

VU Research Portal

Cognition and behaviour of prefrontal lobe damage

Jonker, F.A.

2019

document version

Publisher's PDF, also known as Version of record

[Link to publication in VU Research Portal](#)

citation for published version (APA)

Jonker, F. A. (2019). *Cognition and behaviour of prefrontal lobe damage*.

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal ?

Take down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

E-mail address:

vuresearchportal.ub@vu.nl

This chapter summarizes and discusses the main findings of the studies described in this thesis.

In **Chapter 2**, we described the association between both traditional cognitive tests and tests that are sensitive to the OFC and behavioural disorders. The results of the 15 included studies with described OFC lesions, including case studies, showed a variety of descriptions of behavioural changes (clinical descriptions, DSM-IV diagnoses, questionnaires). We found that some, yet not all, suggested tests are related to behavioural changes after OFC lesions. More specifically, there are three ‘types’ of behaviour following OFC lesions, each with a different underlying cognitive function: 1) behavioural disinhibition related to reversal learning; 2) rigid antisocial behaviour related to deficits in recognition of expressed emotion; 3) impulse-control behaviour related to the IOWA gambling task. Because of the large differences in usage and scoring of the Faux pas test (e.g. the distinction between cognitive and affective ToM), no association was found with specific behaviour. We did find that the traditional executive tests, such as STROOP, Trail Making Test (TMT) and Wisconsin Card Sorting Test, are not sensitive to OFC lesions despite the assumption that executive dysfunctions are related to behavioural changes following frontal lobe lesions.

In **Chapter 3** we describe an exploratory study using graph analysis (i.e. networks) on different tests of traditional executive functioning and behavioural measures for three groups: frontal lesions ($n = 61$), non-frontal brain damage ($n = 66$) and controls ($n = 67$). Cognitive measures used were the Stroop Color Word, TMT, Letter fluency (DAT), 15 word learning test (15T), Recall (15R), and digit span for cognitive functioning; for behavioural changes we used the FrSBe. Networks were estimated in R (R Core Team, 2016) using the *glasso*, *bootnet* and *qgraph* packages. We found three different cognitive phenotypes per

group. Cognitive phenotypes are specific patterns of relations between EF tests: clusters of cognitive tests that are highly interconnected with each other but not with other clusters. The frontal cognitive phenotype involves merely mental processes (working memory, letter fluency, verbal memory) and does not involve visual-motor tasks (TMT, Stroop). No degree of centrality was found between groups for the cognitive and behavioural nodes. Centrality determines how 'central' a variable (node) is within a network, that is, if the node is connected to many other nodes (degree of centrality) [1]. The total performance of the 15T has the most centrality among all tests in all the groups, which means that other cognitive functions are strongly associated with 15T. Both 15T and 15R form a community of 100% for all groups, meaning that 15T and 15R do not differentiate between groups. We found different proportions in the community assignments of nodes between groups. Digit Span backwards (84%) and Fluency (79%) form a community with 15T and 15R in the frontal group but not in the control (25%) and non-frontal (35%) groups. We concluded that the different cognitive clusters, each with a different stability and proportion, might be theoretically interpreted as a potential compensatory mechanism for not finding executive deficits following frontal lobe lesions. As this was an exploratory study, we were not able to compare the results with other studies.

In **Chapter 4**, we tested 18 PFC-damaged patients (4 right, 5 left, 5 bifrontal lobe lesions and 4 with left OFC and basal temporal lesions) using both the JEF[®] and traditional executive tests. Constructs of the JEF[®] were expected to tap onto the OFC functions (e.g. learning and adapting to changing reinforcement contingencies and decision-making tasks) and correlate with the FrSBe. We found no correlation with the FrSBe subscales on self-report and the JEF[®] constructs. Only the Stroop interference score correlated with the Adaptive subscale of the JEF[®]. Lengenfelder and colleagues (2013) suggested that information from the

self-report measure of the FrSBe differed from information gathered from cognitive tests [2]. It is proposed more often that different constructs are being measured to explain the lack of association between cognitive tests and questionnaires [3]. This would imply that a self-report measure for behavioural and cognitive tests does not measure aspects of the same construct as the tests intend to. We did show that the FrSBe Executive dysfunction subscale for proxy ratings displayed significant correlation with the total score of JEF[®]. Lengenfelder and colleagues found no differences in self-report and proxy ratings, but other studies do [4]. Besides, overestimation of behavioural functioning is an often-seen consequence of brain injury [5, 6]. Our findings also support the conclusion that self-report questionnaires might not be sufficient due to alleged behavioural self-awareness deficits. We did conclude the ecological validity of the JEF[®] in relation to the proxy ratings. Based on the above and given the fact that our study only examined lesions in the OFC region with some additional temporal lesions, one can conclude that the cognitive constructs of the JEF[®] do not correlate with self-report behavioural changes following PFC lesions.

In **Chapter 5**, we studied whether behavioural changes following dmPFC and OFC lesions (total $n = 7$), compared with a control group ($n = 22$), are related to deficits in behavioural self-awareness. Behavioural self-awareness was calculated by subtracting family ratings from the patient's self-rating on the FrSBe subscales. A major finding of our study is that the inability to empathize with others (deficits in affective ToM) is due to specific OFC lesions, whereas a lack of behavioural self-awareness (FrSBe Apathy subscale) seems to be due to dmPFC lesions, irrespective of cognitive functioning. This is in line with literature stating that 'affective ToM' is more related to the OFC [7–9]. It has been suggested that the dmPFC (including ACC) does not play a crucial role in cognition per se, but is involved in associating executive functioning with activation of the autonomic nervous system [10, 11].

This is in line with the well-established knowledge, and our results, that lesions to the dmPFC can result in clinically reduced drive and internally motivated behaviour [12]. Self-report questionnaires might not suffice due to behavioural self-awareness deficits caused by dmPFC lesions. We concluded that clinical neuropsychological assessment must be supplemented with MRI data and proxy ratings at the behavioural level in order to link specific behaviour to location of lesion.

In **Chapter 6**, we compared a very small right AHE group ($n = 3$) with controls ($n = 20$) on a broad neuropsychological test battery and several behavioural-psychiatric questionnaires, including proxy ratings. We found a significant difference in affective ToM between groups. In line with previous studies on focal lesions, we confirmed that right hemisphere lesions cause deficits in affective ToM [13, 14]. An unexpected finding is that patients with AHE have the tendency to report significantly fewer negative symptoms on questionnaires, while the proxy rating showed more behavioural problems. These self-awareness deficits (both on behavioural and cognitive questionnaires) in right AHE patients, compared to controls, can be labelled as an 'active coping style'. This discrepancy between self and proxy ratings means that one must be cautious in the choice of empathy measure, as some studies use self-rating questionnaires as a measure of empathy [15]. No difference was found on cognitive measures between the AHE patients and controls. [11]. Overall, patients with right AHE show better improvement on cognitive tests [15–17]. Some suggest that this improvement on cognitive function after right AHE is due to activation of the left hippocampus [18]. This is in line with studies that suggest a lateralization of the amygdala-hippocampus complex in processing both cognitive and affective information, where the left side processes verbal information and the right side visual stimuli [19, 20]. We concluded that these patients end up in (neuro-)psychiatry due to their deficits in monitoring their own

behavioural-psychiatric problems. These findings should be taken into account in the immediate aftercare of AHE patients.