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IS THE PREFRONTAL CORTEX IMPORTANT FOR FLUID INTELLIGENCE? A NEUROPSYCHOLOGICAL STUDY USING MATRIX REASONING

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Abstract

Patients with prefrontal damage and severe defects in decision making and emotional regulation often have a remarkable absence of intellectual impairment, as measured by conventional IQ tests such as the WAIS/WAIS-R. This enigma might be explained by shortcomings in the tests, which tend to emphasize measures of “crystallized” (e.g., vocabulary, fund of information) more than “fluid” (e.g., novel problem solving) intelligence. The WAIS-III added the Matrix Reasoning subtest to enhance measurement of fluid reasoning. In a set of four studies, we investigated Matrix Reasoning performances in 80 patients with damage to various sectors of the prefrontal cortex, and contrasted these with the performances of 80 demographically matched patients with damage outside the frontal lobes. The results failed to support the hypothesis that prefrontal damage would disproportionately impair fluid intelligence, and every prefrontal subgroup we studied (dorsolateral, ventromedial, dorsolateral + ventromedial) had Matrix Reasoning scores (as well as IQ scores more generally) that were indistinguishable from those of the brain-damaged comparison groups. Our findings do not support a connection between fluid intelligence and the frontal lobes, although a viable alternative interpretation is that the Matrix Reasoning subtest lacks construct validity as a measure of fluid intelligence.

INTRODUCTION

Neurological patients with damage to prefrontal cortices, especially in the ventromedial prefrontal (VMPC) sector, typically manifest disruption of complex decision making, planning, social conduct, and emotional regulation. What has made such deficits especially enigmatic is the fact that many affected patients demonstrate a remarkable preservation of conventional intelligence, as measured by standard IQ tests such as the Wechsler Adult Intelligence Scale.¹ Patient EVR, initially reported by Eslinger and Damasio (1985), is a case in point: his WAIS-R IQ scores are well into the superior range (Verbal IQ = 129; Performance IQ = 135; Full Scale IQ = 135), but he is the veritable prototype of someone with severely disrupted decision making, planning, and social conduct following VMPC damage (Koenigs & Tranel, 2006). Similar patients have been described by other investigators (e.g., Blair & Cipolotti, 2000; Burgess & Shallice, 1996; Shallice & Burgess, 1991). These findings have led to the conclusion that the behavioral and emotional dysfunction associated with VMPC damage

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¹For the purposes of the current study, it is important to distinguish the various versions of the WAIS test: the original WAIS, published in 1955; the revised WAIS-R, published in 1981; and the WAIS-III, published in 1997. We will use these designations carefully throughout the manuscript.

cannot be explained by impaired intelligence, as least as measured by standard IQ tests (e.g., Bechara, Damasio, Damasio, & Anderson, 1994; Burgess et al., 2006; Damasio & Anderson, 2003; Eslinger & Damasio, 1985). In fact, classic teaching in this domain has been that standard intelligence tests are not well suited for detecting frontal lobe dysfunction (e.g., Stuss & Benson, 1986; Teuber, 1972).

A longstanding criticism of intelligence tests such as the WAIS/WAIS-R was that there was disproportionate emphasis on measures of “crystallized” intelligence, which refers to one’s knowledge base and ability to solve routine problems relying on well-learned information (Cattell, 1963; Cattell & Horn, 1978; Sternberg, 1995). Vocabulary (defining words) and information (recalling factual data) are examples of such crystallized abilities. By contrast, the WAIS/WAIS-R batteries had weaker representation of measures of “fluid” intelligence, which refers to the ability to solve novel problems and manipulate abstract symbols with minimal dependence on previously acquired knowledge. Classic examples of tests of fluid intelligence are Raven’s Progressive Matrices (Carpenter, Just, & Shell, 1990; Raven, 1938; Raven, Court, & Raven, 1988) and Cattell’s Culture Fair Test, which includes a matrices subtest (Cattell & Cattell, 1960). The lack of detailed measurement of fluid intelligence could help explain why damage to the frontal lobes has typically not been found to adversely affect performance on the WAIS/WAIS-R tests (e.g., Warrington, James, & Maciejewski, 1986).

In a similar vein, Duncan and colleagues (Duncan, 1995; Duncan, Burgess, & Emslie, 1995) have argued that the paradox of frontal patients who manifest disrupted planning and decision making but unimpaired psychometric intelligence might be explained by a more detailed parsing of the construct of intelligence. To begin with, general intelligence is captured by the construct commonly referred to as Spearman’s *g* (Spearman, 1927). In psychometric theory, *g* is considered a general factor of intelligence that contributes to all manner of cognitive activities—in a nutshell, *g* reflects an individual’s overall tendency to perform well or less well on various tasks (Cattell, 1971; Spearman, 1927). It has been proposed that tests of fluid intelligence provide a closer estimate of *g* than do tests of crystallized intelligence (Carroll, 1993). Duncan (1995) suggested that fluid intelligence—or what is most closely related to *g*—may be strongly related to frontal lobe function.

Empirical support for this claim was provided by Duncan et al. (1995), who studied three patients with damage to the frontal lobes and healthy comparison participants matched for age and Full Scale WAIS (or WAIS-R) IQ scores. All participants were administered the Cattell Culture Fair test as a measure of fluid intelligence. The Culture Fair IQ scores for the frontal lobe patients were significantly lower than their own WAIS scores, and also significantly lower than the Culture Fair IQ scores of the normal comparison participants. Also, five patients with lesions to posterior, nonfrontal cortices did not manifest discrepancies between WAIS (or WAIS-R) and Culture Fair IQ scores. Duncan et al. emphasized that tests of fluid intelligence are most appropriate for probing the neuropsychological underpinnings of *g*, and that *g* may be largely a reflection of frontal functions. They also commented (1995, p. 267) that deficits in fluid intelligence may be especially pronounced “whenever a conspicuous frontal deficit is accompanied by high premorbid IQ,” a conclusion supported by the large WAIS IQ vs Culture Fair IQ discrepancies in the three frontal patients (22, 29, and 38 points), and by a subsequent observation that patients with frontal lobe damage and lower premorbid IQs had smaller (albeit still significant) such discrepancies (Duncan, Emslie, Williams, Johnson, & Freer, 1996).

