DOCTORAL THESIS

Primary and Secondary Features in Adults with Attention Deficit Hyperactivity Disorder and Autism Spectrum Disorder.

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Primary and Secondary Features in Adults with Attention Deficit Hyperactivity Disorder and Autism Spectrum Disorder

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This dissertation was submitted in fulfilment of the program requirements for the Doctor of Philosophy Program in the Faculty of Society & Design at Bond University.

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Statement of Originality

I, Ashley C. Stark, certify that all work contained in this thesis is my own unless otherwise cited. This thesis has not been submitted previously in whole, or part, towards a degree at this or any other university.

Signed:

Ashley C. Stark

Dated: 16/02/2018

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Abstract

Attention-Deficit Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD) are considered to be diagnostically distinct disorders with notably divergent primary symptomatology (e.g., repetitive behaviour in ASD vs. inattentive behaviour in ADHD). However, individuals with these disorders often exhibit evidence of secondary or associated features that, whilst not diagnostically required, commonly occur across both groups (e.g., heightened levels of anxiety). Research exploring the primary and secondary features of ADHD and ASD has largely been drawn from clinical samples of individuals in the earlier stages of life (i.e., children and adolescents). Moreover, research on the primary and secondary features of ADHD and ASD has largely focused only on one disorder, with few studies actively conducting a comparative evaluation of the specific aspects of these disorders. To address the paucity of literature on community-based samples of adults with ADHD and ASD, the present research aimed to compare and contrast the features that present secondary to the primary diagnostic properties of these disorders, including cognitive, behaviour, and emotion-based features commonly observed in ADHD and ASD. Moreover, to investigate how both the secondary features of ADHD and ASD influence, and potentially heighten, the presentation of both other secondary and primary features of these disorders, this research aimed to explore the unique relationships between the primary and secondary features common to both disorders. A total of 278 adults participated in the two studies which comprised this research (90 diagnosed with ASD, 96 diagnosed with ADHD, and 92 neurotypical controls) with 107 identifying as male and 169 as female. Participants completed a series of self-report surveys assessing primary (i.e., core sympyomatology) and secondary features (i.e., executive functioning, inhibitory control, alexithymia, aggression, anxiety, and depression) of ADHD and ASD. In the first study, comparative analyses were conducted to assess whether differences existed with respect to the secondary features

exhibited by adults with ADHD and ASD, as well as between adults with either ADHD or ASD and neurotypical controls. In the second study, the secondary features assessed in study one were then included in separate path analyses for the adults with ADHD and ASD to explore the relationships between the primary and secondary features of these disorders. Results from the comparative analyses demonstrated that all secondary features assessed were significantly higher in adults with ADHD and ASD in comparison to neurotypical adults, but that no significant differences existed between the majority of these secondary features in adults with ADHD and ASD. Moreover, path analytic findings demonstrated that the distinct diagnostic behaviours of each disorder were impacted by the six secondary features examined in this study. These findings are important, as greater exploration of the commonalities and distinctions between ASD and ADHD will allow for the development of a more uniform clinical framework for understanding the overarching relationships between these psychopathologies. Moreover, increased knowledge of features that impact symptom presentation in ADHD and ASD may further inform approaches to treatment and the reduction of problematic behaviour (and subsequent functional impairment) in adults with these disorders.

Chapter One: Introduction

Current diagnostic criteria have classified Attention-Deficit Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD) as two distinct neurodevelopmental conditions with notably divergent symptomatology (American Psychiatric Association [APA], 2013). Research has demonstrated however, that individuals with these disorders often experience a range of similar features, secondary to their core diagnostic characteristics, that persist across the lifespan (e.g., Barkley, 1994; 1997; 2012a; 2012b; Rajendran & Mitchell, 2007). These features can involve atypicalities in cognition, behaviour, or emotion (Barkley, 1997; Barnhart & Buelow, 2017; Berthoz & Hill, 2005; Pugliese et al., 2015; Schecklemann et al., 2012), and carry the potential to compound impairments to age-appropriate daily functioning (Barkley & Murphy, 2010; Langberg et al., 2008; 2011; Young, 2005). The research exploring these secondary features in ADHD and ASD spans several decades, but it is limited by several significant methodological gaps. Studies on ADHD and ASD typically focus on clinical samples (e.g., individuals accessing mental health practitioners or facilities; Biederman et al., 1991; 1992; 2008; Pennington & Ozonoff, 1996) of individuals in the earlier stages of life (i.e., children and/or adolescents; Dominick et al., 2007; May, Cornish, & Rinehart, 2012). Moreover, research has largely been conducted exclusively on ADHD or ASD, with few studies actively investigating various aspects of these disorders from a comparative perspective (Davis & Kollins, 2012; Rommelse, Buitelaar, & Hartman, 2017). Consequently, the current literature does not provide a robust account of how the primary and secondary features of ADHD and ASD present in adulthood in the general population and, more importantly, fails to delineate the similarities in secondary features that are often paradoxically presumed to substantially impact the distinct diagnostic characteristics of these disorders.

To address the paucity of literature concerning the secondary features of adults with ADHD and ASD, the present research aimed to explore the nature of these features in a two-stage investigation. First, the cognitive, behaviour, and emotion-based features secondary to the primary diagnostic properties of ADHD and ASD were compared across a community sample of adults meeting the selection criteria for the ADHD, ASD or neurotypical control groups of this study (Study 1). Second, path analytic modelling was conducted on the ADHD and ASD groups, exploring the unique relationships between the primary and secondary features of both disorders (Study 2).

Chapters 2 and 3 of this thesis outline the aetiological underpinnings as well as the diagnostic evolution and reported impairments to functionality in ADHD and ASD, respectively. The literature reviewed in these chapters suggests that ADHD and ASD both tend to persist, diagnostically, well into adulthood. These chapters both further address the importance of continuing to explore all aspects of ADHD and ASD that impact the progression of these disorders across the lifespan, including prominent, non-diagnostic (i.e., secondary) features, and other disorders with similar functional trajectories (i.e., other neurodevelopmental disorders).

Chapter 4 of this thesis discusses the secondary cognitive (executive dysfunction, disinhibition, alexithymia), behaviour (aggression), and emotion-based (anxiety, depression) features of ADHD and ASD as they present in adults with these disorders. The literature reviewed in this chapter reveals points of convergence (e.g., atypicalities in higher order thought processes) and divergence (e.g., deficits in the processing of emotion-based information) across the secondary features of ADHD and ASD, but ultimately suggests that the functionality of adults with either disorder appears to be adversely impacted by the presence of these secondary features. A review of the literature in this chapter, and the points

of ADHD-ASD convergence in particular, highlight the need for further collective research on adults with ADHD and ASD.

Chapter 5 of this thesis outlines the methods and procedures used to apply a Multivariate Analysis of Covariance (MANCOVA) and Discriminant Function Analysis with a community based sample of ADHD, ASD and neurotypical adults (i.e., Study 1). This chapter describes the participants, materials, and data-collection procedures involved in these comparative analyses. Chapter 5 also provides the details of the analyses comparing the secondary cognitive, behaviour, and emotion-based features of ADHD and ASD. The data obtained for the purpose of Study 1 revealed that reportedly higher levels of all secondary features assessed in this study existed in adults in the ADHD and/or ASD groups in comparison to the neurotypical control group.

Chapter 6 of this thesis discusses ways to expand upon the comparative findings of Study 1, including the application of path analytic modelling of the primary and secondary features of ADHD and ASD. This chapter includes a subsequent introduction to behaviour selection theories that could be applied to potential path models of ADHD and ASD, followed by a detailed review of a theoretical model established by psychiatrist Russell Barkley (1997). The literature discussed in relation to Barkley's model addresses the fact that this exploration of behaviour selection has largely focused on children with ADHD, and further argues ways in which it can be applied to people with both ADHD and ASD in adulthood.

Chapters 7 and 9 of this thesis investigate the potential associations between the primary and secondary features of ADHD and ASD, respectively. The literature reviewed in these chapters explores, at length, ways in which Barkley's model (1997) can be applied to the primary (i.e., key symptoms) and secondary (i.e., aggression) behaviour features of adults with these disorders. Relevant research from previous chapters (i.e., 2, 3 and 4) is drawn on

to strengthen the rationale behind using this model to explain these primary and secondary features of ADHD and ASD and to further highlight its unique application to these disorders in this context.

Chapters 8 and 10 of this thesis outline the methods and procedures used to apply path analyses to the ADHD and ASD study groups, respectively (Study 2). These chapters describe the participants, materials, and data-collection procedures involved in the modelling. These chapters also provide the details of the analyses, exploring the exent to which the primary symptoms, and the secondary cognitive (executive dysfunction, disinhibition, alexithymia), behaviour (aggression), and emotion-based features (anxiety, depression) of these disorder impact one another. The data obtained for the purpose of these analyses revealed two separate paths (i.e., one for ADHD, one for ASD) that significantly predicted both the primary (i.e., core sympoms) and secondary behaviour (i.e., aggression) in adults in the ADHD and ASD groups of Study 2.

Chapter 11 of this thesis presents an integrative discussion of the research gaps investigated by Studies 1 and 2 and the major results obtained in each study. The implications of research findings for clinical practice in the areas of reducing functional impairment in ADHD and ASD adults with these disorders are also discussed. This chapter also reviews the limitations inherent in the investigative procedures used to conduct Studies 1 and 2.

Chapter Two: Attention-Deficit Hyperactivity Disorder

ADHD is a neurodevelopmental condition characterised by developmentally-atypical levels of inattention, hyperactivity and impulsivity (APA, 2013). Current diagnostic characterisations of ADHD consider these three primary symptom clusters, as well as the associated impairments in age-appropriate functioning, to be the core features of ADHD. A review of prevalence rates reveals that two prominent trends exist with respect to the presentation of ADHD in the general population. First, ADHD is more commonly found in males, with only one female diagnosed with the disorder for every three males (APA, 2013; Barkley, 2006; Nolan et al., 2001). Second, ADHD is currently one of the most common childhood disorders, existing in approximately 6-7% of children in Australia (Australian Guidelines on ADHD, 2009), 4-5% of children in the United Kingdom (UK; National Institute for Health and Clinical Excellence [NIHCE], 2008; Polanczyk & Rohde, 2007), and 7-10% of children in North America (Visser, Danielson, & Bitsko, 2013). A review of global prevalence rates in adults demonstrates that ADHD carries the potential to persist across the lifespan, existing in approximately 2-4% of individuals aged 18 years or older (APA, 2013; Fayyad et al., 2007; Kessler et al., 2006; Simon, Czobor, Bálint, Mészáros, & Bitter, 2009; Thomas, Sanders, Doust, Beller, & Glasziou, 2015).

Although slight variations exist in ADHD prevalence figures, the global distribution of the disorder does not appear to differ significantly across geographic locations, emphasising the importance of exploring its genetic contributions (Barkley, 2015d; Fayyad et al., 2007; Kessler et al., 2006). That importance is further supported by the fact that a higher prevalence of ADHD symptomatology has been observed in parents and siblings of children with ADHD (Barkley, DuPaul & McMurray, 1990; Biederman et al., 1992; Biederman, Keenan & Faraone, 1990; Cantwell, 1972; Morrison & Stewart, 1971; Smalley et al., 2002; Welner, Welner, Stewart, Palkes, & Wish, 1977), with the frequency of primary ADHD

characteristics (e.g., hyperactivity) in children associated with heightened hyperactive and inattentive behaviour in their biological parents (Bornovalova, Hicks, Iacono & McGue, 2010; Macek, Gosar, & Tomori, 2012). Current considerations of ADHD view a person's genetic composition (the innate part of the individual which, as far as is understood, cannot be changed) as containing certain liabilities or susceptibilities that can then, in combination with specific environmental events, activate a disorder like ADHD through changes in brain structure and functionality (Barkley, 2015d). Consequently, although the exact aetiology of the disorder is yet to be confirmed, a range of genetic and neurobiological factors, and their interactions with the environment, are thought to contribute to the primary presentation of ADHD (Retz & Klein, 2010). This chapter will elaborate on these aetiological factors, as well as discuss the evolution in conceptualisation and diagnostic descriptions of ADHD. This discussion will encompass a review of the research into ADHD conducted over the past century in order to address two key issues: the ways in which research findings have shaped and advanced understanding of the core features of ADHD, and the relative impacts of those core features on daily functioning across the lifespan

The Aetiology of Attention-Deficit Hyperactivity Disorder

Over the last four decades, an extensive exploration of the biological contributions to ADHD in the form of familial, twin and adoption studies has shown that genetic and heritable factors are highly influential in its onset and presentation. Studies involving children with ADHD who were adopted and subsequently raised by non-biological parents have demonstrated a consistent trend towards the biological parents yielding significantly higher levels of ADHD symptomatology than the adoptive parents (Cantwell, 1972; Larsson et al., 2013; 2014; Morrison & Stewart, 1973; Sprich, Biederman, Crawford, Mundy & Faraone, 2000). Additionally, twin studies have demonstrated a greater concordance rate of ADHD symptomatology between monozygotic (identical) versus dizygotic (fraternal) twins (see

meta-analysis by Nikolas & Burt, 2010; also see Goodman & Stevenson, 1989; Larsson et al., 2013; 2014; O'Connor, Foch, Sherry, & Plomin, 1980; Willerman, 1973). Accordingly, current heritability estimates suggest that up to 80% of the variation in the presentation of primary ADHD characteristics (e.g., impulsivity) can be accounted for by genetic factors (Boomsma, Cacioppo, Muthen, Asparouhov, & Clark, 2007; Faraone et al., 2005; Sprich et al., 2000). Nevertheless, despite high heritability rates, the transmission mode of ADHD symptomatology is considered to be non-Mendelian, since concordance rates between monozygotic twins are less than 100%. More specifically, although some form of genetic heritability appears to exist in ADHD, the pattern of inheritance has not been linked to the modulation or mutation of one single gene, with evidence implicating that this complex disorder is polygenetic, and that genetic contributions to ADHD are likely to be influenced by extraneous factors present in the environment (Biederman, Faraone, Monuteaux, Bober, & Catogen, 2004; Brookes et al., 2006; Yan et al., 2013).

Despite the heavy influence of heredity on the expression of ADHD reported in the research, up to one-quarter of ADHD cases are thought to result from exposure to adverse environmental factors which interact with pre-existing genetic susceptibilities for the disorder and subsequently impede neurological development (Nigg, 2006). These environment-gene interactions contribute to the variability in ADHD symptomatology not accounted for by the genetic aetiology of the disorder which possibly occurs during the foetal stage and, to a lesser extent, during postnatal development (see meta-analysis by Nikolas & Burt, 2010; also see Burt, Larsson, Lichtenstein & Klump, 2012). For over 50 years, researchers have explored the extent to which various pregnancy- and birth-related factors may be associated with ADHD. Some studies have not been able establish a clear link between the two (Wagner, Schmidt, Lemery-Chalfant, Leavitt, & Goldsmith, 2009). In contrast, the majority of research mapped a pregnancy/birth-ADHD association, including factors such as a long labour

(Claycomb, Ryan, Miller, & Schnakenberg-Ott, 2004); young maternal age (Claycomb et al., 2004; Denson, Nanson, & McWatters, 1975; Hartsough & Lambert, 1985; Minde, Webb, & Sykes, 1968); low maternal education (Claycomb et al., 2004); maternal obesity (Rodriguez, 2010); premature birth (Galéra et al., 2011); underweight birth (Breslau et al., 1996; Elgen, Holsten, & Odberg, 2013; Galéra et al., 2011; Heinonen et al., 2010; Mick, Biederman, Faraone, Sayer, & Kleinman, 2002; Schothorst & van Engeland, 1996; Sykes et al., 1997; Szatmari, Saigal, Rosenbaum, & Campbell, 1993; Wagner et al., 2009); and the presence of delivery complications (e.g., respiratory distress, birth asphyxia, and preeclampsia; Claycomb et al., 2004; Getahun, Sculier, Sismanidis, Grzemska, & Raviglione, 2012; Sharp et al., 2003). Further, there is evidence to support a link between ADHD and exposure to environmental toxins in utero, such as tobacco (see meta-analyses by Langley, Rice, van den Bree, & Thapar, 2005; also see Chiodo, Jacobsen & Jacobsen, 2004; Fielding, 1985; Galéra et al., 2011; Langley et al., 2007; Mick et al., 2002; Milberger, Biederman, Faraone, Chen, & Jones, 1996); alcohol (see meta-analysis by Langley et al., 2005); combined tobacco and alcohol (see review by Linnet et al., 2003; also see Ekblad et al., 2010; de Zeeuw, Zwart, Schrama, van Engeland, & Durston, 2012; O'Malley & Nanson, 2002; Rodriguez, 2009); genitourinary infections (Mann & McDermott, 2011); and household cleaning products/outdoor pesticides (Nigg, 2006; Polańska, Jurewicz, & Hanke, 2013; Sagiv et al., 2010). There is no robust evidence to suggest that, postnatally, the social environment alone (e.g., poor parenting styles [Danforth, Anderson, Barkley, & Stokes, 1991; Johnston & Mash, 2001], or overexposure to television and video games [Acevedo-Polakovich, Lorch, & Milich, 2007; Barkley, Murphy, & Fischer, 2008; Lingineni et al., 2012; Rietveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma, 2004]) can lead to a child developing ADHD (Barkley, 2015 Ch 14). Nevertheless, socially-oriented, such as low socio-economic status (Goodman & Stevenson, 1989; Hjern, Weitoft, & Linbald, 2010; Langley et al., 2007), or

being raised in a single parent home (Galéra et al., 2011), may interact with neurobiologic and genetic susceptibilities postnatally.

Environmental factors carry the potential to influence the development of ADHD symptomatology in individuals who are genetically vulnerable to neurodevelopmental disorders, by interacting with certain genetic predispositions and, subsequently, influencing neurological development (e.g., neurotransmitters and neurobiological structures within the brain; Barkley, 2015d; Fayyad et al., 2007; Kessler et al., 2006). Early genetic explorations of ADHD focused on dopamine regulating genes, but more recent research has utilised genomewide scans to identify multiple genetic sites related to dopamine, serotonin, and norepinephrine function in the brain, as well as sites associated with cell migration and brain growth, among other important processes involved in neurological development (see meta-analyses by Faraone & Mick, 2010; Franke, Neale, & Faraone, 2009; Gizer, Ficks, & Waldman, 2009). Deficiencies in several serotonin receptors have been identified in people with ADHD (Asherson, 2004), with low levels of this neurotransmitter demonstrating an association with aggressive, impulsive and antisocial behaviour in individuals with this disorder (Flory, Newcorn, Miller, Harty, & Halperin, 2007; Halperin et al., 1994; Pine et al., 1997; Stadler et al., 2007). Additionally, research has demonstrated that there is up to 70% more dopamine transporter activity than normal in the frontal area of the ADHD brain, with the suggestion that this neurotransmitter is being recycled before it is absorbed by the necessary receptors (Selikowitz, 2009). Accordingly, studies have increasingly begun to combine explorations of neurophysiology with molecular genetics in an attempt to further understand potential genetic influences on the topography and function of neurobiological structures commonly associated with ADHD symptomatology (Poelmans, Pauls, Buitelaar, & Franke, 2011; Stergiakouli et al., 2012).

Structural neurophysiological studies utilising neuroimaging research (e.g., positron emission tomography [PET], magnetic resonance imaging [MRI] and functional MRI [*f*MRI]) have revealed some interesting atypicalities in the development of certain neurobiological structures in the brains of children and adults with ADHD. A longitudinal study using MRIs and *f*MRIs to assess cortical maturation discovered that, in comparison to neurotypical controls, children with ADHD were significantly delayed in their neurological development, particularly in the prefrontal cortex (Shaw et al., 2006; 2007). Moreover, research exploring overall brain size in individuals with ADHD has revealed that significantly smaller cerebral volumes are frequently observed in children and adolescents with ADHD in comparison to neurotypical controls (Aylward et al., 1996; Castellanos et al., 1996; 2001; 2002; Durston et al., 2004; Filipek et al., 1997; Seidman, Valera, & Makris, 2005; Tannock, 1998; Valera, Faraone, Biederman, Poldrack, & Seidman, 2005). Subsequent meta-analyses have since intimated this volume reduction can be further localised to particular brain areas (e.g., corpus callosum, cerebellum, basal ganglia), as well as overall grey matter volume in the brain (Ellison-Wright, Ellison-Wright & Bullmore, 2008; Frodl & Skokauskas, 2012; Nakao, Radua, Rubia, & Mataix-Cois, 2011; Proal et al., 2011; Valera, Faraone, Murray, & Seidman, 2007). Research has further demonstrated that most volume differences appear to normalise with age. However, lower mean surface-wide cortical thickness and regional grey matter density can persist into adulthood, particularly in those whose ADHD symptoms persist (Proal et al., 2011; Shaw et al., 2006).

A review of the research on specific neurobiological structures thought to be atypical in the ADHD brain suggests that a smaller and less active anterior cingulate cortex has been observed in the brains of children with ADHD (Bush et al., 1999; Hong et al., 2014; Konrad, Neufang, Hanisch, Fink & Herpertz-Dahlmann, 2006; Makris et al., 2007). For example, in an MRI study of 19 children with ADHD (91% male; 10 to 18 years old), a bilateral

reduction in the anterior cingulate cortex grey matter volume was observed, with abnormality in the right anterior cingulate cortex associated with inattention and disinhibition (Lopez-Larson et al., 2012). Moreover, research on amygdala-cortical intrinsic function and overall amygdala volume has demonstrated that atypicalities exist in some individuals with ADHD; however, research at this stage has not been consistent in its structural (e.g., variations in volume) or functional (e.g., hypo- vs. hyperfunctionality) findings with respect to this particular neurobiological region in ADHD (Hulvershorn et al., 2014; Perlov et al., 2008).

The frontal lobes of the brain have also been reported to be consistently atypical in individuals with ADHD. Assessment of brain function in children with ADHD has yielded smaller amplitudes in a specific type of Evoked Response Potential (ERP) associated with the functionality of the prefrontal regions of the brain (Johnstone, Barry, & Anderson, 2001; Pliszka, Liotti & Woldorff, 2000). In adults with ADHD, studies assessing cerebral glucose metabolism have indicated diminished metabolism particularly in the frontal regions of the brain (Schweitzer et al., 2003; Zametkin et al., 1990). Additionally, assessment of cerebral blood flow using Single-Photon Emission Computed Tomography (SPECT) revealed decreased blood flow to the prefrontal regions and pathways connecting these regions in children with ADHD (Hendren, De Backer, & Pandina, 2000). Lower blood flow in the frontal region of the brain has been correlated with reduced brain activity in ADHD, as measured by electroencephalograph (EEG; Gustafsson, Thernlund, Ryding, Rosen, & Cederblad, 2000). Exploration of the functional interregional interconnectivity in the brain has demonstrated reduced functional connectivity and activity in the fronto-striatal and fronto-parietal circuitry (Arnsten, Steere, & Hunt, 1996; Ashtari et al., 2005; Benton, 1991; Bush, Valera, & Seidman, 2005; Cortese et al., 2012; Cubillo & Rubia, 2010; Dickstein, Bannon, Castellanos, & Milham, 2006; Fassbender & Schweizer, 2006; Karch et al., 2014;

Konrad & Eickhoff, 2010; Konrad, et al., 2012; Makris et al., 2007; Morein-Zamir et al., 2014; Rüsch et al., 2007; Tian et al., 2006).

The neural bases of higher-order cognitive abilities, such as executive functioning, have been associated with the frontal lobes (Goldman-Rakic, 1995; Miller, 1999; Miller & Cohen, 2001; Stuss & Benson, 1984) and, more specifically multiple cortico-cortical and cortical striatal loops within the brain (D'Esposito, 2007; Miller & Cohen, 2001). Historically, research on executive functioning has been associated with analyses of damage to such brain regions, such as the prefrontal cortex (Baddeley, 1998; Shallice & Burgess, 1991). Development of the neural circuitry that forms the core of the prefrontal cortex starts as early as the second trimester of pregnancy (Goldman-Rakic, 1987). However, the frontal lobes are the areas of the brain that take the longest to mature post-natally (Giedd et al., 1999; 1999; Huttenlocher, 1979; 1990; Sowell et al., 1999a; 1999b; Sowell, Delis, Stiles, & Jernigan, 2001). As such, different domains of executive functioning may mature at different rates depending on developmental trajectories within these brain regions (Anderson, Anderson, Northam, Jacobs, & Mikiewicz, 2002; Fischer, Biscaldi, & Gezeck, 1997; Zelazo, Reznick, & Pinon, 1995). More specifically, it has been speculated that, due to the long and complex developmental process of the frontal lobes, there is an increased susceptibility to dysfunction and irregularities in this brain region across the lifespan (Bradshaw, 2001).

Damage to the prefrontal region of the brain has also been associated with behavioural disturbances such as situational inappropriateness, disinhibition, and impulsivity (Barrash, Tranel, & Anderson, 2000; Beer, John, Scabini, & Knight, 2006; Fuster, 1997; Hornak et al., 2003; Stuss & Benson, 1984). Deficits in the prefrontal cortex, and the frontal lobe in general, have also been observed in individuals displaying higher levels of aggression. More specifically, brain lesion studies have revealed that damage to the ventromedial frontal lobe can impair an individual's capacity to consider emotions when making a decision involving a

significant degree of uncertainty (Damasio, Everitt, & Bishop, 1996; Damasio, Tranel, & Damasio, 1991), as well as their ability to self-regulate certain forms of physical aggression (Pietrini, Guazzelli, Basso, Jaffe, & Grafman, 2000). Damage to the orbitofrontal cortex has also been implicated in the self-regulation process, with resultant difficulties in suppressing inappropriate action in response to provocation (Pietrini et al., 2000). Accordingly, in addition to being the most consistently documented atypical neurobiological region of the ADHD brain, functional abnormalities in the circuitry of the frontal lobes have been associated with greater severity of ADHD symptomatology (Mattes, 1980; Monk et al. 2009). It was this connection between front-brain abnormalities and atypical behaviour (e.g., impulsivity) that initially generated neurobiological research across neurodevelopmental disorders such as ADHD (Levin, 1938; Mattes, 1980; Pennington & Ozonoff, 1996).

The Conceptual and Diagnostic Evolution of the Primary Features and Functional Impairments in Attention-Deficit Hyperactivity Disorder

Original accounts of the ADHD symptom profile date back to 1798, when Scottish physician Alexander Crichton described a disease of inattention, or mental restlessness, in his book, *An Inquiry into the Nature and Origin of Mental Derangement* (Crichton, 1798). However, it was not until nearly a century later that English physician George Still (1902) characterised 43 children in his practice as exhibiting overactivity and deficits in concentration that, he speculated, were associated with a decreased threshold for sustained attention and the poor inhibition of inappropriate responses to situations (e.g., acts of aggression and defiance). In these children, Still documented a heightened display of ill-focused passion and emotion that often manifested in acts of presumed jealousy or malice; this was coupled with a consistent inability to act within the confines of the law or in line with any sort of social or moral code. Still (1902) argued this apparent defect in moral control was, at least to some extent, neurobiologically innate, drawing on cases in which children

exhibiting such atypical behaviour also possessed histories of disrupted cerebral development, including head injuries, meningitis, cerebral tumours, and epilepsy.

The biomedical approach to behavioural anomalies gained momentum in 1917, when an encephalitis epidemic in North America and Europe was thought to be linked to extreme restlessness and distractibility in children affected by the illness. In the decades to follow, empirical explorations of inattention and disinhibition in children were heavily focused on potential neurobiological causes (Ebaugh, 1923; Strecker & Ebaugh, 1924; Stryker, 1925), with the underlying belief that severe restlessness in children was simply the result of pathological deficits in the forebrain (Blau, 1936; Levin, 1938). Consequently, in the 1930's the notion of the "brain injured child" was born, which later evolved into the concept of "minimal brain dysfunction" (MBD; Strauss & Lehtinen, 1947). These terms were applied to children with behavioural abnormalities indicative of frontal lobe damage, such as inattention, poor organisational skills, difficulties inhibiting impulsive behaviours, and the need for immediate reward (Khan & Cohen, 1934). It should be noted, however, that many of the children diagnosed as having MBD had little to no physical evidence of the actual brain damage thought to be causing their behavioural abnormalities (Schachar, 1986).

The concept of MBD began to lose its standing in the 1960's as its vague and over-inclusive nature became increasingly recognised (Douglas, Parry, Marton, & Garson, 1976). In the decades to follow, a discernible shift in investigative focus occurred, with the potential neurobiological origins of a disorder giving way to a more in-depth exploration of observable behavioural characeristics (Barkley et al., 1990; Burks, 1960; Chess, 1960; Laufer & Denhoff, 1957; Ounsted, 1955; Prechtl & Stemmer, 1962). Accordingly, research conducted by American psychiatrist Stella Chess (1960) attempted to separate the concept of hyperactivity from brain dysfunction by using behavioural observations of hyperactive children to clinically define relevant recurring behaviour patterns. Chess' (1960) simplified,

behaviour-focused classification of hyperactive children described them as being constantly in motion, "[carrying] out activities at a higher rate of speed than the average child" (p. 2378). This definition, and the disorder's eventual inclusion in the second edition of the Diagnostic and Statistical Manual of Mental Disorders as a *Hyperkinetic Reaction to Childhood* (DSM-II; APA, 1968), represented the shift from more neurobiological accounts of the features of hyperactivity to the exploration of their overt and frequently occurring qualities. Nevertheless, these initial behavioural accounts of overactivity in children were quickly criticised for providing little useful diagnostic information outside of a basic clinical description of the behavioural manifestations of hyperactivity (Barkley et al., 1990).

Shortly following the release of the DSM-II, the amount of research conducted on children with suspected hyperkinetic reactions increased significantly, and with this came another shift in the conceptualisation of the disorder's primary features (Barkley et al., 1990). More specifically, it was around this time that research conducted by physician Paul Wender (1971) and psychologist Virginia Douglas (1972; 1976; 1980; 1983; Douglas & Peters, 1979) documented the inability of hyperactive children to inhibit impulsive responses and sustain attention. Up until the 1970's, behaviours outside of hyperactivity (e.g., impulsivity, inattention, distractibility, low frustration tolerance) had been documented in children with hyperkinetic behaviour, but were largely considered by researchers and clinicians to be on the periphery of the disorder's main features (Chess, 1960; Laufer & Denhoff, 1957). Accordingly, in an attempt to avoid unsubstantiated assumptions made by her predecessors about these behaviours (e.g., clinical judgements based on small sample sizes and non-standardised forms of assessment, such as general observation), Douglas et al.'s (1972; 1976; 1979; 1980; 1983) research explored a range of cognitive and behavioural characteristics in hyperkinetic children through standardised, psychometric assessment (e.g., measures supported through research evaluation and via baseline comparisons to normative samples).

In doing this, Douglas was not only able to establish a definition of the disorder that was more valid and reliable than previous descriptions, but also set up a process by which such classification could continue to be assessed and shaped by future research (Barkley et al., 1990; Sandberg & Barton, 2002). This ongoing process of diagnostic refinement, as well as the inclusion of additional, relevant behavioural characteristics has been represented in diagnostic changes to the DSM with the DSM-III (APA, 1980) and, to a greater extent, its revised edition, the DSM-III-R (APA, 1987), incorporating inattention and impulsivity into the classifications of *Attention-Deficit Disorder (ADD),* and *Attention-Deficit Hyperactivity Disorder (ADHD)*, respectively. This pursuit of diagnostic precision has been further reflected in the creation of separate inattentive, hyperactive-impulsive, and combined diagnostic subtypes listed in the DSM-IV (APA, 1994) and its revised version, the DSM-IV-TR (APA, 2000).

Primary diagnostic features of Attention-Deficit Hyperactivity Disorder as per the Diagnostic and Statistical Manual of Mental Disorders 5th Edition. The current edition of the DSM presents detailed diagnostic criteria for both the inattentive (nine symptom criteria) and hyperactive-impulsive (nine symptom criteria) symptom classes (APA, 2013). At least six out of nine symptoms (five for people aged 17 and older) must exist for each of these two symptom groups in order for a diagnosis to be made (APA, 2013). The hyperactive-impulsive criteria of ADHD encompass a range of symptoms and behaviours associated with poor inhibition and associated hyperactivity (Willcutt et al., 2012). More specifically, individuals with ADHD often respond quickly to situations without waiting for instructions or taking into consideration all situational factors or response-related outcomes (Roberts, Milich, & Barkley, 2015). In contrast, inattentive symptoms in ADHD are associated with poor attentional vigilance and difficulty attending to tasks that are repetitive and not inherently stimulating (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Hoza,

Waschbusch, Owens, Pelham, & Kipp, 2001). Differences both within and between ADHD sub-types demonstrate the innate heterogeneity of the disorder, with the nature of an ADHD diagnosis depending on whether or not criteria are met for inattention, hyperactivity-impulsivity or both, and how behaviour patterns (relating to each criterion) present across different environments (APA, 2013).

The heterogeneity of ADHD also extends to its presentation over time, with variations in the age in which ADHD-related functional impairments are experienced (Keiling, Genro, Hutz, & Rohde, 2010). Adolescents/adults have been observed to manifest key features of the disorder differently to, and sometimes less overtly/intensely than, children (Willcutt et al., 2012). The DSM-5 has acknowledged such variations in ADHD symptom-trajectory by presenting a lower five-symptom threshold for adults and extending the age limit for onset to 12 years (APA, 2013). Additionally, as an extension to the examples of symptom presentation in children with ADHD, the DSM-5 now includes parenthetical clarifications of how symptomatology might be expressed in adolescence or adulthood (APA, 2013). It should be noted that concerns have been raised with respect to how well the modifications to the DSM-5 actually reflect the adult ADHD phenotype (Batstra & Frances, 2012), and that they may inflate prevalence rates in adult samples (Matte et al., 2012). Nevertheless, these evidence-based advances in the disorder's phenomenological description (Lahey & Willcutt, 2010; Roberts et al., 2015; Willcutt et al., 2012) mark an important step forward for adults with ADHD, given the challenges faced by many trying to obtain a diagnosis or secure ongoing support for the ADHD-related impairments they continue to experience past the early developmental years. Studies 1 and 2 of this thesis aimed to focus on ADHD as it presents in adulthood, and thus, will classify the disorder as per current diagnostic criteria described by the DSM-5 for this lifestage.

Age related differences in the presentation of Attention-Deficit Hyperactivity Disorder. Ongoing refinement to diagnostic classification, as well as advances in the screening, treatment, and general public awareness of ADHD have made detection and support increasingly available to children affected by this disorder (Mandell, Thompson, Weintraub, DeStefano, & Blank, 2005). However, questions regarding symptom patterns and prognosis in ADHD in adulthood have only received in-depth examination in the last couple of decades (Barkley, 2012a; 2015b). A recent review of longitudinal follow-up studies of children diagnosed with ADHD found that up to 70% of children experience clinically-significant ADHD symptom levels during adolescence, and 30 to 60% of those children maintained diagnosable ADHD symptoms into adulthood (Barkley et al., 1990; 2002; Kessler et al., 2006; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Weiss & Hechtman, 1993; Willcutt et al., 2012). Despite the existence of several prospective studies on ADHD across the lifespan, little is known about the details of its symptomalogical trajectory (Biederman, Petty, Evans, Small, & Faraone, 2010). More specifically, research over the last two decades has built on the understanding of ADHD's progression across the lifespan, but information on presentation in relation to separate age-specific symptom classes of hyperactivity-impulsivity and inattention in adulthood is yet to be explored at length. The sections which follow discuss the findings on ADHD symptomatology as it uniquely presents in childhood and adulthood, as these two lifestages will permit some examination of variation in symptom presentation across the lifespan and further highlight the adult experiences of ADHD.

Hyperactive-impulsive symptomatology. In children with ADHD, symptoms of hyperactivity-impulsivity are typically displayed when they are required to stay in one location for prolonged periods of time such as in class, in church, out for a family meal, or when riding in the family car (APA, 2013), with restlessness presenting as more severe in

boring or low-stimulation situations (Antrop, Roeyers, Van Oost, & Buysse, 2000). Hyperactive children with ADHD often climb excessively in situations where such behaviour is not appropriate, and typically struggle to play quietly by themselves, sometimes even narrating their own activity (Berk & Potts, 1991; DuPaul et al., 1998). Parents and teachers often report that hyperactive-impulsive children with ADHD are restless and fidgety, engaging in unnecessary, gross bodily movements across a range of settings (e.g., at their desk or the dinner table), and that they often interrupt conversations, games, and activities, or start using others possession's without permission (McGrath, Handwerk, Armstrong, Lucas, & Friman, 2004; Wood, Asherson, Rijsdijk, & Kuntsi, 2009). Moreover, when hyperactive-impulsive children desire something that is controlled by another person (e.g., a trip to the mall or the movies), they may excessively harass that person until access is granted (Roberts, et al., 2015).

In children, there is a large enough correlation between hyperactive and impulsive symptoms to conclude that these features form a single symptom factor (Willcutt et al., 2012). However, in adults with ADHD, hyperactivity appears to form its own symptom dimension, as it appears to occur less frequently than that of impulsivity (Kooij et al., 2005; Murphy & Barkley, 1996). More specifically, as opposed to the unwarranted gross motor movement observed in children with the disorder, hyperactive symptoms in adults with ADHD typically manifest as excessive speech and fidgeting (Barkley et al., 2008; Murphy & Barkley, 1996; Roberts, et al., 2015). Adults with ADHD will often get up from their desk at work or struggle to stay seated for prolonged periods at home, even when engaged in leisure activities; they are often described as being restless and difficult to keep up with due to their need for constant movement and activity (APA, 2013). Similarly to children, adults with ADHD appear to demonstrate more severe symptomatology in low-stimulation situations, with symptoms of hyperactivity-impulsivity typically observed at work, in meetings, when

engaged in chores at home, or when running errands (APA, 2013). In addition to this, experiences of being on hold while at work or paying a bill, or waiting in line while running errands can prove to be particularly taxing for adults with ADHD (APA, 2013).

Hyperactivity-impulsivity and risk taking. In the context of hyperactivity-impulsivity, both children and adults with ADHD often respond quickly and impulsively to situations, failing to listen to instructions (e.g., from a teacher, parent, or supervisor), consider potentially adverse or dangerous consequences (e.g., adverse reactions of others), and appreciate important environmental factors (e.g., other people involved; Roberts, et al., 2015). Moreover, when faced with situations where working towards a more long-term goal would result in a larger reward (e.g., good grades, task completion), both children and adults with ADHD often focus on a more immediate, and typically less rewarding, outcome (e.g., getting up from desk, getting friends to laugh) that requires less effort to achieve (Roberts et al., 2015). With respect to children with ADHD, this means they are more likely to engage in risky physical behaviours such as excessive or inappropriate climbing of objects (Kaya et al., 2008). Adolescents, by contrast, are more likely than their neurotypical peers to engage in risky sexual behaviour, such as unprotected sex and sex with strangers (Barkley, Fischer, Smallish, & Fletcher, 2006; Flory, Molina, Pelham, Gnagy & Smith, 2006), as well as antisocial (e.g., aggressive) and criminal behaviour (e.g., higher likelihood of arrest and criminal conviction than neurotypical peers; Hechtman & Weiss, 1986; Mannuzza, Klein, Konig, & Giampino, 1989; Satterfield, Hoppe, & Schell, 1982; Satterfield, Swanson, Schell, & Lee, 1994). Furthermore, this risk-taking element of hyperactivity-impulsivity can manifest in adults with respect to their driving (e.g., higher instances of speeding, drink driving, and driving without a license in comparison to neurotypical adults; Jerome, Segal, & Habinski, 2006). Adults with ADHD are also more likely to experience difficulties managing

money and paying bills on time, and may spend impulsively, a problem also associated with ADHD-related inattention and disorganisation (Barkley et al., 2008).

Inattentive symptomatology. With respect to inattention, both children and adults with ADHD are typically characterised as being more distractible than their neurotypical peers, often shifting attention toward task-irrelevant stimuli, even when instructed not do so (Roberts, Fillmore, & Millich, 2011; Ross, Harris, Olincy, & Radant, 2000). Inattention is reportedly lower in frequency and intensity in the presence of novel or highly stimulating tasks (e.g., class field trip; Beike & Zentall, 2012; Boonstra et al., 2005; Hoza et al., 2001; Lee & Zentall, 2002), but only if the stimulation originates from the task itself and not another irrelevant environmental factor that may pose as a distraction (Landau, Lorch, & Milich, 1992; Lee & Zentall, 2002). Symptoms of inattention are evident in children with ADHD during schoolwork, extracurricular activities or play with others, and often struggle to keep personal belongings, such as school supplies, in order (APA, 2013). Moreover, children with ADHD often have difficulty paying attention when being given instructions in class or when engaging in conversation with their peers, and spend more time off-task in comparison to their neurotypical peers (Barkley et al., 1990; DuPaul & Stoner, 2003). Adults with ADHD also reportedly experience difficulties with organisation, distraction and task completion, and often lose or mishandle important items such as tools, keys, wallets, paperwork, eyeglasses, and mobile phones (Barkley et al., 2008; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005; Murphy & Barkley, 1996; Roberts et al., 2011). Further, the symptom profiles of adults with ADHD indicate that the behaviours arising from inattention exert a greater impact on functional impairment than those of hyperactivity-impulsivity (Barkley et al., 1990; Biederman, Mick, & Faraone, 2000; Willcutt et al., 2012).

Impacts of Attention-Deficit Hyperactivity Disorder's primary features on daily functioning. Although the primary diagnostic features of ADHD (particularly those of

hyperactivity-impulsivity) are reported to decline with age, difficulties in day-to-day functioning that arise from those features often do not (Fischer, Barkley, Fletcher, & Smallish, 1993; Hart, Lahey, Loeber, Applegate, & Frick, 1995; Hinshaw et al., 2012). More specifically, as children transition into adolescence and, later, adulthood, they are expected to gradually develop adaptive skills, but this does not always appear to be the case in ADHD (Weyandt & Gudmundsdottir, 2015). Impaired adaptive functioning is important in differentiating cases of ADHD from instances in which the individual merely experiences heightened intermittent inattention or hyperactivity, but is otherwise able to manage day-to-day with little difficulty (Gathje, Lewandowski & Gordon, 2008; Wilcutt & Carlson, 2005). Moreover, functional impairments are often the primary reason for clinical referral and the requirement for ongoing treatment (Becker, Chorpita, & Daleiden, 2011). Consequently, identifying the nature of functional impairments in ADHD is essential to understanding how this disorder progresses across the lifespan.

Though often easily confused with one another, it is important to note that *symptoms* (also known as a disorder's *primary features*) are strictly the behavioural expressions of a disorder, and *functional impairments* are the consequences to follow (Barkley et al., 2006). Although not synonymous, symptoms and functional impairments are often at least moderately correlated with one another in ADHD (r = .2 to .5; Gathje et al., 2008; Willcutt et al., 2012). Symptoms of inattention and hyperactivity-impulsivity in children with ADHD have been shown to correlate moderately with functional impairments pertaining to academic performance, as well as peer and familial relationships (Burns, Servera, del Mar Bernad, Carrillo, & Geiser, 2014; Gathje et al., 2008). Similarly, in adults with ADHD, symptoms of inattention have been associated with a range of functional impairments, with distractibility and disorganisation in particular correlating with lower levels of functioning in comparison to neurotypical peers (Barkley et al., 2008; Das et al. 2012; Matte, Rohde, & Grevet, 2012).

Accordingly, studies on individuals with ADHD indicate that impairments in the ability to function well in the context of everyday activities (e.g., socially/communicatively, academically, occupationally) appear to persist across the lifespan (Barkley et al., 1990; Biederman et al., 2012; Jarratt, Riccio, & Siekierski, 2005; Pelham, Fabiano, & Massetti, 2005; Roizen, Blondis, Irwin, & Stein 1994).

Impairments to social-communicative functioning in Attention-Deficit Hyperactivity Disorder. Although children with ADHD often display minimal deficit in initiating social interactions with others (Mikami, Huang-Pollock, Pfiffner, McBurnett, & Hangai, 2007; Pelham & Bender, 1982; Whalen & Henker, 1985; 1992), they have severe difficulties in sustaining peer-based interactions as a result of their symptomatology (Milich & Landau, 1982; Nangle & Erdley, 2001). The tendency of children with ADHD to interrupt, make noise, talk excessively, and be impatient often results in the perception that they are annoying, domineering, self-centred, and intrusive (Normand et al., 2011; Pelham & Bender, 1982; Pelham et al., 2005; Whalen & Henker, 1992). Additionally, inattention in children with ADHD often results in failure to listen and avoid distraction, decreasing the likelihood that peers, and their social cues, will be effectively attended to (Cadesky, Mota, & Schachar, 2000; Hinshaw & Melnick, 1995; Hoza, 2007). As a result of this behaviour, not only are children with ADHD more likely than their neurotypical peers to be bullied or victimised (e.g., verbal teasing, physical violence; Wiener & Mak, 2009), many of their peers view them as bullies (Unnever & Cornell, 2003). Accordingly, research suggests that at least half of children with ADHD experience rejection from their peers (see review by Hoza, 2007; also see Gresham, MacMillan, Bocian, Ward, & Forness, 1998; Hoza et al., 2005), and the relationships children with ADHD do form with their neurotypically developing peers are often reported to be unstable and unsatisfactory with respect to the level of companionship and intimacy that is able to be maintained (Blachman & Hinshaw, 2002; Normand et al.,

2011). Moreover, research has demonstrated that the peers who do form relationships with children with ADHD also often display noncompliant and deviant behaviour (Bagwell, Molina, Pelham, & Hoza, 2001; Blachman & Hinshaw, 2002; Normand et al., 2011; Whalen & Henker, 1985), limiting the type of peers children with ADHD feel comfortable interacting with and not allowing them to learn from more socially competent peers and develop better social skills for later in life (McQuade & Hoza, 2015).

