial assistance. Not only do the results demonstrate the link between ADHD symptoms and academic performance, they show that the impact of ADHD symptoms

is comparable to school-oriented motivation and general cognitive ability. This suggests that intervention for 'high-risk' individuals is of the uppermost importance and justifies the initiation of a randomised trial of appropriate intervention.

To break down barriers to achievement in high-symptom and diagnosed individuals, efforts should be made to develop and implement educational interventions. Stimulant medication and behavioural interventions have been shown to improve off-task and disruptive behaviour (Evans *et al.,* 2001); however the impact of these approaches on academic performance is less pronounced (DuPaul & Eckert, 1997; Raggi & Chronis, 2006). The finding in this thesis that ADHD symptoms negatively impact on GCSE performance suggests that ADHD symptoms impact on both learning and assessment, as GCSEs encapsulate both of these processes. As such, adjustments should be made to the academic environment of high-symptom individuals, with the implementation of alternative teaching and assessment methods that suit the needs of the ADHD individual.

In *Chapter 2*, a range of educational interventions for those with high levels of ADHD symptoms (or a diagnosis of ADHD) was discussed. Utilising these various strategies could be especially important in the GCSE years, where there is pressure on pupils to learn, understand, and memorise large amounts of information. The interventions discussed in *Chapter 2* included: alternative teaching strategies (such as teachers communicating using short clear messages, or teaching the ADHD individual in small groups outside of the main classroom, away from class distractions); study strategies (such as teaching the individual to implement revision goal/reward strategies); distractor strategies (such as using Blu-Tak or stress balls to fidget with during class) in order to improve concentration and to distract the individual from the passing of time; and finally, implementing manipulations to the examination process

(for example reducing exam length). How some of these interventions could be developed, implemented, and evaluated will be discussed in the *Future directions* section of this chapter.

Whilst the results imply that providing academic support for ADHD individuals is of high importance, there are also academic implications for the general adolescent population. In this general population adolescent sample, ADHD symptoms were shown to have almost as strong an impact over GCSE outcome as school-oriented motivation and general cognitive ability. Whilst teachers will be aware that the highly gifted and motivated pupils will be most likely to succeed, the importance of inattentive, hyperactive, and impulsive behaviours may not be fully appreciated. According to the results of this thesis, high achieving pupils seem to possess good cognitive skills, are highly motivated, and do not exhibit high levels of ADHD symptoms, in particular inattention – i.e. they do not make careless mistakes, are able to concentrate, follow instructions, are organised, and are not easily distracted. Teaching all pupils the importance of these behaviours can only increase their chances of academic success. Indeed the classic messages delivered by teachers, such as "*pay attention*", "*do not rush your work*", and "*go back and check over your work*" seem to be closely linked to the ADHD behaviour continuum, and are integral components of school success.

The interventions described in *Chapter 2* could also be used post-16 education settings to assist individuals with ADHD or at least high levels of symptoms. There are some post-16 education support services available for individuals with special needs (such as dyslexia); however, for many adolescents, the educational path ends after the age of 16 and they enter the world of work, where psychological support schemes are less prominent than in educational settings. Currently there are few post-

16 intervention services (Daley, 2006). Research shows that ADHD persists into adulthood (Faraone *et al.*, 2000), and is associated with impairment in academic (Klein & Mannuzza, 1991) and occupational (Mannuzza *et al.*, 1993) domains. As such, the need for the development of intervention services for adolescence, and beyond, is of great importance.

Future directions

The findings of this thesis provide a platform on which future research can be based. Firstly, more research should be carried out to determine the factor structure of ADHD in adolescence. The overall findings of *Study 1* supported the notion that adolescent ADHD is a valid dimension, operating as a continuous variable; the ADHD structure established in *Study 1* did not differentiate between the subtypes, as such further research should be carried out to examine whether the subtypes do indeed merge in the adolescent community. Confirmatory factor analysis should be employed in further research using the present results as a framework on which to impart the data, but within this there would be two alternative ADHD structures: a one-factor framework, in line with the present results, and a two-factor framework, in line with the DSM-IV structure.

The low statistical power in *Study 3* provides a clear opportunity to repeat the study. There is a burgeoning literature on the dual pathway model (Sonuga-Barke, 2002), with knowledge of its relations to functional impairments currently being in the foetal stage (Thorell, 2007). Both a large-scale adolescent community study (although logistically difficult) and a clinically-based study could be carried out, investigating whether indeed it is the cognitive dysregulation (Schachar *et al.*, 2000) pathway and not the motivational style (Sonuga-Barke *et al.*, 1996) pathway that

mediates the link between symptomatology and impairment (Thorell, 2007). As was found in this thesis, a study of this nature is difficult to implement, therefore careful planning would be needed, especially in the areas of recruitment and consent. In the present study a very small number of participants' parents returned the *opt-in* consent forms to allow their child to take part; this severely constricted the sample size. In future research, measures could be taken to ensure that a larger sample size is achieved. For example, perhaps an *opt-out* procedure could be adopted – this consent procedure produced an extremely large sample in *Study 1* and *Study 2*, but could not be used in *Study 3* as the participant would be in a room alone with the principal examiner; perhaps in a future study the computer testing could be carried out with a member of school staff present in the room, thus allowing *opt-out* consent to be used. Alternatively, if an *opt-in* consent process was still to be used, then more could be done to persuade parents to return the consent form; in the present study, the principal examiner was present at parents' evening to discuss the study, parents were sent the study information sheet and consent form, parents were then sent a letter to remind them to return the consent form if they had not already done so, and finally parents were sent a letter stating that the research team would donate money to the school for every consent form returned (this money would be spent on a piece of equipment for the school). Perhaps in future the best incentive to take part would be monetary, with participants receiving cash for taking part; unfortunately PhD research budget constraints dictated that this was not possible in the present study.

A factor that could be examined is reading disability (RD). Research shows that ADHD and RD tend to co-occur (Stevenson *et al.*, 2005), with ADHD children being shown to be at risk for later onset of RD (McGee *et al.*, 2002). As such future research could investigate whether the link between ADHD and academic

performance is mediated by reading problems. Research also shows that ADHD and RD may share a common aetiology (Stevenson *et al.*, 2005; Willcutt *et al.*, 2001), therefore it would be interesting to investigate the extent to which the relationship between ADHD and academic performance can be attributed to RD. As mentioned previously, perhaps an interesting direction for future research could be investigating the role of mediators and moderators in the link between ADHD and academic performance, both in community samples and diagnosed cases. The role of the dual pathway model needs to be investigated, however other factors such as specific reading disorders, socioeconomic status, ethnicity, and parental psychopathology could also be studied.

Study 2 showed that lifetime ADHD symptoms account for more variance in GCSE performance than current symptoms, and that of the current symptoms it is inattention that plays the most prominent role. As such an interesting line for future research could be to carry out a community-based longitudinal study, investigating which aspects of ADHD symptomatology most greatly influence academic performance at different developmental stages. This thesis has shown that ADHD has a negative impact over academic performance in preschoolers, children, adolescents and adults; perhaps these developmental stages could be used as time points for data collection in the study. The results of *Study 2* also point to the possibility of investigating longitudinal risk factors. Such a study could assess which preschool and childhood factors pose significant risk for long-term academic impairment; psychopathology (such as ADHD) and other factors as discussed in *Chapter 1* (such as socio-economic status, personality, parenting style, and peer relationships) could be studied. Also the focus could be shifted from academic outcomes to ADHD, identifying which early factors pose risk for later development of ADHD symptoms,

and perhaps whether there are unique features and circumstances surrounding those who still express ADHD symptoms as older adolescents and adults.

The findings of *Study 2*, and indeed the whole thesis, point to a need to fully research educational interventions for ADHD individuals and those undiagnosed individuals who are identified as exhibiting strong signs of the disorder. Some of the academic interventions discussed in *Chapter 2* have already received some research attention, for example peer tutoring and reducing tasks into sub-units (Raggi & Chronis, 2006). In *Chapter 2* it was proposed that these interventions could be used to assist ADHD individuals who are taking their GCSEs. *Chapter 2* also discussed several possible academic interventions based on existing ADHD knowledge, for example using distractor objects. However, before these interventions are used to help adolescents, their effectiveness and feasibility must be assessed. Examples of how such interventions could be effectively implemented and evaluated will be discussed below. The examples provided are reducing exam length and using distractor objects.

Reducing exam length. Evidence suggests that reducing task length and dividing tasks into subunits is of academic benefit to individuals with ADHD (Raggi & Chronis, 2006). Such tasks could include examinations: when faced with a long examination, ADHD individuals will most likely struggle to maintain their attention, and may also elicit certain behaviours to distract themselves from the passing of time; however these behaviours will most likely stop the individual from working and could be distracting to other pupils. A shorter examination length would be a much more attractive prospect for the ADHD individual, thus increasing the likelihood of full focus on the examination. In order to evaluate the effectiveness of reducing exam length for ADHD adolescents, this strategy would have to be trialled with non-GCSE exams, such as end-of-year school exams or GCSE mock exams. A number of schools

would be invited to participate in a cluster randomised trial, with 15- and 16-year olds who are identified as expressing high levels of ADHD symptoms, or who have a diagnosis of ADHD being offered the opportunity to take part in the trial. Half of the participants would be assigned to the experimental group and half to an age-, gender-, and symptom-matched control group. Those in the experimental group would have their specified exams divided into two parts, perhaps with one part in the morning and one in the afternoon. Any differences in results between the experimental group and control group will provide evidence for or against the effectiveness of the intervention.

If the intervention was deemed to be effective then GCSE exam boards would have to agree to permit its implementation. To make the intervention feasible, there would have to be several logistical problems to overcome, for example: during the period between part one and part two of the examination pupils would have to be kept separate from those who took the examination in one sitting in order to avoid cheating; also, there would have to be enough staff available to invigilate the extra examinations that would be created by the intervention. A different method of adapting examinations to suit the needs of the ADHD individual could be introducing flexible examinations. Here, ADHD individuals could be permitted to leave the examination room for several minutes whenever they feel their attention waning. However an intervention of this nature could prove very problematic. Firstly, each time the individual leaves the room, a member of staff would have to accompany him/her – this could mean that extra staff members are needed to help with invigilation. Secondly, ADHD is highly correlated with disruptive behaviour disorders (Biederman, Newcorn, & Sprich, 1991), as such it is possible that if these

individuals were allowed to leave and re-enter the examination room at will, then this could lead to widespread disruption; the process would have to be heavily monitored.

Using distractor objects. The dual pathway model of ADHD (Sonuga-Barke, 2002) states that many ADHD individuals are averse to delay, therefore in long examinations or school lessons these individuals will tend to elicit certain behaviours to distract themselves from the passing of time, as the prospect of a long delay before the end of the lesson or examination would not be attractive. Unfortunately these behaviours will most likely stop the individual from working and could be distracting to other pupils. A useful intervention could be to design strategies to improve ADHD individuals' in-class focus, thus improving their academic performance: for example these individuals could be given distractor objects – such as Blu-Tak or a stressball – to fidget with during class, thus distracting them from the passing of time and subsequently reducing their delay aversion.

To evaluate the effectiveness of this intervention would require careful planning. Perhaps first the in-class focus and performance of ADHD individuals could be monitored over a set period if time, in order to establish a baseline. Next, in the experimental phase the participants could be given a distractor object at the start of every lesson, and measurements of focus and performance could be taken. If there were significant or meaningful increases in performance and focus, then the intervention could be widely implemented. However due to the close association between ADHD and disruptive behaviour, participants in the evaluation could not simply be given distractor objects and left to work; in-class support assistants would be needed to closely monitor the usage of the objects to ensure that they are being used in the correct manner.

General Conclusion

Collectively the results show that ADHD persists beyond childhood, and that the adolescent ADHD construct is not dichotomous; more, it should be viewed as a continually distributed trait, where the diagnosed individuals represent the extreme upper echelons of the continuum. Moreover, the finding of associated academic impairment in this community sample suggests that adolescent ADHD symptoms are a significant barrier to achievement, not only for diagnosed individuals, but also for those who are at the high – but sub-clinical – end of the continuum.

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Appendices

Appendix 1: Abbreviated version of *Chapter 2* to be published in *Child: Care, Health and Development* in January 2010.

Appendix 2: Information Letters / Invitations to Participate

2 (a) Study 1, Study 2 parental letter

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SEICOLEG
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Study Information Letter

Study Title
The relationship between symptoms of attention deficit hyperactivity disorder (ADHD) and academic performance in adolescents.

Researcher(s)
James Birchwood, PhD Student, University of Wales Bangor,
Dr. D. Daley, Senior Research Tutor, NWCCP, University of Wales Bangor,
Dr M. Hoerger, University of Wales Bangor.

Invitation to participate
Along with every other child in year 11, your child is being invited to take part in a research study. Before you decide to agree to let your child to take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information. If there is anything that is not clear or if you would like more information, please contact me (James Birchwood) by e-mail (my address will be provided at the end of this letter). Also, I will be present at the upcoming Parents' Evening to answer any questions you may have regarding the study.

What is the purpose of the study?
Attention deficit hyperactivity disorder (ADHD) is a condition defined by the presence of severe and pervasive symptoms of inattention, over activity and impulsivity. While originally perceived as a disorder of childhood, there is now both scientific merit and clinical value in investigating ADHD in adolescence and adulthood. While studies of the disorder in childhood have examined the relationship between ADHD symptoms and academic achievement, this has not been extensively researched in adolescent and young adult populations. The current study investigates the relationship between adolescent ADHD and academic achievement. For this study, it is irrelevant whether your child has ADHD or not; we are using a general population sample.

What are the benefits of taking part in the research?
Despite increasing recognition that ADHD persists into adolescence and adulthood, to date there has been little research attention in this area. By allowing your child to take part in this research you will be helping to advance scientific understanding of the relationship between young adult ADHD and academic performance.

Does your child have to take part?
It is up to you whether or not you decide to allow your child to participate. If you agree, your child is still free to withdraw at any time without giving a reason.

What will happen if your child takes part in the research?
The first phase of the study will take place either in your child's classroom or in the school hall during a PSHE session. After being asked for permission to participate, your child will fill out a questionnaire, and take a test of general cognitive ability. This phase of the study will take approximately 40 minutes to complete. A few weeks later we will invite a number of pupils to participate in a second phase of the study, which will take approximately 1 hour to complete; we will send out relevant information letters nearer to the time.

All information collected during the course of the study will be anonymised.
Results of the study will describe overall findings and not information about individuals. You will not find out whether your child has ADHD, as the measures used in this study cannot provide a diagnosis of ADHD. All participants will be sent a summary of the study.

What do you do now?
If you **do not** wish for your child to take part in the study, please return the opt-out slip (see the Consent Form) to the research team, using the addressed FREEPOST envelope provided. If we receive this reply slip, your child **will not** be permitted to take part in the study. If you agree to allow your child to participate, do nothing.