Against this background, the enigma of unimpaired psychometric IQ in patients with frontal lobe damage and egregious behavioral defects might be explained by shortcomings in the tests, and specifically, insufficient measurement of fluid intelligence. In the WAIS-III, the most recent revision of the WAIS battery, the Matrix Reasoning subtest, was added to “enhance measurement of fluid reasoning” (The Psychological Corporation, 1997). The Matrix

Reasoning test was modeled on the Raven's Progressive Matrices, and it entails a series of increasingly difficult visual pattern completion and analogy problems (see Figure 1 for a mock example). For each, the participant is requested to choose from a multiple-choice array the item that best completes the pattern. The test does not require language, and it is untimed.

If the foregoing line of reasoning is correct, and according to the arguments set forth by Duncan and colleagues (1995, 1996), it can be predicted that damage to the frontal lobes will impair performance on Matrix Reasoning, relative to damage outside the frontal lobes. A general objective of the present study was to investigate this hypothesis. Because the frontal lobes comprise anatomically and functionally diverse sectors, we took a more refined approach and focused on various sub-regions of the frontal lobes. Specifically, and following conventional demarcations (Stuss & Knight, 2002), we investigated Matrix Reasoning performance in patients with dorsolateral prefrontal damage (Study 1), ventromedial prefrontal damage (Study 2), or combined dorsolateral plus ventromedial prefrontal damage (Study 3). And in a follow-up exploration (Study 4), we investigated the intriguing proposal from Duncan et al. (1995) that patients with frontal lobe damage and high premorbid IQ might evidence especially large impairments in fluid intelligence.

GENERAL METHOD

Participants

The participants for the experiments reported here (overall $N = 160$) were drawn from our Patient Registry in the Division of Cognitive Neuroscience, Department of Neurology, University of Iowa. In accordance with their enrollment in the Registry, the patients have stable, focal lesions. The specific etiologies for the lesions included cerebrovascular disease ($n = 101$), surgical resection (benign tumor, $n = 23$; hematoma, $n = 9$; anterior temporal lobectomy for pharmacoresistant epilepsy, $n = 16$; arteriovenous malformation, $n = 8$), or herpes simplex encephalitis ($n = 3$). With the exception of some of the temporal lobectomy patients, all of the patients had lesions that were acquired in adulthood (age 18 or greater). All of the benign tumor resections were for meningiomas, and all of these were performed when the patients were age 18 or greater. In the 16 anterior temporal lobectomy patients, there were 14 who had seizure onset before age 18, and 5 who had their operation prior to age 18. All of the epilepsy patients were anterior temporal lobectomy cases (no patients in the various frontal lobe groups described below had epilepsy). Exclusionary criteria included dementia, and a premorbid history of psychiatric disease, developmental abnormalities, and alcohol/drug abuse. We also excluded participants who could not participate validly in the standard WAIS-III battery, e.g., due to aphasia or visuoperceptual impairments (21 such patients were excluded). For the purposes of this study, participants had to complete at least five subtests from the Verbal scales and four subtests from the Performance scales, in order for Verbal IQ, Performance IQ, and Full Scale IQ to be derived. IQ scores were prorated when necessary according to WAIS-III manual instructions. All participants provided informed consent in accordance with the Human Participants Committee of the University of Iowa and Federal regulations.

Measures

Demographic data, including age, gender, education, and handedness, were specified for all participants. The participants completed the WAIS-III as part of a comprehensive neuropsychological evaluation conducted under the auspices of Patient Registry enrollment (Tranel, 2007). The primary dependent variable of interest—namely, fluid intelligence—was indexed with the Matrix Reasoning subtest of the WAIS-III. To round out the analysis, Verbal IQ, Performance IQ, and Full Scale IQ scores were derived from the WAIS-III. Also, the Vocabulary subtest was pulled out as a classic index of crystallized intelligence, to which the Matrix Reasoning subtest could be directly contrasted. In all participants, the WAIS-III data

were collected in the chronic epoch of recovery, more than 3 months after lesion onset. Chronicity data (time between lesion onset and WAIS-III data collection) were specified for all participants. The lesion analysis was performed according to standard methods of our laboratory (H. Damasio & Frank, 1992). Specifically, neuroanatomical analysis was based on magnetic resonance (MR) data obtained in a 1.5 Tesla General Electric Signa scanner with a 3D SPGR sequence yielding 1.5 mm contiguous T₁ weighted coronal cuts. (In a few participants in whom an MR could not be obtained because of a metallic clip or claustrophobia, the neuroanatomical analysis was based on computerized axial tomography [CT] data.) All neuroimaging data were obtained in the chronic epoch, contemporaneous with the neuropsychological evaluation. Each participant's lesion was reconstructed in three dimensions using Brainvox (H. Damasio & Frank, 1992; Frank, Damasio, & Grabowski, 1997).

Design and Statistical Approach

For each of Studies 1–3, we contrasted a frontal lobe target group (dorsolateral, Study 1; ventromedial, Study 2; dorsolateral plus ventromedial, Study 3) with a demographically matched brain-damaged comparison group. The brain-damaged comparison groups were, therefore, unique for each study, and included patients with damage outside the frontal lobes (to parietal, temporal, and/or occipital regions, in either left or right hemisphere). This design permits a test of whether frontal lobe damage per se is related to deficits on Matrix Reasoning and the other IQ measures. Each of the primary contrasts thereby involved a comparison of two groups (target frontal versus brain-damaged comparison) on various demographic and IQ variables. To perform the contrasts, we used *t*-tests or chi squares, and applied alpha correction for multiple comparisons (using the Bonferroni procedure) when the dependent variables were related.

STUDY 1

Study 1 tested the hypothesis that fluid intelligence would be impaired by dorsolateral prefrontal cortex (DLPC) lesions.

Participants

The participants for Study 1 included a target group—patients with lesions that included the dorsolateral prefrontal cortex (DLPC group)—and a brain-damaged comparison group (BDC group)—patients with lesions outside the frontal lobes who were matched to the target group on age, education, gender distribution, and handedness distribution. The target group included a total of 37 patients with either left ($n = 23$) or right ($n = 14$) DLPC damage. For the purposes of this study, the DLPC was defined as the lateral aspects of Brodmann areas 6, 8, and 9, and area 46. Any damage to this sector qualified the patient for entry into the target DLPC group. Many patients ($n = 25$) had damage that extended posteriorly or inferiorly beyond the DLPC sector, but none had damage to the VMPC sector (defined in Study 2 below). The BDC patients had lesions that were outside of all frontal regions.