Similar to children, adults with ADHD display greater impairment in social functioning than their neurotypical peers. More specifically, adults with ADHD have a tendency to interrupt or intrude, become easily frustrated, and fail to listen or pay attention to others in conversation, even in the absence of distraction (Barkley et al., 2008; Shaw-Zirt, Popali-Lehane, Chaplin, & Bergman, 2005). Additionally, adults with ADHD reportedly tend to end relationships impulsively, over trivial matters (e.g., a verbal disagreement; Barkley & Murphy, 2010; Murphy & Barkley, 1996). Moreover, although the number of social relationships adults with ADHD form may not be significantly less than that of their neurotypical peers, the overall quality of their social relationships is reported to be relatively poorer (e.g., fewer close friends, higher friend turnover, greater instance of arguing; Fischer & Barkley, 2006; Klein et al., 2012). There is some evidence to suggest that impairments in this area also place adults with ADHD at higher risk for romantic separation and divorce (Biederman et al., 1993; Murphy & Barkley, 1996); however, further research in this area is required (Barkley, 2015b).

Impairments to academic functioning in Attention-Deficit Hyperactivity Disorder. ADHD symptomatology has been consistently linked to impairment in academic performance. Problematic ADHD-oriented behaviours can result in impairments in educational functioning that significantly impact a child's ability to meet academic expectations (i.e., pay attention to/following instructions, comply with rules, organise class

materials, complete academic tasks; DuPaul & Langberg, 2015). Impairments in academic functioning include failures to perform or engage in learning at a level appropriate to a child's age, intellectual ability and family-school context (DuPaul & Langberg, 2015). At least half of children with ADHD are thought to experience significant problems with respect to academic functioning (Barkley et al., 1990; 2006; DuPaul et al., 2001; Fischer, Barkley, Edelbrock, & Smallish, 1990; Frazier, Youngstrom, Glutting, & Watkins, 2007; Hinshaw et al., 2006; Klein et al., 2012; Lee et al., 2008; Mannuzza et al. 1993), but similar to the heterogeneity seen in ADHD symptomatology, there is significant variability in presentation, severity, and frequency of academic impairment (DuPaul & Langberg, 2015).

Hyperactive-impulsive symptomatology (e.g., talking out of turn, moving around the classroom without permission, playing with objects not related to academic tasks, engaging in activities that yield immediate over long-term benefit) has been associated with impairments of academic functioning, such as breaking class rules (Barkley, 1997; Barkley et al., 1990; Sonuga-Barke, 2003). However, research suggests that it is largely symptoms of inattention that drive problems in academic functioning in children with ADHD (Langberg et al., 2011; Massetti et al., 2008; Rapport, Scanlan, & Denney, 1999). Studies of inattentive children in the classroom have yielded significant associations between inattentive symptoms and emergent literacy skills (e.g., print knowledge, phonological sensitivity) such that higher symptom severity and frequency is associated with lower performance in these areas (Lonigan et al., 1999; Sims & Lonigan, 2013). Additionally, inattentive symptoms, such as distractibility, have been associated with lower task completion rates and lower task accuracy in comparison to neurotypically developing peers (DuPaul & Stoner, 2003). Given the range of these impairments, significantly lower standardised achievement scores and higher rates of grade retention (i.e., being held back a grade) and school dropout are observed in not only children, but adolescents with ADHD (DuPaul & Stoner, 2003).

Although hyperactive-impulsive symptoms often decline in adolescence, inattentive symptoms typically persist or even worsen in some cases (Hart et al., 1995; Martel, von Eye, & Nigg, 2012). This symptom persistence is further compounded by increased and more stringent demands present in the secondary school classroom (Langberg et al., 2008). These demands include increases to academic workloads (e.g., homework and study required for cumulative tests), heightened expectations surrounding independent work, and changes in classroom activities, such as a higher likelihood for lecture-style content delivery (Kent et al., 2011; Langberg et al., 2011). Accordingly, adolescents with ADHD often experience impairments to academic functioning across a broader range of activities than experienced in childhood (Evans, Schultz, DeMars, & Davis, 2011). This is problematic because, by adolescence, chronic and cumulative experiences with misbehaviour and failure in the classroom start to adversely affect academic outcomes (Ackerman et al., 1977; Fischer et al., 1990; Hechtman, 2000; Mendelson, Johnson & Stewart, 1971; Stewart et al., 1973; Wilson & Marcotte, 1996). Moreover, in comparison to their neurotypical peers, adolescents with ADHD have higher rates of course failure (Kent et al., 2011), are more likely to drop out of school (Barkley et al., 2006), and are less likely to enrol in post-secondary education (29.5% of ADHD sample compared to 76.8% neurotypical sample; Kuriyan et al., 2013).

For adults with ADHD who do enrol in tertiary studies, many report continual challenges with respect to inattention, listening, and forgetfulness (Gray, Fettes, Woltering, Mawjee, & Tannock, 2016) and, subsequently, in comparison to their neurotypical peers, tend to obtain lower GPAs (Advokat, Lane, & Luo, 2011; Blase et al., 2009; Frazier et al., 2007; Gropper & Tannock, 2009), experience higher rates of course/program drop out (Advokat et al., 2011; Barkley et al., 2008; Blase et al., 2009; Kuriyan et al., 2013), and are more likely to be on academic probation at some point in their degree (Gropper & Tannock, 2009). Additionally, even for adults with above average GPA scores, many report having to

work harder than their peers to achieve good grades, taking longer than their peers to complete assignments/exams, and having greater concerns about their academic progress and ability to keep up with academic demands (DuPaul, Weyandt, O'Dell, & Varejo, 2009; Gray et al., 2016; Lewandowski, Lovett, Codding, & Gordon, 2008; Rabiner et al., 2008; Sparks, Javorsky, & Philips, 2004).

Impairments to occupational functioning in Attention-Deficit Hyperactivity Disorder. Issues with educational attainment can adversely affect the capacity of adults with ADHD to obtain regular employment later in life (Barkley, 2015b) and, in comparison to their neurotypical peers, they are more likely to enter the workforce unskilled or under-skilled (Currie & Stabile, 2006). Accordingly, as adults with ADHD enter the workforce and take on jobs that require independence, responsibility, and personal development, symptoms of impulsivity, diminished self-control, inattention, and disorganisation may impede successful execution of work-related tasks (Barkley, 2015b). Although they may excel in roles that are non-repetitive, immediately gratifying, novel, physically challenging, and low in organisational/time-management demands, such employment prospects for adults with ADHD are largely dependent on job availability and, more importantly, the way that ADHD has manifested itself during the formative years (Young, 2005).

Similar to impairments in academic functioning, the major employment challenges adults with ADHD experience are associated with symptoms of inattention. Adults with ADHD are typically disorganised at work and demonstrate poor time management, which often results in difficulties meeting deadlines (APA, 2013). Incompletion of routine work-related tasks such as report writing, the review of lengthy documents, and filling out/filing paperwork, is often observed, as well as the use of short-cuts, leading to mistakes on tasks and activities performed in the workplace (Roberts, et al., 2015; Shifrin, Proctor, & Prevatt, 2010). Consequently, adults with ADHD have reported experiencing more employment

problems than adults without ADHD in the form of fewer working hours, higher job turnover, lower work quality, more trouble on the job (e.g., discipline, firing), and lower job performance (Barkley & Fischer, 2011; Barkley et al., 2006; Klein et al., 2012; Kuriyan et al., 2013; Mannuzza et al., 1993; Murphy & Barkley, 1996; Weiss & Hechtman, 1993). Subsequently, in comparison to their neurotypical peers, adults with ADHD are often significantly underemployed (Biederman et al., 2008; Faraone & Biederman, 2005).

The progression of functional impairments in adult Attention-Deficit Hyperactivity Disorder. Adults with ADHD often experience rates of economic activity and income levels that are significantly lower than those of their neurotypical peers (Biederman & Faraone, 2006; Daley, Højberg Jacobsen, Lange, Sørensen, & Walldorf, 2014; Klein et al., 2012; Weiss & Hechtman, 1993). This is one of many problems that arises as a result of ADHD persisting across the lifespan, and highlights the role that age and developmental stage play with respect to functionality in ADHD and the transition into adulthood in particular. The transition from adolescence to adulthood is a period of immense significance and vulnerability for young adults with ADHD, marking the time when they are most likely to move away from home, start university, develop new relationships, explore job opportunities, assume new roles and responsibilities, and otherwise leave behind institutional supports and familiar environments (Schulenberg, Sameroff, & Cicchetti, 2004; Young, 2005). Any disruption during this period carries the potential to create long-lasting adverse effects on a young adult's exploration of his/her potential and personal development, and increased demands posed by new, unstructured environments may lead to worsening of pre-existing impairments (Young, 2005). Research suggests that, in comparison to their neurotypical peers, significant functional impairments in work/study (e.g., failure to complete post-secondary school or secure stable employment) and socialisation (e.g., fewer stable, reciprocal relationships with peers) continue, and even become exacerbated with time for

many adults with ADHD (Barkley et al., 2006). This problem is one of many associated with the progression of functional impairments in individuals with ADHD across the lifespan, because with age, impairments become increasingly disruptive due to their cost, education and employment implications and potential for health consequences (Barkley et al., 2006; Flory et al., 2006; Kleinman, Durkin, Melkonian, & Markosyan, 2009). Consequently, it is important that research on the presentation of ADHD, and its functional impairments in adulthood, is further expanded on.

Common comorbidities in Attention-Deficit Hyperactivity Disorder. In addition to age and developmental stage, the presence of comorbid disorders can further impact the impairments observed in individuals with ADHD across the lifespan (Becker, Luebbe, & Langberg, 2012; Connor, Steeber, & McBurnett, 2010). Comorbidity, or the co-occurrence of two or more disorders in the same individual (Matson & Nebel-Schwalm, 2007), results in presentation of additional symptoms external to an individual's primary disorder. Adults with ADHD who experience ongoing impairments to functionality have been found to be at five times greater risk of developing a comorbid disorder in comparison to adults whose ADHD is not associated with marked impairments to functionality, and at eight times the risk in comparison to adults with no history of ADHD (Yoshimasu et al., 2016). A review of the research on comorbid diagnoses in ADHD over the past several decades suggests that 67-84% of children (Barkley et al., 2008; Kadesjö & Gillberg, 2001) and 76-87% of adults with ADHD (Barkley et al., 2008; Sobanski et al., 2008; Yoshimasu et al., 2016) have been diagnosed with at least one comorbid disorder over the course of their lives. Moreover, over half of all individuals diagnosed with ADHD have had two or more comorbid diagnoses in addition to ADHD at some point in their lives (Barkley et al., 2008; Kadesjö & Gillberg, 2001), with the most common comorbidities including internalising disorders such as anxiety and depression; externalising disorders such as Oppositional Defiance Disorder (ODD) and

Conduct Disorder (CD); and other neurodevelopmental disorders such as Autism Spectrum Disorder (ASD; Becker et al., 2012; Connor, Chartier, Preen, & Kaplan, 2010; Sobanski et al., 2008).

The presentation of multiple disorders within one individual poses an increased risk for impairment as a result of the need to cope with compounded and complex symptomatology contributing to the heterogeneity of ADHD by increasing the likelihood of certain primary symptoms and subsequent impairments to functioning (Barkley, 1997; Barkley, Fischer, Edelbrock, & Smallish, 1990; King & Waschbusch, 2010; Lacourse et al., 2006; McGee, Williams, & Silva, 1984; Moffitt & Silva, 1988; Schachar & Tannock, 1995). Correctly identifying and understanding the nature of potential additional psychopathologies in children and adults with ADHD is important because it can greatly facilitate intervention, resource provision, and long-term prognoses (Matson & Nebel-Schwalm, 2007). The process via which the internalising (e.g., anxiety) and externalising comorbidities (e.g, CD) common to ADHD interact with its primary symptomatology and subsequent impairments to functioning has been of long-term research interest, particularly in children and adolescents. Nevertheless, previous diagnostic requirements of ADHD (i.e., the inability to diagnose comorbid neurodevelopmental disorders) have served to limit the extension of this exploration into the relationship between ADHD and other neurodevelopmental disorders such as ASD (APA, 2000).

Prior to the current version of the DSM (APA, 2013) presentation of ADHD symptomatology in an individual with ASD (e.g., getting distracted by a toy when asked to complete a worksheet) was thought to simply be a reflection of ASD-related impairment (e.g., engaging with a preferred object/circumscribed interest), and vice versa. Until recently, the development, treatment, and exploration of ADHD and ASD as two separate disorders was very clearly articulated due to the primary diagnostic differences that existed between

them, and it has only been within the last decade that systematic investigations of the relationship between ADHD and ASD have begun to emerge (see Clark, Feehan, Tinline, & Vostanis, 1999; Gargaro, Rinehart, Bradshaw, Tonge, & Sheppard, 2011; Lee & Ousley, 2006; Sinzig, Walter, & Doepfner 2009; Thede & Coolidge, 2007). Consequently, ADHD and ASD have largely been studied in isolation from one another (Davis & Kollins, 2012; Rommelse, Buitelaar, & Hartman, 2017; Rommelse et al., 2010). Given the breadth of knowledge on ADHD's presentation in the presence of other comorbid diagnoses (e.g., anxiety and depression) and how this impacts functionality in the ADHD individual, it is likely that further review of the potential overlap of different aspects of ADHD and ASD could provide a broader understanding of how these disorders present across different individuals and, moreover, potentially inform diagnosis and treatment with respect to the formulation of a unifying theory of underlying psychopathology.

Clinically, such an exploration has implications with respect to the collectively informed assessment and treatment of psychological disorders, commonly referred to as a transdiagnostic approach. A transdiagnostic approach to assessment of psychological disorders was formulated in response to difficulties with the clinical utility of diagnostic categories of certain disorders (Brown, Di Nardo, Lehman, & Campbell, 2001). This approach aims to explore psychological disorders in a manner that extends beyond the conceptual structure provided through basic diagnostic classification, and instead formulate and treat disorders through the simultaneous consideration of other disorders that share some form of similar characterisation (Loeb, Lock, Le Grange & Greif, 2012). A review of the research on the transdiagnostic approach demonstrates that such characteristics commonly include aetiological (i.e., genetic, familial, and environmental risks), cognitive (i.e., variants in functioning), affective (i.e., experiences of recurring emotion) and behavioural factors (i.e.,

recurrent behavioural patterns; Mansell, Harvey, Watkins, & Shafran, 2008; Kessler et al., 2005).

Previous psychological disorders explored transdiagnostically include eating disorders, such as anorexia and buleimia nervosa (Fairburn, Cooper, & Shafran, 2003), and internalising disorders, such as anxiety and depression (Newby, McKinnon, Kuyken, Gilbody, & Dalgleish, 2015). Although neurodevelopmental disorders such as ADHD and ASD have not yet been explored in this context, nor has their means of assessment or treatment been aimed to explicitly be inclusive of both, it is possible that investigating ADHD and ASD alongside one another may lead to a greater understanding of additional factors that impact the presentation of primary symptoms and subsequent functional impairments associated with both disorders. Conseqeuently, Study 1 aimed to further explore the presentation of both ADHD and ASD's unique characteristics in adulthood alongside one another, comparing and contrasting points of convergence and divergence in the literature. The literature review of ASD commences in Chapter 3 with a discussion of the potential contributing factors of the primary features and subsequent impairments to functionality in ASD (i.e., aetiology, progression of diagnostic conceptualisation). The discrepancies between ADHD and ASD made apparent by the current body of literature will then be addressed at the end of Chapter 3, with a comparative review on these disorder's common, but non-diagnostic qualities to follow in Chapter 4.

Chapter Three: Autism Spectrum Disorder

ASD is a neurodevelopmental disorder characterised by impairments to social communication and reciprocal social interaction, as well as a presence of restricted and repetitive behaviours, interests and activities (APA, 2013). Diagnostic guidelines suggest that these primary features must be of sufficient intensity to cause subsequent impairments in domains of functioning associated with socialisation, education and employment. Globally, ASD is thought to exist in approximately 1-2% of the general population (APA, 2013) and, on average, the male-to-female ratio of ASD is 4:1 (Fombonne, 2003; 2005; 2009; Idring et al., 2014; Werling & Geschwind, 2013). Epidemiological studies exploring prevalence rates of ASD in children in different parts of the world, including Australia (Australian Bureau of Statistics [ABS], 2015), the UK (Brugha et al., 2012), and North America (Centers for Disease Control and Prevention [CDC], 2016), reveal that figures consistently sit around 1-2% of the general population. Moreover, although ASD is first evident in early childhood it is known to persist across the lifespan, existing in 0.5-1% of adults (Brugha et al., 2012).

Relative uniformity in the global distribution of ASD indicates that the disorder does not appear to discriminate greatly on factors of culture and geographic location, emphasising the importance of exploring potential biological and genetic contributions to the disorder (Bushnell, 2013; Howe, Brand, & Talkowski, 2016). Although the underlying causal factors for ASD are likely to be somewhat influenced by environmental factors, there is substantial evidence to indicate a strong biological (i.e., genetic and neurological) component to its aetiology (Goines & Ashwood, 2013; Howe et al., 2016b; Stamou, Streifel, Goines, & Lein, 2013). The remainder of this chapter will discuss potential underlying factors of ASD; this will start with a review of research on this disorder's aetiology and will follow with a discussion of how ASD's diagnostic conceptualisation over the last century has shaped the

world's understanding of this disorder's primary features and its subsequent impairments to functionality across the lifespan.

The Aetiology of Autism Spectrum Disorder

A review of the genetic research on ASD has yielded heritability estimates that range from 40-90% (Bailey et al., 1995; Bushnell, 2013; Constantino & Todd, 2003; Ronald et al., 2008; Sandin et al., 2014), suggesting that, at a minimum, close to half of the risk for developing ASD is genetic. Studies on families with a child diagnosed with ASD suggest that the risk of the disorder recurring in other siblings is 10-18% (Constantino, Zhang, Frazier, Abbachi, & Law, 2010; Muhle, Trentacoste, & Rapin, 2004; Ozonoff et al., 2011). Studies of twins with ASD over the last several decades have further implicated the importance of genetic factors in the development of ASD, with monozygotic twins having a higher average concordance rate of ASD (64%) in comparison to dizygotic twins (9%; see meta-analysis by Smalley, Asarnow, & Spence, 1988). Given these findings, it is apparent that some form of genetic heritability exists in ASD. It should be noted, however, that the pattern of inheritance has not been linked to the modulation or mutation of one single gene (Talkowski, Minikel & Gusella, 2014), and as such, it is more likely that multiple genes contribute to the susceptibility and phenotypic diversity observed across individuals with ASD (Jorde et al., 1991; Pickles et al., 1995; Risch et al., 1999).

Genetic heterogeneity provides one explanation for the susceptibility and phenotypic diversity observed across individuals with ASD (Howe et al., 2016b). However, diversity in symptom development and presentation could also be influenced by the unique gene-environment interactions experienced by these individuals (D'Amelio et al., 2005; Hallmayer et al., 2011; Sandin et al., 2014). More specifically, ASD symptomatology may, in part, develop as a result of unique pre- or postnatal events interacting with pre-existing neurologic and/or genetic susceptibilities (Glasson et al., 2004). Prenatal events associated with ASD

include maternal diabetes (Gardener, Spiegelman, & Buka, 2009; Krakowiak et al., 2012); maternal stress (Beversdorf et al., 2005; Kinney, Munir, Crowley, & Miller, 2008; Limperopoulos et al., 2007); older parental age (Buizer-Voskamp et al., 2011; Croen, Najjar, Fireman, & Grether, 2007; Gardener et al., 2009; Grether, Anderson, Croen, Smith, & Windham, 2009; Parner et al., 2012; Sandin et al., 2014); exposure to toxins in utero including viral infections (Arndt, Stodgell, & Rodier, 2005; Blattner, 1974; Libbey, Sweeten, McMahon, & Fujinami, 2005; Meyer, Yee, & Feldon, 2007; Patterson, 2009); and maternal medication (e.g., antidepressants) and substance use (e.g., alcohol; Andrade et al., 2008; Croen et al., 2007; Dufault et al., 2012; Gardener et al., 2009; Karr, Solomon, & Brock-Utne, 2007; Kolozsi, MacKenzie, Roullet, deCatanzaro, & Foster, 2009; Roberts et al., 2007; Szpir, 2006). Additionally, a recent meta-analysis identified several birth related complications such as birth injuries, multiple births, maternal haemorrhages, umbilical-cord complications, and low birth weight as potential risk factors for ASD (see Gardener et al., 2009). Postnatal risk factors for ASD include gastrointestinal abnormalities (Iebba, Aloi, Civitelli, & Cucchiarra, 2011; Liu, Li, & Neu, 2005); dysfunction of the immune system (Ashwood, Wills, & Van de Water, 2006); exposure to poisons such as mercury and lead (Cohen, Paul, Anderson, & Harcherik, 1982); and psychosocial factors such as affiliations with a lower socioeconomic status (Larsson et al., 2005; Rai et al., 2012), or having immigrated from another country (Keen, Reid, & Arnone, 2010; Lauritsen, Pedersen, & Mortensen, 2005; Magnusson et al., 2012). It should be noted, however, in settings where healthcare is considered to be more accessible, the impact of psychosocial factors in particular appears to lessen or even be reversed in some cases due to better health literacy and increased potential for diagnosis (Bhasin & Schendel, 2007).

Gene-environment interactions may potentially alter neural connectivity and neurotransmitter signalling pathways during development, resulting in abnormal

neurobiology in some cases (Neale et al., 2012; Sanders et al., 2012; Stamou et al., 2013). Multiple neurotransmitter deficiencies have been reported in ASD, including those of monoamines (i.e., dopamine, norepinephrine and serotonin; Frye, 2010; Frye, Huffman & Elliott, 2010; Frye, Sequeira, Quadros, James, & Rossignol, 2013), acetylcholine (Rossignol & Frye, 2014), and amino acids (i.e., glutamate, gamma-aminobutyric acid [GABA]; Oberman, 2012; Rossignol & Frye, 2014). The exact aetiologies of the atypicalities (e.g., mitochondrial dysfunction) reported for these neurotransmitters is unknown, but studies using animal models suggest they are largely a result of genetic mutations that disrupt neurotransmission through disturbances in the metabolism of the neurotransmitters themselves (Frye, 2010; Frye et al., 2010; Frye et al., 2013; Maloney et al., 2013). In ASD, serotonin, and the pathways responsible for its synthesis in the brain, have been intensively explored in this context (Chugani et al., 1997; Zücher & Hooker, 2016), with decreased levels of serotonin transporter (SERT) having been observed in different areas of the frontal cortex in children with ASD (Makkonen, Riikonen, Kokki, Airaksinen, & Kuikka, 2008). Moreover, the depletion of tryptophan (the precursor of serotonin in the brain) has been associated with an increase in symptomatology in individuals on the autism spectrum (Anderson, 2002; McDougle, Naylor, Cohen, Aghajanian, Heninger, & Price, 1996). Accordingly, much of the research on neurotransmitters in ASD has been motivated by their presumed involvement in the atypicalities in cognitive and emotional processing often observed in this disorder (Anderson, 2002; Flory et al., 2007; Halperin et al., 1994; McDougle et al., 1996; Pine et al., 1997; Stadler et al., 2007).

A review of aetiological links to ASD would suggest that it is a disorder with neurobiological causes related to pathological processes that affect the development and function of neuronal synapses (Howe et al., 2016b). Recent research by Stoner and colleagues (2014) has yielded evidence of focal disruption to cortical laminar architecture in

the cortices of young children with ASD. A transient increase in the number of cortical neurons is expected in the second trimester of pregnancy; however, this increase usually disappears by birth or shortly after. Stoner et al. (2014) have postulated that this disappearance does not happen as abruptly in individuals with ASD. Consequently, Stoner et al.'s (2014) results have indicated a potential link between an increase in the number of neurons in the prefrontal cortex and deficits in the cell-cycle processes of pruning, apoptosis, and neuronal migration initiated in utero. More specifically, their data implicated a probable dysregulation of cortical layer formation in layer-specific differentiation at prenatal developmental stages.

The unrepressed dendritic arborisation often observed in the young ASD brain (Stoner et al., 2014) may be responsible for the observed increase in infantile macrocephaly (i.e., abnormally large head circumferences) in this population (Bauman & Kemper, 2005). Studies of head circumference, a measure that is strongly correlated with brain sizes in young children, have identified greater brain growth in the first year of life in children on the autism spectrum (Courchesne et al., 2001; Courchesne, Campbell, & Solso, 2011). A meta-analysis comprised of MRI data and post mortem head circumference measurement demonstrated that there is a period of early overgrowth followed by reduced growth rates and eventual normalisation of brain volumes in children with ASD (see Bauman & Kemper, 1985; Redcay & Courchesne, 2005). More specifically, during later childhood and adolescence, observed rates of brain growth tend to slow, with the abnormally high levels of enlargement diminishing in magnitude over time (Herbert et al., 2003; Palmen et al., 2005).

Studies suggest that within the cerebrum, the frontal lobe manifests the greatest degree of overgrowth in individuals on the autism spectrum (Carper, Moses, Tigue, & Courchesne, 2002; Palmen, et al., 2005). Structural abnormalities have been found in the frontal lobe of individuals with ASD (Courchesne, 2002; Courchesne et al., 2001) and, more

specifically, within the prefrontal cortex (Castelli, Frith, Happé & Frith, 2002). Brain imaging studies indicate that individuals with ASD demonstrate structural abnormalities such as a thickened cerebral cortex (Bailey et al., 1998), disrupted laminar organisation (Bailey et al., 1998), and decreased pyramidal cell density in the frontal lobe (Ciesielski et al., 1990; Townsend et al., 2001). Moreover, with respect to functional abnormalities in the ASD brain, reduced frontal-posterior connectivity has been associated with diminished capacity of cortical networks to coordinate information processing (Minshew & Keller, 2010). These are important findings, as atypicalities in the structure and function of the frontal lobes of the brain have been associated with heightened symptom expression in individuals with ASD (e.g., inability to process the nonverbal cues expressed by others; Barrash, Tranel, & Anderson, 2000; Beer, John, Scabini, & Knight, 2006; Hornak et al., 2003), and the cognitive (or executive) dysfunction thought to contribute to its presentation (Benton, 1991; Fuster, 1997; Hill & Bird, 2006; Luria, 1966; Pennington & Ozonoff, 1996).

Several structural neuroimaging studies have also implicated atypicalities in regions of the brain outside of the frontal lobes, including the temporal cortex, the limbic system (amygdala, hippocampus, anterior cingulate cortex), caudate nucleus, and cerebellum (Abell et al., 1999; Brieber et al., 2007; Ke et al., 2008; Lange et al., 2010; McAlonan et al., 2005; 2008; Nickl-Jockschat et al., 2012; Nordahl et al., 2012; Via, Radua, Cardoner, Happé, & Mataix-Cols, 2011; Waiter et al., 2004). With respect to the limbic system, overgrowth of the amygdala has been observed early in life in those with ASD (Shaw, Lawrence, Radbourne, Bramham, Polkey, & David, 2004), with both elevated (Dalton et al., 2005; Monk et al., 2010; Weng et al., 2011) and reduced activity in the amygdala (Ashwin, Baron-Cohen, Wheelwright, O'Riordan, & Bullmore, 2007; Bookheimer, Wang, Scott, Sigman, & Dapretto, 2008; Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009; Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2006) demonstrating an association with behavioural abnormalities in

ASD individuals (e.g., atypical facial-emotion recognition). Additionally, volumetric studies on the anterior cingulate cortex (another limbic structure) in individuals with ASD reveal a significantly lower relative volume, coupled with diminished metabolic activity (Haznedar et al., 1997), and, in general, a marked level of hypofunctionality in comparison to neurotypical individuals (Di Martino et al., 2009). Importantly, these neurobiological atypicalities have been considered with respect to potential impacts they may have on individuals with ASD, such as deficits in processing emotions, experiencing heightened levels of adverse emotion (e.g., anxiety, depression), and controlling emotionally driven behaviour such as aggression and, in some instances, the primary symptomatology of ASD (see Blair & Cipolotti, 2000; Blair, Morris, Frith, Perrett, & Dolan, 1999; Blair, Schafe, Bauer, Rodrigues, & LeDoux, 2001; Damasio et al., 1991; 1996; Davidson, Putnam, & Larsen, 2000; Etkin & Wager, 2007; Hulvershorn et al., 2014; Pietrini et al., 2000; Weiger & Bear, 1988; Weniger, Lange, & Irle, 2006).

The Conceptual and Diagnostic Evolution of the Primary Features and Functional Impairments in Autism Spectrum Disorder

Documentation of ASD symptoms first occurred at the turn of the 20th century, when Swiss psychiatrist Eugen Blueler first used the term “autism” to describe what he believed to be a socially withdrawn form of childhood schizophrenia (Bleuler, 1910). It was the presence of autistic characteristics from birth, however, that led child psychiatrist Leo Kanner to conclude that autism and schizophrenia were two distinct disorders, with schizophrenic symptoms typically surfacing after an extended period of normal development during childhood and adolescence (Gallo, 2010; Kanner, 1943). Kanner identified two essential features in the autistic children he observed, extreme aloneness and insistence on sameness (Eisenberg & Kanner, 1956). It was also around this time that paediatrician Hans Asperger (independent from Kanner) used the term *autism* to describe a specific disorder in children

exhibiting strange language patterns, obsessive behaviour and social difficulties; and coined the term *Asperger Syndrome* (AS) to refer to children with similar behavioural qualities as those who were autistic, but who also exhibited more robust language abilities (Asperger, 1944). Asperger further reported the tendency of the autistic child to act like a "little professor", engaging in one-sided conversations and lacking general empathy towards other children (Asperger, 1944).

Despite working independently from one another, both Asperger and Kanner noted the presence of autistic symptomatology from birth, and the inability of their patients to develop and maintain normal relationships with others (Asperger, 1944; Kanner, 1943). Their early explorations of autism in children also documented variations in functional ability, with Kanner noting impairments that ranged from minimally-intrusive inadequacies of social adjustment all the way to complete deterioration in social functioning (Kanner, 1971). Despite the similarities in diagnostic descriptions that independently arose from Kanner and Asperger's research, their observations on core autism features also varied in several ways (e.g., Kanner's heightened focus on over-engagement with trivial changes in the nonsocial world vs. Asperger's focus on language capability). Such variations in the descriptions of autistic children were early indicators of the diagnostic developments to come, which have resulted in formal acknowledgement of an "autism spectrum"

Kanner (1943) believed the most discernible autistic characteristic was the inherent inability to relate to other people, and it was the presence of this characteristic *from birth* coupled with the apparent lack of delusions and hallucinations that differentiated autism from schizophrenia. Nevertheless, other clinicians and researchers in the USA in the 1940s and 1950s disagreed with this differentiation and, consequently, Kanner's initial descriptions of perseverative and socially atypical autistic behaviour were not included in any diagnostic manual until the third edition of the DSM (APA, 1980). Prior to this, in the first and second

editions of the DSM, the characteristics associated with the term *autism* were simply included under childhood and schizotypal disorders (APA, 1952; 1968). By contrast, the DSM-III, listed autism as *infantile autism* and required the early onset of impaired language development, peculiar speech, bizarre and/or rigid responses to the environment, and the absence of symptoms, such as delusions or hallucinations, that might indicate a diagnosis of schizophrenia (APA, 1980). Although this represented the first clinical description of autism in a diagnostic manual (and its differentiation from schizotypal categorisation), this diagnosis was criticised for being extremely narrow, containing the overly stringent diagnostic requirement that all criteria be met, and for focusing strictly on the infantile form of the disorder (Volkmar et al., 2012). These clinical concerns were remedied in the DSM-III-R (APA, 1987), with the inclusion of a disorder whose descriptors allowed for a diagnosis of autism in children who developed impairments after a period of normal development (Volkmar & Klin, 2005); this disorder was known as regressive or *residual autism*, and provided yet another early reminder of autism's complexity and broad scope (e.g., symptom regression vs. linearity) across the lifespan.

Early diagnostic criteria reflected more severe expressions of autism that were usually associated with cognitive and language delays (Hill, Zuckerman, & Fombonne, 2016). By the 1980's, however, a shift in focus to a broader spectrum of autism and its related conditions saw more highly detailed criteria for the disorder in the DSM-III-R (*autistic disorder*; APA, 1987). This conceptual shift was largely a result of the research by psychiatrist Lorna Wing and clinical psychologist Judith Gould who, in the early 1980's, established a triad of impairments they believed to characterise autism (i.e., impairments in social interaction, social communication, and social imagination; Gould, 1982; Wing, 1981a; Wing & Gould, 1979). Wing and Gould's triadic conceptualisation of autism strongly emphasised a marked decline in (1) nonverbal signs of interest in/deriving pleasure from being with others (social

interaction); (2) the ability to negotiate, share ideas/interests, and/or converse in a positive and engaging way (social communication); and (3) the capacity to consider and anticipate the consequences of one's own actions for the self and others (social imagination; Wing & Gould, 1979). However, it was not until the publication of the DSM-IV that a diagnosis of autism required symptom presentation in all three impairment categories, which were then classified as impaired social interaction; impaired communication; and restricted, repetitive, and stereotyped patterns of behaviour, interests, or activities (APA, 1994).

Around this time, Wing (1981a) also made known to the Western world Asperger's early research and, with it, the notion that autism was part of a broader, more complex spectrum. More specifically, Wing and her colleagues noted the potential mixture of Kanner-Asperger clinical descriptors that many autistic individuals would display in combination, and saw the importance of sharing Asperger's findings to broaden the perspective on autism and its varied forms of presentation (Wing & Gould, 1979). Accordingly, in addition to the new triad of symptoms for ASD, both the DSM-IV and its revised version the DSM-IV-TR (APA, 1994; 2000) included descriptions of specific autism-based conditions that were developmentally pervasive, but contained key features that differentiated them into specific diagnoses (e.g., the relatively normal communicative abilities observed in AS). Additionally, for individuals who experienced many diagnostic aspects of ASD but were unable to obtain an official diagnosis due to late onset, atypical symptomatology or sub-threshold symptomatology, a diagnosis of *Pervasive Developmental Disorder- Not Otherwise Specified* (PDD-NOS) could be made (APA, 1994; 2000). These changes certainly presented autism more broadly to the clinical world, however, they subsequently led to increased scrutiny over what the broadening of the autism spectrum should look like going forward (Leekam, Libby, Wing, Gould, & Taylor, 2002).

In the early to mid 1990's it was not uncommon for the term PDD to be used in reference to the separate autistic classifications (e.g., AS and PDD-NOS) of the DSM-IV (APA, 1994). Nevertheless, in the years leading up to the publication of the revised fourth version (DSM-IV-TR; APA 2000), empirical explorations of the different diagnostic categories of PDD (in particular AS vs. ASD) surfaced in response to the uncertainty surrounding their distinct diagnostic differences. Much of the research exploring the different forms of PDD failed to clearly outline discrepancies between the disorders' causal mechanisms, behavioural manifestations, and recommended approach to intervention (Eisenmajer et al., 1996; Ghaziuddin, Butler, & Ghaziuddin, 1994; Ghaziuddin, Alessi, & Greden, 1995; Ghaziuddin & Butler, 1998; Gilchrist et al., 2001; Iwanaga et al., 2000; Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; Klin, 2000; Kurita, 1997; Miller & Ozonoff, 2000; Ozonoff, South, & Miller, 2000; Szatmari, Tuff, Finlyson, & Bartolucci, 1990; Szatmari, Archer, Fisman, Streiner, & Wilson, 1995). Even Wing and her colleagues struggled to achieve clear demarcation between the categories of autism, acknowledging the likelihood of an underlying spectrum of autism throughout their research (Gillberg, 1990; Wing, 1998; Wing & Gould, 1979; Wing, Gould, & Gillberg, 2011). This inability to consistently differentiate these disorders led to the decision to classify them as one diagnosis characterised by persistent deficits in social communication and interaction, and presence of restricted and repetitive patterns of behaviours and/or interests (Frith, 2004; Schopler, 1996). This research, in combination with the observed crossover in genetic, behavioural, and therapeutic characterisations of these varying pervasive developmental disorders, has since led to the separate disorders being classified as one diagnosis on the same spectrum in the most recent version of the DSM (DSM-5, APA, 2013; Frith, 2004).

Primary diagnostic features of Autism Spectrum Disorder as per the Diagnostic and Statistical Manual of Mental disorders 5th Edition. The DSM-5 no longer contains the

separate diagnostic labels (e.g., AS, PDD-NOS) provided in the two previous versions of the diagnostic manual (APA, 1994; 2000; 2013). Instead the latest version of the DSM focuses on a more all-encompassing description of the broad spectrum of topographical characteristics (Birtwell, Willoughby, & Nowinski, 2016) that represent ASD; these include individuals that present as low- (marked symptom severity) to high-functioning (milder symptom expression paired with average or higher intellectual abilities; Eaves & Ho, 2008; Kuhlthau et al., 2010; Starr, Szatmari, Bryson, & Zwaigenbaum, 2003; Wilkinson, 2015). Moreover, the three previous diagnostic categories have been reduced to two: social communicative and interactive symptoms, and restricted and repetitive symptoms.

According to the DSM-5, social communicative and interaction symptoms manifest via impairment in social-emotional reciprocity; nonverbal communication behaviours; and the development and maintenance of relationships appropriate to developmental level (APA, 2013). Restricted and repetitive symptomatology must include at least two of the following: stereotyped or repetitive behaviour; highly restricted, fixated interests; excessive adherence to routines and ritualised patterns of behaviour; and/or hyper- or hypo-reactivity to sensory input (APA, 2013). In addition to this, clinical evidence supporting the presentation of autism alongside other neurodevelopmental, psychological, and psychiatric disorders (Volkmar et al., 2012) has resulted in the allowance of dual diagnoses of ASD and such disorders (e.g., ADHD) where applicable (APA, 2013). It is important to note that due to such changes and the subsequent increase in correspondence between diagnostic criteria and empirical research, the DSM-5 can be utilised for both clinical and research work (Volkmar et al., 2012). Thus, for the purposes of the present research, classification of ASD will adhere to the diagnostic criteria contained in the DSM-5 (APA, 2013).

The changes in the DSM-5 will likely impact the prevalence rates of ASD, however, it is uncertain if higher prevalence rates will result from the broader, more all-encompassing

spectrum of disorder, or if lower prevalence rates will result from the narrowing of diagnostic categories and removal of the more loosely defined PDD-NOS (Hill et al., 2016). One of the main criticisms of the DSM-IV and its revised version was that it widened the criteria for autism and its related disorders so much so that it possibly contributed to the a marked increase in published prevalence rates of ASD observed in the years following publication of these manuals (see Croen, Grether, Hoogstrate, & Selvin, 2002). However, that increases in public awareness and treatment availability, as well as changes to the classification and conceptualisation of autism, have also contributed to the steady growth in prevalence rates and referrals to specialists or special education registers observed over the past several decades (Durkin et al., 2008; Fombonne, 2005; Gurney et al., 2003; Shattuck, 2006). Additionally, the more recent focus on early intervention and improvements to screening for ASD in younger children has resulted in diagnosis at a younger age and, subsequently, the diagnostic inclusion of individuals on the spectrum who may have been missed in earlier years (Wazana, Bresnahan, & Kline, 2007).

Although recent research suggests that behaviour indicative of an ASD diagnosis may be detected before 12 months of age (Ozonoff et al., 2014; Zwaigenbaum et al., 2005), symptoms (e.g., atypical language development, communication patterns, social interest, and patterns of play) are more likely to be noticed between 12-24 months. In some cases, early childhood symptoms may be too subtle to detect and an ASD diagnosis may not be made until late childhood or early adolescence (Birtwell et al., 2016), but overall, large, community-based samples have yielded a median diagnostic age of 4 years old (CDCP, 2016; Shattuck et al., 2009). A review of diagnostic practices over the past several decades reveals significant improvements to the recognition of ASD in children; however, the same cannot be said for adults who were not initially recognised as having ASD in childhood (Rabins, 2016). Individuals presenting for diagnosis for the first time from late childhood onward often lacked

evidence of the profile of infant/toddler behaviours necessary for diagnosis, particularly if they did not have someone who was able to provide an accurate historical account of their early behaviour and development (Wing et al., 2011). Prior to publication of the DSM-5, the age-specific diagnostic criterion for autism required the appearance of symptoms in early childhood (APA, 1952; 1968; 1980; 1987; 1994; 2000). To remedy this, the DSM-5 now states that the onset of symptoms should be in the early developmental period, however, it also notes that deficits may not become fully manifest until social-communication demands exceed limited capacities (APA, 2013). Nevertheless, the developmental trajectory of people with ASD in the later stages of life is yet to be understood in great detail (van Heijst & Geurts, 2015).

Age-related differences in the presentation of Autism Spectrum Disorder. Research on autism has largely focussed on those thought to be most affected by the disorder (i.e., children) and, subsequently, the development of autism-specific early intervention approaches (James, Mukaetova-Ladinska, Reichelt, Briel, & Scully, 2006). Only in the last decade or so have researchers and clinicians begun to question the life course and prognosis of ASD (Howlin & Moss, 2012; Piven & Rabins, 2011; Smith, Maenner, & Selzer, 2012). Consequently, a paucity of systematic investigation exists with respect to the progression of ASD across the lifespan (Howlin & Magiati, 2016). Taking into consideration the recent increases in diagnostic rates of ASD in childhood (CDCP, 2016; Fombonne, 2001) this is problematic, as large numbers of youths with ASD are now transitioning into adulthood, many of whom will face ASD-related impairments for the remainder of their lives, possibly without accurate detection and effective support (Gerhardt & Lainer, 2011).

Although the key characteristics of ASD have, on average, been observed to improve modestly with age (Esbensen, Seltzer, Lam, & Bodfish, 2009; Happé & Charlton, 2012), this does not translate to a progressive decline in symptomatology across all individuals with the

disorder (McGovern & Sigman, 2005). In keeping with the notion that ASD is a disorder of innate neurobiological deficit, a review of longitudinal studies on ASD over the past several decades reveals that, although many individuals show slight behavioural improvements from adolescence into adulthood, most continue to experience marked impairment as a result of their ASD across the lifespan (Anderson, Liang, & Lord, 2014; Farley et al., 2009; Hippler & Klicpera, 2003; Howlin et al., 2004; Howlin, Moss, Savage & Rutter, 2013; Kanner, 1971; Rutter, Greenfield, & Lockyer, 1967). Recent research demonstrates that only a small proportion (8-10%) of individuals diagnosed with ASD in childhood no longer meet diagnostic criteria by adulthood (Anderson et al., 2014; Orinstein et al., 2014; Seltzer, Shattuck, Abbeduto, & Greenberg, 2004). Nevertheless, although there is evidence to support the stability of the ASD diagnosis over time, there is great heterogeneity in the developmental and trajectories of individuals with this disorder (Birtwell et al., 2016). Consequently, further exploration of the progression of both the social-communicative and restricted and repetitive symptomatology in ASD is required.

Social-communicative and interactive symptomatology. Communication involves the verbal, gestural, and written comprehension and expression of information to and from others (American Speech-Language-Hearing Association, 1993). In typically developing children, communication starts in infancy with responses to caregiver cues and environmental stimuli (e.g., facial expressions, vocalisations), and progresses to the formulation of basic vocabulary and, eventually, the production of simple sentences and use of non-verbal body language such as eye contact and basic hand gestures (Howe et al., 2016). Many of these skills develop over the first three years of a child's life and, subsequently, the failure to achieve such verbal and nonverbal communication milestones for children with ASD is often observed between 12-24 months of age (Howe et al., 2016b). This is important to note because problems in the early processing of social stimuli may contribute to the social-communicative impairment

profile of ASD later in life, including skills necessary for reciprocal interaction such as emotion perception, attenuated joint attention, social orienting and affective sharing (Webb, Dawson, Bernier, & Panagiotides, 2006; Dawson, Meltzoff, Osterling, Rinaldi & Brown, 1998).

Both children and adults with ASD rarely respond to the social advances of others aptly, primarily communicating for the purpose of delivering a self-interest based monologue, or to identify a personal need that requires attention (Downs & Smith, 2004). What often results is a consistent inability to comprehend appropriate ways of joining and engaging in conversation with others (Odom et al., 2016). In children, the *social-reciprocity* deficits of ASD often affect their ability to engage in cooperative social behaviour, with diminished interest in working toward a common goal with other children or sharing play experiences (Cohen & Volkmar, 1997). In adults, impairments to social-reciprocity are experienced in the workplace, with difficulties conversing effectively with colleagues and in disclosing feelings of work-related stress or anxiety to superiors (Gallo, 2010). Moreover, in the context of romantic relationships, the frequent exchange of emotions or reciprocation of obvious, known facts can be considered illogical to the ASD adult, so even simple romantic gestures such as saying "I love you" on a daily basis are often not carried out (Aston, 2003).