Further details
If you want to contact the investigator about the research, the details are below:

James Birchwood, PhD student, University of Wales Bangor
E-mail: psp00e@bangor.ac.uk

If you have any complaints about the way that this research is being conducted you are welcome to address unresolved concerns to:

Professor Richard Hastings,
Acting Head,
School of Psychology,
University of Wales Bangor,
Bangor,
Gwynedd
LL57 2DG

2 (b) Study 1, Study 2 participant letter

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Dr. D. Daley, Senior Research Tutor, NWCCP, University of Wales Bangor,
Dr M. Hoerger, University of Wales Bangor.

Invitation to participate
You are being invited to take part in a research study. Before you decide to take part it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. If there is anything that is not clear or if you would like more information, please contact me by e-mail (my address will be provided at the end of this letter).

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Do you have to take part?
It is up to you whether or not you decide to participate. If you decide to take part, you are still free to withdraw at any time without giving a reason. If you decide not to participate, you must remain seated in the hall.

What will happen if you take part in the research?
After being asked to complete the consent form, you will complete the Raven's Standard Progressive Matrices – a test of observation and clear thinking – and then you will be asked to fill out a questionnaire. The study will take between 40 minutes and an hour. **All information collected during the course of the study will be anonymised.** Results of the study will describe overall findings and not information about individuals.

What do you do now?
If you decide to take part, please complete the consent form, and then await instruction from the researchers.

Further details
If you want to contact the investigator about the research, the details are below:

James Birchwood, PhD student, University of Wales Bangor
E.mail: psp00e@bangor.ac.uk

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2 (c) Study 3 parental letter; school sampling

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All information collected during the course of the study will be anonymised.
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2 (d) Study 3 participant letter; school sampling

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Researcher(s)
James Birchwood, PhD Student, University of Wales Bangor,
Dr. D. Daley, Senior Research Tutor, NWCCP, University of Wales Bangor,
Dr M. Hoerger, University of Wales Bangor.

Invitation to participate
Along with every other child in year 11, your child is being invited to take part in a research study. Before you decide to agree to let your child to take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information. If there is anything that is not clear or if you would like more information, please contact me (James Birchwood) by e-mail (my address will be provided at the end of this letter). Also, I will be present at the upcoming Parents' Evening to answer any questions you may have regarding the study.

What is the purpose of the study?
Attention deficit hyperactivity disorder (ADHD) is a condition defined by the presence of severe and pervasive symptoms of inattention, over activity and impulsivity. While originally perceived as a disorder of childhood, there is now both scientific merit and clinical value in investigating ADHD in adolescence and adulthood. While studies of the disorder in childhood have examined the relationship between ADHD symptoms and academic achievement, this has not been extensively researched in adolescent and young adult populations. The current study investigates the relationship between adolescent ADHD and academic achievement. For this study, it is irrelevant whether your child has ADHD or not; we are using a general population sample.

What are the benefits of taking part in the research?
Despite increasing recognition that ADHD persists into adolescence and adulthood, to date there has been little research attention in this area. By allowing your child to take part in this research you will be helping to advance scientific understanding of the relationship between young adult ADHD and academic performance.

Does your child have to take part?

It is up to you whether or not you decide to allow your child to participate. If you agree, your child is still free to withdraw at any time without giving a reason.

What will happen if your child takes part in the research?
The first phase of the study will take place either in your child's classroom or in the school hall during a PSHE session. After being asked for permission to participate, your child will fill out a questionnaire, and take a test of general cognitive ability. This phase of the study will take approximately 40 minutes to complete. A few weeks later we will invite a number of pupils to participate in a second phase of the study, which will take approximately 1 hour to complete; we will send out relevant information letters nearer to the time.

All information collected during the course of the study will be anonymised.
Results of the study will describe overall findings and not information about individuals. You will not find out whether your child has ADHD, as the measures used in this study cannot provide a diagnosis of ADHD. All participants will be sent a summary of the study.

What do you do now?
If you **do not** wish for your child to take part in the study, please return the opt-out slip (see the Consent Form) to the research team, using the addressed FREEPOST envelope provided. If we receive this reply slip, your child **will not** be permitted to take part in the study. If you agree to allow your child to participate, do nothing.

Further details
If you want to contact the investigator about the research, the details are below:

James Birchwood, PhD student, University of Wales Bangor
E-mail: psp00e@bangor.ac.uk

If you have any complaints about the way that this research is being conducted you are welcome to address unresolved concerns to:

Professor Richard Hastings,
Acting Head,
School of Psychology,
University of Wales Bangor,
Bangor,
Gwynedd
LL57 2DG

2 (e) Study 3 parental letter; opportunity sampling

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PSYCHOLOGY

Study Information Letter

Study Title
The relationship between symptoms of attention deficit hyperactivity disorder (ADHD) and academic performance in adolescents.

Researcher(s)
James Birchwood, PhD Student, University of Wales Bangor,
Dr. D. Daley, Senior Research Tutor, NWCCP, University of Wales Bangor,
Dr M. Hoerger, University of Wales Bangor.

Invitation to participate
Your child is being invited to take part in a research study. Before you decide to agree to let your child to take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information. If there is anything that is not clear or if you would like more information, please contact me (James Birchwood) by e-mail (my address will be provided at the end of this letter).

What is the purpose of the study?
Attention deficit hyperactivity disorder (ADHD) is a condition defined by the presence of severe and pervasive symptoms of inattention, over activity and impulsivity. While originally perceived as a disorder of childhood, there is now both scientific merit and clinical value in investigating ADHD in adolescence and adulthood. While studies of the disorder in childhood have examined the relationship between ADHD symptoms and academic achievement, this has not been extensively researched in adolescent and young adult populations. The current study investigates the relationship between adolescent ADHD and academic achievement.

What are the benefits of taking part in the research?
Despite increasing recognition that ADHD persists into adolescence and adulthood, to date there has been little research attention in this area. By allowing your child to take part in this research you will be helping to advance scientific understanding of the relationship between young adult ADHD and academic performance.

Does your child have to take part?
It is up to you whether or not you decide to allow your child to participate. If you agree, your child is still free to withdraw at any time without giving a reason.

What will happen if your child takes part in the research?
If you agree for your child to take part in the research, please fill in the consent form and give it to your child to give to me at the testing session. At the testing session, I will ask your child for permission for him/her to participate. Your child will be asked to fill out a questionnaire, and take a test of general cognitive ability. This phase of the study will take approximately 40 minutes to complete. Next, your child will be asked to complete two computer-based neuropsychological tasks, which will take in total approximately 30 minutes to complete. Finally, we will contact your child for their GCSE results (if your child permits us to view them).

All information collected during the course of the study will be anonymised.
Results of the study will describe overall findings and not information about individuals. You will not find out whether your child has ADHD or not, as the measures used in this study cannot provide a diagnosis of ADHD.

What do you do now?
Please complete the consent form (attached) and give it to your child to bring along to the testing session.

Further details
If you want to contact the investigator about the research, the details are below:

James Birchwood, PhD student, University of Wales Bangor
E-mail: psp00e@bangor.ac.uk

If you have any complaints about the way that this research is being conducted you are welcome to address unresolved concerns to:

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2 (f) Study 3 participant letter; opportunity sampling

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Invitation to participate
You are being invited to take part in a research study. Before you decide to agree to let your child to take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information. If there is anything that is not clear or if you would like more information don't hesitate to ask the experimenter any questions.

What is the purpose of the study?
Attention deficit hyperactivity disorder (ADHD) is a condition defined by the presence of severe and pervasive symptoms of inattention, over activity and impulsivity. While originally perceived as a disorder of childhood, there is now both scientific merit and clinical value in investigating ADHD in adolescence and adulthood. While studies of the disorder in childhood have examined the relationship between ADHD symptoms and academic achievement, this has not been extensively researched in adolescent and young adult populations. The current study investigates the relationship between adolescent ADHD and academic achievement.

What are the benefits of taking part in the research?
Despite increasing recognition that ADHD persists into adolescence and adulthood, to date there has been little research attention in this area. By taking part in this research you will be helping to advance scientific understanding of the relationship between young adult ADHD and academic performance.

Do you have to take part?
It is up to you whether or not you decide to participate. If you agree, you are still free to withdraw at any time without giving a reason.

What will happen if you take part in the research?
If you agree to take part in the research you will fill out a questionnaire, and take a test of general cognitive ability. This phase of the study will take approximately 40

minutes to complete. Next, you will be asked to complete two computer-based neuropsychological tasks. Finally, we will contact you to ask for your GCSE results (if you permit us to view them).

All information collected during the course of the study will be anonymised. Results of the study will describe overall findings and not information about individuals. You will not find out whether you have ADHD, as the measures used in this study cannot provide a diagnosis of ADHD.

What do you do now?
Please complete the consent form, questionnaire and cognitive test, and bring them along to the computer testing session.

Further details
If you want to contact the investigator about the research, the details are below:

James Birchwood, PhD student, University of Wales Bangor
E-mail: psp00e@bangor.ac.uk

If you have any complaints about the way that this research is being conducted you are welcome to address unresolved concerns to:

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2 (g) Study 3 parental letter; ADHD support group sampling

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Study Information Letter

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Dr. D. Daley, Senior Research Tutor, NWCCP, University of Wales Bangor,
Dr M. Hoerger, University of Wales Bangor.

Invitation to participate
Your child is being invited to take part in a research study. Before you decide to agree to let your child to take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information. If there is anything that is not clear or if you would like more information, please contact me (James Birchwood) by e-mail (my address will be provided at the end of this letter).

What is the purpose of the study?
Attention deficit hyperactivity disorder (ADHD) is a condition defined by the presence of severe and pervasive symptoms of inattention, over activity and impulsivity. While originally perceived as a disorder of childhood, there is now both scientific merit and clinical value in investigating ADHD in adolescence and adulthood. While studies of the disorder in childhood have examined the relationship between ADHD symptoms and academic achievement, this has not been extensively researched in adolescent and young adult populations. The current study investigates the relationship between adolescent ADHD and academic achievement.

What are the benefits of taking part in the research?
Despite increasing recognition that ADHD persists into adolescence and adulthood, to date there has been little research attention in this area. By allowing your child to take part in this research you will be helping to advance scientific understanding of the relationship between young adult ADHD and academic performance.

Does your child have to take part?
It is up to you whether or not you decide to allow your child to participate. If you agree, your child is still free to withdraw at any time without giving a reason.

What will happen if your child takes part in the research?
If you agree for your child to take part in the research, we will contact you to arrange a date and time when we could come to your home and carry out the study. Once at your home, we will then ask your child for permission for him/her to participate. Your child will then fill out a questionnaire, and take a test of general cognitive ability. This phase of the study will take approximately 40 minutes to complete. Next, at a pre-arranged later date, your child will be asked to complete two computer-based neuropsychological tasks. Finally, during the summer we will ring you to ask for your child's GCSE results (if your child permits us to view them).

All information collected during the course of the study will be anonymised.
Results of the study will describe overall findings and not information about individuals.

What do you do now?
Please complete the consent form (attached) and return it to us using the Freepost addressed envelope provided. Please remember to provide your telephone number and we will contact you in due course to arrange a study date.

Further details
If you want to contact the investigator about the research, the details are below:

James Birchwood, PhD student, University of Wales Bangor
E-mail: psp00e@bangor.ac.uk

If you have any complaints about the way that this research is being conducted you are welcome to address unresolved concerns to:

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2 (h) Study 3 participant letter; ADHD support group sampling

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Dr M. Hoerger, University of Wales Bangor.

Invitation to participate
You are being invited to take part in a research study. Before you decide to agree to let your child to take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information. If there is anything that is not clear or if you would like more information don't hesitate to ask the experimenter any questions.

What is the purpose of the study?
Attention deficit hyperactivity disorder (ADHD) is a condition defined by the presence of severe and pervasive symptoms of inattention, over activity and impulsivity. While originally perceived as a disorder of childhood, there is now both scientific merit and clinical value in investigating ADHD in adolescence and adulthood. While studies of the disorder in childhood have examined the relationship between ADHD symptoms and academic achievement, this has not been extensively researched in adolescent and young adult populations. The current study investigates the relationship between adolescent ADHD and academic achievement.

What are the benefits of taking part in the research?
Despite increasing recognition that ADHD persists into adolescence and adulthood, to date there has been little research attention in this area. By taking part in this research you will be helping to advance scientific understanding of the relationship between young adult ADHD and academic performance.

Do you have to take part?
It is up to you whether or not you decide to participate. If you agree, you are still free to withdraw at any time without giving a reason.

What will happen if you take part in the research?
If you agree to take part in the research you will fill out a questionnaire, and take a test of general cognitive ability. This phase of the study will take approximately 40

minutes to complete. Next, you will be asked to complete two computer-based neuropsychological tasks. Finally, we will contact you to ask for your GCSE results (if you permit us to view them).

All information collected during the course of the study will be anonymised. Results of the study will describe overall findings and not information about individuals. You will not find out whether you have ADHD, as the measures used in this study cannot provide a diagnosis of ADHD.

What do you do now?
Please complete the consent form, questionnaire and cognitive test, and bring them along to the computer testing session.

Further details
If you want to contact the investigator about the research, the details are below:

James Birchwood, PhD student, University of Wales Bangor
E-mail: psp00e@bangor.ac.uk

If you have any complaints about the way that this research is being conducted you are welcome to address unresolved concerns to:

Professor Richard Hastings,
Acting Head,
School of Psychology,
University of Wales Bangor,
Bangor,
Gwynedd
LL57 2DG

Appendix 3: Consent forms

3(a) Study 1, Study 2 parental consent form (opt-out)

Consent Form

Study Title
The relationship between symptoms of attention deficit hyperactivity disorder (ADHD) and academic performance in adolescents.

Researcher(s)
James Birchwood, PhD Student, University of Wales Bangor,
Dr. D. Daley, Research Tutor, NWCCP, University of Wales Bangor,
Dr. M. Hoerger, University of Wales Bangor.

If you **do not** wish for your son/daughter to participate in the study, please fill in the slip below and return it to the research team using the addressed FREEPOST envelope provided.

Opt-out parental reply slip.

Remember only fill out and return this slip if you do not wish for your son/daughter to participate.

I do not wish for my son/daughter to participate in this study.

Son/daughter's GCSE exam number.....

.....
Parent's Signature

3(b) Study 1, Study 2 participant consent form

Participant Consent Form

Study Title
The relationship between symptoms of attention deficit hyperactivity disorder (ADHD) and academic performance in adolescents.

Researcher(s)
James Birchwood, PhD Student, University of Wales Bangor,
Dr. D. Daley, Research Tutor, NWCCP, University of Wales Bangor,
Dr. M. Hoerger, University of Wales Bangor.

