

Gray's revised Reinforcement Sensitivity Theory in relation to Attention-Deficit/Hyperactivity and Tourette-like behaviors in the general population

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Abstract

Attention-Deficit/Hyperactivity Disorder (ADHD) and Tourette Syndrome (TS) present as distinct conditions clinically; however, they show comorbidity and inhibitory control deficits have been proposed to underlie both. The role of reinforcement sensitivity in ADHD has been studied previously, but no study has addressed this in relation to TS-like behaviors in the general population. The present study examined these associations within the remit of the revised Reinforcement Sensitivity Theory (rRST). One hundred and thirty-eight participants completed psychometric measures of the rRST, and self-report checklists for ADHD- and TS-like behaviors. The results show that whilst ADHD-inattention was only linked to increased anxiety (BIS), ADHD-hyperactivity/impulsivity was linked to increased impulsivity (BAS-fun seeking), anxiety (BIS) and punishment sensitivity (FFFS), and to reduced reward sensitivity (BAS-reward responsiveness), independently of 'comorbid' TS-like behaviors. TS-related phonic tics were associated with increased BIS and FFFS, and TS-related obsessive-compulsive behaviors (OCBs) with increased goal-orientation (BAS-drive) and reduced impulsivity (BAS-fun seeking). However, these associations were driven by ADHD-like behaviors or OCB co-occurrence, respectively, suggesting little role of the rRST in pure TS-like behaviors. The results are discussed in light of mixed findings in the literature and the importance of distinguishing between multiple processing models of the rRST in distinct disorder phenotypes.

Keywords: ADHD; Tourette syndrome; reinforcement sensitivity, BIS/BAS

1. Introduction

It has been suggested that the symptoms of Attention-Deficit/Hyperactivity Disorder (ADHD) derive from a primary executive inhibitory control deficit (Barkley, 1997) though motivational inhibitory deficits have also been proposed (e.g., Newman & Wallace, 1993; Quay, 1997; Nigg 2000). There is a high comorbidity of ADHD in Tourette syndrome (TS) whereby up to 80% of TS patients also exhibit symptoms of ADHD, and these appear to precede the emergence of TS associated tics (Cavanna & Rickards, 2013). Similar to ADHD, it is argued that TS is the result of an inhibitory dysfunction (Sheppard, Bradshaw, Purcell & Pantelis, 1999), though the overall evidence is inconsistent, possibly due to varying levels of comorbidity with ADHD (Pennington & Ozonoff, 1996). Indeed, pure TS may rather be characterized by enhanced executive control (Jackson et al., 2007; Jung et al., 2014) and there is no evidence for automatic inhibitory deficits in TS patients without comorbidity (Ozonoff et al., 1998; Yuen et al., 2005) and independent of medication effects (Kantini et al., 2011). Similar findings were recently shown in relation to TS-like behaviors in the general population when ADHD was controlled for (Heym, Kantini, Checkley & Cassaday, 2014). These findings suggest that TS does not occur in conjunction with deficits in effortful or automatic associative response inhibition. Recently, the application of reinforcement learning models has been proposed to further our understanding of the processes involved in complex symptom patterns in psychiatric and neurological disorders (Bijttebier et al., 2009; Maia & Frank, 2011). Although primarily a motor-disorder, the involvement of fronto-striatal dopaminergic pathways and basal ganglia circuitry in the etiology of TS (Robertson, 2000) and the central role of these pathways in reinforcement learning suggests a role for reinforcement sensitivity and motivational inhibitory processes in this disorder (Maia & Frank, 2011 for review).