Data for the DLPC and BDC groups are provided in Table 1. The groups were well matched, and did not differ on age ($p = .33$), education ($p = .69$), or gender distribution (equal). Handedness distribution was also comparable in the two groups (Pearson $\chi^2 p = .643$), and the groups did not differ on time since lesion onset (chronicity) ($p = .21$).

Results and Discussion

The various WAIS-III measures are provided in Table 2 (Matrix Reasoning and Vocabulary are age-corrected scaled scores). There was no indication that the DLPC group was significantly impaired on Matrix Reasoning. Their overall group mean ($M = 10.4$, $SD = 2.9$)

was slightly below that of the BDC group ($M = 11.0$, $SD = 2.9$), but not statistically different ($p = .38$; 95% Confidence Interval = -1.95 to $+0.76$). These findings do not support the hypothesis that fluid intelligence as indexed by Matrix Reasoning would be impaired by DLPC lesions.

We also looked at the Matrix Reasoning data for the left DLPC and right DLPC lesions separately, given that the test is ostensibly nonverbal (but see Baldo et al., 2005, for a demonstration that solving problems in Raven's Colored Progressive Matrices and the Wisconsin Card Sorting Test is dependent in part on language mediation), and there might be reason to expect a left-right difference (favoring the left, i.e., left-sided damage would produce less impairment than right-sided damage). In fact, the left DLPC group did have a higher Matrix Reasoning score ($M = 10.7$, $SD = 3.0$) than the right DLPC group ($M = 9.8$, $SD = 2.8$), but the difference was small and not significant ($p = .34$). Neither unilateral DLPC group differed from the BDC group ($ps > .20$).

The other WAIS-III variables were also contrasted between the DLPC and BDC groups. The groups were very similar on the Vocabulary subtest, with the means differing by only 0.3 age-corrected scaled score points² ($p = .62$; 95% Confidence Interval = -1.17 to $+1.94$). The DLPC group had slightly lower scores for all three IQ indices, on the order of some 4 to 5 IQ points, but none of the between-group differences was statistically significant: VIQ, $p = .27$, 95% Confidence Interval = -12.85 to $+3.68$; PIQ, $p = .24$, 95% Confidence Interval = -11.57 to $+2.97$; FSIQ, $p = .14$, 95% Confidence Interval = -13.44 to $+2.00$. We also contrasted the left and right DLPC groups on these IQ variables. As the data in Table 2 show, the left DLPC group had a lower Vocabulary score and lower Verbal IQ, and the right DLPC group had a lower Performance IQ (and a lower Matrix Reasoning score, as noted above), a pattern that is broadly consistent with typical verbal-left/nonverbal-right laterality in DLPC systems, but none of the between-group differences was statistically significant. The left DLPC group was marginally different from the BDC group on Verbal IQ ($p = .04$, 95% Confidence Interval = -20.01 to -0.67) and Full Scale IQ ($p = .04$, 95% Confidence Interval = -18.56 to -0.24) (the p levels are not significant after Bonferroni alpha correction, but the Confidence Intervals do not include zero). None of the differences between the right DLPC group and the BDC group approached significance.

As noted, 25 of the 37 DLPC patients had lesions that extended beyond the DLPC region. Thus, we re-ran all of the analyses using the 12 DLPC patients whose lesions were restricted to the DLPC, to get a picture of whether this might change the overall outcomes. It did not. Specifically, the restricted DLPC group had demographics comparable to those of the BDC group (age, $M = 56.2$, $SD = 10.8$; education, $M = 13.8$, $SD = 2.9$), and IQ scores that were statistically indistinguishable from those of the BDC group: Verbal IQ = 103.2 , $SD = 12.7$ ($p = .91$, 95% Confidence Interval = -9.29 to $+10.45$); Performance IQ = 103.4 , $SD = 15.5$ ($p = .85$, 95% Confidence Interval = -9.04 to $+10.98$); Full Scale IQ = 103.5 , $SD = 12.0$ ($p = .93$, 95% Confidence Interval = -9.08 to $+9.90$); Matrix Reasoning = 10.8 , $SD = 2.6$ ($p = .88$, 95% Confidence Interval = -2.06 to $+1.78$); Vocabulary = 11.0 , $SD = 2.7$ ($p = .40$, 95% Confidence Interval = -1.01 to $+2.50$).

Altogether, these data provide no compelling support for the conclusion that DLPC damage leads specifically to IQ defects, in fluid intelligence or otherwise, relative to damage outside the frontal lobes. Patients with DLPC damage did not differ from brain-damaged comparison patients on overall IQ scores, or on Matrix Reasoning or Vocabulary. We had sizeable numbers of participants in each group (37 each), and the groups were carefully matched on age,

²“Points” for the Matrix Reasoning and Vocabulary subtests refers to age-corrected scaled-score points; “points” for the IQ scores refers to IQ points.

education, gender distribution, handedness distribution, and time since lesion onset, so the group contrast is adequately powered and well controlled.³ Moreover, within the DLPC group, a comparison of the fluid intelligence measure of Matrix Reasoning ($M = 10.4$) and the crystallized intelligence measure of Vocabulary ($M = 10.6$) also fails to support the notion that DLPC damage produces disproportionate impairment of fluid intelligence. Finally, when the analyses were re-run on the patients whose lesions are confined to the DLPC region, none of the outcomes changed, again consistent with the conclusion that DLPC damage does not impair Matrix Reasoning (or other measured aspects of IQ) differentially from brain damage outside the DLPC region.

Study 2

Study 2 tested the hypothesis that fluid intelligence would be impaired by ventromedial prefrontal cortex (VMPC) lesions.

Participants

The participants for Study 2 included a target group—patients with lesions that included the ventromedial prefrontal cortex (VMPC group)—and a brain-damaged comparison group (BDC group)—patients with lesions outside the frontal lobes who were matched to the target group on age, education, and gender distribution (the BDC group for Study 2 was comprised of entirely different individuals from the BDC group for Study 1). The target group included a total of 25 patients with either left ($n = 5$), right ($n = 9$), or bilateral ($n = 11$) VMPC damage. For the purposes of this study, the VMPC was defined as the mesial orbital cortex and the inferior mesial prefrontal cortex, encompassing Brodmann areas 11 (mesial sector), 12, 25, and the ventral parts of areas 10 (mesial sector) and 32. Any damage to this sector qualified the patient for entry into the target VMPC group. None of the target patients had damage to the DLPC sector (defined in Study 1 above). The BDC patients had lesions that were outside of all frontal regions.

