

Title: The Neuropsychology of Creativity

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Abstract:

The neuropsychological approach has been instrumental in delivering key insights that have enabled a clearer understanding of the human mind and its workings. Despite the unique promise of this approach and the perspective it affords, it has only been limitedly utilized when exploring creative cognition. This paper provides an overview of three methodologies – single case studies, case series investigations on neurological populations, and case series investigations on psychiatric populations – that have been employed within the neuropsychology of creativity and highlights some of the important revelations that each direction of study has delivered. In doing so, the aim is to make a case for the unique utility of the neuropsychological approach in allowing for a better understanding of the creative mind.

Highlights:

- The neuropsychological approach is limitedly employed in the study of creativity
- An important focus of single case studies is the phenomenon of ‘de novo’ creativity
- Case series investigations in creativity cover neurological and psychiatric groups

Keywords:

creative neurocognition; creative cognition; de novo abilities; neurology; psychiatry; clinical psychology; subclinical groups; cognitive neuropsychology

The brain basis of creativity, or the capacity to conceive of ideas that are original, unique, unusual or novel as well as relevant, fitting, appropriate or satisfying to a particular end (Runco & Jaeger, 2012), is primarily explored through neuroimaging and EEG based approaches (Jung & Vartanian, 2018). Neuropsychological studies are relatively uncommon (Abraham, 2018). This is extremely unusual given the unmistakable utility of the neuropsychological approach in delivering answers about the mechanisms underlying cognition and behavior as well as the unique insights it affords when comparing competing theories of any aspect of these functions (Caramazza & Coltheart, 2006). Although this would also naturally extend to the context of creative neurocognition, neuropsychological studies are rarely leaned on when making inferences on the mechanisms that underlie the same.

The rationale of the neuropsychological approach is that when brain insufficiencies lead to specific changes in behavioral and cognitive function, we can safely assume that the implicated brain regions are not only involved in the said functions, they are likely to be vital for the same. Justifiable critiques of the approach notwithstanding (Fischer-Baum & Campana, 2017; Patterson & Plaut, 2009), it is undeniable that this approach has been instrumental in delivering key knowledge on how the workings of the human mind as typically showcased by the iconic case studies of HM (Squire & Zola-Morgan, 2011), Phineas Gage (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994), and Tan (Domanski, 2013) among many others. In fact, these classic cases continue to be influential even in contemporary studies that map brain structure to brain function (Thiebaut de Schotten et al., 2015).

Neuropsychological investigations of cognitive function typically fall into one of two categories: single case studies of individuals with specific neurological damage and case series investigations, which are group-based studies of individuals who have related brain dysfunctions. While the advantages and disadvantages associated with both approaches is a matter of some debate (Lambon Ralph, Patterson, & Plaut, 2011; Medina & Fischer-Baum, 2017; Nickels, Howard, & Best, 2011; Rapp, 2011; Schwartz & Dell, 2010), it can be maintained that following a multipronged approach would afford the best possible outcomes.

Neuropsychological studies on creativity primarily follow three different methodologies (Figure 1): single case studies of neurological patients, case series investigations of neurological samples, and case series studies of psychiatric samples. The last category is also closely associated with a further methodology, namely the personality based approach. This bears mentioning here as it follows a quasi-neuropsychological logic given that the rationale underlying the linking of specific subclinical personality traits and their associated information processing biases in relation to individual differences in creative cognition (Abraham, 2015; Baas, Nijstad, Boot, & De Dreu, 2016).

Of the single case studies that are relevant to creative neurocognition, the most influential have been the investigations in relation to frontotemporal dementia (FTD). Fascinating examinations of people who develop *de novo* artistic capabilities post neurological insult have been reported in a small subset of patients with the temporal lobe variant of FTD where brain damage is seen in temporal regions

whereas frontal regions remain relatively intact (Liu et al., 2009; Miller et al., 1998; Miller & Hou, 2004; Miller & Miller, 2013). The characterization of 'de novo' is warranted in this context as these (predominantly visual and musical) artistic abilities appear suddenly following brain injury or degeneration and they are unexpected given that the person did not exhibit such tendencies prior to the onset of FTD (Schott, 2012; Zaidel, 2010). This tendency to engage in artistic expression is not short-lived, but tends to be compulsive and highly sustained. Indeed, in the first published report to showcase this phenomenon, all three patients went on to become accomplished painters (Miller, Ponton, Benson, Cummings, & Mena, 1996). The emergence of de novo creativity relevant skills has also been associated with semantic variant primary progressive aphasia (PPA) or semantic dementia (SD) in both visual artistic and literary domains (Midorikawa & Kawamura, 2015; Wu et al., 2015) as well as in Parkinson's disease (Schrag & Trimble, 2001)