Participant
Please write your GCSE exam number in the space below:

.....

This is to certify that I agree to participate as a volunteer in a scientific study as an authorised part of the research undertakings within the School of Psychology at the University of Wales, Bangor, under the supervision of James Birchwood, PhD student.

I have read the information letter and I know what the study is about, and what my part is in it. ☐ (Please tick)

I understand that I don't have to answer every question in the test. ☐

I understand that all data will remain confidential with regard to my identity.☐

I understand that I am free to withdraw my consent and terminate my participation at any time without penalty.☐

I agree to allow the school to release my GCSE results for use in this study. ☐

I understand that I may request a summary of the results of this study.☐

I agree to participate in the study. ☐

In the case of any complaints concerning the conduct of research, these should be addressed to Professor Richard Hastings, Acting Head, School of Psychology, University of Wales, Bangor, Gwynedd, LL57 2DG.

3(c) Study 3 parental consent form (opt-in); school, opportunity, and support group sampling

Consent Form

Study Title
The relationship between symptoms of attention deficit hyperactivity disorder (ADHD) and academic performance in adolescents.

Researcher(s)
James Birchwood, PhD Student, University of Wales Bangor,
Dr. D. Daley, Research Tutor, NWCCP, University of Wales Bangor,
Dr. M. Hoerger, University of Wales Bangor.

Please fill in the slip below and return it to the research team using the addressed FREEPOST envelope provided.

Parental reply slip.

I wish / do not wish (delete as appropriate) for my son/daughter to participate in this study.

Son/Daughter's name*.....
*Once we have all data, we will match your child's name with his/her GCSE exam number, and discard his/her name. This will ensure anonymity.

Son/daughter's GCSE exam number (if known).....

.....
Parent's Signature

3(d) Study 3 participant consent form; school sampling

Participant Consent Form

Study Title
The relationship between symptoms of attention deficit hyperactivity disorder (ADHD) and academic performance in adolescents.

Researcher(s)
James Birchwood, PhD Student, University of Wales Bangor,
Dr. D. Daley, Research Tutor, NWCCP, University of Wales Bangor,
Dr. M. Hoerger, University of Wales Bangor.

Participant
Please write your GCSE exam number in the space below:

.....
if you do not know your GCSE exam number, please provide your name and at a later date we will obtain your exam number.

This is to certify that I agree to participate as a volunteer in a scientific study as an authorised part of the research undertakings within the School of Psychology at the University of Wales, Bangor, under the supervision of James Birchwood, PhD student.

I have read the information letter and I know what the study is about, and what my part is in it. ☐ (Please tick)

I understand that all data will remain confidential with regard to my identity.☐

I understand that I am free to withdraw my consent and terminate my participation at any time without penalty.☐

I agree to allow the school to release my GCSE results to the research team for use in this study. ☐

I understand that I may request a summary of the results of this study.☐

I agree to participate in the study. ☐

In the case of any complaints concerning the conduct of research, these should be addressed to Professor Richard Hastings, Acting Head, School of Psychology, University of Wales, Bangor, Gwynedd, LL57 2DG.

3(e) Study 3 participant consent form; opportunity and support group sampling

Participant Consent Form

Study Title
The relationship between symptoms of attention deficit hyperactivity disorder (ADHD) and academic performance in adolescents.

Researcher(s)
James Birchwood, PhD Student, University of Wales Bangor,
Dr. D. Daley, Research Tutor, NWCCP, University of Wales Bangor,
Dr. M. Hoerger, University of Wales Bangor.

Participant
Please write your GCSE exam number in the space below:

.....
if you do not know your GCSE exam number, please provide your name and at a later date we will obtain your exam number.

This is to certify that I agree to participate as a volunteer in a scientific study as an authorised part of the research undertakings within the School of Psychology at the University of Wales, Bangor, under the supervision of James Birchwood, PhD student.

I have read the information letter and I know what the study is about, and what my part is in it. ☐ (Please tick)

I understand that all data will remain confidential with regard to my identity.☐

I understand that I am free to withdraw my consent and terminate my participation at any time without penalty.☐

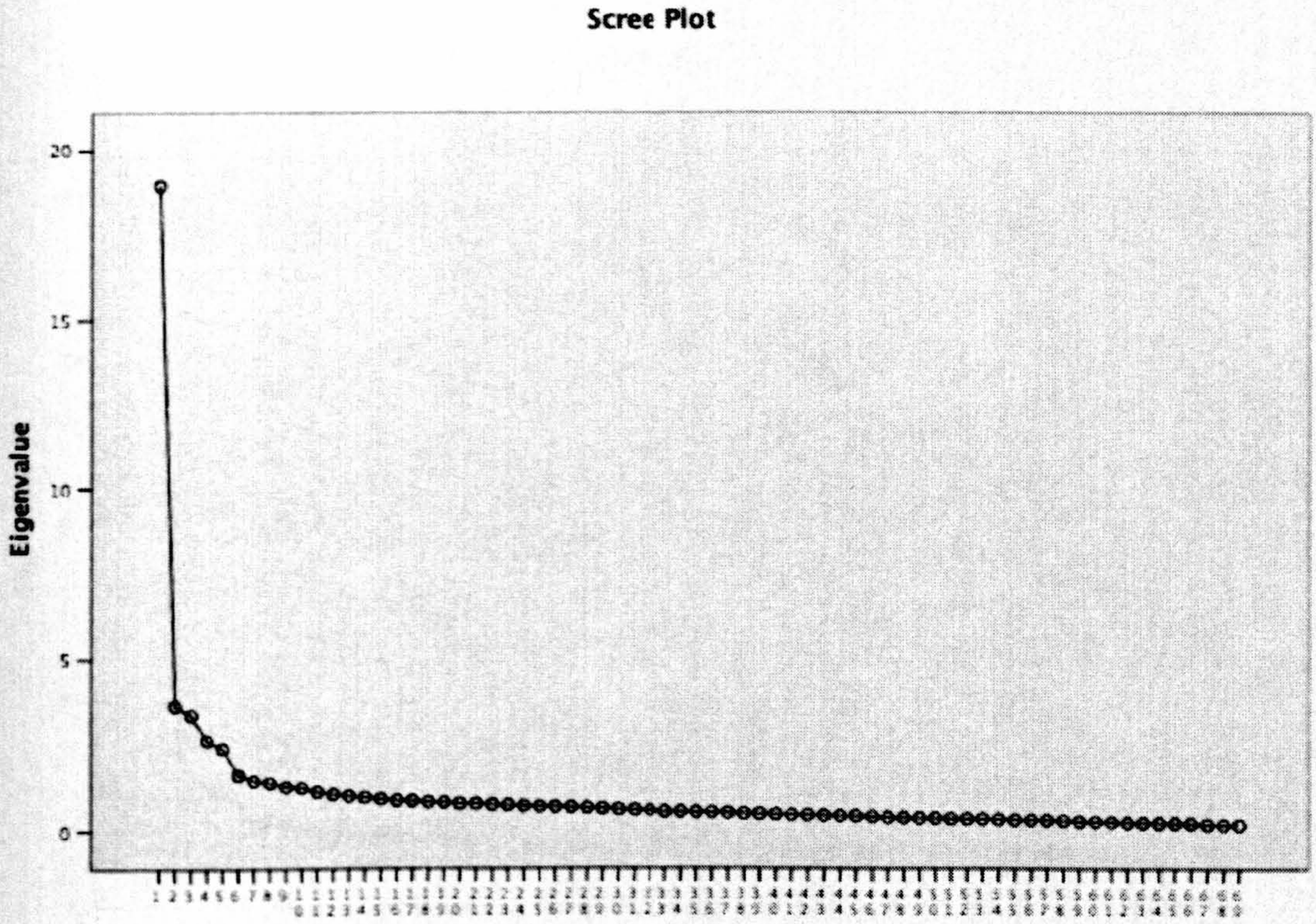
I agree to release my GCSE results to the research team for use in this study. ☐

I understand that I may request a summary of the results of this study.☐

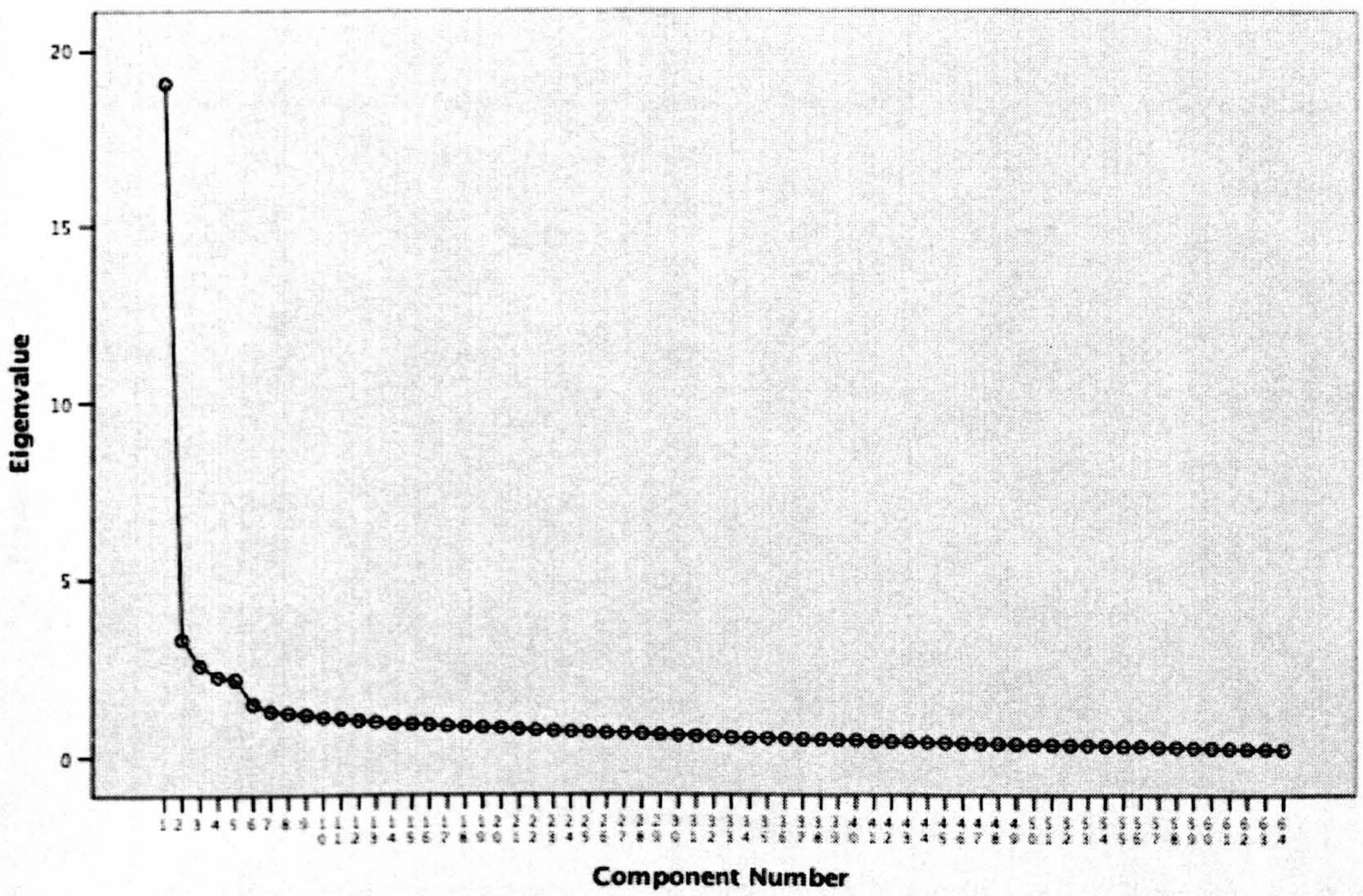
I agree to participate in the study. ☐

In the case of any complaints concerning the conduct of research, these should be addressed to Professor Richard Hastings, Acting Head, School of Psychology, University of Wales, Bangor, Gwynedd, LL57 2DG.

Appendix 5: Scree plot of eigenvalues [Chapter 4 (Study 1)]

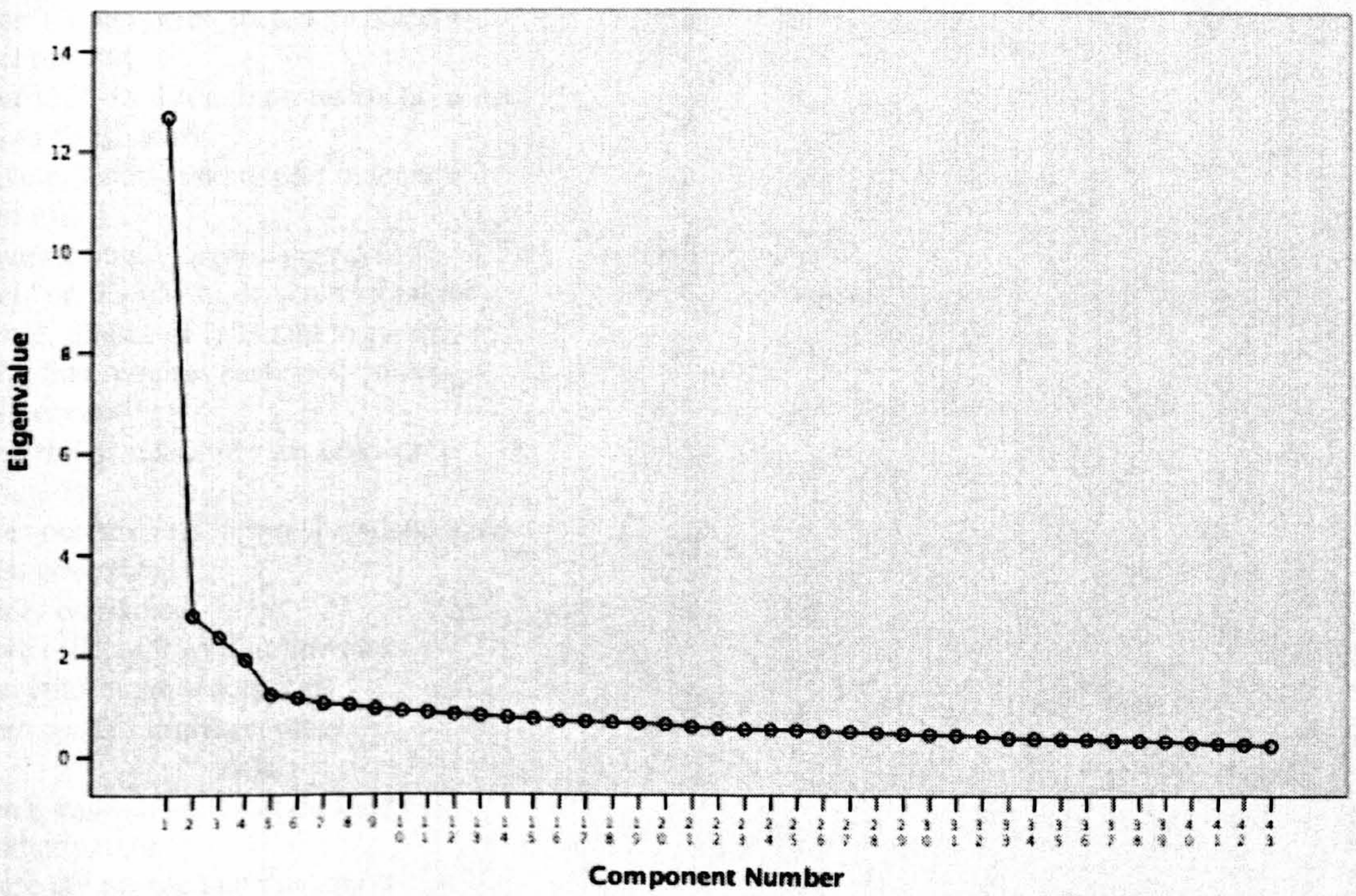


Appendix 6: Scree plot of eigenvalues, overlapping items removed [Chapter 4 (Study 1)]



Appendix 7: Results of factor analysis of AARS, AQ, and HADS items [Chapter 4 (Study 1)]

7(a) Scree plot of eigenvalues



7(b) KMO and Bartlett's test of sphericity

Kaiser-Meyer-Olkin statistic = .938

Bartlett's test of sphericity: *Chi-square* = 9256.03; $p < .00$

These results suggest conditions were appropriate for factor analysis.

