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Adult attention deficit disorder and aggressive behaviour: an exploration of relationships between Brown Attention-Deficit Disorder Scales and the Aggression Questionnaire

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Abstract

Aggressive and violent behaviour is often associated with a diagnosis of attention-deficit hyperactivity disorder (ADHD). This article investigates the relationship between adult attention deficit disorder (ADD) and aggressive cognitions. The Aggression Questionnaire and the Brown Attention-Deficit Disorder Scales were administered to two samples: a "low-risk sample" comprising university students (n = 60), and a "high-risk sample" of prison inmates (n = 117). The prevalence of "probable ADD" was found to be higher in the prison sample (33%), compared with the university sample (20%). There were moderate correlations >r = .5 ($p < .01$) between total ADHD and Aggression Questionnaire scores in both groups. There were also moderate to strong correlations between subscales of both measures, including cognitive processes such as attention and memory and various forms of aggression, which were particularly apparent in the prison sample. The relationship between ADHD and aggressive behaviour suggests that the treatment of adult ADHD may aid in the management of aggressive behaviour.

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Abstract

Aggressive and violent behaviour is often associated with a diagnosis of AD/HD. This paper investigates the relationship between adult Attention Deficit Disorder and aggressive cognitions. Two samples were administered the AQ and the BADDS: a 'low risk sample' comprising university students (n=60), and a 'high risk sample' of prison inmates (n=117). The prevalence of 'probable ADD' was found to be higher in the prison sample (33%), compared to the University sample (20%). There were moderate correlations >r=0.5 (p<0.01) between total AD/HD and AQ scores in both groups. There were also moderate to strong correlations between subscales of both measures, including cognitive processes such as attention and memory and various forms of aggression, which were particularly apparent in the prison sample. The relationship between AD/HD and aggressive behaviour suggests that the treatment of adult AD/HD may aid in the management of aggressive behaviour.

Key Words: Attention Deficit Disorder, Aggression, Prisoners, Treatment

Introduction

Irrespective of comparative rates of crime typology, aggression and violent crime is foremost among the public's perception of crime in general, and of community appraisal of 'society's malaise' (Howitt, 2009). Aggression is a leading public health problem worldwide (Krug et al., 2002). Empirical research into the causes of aggressive behaviour has raised substantial conceptual issues, including the need to differentiate between the behavioural expression of aggression (physical or verbal violence) and the cognitive processes underlying such behaviours, such as hostility and attributional bias (Suris et al., 2004). To this must be added the distinction between reactive, 'anger induced' or impulsive aggression (Dowson & Blackwell, 2010) and instrumental aggression, where the violent act is a purposeful behaviour engaged in pursuit of a predetermined goal (Vitiello et al., 1990). Anger and impulsiveness tend to correlate highly with reactive aggression, but not so with instrumental aggression (Ramirez & Andreu, 2006).

All acts of violence and aggression occur within a context that includes the environment, the victim and the perpetrator. General models and theories of aggression have emerged, the most contemporaneous being the General Aggression Model (GAM: Anderson & Bushman, 2002). According to the GAM, aggressive acts occur when personal predisposing characteristics of the perpetrator converge with precipitating situational variables. Multiple experiences prepare the perpetrator to behave aggressively in varying situations and enable the development of pro-aggressive cognitive schema that are repeatedly accessed in new situations (Collie, Vess & Murdoch, 2007). Access to such schema is influenced by arousal, principally anger (Gilbert & Daffern, 2011), while access to alternative schema, (and hence response pathways), is inhibited by inattention to stimuli inconsistent with the aggressive cognitive schema. The GAM explains, in part, how individual differences, such as cognitive processing variables, contribute to the likelihood of aggressive thoughts and behaviours.

One such contributing variable is the presence or absence of neurodevelopmental disorders, such as attention-deficit hyperactivity disorder (AD/HD). AD/HD is a significant contributor to childhood learning and behavioural difficulties, affecting about 5% of school aged children and 2.5% of adults (APA, 2013). AD/HD is generally persistent from childhood into adulthood (Carlotta et al., 2013; Biederman et al., 2010) and has been associated with aggressive behaviour early in childhood (Kakouros, Maniadaki & Karaba, 2014) as well as with adult difficulties with addictions and with propensity for criminal activity (Johnstone et al., 2010; Barkley, Murphy & Fischer, 2008). This is supported by studies around the world showing 3-15 times greater prevalence rates of AD/HD in prison populations than in the general population (Ginsbert et al., 2010, Roesler et al., 2004, Vernieren et al., 2003).

