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A Meta-Analytic Review of Verbal Fluency Deficits in Schizophrenia Relative to other
Neurocognitive Deficits.

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ABSTRACT

Introduction. A prominent view in the neuropsychological literature is that schizophrenia is particularly associated with executive dysfunction, yet in a meta-analytic review it was concluded that, relative to their general level of intellectual functioning, schizophrenics are not disproportionately impaired on a measure of this construct, the Wisconsin Card Sorting Test (WCST). However, verbal fluency tests may be more valid measures of executive functioning as they are more sensitive to the presence of focal frontal lobe injuries.

Method. A meta-analysis was conducted on 84 studies comparing the performance of schizophrenics and healthy controls on tests of phonemic and semantic fluency, as well as other cognitive measures presumed to impose only minimal demands upon executive functioning.

Results. Neither phonemic or semantic fluency deficits qualified as differential deficits relative to general intelligence or psychomotor speed. Patients with schizophrenia were significantly more impaired on semantic relative to phonemic fluency.

Conclusions. As for the WCST, deficits on tests of verbal fluency reflect a more generalised intellectual impairment and not particular difficulties with executive control processes. The larger deficit for semantic relative to phonemic fluency suggests that, in addition to general retrieval difficulties, schizophrenia is associated with compromises to the semantic store.

Introduction

Schizophrenia is a serious, often debilitating disorder associated with cognitive, behavioural and emotional disturbances. Since no definitive symptom or biological marker has been identified, diagnosis is based on the overall symptomatic profile exhibited (American Psychiatric Association, 1994), and individuals meeting criteria for the disorder may differ markedly. Such heterogeneity has meant that attempts to identify whether schizophrenia is particularly associated with specific types of cognitive deficits have yielded contradictory results. This problem has been exacerbated by the fact that the disorder is often characterised by generalised intellectual impairment, and thus patients often perform poorly on virtually all measures of cognition (Egan et al., 2001; Heinrichs & Zakzanis, 1998). Indeed, David, Malmberg, Brandt, Allebeck and Lewis (1997) found that there was a linear correlation between low IQ and risk for the later development of psychosis, including schizophrenia.

However, it has been suggested that schizophrenic illness is particularly associated with executive dysfunction. Executive functioning is hypothesised to be responsible, not for basic cognitive processes, but for the set of behavioural competencies that integrate these capacities (Della Sala, Gray, Spinnler, & Trivelli, 1998), and thus permits contextually sensitive, flexible responses. Aspects of executive function include self-directed planning and strategy formation, future-orientated, goal-directed and non-habitual behaviour (Crawford & Henry, in press; Perret, 1974; Phillips, 1997; Stuss & Benson, 1986). Whilst it is important not to treat the terms 'frontal' and 'executive' as interchangeable (see; Reitan & Wolfson, 1994), there is nevertheless a great deal of evidence that frontal structures are particularly implicated in executive functioning (see; Shallice, 1988; Stuss & Benson, 1986). What is particularly striking about many of the clinical features of schizophrenia is their phenomenological similarity to those associated with frontal dysfunction, such as spontaneity, avolition, mental rigidity, stimulus boundedness, poor planning and lack of social judgement (Benson & Miller, 1997; David, 1992; Stuss, Alexander, & Benson, 1997).

However, if these cognitive and behavioural features are to be legitimately regarded as reflecting executive dysfunction, it is necessary to demonstrate that schizophrenics are disproportionately impaired on measures of this construct. Fronto-executive hypotheses have been offered to account for the cognitive and behavioural disturbances seen in a vast array of neurological and psychiatric disorders, in addition to schizophrenia. If we are to avoid engaging in what David (1992) has termed "Frontal Lobology: Psychiatry's new pseudoscience", rigorous standards of proof must be applied when evaluating these hypotheses. Thus, given that generalised deficits are associated with schizophrenia, an important stage in evaluating an executive hypothesis is to test whether any observed deficits on executive tasks qualify as differential deficits (Crawford & Henry, in press; Crawford, Obonsawin, & Bremner, 1993; Laws, 1999; Miller, 1984). A deficit on an executive measure is not by itself sufficient to infer the presence of a differential executive deficit; instead, it must be shown that the executive deficit is in excess of the averaged performance deficit across a range of other cognitive tasks that are not considered to impose heavy executive demands.

Meta-Analyses of Executive Dysfunction in Schizophrenia

A number of meta-analytic reviews have quantified the magnitude of deficits upon measures presumed to tap executive processes (Bokat & Goldberg, in press; Heinrichs & Zakzanis, 1998; Johnson-Selfridge & Zalewski, 2001; Laws, 1999). In a meta-analytic review of Wisconsin Card Sorting Test (WCST) performance in schizophrenia, Laws (1999) cumulated results from 29 studies, and concluded that executive deficits do not differ significantly from general intellectual functioning, as relative to IQ level as measured by the Wechsler Adult Intelligence Scale (WAIS), deficits on the WCST were neither selective nor disproportionate ($d_s = 0.91, 0.53$ and 1.23 for WCST categories completed, WCST perseveration and WAIS FSIQ, respectively). Moreover, WCST CC was substantially correlated with WAIS FSIQ ($r = .73$).

Consistent with this study, Heinrichs and Zakzanis (1998) derived 22 mean effects from 204 studies for neurocognitive test variables including memory, motor deficits, attention, general intelligence, spatial ability, language, and executive functioning. Moderate to large deficits were reported for all 22 constructs, and it was concluded that schizophrenia is associated with generalised cognitive impairment. Just as Laws (1999) found, the weighted mean deficit for WAIS FSIQ was substantially larger than the corresponding value for the WCST (Cohen's $d_s = 1.10$ and 0.88 respectively), and again WCST and WAIS-R FSIQ effects were substantially correlated ($r = .56$). Other executive measures (including phonemic fluency and Stroop interference) were also less impaired than FSIQ. Although in another meta-analytic review Johnson-Selfridge and Zalewski (2001) reported that there were deficits upon all executive measures assessed, since measures considered to impose minimal demands on executive functioning were not included in this study, no assessment of the relative magnitude of these deficits could be made.

However, the results of Laws (1999) and Heinrichs and Zakzanis's (1998) meta-analytic reviews are not consistent with the hypothesis that schizophrenic illness is associated with a differential executive deficit, at least as indexed by the WCST. It is important to note, though, that for all three of the meta-analytic reviews cited (Heinrichs & Zakzanis, 1998; Johnson-Selfridge & Zalewski, 2001; Laws, 1999) there may be substantive differences between the studies that assess each of the constructs of interest, i.e., it is not the same studies that contribute to each of the mean effect sizes being compared in each of these reviews. In Heinrichs and Zakzanis's (1998) study, for instance, whilst 35 studies contributed to the mean effect for FSIQ, six contributed to the mean effect for Stroop interference; there may have been little or no overlap between the studies contributing to each of these statistics, and consequently little or no overlap in terms of the participants sampled.