1.1. Reinforcement Sensitivity Theory (RST)

Gray's (1982) original model proposed three neuropsychological systems underpinning approach-avoidance motivation and behavior - the functioning of which was related to personality. Recent revisions to the theory (rRST; Gray & McNaughton, 2000) led to some changes in the conceptualization of the systems involved (Pickering & Corr, 2008). In the rRST, the behavioral approach system (BAS) is a reward-sensitive system - activation leads to goal-oriented approach behavior. BAS is linked to trait impulsivity. The Fight-Flight-Freeze System (FFFS) is a punishment-sensitive system - activation leads to active avoidance behavior, and it is the causal basis of fear. The behavioral inhibition system (BIS) responds to conflicting (aversive and/or appetitive) cues leading to inhibition of the ongoing response, risk assessment and appraisal. BIS is linked to trait anxiety and worry. The main changes in the revision are that punishment sensitivity, originally ascribed to BIS, is now defined by the FFFS, whereas BIS is responsible for resolving goal conflicts. Dysfunctions in these systems have been proposed to lead to various clinical outcomes; for instance, overactivity of the checking mode of the BIS relates to symptoms of Obsessive Compulsive Disorder (OCD), general anxiety and related internalizing disorders, whereas overactivity of BAS relates to externalizing disorders (Gray, 1982; Gray & McNaughton, 2000).

1.2. Reinforcement sensitivity in ADHD and TS

ADHD is an externalizing disorder, and as such, an overactive BAS leading to response modulation deficits has been proposed to underlie ADHD (Newman & Wallace, 1993).

Alternative models have proposed an underactive BIS (Quay, 1988), or an interaction between high levels of BAS relative to low levels of BIS, to be responsible for the inhibition deficits seen in ADHD (Quay, 1997). Experimental studies support the notion of dysfunctional reward processing in ADHD (Luman, Oosterlaan & Sergeant, 2005; Paloyelis, Asherson & Kuntsi, 2009), though taken together, the findings for the effectiveness of reinforcing contingencies in reducing the primary response inhibition deficits in ADHD are mixed (Oosterlaan & Sergeant, 1998). Dual pathway models of ADHD assume however, that (i) deficits in executive or cognitive control underlie inattention symptoms, whereas (ii) deficits in motivational control and reward sensitivity underlie hyperactivity/impulsivity symptoms (Martel & Nigg, 2006). A recent meta-analysis of general personality associations with ADHD suggests executive *and* motivational deficits in both symptom groups, though inattention was more strongly linked to executive and hyperactivity/impulsivity more strongly to motivational traits (Gomez & Corr, 2014). With regards to the RST, ADHD-inattention has been linked to increased levels of BIS (Gomez & Corr, 2010; Hundt, Kimbrel, Mitchel & Nelson-Gray; 2008; Mitchell & Nelson-Gray, 2006), whereas hyperactivity/impulsivity has been mainly associated with increased BAS (Gomes & Corr, 2010), though also with reduced (Hundt et al., 2008) or increased BIS (Mitchell & Nelson-Gray, 2006) in non-clinical samples. These findings are consistent with overactive BAS, but inconsistent regarding the role of an underactive BIS in ADHD. Importantly, the main propositions of the BIS/BAS models for ADHD and the majority of research findings (apart from Gomez & Corr, 2010) have been within the remit of the original RST – as such conflating behavioral inhibition (BIS) with punishment sensitivity (FFFS).

Despite the high comorbidity of ADHD and TS, little is known about the underlying commonalities and differentiations in reinforcement sensitivity of these two disorders. Studies have found (i) greater amygdala activation for fearful, angry and neutral facial expressions in TS patients (though comorbidity was not controlled; Neuner et al., 2010); (ii) impaired punishment learning in unmedicated TS patients, whereas reward sensitivity and reward learning were only reduced in medicated and OCD-comorbid TS patients (Palminteri et al., 2009, 2011; Worbe et al., 2011); and (iii) no differences in reward learning between pure TS patients and healthy controls (Crawford, Channon & Robertson, 2005). These findings suggest increased sensitivity to aversive and ambiguous cues (overactive FFFS and BIS) but deficits in negative reinforcement learning (dysfunctional FFFS or BIS) in TS, whereas reward processing deficits (underactive BAS) appear to be linked to medication status and presence of OCD symptoms. These findings may be due to impairment of distinct cortico-striatal circuits involved in different phenotypes of TS with varying symptom complexity or comorbidities (Worbe et al., 2010), resulting in different patterns of reinforcement sensitivity deficits.