The general lack of social-communicative awareness in ASD can also result in the presentation of *verbal and nonverbal communication* that is not well received by others, with verbal language lacking in appropriate emotion and accompanied physical orientation/gesture, and often presenting as pedantic, overly formal, corrective, and sometimes even argumentative in nature (Ghaziuddin & Gerstein, 1996). Moreover, these symptoms can manifest in an inability to read or comprehend expressions of emotions from others that may offer insight into what they are thinking or feeling over the course of an interaction (Golan & Baron-Cohen, 2006). Accordingly, both children and adults with ASD

often have difficulty with nuances of language such as irony, sarcasm or humour (Howe et al., 2016b; Kleinman, Marciano & Ault, 2001). Children and adults with high-functioning ASD may converse reasonably well in one-on-one interactions, processing individual social cues and using memory of similar social situations to aid their responses. In a group setting, however, they may lose track of a conversation as their ability to process a larger amount of social information takes longer than that of a neurotypical individual; this can result in long pauses in conversation and eye-contact, the use of inappropriate social responses, or complete withdrawal from the conversation altogether (Gallo, 2010). Conversely, children and adults with low-functioning ASD tend to struggle with more basic social situations, such as those involving noisy environments or loud conversations with larger groups of people, with the overstimulation proving to be too distracting or upsetting (Folstein & Carcache, 2016).

Given these issues of initiating, contributing to, and sustaining conversations and interactions, both children and adults with ASD are often unable to *develop and maintain relationships appropriate to developmental level*. Even when basic verbal and nonverbal communicative skills are intact, children and adults with ASD often struggle to engage in the compromises that is required to maintain a relationship with same-age peers (Gallo, 2010). This symptom class has clear associations with impairments to social functionality, and as such, will be addressed in further detail in the section of this chapter discussing functionality in ASD.

Restricted and repetitive symptomatology. In addition to impairments in social interaction, individuals with ASD demonstrate *stereotyped and repetitive* symptomatology. Most children with ASD engage in some form of stereotyped or repetitive speech, motor movements, or object use at some point over the course of their development (Leekam, Prior, & Uljarevic, 2011; Murphy et al., 2005). Repetition in the ASD behavioural repertoire can also manifest as *strict adherence to routine or insistence on sameness*. Children with ASD

might insist on sameness such as always winning, being first, or following their own set of rules for play (Chalfant, 2011), and may be reluctant to try new activities/games, or to go to new places, such as another child's house or venue for a birthday party (Carrington, Templeton, & Papinczak, 2003). Children with ASD may also react adversely (e.g., tantrums, screaming, self-injurious behaviour) to changes in day-to-day activities such as diversion from morning or bedtime routines, taking a different route to school, the child's computer not working, or the misplacement/removal of a favourite possession (Folstein & Carcache, 2016). Into adulthood, these forms of restricted and repetitive behaviours are most commonly affiliated with lower-functioning ASD (Goldman et al., 2009), although most adults with ASD do reportedly experience difficulties breaking routine (Chalfant, 2011) and transgressing from the truth or their own strict ethical codes (Folstein & Carcache, 2016).

One symptom subclass that reportedly becomes more profound with age (as language and cognitive capacities further develop) is *highly restricted or fixated interests* on a range of topics or objects (Barrett, Prior, & Manjiviona, 2004; Bashe & Kirby, 2001; Bishop, Richler, & Lord, 2006; Esbensen et al., 2009; Fecteau, Mottron, Berthiaume, & Burack, 2003; Richler, Huerta, Bishop, & Lord, 2010). In children, fixation on a particular topic or object can result in the persistence of play activities, such as playing with the same doll or action figure, even when their peers have moved on to other areas of interest. Conversely, in adults it can present as the persistent need to study or converse on a particular topic of interest (Howe, Palumbo, & Neumeyer, 2016).

Although circumscribed interests usually centre on a specific topic or object, the preference for certain items and experiences extends to the manner in which sensory sensations are experienced. *Hyper/hypo-reactivity to sensory input* can involve intolerance of particular sounds, sensations, clothing, and, subsequently, avoidance of certain foods and textures, or the excessive pursuit to engage with specific sensory experiences (Howe et al.,

2016a). Studies have demonstrated that problematic reactions to sensory input can persist well into adulthood (Billstedt, Gillberg, & Gillberg, 2007), however, symptoms relating to sensory sensitivity are more commonly observed in children with ASD as opposed to adults (Kern et al., 2006; Leekam et al., 2011; Leekam, Nieto, Libby, Wing & Gould, 2007; Murphy et al., 2005; South, Ozonoff, McMahon, 2005). Children with ASD have been observed to have higher rates of food refusal than their neurotypical peers, demonstrating a more restricted food range, more requirements for the presentation of food/utensils, and a higher likelihood to consume inedible objects (Twachtman-Reilly, Amaral, & Zebrowski, 2008), behaviours that are often linked to rituals reflecting insistence on sameness (Folstein & Carcache, 2016). Persistent preoccupation with sensory stimulating objects or even parts of objects can also take place, resulting in the frequent manipulation and exploration of wheels, fans, lights, light switches, and door-knobs, with interest in these items sometimes exceeding that of interest in regular toys (Gallo, 2010).

A review of the literature on restricted and repetitive symptomatology in ASD demonstrates that concrete and physical/movement symptoms (e.g., repetitive body movements or vocalisations) are more apparent in younger and lower-functioning individuals, with more language- and activity-based behaviours (e.g., highly sophisticated, obsessive interests) typically observed in older or more higher-functioning individuals (Barrett et al., 2004; Bishop et al., 2006; Esbensen et al., 2009; Richler et al., 2010; South et al., 2005). It is not necessarily the systematic form or pattern of restricted and repetitive behaviours that differentiates between high- and low-functioning individuals with ASD, but rather the frequency, with higher frequencies observed in lower functioning individuals (Bodfish, Symons, Parker, & Lewis, 2000; Lam & Aman, 2007; Militerni, Bravaccio, Falco, Fico, & Palermo, 2002; Richler, Bishop, Kleinke, & Lord, 2007). Regardless of symptom severity, both high and low-functioning ASD is associated with risk of impairment in daily

functioning with core symptoms acting to disrupt the individual's capacity to meet age-appropriate demands.

Impacts of Autism Spectrum Disorder's primary features on daily functioning. An individual's ability to meet the demands of everyday living depends on his/her capcity to socialise (e.g., successfully interact with others), communicate meaningfully (e.g., express needs and wants), and execute independent living skills (e.g., personal hygiene; Birtwell et al., 2016). Although, on average, there are improvements to adaptive functioning with age, the overall level of competence in this area for adults with ASD remains relatively low in comparison to neurotypical adults of similar age and IQ (Magiati, Tay, & Howling, 2014; Smith et al., 2012). It is unusual for an individual with ASD to have the same level of impairment across all areas of functioning across the lifespan (Gallo, 2010). As a heterogeneous neurodevelopmental disorder, ASD is composed of a broad spectrum of not only topographical characteristics, but functional impairments with respect to social-communicative, academic, and occupational ability (Farley et al., 2009; Lee & Park, 2007; Starr et al., 2003).

Impairments to social-communicative functioning in Autism Spectrum Disorder. Social-communicative and interactive symptomatology in ASD can lead to difficulty understanding the intentions and thoughts of others (Baron-Cohen, Leslie, & Frith, 1985), making it difficult to discriminate between situations where others are merely joking and where their actions are ill intended (Odom et al., 2016). ASD children and adolescents with such social naïveté can find themselves in situations where they are taken advantage of or that result in teasing or bullying; for example, they may repeatedly give others their lunch money, do others' work-related tasks, or engage in antisocial behaviour at the request of peers in an attempt to gain friendship (Bauminger & Kasari, 2000). Common adverse social experiences of children with this disorder include not having questions answered in conversation;

deliberately being chosen last to be part of a group or team; not being invited to social events; failure to be accepted by peers; ongoing association with a marginalised group; and experiences of bullying, victimisation, or discrimination (Hurlbutt & Chalmers, 2002; Huws & Jones, 2008; MacLeod & Johntson, 2007; Punshon, Skirrow, & Murphy, 2009; Purkis, Goodall, & Nugent, 2016b). Consequently, what is often observed is the tendency of the ASD child to establish relationships with children who are significantly younger and typically much more impressionable, or significantly older and typically more obliging, than same-age peers (Gallo, 2010). Nevertheless, even when children with ASD are able to establish relationships with their same-age classmates, they often report poorer friendship quality and fewer reciprocal relationships than their typically developing peers (Kasari, Locke, Gulsrud, & Rotheram-Fuller, 2011).

Social-communicative impairments carry the potential to worsen with age, with social deficits often becoming more pronounced in adolescence as the social environment becomes more complex (Chalfant, 2011). By adolescence, individuals with ASD are often better able to understand the social differences between them and their typically developing peers, but despite this increase in awareness, ongoing ASD-related social communication and interaction impairments result in a continued battle to establish and maintain good quality relationships (Cederlund, Hagberg, Billstedt, & Gillberg, 2010; Cesaroni & Garber, 1991; Hurlbutt & Chalmers, 2002; Jennes-Coussens, Magill-Evans, & Koning, 2006; MacLeod & Johnston, 2007; Punshon et al., 2009). Accordingly, adolescents with ASD are reportedly less likely to see friends outside of school, get invited to social events, or receive phone calls from friends, and are more likely than their neurotypical peers to experience isolation and low social status (Wagner, Cadwallader, Garza, & Cameto, 2004).

Adults with ASD may initially impress others by conveying a strong sense of social justice and moral conviction, or by appearing reserved and deep-thinking (Aston, 2003). But

their lack of response to and interest in others, and their inability to adhere to social norms, often leads them to present as rude, selfish and inconsiderate, adversely impacting their capacity to engage in conversations and basic interactions with other people (Frith, 2004; Gallo, 2010). For example, adults with ASD are often disciplined for not being a team player in the workplace or for having a dispassionate attitude towards co-workers (National Collaborating Centre for Mental Health, 2012). In addition to this, studies show that approximately half of adults with ASD do not have a group of friends with whom they engage with regularly and that, on average, the friendships they do have tend to be less close and supportive than those observed in neurotypical adults (Baron-Cohen & Wheelwright, 2003; Howlin et al., 2004; Howlin & Moss, 2012; Mawhood, Howlin, & Rutter, 2000; Orsmond, Krauss, & Seltzer, 2004). For example, research demonstrates that only approximately 20% of adults with ASD develop intimate relationships that involve reciprocity and sharing (Howlin & Moss, 2012). The negative experiences that adults with ASD encounter as a result of their enduring symptomatology devalues their perception of themselves and their expectations of living a life full of personal enjoyment and success (Huws & Jones, 2008; Punshon et al., 2008; Purkis et al., 2016b). As these social and interpersonal problems persist into adulthood, so do the feelings of failure and isolation that accompany them (Mazurek, 2014; Orsmond, Shattuck, Cooper, Strezing, & Anderson, 2013), often resulting in increased experiences of marginalisation and loneliness that continue across the lifespan (Head, McGillivray, & Stokes, 2014).

Impairments to academic functioning in Autism Spectrum Disorder. In addition to impacting an individual's ability to engage with others more generally, social-communicative issues in children and adults with ASD can further impact social functioning in the classroom. Schools are highly social contexts, with success often dependent on the relationships formed between students, their peers and their teachers. Early in their studies,

students are expected to continuously interact with their peers and contribute to projects in meaningful ways, engaging in turn taking, cooperative play, and the sharing of classroom materials (Odom et al., 2016). Unfortunately, the difficulty children with ASD experience in discriminating among multiple social cues in the classroom environment makes it more difficult for them to observe their peers and learn skills vicariously (Plavnick & Hume, 2014).

In addition to the social aspect of the classroom, there are tasks relating to academic work that can be impeded by ASD. From kindergarten onward, children are expected to stay on-task and engaged with a range of activities in the absence of adult prompting; this is problematic for children with ASD, however, as they are often reliant on cues from adults to assist in the navigation of changes in class routines (Hume, Loftin, & Lantz, 2009), and are limited in the classroom activities they engage in due to an intense focus on or preoccupation with a narrow range of interests (Estes, Rivera, Bryan, Cali & Dawson, 2011). Diminished reactivity to peers, intense preference to explore a particular topic of interest, or refusal to accept teacher instructions perceived to be illogical by the ASD child can result in children on the spectrum appearing disinterested or challenging in a classroom setting (NCCMH, 2012), and, moreover, can result in difficulty following a teacher's direction or staying on task (Estes et al., 2011). These challenges pose serious limitations to learning, as children who are not able to independently complete tasks or respond to a teacher's direction often struggle to participate in, and benefit from, classroom activities (Machalicek, O'Reilly, Beretvas, Sigafoos, & Lancioni, 2007).

The dynamic of the classroom continues to increase in complexity with age for ASD individuals, with greater emphasis on academic achievement coupled with continued hostility, frustration, and rejection from peers (Sreckovic, Brunsting, & Able, 2014). Accordingly, as measured by performance on academic achievement tests and high school

graduation rates, adolescents with ASD demonstrate a lower level of academic achievement than their neurotypically developing peers (Hendricks & Wehman, 2009), with less than half going on to attend college (Cederlund, Hagberg, Billstedt, Gllberg & Gillberg, 2008; Eaves & Ho, 2008; Howlin, Alcock, & Burkin, 2005; Howlin, Mawhood, & Rutter, 2000; Shattuck et al., 2012; Taylor, & Seltzer, 2011). In young adults with ASD, the period of time after leaving high school is typically met with slowed improvement in symptoms, perhaps due to decreases in daily structure, the number of services and supports available to them, and limited exposure to daytime activities (Shattuck et al., 2012). Subsequently**,** young ASD adults with the intellectual ability to attend and even graduate from post-secondary schooling, often do not pursue a tertiary education due to ongoing symptomological impacts surrounding the challenges of living away from home, navigating the complex social world of a college or university setting, problems managing daily living and finances, and the stress of increases in responsibility (Brodkin, 2016). This latter point demonstrates that even higher-functioning adults with ASD experience sustained impairments in functioning and barriers to meeting their personal goals.

Impairments to occupational functioning in Autism Spectrum Disorder. In line with the findings on school experiences and achievement, adults with ASD often experience difficulty gaining and maintaining steady employment (Hendricks & Wehman, 2009; Hurlbutt & Chalmers, 200). For example, many adults on the spectrum struggle to successfully complete a job interview due to their inability to comprehend social cues and meaningfully interact (Müller, Schuler, Burton & Yates, 2003). Even if these adults are able to make it past this stage, social challenges surrounding interactions with colleagues and office "politics" are typically difficult to navigate (Brodkin, 2016). Moreover, for those on the spectrum who do secure employment, many are underutilised and underpaid, completing menial tasks that they find uninteresting (Taylor & Seltzer, 2011).

Some adults with ASD may be at an advantage as a result of extensive and detailed knowledge in a particular area arising from a perseveration and intense fixation on a particular topic in childhood/adolescence (Howlin, 2003). High-functioning adults with ASD may become real experts in their area of interest and, if able to find a niche in employment, can put their expertise to good use (Howlin, 2003). Nevertheless, a very strong interest can lead also to difficulties in convincing adults with ASD to engage in activities outside of the preferred topic's pursuit, such as obtaining employment in another area if their area of interest contains low employment prospects (Folstein & Carcache, 2016). Accordingly, in addition to being markedly underemployed (approximately 70% of adults with ASD are unemployed; Magiati et al., 2014) and underpaid (Mavranezouli et al., 2014; Roux et al., 2013), adults with ASD tend to experience a high job turnover rate and, overall, find engaging in paid or voluntary work less enjoyable than do their neurotypical peers (Hendricks & Wehman, 2009; Hurlbutt & Chalmers, 2002; Müller et al., 2003).

The progression of functional impairments in adulthood in Autism Spectrum Disorder. In ASD, impairments to academic and occupational functioning often result in a transition into adulthood that is met with diminishing vocational, educational, and employment opportunities and, subsequently, limited access to often poor-quality provisions (Howlin & Magiati, 2016). Without the prospect of stable employment, many adults on the autism spectrum are unable to transition into any form of independent living, remaining economically and emotionally dependent on their families (Barnhill, 2007; Baxter et al., 2014; Buescher, Cidav, Knapp, & Mandell, 2014; Farley et al., 2009; Howlin, Goode, Hutton, & Rutter, 2004). Additionally, a lack of regular daily activities (e.g., work or study) means there is little to no opportunity to meet people their age to form and maintain stable relationships with, contributing to the increase in isolation often observed in adults with ASD (Gantman, Kapp, Prenski, & Laugeson, 2012; Howlin & Magiati, 2016; Howlin et al., 2004;

Farley et al., 2009). Accordingly, follow-up studies on adults diagnosed with ASD in childhood reveal that more than half end up living in some form of supported accommodation (e.g., hospital, psychiatric facility, residential placement; Ballaban-Gil, Rapin, Tuchman, & Shinnar, 1996; Rutter, 1968), less than one-third go on to work or engage in tertiary study (Ballaban-Gil et al., 1996; Rutter, 1968), and more than half report marked difficulties in socialisation (e.g., an inability to maintain successful, stable relationships with peers; Ballaban-Gil et al., 1996). Some adults with ASD are able to live and work independently and live otherwise successful lives; however, many become isolated from society, having little to no autonomy (Balfe & Tantam, 2010; Barneveld, Swaab, Fagel, van Engeland, & de Sonneville, 2014; Cederlund et al., 2008; Eaves & Ho, 2008; Engström, Ekström, & Emilsson 2003; Howlin et al., 2004) and report poorer quality of life than do their neurotypical counterparts (Helles, Gillberg, Gillberg, & Billstedt, 2017; van Heijst & Geurts, 2015).

Common comorbidities in Autism Spectrum Disorder. The variation in functional impairment in ASD is heavily influenced by the heterogeneous nature of the disorder across the lifespan. Other factors, such as psychiatric comorbidity, are also known to influence such outcomes (Helles et al., 2017). Studies on clinical and community samples suggest that individuals with ASD have a diagnosable comorbid psychological disorder of 80% and 30%, respectively (see Ghaziuddin, 2005; Levy & Perry, 2011; Leyfer et al., 2006; Simonoff et al., 2008). This is problematic because individuals with ASD and comorbid disorders exhibit maladaptive behaviour, and subsequent impairments to function, not only stemming from their ASD but from other psychopathologies (Folstein & Carcache, 2016; Gadow, Guttmann-Steinmetz, Rieffe, & Devincent, 2012; Ghaziuddin & Zafar, 2008; Gillberg & Fernell, 2014; Howlin & Moss, 2012; Mazzone et al., 2013). The most common comorbidities include internalising disorders such as depression, anxiety, and Obsessive Compulsive Disorder

(OCD), and other neurodevelopmental disorders such as ADHD (Bellini, 2004; Bradley, Summers, Wood, & Bryson, 2004; Ghaziuddin, Ghaziuddin & Greden, 2002; Ghaziuddin & Zafar, 2008; Hill & Furniss, 2006; Hofvander et al., 2009; Joshi et al., 2013; Kim et al., 2000; LoVullo & Matson, 2009; Mouridsen, Rich, Isager, & Nedergaard, 2008; Simonoff et al., 2013). Similar to ADHD, the research on internalised comorbid diagnoses (e.g., anxiety, depression, OCD) in ASD spans over several decades; the same cannot be said, however, about the comorbid consideration of ASD and ADHD.

As discussed in Chapter 2, further expanding the research on ADHD and ASD may advance the formulation, assessment, and treatment of these neurodevelopmental disorders through the simultaneous consideration of their points of convergence and divergence (Brown et al., 2001; Loeb et al., 2012). A review of the literature on ADHD and ASD in relation to diagnostic development and symptom manifestation reveals some strong similarities in core features exist with respect to difficulties in communication, impulsivity, attention, and varying degrees of restlessness (Bishop & Baird, 2001; Buitelaar, Van der Gaag, Klin, & Volkmar, 1999; Cantwell, 1996; Friedman et al., 2003; Marton, Wiener, Rogers, Moore, & Tannock, 2009; Rao & Landa, 2013; Ronald et al., 2008). However, from a transdiagnostic perspective, the process through which a greater understanding of ADHD and ASD can be achieved should not be solely reliant on the investigation of core diagnostic features (Mansell et al., 2008; Kessler et al., 2005). More specifically, these disorders are best explored within the context of a developmental trajectory where the various secondary features contributing to the diverse outcomes of individuals with these disorders (e.g., aetiological, cognitive, affective, and behavioural factors) can be explored at length. The aetiological underpinnings of ADHD and ASD have been explored, in detail, in Chapters 2 and 3, respectively. Additional features evidenced as common to ADHD and ASD in the

literature (e.g., those of cognition, behaviour and emotion) will be reviewed, in detail, in the next chapter.

Chapter Four: Secondary Features Common to Attention-Deficit Hyperactivity and Autism Spectrum Disorders

Similar to the observation that certain factors (e.g., comorbid diagnoses) further compound functional impairments in ADHD and ASD, it is possible that learning more about features common to these disorders outside of their core symptoms may provide further insight into ways in which functionality can potentially be enhanced (Halperin & Healey, 2011). Common, but non-diagnostic, characteristics of ADHD and ASD, known as *secondary features,* can be cognitive, behavioural, emotional, or physical in nature, and typically present as recurring aspects of these disorders that, though still influential with respect to day-to-day functioning, do not fit easily into psychiatric nomenclature (Barkley et al., 2008; Dominick, Davis, Lainhart, Tager-Flusberg, & Folstein, 2007; Lord & Risi, 1998; Rajendran & Mitchell, 2007). The majority of the studies on the secondary features of ADHD and ASD involve children and adolescents (e.g., Dominick et al., 2007; May et al., 2012). This age-based restriction has resulted in a less detailed understanding of how features with obvious links to long-term functional impairments progress across the lifespan of individuals with ADHD and ASD (Klein et al., 2012; Mannuzza et al., 1998; Rommelse, Buitelaar, & Hartman, 2017; Weiss & Hechtman, 1993). In order to investigate what is currently known about the secondary features of ADHD and ASD beyond childhood, the remainder of this chapter will review, in detail, the literature on these features as they present in adults with these disorders.

Research suggests that common secondary features of adults with ADHD include executive dysfunction, and disinhibition in particular (Barkley, 1994; 1997; 2012a; 2012b; Barkley & Murphy, 2010; 2011; Barnhart & Buelow, 2017; Schecklemann et al., 2012); maladaptive behaviour patterns such as those involving aggression (Jensen et al., 2007; Dowson & Blackwell, 2010) and poor social-communicative skills (Barkley et al., 1990; Jarratt et al., 2005); and heightened levels of adverse emotions such as anxiety (Barkley et al.,

1996; 2008; Kessler et al., 2006; Michielsen et al., 2013) and depression (Barkley et al., 2008; Spencer et al., 2000; Torgersen et al., 2006). Studies pertaining specifically to ASD demonstrate that common secondary features in adults include executive dysfunction (Ambery et al., 2006; Lopez, Licoln, Ozonoff, & Lai, 2005; Rumsey, 1985; Rumsey & Hamburger, 1988; 1990; Wallace et al., 2016); irregularities in the processing of emotion (e.g., alexithymia; Berthoz & Hill, 2005; Hill et al., 2004; Silani et al., 2008); maladaptive behaviour patterns such as aggression (Cohen et al., 2010; Pugliese et al., 2015) and inattention (Nydén et al., 2010); and heightened levels of adverse emotion such as anxiety (Mazefsky, Folstein & Lainhart, 2008; Lugnegård, Hallerbäck, & Gillberg, 2011; Rumsey et al., 1985) and depression (Hill, Berthoz, & Frith, 2004; Mazefsky et al., 2010). Secondary somatic features such as disturbances to sleep and eating patterns have also been associated with ADHD and ASD; however, research suggests that these features are most commonly found in children with these disorders (e.g., Corkum, Tannock, & Moldofsky, 1998; Malow & McGrew, 2008; Najdowski, Wallace, Doney, & Ghezzi, 2003; O'Brien et al., 2003; Richdale & Schreck, 2009; Zimmer et al., 2012). Both social-communicative impairments in ADHD and inattention in ASD have been discussed in Chapters 2 and 3, respectively. As such, the secondary features that will be explored in this chapter include the cognitive features of executive dysfunction, disinhibition, and alexithymia; the behaviour-based feature, aggression; and the emotion-based features of anxiety and depression. The review of these secondary features will include empirically based operational definitions of each, followed by a description of how each presents in adults with ADHD and ASD.

Cognitive Features

Approximately 20% of adults with ADHD (Edel et al., 2010) and 40-65% of adults with ASD (Berthoz & Hill, 2005; Hill et al., 2004; Silani et al., 2008) experience irregularities in recognising and processing emotions; these atypicalities are commonly

associated with a cognitive feature known as *alexithymia* (Geller, 2005). Even more prevalent in adults with ADHD and ASD, are certain cognitive features associated with higher-order thought processes (Ambery et al., 2006; Barkley, 1994; 1997; 2012a; 2012b; Barkley & Murphy, 2010; 2011; Barnhart & Buelow, 2017; Lopez et al., 2005; Rumsey, 19985; Rumsey & Hamburger, 1988; 1990; Schecklemann et al., 2012; Wallace et al., 2016). The cognitive atypicalities in ADHD and ASD related to higher-order thought processes are commonly associated with the neurobiological abnormalities of ADHD and ASD (e.g., frontal lobe abnormalities), and, collectively are referred to as *executive dysfunction*.

Executive functioning. In clinical neurobiology and neuropsychology, executive functioning is considered to represent a number of neurocognitive domains (Suchy, 2009). The exploration of executive functioning with respect to its application and functional properties comprises a vast and rapidly growing area of research, hence the approaches to defining executive functioning in this context vary widely. The underlying, covert operations of executive functioning have been divided into different domains including those of response inhibition, cognitive flexibility, cognitive planning, and working memory (Anderson, 2002; Baddeley, 2000; Barkley, 1997; Hill, 2004; Norman & Shallice, 1986; Rogers & Bennetto, 2000; Zelazo, Carter, Reznick, & Frye, 1997). As such, for the purpose of Studies 1 and 2, executive functioning has been defined as a multifaceted, neuropsychological construct consisting of a set of higher-order neurocognitive processes that facilitate decision-making and the formulation of purposeful, goal-directed, and future-oriented behaviour (Cummings & Miller, 2007; Gazzaley & D'Esposito, 2007; Welsh & Pennington, 1988).

Assessment of executive domains. Studies on executive functioning have largely been conducted by researchers exploring cognitive development, based on the notion that this feature is comprised of a series of cognitive operations measured by their behavioural

outcomes (e.g., impulsive actions; Zelazo et al., 1997; Zelazo & Müller, 2002). Consequently, though the internal domains that comprise executive functioning are covert in nature, the majority of studies exploring these operations have focused on observing and mapping overt responses to task-based psychometric assessments. In the sections to follow, the most common psychometric assessments designed to assess the specific domains of executive functioning will be reviewed, following a brief definition of each operation. Approaches to assessing *multiple domains* of executive functioning will then be discussed, as well as the application of self-report scales to executive functioning.

Psychometric assessment of response inhibition. Response inhibition has been described as the ability to suppress a response that is inaccurate or maladaptive, and has been assessed by a variety of psychometric tools over the last several decades (Rajendran & Mitchell, 2007). Response inhibition is commonly assessed by tasks designed to measure an individual's ability to respond to a target stimulus (e.g., the selection of only red coloured shapes) while withholding, or inhibiting, responses to distracting or irrelevant stimuli (e.g., variations in the type of shapes presented). Examples of such tests of response inhibition include stop-signal tasks (SST; Logan, Cowan, & Davis, 1984); go/no-go discrimination tasks (Trommer, Hoeppner, Lorber, & Armstrong, 1988); Stroop tasks (Stroop, 1935), such as the Stroop Colour-Word Test (Golden, 1978); the Hayling Sentence Completion test (Burgess & Shallice, 1997); and continuous performance tests/tasks (CPT; Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956).

Psychometric assessment of cognitive planning. Cognitive planning involves conceptualising change from the current situation in order to arrive at a situation-appropriate response (Hill & Bird, 2006). Cognitive planning is typically assessed via tasks measuring the ability to devise a cognitive plan and complete a series of problem-solving activities in the shortest number of moves/amount of time whilst adhering to a specific set of rules (e.g.,

move an entire stack of discs from one rod to another, one disc at a time, with larger discs never sitting on top of the smaller ones). Examples of such tests of cognitive planning include the Tower of Hanoi (ToH) and/or Tower of London (ToL; Shallice, 1982). Cognitive planning is also commonly assessed via tasks that require an individual to connect a series of targets in some form of sequence, such as the first half of the Trail Making Test (TMT; Reitan, 1955; 1958).

Psychometric assessment of cognitive flexibility. Cognitive flexibility involves shifting between situation-appropriate thoughts or actions, and as such, is assessed by psychometric tasks measuring cognitive fluidity and adaptability (Hill & Bird, 2006). Cognitive flexibility is most commonly assessed via tasks that measure an individual's ability to switch between different concepts in the face of changing, and often unpredictable, reinforcement schedules (e.g., matching cards according to colour vs. shape). Examples of these tests of cognitive flexibility include the Wisconsin Card Sorting Test (WCST; Heaton, Chelune, Talley, Kay, & Kurtiss, 1993) and the Brixton Spatial Anticipation Test (Burgess & Shallice, 1997). Cognitive flexibility can also be assessed via tasks requiring an individual to engage in multiple activities simultaneously (e.g., sequencing numbered *and* lettered targets), such as the latter half of the TMT (Reitan, 1955; 1958).

Psychometric assessment of working memory. Working memory has been defined as the maintenance and manipulation of task relevant information (e.g., previous responses to similar situations), aimed at guiding future response formation (Baddeley, 2003; Cowan, 2008; 2014). Working memory is typically assessed by tasks exploring memory storage in which items (e.g., words in a sentence) must be recalled whilst an individual engages in competing cognitive tasks (e.g., mathematical calculations). Examples of this type of test of working memory include dual-task or *n*-back paradigms (Daneman & Carpenter, 1980; Jaeggi et al., 2003; Kirchner, 1958), as well as elements of certain intelligence tests, such as

the letter-number-sequencing test (LNS) of the Wechsler Adult Intelligence Test (WAIS; Wechsler, 1939). Working memory can also be assessed via tasks accessing an individual's memory capacity (e.g., tasks of basic problem solving and reading comprehension; Chuderski, 2013; Oberauer, Süß, Wilhelm, & Wittman, 2003).

Psychometric assessment of multiple executive domains. Despite the individual assessments available for various operations within executive functioning, as a whole this form of higher-order thought processing encompasses all of its individual domains and their interactions with one another. Moreover, impairment in any one of these domains can result in diminished executive functioning overall (Cummings & Miller, 2007; Gazzaley & D'Esposito, 2007; Welsh & Pennington, 1988). Accordingly, there are psychometric measures that assess a broad range of executive domains. For example, the Delis–Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001) and the Cambridge Neuropsychological Test Automatic Battery (CANTAB; Luciana & Nelson, 2002) measure a variety of executive operations in both children and adults through a series of unique tasks (e.g., the assessment of response inhibition and cognitive flexibility through design fluency tasks), or through the use of some of the psychometric measures previously established for the specific domains (e.g., Stroop tasks).

More recent research on executive functioning has assessed these operations collectively through a series of self-report scales; these include the Behaviour Rating Inventory of Executive Function (BRIEF-A; Gioia, Isquith, Guy, & Kenworthy, 2000), the Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, & Barratt, 1995), the Impulsive Sensation Seeking subscale (ImpSS; Zuckerman, 2010), the Behavioural Inhibition/Behavioural Approach Scale (BIS/BAS Scale; Carver & White, 1994), the Adaptive Behaviour Assessment System- Second Edition (ABAS-II; Harrison & Oakland, 2003), and the Frontal Systems Behavior Rating Scale (FrSBe; Grace & Malloy, 2001).

These scales encompass a broader scope of executive functioning capabilities, as opposed to the domain-specific nature of many psychometric assessments (e.g., ToL assessing cognitive planning). Additionally, data collected from these scales are derived from personal accounts from the individual and as such, are thought to provide a more accurate representation of the covert features of executive functioning (Barkley, 2012a; 2012b; Barkley & Fischer, 2011; Barkley & Murphy, 2011; Biederman et al., 2006; Boonstra et al., 2005; Burgess, 1997; Burgess, Alderman, Evans, Emslie, & Wilson, 1998; Hervey, Epstein, & Curry, 2004; Jonsdottir, Bouma, Sergeant, & Scherder, 2006; Wallace et al., 2016; Weyandt & Gudmundsdottir, 2015; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005).

Executive functioning in Attention-Deficit Hyperactivity Disorder. Self-report based assessments of executive functioning in ADHD support the existence of executive atypicalities in adults with this disorder (Barkley, 2012a; 2012b; Barkley & Murphy, 2010; 2011; Barnhart & Buelow, 2017). In a study involving 175 adults (M_{age} = 19.06; *SD* = 1.72) with (*n* = 28) and without ADHD (*n* = 147), executive functioning was assessed by the BIS-11 (Patton et al., 1995), the ImpSS (Zuckerman, 2010), the BIS/BAS (Carver & White, 1994), and the FrSBe (Grace & Malloy, 2001). A logistic regression assessing the predictive utility of these measures demonstrated that, when all were entered into the model, it was significant, $X^2(3) = 14.76$, $p < .002$, Nagelkerke's $R^2 = .14$, with an overall prediction of ADHD symptomatology at 85.3%. Overall scores on the BIS-11, and the FrSBe were significant predictors of ADHD symptomatology, with higher scores on these measures resulting in a higher likelihood (1.23x more) of identifying participants in the ADHD group (Barnhart & Buelow, 2017). Results from this study concur with other studies assessing executive functioning in adults with ADHD (e.g., Barkley, 2012a; 2012b; Barkley & Murphy, 2010) and further support the notion of executive atypicalities in adults with this disorder.

There is a strong trend in the research that suggests adults with ADHD exhibit significant weaknesses in mulitpe executive domains. Nevertheless, no other executive domain has been as consistently observed to be in deficit as that of response inhibition (Barkley, 1994; 1997; Dinn, Robbins, & Harris, 2001; Epstein, Connors, Sitarenios, & Erhardt, 1998; Epstein, Johnson, Varia, & Conners, 2001; Lovejoy et al., 1999; Murphy, Barkley, & Bush, 2001; Rapport, Van Voorhis, Tzelepis, & Friedman, 2001; Taylor & Miller, 1997; Walker, Shores, Trollor, Lee & Sachdev, 2000; Willcutt et al., 2005). In a study on 45 adults with ADHD, Schecklmann et al. (2012) assessed response inhibition via an SST (Logan et al., 1984). Results demonstrated significant impairment in the ADHD adults' ability to withhold incorrect responses, as well as longer reaction times in comparison to neurotypical controls. The researchers concluded that these results supported the existence of impaired response inhibition in adults with ADHD (Schecklmann et al., 2012).

The psychometric assessment of other executive domains in adults with ADHD has yielded some findings that indicate significant weaknesses exist in executive domains other than response inhibition. More specifically, in comparison to neurotypical controls, significantly higher levels of impairment in cognitive flexibility (Lovejoy et al., 1999; Taylor & Miller, 1997), cognitive planning (see meta-analysis by Willcutt et al., 2005; also see Lovejoy et al., 1999; Weyandt et al., 2013), and working memory (Lovejoy et al., 1999; Murphy et al., 2001; Schecklmann et al., 2012; Taylor & Miller, 1997) have been reported in adults with ADHD. For example, in the case of cognitive planning, a meta-analysis of 83 studies (Willcutt et al., 2005) on executive functioning demonstrated that 59% of individuals with ADHD experienced significant deficits in their ability to cognitively plan across the lifespan (as assessed by the ToL and ToH; Shallice, 1982). Moreover, in the case of working memory, in Schecklmann et al.'s (2012) study on adults with ADHD, this cognitive domain was assessed via an *n*-back test that required participants to identify previously presented

stimuli by pushing the space bar of a computer. Results demonstrated that, in comparison to neurotypical controls, adults with ADHD were significantly less efficient at identifying previously presented stimuli and did so at a significantly slower rate, indicating that diminished working memory capacity and processing speed characterise some adults with ADHD (Schecklmann et al., 2012). It is important to note that, although these select studies have provided support for atypical functioning on a range of cognitive domains in adults with ADHD, variations in what is observed with respect to certain executive domains (e.g., cognitive planning and flexibility) demonstrate that executive functioning is not impaired as a whole in adults with ADHD.

Executive functioning and the primary and secondary behaviour features of Attention-Deficit Hyperactivity Disorder. Atypicalities in executive functioning are thought to go beyond the scope of information processing deficits, impacting resultant behavioural operations such as the tracking, shifting, initiation, and completion of tasks (Solanto, 2015; Zelazo et al., 1997), and appropriate response selection in the face of competing but erroneous response alternatives (Bradshaw & Sheppard, 2000). From a neurocognitive perspective, given the congruency between the behaviours of patients with prefrontal lobe injuries and the primary and secondary behavioural features of ADHD, it has been theorised that these neurodevelopmental disorders are heavily impacted by atypicalities in cognitive processes and, more specifically, executive functioning (Arnsten & Li, 2005; Barkley, 1997; 2012a; Barrash, Tranel, & Anderson, 2000; Beer, John, Scabini, & Knight, 2006; Brown, 2009; Levin, 1938; Mattes, 1980; Hornak et al., 2003; Pennington & Ozonoff, 1996; Shaw et al., 2006; 2007). Accordingly, it has been proposed that executive dysfunction, and disinhibition in particular, play a key role in the presentation of ADHD symptomatology (Barkley, 1997), with the impulsive and impetuous behaviour frequently observed in adults with ADHD potentially originating from an underlying inability to suppress responses that

may provide access to a desired object or preferred activity (Nigg, 2001; Schachar & Logan, 1990).

Research supports the existence of a relationship between atypicalities in executive functioning and the primary behavioural features of ADHD. More specifically, hyperactive-impulsive and inattentive symptoms have demonstrated significant, positive relationships with executive dysfunction overall (Barkley, 1997; 2012a; Castellanos & Tannock, 2002; Gioia & Isquith, 2002), as well as disinhibition (Barkley & Fischer, 2011). For example, in a study on 105 adults with ADHD (18-37 years old), in which executive functioning was assessed through a battery of psychometric assessments (i.e., ToL, WCST), results yielded significant, positive relationships between deficient executive functioning, and both hyperactive-impulsive ($r = .29$, $p < .05$) and inattentive symptomatology ($r = .36$, $p < .05$) (Stavro, Ettenhofer, & Nigg, 2007). These findings indicate that when irregularities in executive functioning increase in adults with ADHD, so too does their symptomatology.

Research has also linked poor performance on measures of executive functioning to aggression (Damasio et al., 1991; 1996; Giancola, 1995; Giancola & Zeichner, 1994; Hoaken, Giancola, & Pihl, 1998; Lau, Pihl, & Peterson, 1995; Lau & Pihl, 1994; Ogilvie, Stewart, Chan, & Shum, 2011; Pietrini et al., 2000), such that as functionality across executive domains (e.g., response inhibition) increases, the presentation of aggressive behaviour (e.g., hitting another person) decreases (Poland, Monks & Tsermentseli, 2016). In ADHD, there is growing evidence that diminished executive functioning, and response inhibition in particular, is associated with heightened levels of aggression (Allan & Lonigan, 2014; Beauchamp & Anderson, 2010; Hoaken, Shaughnessy, & Pihl, 2003; Jacobson, Williford, & Pianta, 2011; Lau et al., 1995; Riccio, Hewitt, & Blake, 2011; Utendale, Hubert, Saint-Pierre, & Hastings, 2011; Verlinden et al., 2014). It should be noted, however, that the current research exploring aggression and executive functioning in ADHD, and in the general

population, has been criticised for not adequately considering the heterogeneous nature of aggressive behaviour (Poland et al., 2016); this will be further discussed in the section of this chapter on aggression.

Executive functioning in Autism Spectrum Disorder. Similar to ADHD, studies assessing executive functioning via self-report scales in adults with ASD suggest there are deficits in this cognitive domain (Kenworthy, Yerys, Anthony, & Wallace, 2008; Wallace et al., 2016). In a study using the BRIEF (Gioia et al., 2000) and the ABAS-II (Harrison & Oakland, 2003), 35 adults with ASD (18-40 years old; M_{age} = 21.55; SD = 4.12) demonstrated significantly higher levels of dysfunction on all cognitive domains compared to a normative sample ($p < .05$), with the most prominent deficits presenting for cognitive flexibility and metacognition (task initiation, working memory, planning, organisation, and task monitoring) in comparison to standardised cut-off scores of normal executive functioning (Wallace et al., 2016). This study supported the applicability of self-report measures in the assessment of executive functioning, and the persistence of this feature into adulthood in ASD.

Psychometric explorations of executive functioning in ASD suggest that impaired cognitive planning and flexibility are two of the most common executive deficits in adults with ASD (Ambery et al., 2006; Lopez et al., 2005; Rumsey, 1985; Rumsey & Hamburger, 1988; 1990). More specifically, studies using the WCST (Geurts & Vissers, 2012; Towgood, Meuwese, Gilbert, Turner, & Burgess, 2009), the tower tests (Geurts & Vissers, 2012; Hill & Bird, 2006; Nydén et al., 2010) and the TMT (Goldstein, Minshew, Allen, & Seaton, 2002; Geurts & Vissers, 2012; Hill & Bird, 2006; Nakahachi et al., 2006; Towgood et al., 2009) have revealed inflexibility and poor cognitive planning in adults with ASD in comparison to neurotypical controls. In a study of 10 adult males with ASD (18 to 39 years old), cognitive planning was assessed through a task requiring participants to join a series of letters and

numbers in ascending order (Army Individual Test Battery, 1944). In comparison to neurotypical controls, the men with ASD were observed to perform significantly worse on the task, demonstrating marked impairments in their ability to plan and organise information sequentially (Rumsey & Hamburger, 1988). Additionally, to assess cognitive inflexibility in an adult ASD sample, Rumsey and Hamburger (1990) administered the WCST (Heaton et al., 1993) to adult males with (N = 10) and without ASD (N = 15; M_{age} = 26 years old). Results showed that the adults with ASD performed significantly worse, suggesting the presence of cognitive inflexibility in this sample of ASD adults. These findings are important to note given the strong research focus on children and adolescents with ASD to date (Hill & Bird, 2006); however, many of these studies were conducted several decades ago and are in need of replication across larger, more robust samples of adults with ASD.

Studies on other domains of executive functioning in adults with ASD have yielded mixed results. Response inhibition has been found to be significantly impaired in some adults with ASD in comparison to neurotypical controls (Geurts & Vissers, 2012; Lai et al., 2012), but not others (Ambery, Russell, Perry, Morris, & Murphy, 2006; Hill & Bird, 2006; Lopez et al., 2005; Nakahachi et al., 2006; Sachse et al., 2013). Similar results have been found with respect to working memory, with some studies noting impairments in the storage of verbal and spatial information (Geurts & Vissers, 2012; Williams, Goldstein, Carpenter, & Minshew, 2005), with other studies failing to demonstrate the existence of atypicalities in this executive domain in comparison to neurotypical controls (Lopez et al., 2005; Sachse et al., 2013; Towgood et al., 2009; Williams et al., 2005). Nevertheless, these findings are important because they again articulate the complex nature of executive functioning and support the notion that it is not impaired as a whole in adults with ASD.

Executive functioning and the primary and secondary behaviour features of Autism Spectrum Disorder. Over the past several decades, cognitive explorations of ASD have

largely focussed on the relationship between ASD symptomatology and its underlying foundation of executive functioning (Ozonoff, 1997; Ozonoff et al., 1991; Russell, 1997). Consequently, the atypicalities in executive functioning commonly observed in ASD have garnered much attention as possible contributing factors in the manifestation of primary ASD symptomatology (Hughes & Russell, 1993; McEvoy, Rogers, & Pennington, 1993; Ozonoff et al., 1991; Prior & Hoffman, 1990; Rumsey & Hamburger, 1988). With respect to the social-communicative characteristics of ASD, domains of executive functioning such as decision-making abilities, the anticipation of consequences, and the general processing of information are utilised in the perception of social-emotional stimuli such as verbal and nonverbal communication (e.g., facial expressions; Hill, 2004; Hill & Bird, 2006; McEvoy et al., 1993; Ozonoff, 1997; Pennington & Ozonoff, 1996; Rajendran & Mitchell, 2007; Russell, Jarrold, & Hood, 1999). Furthermore, the cognitive inflexibility and response perseveration observed in children and adults with ASD has been associated with diminished initiation of non-routine actions, the experience of extreme distress and resistance to trivial or unanticipated changes in the environment, and a level of detail orientation that eclipses the selection or prioritisation of more situation-relevant information needed to guide responses beyond the immediate situation (Frith, 1972; Hermelin & O'Connor, 1970; Hill & Bird, 2006; Hutt & Hutt, 1965; Koegel & Schreibman, 1977; Lovaas, Koegel, & Shreibman, 1979; Lovaas & Schreibman, 1971; Purkis et al., 2016a).

Rigid, stereotypic preferences may also be impacted by diminished response inhibition in ASD (Mosconi et al., 2009). For example, it has been postulated that inhibitory deficits in ASD may originate from the inability to suppress behaviour that provides access to a preferred activity or topic of interest (Hill, 2004; Hughes & Russell, 1993; Russell, 1997). In a study involving 18 individuals with ASD (8-54 years; M_{age} = 17.7 years old; *SD* = 10.5) and 15 neurotypical individuals (8-55 years; M_{age} = 19.9 years old; *SD* = 11.5), repetitive

behaviours were assessed via the Autism Diagnostic Inventory-Revised (ADI-R; Lord, Rutter, & Le Couteur, 1994) and response inhibition was assessed via a series of tasks in which participants were instructed to generate eye movements away from rather than toward novel targets (Mosconi et al., 2009). Results revealed that individuals with ASD were less likely than the neurotypical controls to inhibit prepotent (familiar/habitual) responses to the stimuli (e.g., staring at the novel stimulus), $F(1, 30) = 4.12$, $p = .05$. These findings were in accordance with previous research exploring this form of response inhibition in adults with ASD (Goldberg et al., 2002; Luna, Doll, Hegedus, Minshew, & Sweeney, 2007; Manoach, Lindregren, & Barton, 2004; Minshew, Luna, & Sweeney, 1999; Thakkar et al., 2008). Additionally, results showed that these types of responses were related to higher-order repetitive behaviours ($r = .65$, $p = .01$), but not sensorimotor repetitive behaviours ($r = .11$, $p = .62$). Mosconi et al., (2009) concluded that these findings highlight a distinct pattern of neurocognitive dysfunction associated with behavioural inflexibility in individuals with ASD with respect to preoccupations and compulsions, and, moreover, that these kinds of symptoms are not exclusively the result of intense emotional attachment to objects and activities.