This raises a potentially important problem, particularly since it has been suggested that there may be important differences between patients with schizophrenia. Thus, it may be that

differential executive deficits are only typical of certain sub-groups of schizophrenics, and indeed, it is now thought that at least three non-mutually exclusive syndromes are necessary to quantify mental state and behaviour (Arndt, Alliger, & Andreasen, 1991; Grube, Bilder, & Goldman, 1998; Peralta, de Leon, & Cuesta, 1992). Liddle (1987) has categorised these as psychomotor poverty (poverty of speech, movement, expressive gesture and flattened affect), disorganisation (incongruity of affect, distractibility, and disorganised speech in which there is derailment, incoherence and poverty of content) and reality distortion (hallucinations and delusions). Thus, whilst psychomotor poverty exemplifies 'negative' symptoms (i.e. behavioural loss), reality distortion is typified by 'positive' symptoms (i.e. behavioural excess and distortion), and disorganisation by a blend of the two. Negative features are believed to be particularly associated with executive dysfunction (Basso, Nasrallah, Olson, & Bornstein, 1998; Berman et al., 1997). In a recent meta-analytic review, for instance, Nieuwenstein, Aleman and de Haan (2001) found that of the three symptom dimensions, negative features demonstrated the most stable and wide-ranging relationship with executive dysfunction as indexed by the WCST and Continuous Performance Test. Thus, when assessing whether a deficit upon one measure is larger than a deficit upon another, it is important that the same participants contribute to both the measures of interest to 'control' for any potential differences between patients, such as the prominence of negative symptoms.

Verbal Fluency Performance in Schizophrenia

In an attempt to resolve whether schizophrenia is associated with a differential executive deficit, verbal fluency performance has been studied extensively. Tests of verbal fluency are amongst the most widely employed measures used to assess cognitive functioning following neurological damage, and involve associative exploration and retrieval of words based on phonemic or semantic criteria (phonemic and semantic fluency, respectively), usually conducted in the setting of a time constraint. Thus, whilst for phonemic fluency participants are asked to generate as many

words as possible beginning with a specified letter (e.g. F), for semantic fluency search is constrained by a specified category (e.g. animals). These measures are considered to impose comparable demands upon executive or supervisory processes because both require efficient organisation of verbal retrieval and recall, as well as self-monitoring aspects of cognition (the participant must keep track of responses already given), effortful self-initiation, and inhibition of responses when appropriate (Crawford & Henry, in press; Ruff, Light, Parker, & Levin, 1997). However, whilst phonemic fluency requires the creation of search strategies based primarily on lexical representations, tests of semantic fluency require searching for semantic extensions of a target superordinate, and thus depend intrinsically upon the integrity of semantic associations within the lexicon (Rohrer, Salmon, Wixted, & Paulsen, 1999). Deficits on tests of semantic fluency may therefore reflect problems with semantic memory, and not executive dysfunction.

In a recent meta-analytic review that included 31 studies and 1791 participants, Henry and Crawford (in press-a) investigated the relative magnitude of cognitive deficits upon tests of phonemic and semantic fluency for patients with focal cortical lesions. The pattern of results suggested that whilst the two types of fluency impose comparable demands upon executive processes, semantic fluency is relatively more dependent upon the integrity of semantic memory. Henry and Crawford (in press-a) found that focal frontal lobe injuries were associated with equivalent phonemic and semantic fluency deficits ($r_s = .52$ and $.54$ respectively). Although as noted earlier, it is important to avoid conflating anatomy and cognition, there is nonetheless a great deal of evidence that frontal structures are particularly implicated in executive functioning (see; Shallice, 1988; Stuss & Benson, 1986). Thus, a pattern of comparable impairment upon tests of phonemic and semantic fluency for patients with schizophrenia may therefore reflect executive dysfunction if, as was found for frontal patients (but not for non-frontal patients), verbal fluency deficits qualify as differential deficits relative to verbal intelligence and psychomotor speed.

However, semantic fluency was more impaired following focal temporal damage ($r = .61$), and this deficit was significantly larger than the corresponding phonemic fluency deficit ($r = .44$). Since there is considerable evidence implicating temporal structures in semantic memory (see; Martin & Chao, 2001) this was presumed to reflect the greater reliance of semantic fluency upon the integrity of semantic memory. Comparison of the relative magnitude of deficits on phonemic and semantic fluency may therefore be used to draw inferences regarding the prominence of executive deficits and semantic store degradation, respectively.

Whilst performance on tests of phonemic and semantic fluency is almost invariably impaired in schizophrenia (Crawford et al., 1993; Kolb & Wishaw, 1983), there has been considerable disagreement as to whether these deficits predominantly reflect a disorganisation of the semantic store (Aloia, Gourovitch, Weinberger, & Goldberg, 1996) or inefficient retrieval of, or access to, semantic representations (Allen, Liddle, & Frith, 1993; Joyce, Collinson, & Crichton, 1996). Whilst this former perspective would predict a larger deficit upon semantic relative to phonemic fluency, the latter would predict comparable deficits on the two measures, since they are presumed to impose comparable demands upon effortful retrieval processes (Henry & Crawford, in press-a). In a recent meta-analytic review, Bokar and Goldberg (in press) found that schizophrenics were more impaired on semantic relative to phonemic fluency (Cohen's $d_s = 1.23$ and 1.01 respectively) in 13 studies that assessed both measures. This suggests that whilst schizophrenia is associated with a deficit in the executive control processes responsible for effortful retrieval (as evidenced by deficits on both types of fluency), the disorder is also associated with compromise to the semantic store. However, the magnitude of the phonemic and semantic fluency deficits relative to other cognitive measures was not assessed, and consequently no assessment was made as to whether verbal fluency deficits could be regarded as differential deficits.

Thus, to date only two meta-analytic studies have quantified mean effects for both phonemic and semantic fluency (Bokar & Goldberg, in press; Johnson-Selfridge & Zalewski, 2001) and

neither of these included measures presumed to impose only minimal demands upon executive control processes. However, it is important to assess the magnitude of phonemic and semantic fluency deficits in the context of performance on other cognitive measures. As noted previously, Laws (1999) concluded that there was no evidence that schizophrenia was associated with disproportionate executive dysfunction as the WCST was less impaired than a measure of FSIQ. However, in a meta-analytic review, Henry and Crawford (in press-a) found that, relative to the WCST, tests of phonemic fluency are substantially more sensitive to the presence of focal frontal lobe lesions. Tests of verbal fluency may therefore be more sensitive to frontal dysfunction in schizophrenia. Moreover, Miyake, Friedman, Emerson, Witzki and Howerter (2000) have identified three dissociable executive processes (shifting, inhibition and updating), and found that the WCST was especially related to the construct of shifting. Thus, even if there is no evidence of a differential deficit in shifting (as indexed by the WCST), other aspects of executive functioning may be particularly disrupted in schizophrenia. It is of note that processes implicated in verbal fluency performance are thought to include updating and inhibition (Cauthen, 1978; Crawford & Henry, in press; Gold & Arbuckle, 1995; Koziol & Stout, 1992).