Whilst researchers have begun to examine the associations of the phenotypes of ADHD in relation to rRST in the general population (Gomez & Corr, 2010), to our knowledge, this approach has not been extended to the examination of TS-like behaviors. Therefore, the aim of the current study was to examine individual differences in reinforcement sensitivity in the different phenotypic expressions of both ADHD- and TS-like behaviors in the general population. In order to tease apart the roles of the rRST constructs, we assess their unique associations with both overall and distinct phenotypical behaviors accounting for sex, age and ‘comorbidity’ with each other (Gomez & Corr, 2010). In line with Gomez and Corr (2010), we predicted that BAS-fun seeking would be related to increased hyperactivity/impulsivity whereas BIS-anxiety should relate to increased inattention ADHD-like behaviors. Given the previous findings in clinical TS (e.g., Palminteri et al., 2009, 2011), we expected a dysfunctional BIS

and/or FFFS to be linked to pure phonic and motor TS-like behaviors and any associations with BAS to be due to ‘comorbidity’ in TS-like behaviors.

2. Method

2.1. Participants and Procedure

The sample consisted of 138 undergraduate participants (90 females and 48 males; mean age = 23.54; SD= 4.62; 17-40 years). The study was approved by the School of Psychology Research Ethics Committee of the University of Nottingham, and the R&D Departments of the Nottinghamshire Lincolnshire Partnership NHS Trust (Derbyshire REC, ref 08/H0401/34, approved April 2008). Written consent was acquired from all participants (or written consent from parents and verbal assent from minor participants) prior to participation.

2.2. Measures

Reinforcement sensitivity was assessed using the BIS/BAS scales (Carver & White, 1994) consisting of: BIS-original (7 items), BAS-drive (4 items), BAS-fun seeking (4 items), and BAS-reward responsiveness (5 items). Following rRST (Heym et al. (2008), the BIS scale was split into BIS-anxiety (4 items) and FFFS-fear (3 items). Items were scored on a 4-point scale (1=very true to 4=very false for me), reversed scored such that higher scores indicate higher endorsement of respective RST constructs, and mean scores were calculated. Previous alphas ranged from .57 to .76 (Heym et al., 2008). In the current study the alphas (and mean inter-item correlations for scales < 5 items) were acceptable ranging from .72 (MIC=.40) for BIS-anxiety to .82 (MIC=.54) for BAS-drive, only the 3-item FFFS-fear scale showed a lower alpha of .60 though the MIC of .34 is deemed reliable (Robinson et al., 1991).

ADHD-like behaviors were assessed using the 18-item adult ADHD Self-Report Screening Scale (ASRS; Kessler et al., 2005) based on DSM-IV symptom criteria for use in the general/non-clinical population. Questions refer to the frequency of occurrence of ADHD-like behaviors over the past six months on a 5-point scale (0=never to 4=very often). The ASRS comprises two subscales: ASRS-inattention (IA) and ASRS-hyperactivity/impulsivity (HI; 9 items each). Total scores were calculated for each scale. Previous alphas ranged from .63 to .72 and the scale has shown validity in relation to clinician symptom and severity ratings in adult ADHD (Kessler et al, 2005). In the current study the alphas for IA, HI and overall ASRS were .72, .73 and .81, respectively.

Tourette-like behaviors were assessed using the 18-item TS behavior checklist based on frequency occurrence of DSM-IV and ICD10 symptoms for TS (except common complex tics as these would be unlikely in an undiagnosed population) and with a similar question format to the ASRS¹ (Heym et al., 2014). The scale comprises two pure TS subscales: TS-phonic (8 items) related to sounds produced through the nose, mouth or throat (e.g., throat clearing, coughing, sniffing), and TS-motor (7 items) related to unintentional physical movements (e.g., blinking, face twitches, random body movements). The questionnaire also includes a subscale for TS-related obsessive-compulsive behaviors (TS-OCB; 3 items), given that these frequently co-occur with TS (Robertson, 2000). Participants indicate frequency of behavior occurrence on a 5-point scale (0=never to 4=very often). The Cronbach’s alphas for TS-overall, phonic, and motor were

¹ Although the ASRS has been proposed as a screening tool and both checklists are based on DSM criteria, this study takes a dimensional approach and no diagnostic cut-off was used. The factorial structure and differential associations of the TS subscales, both with a behavioral and with response inhibition, map onto those seen in clinical TS (Heym et al., 2014).