In a study by Lopez, Lincoln, Ozonoff and Lai (2005), 17 adults with ASD (19-42 years old; $M_{age} = 29$) and 17 neurotypical controls (18-45 years old; $M_{age} = 29$) were assessed with respect to working memory (WAIS-III; Wechsler, 1997), cognitive flexibility (TMT in the D-KEFS; Delis et al., 2001; WCST; Heaton & Chelune, 1993), and response inhibition (Stroop task in the D-KEFS; Delis et al., 2001); ASD symptomatology was assessed via the ADI-R (Lord et al., 1994), the ADOS Generic (ADOS-G; Lord et al., 1989) and the Gilliam Autism Rating Scale (GARS; Gilliam, 1995). Preliminary analyses demonstrated that impairments to cognitive flexibility ($r = .63$, $p < .01$) and response inhibition ($r = .54$, $p < .05$) correlated positively with restricted and repetitive behaviours such that as restricted and

repetitive behaviours increased, so too did cognitive inflexibility and disinhibition. Additionally, working memory (r = -.56, p < .05) was observed to have a significant, negative correlation with restrictive repetitive behaviour, such that as the ability to utilise the working memory system decreased, increases in restrictive, repetitive behaviour were observed. Lopez et al. (2005) suggested that these findings provided preliminary support for a relationship between abnormal functioning in the prefrontal cortex and restricted, repetitive symptoms of ASD. A series of multiple regression analyses further exploring the relationship between executive domains and restricted, repetitive symptomatology demonstrated that impairments to cognitive flexibility yielded predictive utility with respect to restrictive repetitive behaviours, t = 2.56, p = .02, β = .71. Results further demonstrated, however, that when impairments to cognitive flexibility were entered into the same regression model with working memory and response inhibition, none of these executive domains predicted restricted and repetitive behaviour. Lopez et al. (2005) concluded that executive domains are highly interrelated and that the entire executive functioning profile needs to be considered when researching its relationship withASD symptomatology.

A review of the literature on the behaviour features of ASD reveals that cognitive atypicalities, such as the executive functioning deficits common to ASD, may also share a relationship with behaviours secondary to primary diagnostic characteristics. More specifically, research on aggression in ASD has provided preliminary evidence of a link between this type of behaviour and executive functioning. However, the research is predominantly on children and adolescents (e.g., Meza, Owens, and Hinshaw; 2016) and is, therefore, in need of further exploration. Aggression in the context of adult ASD will be discussed in the behaviour section of this chapter.

A comparison of executive functioning across Attention-Deficit Hyperactivity and Autism Spectrum Disorders. The profiles of executive functioning in ADHD and ASD point

to a number of differences in cognitive atypicality between the two groups. Researchers exploring executive functioning across ADHD and ASD (e.g., Pennington, 1997) have suggested that a double dissociation exists in the executive atypicalities between these disorders. More specifically, adults with ASD demonstrate impairments in executive domains such as cognitive flexibility and planning (Happé, Booth, Charlton, & Hughes, 2006; Pennington, 1997), whereas adults with ADHD tend to demonstrate distinct difficulties in response inhibition (Sinzig, Morsch, Bruning, Schmidt, & Lehmkuhl, 2008).

In a study directly comparing executive dysfunction in adults with ADHD and ASD, Johnston, Madden, Bramham and Russell (2011) asked 24 adults with ASD (79.17% male; M_{age} = 27.80 years; *SD* = 8.70), 24 adults with ADHD (79.17% male; M_{age} = 27.30 years; *SD* = 9.00), and 14 neurotypical controls (71.43% male; M_{age} = 28.70 years; *SD* = 11.10) to complete the Stroop Colour-Word Test (Golden, 1978) and the Hayling Sentence Completion Test (Burgess & Shallice, 1997). Results revealed that the ADHD group obtained significantly lower levels of accuracy than both the ASD and control groups on the Stroop Colour-Word Task indicating a more impulsive and less accurate approach to task completion. Conversely, those with ASD had greater response latencies but were more accurate, suggesting a deficit in response initiation but not response inhibition (Johnston, Madden, Bramham, & Russell, 2011). Overall, it was concluded that the adults in this study with ADHD demonstrated deficiencies with specific inhibitory tasks, resulting in differentiation between clinical groups with respect to response inhibition (Johnston et al., 2011). These findings support the implication that adults with ADHD experience heightened levels of inhibitory dysfunction; they also provide further support for the idea that adults with ADHD may demonstrate significantly different executive functioning impairments when compared to adults with other neurodevelopmental disorders such as ASD.

Despite the observation that, in a few select studies, ADHD might be more strongly associated with inhibitory problems and ASD with cognitive flexibility and planning deficiencies (Brandimonte, Filippello, Coluccia, Altgassen, & Kliegel, 2011; Bramham et al., 2009; Christ et al., 2007; Happé et al., 2006; Lipszyc & Schachar, 2010; Ozonoff & Jensen, 1999; Verté et al., 2006), this dissociation is yet to be robustly replicated. Moreover, the observation of such executive atypicalities across the lifespan is yet to be reliably, empirically supported for either neurodevelopmental disorder (Booth, Charlton, Hughes, & Happé, 2003; Corbett & Constantine, 2006; Corbett et al., 2009; Johnson et al., 2007; Johnston et al., 2011; Nydén, Gillberg, Hjelmquist, & Heiman, 1999; Nydén et al., 2010; Raymaekers, Antrop, van der Meere, Wiersman, & Roeyers, 2007; Tsuchiya, Oki, Yahara, & Fujieda, 2005; Verté et al., 2006). This paucity of research poses several challenges with respect to understanding potential contributing factors in the heterogeneous presentation of ADHD and ASD in adulthood. In light of the impact executive functioning seems to have on the presentation of primary symptomatology in these disorders, it would be beneficial to further explore to what extent such a connection exists across different symptoms, and the resultant discrepancies in functional impairment and the presentation of other secondary features. Before explorations into functional impairments are conducted, it is important that the similarities and differences in the cognitive features of ADHD and ASD are understood, including the existence of cognitive atypicalities outside of the executive functioning domain (Barkley, 1997; Ozonoff & Jensen, 1999; Pennington, 1997).

Alexithymia. Defined as a specific cognitive trait associated with atypicalities in the processing of emotion-based information, alexithymia is another cognitive feature that has been explored in ASD and, to a lesser extent, ADHD. Thought to exist in approximately 10% of the population (Fukunishi, Wogan, Berger, & Kuboki, 1999; Salminen, Saarijävi†, Äärelä, Toikka, & Kauhanen‡, 1999), alexithymia is most commonly defined by covert

characteristics such as restricted imaginal processes and the inability to identify and describe one's own feelings (Cameron, Ogrodniczuk, & Hadjipavlou, 2014; Nemiah & Sifneos, 1970). Given its covert nature, alexithymia is typically assessed by self-report scales. A review of the research on alexithymia demonstrates that in adults, it is typically assessed by self-report scales such as the 40-item Bermond Vorst Alexithymia Questionnaire (BVAQ; Vorst & Bermond, 2001) and, more commonly, the 20-item Toronto Alexithymia Scale (TAS-20; Bagby, Parkery & Taylor, 1994).

Though little is known about the potential origins and causes of alexithymia (Bird & Cook, 2013), current research on this cognitive trait suggests that it may be a product of developmental dysfunction and diminished connectivity within structures of the limbic system, such as the anterior insula and the anterior cingulate cortex (Etkin, Egner, & Kalisch, 2011; Kano et al., 2003; Lane et al., 1996; Moriguchi et al., 2006; 2007; Singer, Critchley, & Preuschoff, 2009). The atypical internal processes associated with alexithymia can manifest as adverse behaviours (e.g., display of inappropriate facial expression in response to environmental stimuli, or misreading nonverbal cues of emotion). Nevertheless, along similar lines to executive functioning, alexithymia is very much a covert trait associated with the internal processing of information from the environment. Consequently, for the purpose of this study, alexithymia has been defined as comprising difficulties identifying and describing emotions (including the differentiation of bodily sensations from emotions), and a stimulus-bound, externally oriented cognitive style (Luminet, Bagby, Wagner, Taylor, & Parker, 1999).

Alexithymia in Attention-Deficit Hyperactivity Disorder. Preliminary research on alexithymia in ADHD suggests that this trait may be more associated with hyperactive-impulsive symptomatology over that of inattention (Donfrancesco et al., 2013). Moderately elevated levels of alexithymia have been observed in one study on adults with ADHD

(approximately 20%; Edel et al., 2010). Nevertheless, alexithymia is a trait that has otherwise been sparsely investigated in adults with ADHD (Friedman et al., 2003). Consequently, though there is some preliminary evidence to suggest that alexithymia may be elevated in individuals with ADHD across the lifespan and, to a lesser extent, that this concurrent cognitive atypicality may impact symptom presentation in this disorder, further research is required to understand the extent to which alexithymia presents in adults with ADHD.

Alexithymia in Autism Spectrum Disorder. Alexithymia has received considerable research attention in ASD and is thought to exist in 40-65 percent of individuals with this disorder (Berthoz & Hill, 2005; Hill, Berthoz, & Frith, 2004; Silani et al., 2008). Berthoz and Hill (2005) reported levels of alexithymia in adults with and without ASD. This study included two assessment times with the first involving 27 adults with ASD (55.56% male; M_{age} = 35.07 years; *SD* = 12.26) and 35 neurotypical controls (45.71% male; M_{age} = 32.18 years; *SD* = 11.25); the second assessment time involved a further 19 adults with ASD (63.20% male; M_{age} = 38.21 years; *SD* = 12.55) and 29 neurotypical controls (37.30% male; M_{age} = 33.76 years; *SD* = 11.81). Alexithymia was assessed via the TAS-20 (Bagby et al., 1994) and the BVAQ (Vorst & Bermond, 2001). Results demonstrated that ASD groups reported significantly higher levels of alexithymia for both the first and second assessment times. These results concur with Silani et al.'s (2008) findings of significantly higher levels of alexithymia in adults with ASD in comparison to neurotypical controls as assessed by the TAS-20 ($t(27) = 2.80$, $p < .01$), and support the notion of heightened difficulty experienced by adults with this disorder with respect to their ability to identify and describe states of emotion. It should be noted that, in contrast to the research on the other secondary features of ASD (e.g., executive functioning), the majority of the studies on alexithymia involve the assessments of adults. Consequently, the current body of knowledge on alexithymia in ASD

is centred on the later stages of life, as is the information on how it presents in individuals with this disorder.

Alexithymia and the primary behaviour features of Autism Spectrum Disorder. Adults with ASD tend to be limited in their ability to identify and describe the personal emotions that they experience (Berthoz & Hill, 2005; Hill et al., 2004); even those who demonstrate relatively well-developed language can experience difficulty identifying and expressing emotion (Hill et al., 2004; Perry et al., 2001; Rieffe et al., 2007). Additionally, an exploration of emotion detection in ASD demonstrates that atypicalities in this area extend to the processing of emotions in other people. More specifically, research suggests that many individuals with ASD are less able than their neurotypical peers to identify the emotions of others based on the emotional valence communicated in verbal (e.g., tone of voice) and nonverbal cues (e.g., facial expression; Ashwin et al., 2007; Corden, Chilvers, & Skuse, 2008; Hubert et al., 2007; Humphreys, Minshew, Leonard, & Behrmann, 2007; Philip et al., 2010; Wallace, Coleman, & Bailey, 2008). In ASD, these irregularities are commonly affiliated with alexithymia, as are some of the core behavioural features common to ASD.

Recent research has demonstrated relationships between the primary features of ASD and alexithymia as a whole, as well as its key components (e.g., difficulty identifying and describing feelings; Liss, Mailloux, & Erchull, 2008). Although the inability to identify and describe emotional states does not represent a core symptom of ASD, it has been suggested that some of the behaviour responses common to ASD may be related to such emotional irregularities (Geller, 2005). Furthermore, several recent studies have suggested that primary social-communicative features of ASD such as poor emotional interoception (Silani et al., 2008), deficient processing of non-verbal social cues (Bird, Press, & Richardson, 2011), and inappropriate identification of emotions expressed by others (Cook, Brewer, Shah, & Bird,

2013) may be influenced by co-occurring alexithymia.

In an assessment of alexithymic traits in 18 adults with ASD (M_{age} = 57.20; *SD* = 11.80; 37 to 80 years) in comparison to 18 neurotypical controls (M_{age} = 50.30; *SD* = 14.50; 27 to 72 years), the ability to anticipate the emotive state of others was assessed with respect to alexithymia, as well as brain responses as assessed by fMRI (Bird et al., 2010). Across these groups, results indicated that the higher the self-reported degree of alexithymia, the lower the activity in the left anterior insula when observing another person receive painful stimulation. In this sample, group comparisons of anterior insula activity, independent of the degree of alexithymia, demonstrated no significant difference in brain activity due to the presence or absence of ASD. This finding indicates that empathy deficits in developmental disorders such as ASD may be due to commonalities between alexithymic traits and ASD related emotion dysregulation (Bird et al., 2010). In addition to being congruent with other studies demonstrating a link between alexithymia and ASD symptomatology in adults with the disorder (Samson, Huber, & Gross, 2012; Velotti et al., 2012), these findings not only suggest that adults with ASD may experience elevated levels of alexithymia, but that they might also experience a certain level of emotion dysregulation in relation to these alexithymic characteristics. Accordingly, alexithymia has not only been associated with the presentation of ASD core symptoms, but also with secondary behaviour features such as aggression (Fossati et al., 2009; Manninen et al., 2011; Payer, Lieberman, & London, 2011).

Behaviour Feature: Aggression

Although aggression is not included in the diagnostic criteria for ADHD or ASD in the DSM-5 (APA, 2013), it is a prominent feature of adults with these disorders (Cohen et al., 2010; Dowson & Blackwell, 2010). Deliberate, premeditated and even goal oriented acts of aggression are referred to, collectively, as proactive aggression. Conversely, defensive and/or impulsive aggressive responses to a real or perceived threat are considered acts of reactive

aggression (Anderson & Bushman, 1997; Vittielo & Stoff, 1997). Being a behaviour largely defined by observable actions, aggression is typically characterised by its overt features (e.g., the act of hitting another person), but covert aspects of aggression, such as anger (e.g., feeling like you are going to "explode") and hostility (e.g., adverse feelings toward another person) are also sometimes considered to be characteristics of aggression. Aggression can be verbal (e.g., threatening statements, derogatory or cursory assertions) or physical (e.g., biting, hitting, or kicking another person, self-injurous behaviour, property damage), and varies from person to person with respect to frequency, intensity and duration (Fitzpatrick, Srivorakiat, Wink, Pedapati, & Erickson, 2016).

Studies on aggression reveal that, in accordance with its heterogeneous presentation, this behaviour can be assessed via a range of means. These include direct observation from peers, caregivers, and teachers, as well as self-reported documentation of aggressive acts. However, the most common form of assessment observed in the research on this behaviour in adults is through self-report scales, such as the 28-item Buss Perry Aggression Questionnaire (BPAQ; Buss & Perry, 1992). To encompass its complex and heterogenous nature, for the purpose of Studies 1 and 2, aggression has been defined more broadly as a confrontational, hostile, and/or violent form of verbal and/or physical behaviour directed toward another individual, an object, or the self (Buss & Perry, 1992; de Almeida, Cabral, & Narvaes, 2015).

Aggression in Attention-Deficit Hyperactivity Disorder. Although aggression is not included in the diagnostic criteria for ADHD in the DSM-5 (APA, 2013), it is a common reason for initial diagnostic referral (Jensen et al., 2007; King & Waschbusch, 2010; May & Bos, 2000). The research on aggression in ADHD suggests there is a relationship between this type of behaviour and overall ADHD symptom severity and, especially, the symptom class of hyperactivity-impulsivity in ADHD (see Becker et al., 2012; Cabiya-Morales et al., 2007; Connor, Chartier, Preen, & Kaplan, 2010; Connor & Doerfler, 2008; Dodge, Harnish,

Lochman, Bates, & Petit, 1997; Dowson & Blackwell, 2010; Zalecki & Hinshaw, 2004). Given its inherent heterogeneity, aggression does not present the same way in all individuals with ADHD; however, it has been frequently observed in the form of verbal outbursts (e.g., shouting), physical acts (e.g., hitting/kicking others or inanimate objects), transient loss of behavioural control (e.g., tantrums), and self-harm (e.g., cutting; Dowson & Blackwell, 2010; Wender, Wolf, & Wasserstein, 2001). A review of the research also reveals that a reactive, defensive and impulsive aggressive style is far more common than deliberate or planned acts of aggression in ADHD (see review by King & Waschbusch, 2010).

Dowson and Blackwell (2010) assessed aggression in 73 males (18 to 65 years old) diagnosed with ADHD. Results indicated that aggression was significantly correlated with ADHD symptomatology ($p = .010$) as assessed by the Structured Clinical Interview for the DSM-IV-TR (SCID; APA, 2004). Additionally, aggression was significantly correlated with the specific ADHD symptoms of impulsivity ($p = .012$) and hyperactivity ($p = .018$). Although additional research on adults and adolescents with ADHD has demonstrated that heightened levels of aggression persist into the later stages of life in individuals with this disorder (Linder, Crick, & Collins, 2002; Werner & Crick, 1999), few studies have explored aggression in individuals with ADHD well into adulthood, and as such, further investigation is warranted.

Aggression in Autism Spectrum Disorder. Adults with ASD reportedly experience feelings of aggression toward others, inanimate objects, and themselves more regularly than their neurotypical peers (Cohen et al., 2010; Sotullo, 2010). Research on aggression and ASD symptomatology has demonstrated that a relationship likely exists between this secondary feature and the disorder's core characteristics. In a non-clinical sample of 618 college students (33.50% male; aged 17 to 22 years), ASD symptomatology was found to be significantly related to overall aggression as measured by the BPAQ (Buss & Perry, 1992; $r =$

.34, $p < .001$), as well as to subscales assessing physical aggression ($r = .15$, $p < .001$), verbal aggression ($r = .21$, $p < .001$), anger ($r = .31$, $p < .001$), and hostility ($r = .41$, $p < .001$; White, Kreiser, Pugliese, & Scarpa, 2012). Further analyses demonstrated that ASD symptomatology accounted for a significant amount of variance in hostility and overall aggression. These findings were in accordance with studies assessing the relationship between aggression and ASD symptomatology in other adult, non-clinical samples (Pugliese et al., 2015). White, Kreiser, Pugliese, and Scarpa (2012) suggested that these results highlighted the importance of exploring ASD dimensionally as opposed to categorically when investigating the role of secondary features, such as aggression, in adults with this disorder. It is important to note, however, that these select studies were conducted on non-clinical samples of adults. Accordingly, aggression has not been well characterised in adults with ASD (e.g., Farmer & Aman, 2011), and, consequently, further investigation of this secondary feature in adults with ASD is warranted.

Emotion and Mood Features

The poor emotional awareness, diminished ability to express emotions, and general inability to tone down emotional arousal often observed in ADHD and ASD decreases the likelihood that the adverse emotions experienced by adults with these disorders will be processed and dealt with adequately (Nemiah & Sifneos, 1970; Taylor, Bagby, & Parker, 1997). Individuals with ADHD and ASD are likely to experience heightened levels of adverse emotion, including anxiety and depression, at some point in their lives. Although anxiety and depression in the context of comorbid diagnoses were addressed in Chapters 2 and 3, the remainder of this section will discuss the co-occurrence of the underlying *emotional states* of anxiety and depression as they present in adults with ADHD and ASD; operational definitions of each emotion are provided in the sections to follow.

Anxiety. Anxiety is characterised by an unpleasant state of inner turmoil that is often in response to or in anticipation of a perceived threat (Pliszka, 2009). Covert in nature, anxiety is typically assessed in adults by self-report scales, such as the 20-item State-Trait Anxiety Inventory (STAI: Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), the 14-item Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983), the 24-item Liebowitz Social Anxiety Scale Self-Report (LSAS-SR; Liebowitz, 1987), the 21-item Beck Anxiety Inventory (BAI; Beck, Epstein, & Brown, 1988) and the 42-item Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995). The unpleasant feelings of anxiety are often accompanied by other covert characteristics such as excessive worry and adverse somatic experiences (e.g., dizziness, increased heart rate; Seligman, Walker, & Rosenhan, 2001). These characteristics vary in intensity, frequency, and duration, and can be brief and otherwise manageable, or more recurring and enduring (Rynn & Brawman-Mintzer, 2004). For for the purpose of Studies 1 and 2, the secondary feature of anxiety is explored with respect to these covert characteristics (e.g., excessive worry).

Anxiety in Attention-Deficit Hyperactivity Disorder. Approximately half of all adults with ADHD are expected to experience elevated levels of anxiety at some point in their lives (Kessler et al., 2006). Research on anxiety in ADHD suggests that clinical levels of this secondary feature were present in 16-43% in adults with this disorder (Barkley et al., 1996; 2008; Biederman et al., 1993; Duran et al., 2013; Minde et al., 2003; Shekim et al., 1990; Yoshimasu et al., 2016). However, several longitudinal follow-up studies on adults diagnosed with ADHD in childhood have failed to demonstrate the existence of heightened anxiety across the lifespan (Fischer et al., 2002; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Rasmussen & Gillberg, 2000).

Research on anxiety in adults with ADHD further points to a relationship between heightened levels of this emotion and primary ADHD symptomatology. A sample of adults

with ADHD (N = 231; 60-94 years old; M_{age} = 71.0; SD = 7.70; 59% female) were interviewed about their ADHD symptomatology and assessed with respect to their levels of anxiety through the HADS-A (Zigmond & Snaith, 1983). Results yielded a significant, positive relationship between ADHD symptomatology and levels of anxiety (Michielsen et al., 2013). These findings support a relationship between the primary features of ADHD and anxiety, and are in accordance with research supporting relationships of anxiety with hyperactive-impulsive (Schatz & Rostain, 2006), inattentive (Schatz & Rostain, 2006), and overall symptomatology (Barkley et al., 2008; Miller, Migg, & Faraone, 2007) in adults with this disorder.

Anxiety in Autism Spectrum Disorder. Approximately, 30-50% of adults with ASD are thought to experience clinical levels anxiety (Mazefsky et al., 2008; Lugnegård et al., 2011; Rumsey, 1985). Moreover, comparative studies in adults with ASD have revealed elevated levels of anxiety in comparison to neurotypical controls. In an assessment of anxiety (as assessed by the LSAS-SR; Liebowitz, 1985) in adults with (n = 50; M_{age} = 30.0; 20-47 years old) and without ASD (n = 53; M_{age} = 32.3; 28-32 years old), significantly higher scores on anxiety were reported by the adults with ASD relative to those in the non-ASD comparison group ($p < .001$; Bejerot et al., 2014). These findings were in accordance with other studies demonstrating that ASD adults experience heightened levels of anxiety in comparison to neurotypical adults (see review by van Steensel et al., 2011; also see Gillott & Standen, 2007) and, overall, support the notion that heightened levels of anxiety persist into adulthood in ASD.

The covert characteristics of anxiety in individuals with ASD are typically similar to those of the general population (e.g., worrisome thoughts, adverse somatic experiences; Ghaziuddin & Greden, 1998; Kim et al., 2000); however, similar to ADHD, these anxious thoughts and feelings often result in exacerbation of ASD symptoms manifested as overt

behaviours. Heightened levels of anxiety in adults with ASD have been associated with restricted and repetitive behaviour (Rodgers, Glod, Connolly, & McConachie, 2012; Sukhodolsky et al., 2008; Tantam, 2003). Some researchers have suggested that such symptomatology (particularly with respect to circumscribed interests) may serve as a means to reduce anxiety by engaging in a preferred activity (Joosten, Bundy, & Einfeld, 2009; Ooi et al. 2008; Spiker, Lin, Van Dyke, & Wood, 2011). Conversely, it has been suggested that anxiety in ASD may actually result from certain restricted and repetitive behaviours (e.g., repetitive worried thoughts; Kim et al., 2000; Rodgers et al., 2012; Sofronoff, Attwood, & Hinton, 2005). Thus there is likely an association between anxiety and repetitive and restrictive symptomatology in ASD, however the exact nature of the relationship is unclear (Factor, Condy, Farley, & Scarpa, 2016).

Depression. Depression is characterised by a low and despondent mood, markedly diminished interest or pleasure in enjoyable activities, and excessive feelings of worthlessness or guilt (Pillemar, Suitor, Pardo, & Henderson, 2010; Wilkinson, 2015). Depression may be associated with feelings of helplessness, hopelessness, dejection, and even anger or irritability. Covert in nature, depression in adults is usually assessed via self-report scales, such as the DASS (Lovibond & Lovibond, 1995), the 20-item self-report Centre for Epidemiologic Studies Depressive Scale (CES-D; Radloff, 1977), the HADS (Zigmond & Snaith, 1983), and the 21-item Beck Depression Inventory (BDI; Beck, Steer & Brown, 1996). Similar to anxiety, these covert features can vary in intensity, frequency, and duration, presenting only in the short-term for some, but persisting across time and different settings in others (APA, 2013). For the purpose of Studies 1 and 2, depression has been explored and defined in the context of these covert features (e.g., despondent feelings).

Depression in Attention-Deficit Hyperactivity Disorder. Research suggests that 36-71% of individuals with ADHD will experience clinical levels of depression at some point in

their lives (Barkley et al., 2008; Torgersen, Gjervan, Polit, & Rasmussen, 2006). Longitudinal studies, however, show contradictory results with respect to the persistence of depression across the lifespan in individuals with ADHD, with some studies finding levels of depression in adulthood similar to what was present in childhood (Fischer et al., 2002; Biederman et al., 1996; 2008), and others failing to do so (Mannuzza et al., 1998; Rasmussen & Gillberg, 2000). Additionally, although some studies have suggested that levels of depression in adults with ADHD are significantly higher than in neurotypical adults (Biederman et al., 2008), others have failed to find such discrepancies (Hinshaw et al., 2006). Nevertheless, research suggests that up to half of clinically referred adults with ADHD reportedly experience concurrent dysthymia (depressed mood) and, to a lesser extent, major feelings of depression and despondency (Barkley et al., 1996; Biederman et al., 1993; Kessler et al., 2006; Kooji, 2012; Murphy & Barkley, 1996; Murphy, Barkley & Bush, 2002; Roy-Byrne et al., 1997; Shekim et al., 1990; Sobanski, 2006).

Symptoms of depression tend to magnify the features of ADHD, making it even more difficult for adults with this disorder to sustain concentration on tasks and initiate behaviours (Barkley, 2015c). More specifically, in adults with ADHD, depression often results in increased emotional lability and diminished effortful control over emotion and behaviour and thus has been associated with the disorder's primary symptomatology **(**Chaplin, Cole, & Zahn-Waxler, 2005; Compas, Connor-Smith, & Jaser, 2004; Silk, Steinberg, & Morris, 2003). Michielsen et al. (2013) interviewed 231 adults with ADHD (60-94 years old; M_{age} = 71.0; *SD* = 7.70; 59% female) about their ADHD symptomatology and assessed their levels of depression as per the CES-D (Radloff, 1977). Results revealed a significant, positive relationship between ADHD symptomatology and levels of depression (Michielsen et al., 2013). These findings demonstrated that a relationship between the primary features of ADHD and depression can persist into adulthood and is in accordance with research

supporting such a relationship between overall symptomatology and heightened levels of depression in adults with this disorder (e.g., Biederman et al., 1996).

Depression in Autism Spectrum Disorder. Over half of individuals with ASD are likely to experience clinical levels of depression at some point in their lives (Lugnegård et al., 2011; Mazefsky et al., 2008). In a study by Hill, Berthoz, and Frith (2004), levels of depression were assessed via the BDI (Beck, et al., 1988) in 27 adolescents and adults diagnosed with ASD (88.24% male; aged 16 to 63 years) and 35 typically developing participants (45.71% male; aged 19 to 62 years). ASD participants reported experiencing significantly higher levels of depression in comparison to control subjects ($p < .001$). These results reveal the potential for elevated levels of depression to persist into adulthood in ASD, and for these levels to exceed that of neurotypical adults.

In ASD, heightened levels of depression have been linked to thoughts of personal failure (Schniering & Rapee, 2004) and internal, stable causes (e.g., not being "good enough"; Barnhill & Myles, 2001), and manifest as the presence of a low mood that is noticeably different from that which is normally experienced (Folstein & Carcache, 2016). Research has further suggested a bidirectional relationship between autistic-like symptoms and internalising traits such as symptoms of depression that may exist in ASD. More specifically, in addition to social-communicative and repetitive and restrictive symptomatology being associated with elevated symptoms of depression (Kanne, Abbacchi, & Constantino, 2009; Mayes, Calhoun, Murray, & Zahid, 2011), heightened levels of depression in ASD have been associated with increased compulsive behaviour (Ghaziuddin, 2005), decreased self-care (Clarke et al., 1989; Wing, 1981a), psychomotor retardation (Ghaziuddin & Tsai, 1991), heightened nonverbal communication issues (e.g., monotonous tone of voice, poor eye contact; Ghaziuddin & Zafar, 2008; Rodgers et al., 2012) increased social withdrawal, and alterations in the character of obsessions, with fixations taking on a

more morbid tone (Ghaziuddin, 2005). This is problematic because, although covert states (e.g., adverse thoughts and feelings) mark the key characteristics of this secondary feature, it is the overt behaviours (e.g., social-communicative atypicalities) that result from these internal states that compound the functional impairments experienced by individuals with ASD (Ghaziuddin et al., 2002).

Functional Impairments Associated with the Secondary Features of Attention-Deficit Hyperactivity and Autism Spectrum Disorders

Understanding the ways in which secondary features present in ADHD and ASD and their potential impact on the primary features of these disorders is important, as secondary cognitive, behaviour, and emotion-based atypicalities have been associated with heightened impairments in functionality in both disorders. In the case of executive functioning, longitudinal research suggests that impairments to academic (e.g., lower GPAs, higher levels of grade retention), social (e.g., peer interaction), and occupational functioning (e.g., meeting deadlines, completing projects, engaging in self-motivation) in adults with ADHD (Barkley & Murphy, 2010; Clark, Prior, & Kinsella, 2002; Langberg et al., 2008; 2011; Massetti et al., 2008; Miller, Nevado-Montenegro, & Hinshaw, 2012) and ASD (Hughes, 1996; Lopez et al., 2005; Robinson, Goddard, Dritschel, Wisley, & Howlin, 2009) are more severe in those with deficiencies in executive functioning. Additionally, heightened impairment in social (e.g., peer interaction) and academic functioning (e.g., successful completion of group activities) has been observed in ASD in the context of co-occurring alexithymia (Jahromi, Bryce, & Swanson, 2013).

With respect to secondary behaviour features, research has further demonstrated that the combined presence of ADHD symptomatology and aggression has a stronger impact on peer rejection than ADHD symptomatology alone (Bagwell et al., 2001; Millich & Landau, 1982). In ASD, aggression is a major concern for a number of reasons, not the least of which

involves the potential for physical harm (Kanne & Mazurek, 2011). Heightened levels of aggression in ASD have also been associated with diminished rehabilitative and treatment success (McCracken et al., 2002). Moreover, a review of the functional impacts of aggression on both neurodevelopmental disorders demonstrates that this type of behaviour is one of the strongest predictors of crisis intervention re-referrals (Shoham-Vardi et al. 1996), admission and readmission to hospitals and residential facilities (Benson & Fuchs, 1999; Lakin, Hill, Hauber, Bruininks, & Heal, 1983), the prescription of psychotropic medication (Tsakanikos, Costello, Holt, Sturmey, & Bouras, 2007), and overall quality of life in individuals with ADHD and ASD (Gardner & Moffatt, 1990).

Research on secondary emotion features of ADHD and ASD suggests that anxiety and depression also appear to be particularly prominent with respect to their impact on functionality across the lifespan in individuals with these disorders. In ADHD, heightened levels of anxiety have been associated with poor occupational outcomes such as higher levels of unemployment, greater reliance on social welfare, and a higher instance of criminality and antisocial behaviour (Halmøy, Fasmer, Gillberg, & Haavik, 2009), as well as greater risk of substance abuse and dependence (Biederman et al., 1995; Wilens, Biederman, Mick, 1998). Elevated anxiety in ASD has been associated with disruptions of day-to-day functionality, heightened arousability, loss of engagement in social activities, and an overall diminished quality of life (see review by van Steensel, Bögels, & Perrin, 2011; also see Bejerot, Eriksson & Mörtberg, 2014; Lugnegård et al.,; Mattila et al., 2010; South, Newton, & Chamberlain, 2012; van Steensel, Bögels, & Dirksen, 2012; Clarke, Littlejohns, Corbett, & Joseph, 1989; Lainhart & Folstein, 1994; Perry, Marston, Hinder, & Munden., 2001; Stewart, Barnard, Pearson, Hasan, & O'Brien, 2006). In the context of depression, higher levels of this secondary feature in ADHD have been associated with greater impairments in social (e.g., family conflict) and academic functioning (e.g., sustained attention in class; Angold,

Costello, & Erkanli, 1999; Antshel & Remer, 2003; Bagwell et al., 2001; Biederman, Newcorn, & Sprich, 1991; Biederman et al., 1993; 1996; Blackman, Ostrander, & Herman, 2005; Eiraldi, Power, Karusitis, & Goldstein, 2000; Greene et al., 1996), as well as more negative thoughts and emotions, and heightened rates of suicide (Biederman et al., 1991; Lewinsohn, Rohde, Seeley, & Fischer, 1993). Similarly, impairments to communicative and adaptive functioning are often much worse in individuals with concurrent ASD and depression in comparison to those with ASD alone (Mattila et al., 2010; Mazurek, 2014).

Directions for Future Research on the Secondary Features of Attention-Deficit Hyperactivity and Autism Spectrum Disorders

Research on the secondary features of ADHD and ASD suggests that there are common features outside of the primary diagnostic characteristics that carry the potential to persist across the lifespan and further exacerbate the functional impairments typically associated with these disorders (Keane, 2004; Myles, 2003). As is the case for the primary impairments required for diagnosis, none of the secondary features common to ADHD and ASD appear to be universal across all adults with these disorders (Rodgers et al., 2012). Variations in the extent to which each secondary feature has been documented in both ADHD and ASD are likely heavily influenced by methodological discrepancies in the current body of research (e.g., sample sources, sample sizes, assessment methods; Hoza, Pelham, Milich, Pillow, & McBride, 1993; Kerns & Kendall, 2012; Milberger et al., 1996; van Steensel et al., 2011; White, Oswald, Ollendick, & Scahill, 2009). Nevertheless, such differences are also potentially linked to current gaps in the research and the need for certain aspects of previous studies to be re-visited in further detail. A review of the research on the secondary features common to ADHD and ASD conducted for Study 1 has identified three gaps: 1) a paucity of research on samples of adults with ADHD and ASD, with the majority of research on these disorders conducted on children and, to a lesser extent, adolescents; 2) a paucity of studies on

community-based samples leading to an over-representation of findings obtained from clinical populations; and 3) a paucity of research investigating ADHD and ASD together, with most studies conducted solely on ADHD and/or ASD. These gaps are reviewed in more detail in the context of research on individuals with ADHD and ASD throughout the remainder of this chapter.

Research Gap 1: Child and adolescent versus adult samples in the assessment of the secondary features of Attention-Deficit Hyperactivity and Autism Spectrum Disorders. Although there is some research on the presence of secondary features in adults with ADHD and ASD (e.g., Barkley, 1997; 2012; Dowson & Blackwell, 2010; Michielsen et al., 2013; Wallace et al., 2016), the vast majority of studies exploring such aspects of these disorders are focussed on children and adolescents (e.g., Dominick et al., 2007; May et al., 2012). This is problematic because one of the biggest concerns associated with the secondary features of these disorders is their potential to perpetuate across the lifespan, as such features left unaddressed early in life have been observed to contribute to significant functional deficits (e.g., social, academic, occupational) (Halperin et al., 1994; Tantam, 2003). Moreover, the developmental trajectory of certain secondary features common to ADHD and ASD (e.g., executive functioning) over the first few decades of life is likely to be impacted by the atypical development of prefrontal structures in the brains of individuals with these disorders, resulting in functionality that is subject to fluctuation depending on what the developmental expectations are at each stage of life (Bunge, Dudukovic, Thomason, Vaidya, & Gabriel, 2002; Durston et al., 2003; Lenroot et al., 2007). For example, cognitive flexibility may appear intact in pre-school aged children with ASD, however, when assessed several years later, they may appear markedly more inflexible than neurotypical peers, as a result of the increased environmental demands put on them and insufficient personal resources (Ozonoff et al., 2004). Not only is the range of atypicalities in these disorders'

secondary features likely to be heterogeneous, but this variation is likely to be impacted by age and developmental stage, resulting in changes to presentation of specific impairments/difficulties across the lifespan, and contributing to the current uncertainty of what these atypicalities look like later in life (Nigg et al., 2006; Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003; Sjowall, Roth, Lindqvist, & Torell, 2013; Sonuga-Barke, 2003). Consequently, there is a need to further investigate the presentation of these features outside the context of the early developmental years.

Research Gap 2: Clinical versus community-based samples in the assessment of secondary features of Attention-Deficit Hyperactivity and Autism Spectrum Disorders. A review of the research on the secondary features common to ADHD and ASD suggests that, with the exception of alexithymia (e.g., Berthoz & Hill, 2005; Hill et al., 2004), the vast majority of studies are based on clinical samples. More specifically, as opposed to recruiting study participants from the broader community, assessments of executive functioning, anxiety, depression, and aggression in ADHD and ASD have largely utilised individuals accessing clinics for diagnostic purposes or for treatment and support with the primary symptomatology and related impairments to functionality common to these disorders (e.g., Biederman et al., 1991; 1992; 2008; Chronis-Tuscano et al., 2010; Pennington & Ozonoff, 1996). Clinical samples are often comprised of individuals experiencing heightened levels of symptomatology and impairments to functioning, meaning their primary and secondary pathologies are often greater than what is experienced by the general population (Anderson, Williams, McGee, & Silva, 1987; Costello et al., 1988; McGee et al., 1990). Accordingly, only through a sample of the general community can a true base rate of a disorder and its potential secondary features be established (Caron & Rutter, 1991).

This gap in the research also appears to be associated with a unique issue relating to the conceptualisation of anxiety and depression in ADHD and ASD in particular, in that a

large portion of this clinical research has been heavily focussed on a more dichotomous (diagnosable vs. not diagnosable) exploration of these features, as opposed to that of the features' *level* (e.g., frequency or intensity of underlying emotional states; e.g., Meinzer, Pettit, & Viswesvarab, 2014; van Steensel et al., 2011). This is problematic because it can lead to under-identification individuals who experience heightened levels of anxiety and depression that, while subclinical, still carry the potential to compound impairments to their functionality (Judd, Paulus, Wells, & Rapaport, 1996; Judd et al., 1998). As such, it would be beneficial to consider a continuous spectrum of anxious and depressive characteristics in future research.

Research Gap 3: Direct comparisons of secondary features and how they present in Attention-Deficit Hyperactivity and Autism Spectrum Disorders. Research exploring the secondary features of ADHD and ASD has largely been conducted in isolation (Davis & Kollins, 2012; Rommelse et al., 2010; 2017). Although some studies have directly compared certain secondary features common to these disorders (e.g., executive functioning; Geurts, Verté, Oosterlaan, Roeyers, & Sergeant, 2004; Ozonoff & Jensen, 1999), the majority have only been conducted on groups of adults with either ADHD or ASD. Following a review of the overlap between the features of ADHD and ASD and what is known about the application of a cross-diagnostic approach to assessment and treatment (e.g., see review of transdiagnostic exploration in Chapter 2), it is likely that further review of the potential overlap of different aspects of ADHD and ASD could provide a broader understanding of how these disorders present across different individuals and, moreover, potentially inform diagnosis and treatment with respect to the formulation of a unifying theory of underlying psychopathology. As such, it would be helpful to examine ADHD and ASD togehter, in an attempt to further explore potential points of convergence and divergence that may broaden the clinical understanding of these disorders.

Study One. Research on the secondary features of ASD and ADHD, though limited, does provide some evidence that heightened levels of executive dysfunction (Rumsey, 1985; Rumsey & Hamburger, 1988; 1990; Schecklmann et al. 2012; Young, 2005; Wallace et al., 2016), anxiety (Biederman et al., 1993; Gillott & Standen, 2007; Kessler et al., 2006), depression (Mazefsky et al., 2008; Sobanski, 2006), and aggression (May & Bos, 2000), are common in adults with these disorders. This research further reveals unique atypicalities in the secondary features of these disorders in comparison to neurotypical adults and adults with other neurodevelopmental disorders. In the case of ADHD, response inhibition has relatively consistently been found to be higher in adults with this disorder in comparison to neurotypical controls (Barkley et al., 2010; 2011; Kessler et al., 2006; Murphy & Barkley, 1996; Schecklmann et al., 2012) and adults with ASD (see Johnston et al., 2011; Ozonoff & Jensen, 1999). Additionally, with respect to ASD, levels of alexithymia appear to be significantly higher in adults with this disorder in comparison to neurotypical adults (Berthoz & Hill, 2005; Hill et al., 2004); though no direct comparison studies have been conducted between ADHD and ASD to date, it is important to note that markedly higher levels of alexithymia have been reported in adults with ASD in comparison to those with ADHD (Edel et al., 2010). Consequently, the aim of Study 1 was to examine whether a community-based sample of adults with ADHD and ASD experience heightened levels of atypical cognition (i.e., executive dysfunction, disinhibition, alexithymia), behaviour (i.e., aggression), and emotion (i.e., anxiety, depression), and if so, if these heightened levels exceeded that of neurotypical adults or, where relevant, those with other neurodevelopmental disorders. Based on the existing findings relating to secondary features common to ADHD and ASD, it was hypothesised that:

1. Adults in the ADHD group would report significantly higher levels of executive dysfunction than adults in the neurotypical control group.

2. Adults in the ASD group would report significantly higher levels of executive dysfunction than adults in the neurotypical control group.
3. Adults in the ADHD group would report significantly higher levels of disinhibition than adults in the neurotypical control group.
4. Adults in the ADHD group would report significantly higher levels of disinhibition than adults in the ASD group.
5. Adults in the ASD group would report significantly higher levels of alexithymia than adults in the neurotypical control group.
6. Adults in the ASD group would report significantly higher levels of alexithymia than adults in the ADHD group.
7. Adults in the ADHD group would report significantly higher levels of aggression than adults in the neurotypical control group.
8. Adults in the ASD group would report significantly higher levels of aggression than adults in the neurotypical control group.
9. Adults in the ADHD group would report significantly higher levels of anxiety than adults in the neurotypical control group.
10. Adults in the ASD group would report significantly higher levels of anxiety than adults in the neurotypical control group.
11. Adults in the ADHD group would report significantly higher levels of depression than adults in the neurotypical control group.
12. Adults in the ASD group would report significantly higher levels of depression than adults in the neurotypical control group.

In order to test these hypotheses, study participants were asked to complete a series of self-report measures assessing the primary and secondary features of ADHD and ASD.

Results of these assessments are provided in the chapter to follow, with a more in-depth discussion of the findings presented in Chapter 11.

Chapter Five: Study 1- Comparative Analyses of the Secondary Features of Attention-Deficit Hyperactivity and Autism Spectrum Disorders

Study 1 aimed to conduct an exploratory comparison, in relation to intensity, of six secondary features (i.e., executive dysfunction, disinhibition, alexithymia, anxiety, depression, and aggression), which are suggested to impact on presentation of core features in ADHD and ASD. In order to extend the existing research on the secondary features of ADHD and ASD, which has focused primarily on childhood/adolescence, Study 1 not only explored the presence/intensity of secondary features in adults participants with ADHD/ASD, but also whether the experiences of secondary features in those participants varied to those of neurotypical adults (Kinser & Robins, 2013). As such, in addition to recruiting participants from the community with a singular diagnosis of ADHD or ASD, a group of neurotypical adults was also drawn from the community for comparative purposes. Group membership (e.g., ADHD vs. ASD) was validated via two, self-report screening measures. Participants were required to complete four additional self-report measures assessing the various secondary features focussed on in Study 1. This study was approved by the Bond University Human Research Ethics Committee (BUHREC; protocol number RO1432) in February 2012. Participant recruitment and subsequent data collection took place between December 2012 and September 2017.

Participants

Study 1 participants belonged to one of three groups: ADHD (n = 96), ASD (n = 90), and a neurotypical control group with no diagnosis of mental disorder (n = 92). Formal diagnostic criteria for identification of ADHD and ASD in adulthood first appeared in the fifth edition of the DSM (APA, 2013) possibly contributing to low levels of formal diagnoses in adults who exhibited impairment profiles indicative of ADHD or ASD. For this reason, this study sought to recruit reasonably homogenous ADHD and ASD samples via application

to two inclusion criteria: 1) self-report of formal diagnosis; or 2) self-report of interactions with mental health professionals, such as a psychologist or psychiatrist, for psychotherapeutic purposes, during the course of these interactions, the possibility of having ASD and/or ADHD was seriously discussed.