There is substantial neuropsychiatric evidence that aggressive and antisocial behaviour is related to frontal executive dysfunction (Brower & Price, 2001). Disinhibition theories of AD/HD would suggest that deficiencies in restraining inappropriate responding are related to executive function deficits, including the ability to cognitively process information in working memory so as to generate an appropriate response (Martinussen et al., 2005; Brassett-Harknett & Butler, 2007). Further to this, models of attention deficit disorders without hyperactivity have suggested that the core symptoms of such disorders, especially for adults, are essentially cognitive, irrespective of the presence of impulsivity (Brown, 1996). There is evidence that rates of criminality among people with AD/HD are significantly lower when receiving pharmacotherapy treatment compared to periods without AD/HD treatment (Lichtenstein et al., 2012), and this may be related to reported improvements in working memory subsequent to the administration of methylphenidate (Mehta et al., 2000).

Aggression is common in many disorders co-morbid with AD/HD, in particular personality disorders (Dowson, 2006), and it has been argued that the best response to aggression in the context of AD/HD is to treat the comorbid disorders (Gonzalez, Kallis & Coid, 2013).

However, there is a substantial body of research indicating that impulsive aggression, such as being ‘hot tempered’ or having ‘a short fuse’, should be included in the AD/HD syndrome for adult males (Dowson & Blackwell, 2010; Wender, Wolf & Wasserstein, 2001). This is supported by studies which identify phenotypic and genetic associations between AD/HD and emotional lability, including irritability, low frustration tolerance, temper outbursts and mood volatility (Merwood et al., 2014). Emotional regulation, particularly the management of frustration and anger, is often also impaired (Wolraich et al., 2005).

Given the impact of aggressive behaviour and the potential contribution of AD/HD to our understanding of why some people act aggressively, with cognisance that treating AD/HD can reduce the likelihood of aggressive behaviour, it is useful to investigate the convergence or otherwise of measures of aggression and measures of AD/HD. Measures of aggression vary greatly according to the conceptual framework upon which they have been developed, and there has been some confusion in the literature due to the tendency to use terms such as ‘aggression’, ‘anger’, and ‘hostility’ interchangeably (Suris et al, 2004). Furthermore, measurement issues vary from test to test, including not only how aggression has been defined, but also the method of data collection (such as self-report, observation, analogue or laboratory based studies versus field studies), and the period of time over which the data are collected (Suris et al., 2004). A key issue in the measurement of anger and aggression is whether they are measured as a ‘state’ (related to the person’s response to transient or current circumstances) or a ‘trait’ (related to characterological features of the persons’ general response style: Suris et al., 2004; Ramirez & Andreu, 2006). In the context of AD/HD, which, as a neurodevelopmental disorder would be considered as a trait aspect of an individual’s responding, measurement of aggression utilising a trait based conceptualisation seems appropriate.

While the association between AD/HD and aggression in childhood has been well documented in the literature (Fite et al., 2007), there are few studies investigating such relationships in adults with AD/HD and this would seem particularly pertinent in prison inmates. One exception is the recent investigation into the relationship between AD/HD, trait aggression and drug use (Bacskai, Czobor & Gerevich, 2012). Bacskai and colleagues administered the Buss-Perry Aggression Questionnaire to 150 drug dependent outpatients, who were also assessed for the presence or absence of AD/HD using the Adult Self-Report Scale; a screening test for the presence of symptoms of AD/HD. Results indicated that those with AD/HD symptoms scored significantly higher on measures of trait aggression when compared with those who demonstrated few symptoms of AD/HD. The idea that trait aggression may predict adult AD/HD was further explored by Carlotta and his colleagues (Carlotta et al., 2013). While the principle investigation was to explore diagnostic overlap between Borderline Personality Disorder and AD/HD, Carlotta et al., (2013) reported that self-report retrospective assessment of AD/HD on the Wender Utah Rating Scale (WURS: Ward, Wender & Reimherr, 1993) significantly predicted total scores on the Aggression Questionnaire.