A few caveats are necessary to ward off errors of generalization. First, de novo capabilities are a rare manifestation that occurs in only a tiny proportion of patients affected with the disorders in question. So higher levels of artistry cannot be regarded as central to the neuropsychological profile associated with these disorders. Second, artistic skills displayed in relation to neurodegenerative disorders are rarely prodigious, unlike the case of savants for instance. Nonetheless, it is highly noteworthy that brain injuries that result in reduced function in some cognitive domains, such as semantic understanding, social awareness and speech production, are accompanied by enhanced artistic abilities that emerge unexpectedly.

One hypothesis that has been put forward to explain this phenomenon of what can be seen as evidence of 'paradoxical functional facilitation' (Kapur, 1996) is that the inability to express oneself as one previously did results in the turn towards artistic expression as the drive to communicate is maintained despite the inability to do so (Zaidel, 2014). In fact, longitudinal studies of the creative output of artists who developed FTD (Mell, Howard, & Miller, 2003), PPA (Seeley et al., 2008), left brain injury post-stroke (Takahata et al., 2014) and Parkinson's disease (Chatterjee, Hamilton, & Amorapanth, 2006; Shimura, Tanaka, Urabe, Tanaka, & Hattori, 2012) indicate that their drive for creative expression continued unabated regardless of their altered neurological function. Moreover, fascinating changes in the creative style of these artists, specifically in relation to frontal lobe damage, were documented such that the paintings produced post-injury are characterized by enhanced visual realism and vividness of detail. What this fascinating shift in style following specific forms of brain damage tells us about the workings of the creative brain is as yet unclear as it has received only limited attention thus far. What is clear though is that the damage-resistant capacity of the human brain to engage in artistic expression is attested to by the enormous collection of case discussions of artists who sustained some form of neurological dysfunction yet continued to be productive in a creative capacity (Finger, Zaidel, Boller, & Bogousslavsky, 2013).

The second approach, case series or group-based investigations on neurological populations of interest, has been adopted in a far more limited capacity compared to the first approach, yet at the same time also in a more heterogeneous manner. This makes Disorders of interest include FTD (de Souza et al., 2010; Rankin et al., 2007),

SD (Rankin et al., 2007), Parkinson's disease (Canesi, Rusconi, Isaias, & Pezzoli, 2012; Canesi et al., 2016; Lhommée et al., 2014) and savant syndrome (Motttron, Dawson, & Soulières, 2009; Treffert, 2009, 2014), as well as patients with lesions of the frontal lobe (Abraham, Beudt, Ott, & von Cramon, 2012; Reverberi, Toraldo, D'Agostini, & Skrap, 2005; Shamay-Tsoory, Adler, Aharon-Peretz, Perry, & Mayseless, 2011), the hippocampus (Duff, Kurczek, Rubin, Cohen, & Tranel, 2013; Warren, Kurczek, & Duff, 2016), the parieto-temporal cortex (Abraham et al., 2012; Shamay-Tsoory et al., 2011), and the basal ganglia (Abraham et al., 2012).