7(c) Varimax rotated four-factor solution

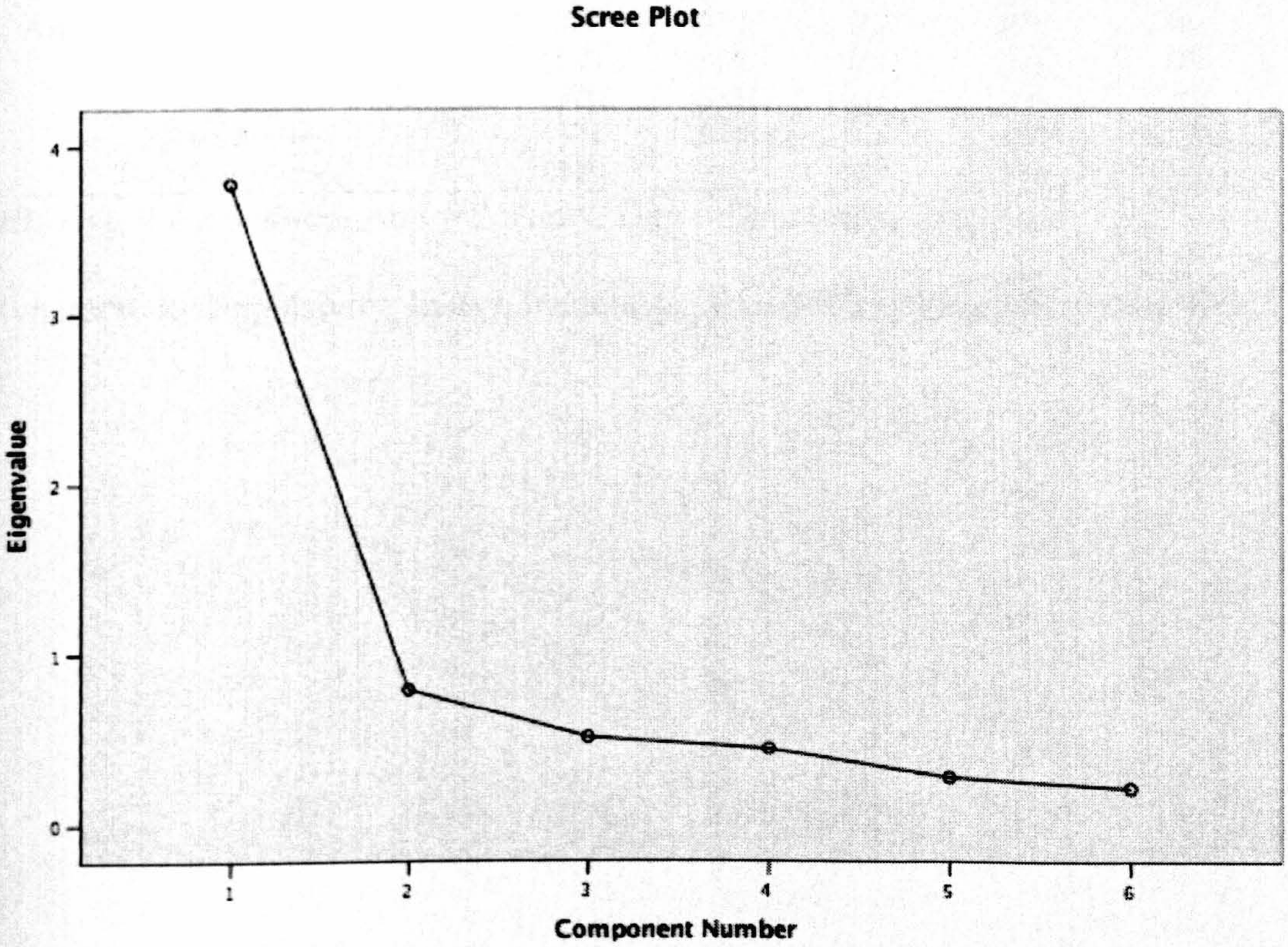
Measure	ADHD	Agg	Anx	Dep
AARS				
Fail to give close attention to details or make careless mistakes at work	**.603**	.115	.194	.091
Fidget with hands, feet or squirm in my seat	**.534**	.158	.245	.017
Difficulty sustaining my attention in tasks	**.628**	.164	.138	.137
Leave my seat in situations in which seating is required	**.592**	.241	-.015	.030
Don't listen when spoken to directly	**.650**	.219	.039	.238
Feel restless	**.511**	.217	.314	.150
Don't follow through on instructions and fail to finish work	**.657**	.195	.115	.216
Have difficulty taking part in leisure activities	.340	.004	.165	**.364**
Have difficulty organising myself.	**.568**	.123	.153	.190
Feel "on the go" or driven by a motor	**.478**	.075	.112	-.184
Avoid, dislike or reluctant to engage in work that requires sustained thinking	**.613**	.193	.102	.079
Talk excessively	**.573**	.230	.223	-.083
Lose things necessary for tasks or activities	**.585**	.154	.169	.201
Blurt out answers before questions have been completed	**.588**	.183	.166	.017
Easily distracted	**.640**	.307	.144	.071
Have difficulty waiting my turn	**.521**	.355	.056	.097
Forgetful in daily activities	**.591**	.181	.205	.170
Interrupt or intrude on others	**.590**	.333	.048	.125
AQ				
Given enough provocation I may hit another person	.198	**.680**	-.038	-.031
There are people that push me so far that we come to blows	.242	**.692**	.061	.048
I have threatened people I know	.263	**.703**	-.046	.080
I often find myself disagreeing with people	.294	**.579**	.127	.107
I can't help getting into arguments when people disagree with me	.221	**.681**	.063	.056
My friends say that I argue a great deal	.241	**.676**	.081	.091
I flare up quickly but get over it quickly	.173	**.649**	.244	-.054
Sometimes I fly off the handle for no good reason	.227	**.694**	.170	.044
I have trouble controlling my temper	.179	**.730**	.072	.141
At times I feel I have had a raw deal out of life	.186	**.536**	.340	.290
Other people always seem to get lucky	.195	**.478**	.387	.255
I wonder why sometimes I feel so bitter about things	.204	**.487**	.460	.258
HADS				
I feel tense or 'wound up'.	.225	.344	**.488**	.301
I still enjoy the things I used to enjoy	.056	-.068	-.142	**.592**
I get a sort of frightened feeling as if something awful is about to happen	.171	.041	**.718**	.043
I can laugh and see the funny side of things	.040	-.014	.031	**.705**
Worrying thoughts go through my mind	.153	.071	**.767**	.058
I feel cheerful	.062	.177	.235	**.654**

Measure	ADHD	Agg	Anx	Dep
I can sit at ease and feel relaxed	.238	.157	.292	**.496**
I feel as if I am slowed down	.191	.177	**.420**	.114
I get a sort of frightened feeling like butterflies in the stomach	.095	.029	**.631**	.029
I have lost interest in my appearance	.055	.073	.168	**.486**
I feel restless as if I have to be on the move	.325	.198	**.415**	.009
I look forward with enjoyment to things	.038	.101	.012	**.693**
I get sudden feelings of panic	.242	.009	**.714**	.137
I can enjoy a good book or radio or TV programme	.159	.153	.064	.293

NB. Agg = Aggression, Anx = Anxiety, Dep = Depression

Appendix 8: Second order factor analysis [Chapter 4 (Study 1)]

8(a) Scree plot of eignvalues



8(b) KMO and Bartlett's test of sphericity

Kaiser-Meyer-Olkin statistic = .860

Bartlett's test of sphericity: *Chi-square* = 1376.01; $p < .000$

These results suggest conditions were appropriate for factor analysis.

8(c) Varimax rotated four-factor solution

Scale score	Agg	Anx	Dep	Hyp/Imp	Inatt	R-ADHD
AARS inattention	.248	.210	.159	*.368*	**.826**	.226
AARS hyperactivity/impulsivity	.266	.224	.107	**.829**	*.364*	.220
WURS total	*.321*	.286	.229	.238	.243	**.805**
AQ total	**.888**	.181	.142	.228	.213	.248
HADS anxiety	.171	**.910**	.186	.183	.172	.210
HADS depression	.119	.162	**.958**	.084	.114	.149

NB. Agg = Aggression, Anx = Anxiety, Dep = Depression, Hyp/Imp = Hyperactivity/Impulsivity, Inatt = Inattention, R-ADHD = Retrospective ADHD.

Appendix 9: Analysis of variance (ANOVA) and Cohen's *d* effect sizes comparing scores on all measures for those who did and did not give consent for the research team to access their GCSE results [Chapter 5 (Study 2)].

Measure	Consenting participants (n = 324) Mean (Standard Deviation)	Non-consenting participants (n = 104) Mean (Standard Deviation)	*F*	*p*	Cohen's *d*
AARS total	14.9 (9.94)	14.49 (9.58)	.139	.71	0.042
AARS inattention	7.43 (5.51)	7.29 (5.15)	.053	.818	0.026
AARS hyp/imp	7.48 (5.06)	7.2 (4.86)	.234	.629	0.056
WURS	52.27 (20.4)	47.94 (21.39)	3.462	.064	0.207
AQ score	30.77 (14.07)	29.38 (12.95)	.787	.375	0.103
HADS depression	4.32 (2.9)	4.13 (3.34)	.333	.564	0.061
HADS anxiety	7.86 (4.13)	6.63 (3.9)	7.121	**.008**	**0.306**
ISM score	132.53 (34.38)	119.35 (48)	9.422	**.002**	**0.316**
RSPM score	43.38 (8.8)	44.31 (7)	.861	.354	-0.117

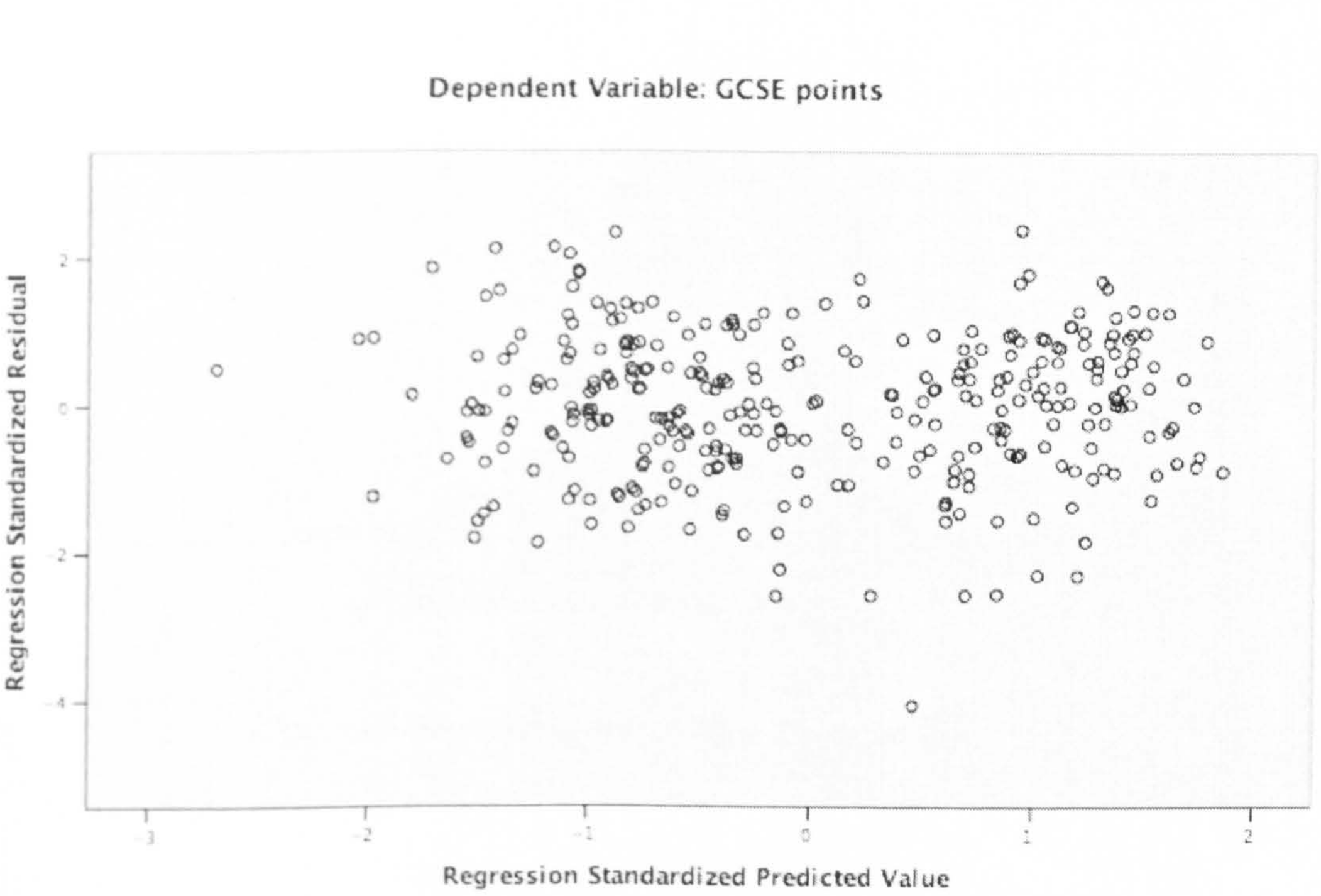
NB. AARS hyp/imp = AARS hyperactivity/impulsivity

Appendix 10: Points allocations for GCSE grades.

