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Article (Accepted Version)

Chan, Edgar, MacPherson, Sarah E, Bozzali, Marco, Shallice, Tim and Cipolotti, Lisa (2018) The influence of fluid intelligence, executive functions and premorbid intelligence on memory in frontal patients. *Frontiers in Psychology*, 9 (926). p. 926. ISSN 1664-1078

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1 **The influence of fluid intelligence, executive functions and**
2 **premorbid intelligence on memory in frontal patients**

3
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1 **The influence of fluid intelligence, executive functions and premorbid**
2 **intelligence on memory in frontal patients**

3 **Abstract**

4 **Objective:** It is commonly thought that memory deficits in frontal patients are a result of
5 impairments in executive functions which impact upon storage and retrieval processes. Yet,
6 few studies have specifically examined the relationship between memory performance and
7 executive functions in frontal patients. Furthermore, the contribution of more general
8 cognitive processes such as fluid intelligence and demographic factors such as age, education
9 and premorbid intelligence has not been considered. **Method:** Our study examined the
10 relationship between recall and recognition memory and performance on measures of fluid
11 intelligence, executive functions and premorbid intelligence in 39 frontal patients and 46
12 healthy controls. **Results:** Recall memory impairments in frontal patients were strongly
13 correlated with fluid intelligence, executive functions and premorbid intelligence. These
14 factors were all found to be independent predictors of recall performance, with fluid
15 intelligence being the strongest predictor. In contrast, recognition memory impairments were
16 not related to any of these factors. Furthermore, age and education were not significantly
17 correlated with either recall or recognition memory measures. **Conclusion:** Our findings
18 show that recall memory in frontal patients was related to fluid intelligence, executive
19 functions and premorbid intelligence. In contrast, recognition memory was not. These
20 findings suggest that recall and recognition memory deficits following frontal injury arise
21 from separable cognitive factors. Recognition memory tests may be more useful when
22 assessing memory functions in frontal patients.

23
24 **Keywords: frontal lobes; recall; recognition; memory intelligence; executive functions**

1. Introduction

It is well-documented that frontal lobe lesions can result in memory difficulties (Kopelman, 2002; Wheeler, Stuss & Tulving, 1995). Memory impairments that result from frontal lobe lesions are thought to be distinct from pure amnesia, which arises from dysfunction of the diencephalon or temporal brain regions (Buckner, Kelly & Petersen, 1999). However, the exact nature of frontal lobe memory impairment is still somewhat unclear. For example, it is still debated whether frontal memory impairment manifests as a deficit in recall, recognition or both recall and recognition. Some argue that only recall memory is impaired while recognition memory remains relatively preserved (e.g., Janowsky, Shimamura, Kritchevsky & Squire, 1989; Milner, Corsi & Leonard, 1991). Others have reported impairments in both recall and recognition (e.g., Alexander, Stuss & Fansabedian, 2003; Baldo, Delis, Kramer & Shimamura, 2002). Recently, memory performance in a large cohort of frontal patients was assessed using the Doors and People battery (Baddeley et al., 1994) which consists of verbal and visual recall and recognition tasks thought to be comparable in terms of difficulty (MacPherson, Turner, Bozzali, Cipolotti & Shallice, 2016). Frontal patients were found to be significantly impaired on both recall and recognition memory tasks compared to healthy controls. However, in line with the pattern of deficits found in an earlier meta-analysis (Wheeler et al., 1995), the effect sizes were greater for recall compared with recognition memory impairment, suggesting that recall memory is more affected following frontal lobe damage.

Although it is commonly thought that frontal memory impairments are secondary to impairment in executive processes, surprisingly few studies have directly examined the relationship between executive dysfunction and memory impairment. In list learning tasks, it is suggested that executive deficits in frontal patients cause a breakdown in top-down supervisory processes. This breakdown leads to the poor use of organisational strategies such as spontaneous categorisation and semantic linkages during memory encoding, and poor search strategies and self-monitoring during memory retrieval (Baldo & Shimamura, 2002). An assumption then is that individuals with greater executive dysfunction will likely have greater memory deficits. Indeed, in the aging literature, it has been argued that memory difficulties in older adults are related to increased vulnerability to executive deficits due to age-related frontal-striatal changes (see Buckner, 2004 for a review). Executive functions have been shown to mediate the relationship between the effects of age and recall memory performance (Crawford, Bryan, Luszcz, Obonsawin & Stewart, 2000; Troyer, Graves & Cullum, 1994). Similarly, in early mild Alzheimer's disease, recall memory performance has been shown to be correlated with performance on executive tasks (Baudic et al., 2006).

In patients with frontal lobe lesions, there has generally only been indirect support for the notion that memory impairments are related to executive deficits. A common finding is that word list-learning performance can be improved in frontal patients by explicitly grouping to-be-remembered words into semantic categories during encoding and by providing category cues during recall, thereby presumably reducing the 'executive load' of the task (e.g., della Rocchetta & Milner, 1993; Gershberg & Shimamura, 1995, but see Turner, Cipolotti, Yousry & Shallice, 2007). Only a very few studies have explicitly examined the relationship between memory performance and performance on executive tasks in frontal patients. In one study, a correlation was found between recall memory performance (total number of words recalled) and phonemic fluency performance (FAS) in left dorsolateral frontal patients (Alexander et al., 2003). Interestingly, no similar correlation between fluency and recognition memory performance was found. However, no other executive measures were included in this study, limiting the conclusions that can be drawn.

1 Besides executive processes, more general cognitive processes may also contribute to
2 memory performance in frontal patients. One prime candidate is fluid intelligence. Deficits in
3 executive tasks in frontal patients have been argued to be underpinned by impairments in
4 fluid intelligence (Duncan et al., 2000). In support of this, it has been shown that differences
5 in performance on some executive tasks between frontal patients and healthy controls can be
6 largely or entirely accounted for by performance on tests of fluid intelligence (Roca et al.,
7 2010; Woolgar et al., 2010; Barbey et al., 2012; Keifer & Tranel, 2013). As such, it may be
8 that memory difficulties in frontal patients might be better explained by impairment in fluid
9 intelligence rather than executive functions. Indeed, fluid intelligence has been found to be
10 the strongest predictor of episodic memory performance in healthy individuals (Aizpurua &
11 Koutstaal, 2010).