Data for the VMPC and BDC groups are provided in Table 3. The groups were well matched, and did not differ on age ($p = .43$), education ($p = .45$), or gender distribution (equal). Handedness distribution was also comparable in the two groups (Pearson $\chi^2 p = .637$), and the groups did not differ on time since lesion onset (chronicity) ($p = .17$).

Results and Discussion

The various WAIS-III measures are provided in Table 4 (Matrix Reasoning and Vocabulary are age-corrected scaled scores). The results failed to support the hypothesis, as there was no indication that the VMPC group was impaired on the fluid intelligence measure of Matrix Reasoning. Their overall group mean ($M = 10.7$, $SD = 2.5$) was very similar to that of the BDC group ($M = 10.4$, $SD = 3.0$), and not statistically different ($p = .68$; 95% Confidence Interval = -1.24 to $+1.88$).

The other WAIS-III variables were also contrasted between the VMPC and BDC groups. On the Vocabulary subtest, the VMPC group outperformed the BDC group slightly (0.9 age-corrected scaled score points), but this difference was not statistically significant ($p = .39$; 95% Confidence Interval = -1.10 to $+2.78$). The VMPC group had slightly higher scores for all three IQ indices, on the order of some three to five IQ points, but none of the between-group

³It should be understood that for any individual participant, the “normality” of an IQ score or an IQ subtest score depends on a contrast between the obtained score and the expected score based on the participant’s estimated premorbid level (typically derived primarily from educational and occupational background). Within each of the DLPC and BDC groups, there could be individuals who have IQ and/or IQ subtest impairments. But the fact that the two groups are well matched on education and occupation makes highly unlikely the possibility that there is any systematic group difference regarding patients with IQ impairments.

differences was statistically significant: VIQ, $p = .41$, 95% Confidence Interval = -5.11 to $+12.42$; PIQ, $p = .45$, 95% Confidence Interval = -5.82 to $+12.87$; FSIQ, $p = .27$, 95% Confidence Interval = -3.83 to $+13.54$.

For the sake of completeness, we broke down the VMPC group as a function of whether the lesion was unilateral left, unilateral right, or bilateral, and demographic (Table 3) and IQ (Table 4) data are presented for these three subgroups. We did not have a priori hypotheses regarding these subgroups, and some of the *Ns* (especially in the unilateral left group) are small, so statistical analyses would not be particularly meaningful and they are not reported. Based on the means, it can be seen from the data in Table 4 that the left–right contrast followed the general pattern that might be expected, with the left VMPC group showing an advantage on Matrix Reasoning (by 3.0 age-corrected scaled score points) and the right VMPC group showing an advantage on Vocabulary (by 1.1 age-corrected scaled score points). In the same vein, the left VMPC group had Matrix Reasoning > Vocabulary (by 2.3 age-corrected scaled score points), and the right VMPC group had Vocabulary > Matrix Reasoning (by 1.8 age-corrected scaled score points).

In summary, the data from Study 2 do not support the conclusion that VMPC damage leads to defects in fluid intelligence, relative to non-frontal damage. Nor was there any indication that VMPC damage produced defects in crystallized intelligence (as indexed by Vocabulary) or in IQ scores more generally. We had sizeable numbers of participants in the VMPC and BDC groups (25 each), and the groups were well matched on education, occupation, gender distribution, handedness distribution, and time since lesion onset, lending confidence to the conclusion that the groups are not different on the various IQ and IQ subtest measures. The specific contrast of fluid intelligence (Matrix Reasoning) and crystallized intelligence (Vocabulary) in the VMPC group also fails to reveal any notable difference, as the means are very similar (10.7 and 11.1, respectively). At a more general level, these findings are consistent with prevailing lore that VMPC damage does not lead to defects in psychometric intelligence, and this appears to hold even when a specific “fluid intelligence” measure is considered.

STUDY 3

Study 3 tested the hypothesis that fluid intelligence would be impaired by lesions that included both the DLPC and VMPC sectors of the frontal lobes.

Participants

The participants for Study 3 included a target group—patients with lesions that included the dorsolateral and ventromedial sectors (DL + VM group)—and a brain-damaged comparison group (BDC group)—patients with lesions outside the frontal lobes who were matched to the target group on age, education, and gender distribution (the brain-damaged comparison participants for this study were different from those used in Studies 1 and 2). The target group included a total of 18 patients with either left ($n = 4$), right ($n = 9$), or bilateral ($n = 5$) DL + VM damage. Anatomically, the DLPC and VMPC were defined as in Studies 1 and 2, and target patients had damage that included both sectors (in three DL + VM patients, the damage extended beyond the frontal lobes). The BDC patients had lesions that were outside of all frontal regions.

Data for the DL + VM and BDC groups are provided in Table 5. The groups were well matched, and did not differ on age ($p = .82$), education ($p = .68$), or gender distribution (equal). Handedness distribution was also comparable in the two groups (Pearson χ^2 $p = .546$), and the groups did not differ on time since lesion onset (chronicity) ($p = .31$).

Results and Discussion

The various WAIS-III measures are provided in Table 6 (Matrix Reasoning and Vocabulary are age-corrected scaled scores). In regard to Matrix Reasoning, the DL + VM group outperformed the BDC group slightly, with a mean ($M = 10.6$, $SD = 3.7$) that was 1.2 age-corrected scaled score points higher than that of the BDC group ($M = 9.4$, $SD = 2.9$); however, the means were not statistically different ($p = .30$; 95% Confidence Interval = -1.09 to $+3.43$). These findings do not support the hypothesis that combined DLPC and VMPC damage would impair fluid intelligence.

The other WAIS-III variables were also contrasted between the DL + VM and BDC groups, and the groups were very similar. In regard to Vocabulary, the groups differed by only 0.6 age-corrected scaled score points (favoring the BDC group), and the difference was not significant ($p = .63$; 95% Confidence Interval = -2.95 to $+1.82$). The BDC group was slightly higher on the three IQ measures, but the differences were small and not significant: VIQ, $p = .52$, 95% Confidence Interval = -14.81 to $+7.59$; PIQ, $p = .77$, 95% Confidence Interval = -14.33 to $+10.66$; FSIQ, $p = .66$, 95% Confidence Interval = -13.48 to $+8.59$.