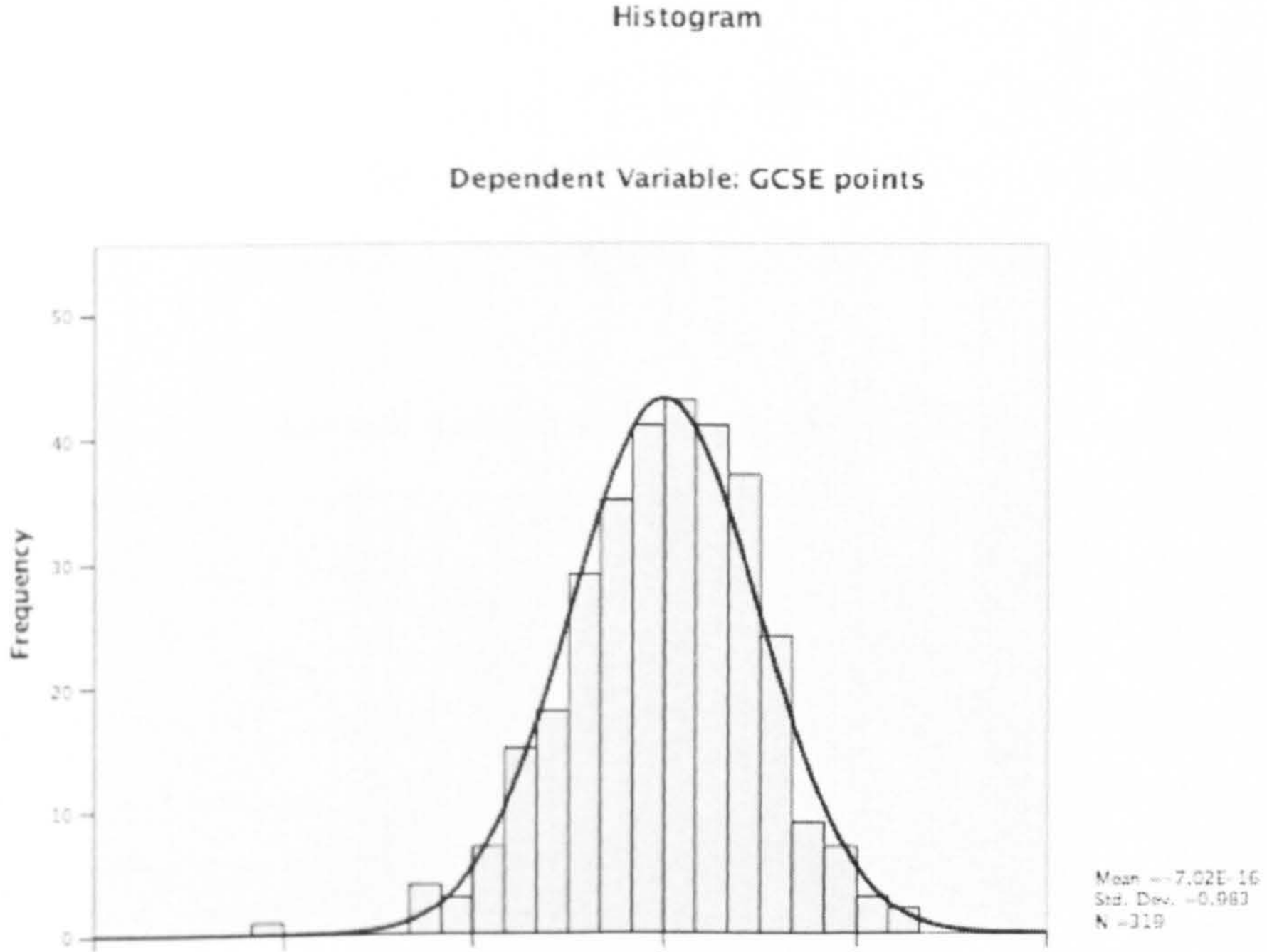
Grade	Points allocation
A*	58
A	52
B	46
C	40
D	34
E	28
F	22
G	16
U	0

Appendix 11: Regression assumption charts, Model *i* [Chapter 5 (Study 2)]

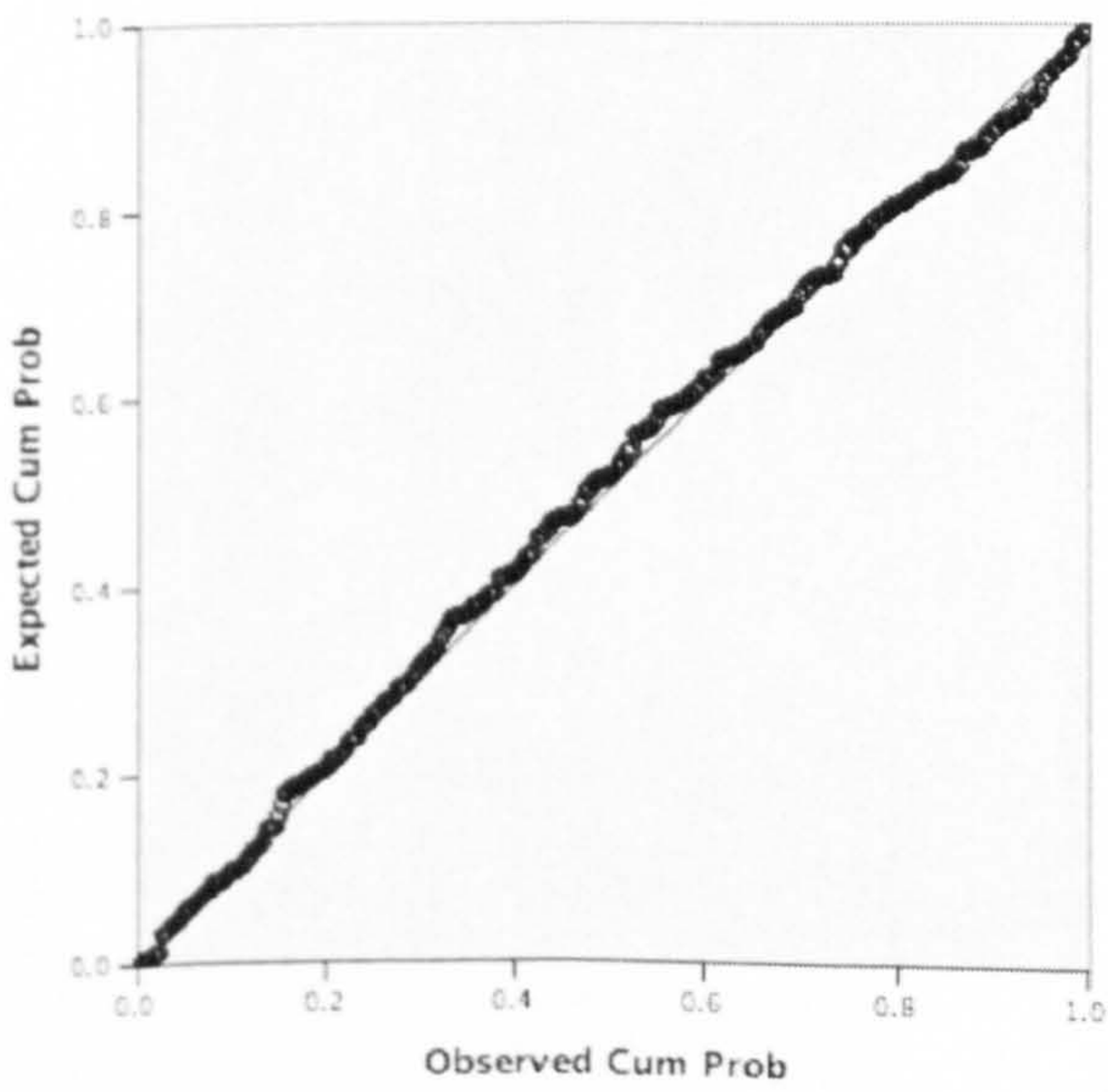
11 (a) Assumptions of linearity and homoscedasticity: Scatter plot of the residuals against the predicted values.



11 (b) Assumption of normally distributed errors: Histograms and normal P-P plots of the residuals.