Therefore, following on from Laws (1999) study, in the present meta-analysis the relative prominence of deficits on tests of phonemic and semantic fluency will be investigated. Moreover, this will be done using a methodology that restricts the studies in each comparison of interest to only those that assess both the measures to be compared; as noted previously, this ensures that comparisons between different cognitive measures are fair as exactly the same participants will have been tested upon each measure. It should also be noted that in each of the previous meta-analytic reviews cited, fixed, as opposed to random effects meta-analytic models were employed for statistical analyses (Bokat & Goldberg, in press; Heinrichs & Zakzanis, 1998; Johnson-Selfridge & Zalewski, 2001; Laws, 1999). As will be discussed, the National Research Council (1992) recommends use of the random effects model, and suggests that the fixed effects model should be

the exception rather than the rule, as it may lead to inappropriately strong conclusions; in the present study, the random effects model will therefore be used. Finally, in the present study, approximately twice as many independent studies contribute to each respective mean for phonemic and semantic fluency relative to previous meta-analytic reviews that have quantified these mean effects, and as noted previously, this is the only meta-analysis to do so whilst also quantifying mean effects for other cognitive measures not considered to impose substantial demands upon executive processes.

Aims

The first aim was to derive effect size estimates for phonemic and semantic fluency for patients with schizophrenia relative to healthy controls. Comparison of the relative magnitude of each will permit an assessment of whether the verbal fluency deficit associated with schizophrenia predominantly reflects executive dysfunction, or problems with semantic memory, such as a specific semantic retrieval deficit (Henry & Crawford, in press-a). Although Bokar and Goldberg (in press) have previously quantified these effect sizes, the present study will permit a rigorous cross-validation of their results, since, as noted, a substantially larger number of studies contribute to the present analyses.

Moreover, a deficit on a test of phonemic or semantic fluency does not by itself provide evidence of executive or semantic memory dysfunction, respectively; it may instead, for instance, reflect a general verbal impairment, or psychomotor slowing. Thus, the second aim was to estimate effect sizes for other cognitive measures in order to provide comparison standards, and thus assess to what extent fluency deficits in schizophrenia qualify as differential deficits.

Premorbid intelligence as estimated by the National Adult Reading Test (NART; Nelson, 1982) or variants upon it, and the reading sub-test of the Wide Range Achievement Test (WRAT; Jastak & Wilkinson, 1984) was included to address the possibility that if a phonemic fluency deficit

is present, it reflects the fact that schizophrenic patients have not been successfully matched to their controls for premorbid ability. Also important was to address the possibility that phonemic and semantic fluency deficits simply reflect a generalised verbal dysfunction (see; Miller, 1984). Thus, the pattern of deficits across fluency versus verbal intelligence as measured by the WAIS (Wechsler, 1955; 1981) Verbal scale (VIQ) will be compared. Deficits upon tests of phonemic and semantic fluency will also be compared with the deficit for FSIQ as measured by the WAIS (Wechsler, 1955; 1981) as this provides an index of patients' general intellectual capacities.

It will also be investigated whether deficits on tests of phonemic and semantic fluency are in excess of deficits on the WAIS Digit Symbol test (Wechsler, 1955; 1981), a widely used measure of psychomotor speed. This will address the possibility that deficits on tests of verbal fluency simply reflect generalised slowing (i.e. a reduction in cognitive speed), rather than executive dysfunction. Performance on tests of phonemic and semantic fluency will also be compared with the Boston Naming Test (BNT; Kaplan, Goodglass, & Weintraub, 1983) as confrontation naming is considered to be very sensitive to deficits in semantic memory (Hart, 1988), yet imposes only minimal demands upon effortful retrieval and cognitive speed. For executive functioning, performance on categories completed and perseverative errors on the WCST (WCST CC and WCST PE respectively; Heaton, 1981; Nelson, 1976) and the interference condition of the Stroop (e.g.; Golden, 1978) will be recorded. A comparison of phonemic and semantic fluency with these other putative measures of executive function will be informative both with respect to their convergent validity, as well as their relative sensitivity to schizophrenia.

Finally, the third aim is to quantify the relationship between patients' age, years of education, duration of illness, overall illness severity, as well level of positive and negative symptoms, with performance on tests of phonemic and semantic fluency. For overall illness severity scores on the Brief Psychiatric Rating Scale (BPRS; Overall and Gorham, 1962) will be recorded, and for positive and negative symptoms the dependent measures will be the Scale for Assessment of

Positive Symptoms (SAPS; Andreasen, 1983b) and the Scale for Assessment of Negative Symptoms (SANS; Andreasen, 1983a) respectively, where total scores are reported. It should be noted that whilst it would also be informative to investigate the relationship between verbal fluency and the disorganisation syndrome, no standardised measure of this construct currently exists that is widely employed.

Method

Sample of Studies

A computer-based search involving the Web of Science, Psych Lit CD-ROM, and Science Direct databases was undertaken, using the following terms as search parameters; 'letter fluency', 'FAS', 'semantic fluency', 'category fluency', 'controlled oral word association', 'COWA(T)', 'word fluency', 'verbal fluency', 'oral fluency', 'phonemic fluency', 'executive test' and 'frontal test'. A manual search of most issues of Cognitive Neuropsychiatry, Neuropsychology, The Clinical Neuropsychologist, Neuropsychologia, the Journal of the International Neuropsychological Society, Neuropsychiatry, Neuropsychology and Behavioural Neurology, Journal of Neuropsychiatry and Clinical Neurosciences, and the Journal of Clinical and Experimental Neuropsychology was also conducted. The search was completed in December 2002.