A total of 278 adults participated in Study 1. The age range was 18 to 73 years (M_{age} = 32.58 years; *SD* = 13.49), with 107 (38.5%) males and 169 (60.8%) females (2 participants in the ASD group did not disclose their gender). Of the 90 participants in the ASD group, 48 (53.33%) were male and 40 (44.44%) were female, with an age range of 18 to 73 years (M_{age} = 34.64 years; *SD* = 13.73). In the ADHD group, 41 (42.71%) of the participants were male and 55 (57.29%) were female, with an age range of 18 to 67 years (M_{age} = 37.30 years; *SD* = 12.69). The 84 participants in the control group were comprised of 18 (19.67%) males and 74 (80.43%) females, with an age range of 18 to 69 years (M_{age} = 25.66 years; *SD* = 11.19). Further participant demographic information is provided in Table 1.

Description of Standardised Measures

The materials for Study 1 included two self-report scales to validate group assignment for ADHD (ADHD Self-Report Scale; Adler, Kessler, & Spencer, 2003) and ASD (Autism Spectrum Quotient; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). Secondary features were assessed via administration of four self-report scales: the BPAQ (Buss & Perry, 1992) for aggression, the DASS (Lovibond & Lovibond, 1995) for anxiety and depression, the FrSBe (Grace & Malloy, 2001) for executive dysfunction and disinhibition, and the TAS-20 (Bagby et al., 1994) for alexithymia. In addition to the fact that the self-report nature of these measures allowed for a broader means of engagement with this study (as opposed to using measures that required participants to attend an in-person assessment), they were selected for use in the study based on a number of important, empirically supported factors. First, following a

Table 1

Participant Demographic Information (N = 278)

	Nationality *n* (%)					Employment *n* (%)			Education *n* (%)			
Group	Australia	North America	Europe	Asia	Other	Paid	NPE	Student	Prim.	Sec.	TAFE/ Trainee	Tert.
ADHD	27	55	6	1	7	57	17	22	2	24	10	60
	(28.13)	(57.29)	(6.25)	(1.04)	(7.29)	(59.38)	(17.71)	(22.92)	(2.08)	(25.00)	(10.41)	(62.50)
ASD	59	10	14	1	6	41	30	19	4	26	19	40
	(65.56)	(11.11)	(15.56)	(1.11)	(6.67)	(45.56)	(33.33)	(21.11)	(4.44)	(28.89)	(21.11)	(44.44)
Control	52	15	13	5	7	16	4	78	2	38	11	41
	(56.52)	(16.30)	(14.13)	(5.43)	(7.61)	(17.39)	(4.34)	(78.26)	(2.17)	(41.30)	(11.96)	(44.56)

Note. “Other” nationalities = countries from the Middle East and Africa, and those who listed multiple nationalities. NPE = no paid employment/unemployed. Prim. = primary education. Sec. = secondary education. Tert. = tertiary education. TAFE = Technical and Further Education. ADHD = Attention-Deficit Hyperactivity Disorder. ASD = Autism Spectrum Disorder.

review of the current literature, the nature of the items and the overall focus of aggregate scores across these measures provided an accurate representation of the study's operational definitions formulated for each secondary feature. Additionally, based on assessments of these measures in empirical studies and in the context of this study, these measures are all appropriate with respect to their applicability (e.g., reliability, validity). These points will be addressed in more detail in the discussions of the individual measures to follow.

Adult ADHD Self Report Scale. The Adult ADHD Self Report Scale (ASRS-v1.1; Adler et al., 2003) is an 18-item self-report measure based on the diagnostic criteria for ADHD in the DSM-IV-TR. The ASRS-v1.1 assesses both inattention and hyperactivity-impulsivity on a 5-point Likert scale, including 0 (*never*), 1 (*rarely*), 2 (*sometimes*), 3 (*often*) and 4 (*very often*). Individuals are asked to answer items with reference to the last six months. There are 9 items (items: 1, 2, 3, 4, 7, 8, 9, 10, 11) assessing inattention; which include statements such as "How often are you distracted by noise or activity around you?" and "How often do you have problems remembering appointments or obligations?" There are 9 items (items: 5, 6, 12, 13, 14, 15, 16, 17, 18) assessing hyperactivity-impulsivity; these items include statements such as "How often do you feel overly active and compelled to do things, as if driven by a motor?" and "How often do you interrupt others when they are busy?" The ASRS-v1.1 takes approximately 10 minutes to complete. Both the inattentive and hyperactive-impulsive items are presented throughout the measure (i.e., the items from each subscale are not all presented together in sequence). All items are positively phrased meaning they all directly assess the extent to which an individual dsiplays symptoms of inattention and/or hyperactivity-impulsivity. Accordingly the scores of each item can be taken at face value, without the need for them to be adjusted in any capacity (e.g., reversed).

There are no cut-off points with respect to different levels of ADHD symptom frequency based on the aggregate scores of this measure, however, the first 6 items of the

ASRS-v1.1 can be used to determine whether or not a diagnosis of ADHD is likely. More specifically, based on empirical exploration of the diagnostic properites of this measure's items, the first 6 items yield the most predictive utility with respect to diagnosis and, therefore, scoring within the clinically high range (selecting *sometimes* to *very often* on at least 4 of the first 6 items) is highly indicative of an ADHD diagnosis (Adler et al., 2003). Because this study aimed to collect data that extended beyond basic categorisation of symptom presentation, a dichotomous index (i.e., clinical vs. subclinical) of ADHD symptom presentation was not used in any of the study's analyses. The overall ASRS-v1.1 score can, however, be used as a *continuous* index of ADHD symptom severity in an adult population, with higher scores representing a greater severity of symptom presentation. Thus, for the purpose of this research, this score was used for the analyses.

In addition to the continuous nature of the data yielded from this measure, empirical explorations of its utility demonstrate that it is reliably applicable for use in screening of ADHD in adults. In a study on 668 community-based participants, both internal consistency (r = .63 to .72) and test-retest reliability (r = .58 to .77) were acceptable (Kessler et al., 2007). Additionally, in a clinical study on 60 adults (M_{age} = 37.5; SD = 10.3; 68% male), internal consistency of overall scores on the ASRSv1.1 was found to be high (r = .88 to .r = .89; Adler et al., 2007). A strong concordance rate with clinical diagnoses of ADHD (r = .90) has also been found (Kessler et al., 2007). In the current study, the Cronbach's alpha for the 18-item ASRS-v1.1 was .84, indicating good internal consistency.

Autism Spectrum Quotient. The Autism Spectrum Quotient (AQ; Baron-Cohen et al., 2001) is a 50-item self-report measure that assesses five different aspects of ASD: social skill, attention switching, communication, attention to detail, and imagination. Social skill contains items 1, 11, 13, 15, 22, 36, 44, 45, 47 and 48, and uses phrases such as "I prefer to do things with others rather than on my own" and "I would rather go to a library than a

party." Attention switching contains items 2, 4, 10, 16, 25, 32, 34, 37, 43 and 46, and uses phrases such as "I prefer to do things the same way over and over again" and "I tend to have very strong interests, which I get upset about if I can't pursue." Communication contains items 7, 17, 18, 26, 27, 31, 33, 35, 38, and 39, and uses phrases such as "When I talk on the phone I am not sure when it is my turn to speak" and "I frequently find that I don't know how to keep a conversation going." Attention to detail contains items 5, 6, 9, 12, 19, 23, 28, 29, 30 and 49, and uses phrases such as "I often notice small sounds when others do not" and "I notice patterns in things all the time." Finally, imagination contains items 3, 8, 14, 20, 21, 24, 40, 41, 42 and 50, and uses phrases such as "I find making up stories easy" and "I don't particularly enjoy reading fiction." The items are rated on a 4-point Likert scale including 1 (*definitely agree*), 2 (*slightly agree*), 3 (*slightly disagree*), and 4 (*definitely disagree*). The test takes approximately 15 minutes to complete. Items 1, 3, 8, 10, 11, 14, 15, 17, 24, 25, 27, 28, 29, 30, 31, 32, 34, 36, 37, 38, 40, 44, 47, 48, 49 and 50 are negatively phrased, meaning that higher scores on them actually represent a lower level of the attribute being measured. These items are included in assessment measures to reduce the chances of response sets appearing in a participant's data (i.e., someone simply selecting the same number for all items; Yamaguchi, 1997). The remainder of the scores are positively phrased and, therefore, can be taken at face value. A score of 32 or greater is indicative of significant autistic symptomatology. For the purpose of this study, only the overall AQ total was utilised as a *continuous* index for ASD symptomatology

Test-retest reliability of the AQ was reported as acceptable (r = .70; Baron-Cohen et al., 2001), with an internal consistency of .67 (p < .010). The internal consistency and Cronbach's Alpha Coefficients of the items in each of the five domains reported by the scale's authors were all moderate to high (communication r = .65; social skill r = .77; imagination r = .65; attention to detail r = .63; attention switching r = .67; Baron-Cohen et

al., 2001). In a study on 12 adults with ASD (19-57 years old), participants in the ASD group scored significantly higher on the AQ in comparison to neurotypical controls (M_{age} = 35.65; *SD* = 6.33; $t = 6.89$, $p < .001$), as well as participants in other diagnostic groups (e.g., OCD [$N = 12$]: $t = 3.99$, $p < .001$; socially anxious [$N = 12$]: $t = 3.99$, $p < .001$). Cronbach's alpha for the AQ in the present study was.85 indicating good internal consistency of the measure with respect to the participants of this study.

Buss-Perry Aggression Questionnaire. The BPAQ (Buss & Perry, 1992) is a 29-item self-report measure that assesses the symptoms of physical aggression, verbal aggression, anger and hostility. Physical aggression is assessed by items 2, 5, 8, 11, 13, 16, 22, 25 and 29, and uses phrases such as "I have become so mad that I have broken things" and "If I have to resort to violence to protect my rights I will." Verbal aggression is assessed by items 4, 6, 14, 21 and 27, and uses phrases such as "I tell my friends openly when I disagree with them" and "I often find myself disagreeing with people." Anger is assessed by items 1, 9, 12, 18, 19, 23 and 28, and uses phrases such as "Some of my friends think I am a hothead" and "When frustrated, I let my irritation show." Hostility is assessed by items 3, 7, 10, 15, 17, 20, 24 and 26, and uses phrases such as "I wonder why sometimes I feel so bitter about things" and "At times I feel I have gotten a raw deal out of life." All items are rated on a 5-point Likert scale including 1 (*extremely uncharacteristic of me*), 2 (*somewhat uncharacteristic of me*), 3 (*neither uncharacteristic nor characteristic of me*), 4 (*somewhat characteristic of me*), and 5 (*extremely characteristic of me*). All of the questions are positively phrased except for items 9 and 16 which are negatively phrased and, therefore, need to be reverse scored. The test takes approximately 10 minutes to complete. Although there are no cut-off scores with respect to high versus low levels of aggression, the higher the score received, the greater the severity of aggression reported by the respondent. For the purpose of this study, the overall score of the BPAQ was considered to be a continuous

dependent measure of the secondary feature of aggression for subsequent comparative analyses across groups.

In addition to demonstrating utility in studies on adults with neurodevelopmental disorders (e.g., White et al., 2012), the items of the BPAQ are in accordance with a more overt characterisation of aggression and, subsequently, the operational definition formulated for this secondary feature based on a review of current literature. Internal consistency for the four subscales has ranged from .72 (verbal aggression) to .89 (total BPAQ score; Buss & Perry, 1992). Test-retest reliability for the BPAQ over nine weeks was also satisfactory with correlations ranging from .72 for anger to .80 for physical aggression and for the total score (Buss & Perry, 1992). Confirmatory factor analyses have demonstrated a good fit for a four-factor model (Buss & Perry, 1992) and also have indicated that the four factors may reasonably be combined into a higher order measure of aggression (subscale correlations ranged from .25 to .48; Buss & Perry, 1992); these findings were replicated in a large, community sample (N = 1200; Gerevich, Bácskai, & Czobor, 2007). Cronbach's alpha for the BPAQ was considered adequate for the present study at .86.

Depression Anxiety and Stress Scales. The DASS (Lovibond & Lovibond, 1995) is a 42-item self-report measure that assesses the symptoms of depression, anxiety, and stress in both adults and adolescents. All items are rated on a 4-point Likert scale including 0 (*did not apply to me at all*), 1 (*applied to me to some degree, or some of the time*), 2 (*applied to me a considerable degree, or a good part of the time*), and 3 (*applied to me very much, or most of the time*). Each subscale has 14 questions allocated to it, all of which are positively phrased. The inventory considers symptoms experienced over the last week, with questions on depression (items: 3, 5, 10, 13, 16, 17, 21, 24, 26, 31, 34, 37, 38, 42) including statements such as "I couldn't seem to experience any positive feeling at all" and "I just couldn't seem to get going." Questions on anxiety (items: 2, 4, 7, 9, 15, 19, 20, 23, 25, 28, 30, 36, 40, 41)

include statements such as “I was aware of the dryness in my mouth” and “I had a feeling of shakiness (e.g., legs going to give way).” Items focussing on stress (items: 1, 6, 8, 11, 12, 14, 18, 22, 27, 29, 32, 33, 35, 39) involve statements such as “I found myself getting upset by quite trivial things” and “I felt that I was using a lot of nervous energy.” The test takes approximately 10 minutes to complete. Scores for all three facets of the DASS range from 0 to 42, with normative scores for depression ranging from 0 to 9, and extremely severe cases having scores of 28 or more. Normative scores for anxiety range from 0 to 7, with extremely severe cases having a score of 20 or more. Normative scores for stress range from 0 to 14, with extremely severe scores totalling 34 or higher. For the purpose of this study, the depression and anxiety subscales were treated as separate, continuous, dependent measures of these secondary features for subsequent comparative analyses across groups.

Participants’ aggregate scores on the anxiety and depression subscales of the DASS (comparable against standardised cut-offs established by research on non-clinical samples) result in informative continuous data, demonstrating the extent to which these features are experienced by each respondent, and not whether respondents are or are not exhibiting clinically diagnosable levels of anxiety or depression (Lovibond & Lovibond, 1995). Items of the DASS are also centred on important covert characteristics used to describe anxiety and depression as per the definitions formulated for this study; the inclusion of items describing the somatic sensations, in particular, might be useful for individuals, such as those with ASD, who often struggle to put in words what it is they are feeling (MacNeil, Prater, & Busch, 2009). Additionally, empirical exploration of the DASS demonstrates its reliable utility in community samples. For example, the psychometric properties of the DASS were assessed with a clinical sample of 152 outpatients as well as 30 non-clinical samples of both adults and adolescents (Lovibond & Lovibond, 1995). Favourable temporal stability was demonstrated based on anxiety (r = .79), depression (r = .71) and stress (r = .81) in test-retest results

obtained two weeks apart from each other (Brown, Chorpita, Korotitsch, & Barlow, 1997). A study by Zlomke (2009) was conducted on the psychometric properties of the DASS utilising undergraduate university students in the USA; of the 1138 participants 73% were female, with an age range of 18 to 41 years old (M_{age} = 20.16, *SD* = 2.01). This study found that the subscales of the DASS were rather highly inter-related (r = .79 to .83, $p < .010$), with the subscales containing good internal consistency ranging from .84 (anxiety subscale) to .91 (depression subscale). An ANOVA comparing the six different diagnostic subsets on the overall scale also yielded significant results, $F(5, 342) = 4.77$, $p < .001$. With respect to convergent validity, Lovibond and Lovibond (1995) found that overall scores on the DASS had satisfactory correlations with both the BAI, r = .81 (Beck et al., 1988), and and the BDI, r = .74 (Beck, et al., 1996). Cronbach's alphas obtained for the DASS anxiety and depression subscales in the present study were both adequate at .85.

Frontal Systems Behaviour Scale. The FrSBe (Grace & Malloy, 2001) is a 46-item rating scale designed to assess frontal lobe related behavioural syndromes in people 18 to 95 years of age. The scale has three factors: apathy (14 items), disinhibition (15 items), and executive dysfunction (17 items). The apathy subscale is assessed by items 1, 8, 11, 14, 16, 21, 23, 24, 29, 38, 39, 41, 42 and 46, and uses phrases such as "I speak only when spoken to" and "I neglect my personal hygiene." The disinhibition subscale is assessed by items 2, 4, 6, 9, 10, 12, 18, 27, 28, 30, 31, 32, 43, 44 and 45, and uses phrases such as "I do things impulsively" and "I do or say embarrassing things." The executive dysfunction subscale is assessed by items 3, 5, 7, 13, 15, 17, 19, 20, 22, 25, 26, 33, 34, 35, 36, 37 and 40, and uses phrases like "I show poor judgment, I am a poor problem solver" and "I am disorganised." All items are rated on a 5-point Likert scale including 1 (*almost never*), 2 (*seldom*), 3 (*sometimes*), 4 (*frequently*), and 5 (*almost always*). The scale takes approximately 15 minutes to complete. Fourteen of the items are negatively phrased (items 33 to 46) and, therefore,

need to be reverse scored. Final scores are converted into *t*-scores using normative samples to compare to standards in the general population. A *t*-score of 65 or greater is indicative of clinical impairment in frontal systems related behaviour. For the purpose of this study, the executive dysfunction and disinhibition subscales were treated as separate continuous, dependent measures for comparative analyses across groups. Total FrSBe scores were not used in the current study.

The majority of the literature on executive functioning in ADHD and ASD has utilised laboratory based tasks that do not necessarily access real-world, day-to-day executive functioning aspects (Barkley, 2012a; 2012b; Barkley & Fischer, 2011; Barkley & Murphy, 2011; Biederman et al., 2006; Boonstra et al., 2005; Burgess, 1997; Burgess et al., 1998; Hervey et al., 2004; Jonsdottir et al., 2006; Wallace et al., 2016; Weyandt & Gudmundsdottir, 2015; Willcutt et al., 2005). Despite the resemblance of these tasks to the everyday environment (e.g., assessing the ability to withhold, or inhibit, particular responses as seen in SSTs), these tasks often fail to actually correlate with measures of everyday executive functioning, leading to concerns that current means of assessment have lead to gross underestimations of executive dysfunction in clinical samples. Research on self-report based assessments of executive functioning in ADHD and ASD has demonstrated the utility of such sales in identifying atypicalities in executive functioning in adults with these disorders (Barkley, 2012a; 2012b; Barkley & Murphy, 2010; 2011; Barnhart & Buelow, 2017; Kenworthy et al., 2008; Mahone & Hoffman, 2007; Wallace et al., 2016). Barkley (2012a; 2012b; Barkley & Fischer, 2011; Barkley & Murphy, 2011) has argued that exploring executive functioning through self-report style rating scales is more likely to yield an accurate depiction of this secondary feature in individuals with ADHD and ASD. Consequently, self-report scales may be more suited to accurately assess executive dysfunction in an individual's everyday life due to higher sensitivity towards specific

domains of impairment and their relationship to daily life activities (Barkley, 2012a; 2012b; Barkley & Fischer, 2011).

With respect to the different self-report scales of executive functioning, the FrSBe was selected for the purposes of this research largely due to the fact that its construction and conceptualisation heavily considered the functional neural networks that underlie executive functioning and not just the behaviours that may result from atypicalities in this area (Bernstein & Waber, 2007); this factor is considered to be extremely important when assessing secondary cognitive features in ADHD and ASD (Hale, How, DeWitt, & Coury, 2001; Kenworthy et al., 2008; Mahone et al., 2002; Solanto, 2015; Sullivan & Riccio, 2007). Additionally, a review of the FrSBe's reliability and validty yielded satisfactory findings. In a study on 436 participants, evidence of adequate internal consistency was found for the overall scale $r = .88$), the executive dysfunction subscale ($r = .79$), the apathy subscale ($r = .72$), and the disinhibition subscale ($r = .75$; Grace, Stout & Malloy, 1999). When compared to the Neuropsychiatric Inventory of Neurobehaviour (NPI; Cummings et al., 1994), a significant correlation was found between the total scores of both scales ($r = .64$, $p < .001$), suggesting convergent validity exists between the measures (Norton, Malloy & Salloway, 2001). Cronbach's alphas obtained for both the FrSBe executive dysfunction (.83), and disinhibition subscales, (.84), were high in the present study.

Toronto Alexithymia Scale-20. The TAS-20 (Bagby et al., 1994) is a 20-item self-report measure of alexithymia. The scale has three factors: difficulties identifying feelings (DIF), difficulties describing feelings (DDF), and externally oriented thinking (EOT). The DIF subscale is assessed by items 1, 3, 6, 9, 11, 13 and 14, and uses phrases such as "I am often confused about what emotion I am feeling" and "I have feelings that I can't quite identify." The DDF subscale is assessed by items 2, 4, 7, 12 and 17, and uses phrases such as "It is difficult for me to find the right words for my feelings" and "I am often puzzled by

sensations in my body." The EOT subscale is assessed by items 5, 8, 10, 15, 16, 18, 19 and 20, and uses phrases such as "I prefer talking to people about their daily activities rather than their feelings" and "Looking for hidden meanings in movies or plays distracts from their enjoyment." All items are are rated on a 5-point Likert scale, including 1 (*strongly disagree*), 2 (*moderately disagree*), 3 (*neither disagree nor agree*), 4 (*moderately agree*), and 5 (*strongly agree*). Items 4, 5, 10, 18, and 19 are negatively phrased, and therefore need to be reverse scored. Scores ranging from 0 to 51 are indicative of non-alexithymic responses or low levels of alexithymia. Scores of 52 to 60 suggest the potential presence of high or borderline high alexithymia. Scores of 61 or higher indicate a high likelihood of alexithymia. For the purpose of this study, overall totals on the TAS-20 were utilised as continuous, dependent measures for all analyses.

A review of the research on alexithymia has demonstrated the TAS-20's utility not only in adult community samples, but in studies directly assessing the link between alexithymia and ASD in adults (e.g, Berthoz & Hill, 2005; Hill et al., 2004). Additionally, research utilising the TAS-20 demonstrates adequate levels of construct validity, with the three-factor structure theoretically congruent with the alexithymia construct (Bagby et al., 1994). The TAS-20 has been found to be a stable and replicable scale across clinical and nonclinical populations, and has demonstrated good internal consistency (r = .81) and test-retest reliability (r = .77, p < .010; Bagby et al., 1994). A meta-analysis on the use of the TAS-20 in 19 different countries yielded findings indicating strong support for the three-factor structure across various languages and cultures (see Taylor, Bagby & Parker, 2002). Cronbach's alpha for the TAS-20 in this study (.84) was considered to be adequate for research purposes.

Procedure

Study recruitment details. This research was advertised on various ADHD and ASD support group websites, social media sites (Facebook, Gumtree), local newspapers (Gold Coast Bulletin), and through advertising means at local universities (research participant pool notice board, digital advertising). Participants were eligibile to win an Apple iPad. Additionally, participants recruited from the ADHD and ASD support groups received a $20 gift voucher. Participants recruited from the university research participation pool were eligible to receive course credit.

Study assessment details. Participants recruited locally (suburbs of Gold Coast or Brisbane in Queensland Australia) had the option of being assessed face-to-face or online; those recruited from interstate and overseas completed the study online. All participants assessed face-to-face completed the assessment scales individually in a consultation room situated at a local university campus. All consultation room doors and windows were closed in an effort to minimize external distractions within each testing environment. Additionally, assessment times were scheduled when campus traffic and external noise were minimal. Participants were provided with an explanatory statement to obtain signed consent and adequately inform them of the study's process and intent. Participants were reminded that their involvement in the study was completely voluntary, and that they were welcome to withdraw from the study at any time during the testing session without penalty. Following the collection of basic demographic information, participants were asked to complete the assessment scales. Data collection was administered in-person by the student researcher. Anticipated assessment time ranged from 60 to 90 minutes and participants were made aware of this prior to arranging a meeting time. Order of completion of measures was counterbalanced at random to mitigate fatigue and response bias effects. With the exception of the counterbalanced measures, each participant group experienced the same assessment

procedure, receiving the same assessment scales. After completion of the assessment measures participants were debriefed and thanked for their participation.

Participants accessing the online version of the assessment were initially requested to provide a password before proceeding to the study site. Following this, participants read the explanatory statement provided on a separate webpage; here, they were reminded that their involvement in the study was completely voluntary, and that they were welcome to withdraw from the study at any time during the testing session without penalty. Consent was required before participants were redirected to the online questionnaires and was obtained by selecting "yes" in agreement to providing consent. Data collection was conducted online through secure PsychData and Survey Monkey accounts. Participants were asked to provide basic demographic information and to complete the assessment scales. Similar to the face-to-face assessment, anticipated assessment time ranged from 60 to 90 minutes and participants were made aware of this on the opening page of the assessment. Order of completion of measures was counterbalanced via the survey generator, to mitigate fatigue and response bias effects. With the exception of the counterbalanced measures, each participant group experienced the same assessment procedure, receiving the same assessment scales. Following completion of the assessment measures participants were directed to a page notifying them of the completion of the study and were provided with the student researcher's contact details should they have further questions or concerns regarding the study.

Research Design

The program of research for Study 1 adopted a one-way, between-groups design, with study group (ADHD, ASD, control) serving as the independent variable. Dependent variables pertaining to the secondary features of ADHD and ASD included the FrSBe disinhibition and executive dysfunction subscales, the DASS anxiety and depression subscales, and the total scores of the BPAQ and TAS-20. Simple bivariate correlations were conducted to test for

significant relationships between the variables of interest, and assignment checks were performed to confirm that participants in each study group adequately represented adults sampled from ADHD, ASD, and neurotypical populations. MANCOVA was first utilised to test for any significant group (ADHD, ASD, control) differences in relation to the requisite dependent measures. In order to control for the influence of extraneous variables, gender and age, these were entered as covariates (being a dichotomous variable, gender was entered as an independent variable; Huitema, 2011). Data were then subjected to a Discriminant Function Analysis to ascertain how ADHD and ASD might differ in relation to symptom (primary impairment) clustering, and to generate visual representation of the interrelationships between secondary features across ADHD, ASD, and control groups. The Discriminant Function Analysis was conducted to further explore significant group differences following the MANCOVA, breaking down the linear combination of outcome variables described by the MANCOVA and further detailing the pattern of differences between variables (Field, 2013).

Assignment Checks

To examine the pattern of ADHD and ASD symptomatology across groups, separate one-way ANOVAs were conducted with study group (ADHD, ASD, control) serving as the independent variable, and scores obtained on either the AQ (Baron-Cohen et al., 2001) or ASRS-v1.1 (Adler et al., 2003) serving as dependent variables. Statistical significance was assessed at $p < .05$. There were significant main effects of both the ADHD group, $F(2, 275) = 69.43$, $p < .001$, and the ASD group, $F(2, 276) = 120.99$, $p < .001$. Employing a Bonferroni adjusted alpha of .017 (.05/3), three a priori comparisons were performed for each of the dependent variables. Linear contrasts demonstrated that the neurotypical participants scored significantly lower with respect to both dependent variables: AQ (control vs. ASD), $t(274) = -15.35$, $p < .001$; AQ (control vs. ADHD), $t(274) = -6.71$, $p < .001$; ASRS-v1.1 (control vs.

ASD), $t(273) = -11.02$, $p < .001$; ASRS-v1.1 (control vs. ADHD), $t(273) = -18.02$, $p < .001$. Additionally, pairwise comparisons revealed that participants diagnosed with ASD scored significantly lower on the ASRS-v1.1 relative to participants diagnosed with ADHD, $t(273) = -7.00$, $p = .002$; and participants with ADHD scored significantly lower on the AQ relative to participants diagnosed with ASD, $t(274) = -8.82$, $p < .001$. The mean scores, standard deviations and *z*-scores obtained on the AQ and ASRS-v1.1 are presented in Table 2.

Table 2

Group Specific Means, Standard Deviations and z-scores for the AQ and ASRS-v1.1

		Independent Variables	
Group		AQ	ASRS-v1.1
ADHD	*M*	23.58	47.84
	SD	(6.72)	(11.23)
	z	-0.06	0.64
ASD	*M*	32.40	40.84
	SD	(6.94)	(11.49)
	z	0.89	0.09
Control	*M*	16.87	29.83
	SD	(6.54)	(8.71)
	z	-0.79	-0.78

N = 278. *Note.* AQ = Autism Spectrum Quotient. ASRS-v1.1 = Adult ADHD Self-Report Scale. ADHD = Attention-Deficit Hyperactivity Disorder. ASD = Autism Spectrum Disorder.

Results

Statistical analysis of the data was performed using IBM Statistical Package for the Social Sciences (SPSS) version 23.0 for Windows. Prior to conducting the planned statistical analyses, the data were screened for outliers and violations to other relevant statistical assumptions.

Data screening and preparation. Due to analyses being performed on the grouped

data, the presence of univariate outliers was assessed separately within each study group. *Z*-scores in excess of plus or minus 3.29 ($p < .05$, two-tailed) were considered to reflect univariate outliers. One univariate outlier was identified in the DASS anxiety scores for both the ADHD ($z = 3.42$) and ASD groups ($z = 3.54$). In the neurotypical control group, univariate outliers were identified in the DASS depression ($z = 3.42$) and FrSBe executive dysfunction scores ($z = 3.69$). All univariate outliers were removed prior to analysis. Across all three groups, Mahalanobis distances ($p < .001$) demonstrated that no multivariate outliers existed across any of the study groups.

The assumption of normality was assessed by inspecting frequency histograms and normal quantile-quantile plots, which were produced for all requisite dependent variables for the overall samples. Additionally, normality was determined by a non-significant ($p > .05$) Shapiro-Wilk statistic, as well as standardised skewness and kurtosis values as calculated and evaluated at $z = +/-1.96$. Based on these criteria, all distributions appeared to meet the assumption of normality with the exception of anxiety and depression, which demonstrated a positive skew across all three study groups. This was anticipated, however, due to the fact that a normal distribution on these subscales of the DASS would represent a study sample that is significantly anxious or depressed. Scores on the TAS-20 appeared to have a negative skew in the ASD group; this distribution of scores appeared to be representative of the underlying population of interest with respect to the standardised cut-off scores for normalcy in groups largely comprised of individuals with a disorder known to be in deficit with respect to emotional processing (Bagby et al., 1994). The assumption of linearity was deemed to be met based on inspection of scatterplots between pairs of variables within each clinical group.

Preliminary analyses. Descriptive statistics on the secondary features of ADHD and ASD groups are presented in Table 3. With respect to all measures, higher scores represented higher levels of the construct being assessed (Bagby et al., 1994; Buss & Perry, 1992; Grace

& Malloy, 2001; Lovibond & Lovibond, 1995). Both the ADHD and ASD groups scored above the normal range for anxiety (0-7) and depression (0-9) as assessed by the DASS (Lovibond & Lovibond, 1995). Both the ADHD and ASD groups showed group scores that exceeded the normal range for the TAS-20 (0-51; Bagby et al., 1994). Participants in the ADHD and ASD groups scored noticeably higher on the BPAQ in comparison to neurotypical controls (Buss & Perry, 1992). Finally, group means on the FrSBe for the ADHD and ASD groups were within the clinically abnormal range for all subscales with respect to the standardised scores ($t \geq 65$; Grace & Malloy, 2001). However, for the purposes of this research, FrSBe raw scores were used in all analyses in order to remain consistent with the non-standarised values of the remaining assessment scales.

Table 3

Means, Standard Deviations and z-scores of the Secondary Features of ADHD and ASD

Group		Executive Dysfunction	Disinhibition	Alexithymia	Anxiety	Depression	Aggression
ADHD	*M*	48.29	38.88	50.65	10.20	14.03	75.27
	SD	(9.85)	(8.50)	(13.72)	(7.56)	(11.68)	(18.70)
	z	0.37	0.27	-0.18	0.14	0.20	0.02
ASD	*M*	48.10	39.05	63.30	10.41	14.66	85.60
	SD	(8.80)	(7.95)	(12.66)	(8.22)	(12.03)	(20.42)
	z	0.36	0.29	0.68	0.17	0.26	0.53
Control	*M*	36.67	31.29	46.43	6.70	6.53	64.33
	SD	(8.27)	(8.39)	(12.23)	(6.71)	(7.36)	(16.11)
	z	-0.73	-0.57	-0.46	-0.31	-0.47	-0.52

Note. $N = 278$. ADHD = Attention Deficit Hyperactivity Disorder. ASD = Autism Spectrum Disorder.

Table 4 reports the confidence intervals (Field, 2013) and intercorrelations for the study variables entered into the MANCOVA. As demonstrated in Table 4, correlations across the secondary features of ADHD and ASD were significant and positive. These relationships demonstrated medium to large effect sizes (Cohen, 1988; 1992). As none of the correlations exceeded .90, the assumption of no multicolinearity was met.

Multivariate Analysis of Covariance. Analyses yielded a Box's M = 187.48, F(105, 31030.15) = 1.65, $p < .001$. Levene's test of equality of error variances was significant for measures of anxiety, depression and aggression at $p < .05$. As such, results were interpreted with no equal variances assumed (Huberty & Petoskey, 2000). Statistical significance was assessed at $p < .05$ for the analyses, including assessment of univariate ANOVAs (Tabachnick & Fidell, 2007). A one-way MANCOVA was conducted with study group (ADHD, ASD, control) serving as the independent variable. Dependent variables included scores on the disinhibition and

Table 4

Intercorrelations and Confidence Intervals of the Secondary Features of ADHD and ASD (N = 278)

Variable	*1.*	*2.*	*3.*	*4.*	*5.*	*6.*
1.Anxiety	-					
2. Depression	.68*** [.62, .74]	-				
3. Executive Dysfunction	.44*** [.34, .53]	.47*** [.38, .56]	-			
4. Disinhibition	.47*** [.37, .56]	.39*** [.29, .49]	.68*** [.61, .74]	-		
5. Alexithymia	.45*** [.35, .54]	.48*** [.38, .56]	.58*** [.49, .65]	.50*** [.41, .59]	-	
6. Aggression	.41*** [.31, .51]	.42*** [.32, .51]	.58*** [.50, .66]	.68*** [.61, .74]	.49*** [.40, .58]	-

Note. 95% confidence intervals for intercorrelations are presented in parentheses. ADHD = Attention-Deficit Hyperactivity Disorder. ASD = Autism Spectrum Disorder.

*$p < .05$. **$p < .01$. ***$p < .001$.

executive dysfunction subscales of the FrSBe (Grace & Malloy, 2001), anxiety and depression subscales of the DASS (Lovibond & Lovibond, 1995), and overall scores on the TAS-20 (Bagby et al., 1994) and the BPAQ (Buss & Perry, 1992). Comparative analyses exploring group differences across demographic variables yielded significant group differences on gender and age. As such, age and gender were entered into the model as

covariates. Due to gender's categorical nature, it was entered into the model alongside the independent variable.

Pillai's Trace demonstrated a significant main effect of group, $F(12, 512) = 13.26$, $p < .001$, partial $\eta^2 = .24$. Follow-up ANOVAs yielded significant variations among clinical groups for executive dysfunction, $F(2, 261) = 33.94$, $MSE = 83.27$, $p < .001$, disinhibition, $F(2, 261) = 19.71$, $MSE = 65.59$, $p < .001$, alexithymia, $F(2, 261) = 36.90$, $MSE = 162.30$, $p < .001$, aggression, $F(2, 261) = 24.50$, $MSE = 329.10$, $p < .001$, anxiety, $F(2, 261) = 9.01$, $MSE = 52.50$, $p < .001$, and depression, $F(2, 261) = 14.06$, $MSE = 106.94$, $p < .001$. Planned comparisons were performed for each measure that yielded a significant ANOVA to delineate the between-group differences. Employing an alpha level of $p < .017$ (.05/3), a priori linear contrasts revealed that the ADHD group obtained significantly higher scores relative to the neurotypical control group for measures of executive dysfunction, $t(180.92) = 8.73$, $p < .001$, disinhibition, $t(184.93) = 6.15$, $p < .001$, aggression, $t(182.56) = 4.29$, $p < .001$, anxiety, $t(183.63) = 3.36$, $p = .001$, and depression, $t(161.57) = 5.27$, $p < .001$. Participants in the ADHD group showed higher levels of alexithymia than neurotypical controls, $t(183.75) = 2.22$, $p = .028$; although this difference was no longer significant once adjusting the *p*-value to accommodate the additional analyses (i.e., $p < .017$ not $< .05$), the mean TAS-20 score for the ADHD adults was in the moderately alexithymic range. Significantly higher scores were also observed in the ASD group in comparison to the neurotypical control group for measures of executive dysfunction, $t(175.40) = 8.95$, $p < .001$, disinhibition, $t(177.98) = 6.37$, $p < .001$, alexithymia, $t(175.55) = 9.05$, $p < .001$, aggression, $t(165.40) = 7.74$, $p < .001$, anxiety, $t(167.98) = 3.31$, $p = .001$, and depression, $t(145.53) = 5.45$, $p < .001$. Pairwise comparisons between the ADHD and ASD groups demonstrated that participants in the ASD group reported significantly higher levels of alexithymia, $t(179.99) = 6.45$, $p < .001$, and aggression, $t(176.22) = 3.56$, $p < .001$. The remainder of comparisons

between the ADHD and ASD groups yielded no significant differences for executive dysfunction, $t(180.77) = -0.14$, $p = .889$, disinhibition, $t(180.98) = 0.13$, $p = .895$, anxiety, $t(176.44) = 0.18$, $p = .858$, or depression, $t(181.00) = 0.36$, $p = .718$. The plotted mean scores for the secondary features of ASD and ADHD are presented in Figure 1.

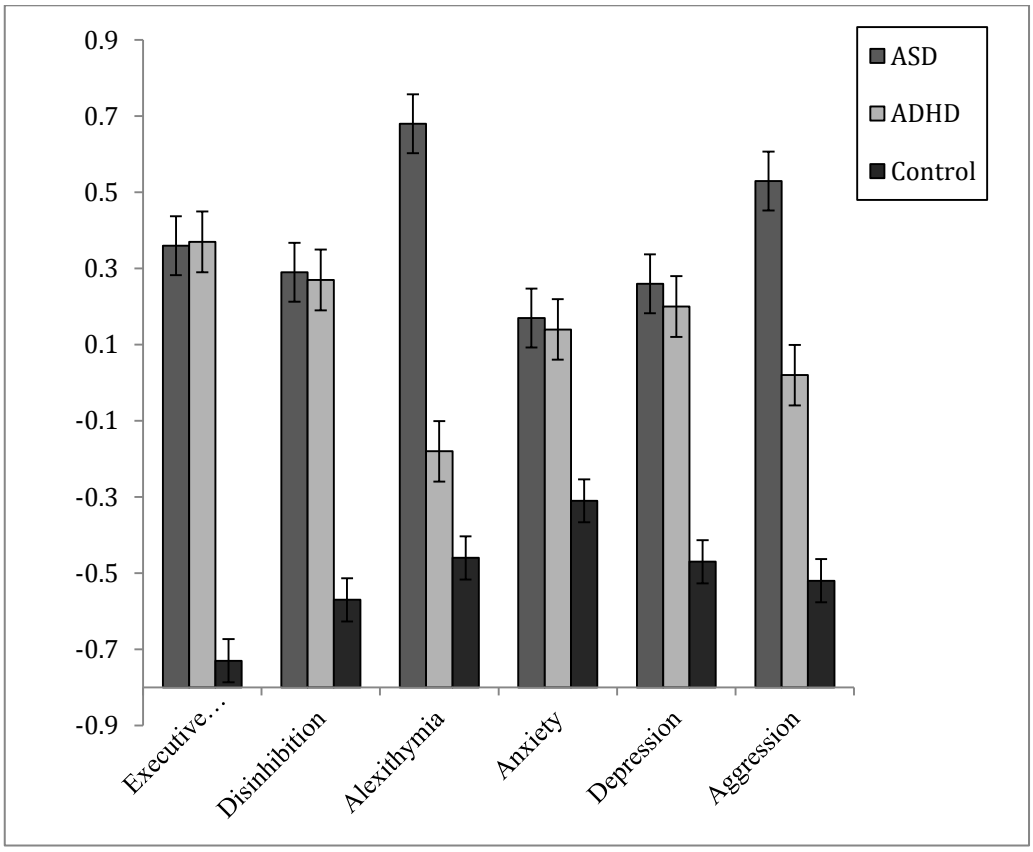

Figure 1. Mean *z*-scores obtained on the secondary features of Attention-Deficit Hyperactivity Disorder and Autism Spectrum Disorder. Error bars represent sample standard error.

Main analysis. To determine whether secondary features could discriminate between clinical groups, a Discriminant Function Analysis was performed. Analyses yielded a Box's $M = 68.66$, $F(42, 207403.99) = 1.58$, $p = .009$. Utilising an alpha level of $p < .05$, one significant discriminant function was obtained with a combined Wilks' lambda of $\Lambda = .53$, χ^2 $(12, N = 269) = 168.78$, $p < .001$. The second function was also observed to be significant

following removal of the first function, $\Lambda = .75$, χ^2 (5, $N = 269$) = 74.62, $p < .001$. Thirty percent of the total association between the variables was accounted for by the first function with a canonical R^2 value of .30. The first function also accounted for 56.8% of the between-group variability. The second function accounted for an additional 24.7% variance between the variables, as well as 43.2% of the between-group variability. The discriminant functions are represented graphically in Figure 2. As demonstrated in Figure 2, the first function differentiated the ASD group from the control group, whereas the second discriminated between the ADHD and ASD groups.

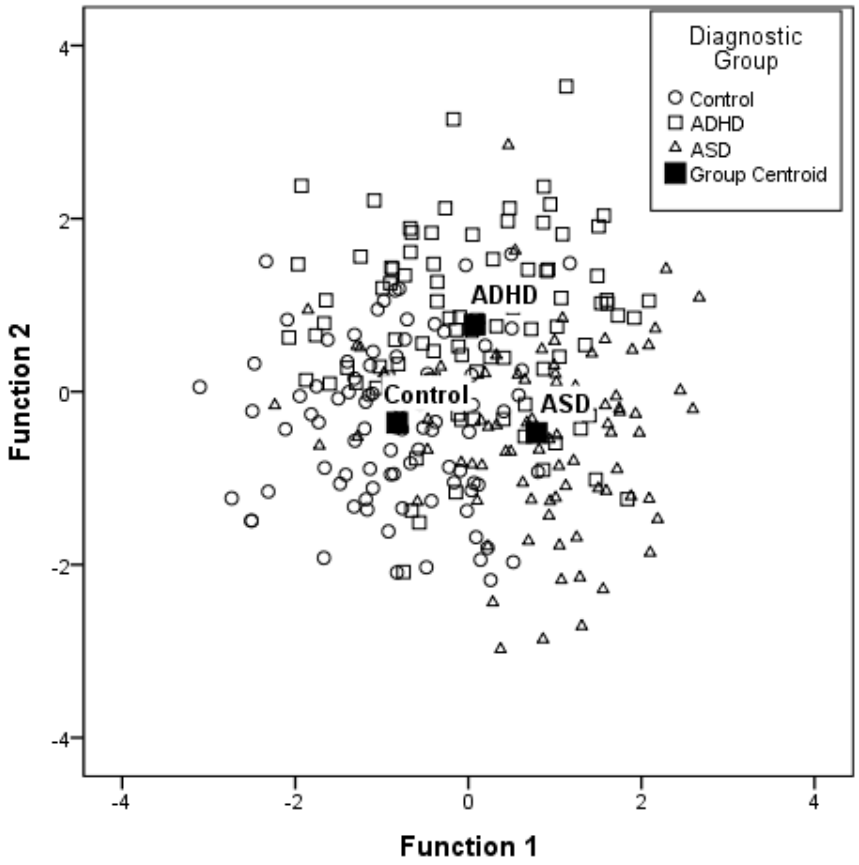

Figure 2. Differentiation of the three study groups on two discriminant functions across secondary features of Attention-Deficit Hyperactivity Disorder and Autism Spectrum Disorder ($N = 278$).

Variable correlations and standardised coefficients on the two discriminant functions are presented in Table 5. All variables on the first function appeared to be effective in discriminating between the ASD and control groups, however, executive dysfunction (*r* = .82) and alexithymia. (*r* = .81) were most effective. The variables on the second function were quite poor at discriminating between the ADHD and ASD groups with the exception of executive dysfunction (.*r* = .48) and alexithymia (*r* = -.35).

Table 5

Variable Correlations and Standardised Coefficients on Two Discriminant Functions Assessed for the Secondary Features of ADHD and ASD

	Standardised Coefficients		Correlation Coefficients	
Variable	Function 1	Function 2	Function 1	Function 2
Disinhibition	0.11	0.48	.65	.32
Executive Dysfunction	0.38	0.87	.82	.48
Alexithymia	0.49	-0.97	.81	-.35
Aggression	0.39	-0.55	.73	-.05
Anxiety	-0.25	0.01	.36	.14
Depression	0.19	0.31	.50	.19

Note. ADHD = Attention Deficity-Hyperactivity Disorder. ASD = Autism Spectrum Disorder. *N* = 278.

As demonstrated in Table 6, jack-knifed, leave-one-out classification demonstrated a higher level of correct classification than could be predicted by chance alone (33.49%), with 171 of the 269 cases (63.5%) being correctly classified. The remaining 98 cases were incorrectly classified, with 23 and 14 of the control participants being incorrectly assigned to the ADHD and ASD groups, respectively. Seventeen participants in the ADHD group were incorrectly assigned to the control group, with a further 12 incorrectly assigned to the ASD group. Finally, 15 participants in the ASD group were incorrectly classified in the ADHD group, and 17 in the control group.