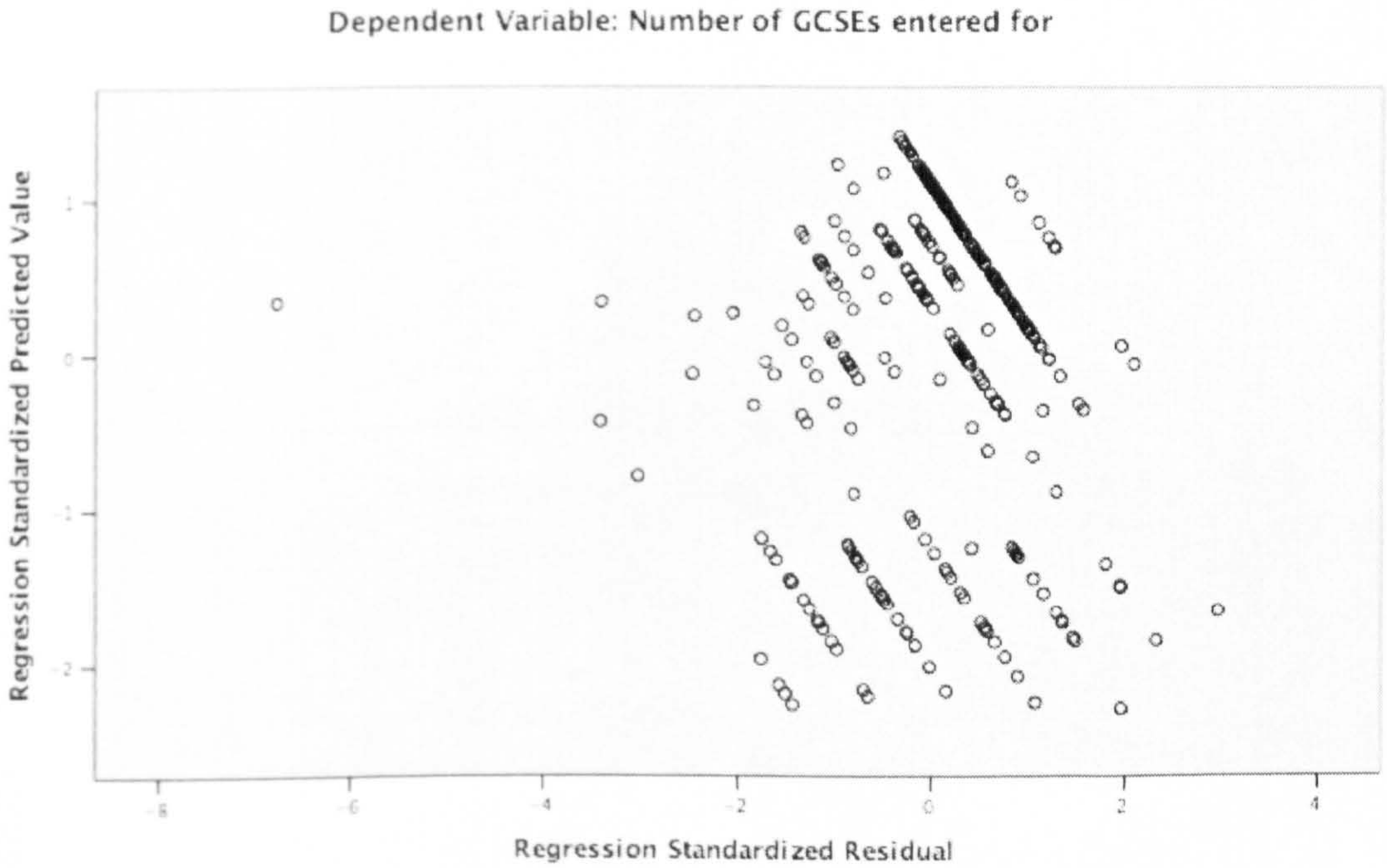


Normal P-P Plot of Regression Standardized Residual

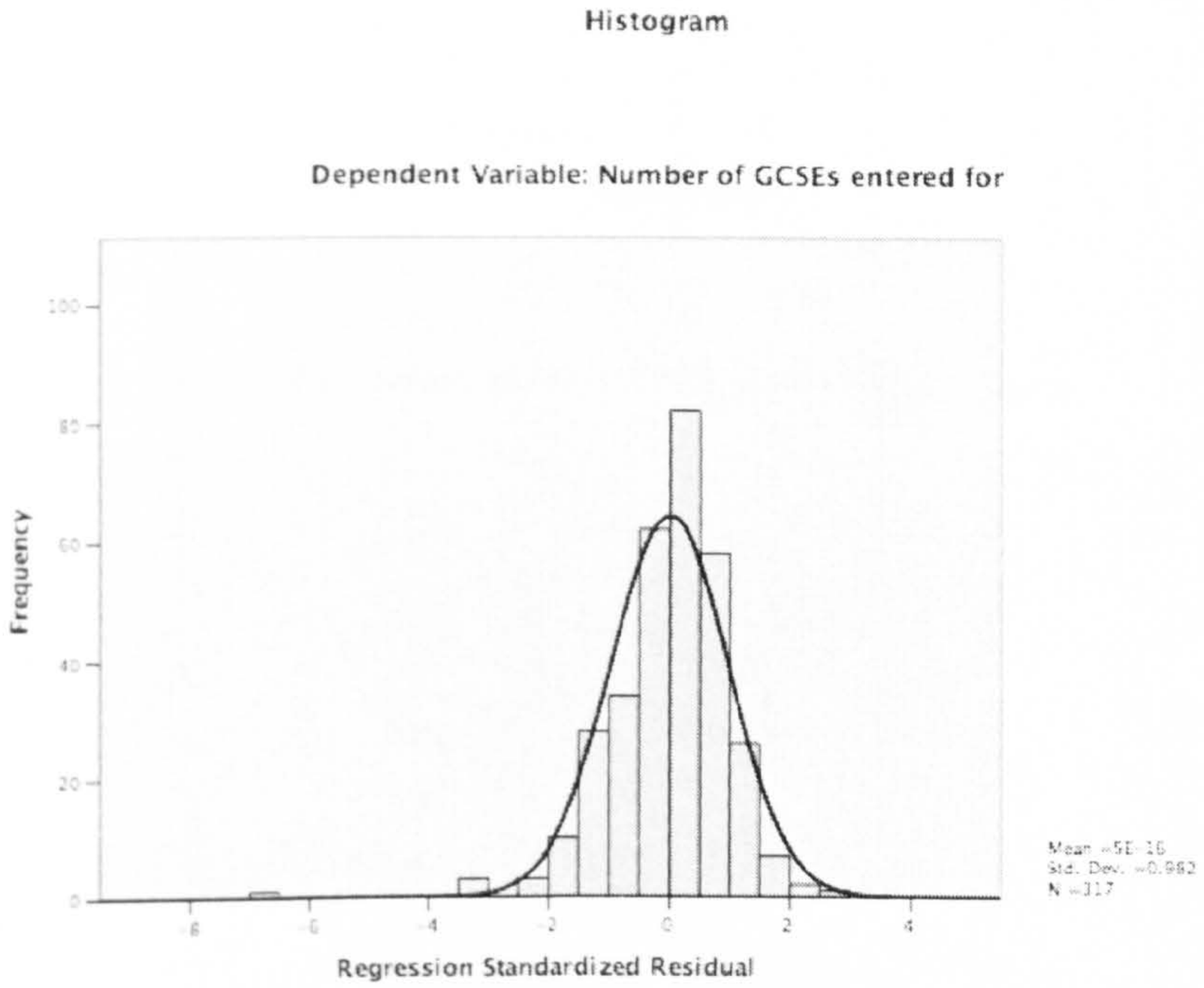


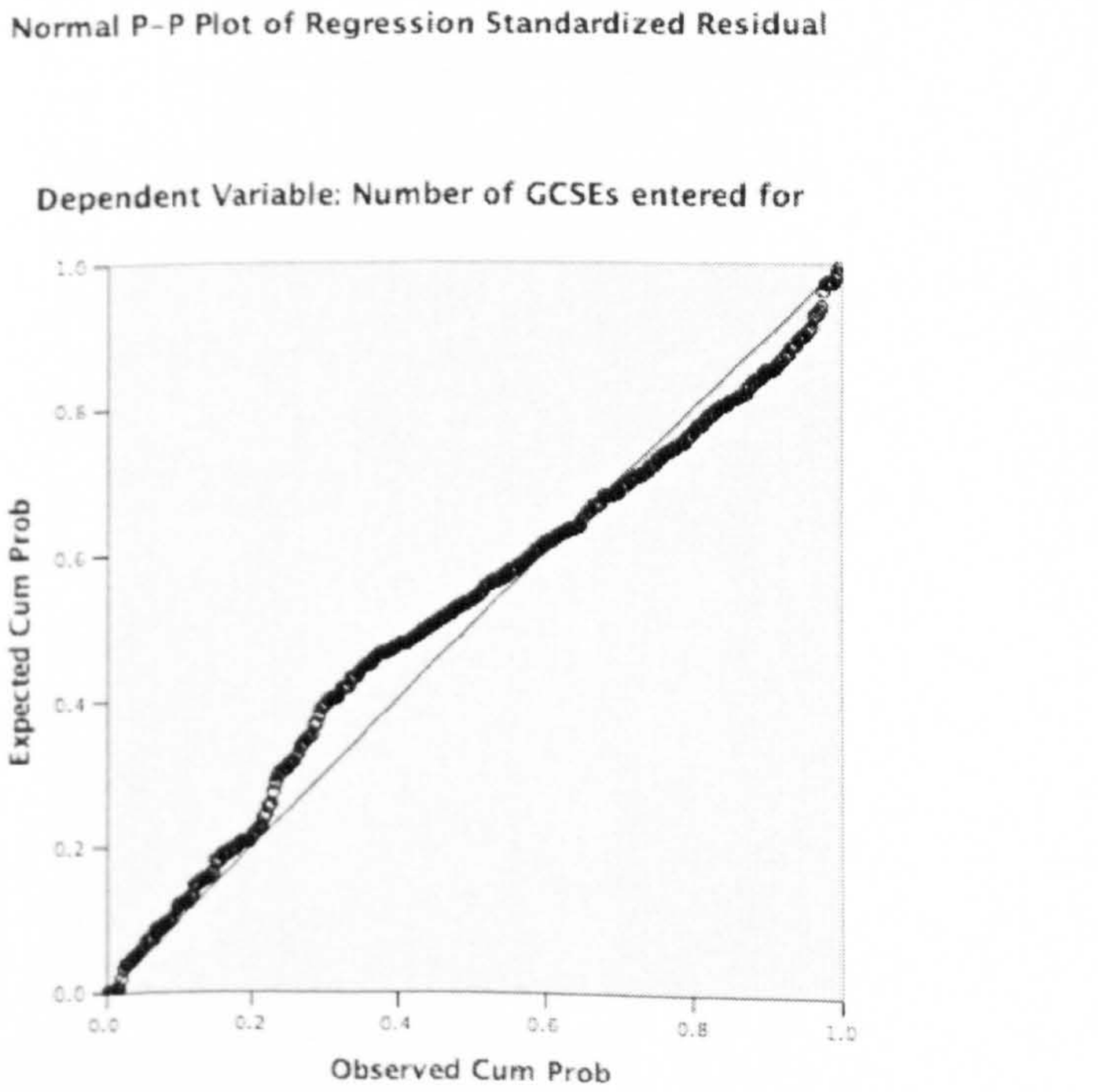
Appendix 12: Regression assumption charts, Model *ii* [Chapter 5 (Study 2)]

12 (a) Assumptions of linearity and homoscedasticity: Scatter plot of the residuals against the predicted values.



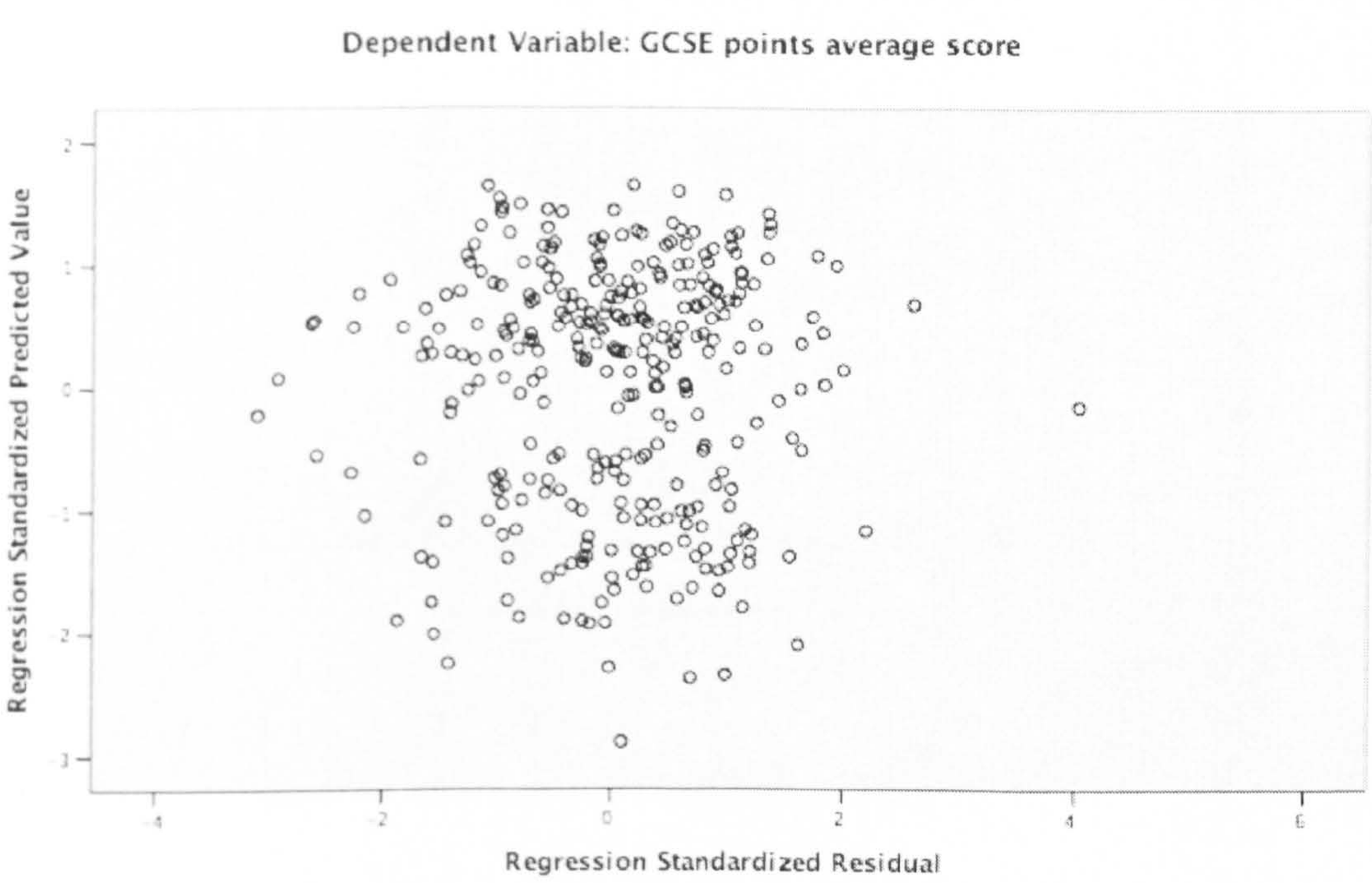
11 (b) Assumption of normally distributed errors: Histograms and normal P-P plots of the residuals.



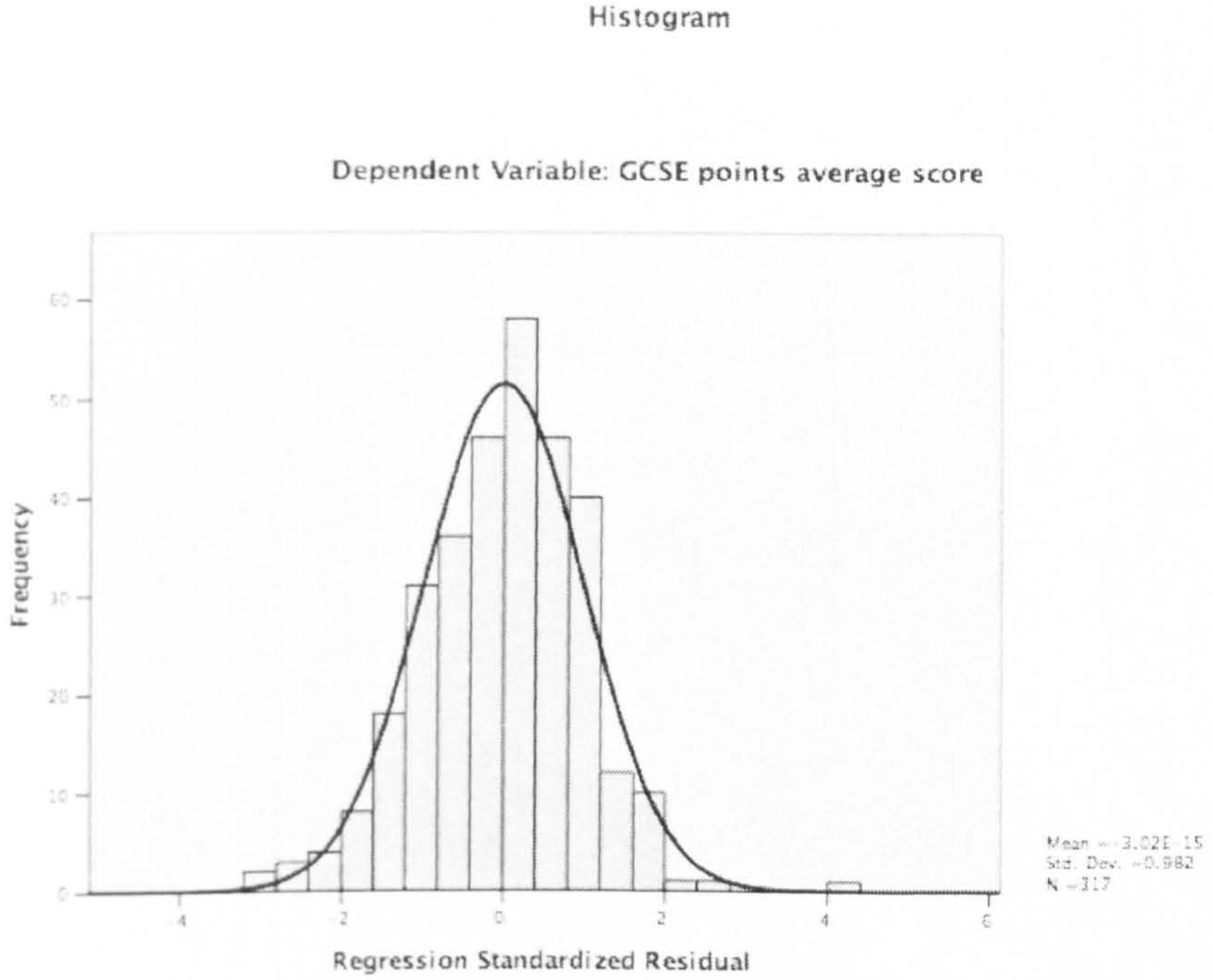


Appendix 13: Regression assumption charts, Model *iii* [Chapter 5 (Study 2)]

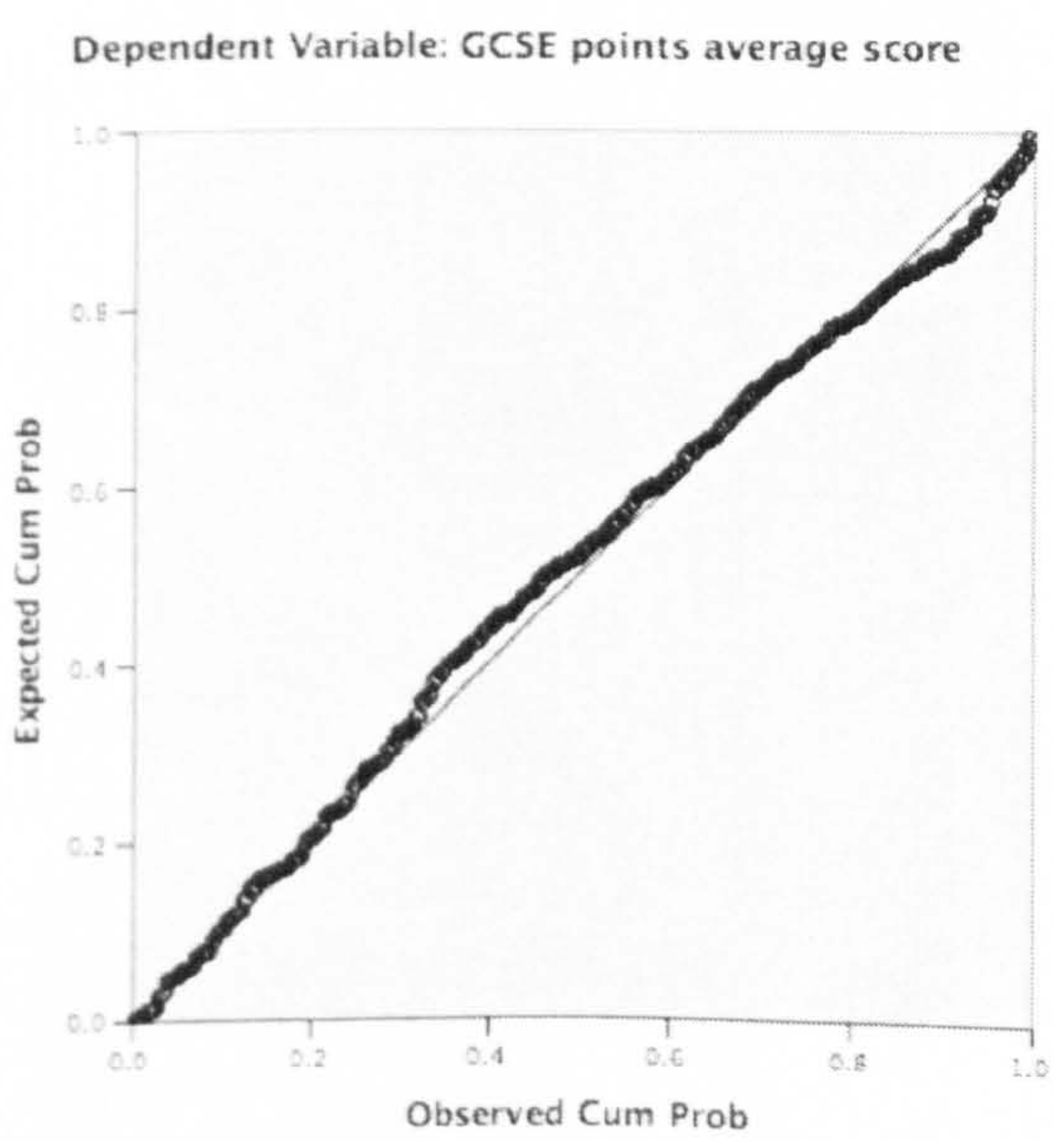
13 (a) Assumptions of linearity and homoscedasticity: Scatter plot of the residuals against the predicted values.