As the samples employed within the case series investigations of FTD and SD do not only include participants with documented de novo artistic capacities, the findings from such case series investigations are difficult to align with the findings from the single case studies of these neurological disorders. Case studies also do not typically include standard divergent thinking psychometric tasks as a form of creativity assessment of the person in question. The limited case series evidence on hand suggests that while differences in artistic style could be seen in the drawings that were generated by these groups compared to neurotypical control participants (Rankin et al., 2007), they also demonstrated poor performance on standardized psychometric measures of creativity (de Souza et al., 2010; Rankin et al., 2007). On the other hand, in a comparison of PD patients with and without increased artistic production relative to healthy control subjects on psychometric tests of creativity revealed that PD patients with enhanced artistic abilities performed comparable to healthy control samples, whereas those without exhibited poorer performance (Canesi et al., 2012). One of the key issues of focus in relation to this patient

population is the influence of dopaminergic therapies on creative capacity in PD with some suggesting that improved creative performance could be attributed to the impact of the drugs on reducing latent inhibition which would abet wider associative thinking (Faust-Socher, Kenett, Cohen, Hassin-Baer, & Inzelberg, 2014). Indeed, recent evidence using an option generation task, where participants are instructed to draw as many different paths as they can between two points in space within a fixed time period, showed that the use of dopaminergic drugs in PD patients and in healthy older adults increased the uniqueness of the responses associated with a given level of fluency (number of responses generated) (Ang et al., 2018).

The idea that cognitive disinhibition can confer advantages in creative cognition has also received support from case series studies of patients with lesions of the basal ganglia (BG) (Abraham et al., 2012). BG patients demonstrated better performance compared to healthy matched control subjects on a specific creativity task (overcoming constraints of knowledge) that requires that participants transcend the mental set imposed by recent knowledge, brought about through the imposition of active contextual salience (presenting examples of new toys generated by others), when creating something new (inventing a new toy). Greater distractibility and disinhibition that typify such populations would be beneficial in this context as it allows one to disregard the salience of the presented information more effortlessly. The study also showed the converse pattern of performance on the same in patients with lesions of the parieto-temporal lobe (PTL), a population that is characterized by perseverative response patterns. Another key point to note was the selectivity of the advantage in relation to BG lesions as the same group showed other patterns of

performance relative to the matched healthy control sample (impaired and comparable) on other aspects of creative cognition.

A more complicated picture emerges from the study of patients with lesions of the frontal lobe (FL) as both advantages (e.g., better insight in problem solving associated with dorsolateral prefrontal damage; better overcoming of knowledge constraints associated with frontopolar and orbitofrontal damage) and disadvantages (e.g., reduced originality associated with lateral and medial prefrontal damage; reduced practicality in creative imagery associated with lateral prefrontal damage) have been reported in select aspects of creative cognition as a function of select types of FL insufficiencies (Abraham et al., 2012; Reverberi et al., 2005; Shamay-Tsoory et al., 2011). The ideas of Boot et al. (2017) appear to have a particular relevance when considering the manifestation of both enhanced and impoverished creative performance in relation to the frontal lobe and the basal ganglia as they make a case for frontostriatal brain networks in orchestrating processes relevant to flexibility (neither low nor high but moderate striatal dopamine) and persistence (neither low nor high but moderate prefrontal dopamine) in creativity (Boot, Baas, van Gaal, Cools, & De Dreu, 2017).

The third approach is that of case series studies on psychiatric groups of interest whose information processing deficits are believed to be of especial relevance to creative cognition. Disorders that have received most of the focus so far are bipolar disorder (Andreasen, 2008; Santosa et al., 2007; Soeiro-de-Souza, Dias, Bio, Post, & Moreno, 2011; Taylor, 2017) and schizophrenia (Abraham, Windmann, McKenna, &

Güntürkün, 2007; Acar, Chen, & Cayirdag, 2018), which is to be expected given the population based studies that indicate a higher degree of these disorders of psychosis in relation to creative professions (Kyaga et al., 2013, 2011; MacCabe et al., 2018) as well as a modest genetic propensity for the same (Power et al., 2015) (also see Keller & Visscher, 2015). Other disorders of interest, which have albeit received comparatively little attention, include attention deficit hyperactivity disorder (ADHD) (Abraham, Windmann, Siefen, Daum, & Güntürkün, 2006; Boot, Nevicka, & Baas, 2017; Healey & Rucklidge, 2006), autism (Craig & Baron-Cohen, 1999; Diener, Wright, Smith, & Wright, 2014; Kasirer & Mashal, 2014) and dyslexia (Kasirer & Mashal, 2016; von Károlyi, Winner, Gray, & Sherman, 2003; Wolff & Lundberg, 2002).