12 We have also previously found that demographic factors such as age, years of
13 education and premorbid intelligence, as measured by literacy attainment assessed using the
14 National Adult Reading Test (NART IQ; Nelson & Willison, 1991), can significantly impact
15 on executive impairments and fluid intelligence following frontal lobe injury (Cipolotti et al.,
16 2015a; MacPherson et al., 2017). In a large cohort of frontal patients, we have shown that age
17 and NART IQ are strongly correlated and predictive of performance on two executive tasks,
18 verbal fluency and the Stroop Colour Word test, over and above other factors such as lesion
19 severity and chronicity. In addition, age, years of education and NART IQ are also related to
20 fluid intelligence, though age seems to account for most of the unique variance. Indeed, age
21 has been shown to exacerbate impairments in executive functions and fluid intelligence
22 following frontal lesions (Cipolotti et al., 2015b). Whether these variables might also be
23 related to, or mediate, memory performance following frontal lobe injury has yet to be
24 investigated.

25 The aim of the current study was to increase our understanding of how executive
26 processes relate to memory performance in patients with frontal lesions. Specifically, we
27 wanted to examine the relationship between recall and recognition memory performance and
28 age, education, premorbid intelligence, fluid intelligence and executive functions.

29 **2. Material and methods**

30 **2.1 Participants**

31 Thirty-nine patients (24 males, 15 females) with focal frontal lesions were
32 prospectively recruited from the National Hospital for Neurology and Neurosurgery, Queen
33 Square, London as part of two larger studies examining cognitive functions of the frontal
34 lobe. Patients had an absence of psychiatric disorders, history of alcohol or substance abuse
35 or previous neurological disorders. Frontal lesions were traced and classified by a neurologist
36 who was blind to the study results based on MRI scans (or CT scans if MRI was unavailable).
37 The aetiologies of the lesions were: glioma = 20; meningioma =14; subarachnoid
38 haemorrhage =1; anterior communicating aneurysm = 3; and traumatic brain injury=1.
39 Importantly, we have previously shown that the grouping together of frontal patients with
40 different aetiologies for the purposes of examining cognitive variables is methodologically
41 justifiable (Cipolotti et al., 2015a). Sixteen patients had lesions confined to the left
42 hemisphere, 18 patients to the right hemisphere and 5 patients had bilateral lesions. **The**
43 **majority of patients had lesions confined to the frontal lobes (n=30; see Supplementary Table**
44 **1).** The mean time since injury to assessment was 3.34 months (SD=8.12 months). In
45 addition, 46 healthy controls (HCs; 21 males, 25 females) with no history of neurological or
46 psychiatric disorders were included for comparison. The study was approved by the National

1 Hospital for Neurology and Neurosurgery & Institute of Neurology Joint Research Ethics
2 Committee and written informed consent was gained according to the Declaration of
3 Helsinki.

4 **2.2 Material and procedure**

5 *2.2.1 Baseline neuropsychological assessment*

6 All patients and HCs were assessed on a series of baseline neuropsychological
7 measures. Premorbid level of optimal functioning ('Premorbid intelligence') was estimated
8 using the National Adult Reading Test (NART; Nelson & Willison, 1991). Naming ability
9 was assessed using the Graded Naming Test (GNT; McKenna & Warrington, 1983) and
10 perceptual ability was assessed using the Incomplete Letters subtest from the Visual Object
11 and Space Perception Battery (VOSP; Warrington & James, 1991).

12 *2.2.2 Fluid intelligence*

13 Fluid intelligence was assessed using Raven's Advanced Progressive Matrices
14 (RAPM; Raven, 1976); an untimed, relatively culture-free, non-verbal test of abstract
15 reasoning. The test requires the selection of the missing piece of a visual pattern from eight
16 possible choices. The total number of correct responses in Set 1 (/12) was recorded and
17 converted into age-adjusted scaled scores based on published norms.

18 *2.2.3 Executive functions – Verbal fluency, Stroop Colour Word test*

19 Two widely-used neuropsychological tasks were administered to assess different
20 aspects of executive functioning. These two tasks were chosen because they have been shown
21 to require executive processes that are distinct from that which can be accounted for by fluid
22 intelligence (Cipolotti et al., 2016; Cipolotti et al., 2018). Verbal generation was assessed
23 using the standard phonemic fluency test ('FAS'; Benton & Hamsher, 1976). The total
24 number of words recalled for all three letters, excluding errors (i.e., proper nouns or
25 repetitions), was recorded. Verbal response inhibition was assessed using the Trenerry et al.
26 (1989) version of the Stroop Colour Word test which requires participants to name the ink
27 colour of 112 colour words (e.g., say 'Blue' when the word Red is written in blue) printed on
28 one A4 sheet. The time taken to read all 112 words was recorded in seconds.

29 *2.2.4 Recall and Recognition memory*

30 All patients and HCs were assessed on a verbal list-learning recall memory test
31 ('Trieste Test'; Turner et al., 2007). Participants were asked to recall six 16-word lists that
32 were each composed of four words from four different semantic categories (for further details
33 on the construction of the word lists and semantic categories, see Turner et al., 2007). For
34 each word list, words were either grouped according to their category ('Blocked') or they
35 were mixed ('Unblocked'). These two types of lists (Blocked or Unblocked) were presented
36 in an alternating fashion across the task (i.e., blocked, unblocked, blocked etc...). For each
37 16-word list, each word was presented on a computer screen for 2 seconds with a 1 second
38 interval between words. Following the list presentation, participants immediately completed a
39 distractor task for 30s (add 1 to a series of random numbers ranging from 1-99). Then,
40 participants were asked to recall as many words as they could from the prior list ('Uncued
41 recall'). Once this was exhausted, the four semantic category labels were provided as prompts
42 (e.g., jewels, occupations) for further recall ('Cued recall'). The total number of words
43 correctly recalled from each list before and after cueing was recorded, as well as separately

1 for blocked and unblocked word lists. We also recorded the total number of errors made
2 during recall (i.e., intrusions of words that were not presented).

