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Executive functioning in schizophrenia: Unique and shared variance with measures of fluid intelligence

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ABSTRACT

Objective: Patients with schizophrenia often display deficits on tasks thought to measure "executive" processes. Recently, it has been suggested that reductions in fluid intelligence test performance entirely explain deficits reported for patients with focal frontal lesions on classical executive tasks. For patients with schizophrenia, it is unclear whether deficits on executive tasks are entirely accountable by fluid intelligence and representative of a common general process or best accounted for by distinct contributions to the cognitive profile of schizophrenia.
Method: In the current study, 50 patients with schizophrenia and 50 age, sex and premorbid intelligence matched controls were assessed using a broad neuropsychological battery, including tasks considered sensitive to executive abilities, namely the Hayling Sentence Completion Test (HSCT), word fluency, Stroop test, digit-span backwards, and spatial working memory. Fluid intelligence was measured using both the Matrix reasoning subtest from the Weschler Abbreviated Scale of Intelligence (WASI) and a composite score derived from a number of cognitive tests.
Results: Patients with schizophrenia were impaired on all cognitive measures compared with controls, except smell identification and the optimal betting and risk-taking measures from the Cambridge Gambling Task. After introducing fluid intelligence as a covariate, significant differences remained for HSCT suppression errors, and classical executive function tests such as the Stroop test and semantic/-phonemic word fluency, regardless of which fluid intelligence measure was included.
Conclusions: Fluid intelligence does not entirely explain impaired performance on all tests considered as reflecting "executive" processes. For schizophrenia, these measures should remain part of a comprehensive neuropsychological assessment alongside a measure of fluid intelligence.

1. Introduction

The term "executive functions" is widely used to refer to a number of abilities involved in complex behaviours (Luria, 1970) such as planning, problem solving, and working memory, and are thought to rely heavily on prefrontal cortical areas. Executive functions are considered key cognitive capacities that allow humans to adapt and flourish and a crucial aspect of what is considered uniquely "human" (Teffer & Semendeferi, 2012). One current theoretical issue is whether impairment in executive functions reflects a common, general process that relies on a multiple demand region (Duncan, 2010, 2013) or whether executive functions comprise many sub-processes supported by distinct neural substrates (Stuss & Alexander, 2007). One avenue for exploring this question is through neuropsychological studies of clinical groups that experience difficulties in executive functions. In helping to understand a heterogeneous disease such as schizophrenia, this question is important for both theoretical grounds, but also to assist in the selection of cognitive test batteries for studies aiming to investigate more homogenous subtypes. In the age of refining clinical phenotypes for genetic discovery and characterization (Cannon & Keller, 2006; Glahn et al., 2014; Gottesman & Gould, 2003; Gur et al., 2007), and with the intention of large research organizations such as the National Institute of Mental Health's (NIMH) Research Domain Criteria (RDoC) outlining a research strategy of classifying psychopathology based on dimensions of observable behaviour and neurobiological processes (Morris & Cuthbert, 2012), it is increasingly important to understand the dimensions of cognitive

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impairment and the most effective and efficient methods for measurement (Joyce & Roiser, 2007).

Understanding the relationship between executive tasks and fluid intelligence is beneficial for our understanding of schizophrenia but also a wide range of diseases known to experience executive dysfunction. Deficits in tasks measuring executive functions are experienced in disorders that affect the prefrontal regions such as Parkinson's disease (Foltynie, Brayne, Robbins, & Barker, 2004; Koerts et al., 2011; Lewis et al., 2005; Muslimovic, Post, Speelman, & Schmand, 2005), Huntington's disease (Bachoud-Levi et al., 2001), frontotemporal dementia (Huey et al., 2009); lesion studies also provide evidence for these deficits (Fiebach, Ricker, Friederici, & Jacobs, 2007; Robinson, Shallice, Bozzali, & Cipolotti, 2010, 2012; Roca et al., 2010). Neuroimaging studies have moreover, provided evidence for the importance of prefrontal regions in a wide range of executive functions (Wagner, Maril, Bjork, & Schacter, 2001; Yuan & Raz, 2014). Cognitive impairment is a core clinical symptom of schizophrenia, a disorder that affects approximately one in a hundred people and often has a devastating effect on social and societal functioning across the lifespan. Impairments in executive functions such as conceptualization, planning, cognitive flexibility, word fluency, ability to solve complex problems, and working memory often occur in schizophrenia (Orellana & Slachevsky, 2013) and is thought, in part, to reflect the frontal dysfunction characteristic of the disorder (Mathalon & Ford, 2008). However, schizophrenia is associated with significant cognitive heterogeneity (Joyce & Roiser, 2007) and coupled with the extensive cognitive dysfunction experienced, this has resulted in the specificity or underlying mechanisms of these deficits remaining poorly understood.