Empirical studies on the association between creativity and schizophrenia have resulted in mixed findings, with the grounds for this inconclusive state of affairs tied to methodological factors, such as varied symptom severity associated with the patient population being tested across studies, type of creativity measure being employed, and so on. A recent meta-analysis (Acar et al., 2018) concluded that the evidence indicates a negative association between schizophrenia and creativity. Similar to postulations made by other researchers (Abraham, 2014; Carson, 2011; Eysenck, 1995), they also hypothesize the presence of an inverted-U type function between creativity and psychopathological traits, such that mild levels of schizophrenia symptoms (but not fully manifested schizophrenia) can confer advantages in creative thinking. A series of meta-analyses of the link between creativity and mood disorders, in contrast, largely revealed a positive association,

and particularly so in relation to bipolar disorder (Taylor, 2017). For instance, apart from dysthymic disorder, mood disorders were more prevalent among creative individuals (defined as professionals or students in the domains of music, writing or the fine arts) than controls, and higher levels of creative performance were associated with cyclothymic and unspecified bipolar disorders, whereas the converse was true of dysthymic disorder. A wide range of neuropsychological deficits are associated with bipolar disorder across domains that include executive function, attention, verbal and nonverbal memory, emotional reactivity, and emotion-related impulsivity (Lima, Peckham, & Johnson, 2018). How the information processing biases that confer negative functionality in these domains of psychological function also confer positive functionality within the domain of creativity is still an open question.

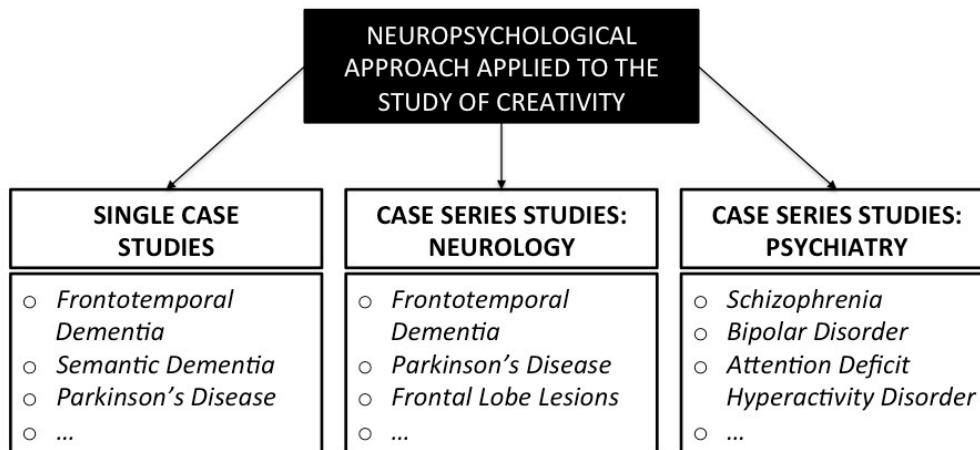
In conclusion, the promise of the neuropsychological approach in delivering many pieces of the puzzle in understanding the workings of the creative mind is not one that has been capitalized on by psychologists, neurologists and neuroscientists nearly enough. The neuropsychological approach enables us to make substantial and unique gains in knowledge that are not afforded by the alternative approaches of neuroimaging and electrophysiology, which are inherently limited by several conceptual and methodological problems (Abraham, 2018). The scientific literature provides us abundant behavioral tasks of creative function that can readily be employed on many different neurological and psychiatric populations of interest. These can be used in combination with chosen behavioral tasks of non-creative function (and even physiological assessments) that are especially relevant in light of

the neuropsychological profile of the population in question to verify whether our theoretical postulations regarding the mechanisms underlying creative cognition (e.g., disinhibition abets creative thinking) actually bears out in empirical investigations of the same. The neuropsychological approach bodes truly fruitful opportunities for the scientific exploration of core ideas in creativity.

Figure Legends

Figure 1: The three approaches employed in the neuropsychology of creativity and examples of populations that have been studied within each approach.

Figure 1



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