3 A subset of frontal patients (n=22) and HCs (n=29) also completed the Doors and
4 People Test battery ('D&P'; Baddeley et al., 1994) which contained two recall tasks and two
5 recognition tasks. Administration was conducted in accordance with procedures outlined in
6 the manual. In brief, the verbal recall task required participants to learn and recall the names
7 of four characters and their associated occupation, while in the visual recall task, participants
8 had to copy and recall four simple line drawings. In both the verbal and visual recall tasks,
9 participants were given three learning and recall trials. Points are awarded for recalled
10 information across all three learning trials and the scores for the two recall tasks were
11 combined to create an age-adjusted recall memory scaled score ('D&P Recall'). For the
12 recognition tasks, participants were asked to remember two sets of 12 stimuli presented for 3s
13 each; the targets were either male/female names in the verbal condition and photographs of
14 different types of doors in the visual condition. Participants were then asked to recognize the
15 target among three distractors. Points were awarded for each correctly identified target and
16 combined to create an age-adjusted recognition memory scaled score ('D&P Recognition').

17 A second smaller subset of frontal patients (n=15) also completed a 30-item three
18 forced choice version (RMT-30) of the classic 50-item two forced choice Recognition
19 Memory Test (Warrington, 1984). In the learning phase, participants were asked to
20 remember 30 photographs of faces presented for 3s each. Photographs were of unfamiliar
21 Caucasian male faces with non-distinctive facial types. Participants were explicitly told to
22 remember the faces and to decide whether the faces were 'pleasant' or 'unpleasant' to
23 encourage encoding. In the recognition phase that immediately followed, target faces were
24 presented again with two distractors each. The total number of targets correctly identified was
25 recorded. Raw scores were converted to z-scores based on available normative data from a
26 separate healthy control sample (see Supplementary Table 2).

27

28 2.3 Statistical analyses

29

30 Statistical analyses were carried out using IBM SPSS Statistics 22
31 (<http://www01.ibm.com/software/analytics/spss/>). Firstly, we investigated differences
32 between frontal patients and HCs, and between left and right frontal patients, on demographic
33 variables and performance on baseline neuropsychological tests, measures of fluid
34 intelligence and executive functions using independent samples t-tests for continuous
35 variables and chi-square test for categorical variables. Performance differences on memory
36 tasks between groups were examined using mixed-design repeated measures Analysis of
37 Variance (ANOVA), except for RMT-30, where patient performance was evaluated using a
38 one-sample t-test with a mean z-score of 0, as healthy control data were not available. An
39 independent samples t-test was again used to compare differences between left and right
40 frontal patients.

41 Secondly, we examined the relationship between recall and recognition memory
42 performance and the different clinical and cognitive variables using two-tailed bivariate
43 Pearson correlation analyses, for the frontal patients only.

44 Finally, for measures that were found to be significantly correlated with memory
45 performance in our frontal patients, we ran a 3-stage hierarchical multiple regression to
46 examine the independent predictive value of each variable. We chose a hierarchical approach
47 because we were particularly interested in how executive functions predicted performance

1 over and above any influences of general intelligence. Our previous work has shown that
2 premorbid intelligence as measured by the NART is the best predictor of cognitive
3 performance in frontal patients (e.g., MacPherson et al., 2017) and so this was entered in
4 stage 1. Fluid intelligence was entered at stage 2 given that it has been argued to account for
5 variance in executive deficits in frontal patients (e.g. Duncan et al., 2000). In stage 3, the two
6 executive measures (Stroop Colour Word test and verbal fluency) were entered together using
7 a forced entry approach as we did not have an a priori hypothesis about the way in which
8 each executive test might contribute to memory performance.

9 For results where p -values were less than 0.05, effect size and r -squared values were
10 reported. For results where p -values were equal or greater than 0.05, additional Bayesian
11 analyses were conducted where appropriate to determine the extent to which the odds were in
12 favour of supporting the null-hypothesis (Gallistel, 2009). According to Jeffreys (1961), odds
13 less than 3 are “weak”, odds between 3 and 10 are “substantial”, and odds between 10 and
14 100 are “strong”.

15 16 **3. Results**

17 *3.1 Demographic and baseline neuropsychological measures*

18 Independent samples t -tests revealed that the frontal patient and HC groups did not
19 significantly differ in terms of age ($p > 0.1$, *Odds=3.58*), premorbid intelligence ($p = 0.077$,
20 *Odds=1.81*) and years of education ($p > 0.1$, *Odds=8.33*; see Table 1a). Chi-squared analysis
21 showed no significant difference in gender ($p > 0.1$). Patients were significantly poorer at
22 naming than HCs ($t(83) = 3.04$, $p < 0.01$, $d = 0.65$) but there was no difference in performance on
23 the test of visuo-perception (VOSP: $p > 0.1$, *Odds=8.23*). Left and right frontal patients were
24 well-matched on the demographic measures ($p > 0.1$; see Table 1b). There was also no
25 difference in performance between left and right frontal patients on naming or visual
26 perception ($p > 0.1$, *Odds=4.22* and *Odds=4.12* respectively).

27 *3.2 Fluid intelligence and executive functions*

28 Compared to HCs, the frontal patients had significantly lower scores on the test of
29 fluid intelligence ($t(81) = 2.11$, $p = 0.038$, $d = 0.46$). Not unexpectedly, the frontal group also
30 performed significantly more poorly compared to HCs on the two measures of executive
31 function - verbal fluency ($t(82) = 5.97$, $p < 0.001$, $d = 1.30$) and Stroop Colour Word test
32 ($t(54) = 2.68$, $p = 0.01$, $d = 0.69$). Table 1a shows the mean scores for each of the tests for the
33 two groups. The difference between patients and HCs remained significant when we co-
34 varied for fluid intelligence (Verbal fluency: $p < 0.001$; Stroop Colour Word test: $p = 0.02$).