.78, .68 and .72, respectively, and TS-OCB had a mean-inter-item correlation of .25. Total scores were calculated for TS-overall (including OCBs) and the three subscales.

3. Results

3.1. Descriptive statistics and zero-order correlations

Descriptive statistics and zero-order correlations for the associations of the rRST with ASRS and TS scales (as well as partial correlations controlling for either ASRS or TS subscales amongst each other) are shown in Table 1. The distributions for BIS-anxiety, BAS-reward responsiveness, BAS-fun seeking and TS-OCB were significantly skewed, and normalized for the subsequent analyses: negatively skewed variables (RST scales) were reflected, then Lg10 transformed (together with positively skewed TS-OCB), and then again reflected. BIS-original, BIS-anxiety and FFFS-fear were significantly positively correlated with all ASRS and TS scales, whereas the BAS scales showed no associations. In the partial correlations, the significant associations of BIS and FFFS with ASRS-IA, TS-motor and TS-OCB became non-significant. BAS-drive became positively associated with ASRS-HI and negatively with IA, when IA or HI were controlled, respectively.

Table 1: *Descriptive statistics and zero-order (and partial) correlations between RST, ADHD and TS scales*

RST scales:		ASRS			Tourette			
		overall	HI	IA	overall	phonic	motor	OCB
	Means (SD)	32.13 (8.16)	14.73 (4.72)	17.40 (4.71)	22.82 (7.73)	11.48 (4.17)	6.32 (3.57)	5.01 (2.38)
BIS-original	3.16 (0.50)	.321**	.318** (.236**)	.238** (.097)	.371**	.355** (.271**)	.234** (.035)	.211* (.138)
BIS-anxiety	3.36 (0.54)	.299**	.275** (.183*)	.243** (.127)	.328**	.302** (.221**)	.208** (.038)	.196* (.131)
FFFS-fear	2.90 (0.62)	.258**	.279** (.229**)	.167* (.034)	.306**	.307** (.242**)	.184** (.009)	.173* (.111)
BAS-drive	2.72 (0.66)	.034	.141 (.210*)	-.082 (-.177*)	.128	.045 (-.026)	.107 (.071)	.164 [^] (.143)
BAS-reward	3.43 (0.44)	.051	.036 (-.012)	.052 (.039)	.135	.110 (.069)	.091 (.030)	.078 (.050)
BAS-fun seeking	2.88 (0.61)	.090	.131 (.138)	.024 (-.048)	-.021	.070 (.122)	-.048 (-.076)	-.115 (-.118)

Note: [^] $p = .055$, statistically significant associations at * $p < .05$, ** $p < .01$ are in bold; HI = hyperactivity/impulsivity, IA = inattention, OCB=obsessive-compulsive behaviors; Correlation coefficients in parentheses are partial correlations controlling for the subscale(s) of the same construct (e.g., the zero-order correlation between ASRS-IA and BIS-original is .238** and .097 when ASRS-HI is controlled for).

3.2. Regressions for unique associations of rRST scales with ASRS and TS

In order to examine the unique contribution of the rRST scales on the ASRS and TS scales, with (and without) controlling for sex, age and TS or ASRS, respectively, linear regression analyses were conducted. The standardized beta values for ASRS are presented in Table 2 and for TS in Table 3. The overall models were statistically significant ($R^2 >.16$; $F_s >2.87$; $ps <.01$). For ASRS, BIS-anxiety was associated with increased IA, and together with FFFS-fear, also with increased HI independent of whether TS was controlled for. BAS-reward responsiveness was associated with reduced HI when TS was controlled for (but only marginally when not), and BAS-fun seeking was only marginally associated with increased HI, independent of TS. BIS-anxiety and FFFS-fear were positively associated with overall and phonic TS when ASRS was not controlled for (FFFS remained a predictor for overall TS independent of ASRS). BAS-drive was positively and BAS-fun seeking was negatively associated with TS-OCB, independent of ASRS. Phonic and motor TS were solely positively associated with ASRS-HI (TS-OCB also negatively with IA).