The inclusion criteria were (1) the patient group had to consist entirely of adults that met accepted criteria for schizophrenia, such as that set out in the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1994), Research Diagnostic Criteria (Spitzer, Endicott, & Robbins, 1978), or the Schedule for Schizophrenia and Affective Disorders (Endicott & Spitzer, 1978). Where the diagnostic criteria differed or was not specified, studies were included if it was stated that the patient group was schizophrenic, and there was sufficient evidence presented that this was the case, such as a history of hospitalisation, or current hospitalisation. Studies in which there were patients with schizophreniform or schizoaffective disorder were also included. In

addition, the study had to include (2) a healthy control group free from neurological or psychiatric disease, and (3) a measure of phonemic or semantic fluency. Effect size estimates for premorbid IQ, VIQ, FSIQ, Digit Symbol, BNT, WCST CC, WCST PE and Stroop interference were derived from studies that also reported verbal fluency results. For inclusion, the study must also have (4) presented precise statistics convertible to effect size r , (5) been published, (6) in English, (7) in a peer-reviewed journal.

Statistical Analysis

Meta-analysis is a rigorous, quantitative alternative to the traditional review process, as it involves statistical integration of results. The basis of this methodology is the effect size, a standardised statistic that quantifies the magnitude of an effect. Two basic types of metric exist that can be used to quantify effect size, known as the r - and the d -families. Although mathematically equivalent, they are associated with different interpretations of what the effect size represents. Whilst exemplars of the r family characterise the degree of correlation between two variables, e.g. the point-biserial correlation between group membership (i.e. presence or absence of schizophrenia), and the variable of interest (i.e. performance on the cognitive measure of interest), d family members exemplify this relationship in terms of the standardised difference between these two variables calibrated in terms of the standard deviation. As a consequence of its greater generality of interpretation, consistency of meaning and more salient practical meaning, r is the more useful effect size estimate (see; Rosenthal & DiMatteo, 2001). Thus, in the present study the effect size r was employed for statistical analyses.

For each construct, effects were pooled to derive an estimate of the mean, with each effect weighted for sample size to correct for sampling error. To do so, the random effects meta-analytic model (Shadish & Haddock, 1994) was selected in preference to the more commonly employed fixed effects model as it yields more generalisable parameter estimates (National Research Council,

1992). This is because, in the fixed effects model, the mean is presumed to reflect a common underlying effect parameter that gives rise to the sample observations. However, in the random effects model the mean represents a hyperparameter, as it allows for substantive differences beyond sampling error that differentiate the effects contributing to each respective mean (Raudenbush, 1994). Statistically, the crucial difference between these methodologies is in the calculation of standard errors and confidence intervals, which for the random effects model are typically larger. Thus, although more technically demanding, it was considered important to use the random effects model in the present work.

To estimate the degree of heterogeneity of the effects contributing to each mean, the homogeneity statistic Q and the random effects variance (σ_{θ}^2) were estimated, as well as the SD of random effects, and the 95% confidence intervals (CI) within which random effects can be expected to fall. Q quantifies within-group heterogeneity (i.e. the degree to which the studies contributing to each respective mean can be regarded as homogenous). If the Q statistic associated with a mean effect is significant, this suggests that there are substantive differences between the studies contributing to that particular mean. In contrast, a non-significant estimate of Q suggests that once sampling error has been removed, no substantive differences between the studies contributing to the respective mean in question remain (i.e. the null hypothesis of homogeneity of effects cannot be rejected).

It was also important to test whether the difference in the magnitude of mean effects between, for instance, phonemic versus semantic fluency, was statistically significant. However, there is no agreed method for statistically comparing mean effects using the random effects meta-analytic model. A particular difficulty is whether the degrees of freedom (df) in such analyses should be based on N (the number of participants) or K (the number of studies). In the present work, a relatively large number of studies were included, and therefore, t -tests were computed using the more conservative K as the df.

As noted earlier, there may be substantive differences between patients with schizophrenia that may moderate the magnitude of deficits across individual studies, and thus, for each statistical comparison of interest, only studies that assessed both variables of interest were included. For example, although in total 89 schizophrenia groups were assessed upon phonemic fluency, and 48 groups were assessed upon semantic fluency, only 30 groups were assessed upon both phonemic and semantic fluency. Therefore, when comparing phonemic and semantic fluency, it was only these 30 groups that were permitted to contribute to the analyses. This ensured that exactly the same participants were being compared upon each of the measures of interest. It should also be noted that because the same participants were compared upon each measure, paired t -tests were employed for all statistical comparisons. Mean effects were also calculated for each of the non-fluency variables identified (premorbid IQ, VIQ, FSIQ, Digit Symbol, BNT, WCST CC, WCST PE and Stroop interference) and compared with the corresponding effects for phonemic and semantic fluency. Again, to control for any potential differences across studies, only studies that assessed both the fluency and non-fluency variable of interest were included in each particular comparison.

To interpret how important a particular effect was in practical terms, Cohen's (1977) guidelines were adopted. These suggest that a correlation of 0.1 should be regarded as representing a small effect, 0.3 as medium, and 0.5 as large. In addition, squares of the effect size multiplied by 100 were also presented as these latter quantities represent the percentage of the variance accounted for (PVAF) by group membership (i.e. the presence of schizophrenia versus being a member of the healthy adult population) on a measure of interest. It should be noted that for inferential statistics comparisons were made using the PVAF by group membership upon each of the measures of interest. This is because the difference between effect sizes is non-linear as r increases, and thus PVAF is the more appropriate index when comparing variables.

Results

Participant Characteristics

Eighty-four studies published between 1981 and 2002 met the inclusion criteria specified, and in total, data from 2947 schizophrenia patients and 2469 controls contributed. Patients and controls were closely matched for age ($M = 35.08$, $SD = 8.11$ versus $M = 33.90$, $SD = 8.35$ respectively), but there was a tendency for patients to be less educated ($M = 11.85$, $SD = 1.69$ versus $M = 13.00$, $SD = 2.03$, years of education, respectively), and a higher proportion to be male (71.94% versus 60.91% male, respectively). The mean duration of schizophrenia was 11.33 ($SD = 6.23$) years.

Effect Sizes for Patients with Schizophrenia Relative to Healthy Controls

In total, eighty nine phonemic fluency, and 48 semantic fluency effect sizes were calculated. Study-level effect sizes for phonemic and semantic fluency for patients with schizophrenia are presented in Appendix A. A positive sign indicates that patients have performed worse than controls, a negative sign the reverse.

[TABLE 1 ABOUT HERE]

Table 1 presents estimates of the mean effects for phonemic and semantic fluency, their variability, and practical importance for studies that include both of these measures. In addition, mean effects are presented for premorbid IQ, VIQ, FSIQ, Digit Symbol, BNT, WCST CC, WCST PE and Stroop interference, and were calculated using only those studies that included the particular non-fluency measures of interest in addition to phonemic or semantic fluency. As noted previously, this methodology ensures that exactly the same participants are contributing to the mean effects for the two variables being compared.