35 Within the frontal group, no significant difference was found between left and right
36 frontal patients on the test of fluid intelligence ($p > 0.1$, *Odds=4.01*). In contrast, patients with
37 left frontal lesions were found to generate significantly fewer words on verbal fluency
38 ($t(31) = -2.18$, $p = 0.037$, $d = 0.76$) and were slower on the Stroop Colour Word test compared
39 with patients with right frontal lesions ($t(18) = 3.69$, $p = 0.002$, $d = 1.65$). The difference
40 between left and right frontal patients remained significant when we co-varied for fluid
41 intelligence (Verbal fluency: $p = 0.021$; Stroop Colour Word test: $p = 0.002$). **Table 1b shows**
42 **the mean scores for each of the tests for the two groups.**

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3.3 Recall memory

Performance on the Trieste test of verbal list-learning was examined using a mixed-design repeated measures Analysis of Variance (ANOVA) with 2 within-subjects factors of Block (Blocked, Unblocked) and Cue (Cue, Uncued) and 1 between-subjects factor of Group (Patients, HCs). There was a significant main effect of Group in which patients recalled fewer words than HCs ($F(1, 83)=6.90, p=0.01, \eta_p^2= 0.08$). There was a significant main effect of Block ($F(1, 83)= 10.63, p=0.002, \eta_p^2= 0.67$) and Cue ($F(1, 83)= 170.52, p<0.001, \eta_p^2= 0.11$) showing that word-lists that were semantically blocked during presentation and providing cues improved recall performance. Crucially however, there was no significant interaction between either factors with Group (Patients or HCs; $p>0.1$). That is, frontal patients did not significantly benefit from blocking or cueing more than HCs (see Table 2a). There was no significant difference in the number of recall errors made between the frontal patients ($M (S.D.)=5.69 (3.95)$) and HCs ($M (S.D.)=4.70 (4.25)$).

Recall performance on the Doors and People test was examined using a mixed-design repeated measures ANOVA with 1 within subjects factor of domain (verbal, visual) and 1 between-subjects factor of Group (Patients, HC). Frontal patients scored significantly more poorly compared to healthy controls overall ($F(1, 50): 7.71, p=0.008, \eta_p^2= 0.13$). There was no significant effect of domain ($p=0.1$) and no interaction between domain and group ($p>0.1$), suggesting that performance on the two recall subtests were relatively comparable.

Within the frontal group, there was no significant difference in the total words recalled on the Trieste test between patients with left and right sided lesions ($p>0.1, Odds=1.69$) and on D&P Recall ($p>0.1, Odds=3.14$; see Table 2b).

3.4 Recognition memory

Recognition performance on the Doors and People test was examined using a mixed-design repeated measures ANOVA with 1 within subjects factor of domain (verbal, visual) and 1 between-subjects factor of Group (Patients, HC). Frontal patients scored significantly more poorly compared to healthy controls overall ($F(1, 50)=6.85, p=0.012, \eta_p^2= 0.12$). There was a significant effect of domain ($F(1, 50)=17.75, p<0.01, \eta_p^2= 0.26$) which showed that the visual recognition test was significantly harder overall ($M (S.D.)=9.47 (0.43)$) compared with the verbal recognition memory test ($M (S.D.)=11.71 (0.46)$). However, there was no significant interaction between domain and group ($p>0.1$).

On the RMT-30, z-score performance of frontal patients was assessed using a one-sample t-test (Mean z-score=0). Mean z-score performance of the frontal patients was statistically different from zero ($t(14)=-2.32, p=0.036, d=0.60$).

Within the frontal group, as with recall performance, there was no significant difference on D&P Recognition between patients with left and right sided lesions ($p>0.1, Odds=3.20$) and on RMT-30 ($p>0.1, Odds=1.30$).

Insert Table 2a and 2b here

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3.6 Relationship between memory performance and the clinical and cognitive variables

We conducted two-tailed bivariate Pearson correlation analyses to examine the relationship between recall and recognition memory performance in frontal patients and their clinical and cognitive variables. Given the lack of significant difference in performance between the left and right frontal patients on all memory measures, the two groups were combined in all correlation and regression analyses to increase power. To reduce the likelihood of false-positives, only the main memory measures that were found to be meaningfully impaired compared with healthy controls were included in the analysis; the Trieste test Total Score, D&P Recall, D&P Recognition, and the RMT-30. Clinical variables included were age, years of education and premorbid intelligence as assessed by the NART. Cognitive variables included were fluid intelligence as measured by Raven's Progressive Matrices and the two executive measures of verbal fluency and the Stroop Colour Word test.

Both recall memory measures were significantly correlated with premorbid intelligence (Trieste test, $p=0.001$; D&P Recall, $p=0.007$), fluid intelligence (Trieste test, $p=0.002$; D&P Recall, $p<0.001$), and verbal fluency (Trieste test, $p<0.001$; D&P Recall, $p=0.026$). Only performance on the Trieste test was related to verbal response inhibition as assessed using the Stroop test ($p=0.035$) but not D&P Recall ($p>0.1$). Performance on the two recall measures were significantly correlated with each other ($p<0.001$). Neither recall memory measures were correlated with age or years of education. The absolute Pearson's correlation coefficient between the two recall memory measures and the clinical and cognitive variables are shown in Figure 1.

In contrast, neither recognition memory measures, D&P Recognition or RMT-30, were correlated with premorbid intelligence ($p>0.1$), fluid intelligence ($p>0.1$) or either executive measures (see Figure 2). Neither recognition memory measures were correlated with age or years of education.

