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Executive function in high-functioning autism: Decision-making consistency as a characteristic gambling behaviour

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ABSTRACT

Restricted and repetitive patterns of behaviours, interests, or activities are a critical diagnostic criterion for autism spectrum disorder (ASD). Previous studies using gambling paradigms with ASD populations have identified that, unlike typically developed control participants, people with a diagnosis of ASD tend to maintain particular response patterns regardless of the magnitude of potential outcomes to uncertain gains or losses. Here we designed a gambling test that permitted calculation of the response consistency in gambling choices in situations that presented varying expected outcomes in terms of gains or losses. The task was administered to 33 adults with a diagnosis of ASDs and compared to a group of 47 typically-developed (TD) control participants who were matched for age and IQ (Intelligence Quotients). When presented with choices where participants could either make a risky gamble or a safe choice in terms of gains or losses (e.g., 20% chance of winning £5 *vs.* 100% chance of winning £1), the ASD participants did not differ from the TDs in their overall risk-taking behaviour. However, they were more consistent in their individual choices from trial to trial. Furthermore, the proportion of participants who either implemented an invariate response strategy (e.g., either always choosing the most risky or most "safe" option) was significantly higher in the ASD group compared with the controls. Additionally, while the ASD group were slower to make their responses in the win frame and the first half of the lose frame, by the end of the task their decision times were the same as the TD controls. These findings suggest that the ASD tendency towards repetitive behaviour may demonstrate itself even in high-level decision-making tasks, which needs to be understood if we are to be sure what such tasks are measuring.

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1. Background

Rumiati and Humphreys (2015) highlight the extraordinarily rapid recent development of the field of "social cognitive neuroscience", and describe this field as facilitating "the development of models attempting to bridge social cognition with neuroscience". One of the ways in which this is being conducted is to apply the methods developed from cognitive neuroscience and neuropsychology to the study of people with autism, particularly those related to "executive dysfunction" (e.g., Spitzer, White, Mandy, & Burgess, 2016). However, the possibility exists that the individual differences in performance on a particular paradigm that exist in people with autism do not share a common basis with typically-developed people, or those with acquired brain damage. This would challenge the drawing of inferences from these findings. For instance, White (2013) argues that performance on tests of executive function may not reflect a true "executive dysfunction", but instead the failure to form an implicit understanding of what the experimenter expects from the participant in performance of the task, leading to odd and idiosyncratic behaviour. It is argued that in these situations, the source of the impairment is actually one of mentalizing or some other social impairment in social cognition rather than of non-social executive function. This is a particularly critical issue, since impairments on tests purportedly of executive function are prevalent in autism (Hill, 2004).

Indeed, there is much debate about the significance of findings on executive function tests in terms of understanding the features of autism (Ozonoff, 1997). Some have argued that the dysexecutive features of autism are primary to the condition (e.g., Russell, 1997), and Pellicano (2007) has argued that executive function may be a necessary precursor to development of theory of mind (see also Ozonoff & McEvoy, 1994). But others have suggested more specific or complex relations between the various features of social cognition (including mentalizing and theory of mind), repetitive behaviour, and "executive dysfunction" (by which is usually meant problems with dealing with novel situations, monitoring and adjusting behaviour, inhibition, initiation etc.). For instance, while several authors have noticed a correlation between executive function problems and impairments in social and communication in autism (e.g., Gilotty, Kenworthy, Sirian, Black, & Wagner, 2002), others have noted a relationship between executive function problems and repetitive behaviours but not sensory features (Boyd, McBee, Holtzclaw, Baranek, & Bodfish, 2009). Others maintain that there may be a relation between repetitive behaviour and only some measures of executive functioning (South, Ozonoff, & Mcmahon, 2007). So there is a general contrast between those who see executive dysfunction as core and probably causal to the presentation of behavioural and social features of autism (e.g., repetitive behaviour, mentalizing), and those who suggest that the relations between these constructs might be more complex.

This latter view would be easy to justify from what we now know about the functions of the frontal lobes and their supporting structures within the brain. Indeed, given what we know about the location of structures within the prefrontal cortex that support social competencies like mentalising and theory of mind, and also various executive functions, it is a possibility that any relation in performance is in effect merely epiphenomena. In neurological patients with acquired damage, there is no "executive (or "frontal lobe") syndrome": the various dysexecutive features show a high degree of dissociation (See Burgess and Stuss, in press for review), with, seemingly, each function having its own neuroanatomical substrates.

For instance, multiple studies of mentalising and social cognition both in neurological patients and also neuroimaging of healthy brains have isolated medial PFC, including caudal medial areas 10 and 11 (frontopolar and orbitofrontal regions) as being a critical part of the brain network that supports social cognition and theory of mind (e.g., Blair & Cipolotti, 2000; Gilbert, Spengler, Simons, Steele et al., 2006; Shammi & Stuss, 1999). This region is extremely close to that which supports executive functions such as multitasking, prospective memory, and task initiation speed (e.g., Burgess, Veitch, Costello, & Shallice, 2000, Burgess, Quayle & Frith, 2001; Burgess & Wu, 2013; Volle et al., 2011). So any developmental or acquired condition that might affect this general region might cause a regular co-occurrence in problems with theory of mind and some executive abilities merely because the anatomical substrates are close together in the brain rather than that the processing is shared or that there is a causal link between them. This may be one explanation for the high frequency of impairments in e.g., multitasking and also theory of mind in people with autism (e.g., White, Burgess, & Hill, 2009). A more complex but related possibility is that executive and social difficulties might be secondary to poor functional connectivity within the brain (Just, Cherkassky, Keller, Kana, & Minshew, 2007). In this case the process that has caused the poor connectivity may just be a mediator variable. But in neither case need there be a direct link at an information processing level between the social or behavioural problems and the executive ones.

Part of the difficulty in attempting to disentangle these various factors and influences is that most, if not all, of the studies that have examined the relation between them have been correlational in design. Typically, measures of social cognition and a separate measure (either psychometric or rating scale) of executive function are administered, and the correlation between them is examined. But these measurements are rarely likely to be independent. Not only might problems with implicit understanding of what is expected of the participant affect what they do on an executive function task (as White's triple-I hypothesis contends), but also behavioural features such as a repetitive tendency might in theory determine behaviour on an executive function task, contributing to variance in performance independently from variance in the "executive function construct (e.g., inhibition, decision-making or whatever) that is the intended focus of measurement of the task.

In these ways the investigation of the relation between social and executive deficits in autism mirrors that which has been conducted in neurological patients with acquired deficits over the last 50 years in particular. For instance, it has long been known that lesions induced through psychosurgery cause mood changes, as well as changes in social behaviour and also poor performance on executive function tasks even

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