Thus, it can be seen in Table 1 that for each non-fluency measure, for instance premorbid IQ, two mean effects have been calculated; one for studies that also assess phonemic fluency ($r =$

.26; $\underline{K} = 23$), and one for studies that also assess semantic fluency ($\underline{r} = .18$, $\underline{K} = 16$). Each fluency mean effect was also re-calculated for each of these comparisons; these data are presented in the final column of Table 1.

All the mean effects are significantly different from zero, and in terms of practical importance, at least small in magnitude, with the PVAF by group membership ranging from 3.1% to 34.5%. Eight of the 18 mean effects are associated with significant heterogeneity, as indexed by Q. It should be noted that four outliers were identified in the present study (for the comparisons with studies that also assessed phonemic fluency, one outlier was identified for semantic fluency, one for FSIQ, and two for Digit Symbol). Although excluding these outliers reduces the heterogeneity observed for the three respective means in question, their exclusion does not alter the pattern of results for any of the inferential tests on differences and they were therefore retained. It should be noted that whilst in some instances outliers may be attributable to recording errors or system noise, on other occasions they may represent an important part of the data, for instance, a group of schizophrenics may perform particularly well or particularly badly upon the cognitive measure of interest. Since one of the aims of the present study was to investigate potential sources of heterogeneity (such as level of positive or negative symptoms), it was considered important to retain the full dataset, particularly since there were no obvious flaws in the studies that yielded outliers. Thus, the advantage of retaining outliers in the present study is that it means any potentially important data have not been omitted; the disadvantage is, of course, that it increases the heterogeneity of the effects contributing to the mean.

The results of inferential statistics are presented in Table 2. For each comparison the mean difference between the two measures (PVAF) is also presented. It can be seen that for studies that assessed both phonemic and semantic fluency ($\underline{r}_s = .43$ and $.49$, respectively) the deficit for the latter is significantly larger ($\underline{p} = .009$). The effect sizes for phonemic and semantic fluency are both significantly larger than the effect sizes for premorbid IQ ($\underline{r}_s = .43$ versus $.26$; $\underline{r}_s = .52$ versus $.18$,

respectively), but do not differ significantly from the deficits for VIQ or FSIQ, and are significantly smaller than the deficits for Digit Symbol. Thus, for both of these types of fluency there is no evidence of a deficit that is disproportionate to general intellectual functioning or cognitive speed. Indeed, the correlation between phonemic fluency and WAIS FSIQ effect sizes is large according to Cohen's (1977) criteria ($r = .57$, $p = .042$, $K = 13$), and it is of note that this correlation is of a comparable magnitude to the correlation between phonemic and semantic fluency ($r = .58$, $p = .001$, $K = 30$). The correlation between FSIQ and semantic fluency is of a lesser magnitude ($r = .42$, $p = .255$, $K = 9$).

[TABLE 2 ABOUT HERE]

Although relative to the BNT both types of fluency are more impaired ($r_s = .43$ versus $.36$ for phonemic fluency versus the BNT, respectively; $r_s = .47$ versus $.26$ for semantic fluency versus the BNT, respectively), neither of these comparisons attained significance. Relative to measures of executive functioning, phonemic fluency is comparably impaired; indeed, the degree of similarity in these measures' sensitivity to schizophrenia is quite striking. For studies that assessed WCST CC and phonemic fluency, for both measures the mean effect is estimated to be $.47$; for studies that assessed WCST PE and phonemic fluency, for both the mean effect is $.44$. The effect sizes for Stroop interference and phonemic fluency are also virtually identical in magnitude ($r_s = .44$ and $.43$, respectively). Relative to WCST CC, WCST PE and Stroop interference, semantic fluency is more impaired, although only the comparison with the WCST PE is significant.

Moderators of Verbal Fluency Deficits in Schizophrenia

Patients' age, years of education, duration of illness, BPRS scores, as well as level of positive and negative symptoms as indexed by the SAPS and SANS respectively, were correlated with the PVAF by group membership upon tests of phonemic and semantic fluency. The results are presented in Table 3.

[TABLE 3 ABOUT HERE]

It can be seen that patients' age is not significantly related to the magnitude of the deficit for either phonemic or semantic fluency. Whilst duration of illness is significantly correlated with semantic but not phonemic fluency ($r_s = .36$ and $-.08$, respectively), phonemic but not semantic fluency deficits were significantly positively correlated with overall illness severity as indexed by the BPRS ($r_s = .57$ and $.23$, respectively) and significantly negatively correlated with years of education ($r_s = -.28$ and $-.22$, respectively).

Despite the fact that the levels of positive and negative symptoms are themselves very highly correlated ($r = .77$, $p < .001$, $K = 16$), phonemic fluency is significantly related to the level of negative symptoms ($r = .70$, $p = .011$, $K = 12$), but not to the level of positive symptoms experienced ($r = .17$, $p = .626$, $K = 11$). Whilst the semantic fluency deficit was highly correlated with both types of symptoms ($r_s = .72$ and $.54$ respectively), due to a lack of statistical power, neither of these correlations attained significance (for both, $K = 5$, and $p > .05$).

Assessing the Possibility of Publication Bias

A number of validity threats have been identified that may lead to imprecise conclusions in both non-quantitative and meta-analytic reviews. Particularly problematic is the 'file drawer problem', which refers to the fact that significant results are more likely to be published than non-significant results (Easterbrook, Berlin, Gopalan, & Mathews, 1991). Two procedures were employed to assess whether this bias posed a threat to the results of the present study. The first was Rosenthal's (1979) 'Failsafe N ', a statistic that quantifies the number of studies with no relation between variables ($r = 0$) needed to reduce the significance of the mean effect to a defined critical level ($p = .05$).

[TABLE 4 ABOUT HERE]

The results are presented in Table 4. It can be seen that all values of Failsafe \underline{N} substantially exceed the critical value (which is Rosenthal's (1979) criteria, five multiplied by \underline{K} , plus ten), with the exception of the BNT and VIQ for studies that also assess semantic fluency. This reflects the fact that, although of a moderate magnitude, these mean effects were calculated from a relatively small number of effects (six and four, respectively).

The second procedure that was used to evaluate the potential presence of publication bias was visual inspection of a funnel-plot diagram, a graph that plots sample size against the corresponding study-level effect. If statistically non-significant results have been discriminated against, there should be a relative absence of studies with small sample sizes that report weak effects. Whilst mean effects based on a relatively small number of studies can be regarded as less robust to the potential problem of publication bias than those based on larger number of studies, for none of the variables was there any actual evidence of this confound operating in the funnel plots constructed.