Insert Figure 1 & 2 here

3.7 Predictors of recall memory performance

Given that the recall memory measures were significantly correlated with premorbid intelligence, fluid intelligence and performance on the executive tasks, we examined the relative predictive value of these three variables using a **3-stage hierarchical multiple regression. Premorbid intelligence was entered at stage 1, fluid intelligence was entered at stage 2 and the two executive tasks (Stroop Colour Word test and verbal fluency) were entered at stage 3.**

Using performance on Trieste Total Recall as the dependent variable, the hierarchical multiple regression revealed that at **stage 1**, premorbid intelligence contributed significantly to the regression model ($F(1,22) = 11.76, p<0.01$) and accounted for 36% of the variance in recall memory performance. Introducing fluid intelligence at **stage 2** explained an additional 26% of the variation, explaining a total of 62% variance in recall memory performance, and this change in R^2 was significant ($F(1,20) = 13.57, p<0.01$). Adding the two executive tasks

1 explained an additional 19% of the variance to the model and this change was significant (F
2 (2,18) = 8.82, $p < 0.01$). The final model accounted for 81% of the variance in Trieste Total
3 Recall (F(4,12)=18.83, $p < 0.01$). Premorbid intelligence ($\beta = -0.45$, $p = 0.041$), fluid intelligence
4 ($\beta = 1.24$, $p < 0.01$), and Stroop Colour Word test ($\beta = -0.59$, $p < 0.01$) were all significant
5 predictors whereas verbal fluency was not ($p > 0.1$).

6 The same analysis was repeated with D&P Recall as the dependent variable. The
7 hierarchical multiple regression revealed that at **stage 1**, premorbid intelligence contributed
8 significantly to the regression model (F (1,19) = 7.73, $p = 0.012$) and accounted for 29% of the
9 variance in recall memory performance. Introducing fluid intelligence at **stage 2** explained an
10 additional 20% of the variation, explaining a total of 49% variance in recall memory
11 performance, and this change in R^2 was significant (F (1,18) = 7.09, $p = 0.016$). Unlike Trieste
12 Total Recall, adding the two executive tasks did not significantly add to the variance
13 explained by the model for D&P Recall performance ($p > 0.1$). At **stage 2**, only fluid
14 intelligence ($\beta = 0.85$, $p = 0.016$) was a significant predictor of recall performance, whereas
15 premorbid intelligence was not ($p > 0.1$).

16 Given that recognition memory performance was not correlated with any of the
17 clinical or cognitive variables, multiple regression was not performed.

18 19 **4. Discussion**

20 For the first time, we investigated how demographic factors of age and education,
21 premorbid intelligence, fluid intelligence and executive functions relate to and account for
22 recall and recognition memory performance in frontal patients. Our frontal patients were
23 found to be impaired on two different measures of recall memory and two different measures
24 of recognition memory compared with healthy controls. This finding supports previous
25 suggestions that frontal injury can result in both recall and recognition memory deficits (e.g.,
26 MacPherson et al., 2016). Crucially however, we show that the nature of these deficits may
27 be separable in how they relate to other clinical and cognitive factors.

28 For recall memory, performance in frontal patients on both recall memory measures
29 was correlated with premorbid intelligence, fluid intelligence and verbal fluency.
30 Performance on the list learning task was also related to the Stroop Colour Word test.
31 Investigation into the individual contributions of premorbid intelligence, fluid intelligence
32 and executive functions on predicting recall memory performance revealed slightly different
33 but converging results for our two measures. For the Trieste list-learning task, all three
34 variables were significant independent predictors of recall performance. Of the executive
35 tasks, although both verbal fluency and Stroop Colour Word were correlated with
36 performance, only the Stroop Colour Word test was a significant predictor of performance
37 when all variables were taken into account. Of all the significant predictors, fluid intelligence
38 was the strongest predictor of performance. For D&P Recall, fluid intelligence was the only
39 significant predictor of recall performance. Despite D&P Recall performance being
40 correlated with both premorbid intelligence and verbal fluency, neither variable contributed
41 significantly over and above the variance accounted for by fluid intelligence. Overall, our
42 findings suggest that recall memory deficits in frontal patients are best accounted for by fluid
43 intelligence. The difference in findings between our two recall measures might reflect
44 inherent differences in the two measures. The Trieste list learning task has 16-items per word
45 list and one learning trial per list whereas the D&P Recall tasks only contain 4-items and
46 have 3 repeated learning trials. Thus, it may be that the Trieste test requires greater demand

1 on supervisory processes such as strategy and inhibition to encode the multiple word lists
2 efficiently and avoid interference across lists (Baldo & Shumamura, 2002). However,
3 investigation into the differences between the demands of the tasks warrants further study.

4 The finding that recall memory in frontal patients is related to fluid intelligence
5 processes is in keeping with a specific theoretical proposal regarding the neurocognitive
6 architecture of the frontal lobe. Fluid intelligence is taken as a measure of some general or *g*
7 factor that can broadly account for performance across a range of different tasks (Duncan et
8 al., 2000). It captures the mental processes required for breaking tasks down into
9 subcomponents that are thought to be necessary to perform most cognitive tasks, particularly
10 novel or complex ones. It has been argued that fluid intelligence can be mapped onto a
11 multiple-demand (MD) network in the brain that involves predominantly frontal-parietal
12 regions (Woolgar et al., 2010). As such, damage to frontal brain regions often results in
13 impairment in fluid intelligence (Duncan et al., 2000). It has been shown that fluid
14 intelligence can account for some executive deficits that result from frontal lobe injury (Roca
15 et al., 2010). Furthering this, our data suggests that impairment in fluid intelligence
16 following frontal lesions may also account for performance in recall memory tasks.

17 Recall performance in frontal patients was also correlated with premorbid intelligence
18 as assessed by the NART but not years of education. NART was also a significant
19 independent predictor of Trieste performance. Both NART and years of education are often
20 thought of as comparable indicators of premorbid intelligence. However, we have recently
21 shown that these two variables do not represent the same proxy measure, at least following
22 frontal injury, with NART being a better predictor of executive functions (MacPherson et al.,
23 2017). Our findings further extend the important role of premorbid intelligence as measured
24 by the NART in protecting against the impact of frontal brain injury on memory functions.