Again for the sake of completeness, we broke down the DL + VM group as a function of whether the lesions were unilateral left, unilateral right, or bilateral, and the demographic and IQ data for these subgroups are presented in Tables 5 and 6, respectively. For the same reasons noted in Study 2, statistical analyses of these data are not reported. Inspection of the means for the IQ data in Table 6 indicates the same general pattern as in Studies 1 and 2, whereby the unilateral left group tended to perform slightly better than the unilateral right group on Matrix Reasoning, and slightly worse on Vocabulary. The magnitudes of these differences were small (0.4 and 0.2 age-corrected scaled score points, respectively).

As noted, of the 18 DL + VM patients 3 had lesions that extended beyond the prefrontal region. Thus, we re-ran all of the analyses using the 15 DL + VM patients whose lesions were restricted to the DLPC/VMPC, to get a picture of whether this might change the overall outcomes. It did not. Specifically, the restricted DL + VM group had demographics comparable to those of the BDC group (age, $M = 51.2$, $SD = 17.7$; education, $M = 13.6$, $SD = 2.9$), and IQ scores that were statistically indistinguishable from those of the BDC group: Verbal IQ = 96.7 , $SD = 17.3$ ($p = .57$, 95% Confidence Interval = -15.64 to $+8.78$); Performance IQ = 92.9 , $SD = 18.0$ ($p = .56$, 95% Confidence Interval = -16.39 to $+9.04$); Full Scale IQ = 94.9 , $SD = 16.3$ ($p = .59$, 95% Confidence Interval = -14.81 to $+8.56$); Matrix Reasoning = 10.4 , $SD = 3.6$ ($p = .38$, 95% Confidence Interval = -1.29 to $+3.32$); Vocabulary = 10.0 , $SD = 3.4$ ($p = .90$, 95% Confidence Interval = -2.81 to $+2.48$).

Overall, the findings from Study 3 are consistent with those from Studies 1 and 2, in as much as damage to the dorsolateral and ventromedial prefrontal sectors did not produce defects in fluid intelligence, crystallized intelligence, or IQ more generally, relative to nonfrontal damage. Study 3 had reasonable numbers of participants in both groups (18 each) and careful matching on age, education, gender distribution, handedness distribution, and time since lesion onset, permitting confidence in the conclusion that combined dorsolateral and ventromedial prefrontal damage does not impair psychometric intelligence, relative to damage outside the frontal lobes. Moreover, when the analyses were rerun on the DL + VM patients whose lesions were restricted to prefrontal cortices, none of the outcomes changed, again consistent with the conclusion that DL + VM damage does not impair Matrix Reasoning (or other measured aspects of IQ) differentially from damage outside the DL/VM regions.

To provide an overview of the entire set of results across the different frontal groups, we collapsed all of the prefrontal patients (total $N = 80$) and contrasted them with all of the brain-damaged comparison patients (total $N = 80$). The data are provided in Table 7. It is quite obvious

that the prefrontal group does not differ from the brain-damaged comparison group on any of the IQ variables, including both subtests (Matrix Reasoning, Vocabulary) and all three IQ indices. We did not analyze these data statistically, as this contrast was simply for descriptive purposes (the planned statistical contrasts have already been performed), but it is abundantly clear that the various IQ and IQ subtest scores are very similar between groups, falling within a few points for the IQ scores and within a few tenths of a point for the subtests. Nor is there any indication that the fluid intelligence measure (Matrix Reasoning) and crystallized intelligence measure (Vocabulary) differed in the Prefrontal group—in fact, the means were identical (10.5).

Table 7 also includes data for the three prefrontal subgroups (DLPC, VMPC, DL + VM), which were reported previously but are included in Table 7 for the sake of direct comparisons. (Again, these data were not analyzed statistically because the main contrasts have already been performed.) Descriptively, the most obvious message from these data is the comparability of the various groups across all of the IQ and IQ subtest measures. Relative to the BDC group, the VMPC group tended to have slightly higher scores on the various IQ measures, and the DL + VM group tended to have slightly lower scores on most measures. The group differences are small in magnitude, however, and clearly not indicative of notable, systematic effects of prefrontal lesions on IQ performances.

A variable that warrants mention in this context is lesion volume. In the various groups reported in Studies 1–3, the patients whose lesions tend to be the largest are the VMPC cases, mainly because many of those lesions (11/25 in the VMPC group and 5/18 in the DL + VM group) are bilateral (some of the DLPC patients also have fairly sizeable lesions). None of the patients in the BDC groups had bilateral lesions. We did not address lesion volume explicitly as an experimental variable, because it was so clearly irrelevant to the main findings. Specifically, since the VMPC patients, in particular, tend to have larger lesion volumes, this would operate against our null findings (assuming that larger lesions per se would be expected to cause more IQ decrement). For the same reasons, we did not analyze the relationship between lesion volume and Matrix Reasoning scores—again, the largest lesion volumes are in the VMPC patients, but the between-group differences in Studies 1–3 are patently nil.

STUDY 4

In the final study, we investigated the proposal that frontal lobe patients with high premorbid IQ might evidence especially large impairments in fluid intelligence.

Participants

We selected from the 80 frontal patients reported in Studies 1–3 above those patients who met the dual criteria of: (1) high premorbid IQ, and (2) impaired decision making, social conduct, and emotional regulation. To meet the criterion of high premorbid IQ, the patient had to have (a) a WRAT-3 reading standard score greater than 115, or (b) a Barona estimated IQ above 115, and (c) a current WAIS-III Verbal IQ above 115—i.e., either (a) or (b) had to apply, and (c) had to apply. To meet the second criterion, the patient had to have impairments on the Iowa Gambling Task and evidence of “acquired sociopathy” and emotional dysregulation on the Iowa Rating Scales of Personality Change (see Tranel, Damasio, Denburg, & Bechara, 2005, for details of this assessment). Seven patients met these criteria and, not unexpectedly, all had damage in the ventromedial prefrontal sector.