Discussion

Quantifying Verbal Fluency Deficits in Schizophrenia

Although the presence of schizophrenia was associated with mean deficits upon both phonemic and semantic fluency that were significantly different from zero and moderate to large in practical importance according to Cohen's (1977) criteria, for studies that assessed both measures, the semantic fluency deficit was significantly larger ($\bar{r}s = .43$ and $.49$, respectively). As discussed earlier, two competing hypotheses have been proposed to explain why schizophrenics perform poorly on measures of verbal fluency, the first of which is that the disorder is associated with a disrupted semantic store (McKay et al., 1996), the second that retrieval difficulties reduce access to the semantic store (Allen et al., 1993; Joyce et al., 1996). The present results concur with the results of Bokar and Goldberg's (in press) meta-analysis, in which it is argued that whilst participants have general retrieval difficulties (as indicated by deficits of a moderate magnitude on

both tests of verbal fluency), the larger deficit for semantic fluency suggests that the integrity of the semantic store is also compromised. As discussed previously, drawing such inferences on the basis of the relative prominence of phonemic and semantic fluency deficits is consistent with Henry and Crawford's (in press-a) meta-analytic review. As noted, Henry and Crawford (in press-a) found that phonemic and semantic fluency were associated with comparable deficits for patients with focal frontal lesions, suggesting that the measures implicate executive control processes such as effortful retrieval to a comparable degree. Since semantic fluency was more sensitive to the presence of focal temporal lesions, this is consistent with the possibility that semantic fluency is relatively more dependent upon the integrity of the semantic system.

It is however important to point out that in interpreting data of these sort, there is the possibility of identity fallacies. Thus, whilst it is suggested that a similar pattern in phonemic and semantic fluency effect sizes for patients with schizophrenia and patients with temporal lobe damage provides evidence that schizophrenic patients are like temporal patients and therefore have greater difficulties with semantic memory than with executive control processes, other interpretations of these data are possible. Relatedly, if we were to find a profile of phonemic and semantic fluency deficits in schizophrenia that paralleled the profile for frontal patients, this would not constitute evidence of either frontal or executive dysfunction, but could only be regarded as consistent with this possibility. Thus it may be that patients with schizophrenia show greater impairment in semantic fluency for reasons that are unrelated to a degradation of the semantic store. For instance, the retrieval of semantic items in semantic fluency tasks may depend on retrieval mechanisms that are different to those required in phonemic retrieval (e.g. if depth of encoding differs, retrieval mechanisms may differ as well). However, as will be discussed, the present authors favour the interpretation that semantic memory is abnormal in schizophrenia, because there is a great deal of other evidence consistent with this perspective.

Semantic Memory Dysfunction in Schizophrenia

A number of researchers have found evidence of semantic memory impairment in schizophrenia in the form of multiple weak or idiosyncratic associations. Multidimensional scaling and clustering techniques, for instance, indicate that the semantic network of patients with schizophrenia lacks organisation and logical associations (Aloia et al., 1996; Chen, Wilkins & Mckenna., 1994). Moreover, Vinogradov, Kirland, Poole, Drexler, Ober and Shenaut (2002) found that for patients with schizophrenia, as the complexity of semantic memory networks increased (i.e. as the semantic networks became increasingly disorganised), fewer items were retrieved from the semantic domain for a test of semantic fluency and a similarity rating task. Thus, it is probable that a breakdown in the organisation of semantic memory at least partially underlies the deficits on tests of semantic fluency observed. However, it has also been found that for patients with schizophrenia, there may be a reduction in the size of the lexicon. Chen, Chen, Chan, Lam and Lieh-Mak (2000) for instance, found that relative to healthy controls, patients with schizophrenia were significantly impaired on a test of semantic fluency, and their estimated lexicon size was significantly smaller. Thus, there is evidence that the semantic store is degraded in schizophrenia, both in terms of being abnormally organised, and also in terms of the overall size of the semantic store being reduced.

In the present study, in addition to there being a significantly larger deficit for semantic relative to phonemic fluency, a deficit of moderate magnitude was found for the BNT, consistent with there being a degradation of the semantic store. However, although the PVAF did not differ significantly for semantic fluency versus the BNT, group membership accounted for over three times more variance for the former (PVAF = 22.09% versus 6.76%, respectively). As noted previously, comparisons based on K as opposed to N can be regarded as conservative. Although only six studies contributed to the comparisons of the BNT versus semantic fluency, and were thus associated with relatively low statistical power when using K as df, these studies included a total of 234 participants. Moreover, the associated Q statistics for the BNT were homogeneous, suggesting

that there is relatively little variation on this measure between patients with schizophrenia. Thus, although there is a great deal of evidence that confrontation naming is very sensitive to the integrity of the semantic system (Hart, 1988), the present results indicate that it is less sensitive to the presence of schizophrenia than semantic fluency. It is suggested that the larger deficit for the latter measure reflects the fact that semantic fluency but not the BNT additionally imposes substantial demands upon effortful retrieval and/or cognitive speed.

Indeed, the present results indicate that patients with schizophrenia experience particular difficulties with tasks that require cognitive speed. Whilst a deficit in this aspect of cognition was to be expected given that psychomotor poverty is often associated with schizophrenia, and manifests itself in a poverty of speech and behaviour (Liddle, 1987), the magnitude of the deficit for the measure of this construct can be considered very large. The mean effect for Digit Symbol was the largest deficit observed, and, like the BNT, was associated with homogeneity statistics that were not significant. This indicates that a deficit of a large magnitude on the Digit Symbol test is a relatively consistent feature of schizophrenic illness, and suggests that the generalised dysfunction associated with schizophrenia may at least partially reflect cognitive slowing.

Evidence for a Differential Executive Deficit?

As discussed previously, the presence of a deficit on a test that is sensitive to executive dysfunction is not sufficient to infer the presence of a differential executive deficit; instead, it must be demonstrated that patients exhibit greater impairment on measures of this construct than they do on measures presumed to impose only minimal executive demands (Crawford et al., 1993). In the present study, this pattern of results was not found, and indeed, FSIQ was substantially (although not significantly) more impaired than both phonemic and semantic fluency. These results are consistent with the conclusions from other meta-analytic studies. Laws (1999) employed meta-analysis to investigate performance on the WCST relative to FSIQ and found that deficits on the

former were neither selective nor disproportionate to those associated with the latter; Heinrich and Zakzanis (1998) also found that none of the mean effects for the executive measures assessed differed substantially from the deficit on FSIQ. Moreover, in the present study, there was a substantial correlation between phonemic fluency and FSIQ ($r = .57$); both Laws (1999) and Heinrich and Zakzanis (1998) also reported substantial correlations between the WCST CC and FSIQ (.73 and .56, respectively).