25 Recall memory impairment in our frontal patients was correlated with impairment in
26 executive processes. Consistent with Alexander and colleagues (2003), we found that recall,
27 but not recognition memory was related to performance on verbal fluency. In addition,
28 Trieste recall was also related to response inhibition as measured by the Stroop Colour Word
29 Test. As far as we know, this is the first time in which the contribution of different executive
30 measures to recall memory in frontal patients has been examined independently. Previous
31 work has generally combined different executive measures into a composite, thereby limiting
32 the potential for differences between tests to be explored (e.g., Crawford, Bryan, Luszcz,
33 Obonsawin & Stewart, 2000; Troyer, Graves & Cullum, 1994). In our study, although both
34 verbal fluency and Stroop performance were correlated with recall, only performance on the
35 Stroop, but not verbal fluency, was a significant predictor independent of premorbid
36 intelligence and fluid intelligence. Our findings show that different executive functions may
37 contribute to recall performance differently. Furthermore, our findings support the notion
38 that some executive abilities are dissociable from fluid intelligence following frontal injury
39 (Cipolotti et al., 2016; Cipolotti et al., 2018). In future, it would be important to consider this
40 in further detail with a wider variety of tasks tapping different known executive functions.

41 In contrast to recall memory, performance on recognition memory measures in our
42 frontal patients were not significantly related to premorbid intelligence, fluid intelligence or
43 either executive measure. Importantly however, frontal patients were significantly impaired
44 on the recognition memory measures, which is consistent with previous findings
45 (MacPherson et al., 2016; Wheeler et al., 1995). The lack of relationship between recognition
46 memory impairment and performance on other cognitive tests suggests that recognition
47 memory performance is dissociated from premorbid intelligence, fluid intelligence and

1 executive functions. It may be that poor performance on the recognition memory task reflects
2 some genuine deficit in memory processes (Cipolotti et al., 2001). Alternatively, it has been
3 shown that poor recognition performance in frontal patients may be related to specific
4 impairment in familiarity judgements; a difficulty in frontal patients to inhibit responding
5 ‘yes’ to similar distractors (Alexander, Stuss & Fansabedian, 2003; MacPherson et al., 2008).

6 We did not find any significant relationship between performance on any of our
7 memory measures and patients’ age. In our previous work, we have demonstrated that age
8 predicts performance on executive tasks in frontal patients (MacPherson et al., 2017) and
9 modulates the magnitude of their impairment, whereby middle-aged and older frontal patients
10 had exacerbated executive impairment compared to younger adults (Cipolotti et al., 2015b).
11 This latter effect was not found for performance on non-executive tasks that do not rely on
12 frontal functions. The lack of relationship between age and memory performance in our
13 current study appears inconsistent also with what is shown in the healthy and pathological
14 aging literature (Buckner, 2004). It may be that the impact of frontal lesions decompensates
15 for any premorbid relationship between age and memory performance (but see Cipolotti et
16 al., 2015b).

17 Our study represents a first step into exploring the relationship between memory
18 performance and fluid intelligence, executive functions and premorbid intelligence in frontal
19 patients. **Given our findings, it would be important to examine these underlying mechanisms
20 further in a larger sample of frontal patients to allow for grouping of patients into different
21 subregions and more detailed examination of neuropathological factors such as proportionate
22 grey matter loss or white matter tract involvement.** It has been shown that the pattern of
23 memory impairment may vary depending on the frontal subregion injured consistent with the
24 known specialisation of function in different frontal areas (Stuss & Alexander, 2005; Turner
25 et al., 2007). It may be that factors such as premorbid intelligence and fluid intelligence
26 impact upon recall performance across frontal subregions whereas different executive
27 functions have a more location-specific effect. Furthermore, our slightly different pattern of
28 findings across our two recall memory tasks suggests a more systematic exploration of frontal
29 memory processes is necessary to further examine the different influences of fluid
30 intelligence and executive tasks on recall task demands.

31 Overall, we have shown that recall memory performance in frontal patients can
32 largely be accounted for by fluid intelligence, executive functions and premorbid intelligence.
33 **Future studies examining memory performance in frontal patients should consider how these
34 factors might mediate any deficits observed.** Although all three variables were related to
35 recall memory performance, general fluid intelligence appears to be the strongest predictor.
36 This was not replicated in recognition memory performance. Our findings suggest that it may
37 be more meaningful to assess memory functions in frontal patients using recognition
38 memory, as recall performance may likely be affected by non-memory related processes.

39 **5. Acknowledgement**

40 This work was supported by funding from the Wellcome Trust (grant numbers
41 066763, 089231/A/09/Z).

42 **6. Author Contributions Statement**

1 All authors were involved in the conception of the study, EC and SM was involved in
2 the collection of the data, EC, SM and MB were involved in the analyses of the data, EC,
3 SM, LC was involved in the writing and editing of the manuscript, MB and TS reviewed the
4 manuscript.