Table 2: *Regression analyses of ADHD-like behaviors on the rRST scales*

ASRS:	overall	HI	IA
Sex	-.103	-.167	-.039
Age	-.139	-.071	-.173*
TS-overall	.347***		
TS-phonic		.355***	.226*
TS-motor		.064	.143
TS-OCB		.050	-.193*
BIS-anxiety	.213* (.273**)	.184* (.236*)	.184 (.237*)
FFFS-fear	.094 (.145)	.154 (.200*)	.007 (.050)
BAS-drive	-.062 (-.007)	.104 (.139)	-.115 (-.152)
BAS-reward	-.116 (-.103)	-.193* (-.184 [^])	.002 (.006)
BAS-fun seeking	.202* (.174)	.176 [^] (.179 [^])	.065 (.123)
R^2	.261*** (.129**)	.330*** (.157***)	.211** (.082*)

Note: [^] $p <.06$, statistically significant associations at * $p <.05$, ** $p <.01$, *** $p <.001$ are in bold; R^2 and standardized beta coefficients for models excluding sex, age and TS in parentheses.

Table 3: *Regression analyses of TS-like behaviors on the rRST scales*

Tourette:	overall	phonic	motor	OCB
Sex	-.120	-.021	-.197*	-.044
Age	-.013	-.017	.015	-.074
ASRS-overall	.342***			
ASRS-HI		.378**	.213*	.257*
ASRS-IA		.083	.119	-.244*
BIS-anxiety	.150 (.213*)	.097 (.213*)	.104 (.129)	.103 (.101)
FFFS-fear	.181 [^] (.198*)	.123 (.210*)	.123 (.115)	.083 (.115)
BAS-drive	.136 (.147)	-.044 (.007)	.109 (.143)	.192 [^] (.266**)
BAS-reward	.031 (.004)	.056 (-.014)	.038 (.011)	.038 (-.005)
BAS-fun seeking	-.092 (-.051)	.037 (.119)	-.115 (-.095)	-.228* (-.216*)
R^2	.271*** (.151***)	.288*** (.135**)	.185** (.069)	.168** (.108*)

Note: $\wedge p < .06$, statistically significant associations at * $p < .05$, ** $p < .01$, *** $p < .001$ are in bold; R^2 and standardized beta coefficients for models excluding sex, age and ADHD in parentheses.

4. Discussion

ADHD-related individual differences in the general population have previously been studied using rRST (Gomez & Corr, 2010). In the present study, BAS-reward responsiveness was associated with reduced, whereas BAS-fun seeking tended (marginally at $p = .06$) to be associated with increased hyperactivity/impulsivity in the regression models. Whilst Gomez and Corr (2010) found increased BAS-fun seeking in hyperactivity/impulsivity independent of Oppositional Defiant Disorder (ODD) symptoms, in the current study this association was independent of TS 'comorbid' disposition. However, these relationships were not seen in the correlations (most likely due to the relatively low sample size), and only reflect the unique contribution of these BAS scales, independent of the shared variance with the other RST constructs. BAS-drive was only associated with increased hyperactivity/impulsivity and reduced inattention when each was controlled for the other in the partial correlations. Multiple-processing models for BAS emphasize the distinction between reinforcement processes redirecting (i) attention towards reward (reward responsiveness), and (ii) behavior towards gaining rewards (drive) (Pickering & Smillie, 2008). Thus, reward processing and goal-directed behavior may play a differential role in pure Attention Deficit or Hyperactivity Disorder. Moreover, BAS-reward responsiveness and BAS-drive cluster under a reward reactivity factor that is distinct from a trait impulsivity factor, which encompasses BAS-fun seeking (Smillie, Pickering, & Jackson, 2006; Heym & Lawrence, 2010). Given this distinction between reward reactivity and trait impulsivity, the current findings question a simple overactive BAS account (if conceptualized as reward sensitivity rather than impulsivity) in ADHD and may explain inconsistent findings in the past (Oosterlaan & Sergeant, 1998; Paloyelis et al., 2009).