The purported link between AD/HD and trait aggression may be elucidated further through examination of current symptoms of AD/HD (as opposed to retrospective assessment) and current reports of trait aggression. This study sought to explore the extent to which two established measures of these variables correlated in both a low risk (university student) and a high risk (prison inmate) sample; hence two related but separate studies were conducted. If, as proposed by previous research, trait aggression is a facet of AD/HD, we would expect significant correlations across measures in both subject samples. The low risk sample included both males and females, while only male participants were included in the high risk sample.

Methods

Participants

Ethics approval for both studies was provided by the Human Research Ethics Committee of the University of Wollongong. Participants for the 'low risk' sample comprised a convenience sample of Australian undergraduate university students, recruited through the student research participation scheme which comprises an aspect of their undergraduate curriculum. Clinically significant symptoms of AD/HD have been reported as affecting between 2% and 8% of college/university students, with implications not only for their educational achievement, but also with respect to social and behavioural problems (Anastopoulos & King, *in press*). The university student sample was administered a brief demographic questionnaire, then asked to complete the aggression and attention deficit measures. There was no prompting and measures were administered in random order to counteract priming effects. After completion of the study, students were debriefed if they elected to receive feedback.

Participants in the 'high risk' sample were 117 inmates of a mixed security-level prison in New South Wales. Participants included both high and medium security classifications, and were recruited as part of a larger study into the effects of omega 3 and micronutrient supplementation on within-prison behaviour (Meyer et al., 2014). Participation was voluntary, however a small financial incentive was provided to encourage inmate engagement. Prisoners were assigned a code that de-identified their data to attenuate concerns about institutional use of information and as part of the over-all double-blinding associated with the larger study. Demographic and research questionnaires were administered in a group room on the prison site and where necessary, some assistance in reading and completing

questionnaires was provided. Order of aggression and attention deficit questionnaires was randomised as per the university student population.

Measures

Trait Aggression

Trait aggression was measured using the Aggression Questionnaire (AQ: Buss & Perry, 1992). The AQ is among the most widely researched self-report trait based measures of aggression, anger and hostility (Ramirez & Andreu, 2006; Harris, 1997; Harris, 1994). Furthermore, the AQ has been found to be reasonably robust with respect to social desirability (Becker, 2007), an issue some have argued limits the reliability of self-report measures of aggression (Suris et al., 2004). Additionally, the AQ has been subjected to broad cross-cultural validation and is applicable to non-Anglo-Saxon samples (Ramirez & Andreu, 2006).

The AQ comprises 29 items, each associated with a 5-point Likert scale, ranging from 1 ('extremely uncharacteristic of me') through to 5 ('extremely characteristic of me'). The AQ yields five subscale scores: Physical Aggression (PA); Verbal Aggression (VA); Anger (AN); Hostility (HS); and Indirect Aggression (IND). The AQ also yields a composite total score.

AD/HD

Selection of an appropriate adult measure of the AD/HD variable was influenced by the debate surrounding whether impulsivity or attentional deficit contributes foremost to aggressive behaviour. Recent research by Garcia-Forero and his colleagues (Garcia-Forero et al., 2008) suggests that impulsivity and aggressiveness are separate concepts and that to confuse the two has resulted in misconceptions in the literature. In keeping with AD/HD theories that promote the importance of dysexecutive syndromes, and in particular, working

memory capacity (Burgess et al., 2010), and with the observation that improved attention is associated with increased behavioural control in children with AD/HD (Johnstone et al., 2012), this study chose to focus on the attentional deficits associated with an AD/HD diagnosis.

Attention-deficit was assessed using the Brown Attention-Deficit Disorder Scales (BADDS: Brown, 1996). The BADDS is a 40-item self-report scale that measures a range of symptoms associated with inattention, but does not assess either hyperactivity or impulsivity. Compared to other rating instruments, the BADDS is effective at predicting clinical diagnosis (Kooij et al., 2008). Items are rated on a scale of 0 (never) through to 3 (almost daily) and combine to provide five subscales relating to executive function impairment: Activation (organizing, prioritizing, and activating work); Focus/Attention (focussing, sustaining, and shifting attention to tasks); Effort (regulating alertness, sustaining effort, and processing speed); Emotion/Affect (managing frustration and modulating emotions); and Memory (utilizing working memory and accessing recall). BADDS scores can range from 0 to 120, with increasing scores indicating more impairment. The clinical cut off score in adults is a BADDS total score of 50, producing 4% false negatives and 6% false positives (Brown et al., 2011).