5 **7. Conflict of Interest Statement**

6 There is no known conflict of interest.

7 **8. References**

- 8 Alexander, M.P., Stuss, D.T., & Fansabedian, N. (2003). California Verbal Learning Test:
9 Performance by patients with focal frontal and non-frontal lesions. *Brain*, *126*, 1493-1503.
- 10 Alexander, M.P., Stuss, D.T., & Gillinham, S. (2008). Impaired list learning is not a general
11 property of frontal lesions. *Journal of Cognitive Neuroscience*, *21*, 1422-1434.
- 12 Aizpurua, A. & Koutstaal, W. (2010). Aging and flexible remembering: contributions of
13 conceptual span, fluid intelligence, and frontal functioning. *Psychology and Aging*, *25*(1),
14 193-207.
- 15 Baddeley, A. D., Emslie, H., & Nimmo-Smith, I. (1994). The Doors and People Test: A test
16 of visual and verbal recall and recognition. *Bury-St-Edmunds, UK: Thames Valley Test*
17 *Company*.
- 18 Baldo., J.V., Delis, D., Kramer, J., & Shimamura, A.P. (2002). Memory performance on the
19 California Verbal Learning Test – II: Findings from patients with focal frontal lesions.
20 *Journal of the International Neuropsychological Society*, *8*, 539-546.
- 21 Baldo, J.V., & Shimamura, A.P. (2002). Frontal lobes and memory. In A. Baddeley, B.
22 Wilson, & M. Kopelman (Eds.), *Handbook of Memory Disorders* (pp. 363-379). London,
23 UK: John Wiley & Co.
- 24 Barbey, A.K., Colom, R., Solomon, J., Krueger, F., Forbes, C. & Grafman, J. (2012). An
25 integrative architecture for general intelligence and executive function revealed by lesion
26 mapping. *Brain*, *135*, 1154–1164.
- 27 Baudic, S., Barba, G.D., Thibaudet, M.C., Smagghe, A., Remy, P. & Traykov, L. (2006).
28 Executive function deficits in early Alzheimer’s disease and their relations with episodic
29 memory. *Archives of Clinical Neuropsychology*, *21*, 15-21.
- 30 Benton, A.L. & Hamsher, K. (1976). *Multilingual Aphasia Examination Manual*. University
31 of Iowa; Iowa City.
- 32 Buckner, R.L., Kelley, W.M. & Petersen, S.E. (1999). Frontal cortex contributes to human
33 memory formation. *Nature Neuroscience*, *2*, 311-314.

- 1 Buckner, R.L. (2004). Memory and executive function in aging and AD: Multiple factors that
2 cause decline and reserve factors that compensate. *Neuron*, 44, 195-208.
- 3 Cipolotti, L., Shallice, T., Chan, D., Fox, N., Scahill, R., Harrison, G., Stevens, J. & Rudge,
4 P. (2001). Long-term retrograde amnesia...the crucial role of the hippocampus.
5 *Neuropsychologia*, 39 (2), 151-172.
- 6 Cipolotti, L., Healy, C., Chan, E., Bolsover, F., Lecce, F., White, M., Spano, B., Shallice, T.,
7 & Bozzali, M. (2015a). The impact of different aetiologies on the cognitive performance of
8 frontal patients. *Neuropsychologia*, 68, 21-30.
- 9 Cipolotti, L., Healy, C., Chan, E., MacPherson, S.E., White, M., Woollett, K., Turner, M.,
10 Robinson, G., Spano, B., Bozzali, M. & Shallice, T. (2015b). The effect of age on cognitive
11 performance of frontal patients. *Neuropsychologia*, 75, 233-241.
- 12 Cipolotti, L., Spano, B., Healy, C., Tudor-Sfetea, C., Chan, E., White, M., Biondo, F.,
13 Duncan, J., Shallice, T., & Bozzali, M. (2016). Inhibition processes are dissociable and
14 lateralised in human prefrontal cortex. *Neuropsychologia*, 93, 1-12.
- 15 Cipolotti, L. (2018). Lateralisation of verbal and non-verbal generation processes in frontal
16 patients. *Manuscript in preparation*.
- 17 Crawford, J.R., Bryan, J., Luszcz, M.A., Obonsawin, M.C. & Stewart, L. (2000). The
18 executive decline hypothesis of cognitive aging: Do executive deficits qualify as differential
19 deficits and do they mediate age-related memory decline? *Aging, Neuropsychology, and*
20 *Cognition*, 7(1), 9-31.
- 21 della Rocchetta A.I., & Milner B. (1993). Strategic search and retrieval inhibition: The role of
22 the frontal lobes. *Neuropsychologia*, 31, 503-524
- 23 Duncan, J., Seitz, R.J., Kolodny, J., Bor, D., Herzog, H., Ahmed, A., et al. (2000). A neural
24 basis for general intelligence. *Science*, 289, 457-460.
- 25 Gallistel, C.R. (2009). The importance of proving the null. *Psychological Review*, 116(2),
26 439-453.
- 27 Gershberg, F.B., & Shimamura, A.P. (1995). Impaired use of organizational strategies in free
28 recall following frontal lobe damage. *Neuropsychologia*, 33, 1305-1333.
- 29 Janowsky J. S., Shimamura A. P., Kritchevsky M., & Squire L. R. (1989). Cognitive
30 impairment following frontal lobe damage and its relevance to human amnesia. *Behavioral*
31 *Neuroscience*, 103, 548-560.
- 32 Jeffreys, H. (1961). *Theory of probability* (3rd ed.). Oxford, England: Oxford University
33 Press.