In line with our prediction, BIS-anxiety was associated with increased inattention, however, together with FFFS-fear, also with increased hyperactivity/impulsivity. Although the current findings do not support the notion of an underactive BIS leading to dysfunctional inhibition in ADHD (Quay, 1988), an overactive BIS has been proposed to lead to anxious impulsivity in ADHD (Wallace, Newman & Bachorowski, 1991). A multiple-pathway model for BIS considers (i) anxiety, worry and rumination, and (ii) conflict detection/risk assessment as distinct processes governed by separate neural levels (McNaughton & Corr, 2008). Accordingly, it is feasible that (i) high anxiety levels combined with (ii) dysfunctional risk assessment lead to such anxious impulsivity. Importantly, the BIS scale measures only aspects of anxiety, and does not cover the full repertoires of BIS (conflict detection/risk assessment). As such, these associations cannot be extended to the functioning of the whole system. Clearly, this issue needs to be readdressed once better measurement tools, taking both aspects of BIS into account, are available. Furthermore, the underactive BIS account for ADHD based on the assumption of punishment insensitivity (Quay, 1988, 1997) - now FFFS - is not supported by the current data given the positive association between FFFS-fear and hyperactivity/impulsivity. It is possible that some impulsive aspects are driven by hypersensitivity to aversive cues leading to fight/flight responses. Indeed, high levels of FFFS have been shown to increase (moderate) trait impulsivity (Heym & Lawrence, 2010).

Regarding TS, BIS-anxiety and FFFS-fear were consistently and uniquely associated with increased overall and phonic TS, but these associations disappeared when ADHD was taken into

account. Thus, findings regarding increased sensitivity to aversive and ambiguous cues (Neuner et al., 2010) may have been due to ADHD comorbidity. BIS-anxiety and FFFS-fear were also associated with motor TS and OCBs in the zero-order correlations, but did not uniquely contribute to either. FFFS and BIS are proposed to facilitate simple (motor) and complex (cognitive) obsessional avoidance behaviors of OCD, respectively (Gray & McNaughton, 2000), suggesting that overactivity of BIS and FFFS in TS may be due to high OCD comorbidity (Cavanna & Rickards, 2013). However, our findings do not support a strong role for these systems in OCBs. On the other hand, though pure TS-like behaviors were not related to BAS, TS-OCB was linked to reduced BAS-fun seeking and increased BAS-drive (independent of ADHD dispositions). This opposite pattern suggests that OCBs seen in TS are linked to high goal orientation and reduced impulsivity, which may reflect the compensatory strategy of increased cognitive control in TS (Jung et al., 2012). TS tics are preceded by strong premonitory sensations that urge for relief, and if they are suppressed, they are followed by a rebound worsening of symptoms (Leckman, Walker & Cohen, 1993). Increased drive in combination with punishment sensitivity may be reflected in the persistence of compensatory OCBs for relief. The apparent involvement of BAS in TS is in line with the role of dopaminergic pathways (Robertson, 2000), though the findings suggest this may be due to OCD comorbidity. Nevertheless, given the low reliability of the (three-item) TS-OCB scale this should be further examined using more specifically designed psychiatric rating scales for OCD. The current OCB scale only reflects behaviors characteristic of TS, which appear to be clinically different from those seen in OCD (Robertson, 2000), and can therefore not be equated with the full repertoire of OCD.

Taken together, our findings regarding the role of the rRST in ADHD are different to Gomez and Corr's (2010) findings. This may be due to (i) different 'comorbidities' taken into account – (ODD as opposed to TS); and/or (ii) different assessment methods for ADHD-like behaviors (we used a measure adapted for use in non-clinical samples as opposed to a clinical measure of DSM-IV symptoms). This clearly has implications for the operationalization and measurement of ADHD-like behaviors in the general population. To our knowledge, this is the first study that examined the role of the rRST constructs in TS. We found some evidence for an overactive BIS and FFFS, and opposite associations of BAS-drive/reward and BAS-fun seeking. However, these associations were influenced by either ADHD-like behaviors or co-occurring OCBs, respectively. The differential associations of the BAS subsystems with distinct ADHD and TS phenotypes highlights the importance of incorporating multiple-process models for BAS specific pathways (Pickering & Smillie, 2008) in explaining the distinct phenotypes of such disorders. The unique roles of the rRST subsystems in various disorder subtypes and related comorbidities should be further systematically examined – ideally in clinically pure versus comorbid patient groups – to clarify the involvement of specific reinforcement pathways as potential underlying mechanisms.

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