13 (b) Assumption of normally distributed errors: Histograms and normal P-P plots of the residuals.

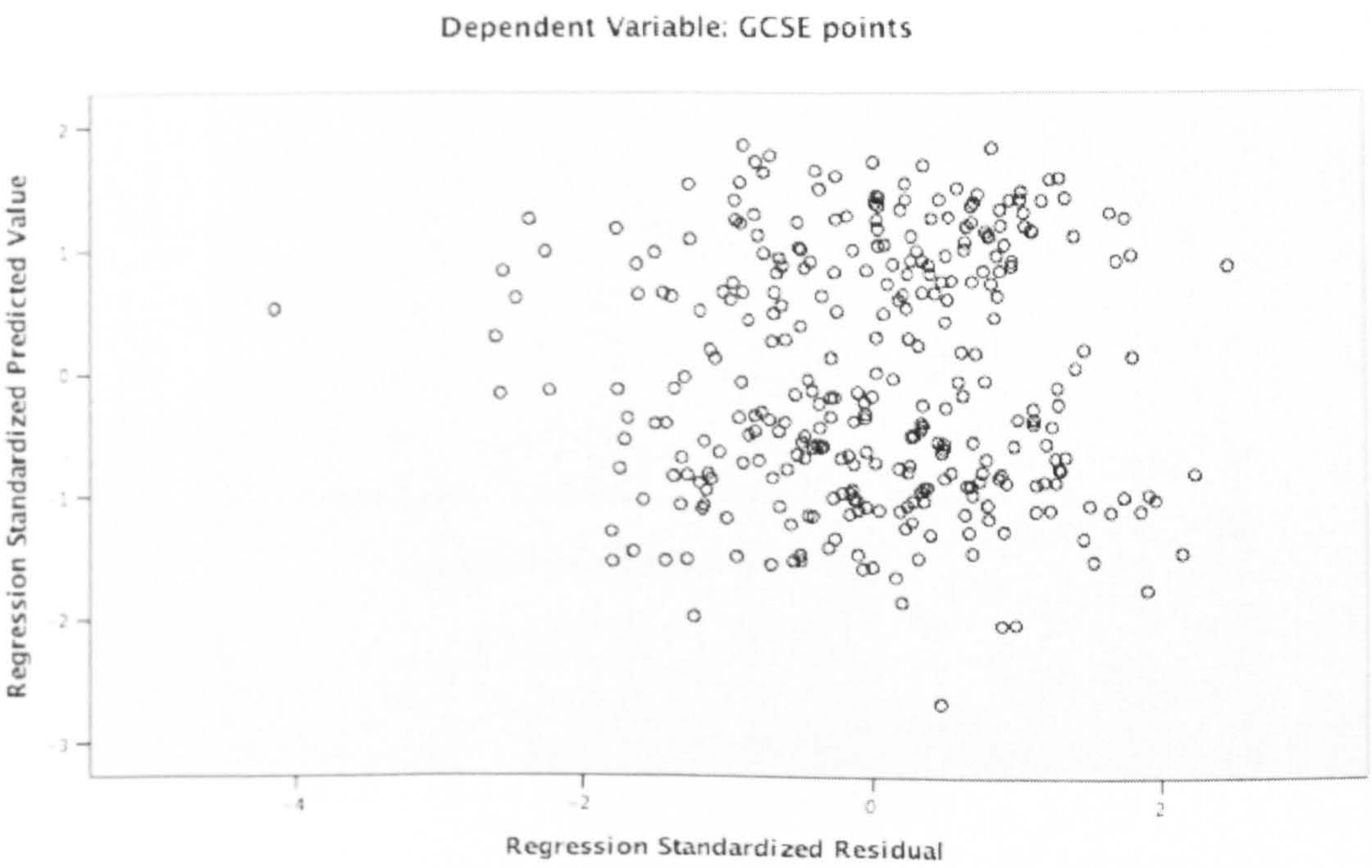


Normal P-P Plot of Regression Standardized Residual

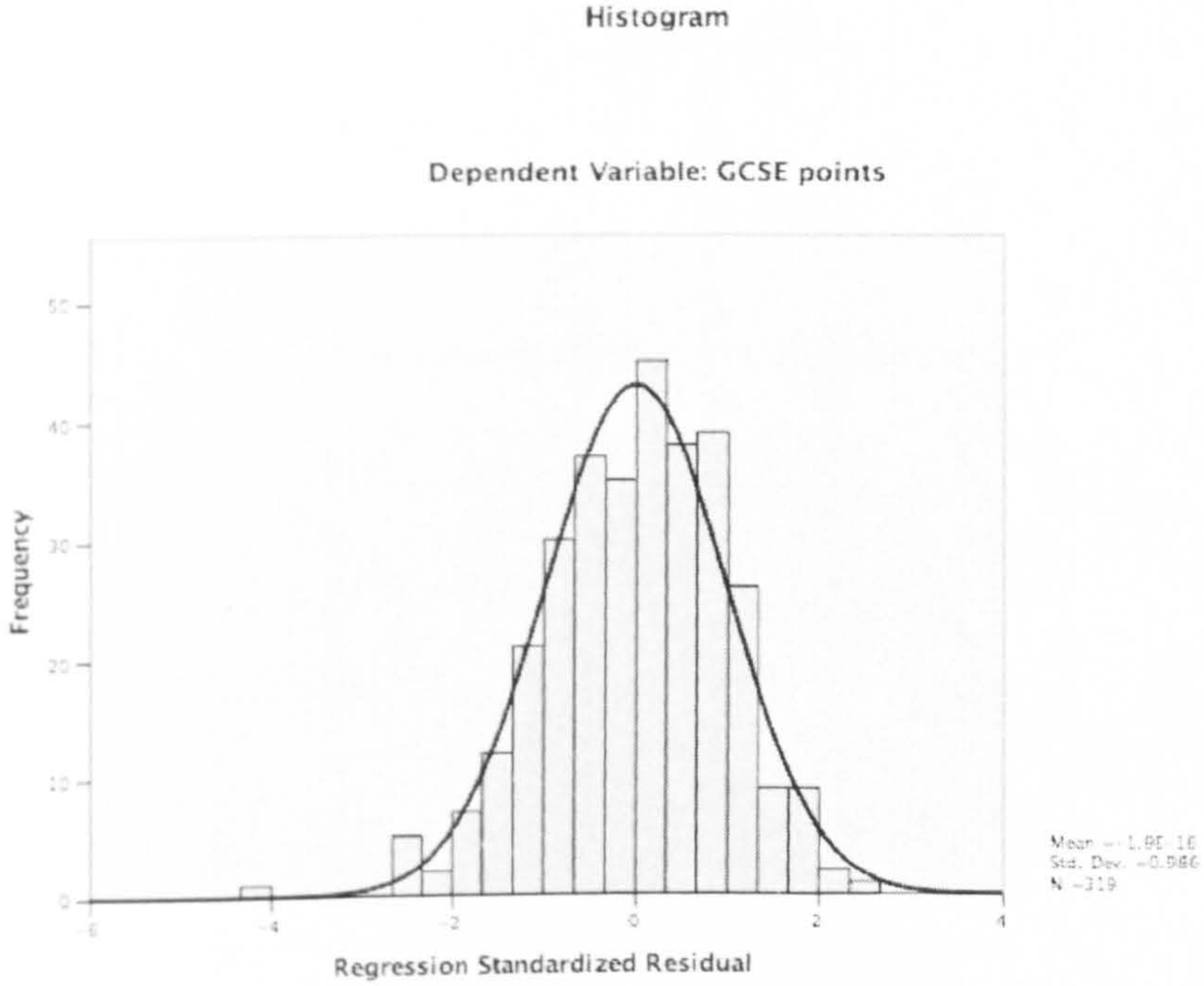


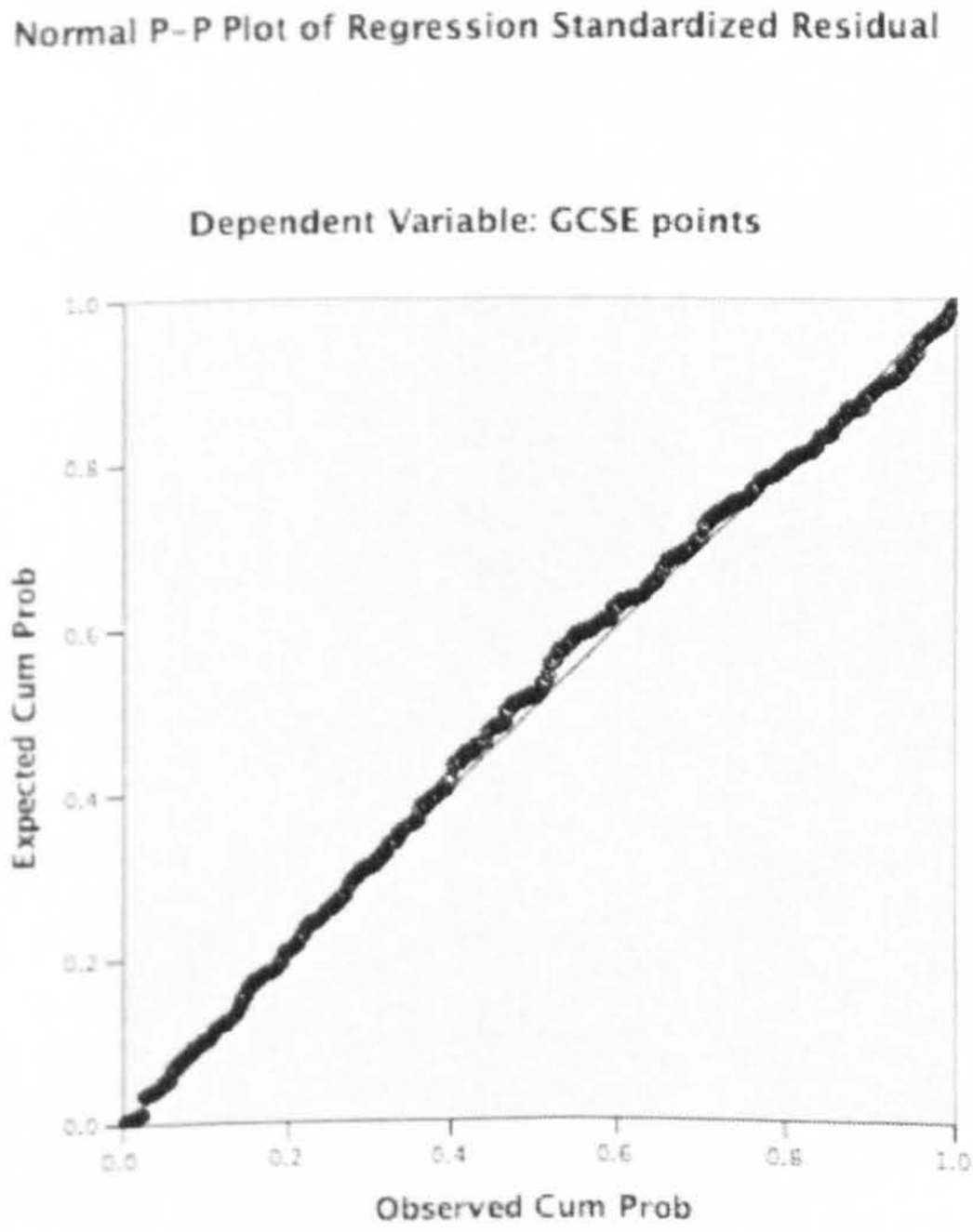
Appendix 14: Regression assumption charts, Model *iv* [Chapter 5 (Study 2)]

14 (a) Assumptions of linearity and homoscedasticity: Scatter plot of the residuals against the predicted values