- 1 Keifer, E., & Tranel, D. (2013). A neuropsychological investigation of the Delis-Kaplan
2 executive function system. *Journal of Clinical and Experimental Neuropsychology*, *35*(10),
3 1048–1059.
- 4 Kopelman M. D. (2002). Disorders of memory. *Brain*, *125*, 2152–2190.
- 5 MacPherson, S.E., Bozzali, M., Cipolotti, L., Dolan, R.J., Rees, J.H., & Shallice, T. (2008).
6 Effect of frontal lobe lesions on the recollection and familiarity components of recognition
7 memory. *Neuropsychologia*, *46*, 3124-3132.
- 8 MacPherson, S.E., Turner, M.S., Bozzali, M., Cipolotti, L., & Shallice, T. (2016). The Doors
9 and People test: The effect of frontal lobe lesions on recall and recognition memory
10 performance. *Neuropsychology*, *30*(3), 332-337.
- 11 MacPherson, S. E., Healy, C., Allerhand, M., Spano, B., Tudor-Sfetea, C., White, M., Smirni,
12 D., Shallice, T., Chan, E., Bozzali, M. & Cipolotti, L. (2017). Cognitive reserve and cognitive
13 performance of patients with focal frontal lesions. *Neuropsychologia*, *96*, 19-28.
- 14 McKenna, P. & Warrington, E. (1983). *The Graded Naming Test*. NFER-Nelson, Windsor.
- 15 Milner B., Corsi P., & Leonard G. (1991). Frontal-lobe contribution to recency judgments.
16 *Neuropsychologia*, *29*, 601–618.
- 17 Nelson, H. E., & Willison, J. (1991). *National Adult Reading Test (NART)*. Windsor: NFER-
18 Nelson.
- 19 Raven, J.C. (1976). *Manual for the Advanced Progressive Matrices: Set 1*. Psychological
20 Corporation, San Antonio.
- 21 Roca, M., Parr, A., Thompson, R., Woolgar, A., Torralva, T., Antoun, N., et al. (2010).
22 Executive function and fluid intelligence after frontal lobe lesions. *Brain*, *133*, 234-247.
- 23 Stuss, D.T. & Alexander, M.P. (2005). Does damage to the frontal lobes produce impairment
24 in memory. *Current Directions in Psychological Science*, *14*:2, 84-88.
- 25 Trenerry M. R., Crosson B., Deboe J., & Leber W. R. (1989). Stroop Neuropsychological
26 Screening Test. *Odessa, FL: Psychological Assessment Resources*.
- 27 Troyer, A.K., Graves, R.E., & Callum, C.M. (1994). Executive functioning as a mediator of
28 the relationship between age and episodic memory in healthy aging. *Aging and Cognition*, *1*,
29 45-53.
- 30 Turner, M.S, Ciplotti, K., Yousry, T. & Shallice, T. (2007). Qualitatively different memory
31 impairments across frontal lobe subgroups. *Neuropsychologia*, *45*, 1540-1552.
- 32 Wheeler M. A., Stuss D. T., & Tulving E. (1995). Frontal lobe damage produces episodic
33 memory impairment. *Journal of the International Neuropsychological Society*, *1*, 525–536.

1 Woolgar, A, Parr, A., Cusack, R., Thompson, R., Nimmo-Smith, I., Torralva, T., et al (2010).
2 Fluid intelligence loss linked to restricted regions of damage within frontal and parietal
3 cortex. *Proceedings of the National Academy of Sciences USA*, 107, 14899-148902.

4 Warrington, E. K. (1984). *Recognition Memory Test: Manual*. Berkshire, UK: NFER-Nelson.

5 Warrington, E.K. & James, M. (1991). *The Visual Object and Space Perception Battery*.
6 Bury St Edmunds, England; Thames Valley Test Company.

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1 **Table 1a**

2 *Clinical and cognitive neuropsychological data for Patients and Healthy Controls*

	Frontal			Healthy Control		
	n	M	SD	n	M	SD
Age (years)	39	46.64	15.24	46	50.65	14.65
Education (years)	39	13.56	2.88	46	13.59	2.83
Premorbid Intelligence - NART (FSIQ)	37	107.65	13.04	44	112.09	9.19
Fluid Intelligence - Raven's Advanced Progressive Matrices (Scaled Score)	37	10.97*	3.11	46	12.30	2.62
FAS (Total words)	38	31.42**	15.41	46	49.80	12.81
Stroop Colour-Word test (sec)	23	154.40**	53.22	33	125.23	27.76

3 Difference between groups - *p<0.05, **p<0.01

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6 **Table 1b**

7 *Clinical and cognitive neuropsychological data for Left and Right Hemisphere Patients*

	Left Frontal			Right Frontal		
	n	M	SD	n	M	SD
Age (years)	16	47.19	13.09	18	46.06	15.73
Education (years)	16	14.19	2.48	18	13.56	3.09
Premorbid Intelligence - NART (FSIQ)	15	111.13	12.24	18	108.67	8.55
Fluid Intelligence - Raven's Advanced Progressive Matrices (Scaled Score)	14	11.64	3.10	18	11.00	3.01
FAS (Total words)	16	27.00*	15.68	17	38.18	13.80
Stroop Colour-Word test (sec)	10	179.79**	52.45	10	114.28	19.87

8 Difference between groups - *p<0.05, **p<0.01

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1 **Table 2a**
 2 *Recall and Recognition memory performance for Patients and Healthy Controls*

	Frontal		Healthy Control		
		M	SD	M	SD
Trieste Test	n=39			n=46	
Total Correct (/96)		60.67**	19.00	69.76	13.58
Blocked+Uncued (/48)		26.62	10.80	31.59	8.67
Blocked+Cued (/48)		31.77	9.65	35.87	6.57
Unblocked+Uncued (/48)		24.23	11.84	30.15	9.75
Unblocked+Cued (/48)		28.95	9.93	33.85	8.05
Doors and People test (D&P)	n=22			n=29	
D&P Recall (SS)		9.00**	2.86	11.48	3.40
Verbal (SS)		8.35	4.04	11.03	3.91
Visual (SS)		9.87	2.24	11.38	2.87
D&P Recognition (SS)		10.09*	3.13	11.93	2.83
Verbal (SS)		10.78	3.49	12.66	3.06
Visual (SS)		8.57	3.09	10.38	3.05
RMT-30	n=15				
RMT-30 (z-score)		-0.67*	1.11	-	-

3 Difference between groups- *p<0.05, **p<0.01

6 **Table 2b**
 7 *Recall and Recognition memory performance for Left and Right Hemisphere Patients*

	Left Frontal			Right Frontal		
	n	M	SD	n	M	SD
Trieste Test	n=16			n=18		
Total Correct/96		59.75	18.42		65.61	16.18
Doors and People test (D&P)	n=9			n=11		
D&P Recall (SS)		9.33	2.30		9.18	3.22
D&P Recognition (SS)		10.11	3.89		10.54	2.62
RMT-30	n=6			n=7		
RMT-30 (z-score)		-.82	.10		-.31	1.23

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1 **Figure 1.** Pearson's correlation coefficient (r) between the two recall memory measures and
2 the clinical and cognitive variables for frontal patients (* Sig. correlation, $p < 0.05$; **Sig.
3 correlation, $p < 0.01$). † Significant predictor of memory performance in the hierarchical
4 multiple regression.

5

6 **Figure 2.** Pearson's correlation coefficient (r) between the two recognition memory measures
7 and the clinical and cognitive variables for frontal patients.

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