Results

Study 1: A total of 60 undergraduate university students aged aged 17-59 (M=21.8 SD=7.2) participated (26.7% male). The mean total AQ t-score was 51.7 (*SD*=9.97) and the mean total BADDS score t-score was 58.5 (SD=10.16). Twenty percent of the sample (*n*=12) had a raw total BADDS score > 55, indicative of 'highly probable' ADD (Brown, 1996). Stratification of the sample by age and gender yielded no significant differences for either total or subscale scores. Shapiro-Wilk assessment indicated that neither the BADDS nor the AQ distributions

were normally distributed and thus non-parametric analysis using Spearmans rho was undertaken. Total t-scores for the AQ and the BADDS evidenced a moderate correlation of r=0.57 (p<0.01). Correlations between subscale t-scores (age and gender adjusted) are presented in Table 1.

Insert Table 1. About Here

Study 2: A total of 117 male prison inmates aged 18-79 (M=34.4, SD=11.22) completed the questionnaires. The mean t-score for total AQ was 55.4 (SD=10.31) and for BADDS was 78.2 (SD=9.30). Thirty nine participants had a raw total BADDS score over 55, suggesting that 33% of the sample have 'highly probable' ADD (Brown, 1996). Shapiro-Wilk assessment again showed that the BADDS and AQ subscales were not normally distributed so Spearmans rank order correlation was used. The correlation between total AQ and BADDS was again moderate (r=0.56, p<.01). Correlations between subscale t-scores (age adjusted) are presented in Table 2.

Insert Table 2. About Here

Discussion

This study examined the relationship between the AQ, a measure of trait aggression, and the BADDS, a measure of attention deficit disorder. Two samples were selected, broadly representing a 'normal' and a 'pathological' cohort. As hypothesised, trait aggression was associated with attention deficit in both samples, with the prison sample displaying the strongest relationships. While the pattern of relationships within subscales of the two measures was similar across the samples, age-adjusted t-scores indicated that neither memory nor attention remained significantly associated with physical and verbal aggression for the

University sample. However, the same adjustment did not change the significance of these relationships for the prison sample.

Within the subscale analyses, the strength of the correlations supported the contention that deficits in cognitive processes associated with AD/HD potentiate trait aggression; specifically, the BADDS subscales of “Attention’ and “Affect”. “Attention” related to the individual’s ability to sustain focus and behaviours which appear impulsive may indeed reflect a failure to adequately attend to salient social cues. In this study, the moderately strong correlations between both “Anger” and “Hostility” with “Attention” support such a proposition. Similarly, the ‘Affect’ subscale of the BADDS, which reflects difficulties managing frustration and emotion, correlated significantly with both “Anger” and “Hostility” subscales of the AQ. This suggests that individuals with AD/HD who also evidence trait aggression are likely to experience difficulty containing their hostile and angry reactions, as well as experiencing difficulties generating socially adaptive behaviours and executing skills to avoid aggressive responses (Fishbein et al., 2009).

While trait aggression is not caused by attention deficits, our data would suggest that the attention deficit component of AD/HD is an important mediating variable to an individual acting upon such dispositions. Research indicating a reduction in criminal behaviour and antisociality following effective treatment for AD/HD (for example, Lichtenstein et al., 2012) is consistent with such an interpretation. However, while treatment of AD/HD using stimulant medications have been shown to effectively ameliorate inattentive and impulsive behaviours (Clarke et al., 2007), as well as the frequency of reactive aggression (Faraone & Upadhyaya, 2007), the potential iatrogenic impact of the use of ongoing prescription medications (Graham & Coghill, 2008) has prompted investigations into alternative and less intrusive therapies (Johnstone et al., 2010). This has included efforts to enhance executive functioning through computer-assisted working memory training (Klingberg et al., 2005), on

the basis that improved executive functioning will enhance self-control skills (Geurts et al., 2005). Studies aimed at non-medication based interventions to improve cognitive function have demonstrated improved attention and increased behavioural control in children with AD/HD (Johnstone et al., 2012).

