



# The neuropsychology of creativity

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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 promise of this approach and the unique perspective it affords, it has only been limitedly utilized when exploring creative cognition. This paper presents a brief 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 approach has delivered. In doing so, the aim is to make a case for the utility of the neuropsychological approach in allowing for a better understanding of the creative mind.

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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 [1], is primarily explored through neuroimaging and EEG-based approaches [2••]. Neuropsychological studies are relatively uncommon [3••]. This is extremely unusual given the unmistakable usefulness 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 such functions [4]. 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

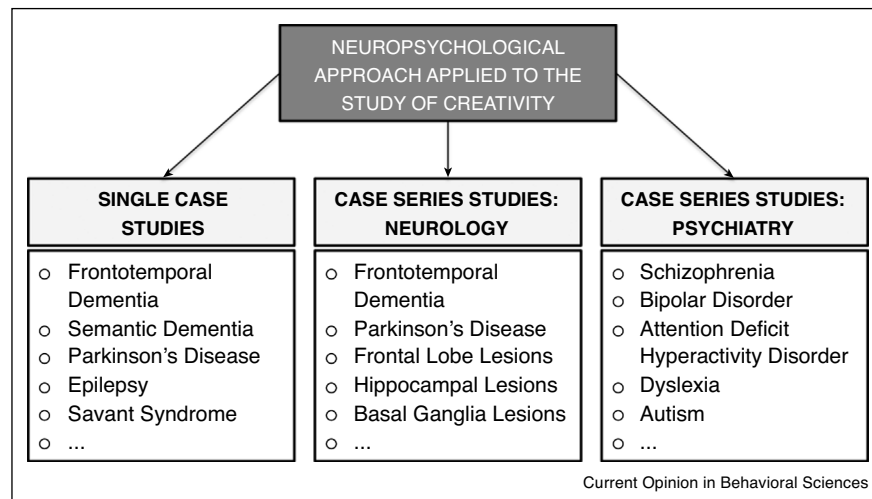
that that the implicated brain regions are not only involved in the said functions, they are likely to be vital for the same. Justifiable critiques of the approach notwithstanding [5•,6], it is undeniable that this approach has been instrumental in delivering key knowledge on the workings of the human mind as typically showcased by the iconic case studies of HM [7], Phineas Gage [8], and Tan [9] among many others. In fact, these classic cases continue to be influential even in contemporary studies that map brain structure to brain function [10].

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 [11,12•,13–15], it can be maintained that following a multipronged approach would logically 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 the rationale underlying the linking of specific subclinical personality traits and their associated information processing biases in relation to individual differences in creative cognition [16,17].

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 [18–21]. 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 before the onset of FTD [22,23]. This tendency to engage in artistic expression is not short-lived; it tends to be compulsive and highly sustained. Indeed, in the first published report to showcase this phenomenon, all three

Figure 1



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

patients went on to become accomplished painters [24]. 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 [25,26] as well as in Parkinson's disease [27].

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 been put forward to explain this phenomenon of what can be seen as evidence of 'paradoxical functional facilitation' [28] 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 [29]. In fact, longitudinal studies of the creative output of artists who developed FTD [30], PPA [31], left brain injury post-stroke [32] and Parkinson's disease [33,34] 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 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 [35].

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. Disorders of interest include FTD [36,37], SD [37], Parkinson's disease [38,39,40] and savant syndrome [41–43], as well as patients with lesions of the frontal lobe [44–46], the hippocampus [47,48], the parieto-temporal cortex [44,46], and the basal ganglia [44].

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 [37], they also demonstrated poor performance on standardized

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