Table 6

Jack-knifed Classification of Participants into Study Groups Based on Measures of Secondary Features

	Predicted Group Membership			
Study Group	Control	ADHD	ASD	Total
Control	58 (.64)	17	15	90
ADHD	23	54 (.57)	17	94
ASD	14	12	59 (.69)	85
Total	95	83	91	*N* = 269

Note: Group hit rates are reported in parentheses, expressed as decimals. ADHD = Attention Deficit Hyperactivity Disorder. ASD = Autism Spectrum Disorder.

Discussion

Study 1 aimed to compare the secondary features of cogniton, behaviour, and emotion across ADHD, ASD, and neurotypical study groups. A review of reported levels of executive dysfunction, disinhibition, alexithymia, aggression, anxiety, and depression across study participants yielded heightened levels of each feature in adults in the ADHD and ASD study groups in comparison to a sample of neurotypical adults. These results are consistent with what would be expected for adults with these conditions, and are displayed in Table 3 of the Results section.

Multivariate Analysis of Covariance. The comparative analysis of Study 1 aimed to explore the presence and intensity of secondary features differentiated adults in the ADHD and ASD study groups from a group of their neurotypical peers. The MANCOVA conducted on executive dysfunction, disinhibition, alexithymia, aggression, anxiety, and depression yielded a significant main effect of group type, indicating that there were significant overall differences between study groups on these domains.

Cognitive features of Attention-Deficit Hyperactivity Disorder. Planned comparisons further revealed that there were significant differences between the control and ADHD

groups on executive dysfunction, with adults in the ADHD group reporting significantly higher levels of executive dysfunction than adults in the neurotypical control group. These findings supported the hypothesis that adults with ADHD would report significantly higher levels of executive dysfunction in comparison to neurotypical controls and were congruent with research demonstrating significantly higher levels of executive dysfunction in adults with ADHD in comparison to neurotypical controls (Lovejoy et al., 1999; Murphy et al., 2001; Schecklmann et al., 2012; Taylor & Miller, 1997). Adults in the ADHD group also reported significantly higher levels of disinhibition than adults in the neurotypical control group. These findings supported the hypothesis that the ADHD group would report significantly higher levels of disinhibition in comparison to neurotypical controls and again were in agreement with previous research demonstrating elevated levels of this executive dyscontrol in adults in comparison to neurotypical controls (Barkley, 1994; 1996; 1997; Dinn et al., 2001; Epstein et al., 1998; 2001; Lovejoy et al., 1999; Murphy et al., 2001; Rapport et al., 2001; Taylor & Miller, 1997; Walker et al., 2000; Willcutt et al., 2005; Schecklmann et al., 2012). Alexithymia was also found to be elevated in adults with ADHD in this study; though there were no hypotheses pertaining to this feature for the ADHD sample in this study, this finding adds to the few studies on alexithymia in ADHD and its potential persistence across the lifespan in individuals with this disorder (Edel et al., 2010; Friedman et al., 2003).

Behaviour features of Attention-Deficit Hyperactivity Disorder. Planned comparisons revealed that adults with ADHD reported significantly higher levels of aggression than adults in the neurotypical control group. These results supported the hypothesis that adults with ADHD would report significantly higher levels of aggression than neurotypical adults assessed in this study and were congruent with research also demonstrating heightened aggression in adults with this disorder (e.g., Dowson & Blackwell, 2010).

Emotion features of Attention-Deficit Hyperactivity Disorder. Planned comparisons further revealed that adults with ADHD reported significantly higher levels of anxiety than adults in the neurotypical control group. These findings supported the hypothesis that reports of anxiety would be significantly higher in the ADHD group in comparison to the neurotypical control group and concurred with previous research findings that levels of anxiety are heightened in adults with this disorder (Barkley et al., 2008; Biederman et al., 1993; Duran et al., 2013; Minde et al., 2003; Shekim et al., 1990; Yoshimasu et al., 2016). Planned comparisons assessing depression further supported the hypothesis that adults in the ADHD study group would report significantly higher levels of depression than adults in the neurotypical control group, a finding supported by pre-existing research yielding heightened levels of depression in adults with ADHD (Biederman et al., 1996; 2008; Fischer et al., 2002; 2007; Michielsen et al., 2013; Roy-Byrne et al., 1997; Torgersen et al., 2006).

Cognitive features of Autism Spectrum Disorder. Planned comparisons revealed that adults in the ASD group reported significantly higher levels of executive dysfunction and alexithymia than adults in the neurotypical control group. These findings supported the hypothesis that individuals with ASD would report significantly higher levels of executive dysfunction in comparison to neurotypical controls and were congruent with research demonstrating significantly higher levels of executive dysfunction in adults with ASD in comparison to neurotypical controls (Ambery et al., 2006; Geurts & Vissers, 2012; Lai et al., 2012; Lopez et al., 2005; Rumsey, 1985; Rumsey & Hamburger, 1988; 1990; Williams et al., 2005). These findings also supported the hypothesis that the ASD group would report significantly higher levels of alexithymia in comparison to neurotypical controls and again were in agreement with previous research demonstrating elevated levels of this trait in adults with ASD (Berthoz & Hill, 2005; Hill et al., 2004; Silani et al., 2008).

Disinhibition was also found to be significantly higher in the ASD group in comparison to the neurotypical control group. Although there were no hypotheses specified for this feature for the ASD sample in this study, this finding adds to the small body of research yielding heightened levels of disinhibition in some adults with ASD (e.g., Lopez et al., 2005). In ASD, inhibitory deficits may originate from an inability to suppress behaviour that provides access to a preferred activity or topic of interest (Hughes & Russell, 1993). Moreover, the repetitive and perseverative behaviour often observed in individuals with ASD provides access to enjoyable activities and familiar structure in place of more unpredictable and potentially aversive activities (Folstein & Carcache, 2016). As such, although not as overt as the impulsive behaviour observed in individuals with ADHD, it is possible that inhibitory deficits in individuals with ASD do exist, and potentially contribute to the presentation of repetitive and familiar behaviour through a lack of suppression of more automatic responses.

Behaviour features of Autism Spectrum Disorder. Planned comparisons revealed that adults in the ASD group reported significantly higher levels of aggression than adults in the neurotypical control group. These results supported the hypothesis that adults with ASD would report significantly higher levels of aggression than neurotypical controls and were in agreement with previous research demonstrating elevated levels of aggression in adults with ASD (Brown & Radford, 2007; Farmer & Aman, 2011; Kanne & Mazurek, 2011; Pugliese et al., 2015; White et al., 2012).

Emotion features of Autism Spectrum Disorder. Planned comparisons further yielded reportedly higher levels of anxiety and depression in the ASD group in comparison to the neurotypical control group. These findings supported the hypothesis that reported levels of anxiety would be significantly higher in adults with ASD in comparison to neurotypical controls and concurred with previous research findings that levels of anxiety are heightened

in adults with this disorder (see review by van Steensel et al., 2011; also see Bejerot et al., 2014; Gillott & Standen, 2007; Lugnegård et al., 2011; Mazefesky et al., 2008; Rumsey, 1985). Additionally, these findings supported the hypothesis that adults in the ASD group would report significantly higher levels of depression than adults in the neurotypical control group; a finding supported by pre-existing research yielding heightened levels of depression in adults with ASD (Hill et al., 2004; Mazefsky et al., 2008).

A comparison of secondary features across Attention-Deficit Hyperactivity and Autism Spectrum Disorders. Comparisons between the ADHD and ASD groups yielded a significantly higher level of alexithymia in the ASD group in comparison to the ADHD group. This finding supported the hypothesis that adults in the ASD group would report significantly higher levels of alexithymia in comparison to adults in the ADHD group, and concurs with research demonstrating that adults with ASD often experience higher levels of alexithymia (Berthoz & Hill, 2005; Hill et al., 2004; Silani et al., 2008) than those reported in adults with ADHD (Edel et al., 2010). Non-significant differences across all remaining variables were found between ADHD and ASD groups in Study 1. The majority of the secondary features of ADHD and ASD were not hypothesised to differentiate significantly, however, these findings provided no support for the hypothesis that the ADHD group would report significantly higher levels of disinhibition in comparison to the ASD group. Although this largely goes against the literature supporting a difference between adults with ADHD and ASD on this executive domain (Johnston et al., 2011; Ozonoff & Jensen, 1999), some research has failed to differentiate groups of children and adolescents with ADHD and ASD on disinhibiton (Nydén et al., 1999). Disinhibition has not been identified as a key executive impairment across all individuals with ASD, but has been observed in some people with this disorder (see Ames & Jarrold, 2007; Biró, & Russell, 2001; Bishop & Norbury, 2005; Christ et al., 2007).

Discriminant Function Analysis. Further delineations of the complex relationships across secondary features of the adults assessed in Study 1 were conducted in the form of a Discriminant Function Analysis. In this analysis, both functions were found to be significant, and the overall model yielded significant predictive utility of the study variables. Function one of this analysis revealed executive dysfunction and alexithymia to be the most important secondary features when differentiating between adults in the ASD and adults in the neurotypical control groups. Function two identified executive dysfunction and alexithymia to be most effective at discriminating between adults in the ASD and ADHD groups. A review of the executive dysfunction scores across study groups revealed that the difference on executive dysfunction between the ADHD and ASD groups in this study was marginal at best (the mean ADHD executive dysfunction score was only .2 higher than that of ASD). Accordingly, this difference was not significant, suggesting that it might be the *nature* of the responses to the executive dysfunction subscale of the FrSBe that differentiated the adults in the ADHD and ASD groups of Study 1, not the overall scores. Moreover, the variables of the second function were not nearly as predictive as the variables of the first function, suggesting that although it was easy to differentiate between one of the clinical (i.e., ASD) and control groups of this study, it was not as easy to differentiate between the two clinical groups of the study (i.e., ADHD and ASD). These findings further support the notion that although the primary features of ADHD and ASD are distinctly unique, their secondary features are not as easy to differentiate.

Conclusion

Overall, a review of the data from the comparative analyses conducted in Study 1 revealed that the secondary features of ADHD and ASD carry the potential to persist into adulthood. More specifically, self-reported indices of executive dysfunction, disinhibition, alexithymia, aggression, anxiety, and depression were all significantly higher in the ADHD

and ASD groups in comparison to a group of neurotypical controls in an adult, community sample, even once age and gender were controlled for. Furthermore, secondary features not anticipated to be elevated in ADHD (e.g., alexithymia) and ASD (e.g., disinhibition) in accordance with the existing literature, were also observed. Consequently, though the assignment check of the adults in this sample revealed that the primary diagnostic features of the ADHD and ASD groups remained distinctly different, further comparisons of the secondary features of ADHD and ASD demonstrated very little differentiation on the cognitive, behaviour, and emotion-based features of these disorders, with the exception of alexithymia.

Chapter Six: Barkley's Model of Behaviour Selection

ADHD and ASD are both neurodevelopmental disorders characterised by primary (e.g., core impairments) and secondary features (e.g., cognitive, behaviour, and emotion patterns) associated with detriments to age-appropriate functioning across contexts and environments. The comparative analyses conducted in Study 1 revealed that the primary and secondary features (i.e., executive dysfunction, alexithymia, anxiety, depression, aggression) of ADHD and ASD proved to be significantly higher than neurotypical controls in a community sample of adults. Overall, these findings provided a necessary step in confirming that both the primary and secondary features commonly associated with these disorders in childhood are evident in adults with ADHD and ASD.

The basic data analyses reported in Chapter 4 involved the testing of specific differences (e.g., MANCOVA) and coefficients (e.g., bivariate correlations). Results from these forms of analysis typically provide evidence for or against hypothesised differences or relationships between variables, however, yield little indication as to whether study variables contribute to the presentation of one another (Hoyle, 2012). Therefore, in order to illuminate any between-variable contributions, the elevated secondary features identified in the comparative analyses of this study could be further explored in relation to one another, and the core diagnostic features of ADHD and ASD, by utilising an analytic approach known as data modelling (Rodgers, 2010). Data modelling explores the system of influence and interrelation between study variables that is often neglected by more basic analytic approaches (Hoyle, 2012). In the context of Study 1, the extent to which the primary and secondary features of ADHD and ASD influence and are interrelated to one another could be further investigated by establishing some form of model that incorporates all of these features and how they interact with one another. However, that the development of such a model should be heavily influenced by relevant pre-existing research and theory (Rodgers, 2010).

Current theoretical models of ADHD include explorations of dysfunctional processing of reward and/or punishment contingencies (Bechara, Damasio, & Damasio, 2000; Luman, Oosterlaan, & Sergeant, 2005; Rolls, 2004), extreme aversion to delayed reinforcement (Bitsakou, Psychogiou, Thompson, & Sonuga-Barke, 2009; Sonuga-Barke, 2003), and deficiencies in the ability to process situational information quickly and efficiently (McGrath et al., 2011; Rucklidge & Tannock, 2002; Shanahan et al., 2006). Nevertheless, research supporting these models has yet to robustly replicate consistent findings across samples of adults with ADHD (Hartung, Milich, Lynam, & Martin, 2002; Pauli-Pott & Becker, 2011; Scheres et al., 2006; Shanahan et al., 2006; Wilbertz et al., 2013; Willcutt et al., 2005), implicating that not any one universal deficit causes the behavioural patterns associated with ADHD. Instead, a comprehensive, neurocognitive model of ADHD should reflect the impairment of multiple cognitive processes within the brain, with the patterns in behaviour selection a product of atypicalities in several executive operations (Pennington, 2006; Willcutt et al., 2010).

In the case of ASD, Frith and colleagues have explored models of this disorder in the context of central coherence (Frith & Happé, 1994) with the postulation that individuals with ASD have a tendency to focus on the minute details of a situation, neglecting the big picture. Baron-Cohen, Knickmeyer, and Belmonte (2005) have also explored this idea of weak central coherence in ASD, describing it as a cognitive style that is inherently linked to an atypical proclivity for systemising and processing information that attends to specific rules which govern the behaviour of objects. Additionally, as part of their Theory of Mind hypothesis of ASD, Baron-Cohen and colleagues (1985; 1989) further postulated that individuals with ASD fail to take into account other people's mental states. However, research on both Theory of Mind and Weak Central Coherence does not support the notion of a universal deficit in either of these constructs across all individuals with ASD, nor has either

theory been able to account for all symptomatology in the disorder (Baron-Cohen, 1985; 1989; South, Ozonoff, & McMahon, 2007). Starting in the 1980s, causal investigations of ASD largely focussed on the relationship between ASD symptomatology and the functions associated with the frontal areas of the brain, resulting in the current exploration of ASD's underlying foundation of executive functioning deficits (Hill, 2004; Ozonoff, 1997; Ozonoff et al., 1991; Pennington & Ozonoff, 1996; Rozga et al., 2011; Russell, 1997; Sergeant et al., 2003). Consequently, similar to ADHD, current research suggests that a comprehensive, neurocognitive model of ASD is more appropriate; one that takes into account the impact that multiple cognitive processes within the brain have on the behavioural responses of adults with this disorder (Pennington, 2006; Willcutt et al., 2010).

A review of the research on ADHD and ASD indicates that exploration into the potential causal factors of these disorders' core features should heavily consider currently recognised neurobiological atypicalities and their cognitive and behavioural correlates (e.g., Pennington, 2006; Willcutt et al., 2010). Such an exploration would facilitate the development of a broader causal model of these disorders, and is likely to avoid the formulation of single-cause reductionist conceptual frameworks that do not capture the complexity of either disorder's core characteristics (Tannock, 1998). In his research on cognition and behaviour, psychiatrist Russel Barkley (1997) has established a model that explores the cognitive framework associated with the selection of behaviour that could be applied to the primary and secondary features common to ADHD and ASD. More specifically, Barkley has posited that behaviour is selected through various higher-order cognitive processes of executive functioning and has explored, at length, the extent to which atypicalities in this area of functioning impact subsequent behavioural patterns (Barkley, 1994; 1997; 2000; 2015). Consequently, in an attempt to establish a foundation for a model that incorporates the common secondary features (e.g., behavioural manifestations of

executive dysfunction) in the context of the core symptoms of ADHD and/or ASD, the remainder of this chapter will explore Barkley's evidence-based behaviour selection process.

Barkley's Model of Behaviour Selection: An Overview

The behaviour humans engage in is heavily influenced by its resultant consequences and their potentially reinforcing or punishing nature. Behaviour that is acted out in response to an immediate situation is most likely to yield some form of momentary benefit, with functional utility generally restricted to that situation (Strathman, Gleitcher, Boninger, & Edwards, 1994). Conversely, behaviour that is acted out in anticipation of a future state is most likely to yield a delayed benefit that carries the potential to serve a more long-term adaptive function for the individual (Strathman et al., 1994). Both forms of behaviour are influenced by the nature of reinforcement they are expected to yield, with some forms of behaviour predicted to provide something pleasurable (positive reinforcement) and others to escape or avoid aversive, punitive or otherwise undesirable events (negative reinforcement; Flora, 2004; Nikoletseas, 2010; Schulz, 2015). Future-oriented behaviour is typically expected to yield greater, albeit delayed, reinforcement in comparison to immediate-oriented behaviour due to its consideration of goal attainment and the maintenance of key personal and societal values (Barkley, 2015e; Cicchetti, Ackerman, & Izard., 1995; Beenstock, Adams, & White, 2011; Joireman, 1999; Joireman, Sprott, & Spangenberg, 2005; Peters, Joireman & Ridgeway, 2005; Strathman et al., 1994; Thompson, 1991). However, the pursuit of adaptive, future-oriented behaviour typically involves a greater level of conscious effort across a range of executive processes (Barkley, 1994; 1997).

In his model of behaviour selection, Barkley (1997) has posited that individuals either act in accordance with an immediate state or in anticipation of a future state through a series of self-directed operations that, collectively, represent the higher-order cognitive process of executive functioning. As demonstrated in Figure 3, within Barkley's model of

behaviour selection is a proposed sequence of executive operations including 1) awareness that an event has taken place in the environment that has evoked an internal response (self-awareness); 2) the inhibition of an initial response directed at the event (response inhibition); 3) the consideration of pre-existing information or novel concepts relevant to the event (working memory; cognitive reconstitution through means of cognitive flexibility and planning); and 4) the analysis and synthesis of relevant information in the formulation of suitable response alternatives (working memory; cognitive reconstitution through means of cognitive flexibility and planning). Although the individual domains that comprise executive functioning in Barkley's model represent private, covert operations through which an individual is able to generate important functional actions in the mind (Zelazo et al., 1997; Zelazo & Müller, 2002), Barkley has argued that it is the *interaction* between these executive operations that is essential in the conceptualisation of hypothetical future states and the cross-temporal organisation of behaviour for the attainment of future goals (Barkley, 2012a).

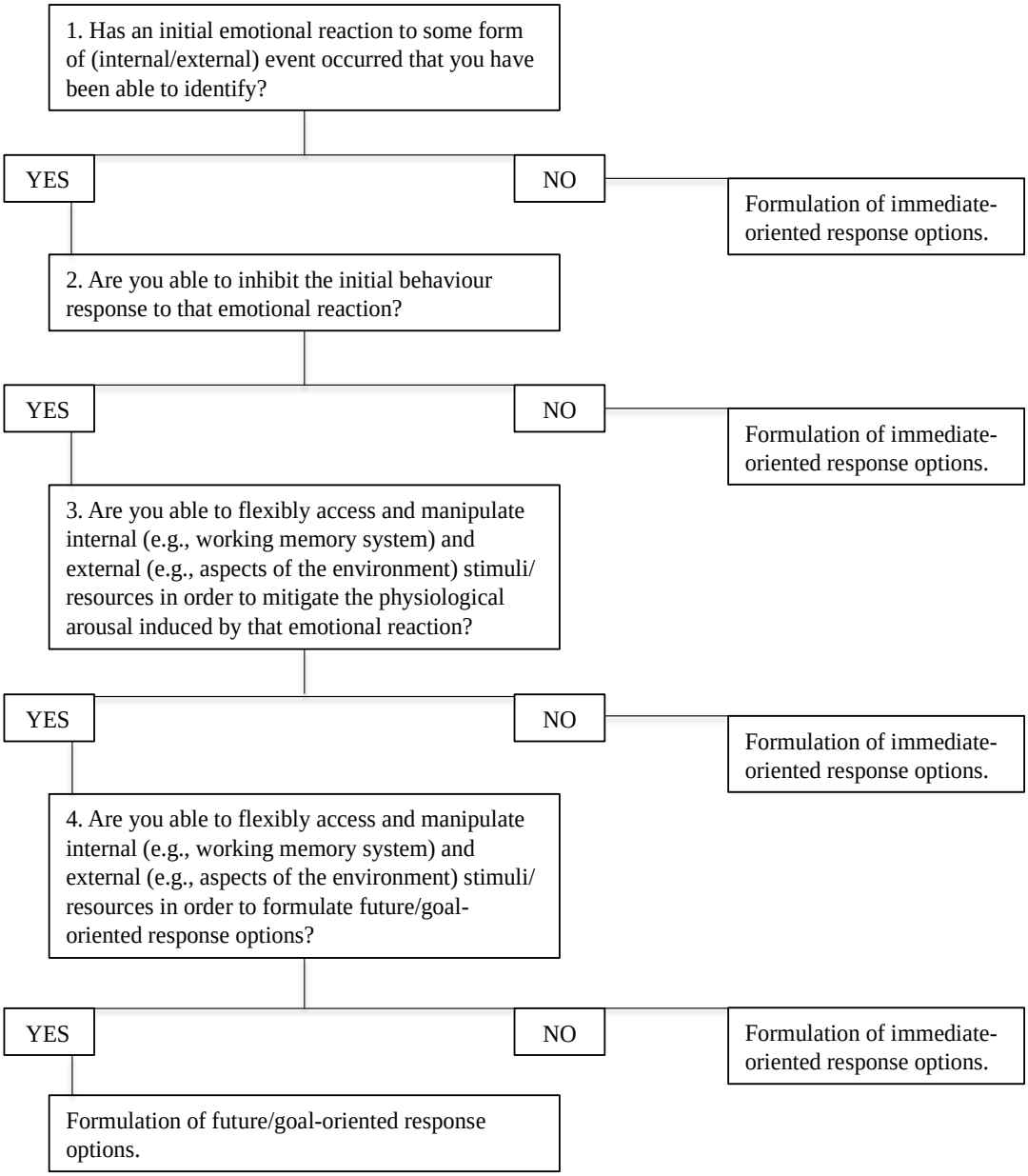

Figure 3. Barkley's (1997) model of behaviour selection.

Awareness and emotion identification. When there is synchronous functioning across executive operations, an individual is able to delay initial responses to an event and use self-directed, reflective behaviour to construct alternative responses for future situations, novel contexts, and complex problems (Barkley, 2012a). Barkley has posited, however, that before any other executive operations can be utilised to respond to a situation, an individual

must first be self-aware (Step 1 of Figure 3). Moreover, Barkley (1997) has suggested that before an individual can direct any further action back on him/herself there must be an initial sense of self to begin with. Self-awareness is the process involving consciousness of what is occurring outside of the person (e.g., external events in the environment), and any internal states (e.g., thoughts and emotions) that may arise in response to those external events (Lezak, 1995). An individual who is self-aware is able to recognise that personal interpretations and meanings attributed to events in the environment are what motivate initial emotional reactions to those events (Dryden, DiGiuseppe, & Neenan, 2010).

Emotions generate physiological arousal that contributes to the degree of activation, force, or intensity with which someone is motivated to act in response to them (Gray, 1994; Lang, 1995). Developing adequate means of regulating internal emotional states has been identified as an important factor in successful functioning across the lifespan (Cicchetti et al., 1995; Thompson, 1991). The regular tasks and activities people engage in each day can provoke unpleasant feelings and in order to overcome these feelings and continue to function, a repertoire of adaptive responses and self-regulatory capabilities is required (Barkley, 2015b). If an individual can mitigate the physiological arousal an emotional reaction induces, that individual can then shift focus away from the immediate emotional experience (Gottman & Katz, 1989; Gross & Thompson, 2007); this is important because the more an individual is able to modify the dynamic features of emotion, such as magnitude and duration of physiological arousal, the more likely that individual is to also evaluate and modify emotion in the service of a future state (Gottman & Katz, 1989; Gross & Thompson, 2007).

Response inhibition. Barkley has posited that once an individual is able to acknowledge that a change in emotion has been elicited in response to a situation, self-directed action in the form of response inhibition (Step 2 of Figure 3) can be undertaken to suppress the initial behavioural reaction to that emotional response (Barkley, 2015e). This

postulation is supported by research suggesting not only an association between self-awareness and executive functioning in general (e.g, Bogod, Mateer, & MacDonald, 2003), but self-awareness and response inhibition in particular (e.g., Eimer & Schleghecken, 1998). Response inhibition involves the suppression of an impulsive behavioural response before it occurs, or the interruption of an ongoing response no longer proving to be functionally effective (Barkley, 2015e; Rajendran & Mitchell, 2007). According to Barkley (1997), this executive operation is an essential pre-requisite to the remainder of the behaviour selection process due to the fact that before an adaptive and future-oriented response to an event can be selected, immediate, and often inappropriate, reactions to that event need to be inhibited or delayed. More specifically, no further executive operations can be engaged until an individual stops, however briefly, directing action toward an environmental event; in doing so, there is a separation between the event and the eventual sensorimotor responses that will be engaged in response to it, creating a temporal gap that provides the opportunity for further executive action without interruption (Barkley, 2015e). Without the cessation of such actions, attention cannot be shifted away from external reality and toward the contemplated future for the self (Barkley, 1997). Accordingly, in the presence of diminished response inhibition, the behavioural response selected is most likely to be a dominant or familiar response for which immediate reinforcement is anticipated or has previously been associated with (Barkley, 1997; 2000).

Cognitive reconstitution and the working memory system. Barkley (1997) has suggested that the initial stages of the behaviour-selection process, self-awareness and response inhibition, are paramount in establishing a foundation for a unified executive system. Once temporal distance has been created between an event and an individual's initial reaction to it, that individual can incorporate data from the external environment, as well as information stored in the working memory system to consider a range of relevant

factors while formulating an adaptive behavioural response (Barrett, Tugade, & Engle, 2004; Kuntsi, Oosterlaan, & Stevenson, 2001; Pennington, 1997). The working memory system is the storage centre for various forms of information that can be used to service goals and guide future-directed behaviour (Baddeley, 2003; Cowan, 2014; Norman & Shallice, 1986). The information stored in the working memory system includes important attributes of the current environment, plans for future states, and memories of previous experiences or similar situations (Hill & Bird, 2006; Lazarus & Folkman, 1984). According to Barkley (1997), the relevant mental constructs of the working memory system, are deconstructed and then recombined, or synthesised, to formulate new behavioural sequences that can be considered in the context of the problem to be solved through cognitive reconstitution. More specifically, by way of cognitive flexibility and planning, cognitive reconstitution generates response options by shifting between working memory system data to conceptualise change from the current situation (Hill & Bird, 2006). Together, cognitive reconstitution and the working memory system function to regulate emotions and, ultimately, formulate behavioural responses (Corballis, 1986; Fuster, 1997).

Barkley has proposed that certain aspects of the working memory system can be used to regulate emotions and mitigate the physiological arousal they produce (Barkley, 2015e). The regulation of emotion critically relies on the extent to which the contents of the working memory system are deconstructed and applied to new information as it becomes available (Pe, Raes & Kuppens, 2013). Barkley has stipulated that private images and self-verbalisations created by the working memory system can be accessed to evoke pleasurable emotional responses in an individual even in the absence of an actual situation or stimulus in the environment (Barkley, 1997; 2000; Fuster, 1997; Goldman-Rakic, 1995); these include covert reflections, self-instructions, self-questioning (Diaz & Berk, 1992), and the visualisation of past or future experiences (Dryden et al., 2010; Ellis, 1988; Ellis & Dryden,

1987; 2007). The images and self-statements created by the working memory system can redirect focus onto a relevant, positive memory or an ideal situational outcome, counter-acting any adverse emotional response that might otherwise demand an immediate behavioural response (Wilkinson, 2015). Accordingly, it is through private images and self-statements that the working memory can act to provide an essential link between perception and higher cognitive functions, allowing for the active maintenance of information about stimuli no longer in view (Harrison & Tong, 2009). In the context of Barkley's model, this is the stage of the behaviour selection process that can reduce the incentive or motivation to act in service of the immediate situation, and instead allow for focus on the generation of behaviour responses oriented toward a future state (Step 3 of Figure 3).

Barkley's (1997) model further posits that, following the amelioration of an emotional response to a situation, cognitive reconstitution and the working memory system can work together to formulate adaptive and future-oriented response options. Though all considerations of the working memory system are important in the formulation of future-oriented responses, Barkley has proposed that the reflection on previous, similar experiences and recreation of potential future experiences is essential in bridging the temporal gap between an action and its potential reinforcement (Step 4 of Figure 3). In this context, individuals can use mental recreations of past sensorimotor events to repeatedly re-experience situations they are not presently in and re-enact responses to stimuli that are not physically present, exploring and perfecting future responses to new and complex environmental events (Garnefski, Kraaij, & Spinhoven, 2001). It is through this process that cognitive reconstitution and the working memory system permit the private simulation of actions within specific settings that allow for an assessment of probable behavioural outcomes devoid of real-world consequence (Barkley, 2015e). When the working memory system is provided an opportunity to integrate such information, the temporal bridge between

response and reinforcement can be constructed by way of visualisation and conceptualisation and the complex patterns of behaviour directed, over time, to an ever more distant goal of the future (DeWall et al., 2007; Johns et al., 2008; Wagner & Heatherton, 2013).

Barkley's Model of Behaviour Selection: Summary and Directions for Future Research

Overall, according to Barkley (1997), self-awareness, response inhibition, working memory, and cognitive reconstitution provide an exceptionally powerful set of executive tools that greatly facilitate the selection of future-oriented behaviour. When working in conjunction with one another, these executive operations allow for an understanding of the temporal ordering of events, and requisite responses to them (Shimamura, Janowsky & Squire, 1990), that facilitate the generation of sequential steps needed to appropriately react to them (Sirigu et al., 1995). In doing so, Barkley (2015e) has posited that these executive operations allow for a change in focus from immediate reinforcement to delayed gratification, and, ultimately, from the immediate context to the conjectured future. Accordingly, higher levels of executive capacity have been associated with a greater ability to delay personal gratification (see Eigsti et al., 2006). Conversely, lower levels of executive capacity have been associated with higher usage of maladaptive, immediate-oriented behaviour (see Rabin, Fogel, & Nutter-Upham, 2011). Moreover, a review of the research on the specific executive operations in Barkley's model of behaviour selection reveals that a relationship exists between adaptive, future-oriented behaviour and heightened levels of self-awareness (Bagozzi, 1992; Hoyle & Sherrill, 2006), response inhibition (Christodoulou, Lewis, Ploubidis, & Frangou, 2006; Spinella, 2004), working memory (Byrne, Becker, & Burgess, 2007; McDaniel & Einstein, 2000), and cognitive flexibility and planning (Bagozzi, 1992).

Barkley (2012) has proposed that with increases in age and level of developmental maturation (e.g., transition from adolescence to young adulthood), so too come increases in executive capacity, the ability to be guided by more covert representations of the external

environment, and deferals of gratification in service of more future-directed goals. In this context, human beings demonstrate a substantial shift over the first three decades of life, discounting smaller, more immediate rewards and favouring larger, more delayed ones in adulthood (Greene et al., 1996). Nevertheless, given the number of the executive processes required to adequately select adaptive, future-oriented behaviour, deficits in any one of these underlying processes carry the potential to impede the formulation of an appropriate response to a situation, and therefore, can result in the selection of behaviour that is inappropriate and maladaptive in the long-term. Consequently, it could be anticipated that, in individuals known to experience atypicalities in executive functioning, such as those with ADHD and ASD, there might be a reduced likelihood that adaptive, future-oriented behavioural responses would be selected regularly, even in the later developmental stages of life (i.e., adulthood).

Barkley's empirical exploration of behaviour selection (1994; 1997; 2012b; 2015e) has largely been dedicated to the investigation of this process, and the executive operations that underlie it, in individuals with ADHD. Nevertheless, the majority of studies on ADHD and atypicalities in executive functioning continue to focus on children and adolescents (Barkley et al., 2010; 2011; Kessler et al., 2006; Murphy & Barkley, 1996). Barkley's model has also not been explored in adults with other neurodevelopmental disorders such as ASD who also experience deficits in executive functioning, limiting the empirical exploration of his model to that of ADHD (Thede & Coolidge, 2007). Consequently, what is unclear at this stage is the specific role that the processes of executive functioning play in the presentation of primary and secondary features in these disorders, particularly into adulthood. Moreover, a direct assessment of the potential causal path(s) for the primary and secondary behavioural patterns typically observed in adults with ADHD and ASD is yet to be conducted at length.

Further exploration of how individuals with ADHD and ASD manage and utilise competing information to guide controlled behaviour later in life, once behavioural patterns have been well established (Denckla, 1985; Ozonoff & Jensen, 1999), would be beneficial in this context. Such investigation may yield more information on the executive discrepancies across ADHD and ASD, including how individuals with these disorders process and respond to environmental stimuli and how this impacts symptom presentation. With respect to the clinical application of such an exploration, if the secondary features common to ADHD and ASD could be shown to be related to specific symptoms of these disorders, treatment planning might focus on these secondary features as a method of reducing such symptoms and the functional impairments associated with them. More specifically, if the goal of exploring the primary diagnostic criteria of a disorder is to inform treatment decisions, then identifying the secondary features that may influence such primary features is paramount in developing a treatment plan that alters the presentation of problematic symptomatology.

In the chapters to follow, the behaviour selection process according to Barkley (1997) will be explored, in depth, in the context of adults with ADHD and ASD. Self-awareness, response inhibition, and cognitive reconstitution will be discussed, at length, in light of current research on adults with ADHD and ASD in Chapters 7 and 9, respectively. Additionally, in an attempt to extend the comparative analyses of Study 1 and explore the extent to which the primary and secondary features of ADHD and ASD interrelate and impact one another, a form of data modelling known as path analysis will be applied to both the ADHD and ASD groups assessed in this study (Study 2). Results of these analyses for ADHD and ASD will be further addressed in Chapters 8 and 10, respectively. Finally, a comprehensive discussion of these behaviour selection paths in terms of relevant literature and clinical implications for both ADHD and ASD will be provided in Chapter 11.

Chapter Seven: Barkley's Model of Behaviour Selection as Applied to Adults with Attention-Deficit Hyperactivity Disorder

Barkley's model implicates the role of various neural networks across the brain with respect to executive processes (Barkley, 1997; 2012). In the context of a neurodevelopmental disorder like ADHD, such a model does not require all behavioural manifestations of the disorder to be linked to one universal deficit, but rather suggests these problems are linked to various vulnerabilities and abnormalities that exist within the same complex neural network. In ADHD, atypicalities exist in multiple stages of the behaviour selection process, including the initial response inhibition process, and insufficiencies in several aspects of the later executive functioning stages (e.g., working memory, cognitive planning). Nevertheless, an interactive model assessing the extent to which these cognitive features interact and influence other primary and secondary features in adults with ADHD is yet to be directly explored (Bloemsma et al., 2013).

The aim of Study 2 was to further investigate the role that cognitive factors such as disinhibition and executive dysfunction, and emotions such as heightened levels of anxiety and depression, play with respect to the formulation of behaviour responses typically observed in adults with this disorder. The formulation of the behaviour selection model used in Study 2 was based on Barkley's model (1997) and the premise that the same behaviour selection processes that result in the presentation of the primary diagnostic features of ADHD also result in the formulation of secondary aggressive behaviour. The complex network of relationships between these factors is depicted in Figure 4, with a description of the supporting research to follow.

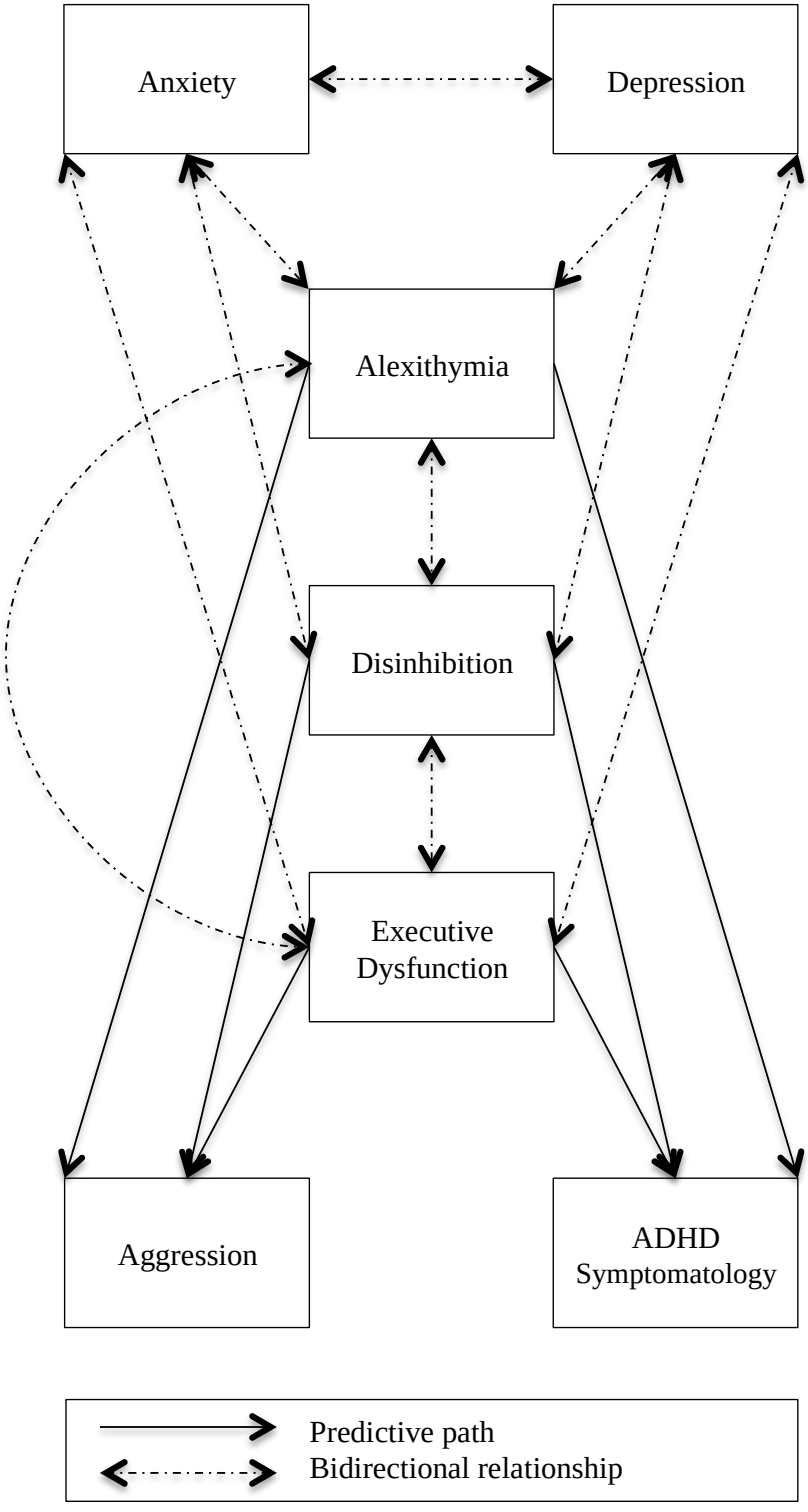

Figure 4. A proposed procedural path for behaviour selection in adults with Attention-Deficit Hyperactivity Disorder.

Awareness and Identification of Emotions in Attention-Deficit Hyperactivity Disorder

Although the ability to identify emotional states within the self does not appear to be universally problematic in adults with ADHD (Matuszak, Miller, Kemmelmeier, & Mason, 2013), the few studies on the cognitive trait of alexithymia in ADHD suggest that adults with this disorder are more likely to experience difficulties identifying and describing emotions than their neurotypical peers (e.g., Edel et al., 2010). Additionally, research on children with ADHD suggests that alexithymia may play a role in the presentation of hyperactive-impulsive symptomatology (e.g., Donfrancesco et al., 2013). Nevertheless, the decision to include alexithymia in Study 2 was heavily reliant on pre-existing theory on behaviour selection in this disorder, and the importance of emotional awareness at the start of the behaviour selection process (Barkley, 1997). Additionally, the significantly elevated rates of alexithymia demonstrated in the ADHD group in the comparative analyses of Study 1 demonstrated that, despite inconsistencies in the literature on alexithymia in adults with ADHD, the adults in the ADHD group of that study experienced this cognitive feature in markedly higher levels than their neurotypical peers. Consequently, the path analytic model developed for this study implied that the presentation of primary and secondary behavioural features of ADHD were likely directly influenced by heightened alexithymia.

In addition to showing potential relationshps with ADHD symptomatology, explorations of alexithymia in ADHD may also allow for further insight on the impact of specific emotions on behaviour selection. Research exploring alexithymia in adults has demonstrated that a relationship does appear to exist with respect to both anxiety (Devine, Stewart, & Watt, 1999; Marchesi, Fontò, Balista, Cimmino, & Maggini, 2005) and depression (Bankier, Aigner, & Bach, 2001; Berthoz et al., 2002; Honkalampi, Hintikka, Tanskanen, Lehtonen, & Viinamäki, 2000; Karukivi et al., 2010), such that as levels of alexithymia increase so do levels of anxiety and/or depression. As assessed via the TAS-20,

adults experiencing difficulties processing and regulating emotions are reportedly at risk of developing clinically diagnosable forms of anxiety (e.g., Generalised Anxiety Disorder; Berthoz et al., 2002; Marchesi et al., 2005) and depression (e.g., Major Depressive Disorder; Bankier et al., 2001; Berthoz et al., 2002; Honkalampi et al., 2000; Luminet, Bagby, & Taylor, 2001; Saarijävi, Salminen, & Toikka, 2001; 2006; Taylor & Bagby, 2004). Research assessing levels of alexithymia in adults with depression in particular reveals that overall scores on the TAS-20, as well as scores on the difficulty identifying and difficulty describing emotions subscales, are associated with depression over and above that of what is observed in non-depressed adults (Bamonti et al., 2010; Leweke, Leichsenring, Krus, & Hermes, 2012; Liss et al., 2008; Marchesi, Bertoni, Cantoni, & Maggini, 2008; Saarijävi et al., 2001, 2006).

The decision to include the specific emotional states of depression and anxiety in the path analytic model of ADHD conducted in Study 2 was based on research demonstrating the persistence of anxiety and depression in this disorder across the lifespan (see Chapters 2 and 4). Further, in adults with ADHD, both anxiety (Eysenck, Derakshan, Santos & Calvo, 2007; Quay, 1988; 1996; Tannock, 2009) and depression (Ottowitz, Todo, Dougherty, & Savage, 2002) have yielded significant, negative relationships with executive functioning, such that as the functionality of executive operations decreases, experiences of anxiety and depression increase. Additionally, in adolescents and adults with ADHD, both anxiety and depression have yielded significant, negative relationships with response inhibition, such that as the functionality of executive operations decreases, experiences of anxiety and depression increase (Oosterlaan & Sergeant, 1996). Nevertheless, although a review of the literature on depression and anxiety in ADHD suggests that some form of relationship exists between these emotions and the behaviour patterns of adults with this disorder, there is no substantial body of evidence suggesting that either anxiety or depression are directly, causally related to the primary (i.e., core symptoms) and secondary behaviours (i.e., aggression) common to

ADHD. As such, it was not anticipated that either emotion would act as a direct predictor in any of the causal pathways for behaviour selection in this model, because their impact on behaviour formulation is likely delivered *through* cognitive processes such as executive functioning. Anxiety and depression were incorporated into the model as variables that contributed to the presentation of primary and secondary behaviour in ADHD through their connections to other influential factors (e.g., executive dysfunction, disinhibition), with the reciprocal relationship between executive functioning and emotionality reflected in the initial stages of the model.

Response Inhibition in Attention-Deficit Hyperactivity Disorder

Barkley has proposed that the initial stages of the behaviour-selection process, self-awareness and response inhibition, can be used to override immediate reactions to situations. Nevertheless, adults with ADHD have demonstrated marked difficulties suppressing impulsive responses to the thoughts and emotions initially evoked by events in their environment (Barkley, 1994; 1997; 2015e). In the context of Barkley's model (1997), the initial emotional experiences or thoughts in response to a situation in adults with ADHD are less likely to be alleviated, or altered, due to the fact that adults with this disorder struggle to inhibit their initial responses to situations, and subsequently do not take into account information that could be used to refocus attention on more helpful and future-oriented information. Accordingly, it is not necessarily that the reactive thoughts and emotions experienced by adults with ADHD are more intense than those without the disorder, rather that adults with ADHD are more likely to display impulsive or aggressive behaviour in response to these primary reactions due to their diminished ability to moderate responses elicited by these primary reactions.

Deficits in response inhibition pose serious threats to the initial stages of the behaviour selection process. Even if an adult with ADHD is unable to recognise that an event

has evoked some sort of emotional reaction within the self, he/she may not be able to supress an immediate behavioural reaction to this emotional response long enough to try and mitigate the physiological arousal associated with it (Barkley, 1997; 2012a; 2015; Cowan, 2008; Pennington, 1997). More specifically, adults with ADHD may select responses to situations that focus on the immediate moment because deficits in response inhibition do not allow them to pause long enough to see past the immediate moment (Bitsakou et al., 2009; Sonuga-Barke et al., 1992). Research on deficits in response inhibition in adults with ADHD supports the postulation that diminished response inhibition is associated with heightened levels of primary ADHD symptomatology (see Chapter 4; also see Barkley et al., 1990; 2000; 2015e), as well as secondary behavioural features such as aggression (see Chapter 4; also see Connor & Doerfler, 2008; Zalecki & Hinshaw, 2004). These findings provide support for the relationships depicted in this study's model between cognitive processes and the primary, core symptoms of ADHD, as well as a secondary behaviour, aggression. Consequently, the path analytic model developed for this study proposed that the presentation of primary diagnostic symptoms in ADHD (i.e., hyperactivity, impulsivity, inattention) and secondary behaviour features (i.e., aggression) were likely directly influenced by atypicalities in response inhibition.