Results and Discussion

The results did not support the prediction, either at a group level or at an individual level (Table 8). As a group, the seven patients had Matrix Reasoning scores that were nearly identical (*M*

= 13.6, $SD = 2.1$) to the Vocabulary scores ($M = 13.9$, $SD = 1.7$). At an individual level, three patients had Matrix Reasoning scores lower than Vocabulary scores, two patients had the reversed pattern, and two patients had equal scores. Only two patients showed differences between Matrix Reasoning and Vocabulary that were more than 2 age-corrected scaled score points, and the patterns were opposite: one favored Vocabulary (patient 2570) and one favored Matrix Reasoning (patient 2855). The group difference between Matrix Reasoning and Vocabulary was not significant in a t -test ($p = .76$; 95% Confidence Interval = -1.90 to $+2.47$).

In terms of the overall IQ scores, the group of seven patients showed stronger verbal abilities on average, with a mean VIQ (126.4) that was nearly 12 IQ points higher than the mean PIQ (114.6). This difference was not statistically significant in a t -test ($p = .28$; 95% Confidence Interval = -12.33 to $+36.05$), although it is probably underpowered due to the small N . Individually, all but one patient accorded with the pattern of $VIQ > PIQ$. This outcome is not particularly surprising, though, and in fact a $VIQ > PIQ$ pattern is not uncommon in healthy individuals with high intellect (e.g., Hsu, Hayman, Koch, & Mandell, 2000; Mitrushina & Satz, 1995; Winner, 2000). So there is no convincing indication in our findings that the IQ patterns in the “High IQ” participants reported in Table 8 are anything outside of the ordinary.

DISCUSSION

We found no support for the hypothesis that fluid intelligence would be disproportionately impaired by frontal lobe damage, relative to brain damage outside the frontal lobes. The negative outcome obtained for the prefrontal cortex generally, and for all subregions we investigated, including dorsolateral, ventromedial, and dorsolateral plus ventromedial. In no case did patients with damage to these prefrontal sectors demonstrate fluid intelligence performances significantly inferior to those of demographically matched comparison patients with damage outside the frontal lobes. Nor was there any case in which prefrontal patients demonstrated significant inferiority of fluid intelligence as contrasted with crystallized intelligence (indexed by Matrix Reasoning and Vocabulary, respectively); again, this was true of all three prefrontal subgroups. We did find group trends consistent with typical laterality effects, in as much as unilateral left prefrontal lesions led to relative weakness on Vocabulary compared to Matrix Reasoning, whereas unilateral right prefrontal lesions led to relative weakness on Matrix Reasoning compared to Vocabulary, although the magnitude of these effects was small. The comparability of prefrontal groups with nonfrontal groups extended to other WAIS-III IQ measures as well, including Verbal IQ, Performance IQ, and Full Scale IQ—we failed to find significant differences between prefrontal and nonfrontal groups on any of these measures. A bird’s eye view of the data is provided in Table 7, where it can be seen that the three prefrontal subgroups are very comparable to one another and to the brain-damaged comparison group on all of the IQ subtest and IQ measures.

In designing our study, we chose to contrast various prefrontal subgroups with demographically matched brain-damaged comparison groups with lesions outside the frontal lobes. The matching was successful, and for all three prefrontal subgroups (DLPC, VMPC, DL + VM) the brain-damaged comparison groups were highly similar on age, education, gender balance, handedness, and time since lesion onset. This design helps to isolate scientifically the factor of prefrontal damage, and the reliable lack of significant between-group differences provides strong evidence that prefrontal damage, per se, does not lead to disproportionate defects in IQ scores or on the Matrix Reasoning and Vocabulary subtests, relative to damage outside the frontal region. This conclusion was further bolstered when we re-ran all of the analyses using prefrontal patients whose lesions were restricted to the dorsolateral and/or ventromedial sectors, and again there were no disproportionate defects on any IQ measures in the prefrontal patients. Overall, the results were consistent and robust, and for all of the main contrasts, every confidence interval for the various IQ and IQ subtest measures included zero. Our design does

not speak to the issue of IQ impairment in individual participants, which requires a pre–post lesion contrast, but the close demographic matching makes it very unlikely that there would be systematic between-group differences in the numbers of patients with IQ impairments.

As far as the ventromedial prefrontal sector is concerned, our findings support and extend the classic lore that such patients frequently and even typically lack impairments in psychometric—or what can be termed “cognitive”—intelligence (e.g., Bar-On, Tranel, Denburg, & Bechara, 2003; Damasio & Anderson, 2003). This notion has prevailed for many years in regard to the WAIS/WAIS-R and frontal lobes generally (Black, 1976; Janowsky, Shimamura, Kritchevsky, & Squire, 1989; Milner, 1963; Stuss & Benson, 1986; Teuber, 1972; Warrington et al., 1986), and it would appear to hold equally strongly for the WAIS-III, even with its increased emphasis on the measurement of fluid intelligence. In fact, in our overall group of 25 VMPC patients there was no indication that fluid intelligence as measured by Matrix Reasoning was any differently affected than crystallized intelligence as measured by Vocabulary. Moreover, in seven VMPC patients who had high premorbid intellect and acquired defects in decision making, social conduct, and emotional regulation, there was no indication of disproportionate defects in fluid intelligence relative to crystallized intelligence. This result fails to support Duncan et al.’s (1995) proposal that deficits in fluid intelligence might be especially pronounced whenever conspicuous frontal defects are accompanied by high premorbid IQ. It should be emphasized that we are not arguing that VMPC patients have normal intelligence, in the broader sense of the term—in fact, if one follows Wechsler’s classic definition of intelligence as “the capacity of the individual to act purposefully, to think rationally, and to deal effectively with his environment” (1944, p. 3), there is no question that VMPC patients surely do have major intellectual impairments. They just do not fail IQ tests.