The present study has therefore found results that parallel the conclusions of previous meta-analytic reviews, but has done so using a more rigorous method of meta-analysis; as noted earlier, for each comparison, only studies that assessed both variables of interest were included. Thus, the results of these other meta-analytic reviews coupled with the results of the present study suggests that the role of executive dysfunction in schizophrenia may have been over-estimated, particularly since in neuropsychological research the WCST and phonemic fluency represent the most widely used measures of this construct, and evidence for the validity of the latter is particularly strong (Henry & Crawford, in press-a). It is also of interest that compared with other non-fluency measures of executive function, phonemic fluency was associated with virtually identical effect size estimates. Relative to the WCST, phonemic fluency has proven to be far more sensitive to both the presence of focal frontal cortical lesions (Henry & Crawford, in press-a) and traumatic brain injury (Henry & Crawford, in press-b), yet the present results suggest that these measures are equivalent in sensitivity to schizophrenia. As noted earlier, there is evidence for at least three dissociable executive processes (Miyake et al., 2000). Despite the differential reliance of these executive measures upon different component processes, the present study could not differentiate between them in terms of their relative sensitivity to schizophrenia. Thus, the present results provide no evidence that different cognitive control processes are differentially affected by schizophrenia. Whilst there was a tendency for semantic fluency to be more impaired than all the other executive

measures (although only the comparison with WCST PE attained significance), this is consistent with the additional demands this task imposes upon the integrity of the semantic store.

Moderators of Performance on Tests of Verbal Fluency

For eight of the variables assessed in the present study, the homogeneity statistic Q was significant. Since corrections had been implemented for sampling error, the most serious source of artefactual variance (Hunter, Schmidt, & Jackson, 1982), this suggests that substantive differences between studies remain. Moreover, it is also important to note that where significant heterogeneity was observed, this can not simply be attributed to the presence of a few extreme outliers, as outliers were only identified for three of the mean effects. The present results are therefore consistent with the possibility that there are a number of important moderating factors that result in the heterogeneity observed which may include the age of onset (Basso, Nasrallah, Olson, & Bornstein, 1997), the level of thought disorder (Faber & Reichstein, 1981; Kuperberg, McGuire, & David, 1998) and typical versus atypical antipsychotics (Simms, Patel, Burke, Reveley, & Dursun, 1996).

Fortunately, for certain variables, and specifically, patients' age, years of education, and duration of illness, as well as scores on the BPRS, SAPS and SANS, information was presented sufficiently often to enable an assessment of their relationship with performance upon tests of phonemic and semantic fluency. It was found that whilst patients' age was not related to the magnitude of the deficit for either phonemic or semantic fluency, duration of illness was significantly positively correlated with semantic but not phonemic fluency ($r_s = .36$ and $-.08$, respectively). Since the former correlation is positive, this indicates that the longer the duration of illness, the larger the magnitude of the semantic fluency deficit observed. Scores on the BPRS as well as years of education were significantly positively related to deficits upon phonemic, but not semantic fluency. Thus, semantic fluency deficits will tend to be more pronounced in patients with schizophrenia for whom the condition is chronic, and more severe episodes can be expected to be

associated with particularly impaired phonemic, but not semantic, fluency. The negative correlation between years of education with both phonemic and semantic fluency (although only the former was significant) indicates that the magnitude of verbal fluency deficits may be attenuated in patients who have received more years of education.

However, of particular interest was the finding that negative symptoms as measured by the SANS, but not positive symptoms as measured by the SAPS, was significantly correlated with the level of phonemic fluency impairment. This is consistent with other evidence that suggests that negative symptoms are particularly related to frontal dysfunction (Basso et al., 1998; Berman et al., 1997). However, it is important to note that there remains the caveat that it is important to demonstrate that FSIQ does not demonstrate the same pattern of correlations with positive and negative symptoms, i.e. it may simply be that in general cognitive problems are more highly correlated with negative symptoms than with positive symptoms. Unfortunately, this possibility could not be addressed in the present study as very few studies presented the prerequisite data.

Although a number of studies have investigated whether symptom scores are differentially related to performance on different cognitive measures, a consistent pattern of results has not emerged. Schuepbach, Keshavan, Kmiec and Sweeney (2002) found that a reduction in negative symptoms was associated with improvement on tests of phonemic fluency and attention, but a reduction in positive symptoms was not associated with a change in performance on any cognitive measure in a sample of unmedicated first-episode schizophrenic patients. However, Addington, Addington and Maticka-Tyndale (1991) assessed a group of acutely ill hospitalised schizophrenics ($N = 38$) at baseline and six-months later. It was found that, at both time intervals, negative symptoms were significantly correlated with deficits on WAIS FSIQ, the WCST and a test of semantic fluency that incorporated a switching component, but not with phonemic fluency. Moreover, a resolution in positive but not negative symptoms was associated with improved cognitive functioning. Atre-Vaidya, Taylor, Seidenberg, Reed, Perrine and Glick-Oberwise (1998)

reported that in general cognitive impairment was more pronounced the more negative symptoms patients with bipolar mood disorder experienced, whilst Daban et al. (2002) found that deficits on a test of working memory and the WCST were highly correlated with disorganised symptoms, modestly with positive symptoms, but not at all to negative symptoms.

Thus, it is difficult to draw clear conclusions from the existing literature, particularly given that the studies cited vary in terms of patients' level of chronicity, medication status, as well as whether it is the deficits on cognitive measures, or the change in cognitive performance that is correlated with symptom scores. Thus, although the present study indicates that negative but not positive symptoms are significantly correlated with phonemic fluency performance, it remains unclear whether this reflects a specific relationship between executive dysfunction and negative symptoms, or whether it is simply that, in general, cognitive deficits correlate with negative symptoms more than with positive symptoms. Irrespective of which perspective is correct, although the present meta-analysis indicates that semantic fluency will typically be more disrupted than phonemic fluency, this should not be regarded as a fixed characteristic of schizophrenia, for as noted substantial heterogeneity was associated with performance on many of the cognitive measures. However, if the former perspective is correct, it may be that patients for whom negative symptoms are especially prominent present with a more typically 'frontal' pattern of impairment (i.e. comparable deficits upon the two types of fluency). Relatedly, it may be that the degree of semantic memory disruption varies between schizophrenics; Rossell, Rabe-Hesketh, Shapleske and David (1999), for instance, found that patients with delusions exhibited greater deficits on semantic fluency than patients without.

Summary and Conclusions

Although patients with schizophrenia were impaired on both phonemic and semantic fluency, they were significantly more impaired on the latter measure. This suggests that whilst

schizophrenia is associated with a deficit in the executive control processes responsible for effortful retrieval (as evidenced by deficits upon both types of fluency), the disorder is also associated with compromise to the semantic store. However, although confrontation naming is also very sensitive to the integrity of the semantic store, relative to semantic fluency the BNT was substantially less impaired. Since the deficit for a measure of cognitive speed was the largest of all the deficits observed, it may be the additional speed component in tests of semantic fluency that particularly differentiates this measure from the BNT.