Cognitive Reconstitution and the Working Memory System in Attention-Deficit Hyperactivity Disorder

Barkley has suggested that if the initial reaction to a situation is not suppressed, memories of previous experiences and knowledge of future plans stored within the working memory system cannot be used to de-escalate or alter an individual's emotional state (or the physiological arousal it induces) long enough to even consider the reinforcement that may lie outside of the immediate situation (Barkley, 1997; 2015a). Consequently, an inability to retrieve constructive content from the working memory system not only hinders the

mitigation of the physiological arousal associated with adverse emotionality, it carries the potential to further exacerbate the experience of such adverse emotionality. In the case of ADHD, enduring a lifetime of the disorder's associated functional challenges often results in the development of negative self-schemas and an increase in the salience of failure experiences in adults with the disorder (Knouse, Zvorsky & Safren, 2013; Ramsay & Rostain, 2003; Safren et al., 2005). Accordingly, the private images and verbalisations produced by the working memory of an adult with ADHD are at risk of being flooded with unfavourable imagery of past adversity and negative self-rhetoric that casts doubt on an adult's ability to navigate future challenges (Fuster, 1997; Goldman-Rakic, 1995). What can result from this is a pessimistic and catastrophising style of thinking that misconstrues ambiguous information as threatening, fixates on the worst possible outcome of a situation, and, ultimately, perpetuates states of anxiety and depression that are difficult to look beyond (Hirsch, Clark, Matthews, & Williams, 2003; Hirsch, Maynen, & Clark, 2004; Holmes, Lang & Deeprose, 2009; Holmes & Matthews, 2005; Moscovitch et al., 2011). As such, in addition to potentially explaining why adults with ADHD are at greater risk of experiencing anxiety and depression, the perpetuation of negative cognitions in the working memory system coupled with the disinhibited ADHD cognitive style may result in the selection of behaviour that continually pursues emotional reprieve above all other consequences. More specifically, the anxious and depressive states induced by the environment may continue to propagate inside the minds of adults with ADHD, acting as means of continuous prompting to immediately address the adverse emotion being experienced.

Barkley has suggested that it is possible to focus on more than just negative constructs of the working memory system or the adverse aspects of a problematic situation through cognitive reconstitution, and, more specifically, the fluency and flexibility of adequate executive functioning. Nevertheless, although cognitive flexibility appears to be relatively

intact in adults with ADHD (see Fischer et al., 1990), adults with this disorder have demonstrated marked impairment in the area of cognitive planning and the ability to adequately integrate positive, helpful, and future-oriented information into the formulation of response options (Young, 2005). Research has demonstrated that adults with ADHD often struggle to pay attention to all relevant situational data, encoding fewer social cues and neglecting to coherently incorporate the information they do encode into the behaviour selection process (Andrade et al., 2012; Barkley, 1997; Matthys, Cuperus, & van Engeland, 1999). Moreover, adults with ADHD are more likely to consider recent over more relevant situational information when formulating perceptions of an environmental event (Milich-Reich, Campbell, Pelham, Connely, & Geva, 1999). In adults with ADHD, the most noteworthy consequence of missing such information is the diminished presentation of controlled and purposeful responses that project outside the scope of the immediate situation (Willcutt et al., 2012). Accordingly, research on executive functioning in ADHD demonstrates a significant, positive correlation between overall executive dysfunction and the primary symptomatology (Ozonoff & Jensen, 1999; Pennington, 1997; Pennington & Ozonoff, 1996; Stavro et al., 2007), as well as secondary behaviour features, such as aggression, in this disorder (McQuade, Breaux, Miller, & Mathias, 2016). Consequently, the path analytic model developed for Study 2 implied that the presentation of primary and secondary behavioural features of ADHD was likely directly influenced by atypicalities in executive functioning.

Study 2. Although some studies have started to explore the relationship between the primary and secondary features of ADHD in the context of executive functioning (e.g., Jarrett, Rapport, Rondon, & Becker, 2014; Wood, Lewandowski, Lovett, & Antshel, 2014), little research has been conducted on the relationships between heightened emotionality and the executive processes impacting behaviour selection in adults with this disorder (Jarrett,

2015). Additionally, many studies only contain preliminary correlational associations between variables or regression analyses that have only explored certain aspects of the proposed behaviour selection model (e.g., Barnhart & Buelow, 2017; Smitherman, Huerkamp, Miller, Houle, & O'Jile, 2007). Consequently, a gap exists in the empirical exploration of the distinct cognitive processes involved in Barkley's model and the effect they have on behaviour formulation in adults with ADHD (Nigg et al., 2006; Pennington, 2006; Sonuga-Barke et al., 2003; Willcutt et al., 2010).

The current research on behaviour patterns characteristic of adults with ADHD, though limited, does provide some evidence that impairments in executive functioning (MacQuade et al., 2016; Ozonoff & Jensen, 1999; Pennington, 1997; Pennington & Ozonoff, 1996; Stavro et al., 2007) and, more specifically, response inhibition (Barkley et al., 1990; 2000; 2015; Connor & Doerfler, 2008; Zalecki & Hinshaw, 2004), are associated with the primary and secondary behavioural features of this disorder. As such, it was hypothesised that:

1. The model would significantly predict the primary and secondary behaviour features reported by adults in the ADHD group.
2. The level of disinhibition reported by adults in the ADHD group would act as a significant, positive predictor of aggression.
3. The level of disinhibition reported by adults in the ADHD group would act as a significant, positive predictor of ADHD symptomatology.
4. The level of executive dysfunction reported by adults in the ADHD group would act as a significant, positive predictor of aggression.
5. The level of executive dysfunction reported by adults in the ADHD group would act as a significant, positive predictor of ADHD symptomatology.

Results of the analysis of this model (Study 2), as well as a comprehensive discussion of a possible behaviour selection path in the context of of relevant literature, are further addressed in Chapter 8. The findings of this analysis are discussed in terms of clinical implications and directions for future research in Chapter 11.

Chapter Eight: Study 2- Path Analytic Modelling of the Primary and Secondary Features of Attention-Deficit Hyperactivity Disorder

In order to further assess the path through which adults with ADHD engage in behaviour primary and secondary to their diagnosis, Study 2 utilised data (obtained in Study 1) from the ADHD participant group (N = 96); the demographic information relevant to this study group is provided in Chapter 5. Participants in the ADHD study group were either assessed face-to-face or online; procedural information on both forms of assessment is also provided in Chapter 5. Relevant assessment scales for this analysis included the FrSBe (Grace & Malloy, 2001), BPAQ (Buss & Perry, 1992), DASS (Lovibond & Lovibond, 1995), ASRS-v1.1 (Adler et al., 2003), and the TAS-20 (Bagby et al., 1994). Detailed descriptions of all five scales are located in Chapter 5 of this thesis.

Research Design

In order to examine the ways in which the primary and secondary features interacted in the ADHD study participants, a path analysis was performed. Path analysis allows for the exploration of influence and interrelation within complex models of multiple exogenous and endogenous variables (Hoyle, 2012). Exogenous variables are those whose values are wholly causally independent from the other variables in the model (Wuensch, 2016). Conversely, endogenous variables are those whose variances are considered to be explained by other variables entered into the model (Wuensch, 2016). In Study 2, the exogenous variables of disinhibition, executive dysfunction, alexithymia, anxiety and depression were theorised to contribute to the presentation of the endogenous variables (i.e., primary, core symptoms and aggression) entered into the model. More specifically, the effect that cognitive factors (disinhibition, executive dysfunction, and alexithymia), and emotions (anxiety and depression), had on ADHD symptomatology and aggression were explored in the path

analysis model. Preliminary analyses to examine the bivariate relationships between model variables were conducted.

Results

Statistical analysis of the data was performed using IBM SPSS AMOS version 23.0 for Windows. Prior to conducting the planned statistical analyses, the data were screened for outliers and violations to other relevant statistical assumptions.

Data screening and preparation. The one univariate outlier (on DASS anxiety) identified in Study 1 was removed prior to analysis. Mahalanobis distances demonstrated that no multivariate outliers existed within the ADHD group. Based on screening analyses conducted in Study 1, all study variables appeared to meet the assumption of linearity. All variables appeared normal, with the exception of anxiety and depression, which were appropriately positively skewed (see Chapter 5 for review). Additional tests of normality demonstrated that ADHD scores on the ASRS-v1.1 were normal.

Preliminary analyses. Descriptive statistics on the secondary features of ADHD are presented in Table 7. Table 7 reports the means, standard deviations, confidence intervals (Field, 2013) and intercorrelations for the study variables entered into the ADHD path analysis. As demonstrated in Table 7, correlations across the cognitive, behaviour and emotion-based features of ADHD were significant and positive. These relationships demonstrated medium to large effect sizes (Cohen, 1988; 1992). As none of the correlations exceeded .90, the assumption of no multicolinearity was met.

Table 7

Means, Standard Deviations and Intercorrelations Across the Primary and Secondary Features of ADHD (N = 96)

Variable	*M*	*SD*	*1.*	*2.*	*3.*	*4.*	*5.*	*6.*	*7.*
1. Anxiety	10.20	7.56	-						
2. Depression	14.03	11.68	.66*** [.52, .76]	-					
3. Executive Dysfunction	48.29	9.85	.44*** [.26, .59]	.43*** [.25, .58]	-				
4. Disinhibition	38.88	8.50	.48*** [.30, .62]	.35*** [.16, .52]	.60*** [.45, .72]	-			
5. Aggression	75.27	18.70	.44*** [.27, .59]	.36*** [.17, .52]	.48*** [.31, .62]	.60*** [.45, .72]	-		
6. Alexithymia	50.56	13.62	.56*** [.40, .68]	.50*** [.33, .64]	.59*** [.44, .71]	.57*** [.42, .69]	.46*** [.28, .61]	-	
7. ADHD Symptoms	47.84	11.23	.43*** [.25, .58]	.25* [.05, .43]	.57*** [.42, .69]	.61*** [.47, .73]	.42*** [.24, .57]	.54*** [.38, .67]	-

Note. 95% confidence intervals for intercorrelations are presented in parentheses. *M* = Mean. *SD* = Standard deviation. ADHD = Attention-Deficit Hyperactivity Disorder. ASD = Autism Spectrum Disorder. *$p < .05$. **$p < .01$. ***$p < .001$.

Path analysis. Model fit indices are provided in Table 8. The normed fit index (NFI) and comparative fit index (CFI) were both above the recommended cut-off of .9, suggesting the model was comparable to that of the best fit, saturated model (Hu & Bentler, 1999). The Root Mean Square Error of Approximation (RMSEA) demonstrated the model was close to a perfect fit (Browne & Cudeck, 1993; Hu & Bentler, 1999; Kline, 2011). Moreover, when all variables were entered into the path analysis, the model was not significantly different from that of the saturated model, $\chi^2(5) = 7.51$, $p = .185$, suggesting there were no significant differences between the proposed model and a model that perfectly explains the presentation of primary symptoms and aggressive behaviour in ADHD.

Table 8

Model Fit (N = 96)

	NFI	CFI	RMSEA
Model	.98	.99	.07
Saturated Model	1.00	1.00	

Note. NFI = normed fit index. CFI = comparative fit index. RMSEA = root mean square error of approximation.

Table 9 reports the standardised and unstandardised beta weights of the individual paths within the model. Results demonstrated that executive functioning was a significant predictor of ADHD symptomatology, but not aggression. Results further demonstrated that disinhibition was a significant, positive predictor of ADHD symptomatology and aggression. Alexithymia did not appear to significantly predict either ADHD symptomatology or aggression. Figure 5 depicts the full path model including beta weights and correlation coefficients between model variables.

Table 9

Individual Paths within the Model for the Primary and Secondary Features of ADHD (N = 96)

Path	*B*	*SE B*	*β*
Executive dysfunction → ADHD Symptomatology	0.28	.12	.24*
Executive dysfunction → Aggression	0.28	.21	.15
Disinhibition → ADHD Symptomatology	0.48	.13	.36***
Disinhibition → Aggression	0.98	.24	.45***
Alexithymia → ADHD Symptomatology	0.15	.08	.19
Alexithymia → Aggression	0.16	.14	.12

Note. ADHD = Attention-Deficit Hyperactivity Disorder.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Discussion

Study 2 aimed to assess the extent to which the cognitive and emotion-based features of ADHD contributed to the presentation of this disorder's primary and secondary behaviour. A review of the cognitive (executive dysfunction, disinhibition, alexithymia), emotion (anxiety, depression) and behaviour (primary symptomatology, aggression) variables across study participants demonstrated elevated levels across all secondary features in adults of the ADHD group. These results are consistent with what would be expected for individuals with a diagnosis of ADHD, and are displayed in Table 7 of the Results section.

A review of the model demonstrates that it was not significantly different from a saturated model (i.e., a model that perfectly explains the primary and secondary behavioural features of ADHD as predicted by executive dysfunction, disinhibition, alexithymia, anxiety, and depression). These findings are in support of the hypothesis that the model created to assess behaviour patterns in adults with ADHD would significantly predict both the primary and secondary behaviour features of this disorder. These findings also concur with Barkley's model of behaviour selection

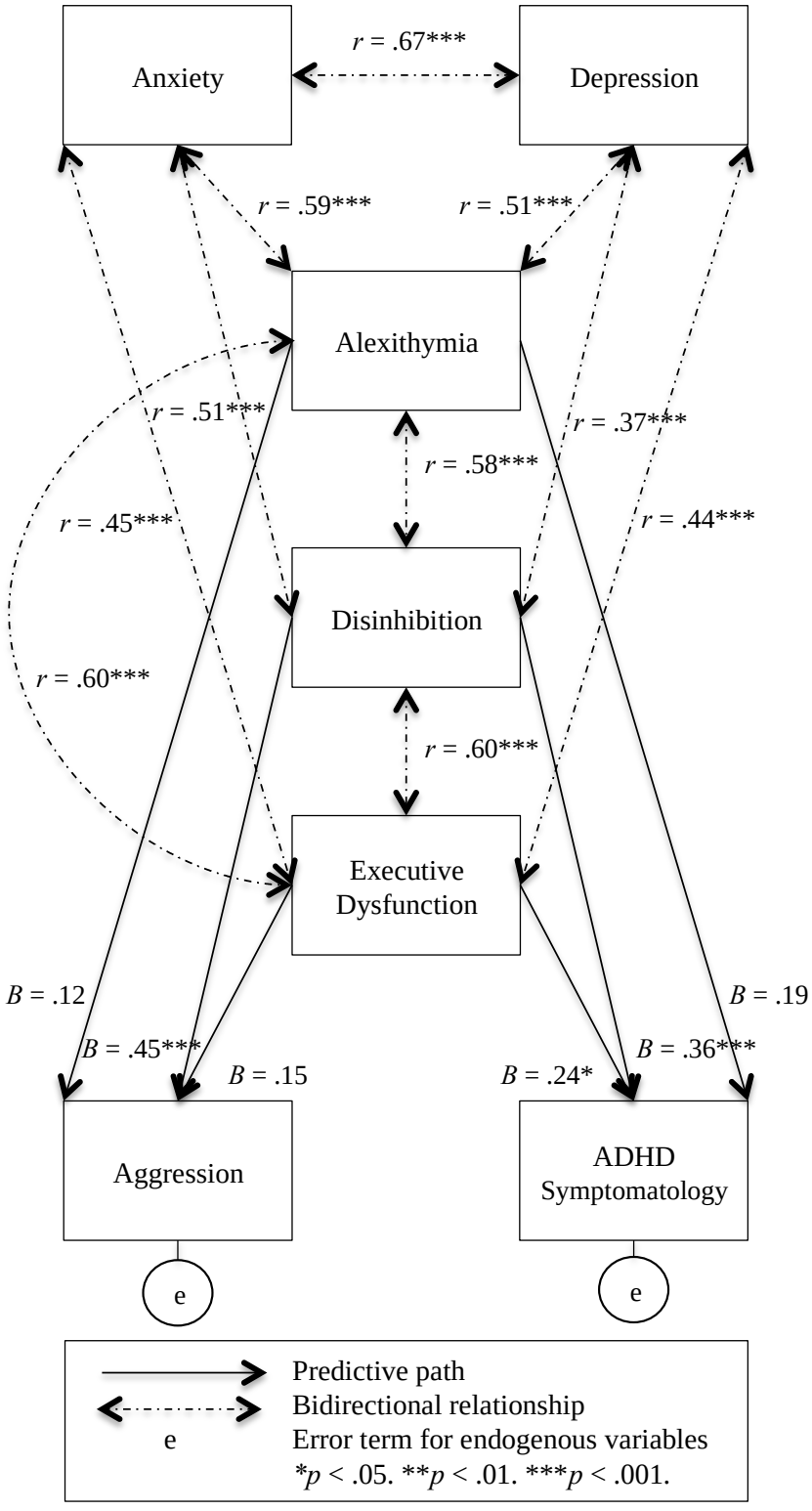

Figure 5. Path model for primary and secondary behaviour features of Attention-Deficit Hyperactivity Disorder (*N* = 96).

and, more specifically, the proposition that deficits in response inhibition, and the executive operations that facilitate it, contribute to the presentation of primary ADHD symptomatology and secondary behaviour features such as aggression.

A review of the correlations between the cognitive and emotion variables entered into the model demonstrated that significant, positive relationships existed between all study variables anticipated to have bidirectional relationships. These relationships were in accordance with research demonstrating links between the study variables (see Chapters 4 and 7 for an extensive review of this literature). These findings did not support any particular set of hypotheses pertaining to the behaviour selection model of ADHD. However, they did provide support for the inclusion of these specific variables in the model, and the notion that the internal processes involved in behaviour selection (e.g., integration of information from the working memory system and the environment) comprise a heavily integrated and complex network of covert operations (Barkley, 2012a; Cummings & Miller, 2007; Gazzaley & D'Esposito, 2007; Welsh & Pennington, 1988).

The unidirectional paths of the model revealed that several paths successfully predicted both the primary and secondary behaviour features assessed in the adults in the ADHD group. More specifically, executive dysfunction successfully predicted ADHD symptomatology. This finding supported the hypothesis that executive dysfunction would act as a significant predictor of the primary behaviour features of ADHD and is congruent with research supporting a link between executive dysfunction and primary ADHD symptomatology (Ozonoff & Jensen, 1999; Pennington, 1997; Pennington & Ozonoff, 1996; Stavro et al., 2007). Executive dysfunction did not, however, predict aggression. This finding is in contrast to current research supporting a link between executive dysfunction and aggression in ADHD (McQuade et al., 2016) and fails to provide support for the hypothesis that executive dysfunction would successfully predict aggression in adults with ADHD.

Conversely, disinhibition was found to successfully predict both ADHD symptomatology and aggression, supporting the hypotheses that disinhibition would act as a significant predictor of both ADHD symptomatology and aggression in an adult sample. These findings are in accordance with research supporting a link between disinhibition and ADHD symptomatology (Barkley et al., 1990; 2000; 2015; Barkley & Fischer, 2011), and disinhibition and aggression in ADHD (Connor & Doerfler, 2008; Zalecki & Hinshaw, 2004). The fact that disinhibition appeared to significantly predict aggression, even though executive dysfunction did not, perhaps further emphasises the importance of disinhibition with respect to the presentation of impulsive, emotion-driven behaviour in adults with ADHD. Although alexithymia's placement in the model contributed to the model's overall robustness and strength as a predictor of ADHD-related behaviour, this variable did not appear to significantly predict either ADHD symptomatology or aggression in adults in the ADHD group.

Conclusion

The data yielded from the path analysis conducted in Study 2 provides further support for the role that both emotion and cognitive factors play with respect to the presentation of primary and secondary behaviour patterns in adults with ADHD. Moreover, though not all hypotheses pertaining to the model were significant (e.g., executive dysfunction's failure to predict aggression), the results demonstrated that, in combination with the recognition of specific emotions, cognitive atypicalities common to ADHD do yield predictive utility with respect to behaviours selected in this disorder. In the context of Barkley's model of behaviour selection in ADHD, the results of this path analysis further support the important role of response inhibition with respect to not only the primary features of ADHD, but secondary behavioural patterns as well (i.e., aggression). These results will be further addressed in

comparison to path analytic findings in ASD, and in the context of their clinical applications in Chapter 11.

Chapter Nine: Barkley's Model of Behaviour Selection as Applied to Adults with Autism Spectrum Disorder

Similar to ADHD, Barkley's model (1997) of behaviour selection appears to be well suited to individuals with ASD, as it does not imply that the dysfunction of one, universal element of the behaviour selection process is responsible for all of the behavioural features associated with the disorder. Instead, Barkley's model suggests that impairments to behaviour selection are likely to be spread across several executive operations, something that is highly likely in ASD given the current literature on executive functioning in this disorder (Pennington, 2006; Willcutt et al., 2010). Nevertheless, causal paths of behaviour in adults with ASD are yet to be explored at length, and the majority of research that currently does exist on executive functioning in this disorder is largely on children and adolescents (e.g., Anderson et al., 2014; Milosavljevic et al., 2016; Trevisan, Bowering, & Birmingham, 2016).

Study 2 aimed to further investigate the role that cognitive factors such as alexithymia and executive dysfunction, and emotions such as heightened levels of anxiety and depression, play with respect to the formulation of behaviour responses typically observed in adults with this disorder. The formulation of the ASD behaviour selection model was based on Barkley's model (1997) and the premise that the executive deficits in the behaviour selection process are responsible for both the primary diagnostic features of ASD and the secondary behaviour feature of aggression. The complex network of relationships between these factors is depicted in Figure 6, with a description of the supporting research to follow.

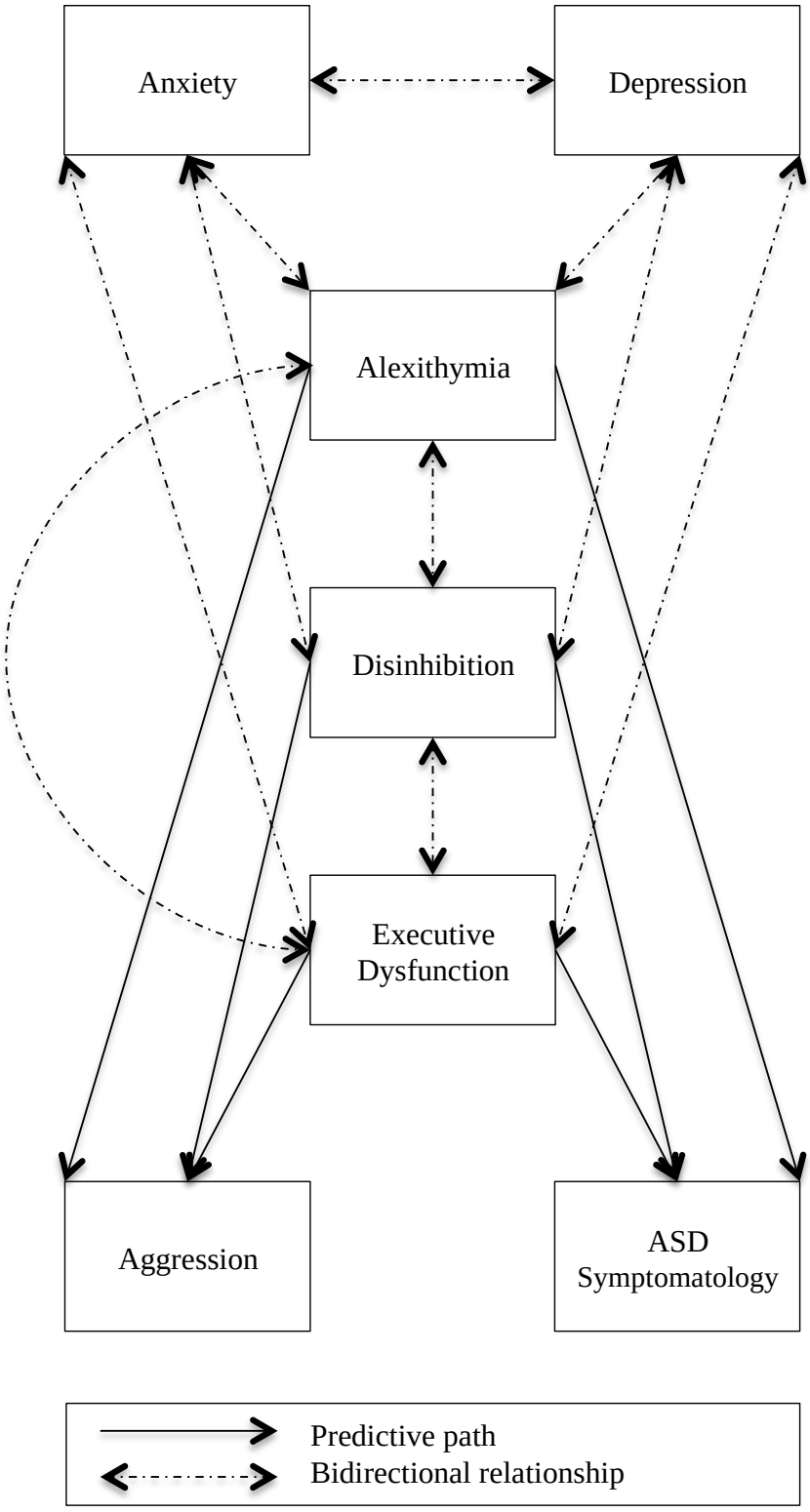

Figure 6. A proposed procedural path for behaviour selection for adults with Autism Spectrum Disorder.

Awareness and Identification of Emotions in Autism Spectrum Disorder

Barkley has emphasised the important role that emotion plays in the selection of behaviour. More specifically, he has stated that once an individual is able to acknowledge that a change in emotion has been elicited in response to a situation, self-directed action can be undertaken to suppress the initial behavioural reaction to that emotional response (Barkley, 2015e). This process appears to be hindered in adults with ASD, as they often struggle to recognise that what they are experiencing in response to a situation is in fact emotion, particularly when the associated physiological arousal is at a less detectable, albeit more manageable, threshold (Gotham, Unruh & Lord, 2014; Hill et al., 2004; Perry et al., 2001; Purkis et al., 2016a; Rieffe et al., 2007). Research suggests that adults with this disorder tend to be limited in their ability to identify and describe the personal emotions that they experience (e.g., Berthoz & Hill 2005; Hill et al. 2004).

A study by Bird and colleagues (see 2010; 2011; 2013) has revealed that, in adults with ASD, the inability to detect emotional states within the self is largely due to co-occurring alexithymia, a trait known to be highly prevalent in ASD (Berthoz & Hill 2005; Hill et al. 2004). Although alexithymia is most commonly known for the limitations it places on individuals' abilities to engage in abstract thought and identify and describe their own feelings, it has also been associated with difficulty differentiating bodily sensations from emotions (Luminet et al., 1999; Nemiah & Sifneos, 1970; Sifneos, 1973). Individuals with elevated alexithymia show a diminished ability to integrate multisensory information and are less likely to register changes in physiological arousal within the body such as an increase in heart rate (Grynberg & Pollatos 2015; Herbert, Herbert, & Pollatos, 2011; Lyvers et al. 2014). This is important to note because the sensory modulation atypicalities within alexithymia may mirror the emotional awareness difficulties that are also characteristic of this trait, implying that individuals with ASD who have heightened levels

of alexithymia may struggle to process the physiological markers of emotion and to adequately use this information to facilitate the regulation of such emotion.

In the context of Barkley's model (1997), heightened levels of alexithymia are thought to disrupt the individual's capacity to inhibit an impulsive or adverse behavioural reaction to an emotion through a lack of understanding of that initial emotional reaction. Research on alexithymia in adults with ASD supports the postulation that heightened levels of this trait are associated with increased presentation of primary ASD symptomatology (Bird et al., 2010; Samson et al., 2012; Velotti, Di Folco, & Zavattini, 2012), as well as secondary behaviour features, such as aggression (Konrath, Novin, & Li, 2012; Taylor et al., 1997). These findings provide some support for the relationships depicted in Study 2's model surrounding alexithymia and behaviour patterns in ASD. Consequently, the path analytic model developed for this study implied that the presentation of primary and secondary behaviour features of ASD was likely directly influenced by heightened levels of alexithymia.

Research exploring alexithymia in adults with ASD suggests that a relationship with both anxiety and depression. In a study on 27 adults with ASD and 27 neurotypical controls aged 18 to 64 years old, emotion regulation, alexithymia, and emotional experience were assessed via the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003), the TAS-20 (Bagby et al., 1994) and the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegan, 1988), respectively. Results showed elevated negative emotion in the ASD adults, as well as significantly higher levels of difficulty identifying and describing emotions. Additionally, adults in the ASD group demonstrated poorer emotion regulation than the neurotypical controls (Samson et al., 2012). These findings concur with other research exploring alexithymia and heightened adverse emotion in ASD (e.g., Berthoz, Lalanne, Crane, & Hill, 2013). However, these studies have not explored the relationship between

these factors; this is similar to many of the studies exploring alexithymia in ASD (e.g., Milosavljevic et al., 2016) and, subsequently, the relationship between alexithymia and heightened adverse emotion is largely assumed to be bidirectional.

The decision to include depression and anxiety in the model for Study 2 was based on research indicating these secondary feature's persistence in ASD across the lifespan (see Chapters 3 and 4) and their relationship with the other variables in the model. In ASD, although some research has failed to support a clear link between executive dysfunction and heightened adverse emotion (e.g., depression; Hollocks, Howlin, Papadopoulos, Khondoker, & Simonoff, 2014; Simonoff et al., 2012), both anxiety (Eysenck et al., 2007; Tannock, 2009; Wallace et al., 2016) and depression (Cederlund et al., 2010; Kleinhans et al., 2001; Wallace et al., 2016) have been observed to have significant, positive relationships with executive functioning deficits in adults with the disorder. Accordingly, current research on anxiety and depression in ASD does not suggest that these are causally related to the behavioural patterns common in ASD, but instead suggests that they likely impact behaviour *through* cognitive processes, such as executive functioning. Thus, similar to ADHD, it was not anticipated that negative emotion would act as a direct predictor in any of the causal pathways for behaviour selection in this model; the reciprocal relationship between executive functioning and emotion were instead reflected in the initial stages of the model.

Response Inhibition in Autism Spectrum Disorder

Barkley has proposed that the initial stages of the behaviour selection process, self-awareness and response inhibition, can be used to push past adverse interpretations of environmental situations and the emotional or physiological arousal they evoke (Barkley, 2015e). Research has indicated that a deficit in the ability to adequately identify and process initial emotional responses to situations may be linked to some of the behaviour seen in ASD. However, research on response inhibition and its impact on behaviour in this disorder is less

clear. Although some studies provide support for a link between diminished response inhibition and heightened levels of restricted and repetitive behaviour (Hill, 2004; Lopez et al., 2005; Mosconi et al., 2009; Goldberg et al., 2002; Luna et al., 2007; Manoach et al., 2004; Minshew et al., 1999; Thakkar et al., 2008), other studies have failed to consistently demonstrate that atypicalities in response inhibition even exist in ASD (Johnston et al., 2011; Kana, Keller, Minshew, & Just, 2007; Luna et al., 2007; Perry, Minassian, Lopez, Maron, & Lincoln, 2007; Raymaekers, van der Meere, & Roeyers, 2004). Consequently, the decision to include response inhibition in this study's path analytic model for ASD was heavily reliant on the behaviour selection theory established for this disorder and the importance of the suppression of initial, and often inappropriate, behavioural responses to situations (Barkley, 1997). Additionally, the significantly elevated rates of disinhibition demonstrated in the adults in the ASD group in Study 1 indicated that, despite inconsistencies in the literature on response inhibition in adults with ASD, the adults in the ASD group of this study experienced this cognitive feature in markedly higher levels than the neurotypical adults of this study. Consequently, the path analytic model developed for this study implied that the presentation of primary and secondary behavioural features of ASD was likely directly influenced by atypicalities in response inhibition.

Cognitive Reconstitution and the Working Memory System in Autism Spectrum Disorder

Barkley has posited that once an individual suppresses an initial response to a situation, he/she can incorporate data from the external environment, as well as information stored in the working memory system, to consider a range of relevant factors while formulating an adaptive behavioural response (Barkley, 1997; 2015e). Moreover, Barkley (1997) has suggested that emotion can be mitigated by redirecting focus to a positive memory or ideal future outcome within the working memory system. Nevertheless, many adults with

ASD have experienced repeated exposure to stigma and discrimination over the course of their lives as a result of their behavioural differences (Purkis et al., 2016c; 2016d) and thus, similar to ADHD, are often drawing from a memory bank filled with adverse imagery and negative self-statements (Fuster, 1997; Goldman-Rakic, 1995). The years of invalidating and traumatic experiences that often accompany ASD can result in extreme scepticism and dislike for both the self and others (Purkis et al., 2016c; 2016d). Accordingly, adults with ASD have reported engaging in significantly more frequent depressive thoughts than their neurotypical peers (Crane, Goddard, & Pring, 2013), a problem thought to be made worse by the ASD tendency to perseverate and fixate incessantly on certain thoughts (Liss et al., 2008). Barkley has posited that it is possible to access more than just negative constructs of the working memory system by way of cognitive reconstitution and, more importantly, the flexibility and fluency of the mind. But the rigid and hyper-focussed nature of the ASD cognitive style often results in a failure to consider other relevant sources of information that could otherwise refocus attention on more positive and future-oriented aspects of a situation (Frith, 1972; Hermelin & O'Connor, 1970; Hutt & Hutt, 1965; Koegel & Schreibman, 1977; Lovaas et al., 1979; Lovaas & Schreibman, 1971).

Frith (1972) postulated that, as opposed to taking a more flexible approach to challenging situations, individuals with ASD often appear to adhere to self-imposed rules and restrictions that narrow their patterns of responding. Accordingly, the cognitive rigidity common in ASD may be beneficial in areas where there are well-established rules that the individual is familiar with. Behaviour selection is, however, a process that involves constant alteration of cognitive constructs to manage situational changes and informational demands (Lazarus & Folkman, 1984), thus, the ASD tendency to be rigid in thought often presents a serious challenge in the effort to consistently meet the demands of a changing environment (Szatmari, Bremner, & Nagy, 1989). As opposed to conceptualising situations as a whole,

what tends to result from this rigid and repetitive cognitive style is the restricted processing of situational details that prove salient to the ASD individual (Wilkinson, 2015). For example, an adult with ASD may ignore social context, adhere to literal word meaning, fail to consider past events and hypothetical futures when interpreting social interactions, and restrict attentional focus to obsessional interests (Martin & McDonald, 2004). Consequently, adults with ASD often experience disjointed and narrow perspectives of the environment that fail to utilise all information required to adequately reorient focus outside of the immediate situation, formulate novel and forward-thinking solutions to problems, and mitigate emotional arousal (Adams & Sheslow, 1983; Bogte, Flamma, van der Meere, & van Engeland, 2007; Goddard, Howlin, Dritschel & Patel, 2006; Hill & Bird, 2006; Pennington & Ozonoff, 1996; Purkis et al., 2016a; Stuss, 1992; Wilkinson, 2015).

Behaviour that is selected to immediately alleviate some form of emotional adversity is typically done so in pursuit of instant relief and instant gratification (Solanto et al., 2001). In adults with ASD, the selection of such responses potentially contributes to both the primary and secondary behaviours associated with the disorder. For example, when unable to select a more appropriate means of responding to distressing events, individuals with ASD may resort to communicating through aggressive behaviours, such as verbal outbursts, self-injury or even physical violence as a way of expressing frustration and alleviating the physiological arousal that comes with it (Mazur, 1993). With respect to the disorder's primary behavioural features, ritualistic and perseverative behaviours can act to promote a sense of immediate order and control over situations that may initially present as somewhat confusing or chaotic (Joosten et al., 2009; Purkis et al., 2016a; Ridley, 1994); this can be achieved via the calming self-stimulatory benefits ritualistic and repetitive movements can provide and the inherent familiarity associated with fixating on rigid routines and circumscribed interests (Lipsky & Richards, 2009). Moreover, checking, ordering, and strict

adherence to personal rules are ritualised means by which individuals with ASD attempt to avoid chaos or disorganisation within the environment (Lewis & Bodfish, 1998; Rodgers et al., 2012). In this view, repetitive behaviours are thought to be an adaptive mechanism that limits environmental variance so that the world becomes more predictable, again providing immediate benefit to the individual. In addition to this, the social symptomatology of ASD, though seemingly less functional, can action immediate evasion of situations and interactions that do not result in personal satisfaction (Chevallier, Kohls, Troiani, Brodkin, & Schultz, 2012), and, subsequently, grant instantaneous access to a preferred object or activity (Hughes & Russell, 1993; Russell, 1997).

Research on the behaviour patterns common to ASD with respect to executive operations further supports the notion of a relationship between executive dysfunction and primary symptomatology (Frith, 1972; Hermelin & O'Connor, 1970; Hill & Bird, 2006; Hutt & Hutt, 1965; Koegel & Schreibman, 1977; Lovaas et al., 1979; Lovaas & Schreibman, 1971; Pennington, 1997; Pennington & Ozonoff, 1996; Samson et al., 2014) and secondary behaviour features, such as aggression, in this disorder (Meza, Owens, & Hinshaw, 2016). These findings provide support for the relationships depicted in Study 2's model between the cognitive and behaviour features of ASD. Consequently, the path analytic model developed for Study 2 implied that the presentation of primary and secondary behaviour features of ASD was likely directly influenced by atypicalities in executive functioning.

Study 2. Although some studies have started to explore the relationships between the primary and secondary features of ASD in the context of executive functioning and alexithymia (e.g., Berthoz, Lalanne, Crane, & Hill, 2013; Samson et al., 2012), there is little research on the relationships between emotion and the executive processes effecting behaviour selection in adults with this disorder. Instead, the primary focus of these studies tends to be the exploration of preliminary correlational associations between variables (e.g.,

Liss et al., 2008) or regression analyses that only explore certain aspects of the proposed behaviour selection model (e.g., Meza et al., 2016; Trevisan et al., 2016). Consequently, a gap exists in the empirical exploration of the distinct cognitive processes involved in Barkley's model and the effect they may have on behaviour formulation in adults with ASD.

The research on the behavioural trends of adults with ASD, though limited, does provide some evidence that atypicalities in executive functioning (Frith, 1972; Hermelin & O'Connor, 1970; Hill & Bird, 2006; Hutt & Hutt, 1965; Koegel & Schreibman, 1977; Lovaas et al., 1979; Lovaas & Schreibman, 1971; Meza et al., 2016; Pennington, 1997; Pennington & Ozonoff, 1996; Samson et al., 2014) and alexithymia (Bird et al., 2010; Samson et al., 2012; Trevisan et al., 2016; Velotti et al., 2012) are associated with the primary and secondary behavioural features of this disorder. Based on such evidence, the following hypotheses were proposed:

1. The model would significantly predict the primary and secondary behaviour features reported by adults in the ASD group.
2. The level of alexithymia reported by adults in the ASD group would act as a significant, positive predictor of ASD symptomatology.
3. The level of executive dysfunction reported by adults in the ASD group would act as a significant, positive predictor of aggression.
4. The level of executive dysfunction reported by adults in the ASD group would act as a significant, positive predictor of ASD symptomatology.

Results of the analysis of this model (Study 2), as well as a comprehensive discussion of this behaviour selection path in terms of relevant literature, are further addressed in Chapter 10. The findings of this analysis are further discussed in terms of clinical implications and directions for future research in Chapter 11.

Chapter Ten: Study 2- Path Analytic Modelling of the Primary and Secondary Features of Autism Spectrum Disorder

In order to further assess the path through which adults with ASD engage in behaviour primary and secondary to their diagnosis, Study 2 utilised all study data from the ASD participant group (N = 90); demographic information on this study group is provided in Chapter 5. Participants in the ASD study group were either assessed face-to-face or online; procedural information on both forms of assessment is also provided in Chapter 5. Relevant assessment scales for this analysis included the FrSBe (Grace & Malloy, 2001), TAS-20 (Bagby et al., 1994), BPAQ (Buss & Perry, 1992), DASS (Lovibond & Lovibond, 1995) and AQ (Baron-Cohen et al., 2001). Detailed descriptions of all five scales are located in Chapter 5 of this thesis.

Research Design

In order to examine the path through which primary and secondary features might interact in ASD based on data obtained from study participants, a path analysis was performed with executive dysfunction, disinhibition, alexithymia, anxiety and depression entered into the model as exogenous variables, and aggression and ASD symptomatology entered into the model as endogenous variables. The influence of cognitive factors (alexithymia, executive dysfunction, and disinhibition), and emotions (anxiety and depression), on ASD symptomatology and aggression were explored in the path analysis model. Preliminary analyses to examine the bivariate relationships between model variables were conducted.

Results

Statistical analysis of the data was performed using IBM SPSS AMOS version 23.0 for Windows. Prior to conducting the planned statistical analyses, the data were screened for outliers and violations to other relevant statistical assumptions.

Data screening and preparation. The one univariate outlier (on DASS anxiety) identified in Study 1 was removed prior to analysis. Mahalanobis distances ($p < .001$) demonstrated that no multivariate outliers existed within the ASD group. Based on screening analyses conducted in Study 1, all study variables appeared to meet the assumption of linearity. All variables appeared normal, with the exception of anxiety and depression, which were appropriately positively skewed (see Chapter 5 for review). Additional tests of normality demonstrated that ASD group scores on the AQ were normal.

Preliminary analyses. Descriptive statistics on the secondary features of ASD are presented in Table 10. Table 10 reports the means, standard deviations, confidence intervals (Field, 2013) and intercorrelations for the study variables entered into the ASD path analysis. As demonstrated in Table 10, correlations across the cognitive, behaviour and emotion-based features of ASD were significant and positive, with the exception of most of the correlations with ASD symptomatology (all non-significant with the exception of the significant, positive relationships with disinhibition and alexithymia). All significant, positive relationships demonstrated medium to large effect sizes (Cohen, 1988; 1992). As none of the correlations exceeded .90, the assumption of no multicolinearity was met.

Path analysis. Model fit indices are provided in Table 11. The NFI and CFI were both above the recommended cut-off of .9, suggesting the model was comparable to that of the best fit, saturated model (Hu & Bentler, 1999). The RMSEA demonstrated the model was close to a perfect fit (Browne & Cudeck, 1993; Hu & Bentler, 1999; Kline, 2011). Moreover, when all variables were entered into the path analysis, the model was not significantly different from that of the saturated model, $\chi^2(5) = 8.83$, $p = .116$, suggesting there were no significant differences between the

Table 10

Means, Standard Deviations and Intercorrelations Across the Primary and Secondary Features of ASD (N = 90)

Variable	*M*	*SD*	*1.*	*2.*	*3.*	*4.*	*5.*	*6.*	*7.*
1.Anxiety	10.41	8.22	-						
2. Depression	14.66	12.03	.67*** [.53, .77]	-					
3. Executive Dysfunction	48.10	8.80	.40*** [.21, .56]	.38*** [.19, .55]	-				
4. Disinhibition	39.05	7.95	.44*** [.25, .60]	.25* [.04, .44]	.58*** [.42, .70]	-			
5. Alexithymia	63.30	12.66	.32** [.11, .50]	.38*** [.18, .55]	.53*** [.36, .67]	.24* [.03, .43]	-		
6. Aggression	85.60	20.42	.38*** [.19, .55]	.41*** [.22, .57]	.54*** [.37, .67]	.67** [.53, .77]	.32** [.12, .50]	-	
7. ASD Symptoms	32.40	6.94	.05 [-.16, .26]	.01 [-.20, .22]	.113 [-.10, .32]	.26** [.05, .95]	.28** [.07, .46]	.18 [-.04, .37]	-

Note. 95% confidence intervals for intercorrelations are presented in parentheses. *M* = mean. *SD* = standard deviation. ADHD = Attention-Deficit Hyperactivity Disorder. ASD = Autism Spectrum Disorder. *$p < .05$. **$p < .01$. ***$p < .001$.

proposed model and a model that perfectly explains the presentation of primary symptomatology and aggressive behaviour in ASD.

Table 11

Model Fit (N = 90)

	NFI	CFI	RMSEA
Model	.96	.98	.09
Saturated Model	1.00	1.00	

Note. NFI = normed fit index. CFI = comparative fit index. RMSEA = root mean square error of approximation.

Table 12 reports the standardised and unstandardised beta weights of the individual paths within the model. Results demonstrated that disinhibition was a significant predictor of both ASD symptomatology and aggression. Results further demonstrated that alexithymia was a significant, positive predictor of ASD symptomatology, but not aggression. Executive dysfunction did not appear to significantly predict either ASD symptomatology or aggression. Figure 7 depicts the full path model, including beta weights for the model paths and correlation coefficients between study variables.

Table 12

Individual Paths within the Model for the Primary and Secondary Features of ASD (N = 90)

Path	*B*	*SE B*	*β*
Executive dysfunction → ASD Symptomatology	-0.21	.11	-.27
Executive dysfunction → Aggression	0.40	.25	.17
Disinhibition → ASD Symptomatology	0.27	.11	.31*
Disinhibition → Aggression	1.39	.24	.54***
Alexithymia → ASD Symptomatology	0.20	.07	.36**
Alexithymia → Aggression	0.15	.15	.10

Note. ASD = Autism Spectrum Disorder.