The failure of our study to find disproportionate defects in fluid intelligence associated with dorsolateral prefrontal damage was more unexpected. There is by now a fairly consistent literature, especially from functional imaging approaches, suggesting a relationship between DLPC and fluid intelligence. Specifically, DLPC activation has been reported in “high *g*” tasks that ostensibly require fluid intelligence, such as the Raven Progressive Matrices task and similar reasoning tasks (Duncan et al., 2000; Esposito, Kirkby, Van Horn, Ellmore, & Berman, 1999; Gray, Chabris, & Braver, 2003; Njemanze, 2005; Prabhakaran, Smith, Desmond, Glover, & Gabrieli, 1997). There is some consistent evidence from lesion work, as well. For example, Waltz and colleagues (Waltz et al., 1999) reported six patients with damage to prefrontal cortex caused by degenerative disease, who had severe damage in DLPC sectors and were selectively impaired on tasks requiring multiple relational premises, including matrix-reasoning-like tasks. (It should be noted that some older work did not point in this direction—for example, it was found in a neuropsychological study that performance on the Raven Colored Matrices was impaired by damage to the right parietal region or to the left perisylvian region: Basso, De Renzi, Faglioni, Scotti, & Spinnler, 1973.) From a behavioral perspective, it has been found that performance on traditional “frontal” tasks (e.g., Wisconsin Card Sorting Test; Tower of London) is correlated with measures of fluid intelligence (Isingrini & Vazou, 1997; Parkin & Java, 1999). In a literature review a few years ago, Kane and Engle (2002) acknowledged that although the evidence for a relationship between fluid intelligence and the DLPC was limited by the small number of studies available and the small sample sizes in each study, there was every reason to predict that further investigations would confirm a prominent role for the DLPC in novel reasoning and psychometric *g*.

In this context, it behooves us to consider alternative explanations for the current findings. An immediate question is in regard to the construct validity of the Matrix Reasoning subtest. As noted earlier, Matrix Reasoning was modeled on and derived from established measures of fluid intelligence, and was added to the WAIS-III to provide enhanced measurement of fluid intelligence (The Psychological Corporation, 1997). The construct validity and neural

correlates of the Matrix Reasoning subtest, however, have not been well studied, and previous work has focused primarily on the issue of how “nonverbal” the task really is (e.g., Dugbartey et al., 1999). Although the Matrix Reasoning subtest would appear to have high face validity as an index of fluid intelligence (and it does, in fact, correlate highly [$r = .80$] with performance on the Raven Progressive Matrices; The Psychological Corporation, 1997), the empirical support for this is surprisingly meager. Lezak and colleagues (Lezak, Howieson, & Loring, 2004, p. 582) cautioned that the utility of the Matrix Reasoning subtest with patient populations has “yet to be established,” and Donders, Tulsky, and Zhu (2001) found that patients with traumatic brain injury, even when severe, performed as well as normal comparison participants on Matrix Reasoning. In other applications, especially in aging research, investigators have tacitly assumed that Matrix Reasoning probes “fluid reasoning” (e.g., Bugg, Zook, DeLosh, Davalos, & Davis, 2006; Wright, Kunz-Ebrecht, Iliffe, Foese, & Steptoe, 2005; Zook, Welsh, & Ewing, 2006), but the accuracy of this has gone largely unquestioned, and is not obviously established by empirical data.

Our findings raise some challenging questions about whether the Matrix Reasoning subtest measures fluid reasoning in the manner intended and purported by the WAIS-III developers (The Psychological Corporation, 1997). If Matrix Reasoning does tap fluid reasoning, and to the extent that fluid intelligence is linked to dorsolateral prefrontal function, it is surprising that lesions to this region do not yield disproportionate deficits on Matrix Reasoning. Perhaps if Matrix Reasoning were combined with other WAIS-III subtests that have been touted as measures of fluid intelligence, such as Digit Span, Arithmetic, and Block Design (e.g., Caruso & Cliff, 1999), a more distinct pattern would emerge. The fact that Matrix Reasoning is untimed, unlike all of the other subtests on the Performance scales, may also contribute to its potential lack of sensitivity to fluid reasoning deficits. The findings from our study are even consistent with the notion that in its current, untimed, form, Matrix Reasoning may actually perform like a “hold” test, more akin to Vocabulary (e.g., Krull, Scott, & Sherer, 1995). Of course, the putative brain–behavior relationship, i.e., that fluid reasoning is associated with dorsolateral prefrontal cortices, could also be incorrect, and further studies are needed to resolve this issue. It is probably not unimportant that the DLPC subsumes a broad expanse of cortex that is functionally heterogeneous, and more fine-grained experimental parcellation may illuminate this issue.

In sum, findings from the current study are consistent with the conclusion that damage to prefrontal brain regions does not lead to disproportionate impairment of fluid intelligence, relative to damage outside the frontal lobes. But an alternative explanation, namely, that the fluid intelligence measure we used in this study—Matrix Reasoning—is not a good measure of fluid intelligence, cannot be ruled out. Our results support and extend previous observations that prefrontal damage does not lead to IQ impairment, as measured by conventional psychometric tests such as the WAIS-III, and they also challenge researchers to find better empirical support for the construct validity of Matrix Reasoning as an index of fluid intelligence, and to establish whether Matrix Reasoning has reliable and discrete neural correlates.

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Figure 1.
Mock example of a Matrix Reasoning problem.

Table 1

Demographic data for Study 1: DLPC target group

Group	N	Age	Education	Gender	Handedness	Chronicity
DLPC	37	57.7 (12.7)	13.9 (2.8)	21M/16F	35R/2L	6.7 (7.5)
Left	23	59.1 (12.7)	13.8 (2.7)	14M/9F	23R/0L	6.0 (6.2)
Right	14	55.2 (12.9)	14.0 (3.1)	7M/7F	12R/2L	7.7 (9.5)
BDC	37	55.0 (10.9)	13.6 (2.4)	21M/16F	34R/3L	4.6 (6.6)

Age and Education are in years; Chronicity is years between lesion onset and collection of WAIS-III data. DLPC = dorsolateral prefrontal cortex; BDC = brain-damaged comparison group.

Table 2

WAIS-III data for Study 1: DLPC target group

Group	Matrix Reasoning	Vocabulary	VIQ	PIQ	FSIQ
DLPC	10.4 (2.9)	10.6 (3.4)	98.0 (17.9)	98.1 (16.0)	97.4 (16.3)
Left	10.7 (3.0)	9.7 (3.6)	92.3 (17.3)	98.7 (15.9)	93.7 (15.9)
Right	9.8 (2.8)	11.6 (3.1)	104.6 (16.8)	97.4 (16.6)	101.6 (16.2)
BDC	11.0 (2.9)	10.3 (2.5)	102.6 (15.2)	102.4 (14.7)	103.1 (14.6)

Matrix Reasoning and Vocabulary are age-corrected scaled scores. VIQ = Verbal IQ; PIQ = Performance IQ; FSIQ = Full Scale IQ; DLPC = dorsolateral prefrontal cortex; BDC = brain-damaged comparison group.