Recent research has also demonstrated that diet and dietary supplements can ameliorate symptoms of AD/HD (Sinn, 2008a). Furthermore, Sinn and her colleagues found improved parent-reported symptoms in hyperactivity, impulsivity and inattention following supplementation with omega-3 fatty acids and parent-reported improvements were mediated by improvements in cognitive tests, including a test of attention control (Sinn et al., 2008b). This provides theoretical support for cognitive underpinnings of impulsivity and hyperactivity – and possibly by extension control of emotions like anger. Furthermore, both Gesch et al., (2002) and Zaalberg et al., (2010) reported a reduction of antisocial behaviour among prison inmates following nutritional supplementation. While neither Gesch nor Zaalberg related their results to improvements in executive functioning, it is plausible that the therapeutic benefit of nutritional supplementation on AD/HD symptoms was causal in their results, and is consistent with our findings.

This study had several limitations which limit the generalizability of our results. First, both the AQ and the BADDS are self-report measures and the propensity for positive self-promotion among respondents is a tangible threat to the validity of their responses. However, while this may result in an underestimation of the total scores, the relationship between the measures would not be affected by a uniform positive impression management strategy. Nevertheless, future research should incorporate an objective measure of behaviour and relate this back to the BADDS and AQ. Secondly, the prison sample included only male inmates and the generalizability to female inmates is uncertain. However, the study among university students, who were predominantly female, evidenced similar correlations suggesting that

gender may not be an important covariate. Third, this was a correlational study only, confounded by the diagnostic relevance of aggressive behaviour in current conceptualisations of AD/HD. That is, if aggressive behaviour is a relevant diagnostic feature of AD/HD as some suggest (Dowson & Blackwell, 2010; Wender, Wolf & Wasserstein, 2001), then observing high trait aggression among those with AD/HD is of diagnostic not causal significance. Future studies should determine if the relationship between trait aggression and Attention Deficit Disorder holds when the symptoms of Attention Deficit Disorder are effectively treated. If trait aggression remains but is not behaviourally acted upon, then the mediating role of Attention Deficit Disorder in antisocial behaviour becomes more plausible. Should that be the case, management and treatment of aggressive behaviours will necessarily involve strategies for the treatment of Attention Deficit Disorder where such a diagnosis has been made.

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Table 1. Spearman's correlations between sub-scale t-scores of AQ and BADDS for university sample

Factor	Physical	Verbal	Anger	Hostility	Indirect	Activation	Attention	Effort	Affect	Memory
Physical	1.00	0.480**	0.563**	0.363**	0.426**	0.292*	0.170	0.370**	0.213	0.092
Verbal		1.00	0.629**	0.524**	0.465**	0.378**	0.292*	0.398**	0.362**	0.266
Anger			1.00	0.631**	0.602**	0.396**	0.353**	0.511**	0.535**	0.345*
Hostility				1.00	0.663**	0.606**	0.525**	0.583**	0.575**	0.271*
Indirect					1.00	0.409**	0.455**	0.525**	0.490**	0.366**
Activation						1.00	0.623**	0.724**	0.670**	0.417**
Attention							1.00	0.646**	0.476**	0.606**
Effort								1.00	0.632**	0.599**
Affect									1.00	0.387**
Memory										1.00

** $P<0.01$

* $P<0.05$

Table 2. Spearman's correlations between sub-scale t-scores (age adjusted) of AQ and BADDS for prison sample

Factor	Physical	Verbal	Anger	Hostility	Indirect	Activation	Attention	Effort	Affect	Memory
Physical	1.00	0.667**	0.732**	0.487**	0.649**	0.354**	0.362**	0.299**	0.327**	0.303**
Verbal		1.00	0.602**	0.506**	0.615**	0.289**	0.238*	0.250**	0.341**	0.219*
Anger			1.00	0.631**	0.666**	0.469**	0.438**	0.434**	0.541**	0.365**
Hostility				1.00	0.691**	0.626**	0.576**	0.606**	0.717**	0.589**
Indirect					1.00	0.518**	0.545**	0.492**	0.511**	0.509**
Activation						1.00	0.808**	0.847**	0.731**	0.741**
Attention							1.00	0.757**	0.684**	0.771**
Effort								1.00	0.725**	0.742**
Affect									1.00	0.610**
Memory										1.00

** $P<0.01$

* $P<0.05$