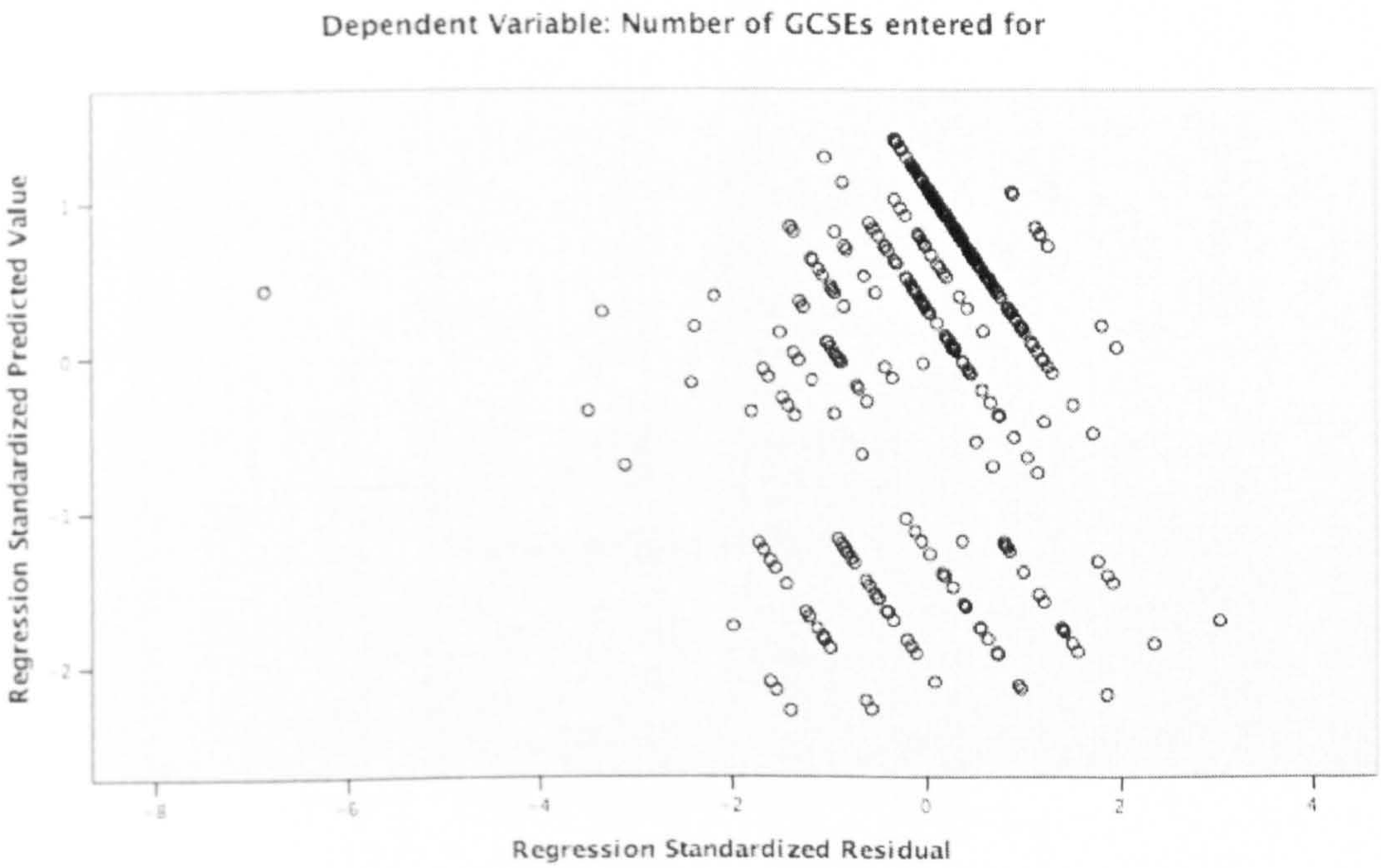
14 (b) Assumption of normally distributed errors: Histograms and normal P-P plots of the residuals.



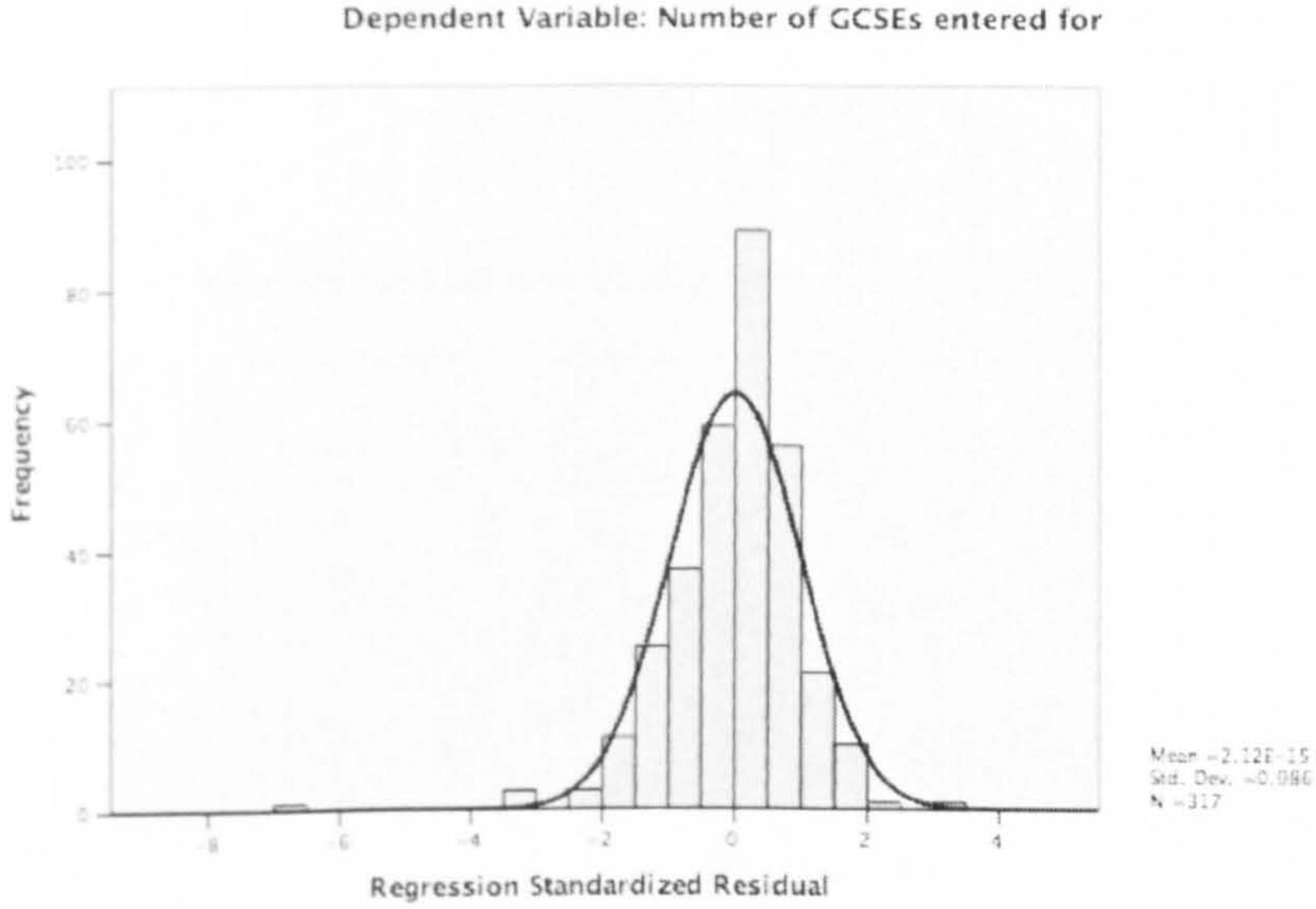


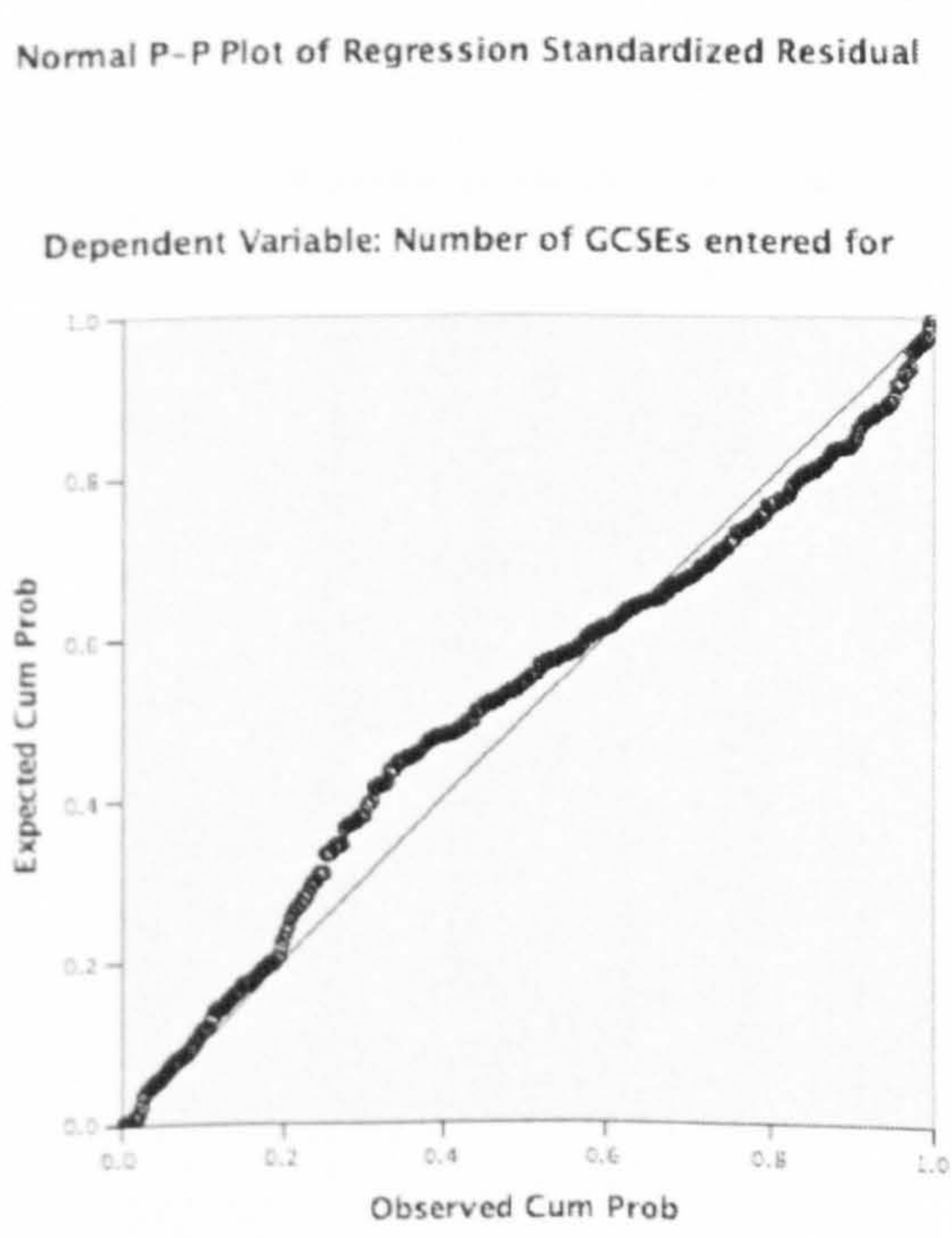
Appendix 15: Regression assumption charts, Model *v* [Chapter 5 (Study 2)]

15 (a) Assumptions of linearity and homoscedasticity: Scatter plot of the residuals against the predicted values



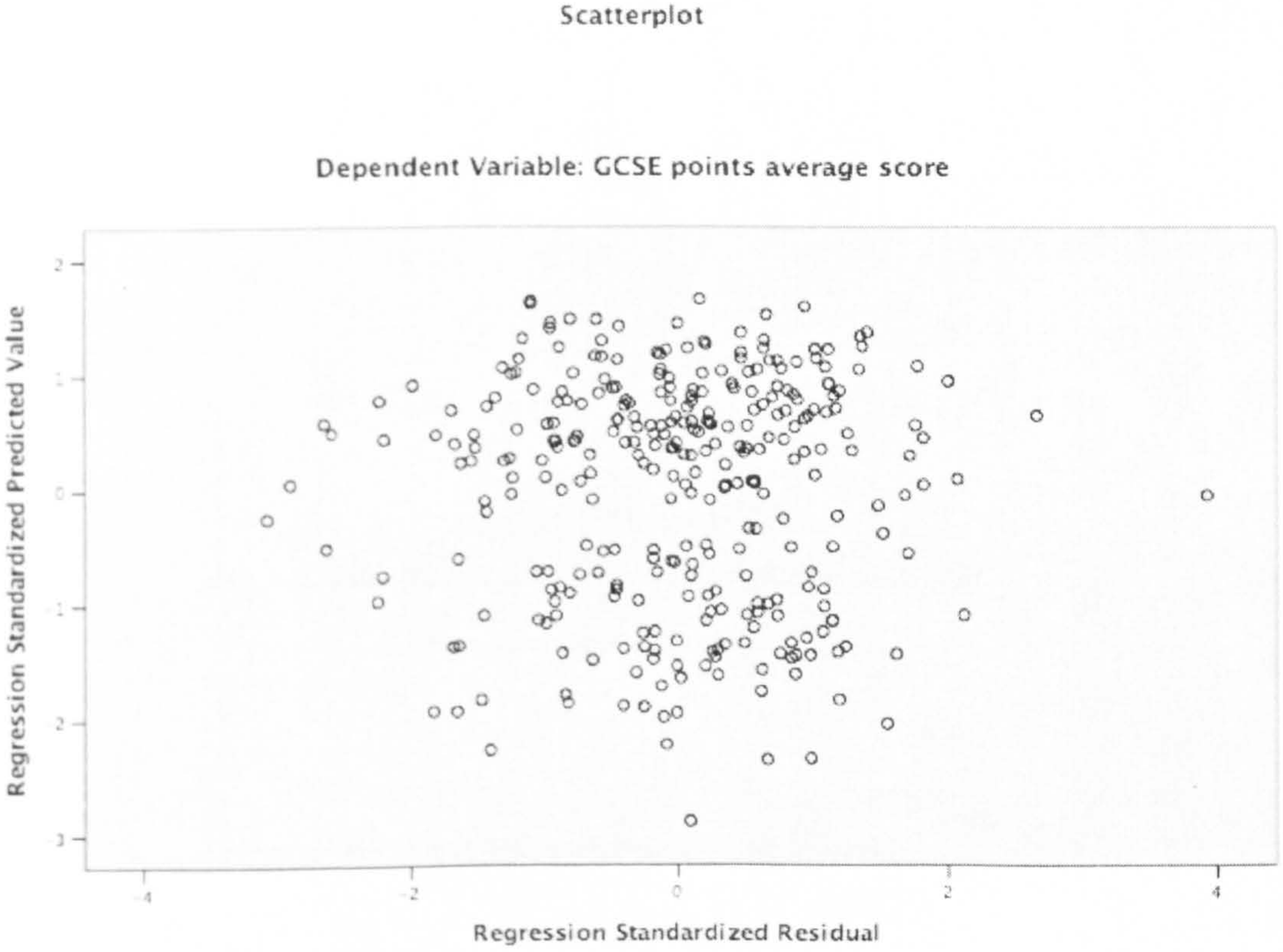
15 (b) Assumption of normally distributed errors: Histograms and normal P-P plots of the residuals.





Appendix 16: Regression assumption charts, Model *vi* [Chapter 5 (Study 2)]

16 (a) Assumptions of linearity and homoscedasticity: Scatter plot of the residuals against the predicted values



15 (b) Assumption of normally distributed errors: Histograms and normal P-P plots of the residuals.