Table 3

Demographic data for Study 2: VMPC target group

Group	N	Age	Education	Gender	Handedness	Chronicity
VMPC	25	53.3 (13.7)	13.6 (2.2)	15M/10F	23R/2L	4.7 (7.5)
Left	5	48.8 (20.5)	13.8 (1.8)	3M/2F	5R/0L	1.9 (1.8)
Right	9	58.0 (14.2)	14.2 (2.0)	5M/4F	8R/1L	2.1 (1.2)
Bilateral	11	51.5 (9.4)	13.1 (2.6)	7M/4F	10R/1L	8.2 (7.8)
BDC	25	50.1 (14.5)	14.2 (2.6)	15M/10F	22R/3L	2.9 (2.4)

Age and Education are in years; Chronicity is years between lesion onset and collection of WAIS-III data. VMPC = ventromedial prefrontal cortex; BDC = brain-damaged comparison group.

Table 4

WAIS-III data for Study 2: VMPC target group

Group	Matrix Reasoning	Vocabulary	VIQ	PIQ	FSIQ
VMPC					
Left	10.7 (2.5)	11.1 (2.5)	105.7 (14.8)	102.6 (19.1)	105.7 (16.9)
Right	12.8 (2.3)	10.5 (1.7)	103.3 (11.4)	104.5 (21.7)	104.3 (15.3)
Bilateral	9.8 (2.1)	11.6 (2.9)	107.4 (15.9)	105.7 (22.7)	110.3 (18.9)
BDC	10.5 (2.5)	10.9 (2.7)	105.1 (16.1)	99.0 (15.6)	102.7 (16.8)
	10.4 (3.0)	10.2 (3.5)	102.0 (14.7)	99.0 (11.5)	100.9 (11.6)

Matrix Reasoning and Vocabulary are age-corrected scaled scores. VIQ = Verbal IQ; PIQ = Performance IQ; FSIQ = Full Scale IQ; VMPC = ventromedial prefrontal cortex; BDC = brain-damaged comparison group.

Table 5
Demographic data for Study 3: DLPC + VMPC target group

Group	N	Age	Education	Gender	Handedness	Chronicity
DL + VM	18	50.1 (16.6)	13.5 (2.9)	11M/7F	17R/1L	4.0 (5.8)
Left	4	49.0 (11.8)	14.0 (4.2)	4M/0F	4R/0L	2.2 (2.1)
Right	9	44.9 (19.2)	14.0 (3.1)	4M/5F	9R/0L	6.0 (7.8)
Bilateral	5	60.2 (11.9)	12.2 (0.4)	3M/2F	4R/1L	1.7 (1.2)
BDC	18	48.8 (15.8)	13.1 (2.7)	11M/7F	16R/2L	6.1 (6.4)

Age and Education are in years; Chronicity is years between lesion onset and collection of WAIS-III data. DLPC and DL = dorsolateral prefrontal cortex; VMPC and VM = ventromedial prefrontal cortex; BDC = brain-damaged comparison group.

Table 6
WAIS-III data for Study 3: DLPC + VMPC target group

Group	Matrix Reasoning	Vocabulary	VIQ	PIQ	FSIQ
DL + VM					
Left	10.6 (3.7)	9.6 (2.9)	96.6 (16.1)	94.8 (19.2)	95.6 (16.1)
Right	11.3 (4.2)	9.3 (3.8)	93.8 (14.8)	98.3 (23.4)	95.5 (17.7)
Bilateral	10.9 (3.0)	9.5 (3.0)	100.0 (17.5)	94.8 (16.3)	97.8 (15.2)
BDC	9.4 (4.9)	10.3 (2.3)	92.6 (16.5)	92.0 (24.5)	91.8 (19.5)
	9.4 (2.9)	10.2 (3.7)	100.2 (16.9)	96.6 (17.7)	98.1 (16.5)

Matrix Reasoning and Vocabulary are age-corrected scaled scores. VIQ = Verbal IQ; PIQ = Performance IQ; FSIQ = Full Scale IQ; DLPC and DL = dorsolateral prefrontal cortex; VMPC and VM = ventromedial prefrontal cortex; BDC = brain-damaged comparison group.

Table 7

WAIS-III data for all participants

Group	N	Matrix Reasoning	Vocabulary	VIQ	PIQ	FSIQ
Prefrontal	80	10.5 (2.9)	10.5 (3.0)	100.1 (16.7)	98.7 (17.7)	99.5 (16.7)
DLPC	37	10.4 (2.9)	10.6 (3.4)	98.0 (17.9)	98.1 (16.0)	97.4 (16.3)
VMPC	25	10.7 (2.5)	11.1 (2.5)	105.7 (14.8)	102.6 (19.1)	105.7 (16.9)
DL + VM	18	10.6 (3.7)	9.6 (2.9)	96.6 (16.1)	94.8 (19.2)	95.6 (16.1)
BDC	80	10.4 (3.0)	10.2 (3.1)	101.8 (15.3)	100.1 (14.6)	101.2 (14.2)

Matrix Reasoning and Vocabulary are age-corrected scaled scores. VIQ = Verbal IQ; PIQ = Performance IQ; FSIQ = Full Scale IQ; DLPC and DL = dorsolateral prefrontal cortex; VMPC and VM = ventromedial prefrontal cortex; BDC = brain-damaged comparison group.

Table 8

High IQ participants

ID	Age	Educ.	Sex	Matrix	Vocab.	VIQ	PIQ	FSIQ
0318	59	14	M	14	16	142	134	143
0770	57	16	F	13	14	119	94	108
2167	41	15	F	14	12	117	110	115
2570	58	16	M	10	14	140	94	119
2590	60	16	M	12	12	118	155	146
2854	81	20	M	16	16	131	102	122
2855	56	12	M	16	13	118	113	117
Mean	58.9	15.6	—	13.6	13.9	126.4	114.6	124.3
SD	11.7	2.4	—	2.1	1.7	11.1	22.5	14.5

Age and Education are in years. Matrix = Matrix Reasoning; Vocab. = Vocabulary; both are age-corrected scaled scores. VIQ = Verbal IQ; PIQ = Performance IQ; FSIQ = Full Scale IQ.