* $p < .05$. ** $p < .01$. *** $p < .001$.

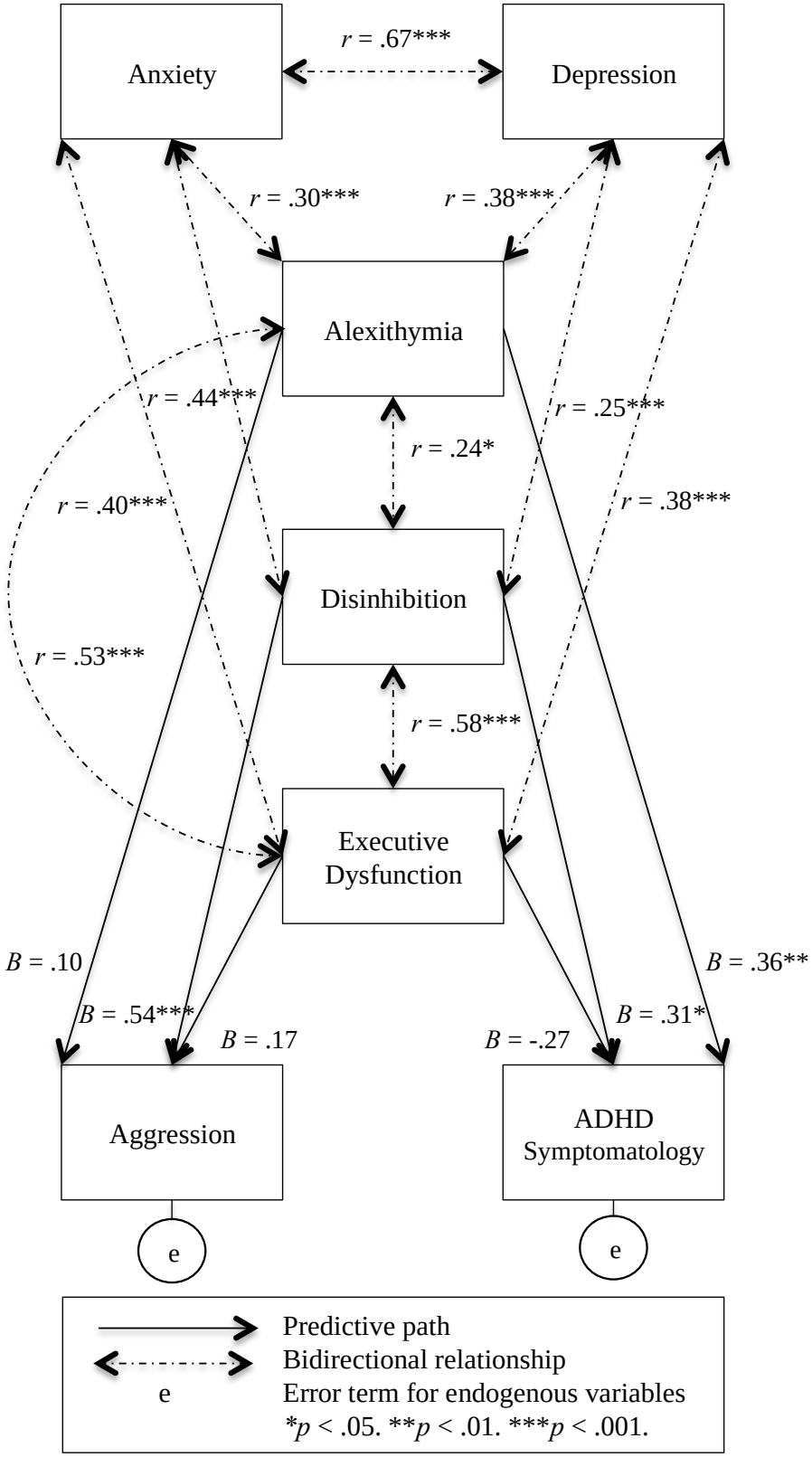

Figure 7. Path model for primary and secondary behavioural features of Autism Spectrum Disorder (*N* = 90).

Discussion

This study aimed to assess the extent to which the secondary cognitive and emotion features of ASD contributed to the presentation of primary (i.e., social-communicative and/or restricted/repetitive) and secondary (i.e., aggressive) behaviour patterns in adults with this disorder. A review of the cognitive (executive dysfunction, disinhibition, alexithymia), emotion (anxiety, depression) and behaviour (primary symptomatology, aggression) variables across study participants demonstrated that elevated levels of all features were reported in adults in the ASD group of Study 2. These results are consistent with what would be expected for individuals with a diagnosis of ASD, and are displayed in Table 10 of the Results section.

A review of the model demonstrates that it was not significantly different from a saturated model (i.e., a model that perfectly explains the primary and secondary behavioural features of ASD as predicted by executive dysfunction, disinhibition, alexithymia, anxiety, and depression). These findings are in support of the hypothesis that the model would significantly predict both the primary and secondary behavioural features of ASD. These findings also concur with Barkley's model (1997) of behaviour selection and, more specifically, the proposition that deficits in alexithymia, and the executive operations associated with it, contribute to the presentation of primary ASD symptomatology and secondary behavioural features such as aggression.

A review of the correlations between the cognitive and emotion variables entered into the model demonstrated significant, positive relationships between all variables anticipated to yield bidirectional relationships. These relationships were in accordance with research demonstrating links between the study variables (see Chapters 4 and 9 for an extensive review of this literature). These findings did not support any particular set of hypotheses pertaining to the behaviour selection model of ASD; however, they did provide support for the inclusion of the specific variables in the model, and the notion that the internal processes

involved in behaviour selection comprise a heavily integrated and complex network of covert operations (Cummings & Miller, 2007; Gazzaley & D'Esposito, 2007; Welsh & Pennington, 1988).

A review of the unidirectional paths of the model revealed that several paths successfully predicted both the primary and secondary behavioural features assessed in adults in the ASD group, however, that many of the predictive paths were not anticipated by the existing literature. In the current model, alexithymia successfully predicted ASD symptomatology. This finding supported the hypothesis that alexithymia would act as a significant predictor of the primary behavioural features of ASD, and is congruent with research supporting a link between alexithymia and ASD symptomatology (Bird et al., 2010; Samson et al., 2012; Velotti et al., 2012). Conversely, executive dysfunction did not appear to successfully predict either ASD symptomatology or aggression. Although some studies have failed to find a link between executive dysfunction and ASD symptomatology (Hollocks et al., 2014; Simonoff et al., 2012), the findings from these studies are in contrast to the majority of current research supporting a link between executive dysfunction and ASD symptomatology (Frith, 1972; Hermelin & O'Connor, 1970; Hill & Bird, 2006; Hutt & Hutt, 1965; Koegel & Schreibman, 1977; Lovaas et al., 1979; Lopez e al., 2005; Lovaas & Schreibman, 1971; Meza et al., 2016; Pennington, 1997; Pennington & Ozonoff, 1996; Samson et al., 2014), and executive dysfunction and aggression in ASD (Meza et al., 2016). In the context of Study 2, the findings fail to provide support for the hypotheses that executive dysfunction would successfully predict both ASD symptomatology and aggression in adults with ASD.

Two predictive paths not greatly anticipated by the literature were observed in Study 2. Disinhibition acted as a significant predictor of ASD symptomatology and aggression, demonstrating marked predictive utility with respect to both primary and secondary

behaviour features of this disorder in adults. Although explorations of response inhibition in relation to ASD symptomatology have yielded some evidence that deficit in this executive domain may influence the presentation of restricted and repetitive behaviour in particular (Hughes & Russell, 1993; Mosconi et al., 2009; Lopez et al., 2005; Russell, 1999), research on the persistence of disinhibition across the lifespan of individuals with ASD is relatively inconsistent. In line with the postulation that inhibitory deficits in ASD may originate from the inability to suppress behaviour that provides access to a preferred activity or topic of interest, it is possible that the cognitive rigidity considered to be characteristic of ASD may be more heavily impacted by the early stages of the behaviour selection process than originally anticipated. Hill's (2004) suggestion that disinhibition in ASD may be limited to an inability to stray from the preferred and/or familiar perhaps represents a form of disinhibition that, though seemingly minor in comparison to the more overt behavioural manifestations of disinhibition associated with disorders such as ADHD (e.g., acts of impulsivity), may have been largely overlooked in adults with ASD.

Another finding of the path analysis that contradicted current literature was the lack of predictive utility of executive dysfunction outside of disinhibition. More specifically, the measure of cognitive reconstitution (e.g., cognitive planning and flexibility) represented by the executive dysfunction variable of the model did not appear to predict primary or secondary behaviour features of adults with ASD. In the case of ASD symptomatology, this executive domain's relationship with the primary behavioural features of ASD actually appeared to be inverted, such that as executive dysfunction lessened, ASD symptomatology actually appeared to get worse; this is in contrast to research supporting the notion that ASD symptomatology is more likely to improve as impairments to executive function lessen (Frith, 1972; Hermelin & O'Connor, 1970; Hill & Bird, 2006; Hutt & Hutt, 1965; Koegel &

Schreibman, 1977; Lovaas et al., 1979; Lovaas & Schreibman, 1971; Meza et al., 2016; Pennington, 1997; Pennington & Ozonoff, 1996; Samson et al., 2014).

One possible explanation for this discrepancy is that the measure of executive dysfunction in this study did not appropriately differentiate between domains of executive dysfunction that are common and uncommon to ASD (i.e., cognitive flexibility vs. working memory) and, therefore, executive deficits that may have contributed to ASD symptomatology were subsequently "cancelled out" by strengths in other areas. The successful prediction of behaviour through alexithymia and disinhibition, however, perhaps reveals a more complex relationship between executive operations in the case of ASD, particularly in the later stages of life. In accordance with research conducted by Bird and colleagues (2010; 2013), the results of this study also potentially support the postulation that alexithymia plays a much bigger role in the presentation of ASD symptomatology than once thought, and that the impact of this atypical form of emotion processing exceeds that of atypicalities in cognitive processing (i.e., executive functioning) on behavioural responding in adults with this disorder.

The results demonstrating the non-predictive utility of executive dysfunction in this ASD sample also potentially suggest strengths within this cognitive domain. For example, these results may suggest that despite executive domains such as cognitive planning and flexibility continuing to operate in deficit in adulthood, perhaps in the later stages of life, individuals with ASD have learned to utilise such rigid thought processes to their advantage and to moderate their own primary symptoms. The cognitive rigidity often demonstrated by individuals with ASD has been implicated in efficient problem solving in areas where there are well-established rules that the ASD individual is familiar with. As such, it is possible that the atypicalties in executive functioning observed in the adults of this study with ASD may

not have been as strongly associated with heightened symptomatology as based on previous work.

Conclusion

The data yielded from the path analysis in this study provides further support for the role that both emotion and cognitive factors play with respect to the presentation of primary and secondary behavioural patterns in adults with ASD. Moreover, although not all hypotheses surrounding the model were significant (e.g., the failure of the current index of executive dysfunction to predict ASD symptomatology and aggression), the results demonstrate that, in combination with issues surrounding the recognition of specific emotions, cognitive atypicalities common to ASD (e.g., alexithymia) do yield predictive utility with respect to behaviours selected in this disorder. In the context of Barkley's model (1997) of behaviour selection, the data from this path analysis further support the importance of the early stages of the behaviour selection process with respect to not only the primary features of ASD, but secondary behavioural patterns as well (i.e., aggression). These results will be further addressed in comparison to path analytic findings in ADHD, and in the context of their clinical applications, in Chapter 11.

Chapter Eleven: General Discussion

In order to allow for a more robust conceptualisation of ADHD and ASD both clinically and functionally, Studies 1 and 2 included empirical explorations of adults with ADHD and ASD that extended beyond these disorders' primary characterisations (Grzadzinski, Huerta, & Lord, 2013). Study 1 investigated secondary features common to ADHD and ASD, with the community-based sample of adults in the ADHD and ASD groups of this study indicating significantly higher levels of all secondary features assessed in comparison to neurotypical controls. Through this exploration, Study 1 provided a good baseline of knowledge on factors that carry the potential to impact functionality in adults with ADHD and ASD, however, the study was limited in its ability to provide an in-depth understanding of how these features interact with one another and the primary features of the disorder. Accordingly, Study 2 explored the primary and secondary features of ADHD and ASD alongside one another, with the path models for each group of adults demonstrating the predictive utility of cognitive and emotional atypicalities in the presentation of primary and secondary behaviours in these disorders.

This chapter summarises and integrates the main findings of Studies 1 and 2. All major findings will first be reviewed in detail in order to communicate their contribution to the current body of literature on adults with ADHD and ASD. This chapter will then discuss the strengths and limitations of this program of research and resultant clinical implications of the study findings. Finally, this chapter will provide an overall conclusion of the research discussed.

Major Findings

Overall, the adults in the ADHD and ASD groups of Study 1 differentiated themselves from neurotypical controls across all secondary features assessed. This divergence extended to specific cognitive features not anticipated to be elevated in both

clinical groups based on a review of the existing literature. First, although it was not hypothesised, alexithymia was elevated in adults with ADHD, so much so that approximately half of all adults with this disorder received scores on the TAS-20 indicative of high alexithymia. This finding is in accordance with the limited body of research on alexithymia in ADHD (e.g., Edel et al., 2010), however, further exploration is warranted to advance understandings of how this feature presents in adults with this disorder across a range of settings and situations. Second, differences were not observed between adults in the ADHD and ASD groups with respect to disinhibition, with the adults in the ASD group even showing slightly higher scores on the disinhibition subscale of the FrSBe. Additionally, path analyses indicated that disinhibition significantly predicted not only aggression in the adults in the ASD group, but also ASD symptomatology. Although the prevalence of disinhibition has been relatively well documented in ADHD (e.g., Coccaro & Siever, 1995; Connor & Doerfler, 2008; Nigg, 2001), this was not the case for ASD. A plausible interpretation, however, is that inhibitory control over more automatic, repetitive responses in the ASD individual is impaired or diminished to some extent. The repetitive and perseverative behaviour typically observed in ASD may occur in part, as a result of the inability to suppress routine behaviour that provides access to a preferred activity or topic of interest (Hill, 2004; Russell, 1991). Such theorising may provide some explanation as to why the adults in the ASD group of this study did not report a significantly lower level of inhibitory impairment in comparison to the adults in the ADHD group and, moreover, why disinhibition appeared to play such a prominent role in the path analytic model exploring the factors involved in ASD behaviour selection.

Another issue that may account for these findings concerns methodology. First, as discussed in Chapter 10, though disinhibition was assessed by a separate subscale, the general representation of the other aspects of executive functioning obtained via the FrSBe (Grace &

Malloy, 2001) may have missed the intricacies of the ASD response patterns reflecting this cognitive feature. Future research could include a more detailed assessment of the various executive domains, employing a self-report measure such as the BRIEF to ascertain the extent to which individual executive functions contribute to the presentation of primary and secondary behaviours (Gioia et al., 2000; see Chapter 10 for further discussion). Second, the current study had enough statistical power to run the path analytic models as they were, however, should additional variables/paths be added (e.g., through use of a more detailed scale such as the BRIEF), data would need to be from a larger sample. Future studies could aim to recruit a larger sample of adults with ADHD and/or ASD so a more detailed path analysis could be conducted, such as one assessing various executive domains (e.g., cognitive flexibility, cognitive planning) in the context of specific symptom subclasses of either disorder (e.g., social-communicative symptoms in ASD; inattentive symptoms in ADHD). Third, samples of ADHD and ASD participants in this study were comprised of relatively high functioning adults limiting the capacity of the study results to be generalised to the broader populations of adults, with severely impairing developmental difficulties. Future studies could further explore secondary features in lower functioning adults with these disorders or make official diagnosis a study requirement, obtaining more detailed information on the nature of participant diagnoses and subsequent levels of impairment as a point of comparison.

Study Limitations

This research was exploratory and cross-sectional in nature, with two distinct modes of data-collection (i.e., online vs. face-to-face) via survey using six standardised self-report scales (Adler et al., 2003; Bagby et al., 1994; Baron-Cohen et al., 2001; Buss & Perry, 1992; Grace & Malloy, 2001; Lovibond & Lovibond, 1995). This data-collection process allowed for a reasonably broad and large sample of community adults to be relatively quickly and

systematically assessed. However, despite these advantages, the cross-sectional nature of this research did not provide information on any of the features assessed with respect to their presentation in the long-term. For example, assessment of anxiety and depression by way of the DASS which is essentially a screening device and only requires the informant to reflect on symptomatology present within the last seven days, providing no indication of such clinical function past this point. The study would have been improved by the application of a formal clinical assessment to ascertain details surrounding factors such as impedement to functionality, as well as past and present experiences with depression and anxiety.

In addition to limitations in data-collection, there were also challenges posed by the measurements themselves. The inclusion of only one test per psychological construct did not allow for the provision of additional information, including that of past versus current levels of relevant functioning. Moreover, the accuracy of the self-report style questionnaires was not validated by any other means in this study. Future research could further include inventories completed by a reliable informant who engages with the participant on a regular basis, such as a spouse or partner. These additional reports could provide external validation of individual levels of social and cognitive function. Additionally, they could reveal discrepancies with self-reports that further indicate prominent areas of interpersonal problems, as well as specific primary and secondary features to be targeted therapeutically (Friedman et al., 2003).

Clinical Implications

Research has demonstrated links of prominent secondary features of ADHD and ASD to functional impairments in adults with these disorders (e.g., Barkley & Murphy, 2010; Biederman et al., 1991; 1995; Langberg et al., 2008; 2011; van Steensel et al., 2011). These functional impairments make it difficult for people with these disorders to successfully interact with others and achieve key milestones (e.g., graduate from college, obtain stable

employment), particularly as they transition into adulthood (Barkley et al., 2006; Cederlund et al., 2008; Eaves & Ho, 2008; Howlin et al., 2004; Young, 2005). Accordingly, over the course of their lives, many adults with ADHD and ASD have been repeatedly subjected to expressions of disappointment from other people with respect to their behavioural or interpersonal performances across a range of settings (Murphy, 2015; Purkis et al., 2016a). The cumulative effect of these experiences can lead to despondency and demoralisation, as well as a very negative outlook on the future, and as a result, many adults with ADHD and ASD report a chronic and pervasive sense of frustration over lost opportunities and an inherent inability to yield more positive outcomes for themselves (Murphy, 2015; Cesaroni & Garber, 1991; Punshon et al., 2009). Consequently, it is important that research aimed at informing, treating and enhancing the quality of life of adults with ADHD and ASD provide further insight into all potential contributing factors of the key symptoms and subsequent functional impairments that heighten the challenges faced by people with these disorders across the lifespan.

The research conducted in Study 1 of this thesis provided evidence that features outside of the primary diagnostic characteristics of these disorders can persist into adulthood. Results of the path analyses in Study 2 further demonstrated that both the ADHD and ASD models yielded significant predictive utility with respect to the behavioural features assessed, with all bidirectional relationships between model variables demonstrating significance, and several unidirectional paths yielding predictive utility of specific behaviour in the adults in the ADHD and ASD groups of this study. Clinically, the results of Studies 1 and 2 suggest that the primary and secondary behaviour patterns commonly observed in adults with ADHD and ASD not only persist past childhood, they are most likely a result of multiple underlying factors that contain complex and unique relationships with one another. In accordance with this postulation, should therapeutic efforts fail to address even one secondary aspect prevalent

in an adult with either disorder, it is possible that primary diagnostic behaviour or other maladaptive behaviour (i.e., aggression) will continue to be selected in place of a more appropriate or adaptive response. As such, the key clinical implication of this research is that treatment of ADHD and ASD in adulthood should reflect the constellation of underlying factors that may be influencing or escalating the maladaptive behaviour patterns common to these disorders. This could require therapeutic efforts to focus on multiple features of either disorder over the course of treatment, but, at a minimum, should involve ongoing assessment of the extent to which such secondary features present throughout the therapeutic process.

Overall, these findings emphasise the importance of filling the gaps in research on features common to, and distinct from, ADHD and ASD, and potentially considerating a transdiagnostic approach to neurodevelopmental disorders. Some of the present findings would not have been obtained had ADHD or ASD been examined exclusively, yet the literature review encompassed research that was largely limited to only ADHD or ASD. For example, the literature used to justify the analyses of this study would not have necessarily indicated that an assessment of disinhibition in ASD was warranted, nor would it have implied that an assessment of alexithymia in ADHD should be conducted. These were both important factors in the path analytic models of this study, and otherwise would not have been included if they did not present as such prominent secondary features in the comparative analyses of this study. Additionally, it is likely that this study would not have applied Barkley's (1997) behavioural selection model to the path analytic model for ASD and, or at least would not have had a solid rationale to do so. Consequently, by addressing two separate samples not extensively explored together in past research, findings were yielded that carry the potential to enlighten future research and therapeutic approaches.

Conclusion

On the basis of mounting research, it is becoming increasingly evident that a comprehensive understanding of ADHD and ASD needs to encompass awareness of features outside of their primary diagnostic characteristics. As indicated by the results of this study, underlying cognitive and emotional factors need to be considered in the clinical assessment and treatment of maladaptive behaviour in adults with ADHD or ASD. A more developed understanding of the presentation and course of these features in adults with ADHD or ASD may facilitate ongoing support of people with these disorders later in life, while guiding the development of new treatment approaches and interventions. This study aimed to fill the current gaps in the research surrounding the secondary features of these disorders by directly comparing them in adults with a diagnosis of ASD or ADHD and by establishing a path model for these features in both disorders. Consequently, this study has contributed to a necessary and growing body of research that can be utilised to assist in the development of appropriate interventions for individuals with these disorders across the lifespan. By comparing and contrasting the nature in which such features present themselves in two disorders once considered so similar they could not be differentially diagnosed, further insight may be provided with respect to the ways in which these secondary features can impact the functionality of individuals with these disorders and ways in which treatment and functional improvements can be further facilitated in this context.

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Appendix A

Project title: A comparative analysis: Attention Deficit Hyperactivity Disorder and Autism Spectrum Disorder with Respect to Primary and Secondary Diagnostic Features

Project protocol number: RO1432

This research is being conducted by Ashley Stark, as a thesis project under the direct supervision of Professor Vicki Bitsika and Dr Mike Lyvers at the Centre for Autism Spectrum Disorder at Bond University. This project will be conducting research on the clinical and cognitive function of people diagnosed with an Autism Spectrum Disorder (ASD).

The purpose of this program of research is to compare and contrast, at length, ASD and Attention Deficit Hyperactivity Disorder (ADHD) in terms of clinical and cognitive functioning. By elucidating the precise nature of the relationship between these concepts in this context, clinical practice will be able to, more successfully, consider alternative diagnoses and explore potential neurological links with respect to brain function. At a theoretical level, this program of research seeks to identify the commonalities between the concepts to provide a more uniform framework for the understanding of these disorders.

We are looking for individuals (aged 18 years & over) who have a singular diagnosis of ASD or ADHD. The project involves the completion of a series of psychometric measures as part of an online or in-person questionnaire. Each measure will have a brief explanation of how to properly complete the assessment to further assist this process. It is anticipated that the completion of the measures will take 60-90 minutes. Participation in this research study is voluntary and participants are free to withdraw from the study at any time.

Please be assured that all responses will be kept confidential, and only accessed by the researchers involved in the project. The data will be securely stored at Bond University for a period of five years and will be subsequently disposed of securely.

If you have any queries or would like to be informed of the research findings, please contact the Student Researcher, Ashley Stark, via email (astark@bond.edu.au) or phone (0451 006 527).

We thank you very much for your time and consideration, and look forward to hearing from you soon.

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We are looking for individuals (aged 18 years & over) who do not have a diagnosis of ASD or ADHD. The project involves the completion of a series of psychometric measures as part of an online or in-person questionnaire. Each measure will have a brief explanation of how to properly complete the assessment to further assist this process. It is anticipated that the completion of the measures will take 60-90 minutes. Participation in this research study is voluntary and participants are free to withdraw from the study at any time.

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If you are feeling any distress upon completion of the surveys please contact either: Bond Counseling Service: 07 5595 4002 or Lifeline: 131114 or a private practitioner.

We thank you very much for your time and your participation.

I understand that I am at least 18 years old and that the purposes and procedures of this research are to my satisfaction. I understand that I am free to withdraw at any time.

Participant's signature_____ Date _____

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Appendix B

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We are looking for individuals (aged 18 years & over) who have a singular diagnosis of ASD. The project involves the completion of a series of psychometric measures as part of an online or in-person questionnaire. Each measure will have a brief explanation of how to properly complete the assessment to further assist this process. It is anticipated that the completion of the measures will take 60-90 minutes. Participation in this research study is voluntary and participants are free to withdraw from the study at any time.

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We are looking for individuals (aged 18 years & over) who have a singular diagnosis of ADHD. The project involves the completion of a series of psychometric measures as part of an online or in-person questionnaire. Each measure will have a brief explanation of how to properly complete the assessment to further assist this process. It is anticipated that the completion of the measures will take 60-90 minutes. Participation in this research study is voluntary and participants are free to withdraw from the study at any time.

Please be assured that all responses will be kept confidential, and only accessed by the researchers involved in the project. The data will be securely stored at Bond University for a period of five years and will be subsequently disposed of securely.

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This research is being conducted by Ashley Stark, as a thesis project under the direct supervision of Professor Vicki Bitsika and Dr Michael Lyvers in the Centre for Autism Spectrum Disorder at Bond University. This project will be conducting research on the functional and behavioural experiences of adults diagnosed with Autism Spectrum Disorder (ASD).

The purpose of this program of research is to compare and contrast, at length, ASD and Attention Deficit Hyperactivity Disorder (ADHD) in terms of cognitive and behavioural functioning. By elucidating the precise nature of the relationship between these concepts, clinical practice will be able to more successfully consider alternative diagnoses and explore potential neurological links with respect to brain function. At a theoretical level, this program of research seeks to explore the complex relationship between cognitive functioning, presenting behaviour, and comorbidities to provide a more uniform framework for understanding these disorders.

Your participation is voluntary and you are free to withdraw from the study at any point without penalty.

Please note, there are several questionnaires used in this study resulting in over 200 items. Each questionnaire is of its own question style and response options, so please read the instructions provided throughout to ensure you are answering each section most accurately and appropriately.

Please be assured that your participation in this study will be kept confidential, as will be any responses you give. Only the researchers involved in this study will have access to the original data. The data will be securely stored at the Centre for Autism Spectrum Disorder for a period of five years and will be subsequently disposed of securely.

If you have any queries or would like to be informed of the research findings, please contact the student researcher, Ashley Stark (astark@bond.edu.au).

If you are feeling any distress upon completion of the survey, please contact your local support service or a private practitioner.

Should you have any complaints concerning the manner in which this research is being conducted, please make contact with Bond University Human Research Ethics Committee, c/o Bond University Office of Research Services, Bond University, Gold Coast 4229 Tel: +61 7 5595 5039 Email: research@bond.edu.au

If you agree to take part in this project, please provide consent below and proceed to the survey by clicking the "Next" button at the bottom of this page.

* I acknowledge that I am at least 18 years old and that the purposes and procedures of this research are to my satisfaction. I understand that I am free to withdraw at any time.

- ◯ Yes
- ◯ No

Appendix C

Demographic Questionnaire

Gender: ☐ Male ☐ Female

Age: _____

Nationality: _____

Occupation:_____

Are you: Right handed ☐ Left handed ☐ Able to use both hands ☐

Is your hearing: Normal ☐ Corrected (e.g. cochlear implant, hearing aid, etc.) ☐
Impaired (partial or total inability to hear due to disease, disorder or injury) ☐

Is your vision: Normal ☐ Corrected (e.g. use of eye glasses, contact lenses, etc.) ☐
Impaired (poor vision even with assistance of eye glasses, contact lenses, medicine, or surgery) ☐

Relationship Status:_____

Highest level of education achieved: Primary school ☐ Secondary school ☐
TAFE/Traineeship ☐ University (undergraduate) ☐ University (postgraduate) ☐

Are you currently taking any form of medication? If yes, please specify:

_____

Have you ever received a diagnosis of the following:
Depression: Yes ☐ No ☐
Anxiety: ☐ Yes ☐ No
Autism Spectrum Disorder: Yes ☐ No ☐ Informal ☐
If yes or informal, please provide a brief description of this, including the date(s) of discussion/diagnosis and who it was with:
_____
_____
Asperger's Syndrome: Yes ☐ No ☐ Informal ☐
If yes or informal, please provide a brief description of this, including the date(s) of discussion/diagnosis and who it was with:
_____
_____
ADHD: Yes ☐ No ☐ Informal ☐
If yes or informal, please provide a brief description of this, including the date(s) of discussion/diagnosis and who it was with:
_____
_____

ADD: Yes ☐ No ☐ Informal ☐
If yes or informal, please provide a brief description of this, including the date(s) of discussion/diagnosis and who it was with:
_____
_____

Has a family member ever received a diagnosis of any of the above? If yes, please specify:
_____
_____

Appendix D

ASRS-v1.1

Please answer the questions below, rating yourself on each of the criteria shown using the scale: Never, Rarely, Sometimes, Often, Very Often in a way that best describes how you have felt and conducted yourself over the past 6 months.

1. How often do you have trouble wrapping up the final details of a project, once the challenging parts have been done?
Never **Rarely** **Sometimes** **Often** **Very Often**

2. How often do you have difficulty getting things in order when you have to do a task that requires organization?
Never **Rarely** **Sometimes** **Often** **Very Often**

3. How often do you have problems remembering appointments or obligations?
Never **Rarely** **Sometimes** **Often** **Very Often**

4. When you have a task that requires a lot of thought, how often do you avoid or delay getting started?
Never **Rarely** **Sometimes** **Often** **Very Often**

5. How often do you fidget or squirm with your hands or feet when you have to sit down for a long time?
Never **Rarely** **Sometimes** **Often** **Very Often**

6. How often do you feel overly active and compelled to do things, like you were driven by a motor?
Never **Rarely** **Sometimes** **Often** **Very Often**

7. How often do you make careless mistakes when you have to work on a boring or difficult project?
Never **Rarely** **Sometimes** **Often** **Very Often**

8. How often do you have difficulty keeping your attention when you are doing boring or repetitive work?
Never **Rarely** **Sometimes** **Often** **Very Often**

9. How often do you have difficulty concentrating on what people say to you, even when they are speaking to you directly?
Never **Rarely** **Sometimes** **Often** **Very Often**

10. How often do you misplace or have difficulty finding things at home or at work?
Never **Rarely** **Sometimes** **Often** **Very Often**

11. How often are you distracted by activity or noise around you?
Never **Rarely** **Sometimes** **Often** **Very Often**

12. How often do you leave your seat in meetings or other situations in which you are expected to remain seated?
Never **Rarely** **Sometimes** **Often** **Very Often**

13. How often do you feel restless or fidgety?
Never **Rarely** **Sometimes** **Often** **Very Often**

14. How often do you have difficulty unwinding and relaxing when you have time to yourself?
Never **Rarely** **Sometimes** **Often** **Very Often**

15. How often do you find yourself talking too much when you are in social situations?
Never **Rarely** **Sometimes** **Often** **Very Often**

16. When you're in a conversation, how often do you find yourself finishing the sentences of the people you are talking to, before they can finish them themselves?
Never **Rarely** **Sometimes** **Often** **Very Often**

17. How often do you have difficulty waiting your turn in situations when turn taking is required?
Never **Rarely** **Sometimes** **Often** **Very Often**

18. How often do you interrupt others when they are busy?
Never **Rarely** **Sometimes** **Often** **Very Often**

AQ

		Definitely agree	Slightly agree	Slightly disagree	Definitely disagree
1	I prefer to do things with others rather than on my own.	○	○	○	○
2	I prefer to do things the same way over and over again.	○	○	○	○
3	If I try to imagine something, I find it very easy to create a picture in my mind.	○	○	○	○
4	I frequently get so strongly absorbed in one thing that I lose sight of other things.	○	○	○	○
5	I often notice small sounds when others do not.	○	○	○	○
6	I usually notice car number plates or similar strings of information.	○	○	○	○
7	Other people frequently tell me that what I've said is impolite, even though I think it is polite.	○	○	○	○
8	When I'm reading a story, I can easily imagine what the characters might look like.	○	○	○	○
9	I am fascinated by dates.	○	○	○	○
10	In a social group, I can easily keep track of several different people's conversations.	○	○	○	○
11	I find social situations easy.	○	○	○	○
12	I tend to notice details that others do not.	○	○	○	○
13	I would rather go to a library than to a party.	○	○	○	○
14	I find making up stories easy.	○	○	○	○
15	I find myself drawn more strongly to people than to things.	○	○	○	○
16	I tend to have very strong interests, which I get upset about if I can't pursue.	○	○	○	○
17	I enjoy social chitchat.	○	○	○	○
18	When I talk, it isn't always easy for others to get a word in edgewise.	○	○	○	○
19	I am fascinated by numbers.	○	○	○	○
20	When I'm reading a story, I find it difficult to work out the characters' intentions.	○	○	○	○
21	I don't particularly enjoy reading fiction.	○	○	○	○
22	I find it hard to make new friends.	○	○	○	○
23	I notice patterns in things all the time.	○	○	○	○
24	I would rather go to the theater than to a museum.	○	○	○	○
25	It does not upset me if my daily routine is disturbed.	○	○	○	○
26	I frequently find that I don't know how to keep a conversation going.	○	○	○	○
27	I find it easy to 'read between the lines' when	○	○	○	○

someone is talking to me.

28 I usually concentrate more on the whole picture, rather than on the small details.

29 I am not very good at remembering phone numbers.

30 I don't usually notice small changes in a situation or a person's appearance.

31 I know how to tell if someone listening to me is getting bored.

32 I find it easy to do more than one thing at once.

33 When I talk on the phone, I'm not sure when it's my turn to speak.

34 I enjoy doing things spontaneously.

35 I am often the last to understand the point of a joke.

36 I find it easy to work out what someone is thinking or feeling just by looking at their face.

37 If there is an interruption, I can switch back to what I was doing very quickly.

38 I am good at social chitchat.

39 People often tell me that I keep going on and on about the same thing.

40 When I was young, I used to enjoy playing games involving pretending with other children.

41 I like to collect information about categories of things (e.g., types of cars, birds, trains, plants).

42 I find it difficult to imagine what it would be like to be someone else.

43 I like to carefully plan any activities I participate in.

44 I enjoy social occasions.

45 I find it difficult to work out people's intentions.

46 New situations make me anxious.

47 I enjoy meeting new people.

48 I am a good diplomat.

49 I am not very good at remembering people's date of birth.

50 I find it very easy to play games with children that involve pretending.

BPAQ

Using this 5 point scale, indicate how uncharacteristic or characteristic each of the following statements is in describing you.1 = Extremely uncharacteristic 2 = Somewhat uncharacteristic 3 = Neither uncharacteristic nor characteristic 4 = Somewhat characteristic 5 = Extremely characteristic.

	1	2	3	4	5
1. Some of my friends think I am a hothead	○	○	○	○	○
2. If I have to resort to violence to protect my rights, I will.	○	○	○	○	○
3. When people are especially nice to me, I wonder what they want.	○	○	○	○	○
4. I tell my friends openly when I disagree with them.	○	○	○	○	○
5. I have become so mad that I have broken things.	○	○	○	○	○
6. I can't help getting into arguments when people disagree with me.	○	○	○	○	○
7. I wonder why sometimes I feel so bitter about things.	○	○	○	○	○
8. Once in a while, I can't control the urge to strike another person.	○	○	○	○	○
9. I am an even-tempered person.	○	○	○	○	○
10. I am suspicious of overly friendly strangers.	○	○	○	○	○
11. I have threatened people I know.	○	○	○	○	○
12. I flare up quickly but get over it quickly.	○	○	○	○	○
13. Given enough provocation, I may hit another person.	○	○	○	○	○
14. When people annoy me, I may tell them what I think of them.	○	○	○	○	○
15. I am sometimes eaten up with jealousy.	○	○	○	○	○
16. I can think of no good reason for ever hitting a person.	○	○	○	○	○
17. At times I feel I have gotten a raw deal out of life.	○	○	○	○	○
18. I have trouble controlling my temper.	○	○	○	○	○
19. When frustrated, I let my irritation show.	○	○	○	○	○
20. I sometimes feel that people are laughing at me behind my back.	○	○	○	○	○
21. I often find myself disagreeing with people.	○	○	○	○	○
22. If somebody hits me, I hit back.	○	○	○	○	○
23. I sometimes feel like a powder keg ready to explode.	○	○	○	○	○
24. Other people always seem to get the breaks.	○	○	○	○	○
25. There are people who pushed me so far that we came to blows.	○	○	○	○	○
26. I know that "friends" talk about me behind my back.	○	○	○	○	○
27. My friends say that I'm somewhat argumentative.	○	○	○	○	○
28. Sometimes I fly off the handle for no good reason.	○	○	○	○	○
29. I get into fights a little more than the average person.	○	○	○	○	○

DASS

DASS

Please read each statement and circle a number 0, 1, 2 or 3 that indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1	I found myself getting upset by quite trivial things	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I just couldn't seem to get going	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I had a feeling of shakiness (eg, legs going to give way)	0	1	2	3
8	I found it difficult to relax	0	1	2	3
9	I found myself in situations that made me so anxious I was most relieved when they ended	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting upset rather easily	0	1	2	3
12	I felt that I was using a lot of nervous energy	0	1	2	3
13	I felt sad and depressed	0	1	2	3
14	I found myself getting impatient when I was delayed in any way (eg, elevators, traffic lights, being kept waiting)	0	1	2	3
15	I had a feeling of faintness	0	1	2	3
16	I felt that I had lost interest in just about everything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I perspired noticeably (eg, hands sweaty) in the absence of high temperatures or physical exertion	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life wasn't worthwhile	0	1	2	3

Reminder of rating scale:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

22	I found it hard to wind down	0	1	2	3
23	I had difficulty in swallowing	0	1	2	3
24	I couldn't seem to get any enjoyment out of the things I did	0	1	2	3
25	I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)	0	1	2	3
26	I felt down-hearted and blue	0	1	2	3
27	I found that I was very irritable	0	1	2	3
28	I felt I was close to panic	0	1	2	3
29	I found it hard to calm down after something upset me	0	1	2	3
30	I feared that I would be "thrown" by some trivial but unfamiliar task	0	1	2	3
31	I was unable to become enthusiastic about anything	0	1	2	3
32	I found it difficult to tolerate interruptions to what I was doing	0	1	2	3
33	I was in a state of nervous tension	0	1	2	3
34	I felt I was pretty worthless	0	1	2	3
35	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
36	I felt terrified	0	1	2	3
37	I could see nothing in the future to be hopeful about	0	1	2	3
38	I felt that life was meaningless	0	1	2	3
39	I found myself getting agitated	0	1	2	3
40	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
41	I experienced trembling (eg, in the hands)	0	1	2	3
42	I found it difficult to work up the initiative to do things	0	1	2	3

FrSBe

1 Almost never 2 Seldom 3 Sometimes 4 Frequently 5 Almost always

	Before illness or injury	At the present time
1. I speak only when spoken to.	1 2 3 4 5	1 2 3 4 5
2. I am easily angered or irritated; I have emotional outbursts without good reason.	1 2 3 4 5	1 2 3 4 5
3. Repeat certain actions or get stuck on certain ideas.	1 2 3 4 5	1 2 3 4 5
4. I do things impulsively.	1 2 3 4 5	1 2 3 4 5
5. Mix up a sequence, get confused when doing several things in a row.	1 2 3 4 5	1 2 3 4 5
6. Laugh or cry too easily.	1 2 3 4 5	1 2 3 4 5
7. Make the same mistakes over and over, do not learn from past experience.	1 2 3 4 5	1 2 3 4 5
8. Have difficulty starting an activity, lack initiative, motivation.	1 2 3 4 5	1 2 3 4 5
9. Make inappropriate sexual comments and advances, am too flirtatious.	1 2 3 4 5	1 2 3 4 5
10. Do or say embarrassing things.	1 2 3 4 5	1 2 3 4 5
11. Neglect my personal hygiene.	1 2 3 4 5	1 2 3 4 5
12. Can't sit still, am hyperactive.	1 2 3 4 5	1 2 3 4 5
13. Am unaware of my problems or when I make mistakes.	1 2 3 4 5	1 2 3 4 5
14. Sit around doing nothing.	1 2 3 4 5	1 2 3 4 5
15. Am disorganized.	1 2 3 4 5	1 2 3 4 5
16. Lose control of my urine or bowels and it doesn't seem to bother me.	1 2 3 4 5	1 2 3 4 5
17. Cannot do two things at once (for example, talk and prepare a meal).	1 2 3 4 5	1 2 3 4 5
18. Talk out of turn, interrupt others in conversations.	1 2 3 4 5	1 2 3 4 5
19. Show poor judgment, poor problem solver.	1 2 3 4 5	1 2 3 4 5
20. Make up fantastic stories when unable to remember something.	1 2 3 4 5	1 2 3 4 5
21. Have lost interest in things that used to be fun or important to me.	1 2 3 4 5	1 2 3 4 5
22. Say one thing, then do another thing.	1 2 3 4 5	1 2 3 4 5
23. Start things but fail to finish them, "peter out."	1 2 3 4 5	1 2 3 4 5
24. Show little emotion, am unconcerned and unresponsive.	1 2 3 4 5	1 2 3 4 5

1 Almost never	2 Seldom	3 Sometimes	4 Frequently	5 Almost always	Before illness or injury	At the present time
25. Forget to do things but then remember when prompted or when it is too late.					1 2 3 4 5	1 2 3 4 5
26. Am inflexible, unable to change routines.					1 2 3 4 5	1 2 3 4 5
27. Get in trouble with the law or authorities.					1 2 3 4 5	1 2 3 4 5
28. Do risky things just for the heck of it.					1 2 3 4 5	1 2 3 4 5
29. Am slow moving, lack energy, inactive.					1 2 3 4 5	1 2 3 4 5
30. Am overly silly, have a childish sense of humor.					1 2 3 4 5	1 2 3 4 5
31. Find that food has no taste or smell.					1 2 3 4 5	1 2 3 4 5
32. Swear.					1 2 3 4 5	1 2 3 4 5
Read each of the following items carefully before responding.						
33. Apologize for misbehavior (for example, apologize for swearing).					1 2 3 4 5	1 2 3 4 5
34. Pay attention, concentrate even when there are distractions.					1 2 3 4 5	1 2 3 4 5
35. Think things through before acting (for example, consider finances before spending money).					1 2 3 4 5	1 2 3 4 5
36. Use strategies to remember important things (for example, write notes to myself).					1 2 3 4 5	1 2 3 4 5
37. Am able to plan ahead.					1 2 3 4 5	1 2 3 4 5
38. Am interested in sex.					1 2 3 4 5	1 2 3 4 5
39. Care about my appearance (for example, daily grooming).					1 2 3 4 5	1 2 3 4 5
40. Benefit from feedback, accept constructive criticism from others.					1 2 3 4 5	1 2 3 4 5
41. Get involved with activities spontaneously (such as hobbies).					1 2 3 4 5	1 2 3 4 5
42. Do things without being requested to do so.					1 2 3 4 5	1 2 3 4 5
43. Am sensitive to the needs of other people.					1 2 3 4 5	1 2 3 4 5
44. Get along well with others.					1 2 3 4 5	1 2 3 4 5
45. Act appropriately for my age.					1 2 3 4 5	1 2 3 4 5
46. Can start conversations easily.					1 2 3 4 5	1 2 3 4 5

TAS-20

Using the scale provided as a guide, indicate how much you agree or disagree with each of the following statements by circling the corresponding number. Give only one answer for each statement.

1 = STRONGLY DISAGREE
2 = MODERATELY DISAGREE
3 =NEITHER DISAGREE NOR AGREE
4 = MODERATELY AGREE
5 = STRONGLY AGREE

		Strongly disagree	Moderately disagree	Neither disagree nor agree	Moderately agree	Strongly agree
1	I am often confused about what emotion I am feeling	1	2	3	4	5
2	It is difficult for me to find the right words for my feelings	1	2	3	4	5
3	I have physical sensations that even doctors don't understand	1	2	3	4	5
4	I am able to describe my feelings easily	1	2	3	4	5
5	I prefer to analyze problems rather than just describe them	1	2	3	4	5
6	When I am upset, I don't know if I am sad, frightened, or angry	1	2	3	4	5
7	I am often puzzled by sensations in my body	1	2	3	4	5
8	I prefer to just let things happen rather than to understand why they turned out that way	1	2	3	4	5
9	I have feelings that I can't quite identify	1	2	3	4	5
10	Being in touch with emotions is essential	1 Strongly disagree	2 Moderately disagree	3 Neither agree nor disagree	4 Moderately agree	5 Strongly agree

11	I find it hard to describe how I feel about people	1	2	3	4	5
12	People tell me to describe my feelings more	1	2	3	4	5
13	I don't know what's going on inside me	1	2	3	4	5
14	I often don't know why I am angry	1	2	3	4	5
15	I prefer talking to people about their daily activities rather than their feelings	1	2	3	4	5
16	I prefer to watch 'light' entertainment shows rather than psychological dramas	1	2	3	4	5
17	It is difficult for me to reveal my innermost feelings, even to close friends	1	2	3	4	5
18	I can feel close to someone, even in moments of silence	1	2	3	4	5
19	I find examination of my feelings useful in solving personal problems	1	2	3	4	5
20	Looking for hidden meanings in movies or plays distracts from their enjoyment	1	2	3	4	5