One line of converging evidence from neuropsychological and neuroimaging studies suggest that there may be a common general process able to explain all variance on classic executive measures. Functional neuroimaging studies of executive processes consistently show recruitment of regions including the inferior frontal sulcus, anterior insula and adjacent frontal operculum, the pre-supplementary motor area and adjacent anterior cingulate cortex, and the intraparietal sulcus, with tasks considered to tap dissociable executive factors, such as task-switching and response inhibition, unable to recruit unique regions or networks (Duncan, 2010; Duncan & Owen, 2000), although some evidence exists for specificity of neural processes of executive abilities (Collette, Hogge, Salmon, & Van der Linden, 2006). In terms of neuropsychological measures, the phenomenon of 'g' emerged from the observation of a consistent positive correlation between cognitive tests (Spearman, 1904, 1927). Operationally, 'g' has been measured using a composite score, usually the first component of a factor analysis containing a wide-range of varied cognitive measures, or a single test approximate of fluid intelligence, such as matrix reasoning from the Wechsler scales (Weschler, 1999, 2008), Cattell's Culture Fair (Cattell & Cattell, 1960) or Raven's progressive matrices (Raven, 1938).

Recently, several studies have contributed to our understanding of the extent that performance on measures of executive functions are reflective of multiple sub-processes or a common underlying process that, when damaged, subsequently leads to impairments across a number of tasks. By including a measure of fluid intelligence in addition to tests thought to tap specific executive abilities, it was possible to address whether specific measures explained unique variance not attributable to fluid intelligence. In a group of frontal lesion patients, fluid intelligence, as measured by the Culture Fair IQ, was found to explain the difference with controls on the Wisconsin Card Sorting Test, Word Fluency and Iowa Gambling tasks, all classic tasks of executive functions (Roca et al., 2010). The authors proposed that such tasks might not adequately assess specific deficits, but rather reflect a more general cognitive loss and put forward the multiple demand system as one candidate that may explain these deficits. However, other executive measures, the Hayling and Go-No go tasks, remained significant with fluid intelligence in the model. Roca and colleagues hypothesized this reflected involvement of the anterior frontal cortex in the processes of multi-tasking (Braver & Bongiolatti, 2002; Burgess, Quayle, & Frith, 2001; Gilbert et al., 2006) and ability to switch between different cognitive contexts (Badre & D'Esposito, 2007; Burgess, 2005; Koechlin, Ody, & Kouneiher, 2003). Word fluency was only marginally non-significant ($p = 0.07$) after fluid intelligence was entered as a covariate and evidence has since emerged that word fluency deficits in frontal lesion patients may represent a distinct cognitive process supported by specific neuroanatomical regions. A study by Robinson et al. (2012) comparing healthy controls to patients with either focal frontal or posterior lesions on a range of verbal and non-verbal fluency tests found that phonemic word fluency did not correlate with fluid intelligence. It was also observed that the deficit was most severe in patients with left inferior frontal gyrus lesions, supporting a specialized role of this region in word fluency constrained by phonology.

Employing a similar methodology in Parkinson's disease, the role of fluid intelligence in executive tasks has been explored (Roca et al., 2012). Using the Raven's Colored Progressive Matrices (RCPM) as a measure of fluid intelligence, similar results were observed to those in the frontal lesion patients. Variance between patients and controls on the Wisconsin Card Sorting Task and word fluency task was removed when fluid intelligence was introduced as a covariate. Again, variance on the Hotel Task, a task measuring an individual's ability to manage multiple goals and tasks, remained significant after fluid intelligence was included in the model, suggesting distinct processes for tasks involving multitasking. It should be noted that word fluency again only just failed to reach significance when fluid intelligence was included as a covariate ($p = 0.07$), and although it may explain a significant proportion of word fluency capacity, it remains unclear as to whether word fluency tests simply reflect a more general process as reflected by fluid intelligence measures.

The contribution of fluid intelligence to processes traditionally thought to tap executive functions has also been investigated in behavioural-variant frontotemporal dementia (bvFTD) (Roca et al., 2013). Using a composite score derived from six varied cognitive tests, similar results to frontal lesion and Parkinson's patients were observed. A notable difference concerned the Iowa Gambling Task, which remained significant unlike in previous studies. The authors suggest that specific deficits in risky decision-making for bvFTD patients may explain the difference with previous clinical groups and could reflect ventromedial frontal pathology.

This leads to the purpose of the current study and schizophrenia. Although patients with schizophrenia are impaired on a wide range of cognitive tests and are known to be impaired on measures of fluid intelligence (Caspi et al., 2003; Johnson et al., 2013), few studies have explored whether deficits on measures of executive functions are indicative of a common general process or specific sub-processes. Factor analyses have been conducted on large groups of schizophrenia samples with results suggesting that a single component model was the best fitting model to describe cognitive functioning in schizophrenia (Dickinson, Goldberg, Gold, Elvevag, & Weinberger, 2011; Keefe et al., 2006), although these only accounted for 45–63% of variance, leaving substantial residual variance to be explained. To date, only one study with patients with schizophrenia has investigated the unique variance explained by a number of tasks considered measures of specific executive functions (Roca et al., 2014). In a small group of 15 patients with schizophrenia, deficits in the performance of patients, compared to controls, on executive measures (WCST, word fluency, and