The mean effect sizes for measures of phonemic and semantic fluency were both of a smaller or comparable magnitude to the deficits for a measure of general intelligence and psychomotor speed. Thus, the present results are consistent with Laws's (1999) meta-analysis of WCST studies in suggesting that schizophrenia is not characterised by a differential executive deficit. The SANS but not the SAPS was significantly correlated with the magnitude of the phonemic fluency deficit. However, it remains unclear whether this reflects a specific relationship between executive dysfunction and negative symptoms, or whether it is simply that, in general, cognitive deficits correlate with negative symptoms more than with positive symptoms.

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Table 1

Meta-Analysis Summary of Effect Sizes for Tests of Phonemic Fluency (PF), Semantic Fluency (SF), and other Cognitive Measures

	r	K	N	SE	<u>CIs of mean</u>		Z	$PVAF$	Q	σ^2	SD	<u>CIs of mean effects</u>		
					Lower	Upper						Lower	Upper	
<u>Studies that include PF</u>														
Semantic Fluency	.49	30	1633	.032	.43	.56	15.5	24.1	91.4*	.019	.139	.22	.77	PF M .43 ($K = 30$)
Premorbid IQ	.26	23	1051	.042	.18	.34	6.2	6.6	50.7*	.021	.145	-.03	.54	.43 ($K = 23$)
VIQ	.40	15	607	.039	.33	.48	10.2	16.1	22.1	.008	.091	.23	.58	.48 ($K = 15$)
FSIQ	.57	13	587	.053	.46	.67	10.6	32.0	51.4*	.027	.164	.25	.89	.50 ($K = 13$)
Digit Symbol	.59	18	1077	.019	.55	.63	30.7	34.5	12.3	—	—	—	—	.39 ($K = 18$)
BNT	.36	8	361	.045	.27	.44	8.0	12.7	3.4	—	—	—	—	.43 ($K = 8$)
WCST CC	.47	40	2295	.023	.42	.51	20.0	21.9	84.0*	.011	.103	.27	.67	.47 ($K = 40$)
WCST PE	.44	43	2525	.021	.39	.48	20.3	19.1	78.6*	.008	.092	.26	.62	.44 ($K = 43$)
Stroop interference	.44	20	1132	.046	.35	.53	9.6	19.4	74.8*	.030	.172	.10	.78	.43 ($K = 20$)
<u>Studies that include SF</u>														
SF M														
Phonemic Fluency	.43	30	1633	.024	.39	.48	18.8	18.7	41.2	.005	.069	.30	.57	.49 ($K = 30$)
Premorbid IQ	.18	16	787	.034	.11	.24	3.2	3.1	8.6	—	—	—	—	.52 ($K = 16$)
VIQ	.34	4	130	.063	.22	.46	11.6	10.9	2.7	—	—	—	—	.36 ($K = 4$)
FSIQ	.51	9	475	.052	.41	.61	26.0	26.0	20.4*	.014	.118	.28	.74	.45 ($K = 9$)
Digit Symbol	.59	5	282	.039	.51	.66	34.3	34.3	2.6	—	—	—	—	.62 ($K = 5$)
BNT	.26	6	234	.078	.11	.41	6.7	6.8	9.2	.016	.128	.01	.51	.47 ($K = 6$)
WCST CC	.53	13	653	.034	.46	.59	27.6	27.6	18.2	.005	.070	.39	.66	.55 ($K = 13$)
WCST PE	.46	14	748	.036	.39	.53	20.9	20.9	19.1	.005	.072	.32	.60	.57 ($K = 14$)
Stroop interference	.48	13	610	.044	.40	.57	23.3	23.3	27.3*	.014	.118	.25	.71	.54 ($K = 13$)

* $p < .05$

— indicates that the random effects variance component is estimated to be zero.

Note; For each variable of interest, the mean effect for PF was recalculated using only those studies that assessed both; i.e. 23 studies assess both phonemic fluency and premorbid intelligence; in addition to calculating a mean effect for premorbid IQ based on these 23 studies ($r = .26$), the mean effect for phonemic fluency was also recalculated based only on these 23 studies (i.e. $r = .43$). Thus, when assessing whether or not the effect size for phonemic fluency is in excess of that for premorbid intelligence, exactly the same participants have been tested upon each measure, effectively 'controlling' for any substantive differences between studies. This procedure was also adopted for comparisons of SF with other cognitive measures. This is a more rigorous method of comparing performance on different measures at the level of meta-analysis.

Table 2.

Inferential Statistics Comparing Phonemic and Semantic Fluency Deficits with one another and with Deficits on other Cognitive Measures

Comparison		Δ PVAF	t	df	p
Phonemic Fluency	Semantic Fluency	5.5	-2.78	29	.009
Phonemic Fluency	Premorbid IQ	11.7	3.51	22	.002
Phonemic Fluency	VIQ	7.0	1.74	14	.104
Phonemic Fluency	FSIQ	12.5	-1.60	12	.135
Phonemic Fluency	Digit Symbol	19.6	-7.08	17	< .001
Phonemic Fluency	BNT	5.5	1.03	7	.338
Phonemic Fluency	WCST CC	0.1	0.18	39	.856
Phonemic Fluency	WCST PE	0.1	0.37	42	.714
Phonemic Fluency	Stroop	0.9	-0.44	19	.667
Semantic Fluency	Premorbid IQ	23.8	6.82	15	< .001
Semantic Fluency	VIQ	1.4	0.99	3	.396
Semantic Fluency	FSIQ	5.8	-1.14	8	.287
Semantic Fluency	Digit Symbol	3.6	4.36	4	.012
Semantic Fluency	BNT	15.3	1.64	5	.162
Semantic Fluency	WCST CC	2.2	1.23	12	.229
Semantic Fluency	WCST PE	11.3	2.64	13	.020
Semantic Fluency	Stroop	6.1	0.40	12	.700

Table 3

Moderators of Phonemic and Semantic Fluency Performance in Schizophrenia

	<u>K</u>	<u>r</u>	<u>p</u>
<u>Phonemic Fluency</u>			
Age	79	.01	.920
Education	53	-.28	.039
Duration of illness	58	-.08	.560
BPRS	18	.57	.013
SANS	12	.70	.011
SAPS	11	.17	.626
<u>Semantic Fluency</u>			
Age	47	.27	.071
Education	33	-.22	.226
Duration of illness	33	.36	.041
BPRS	10	.23	.531
SANS	5	.72	.175
SAPS	5	.54	.347

Table 4

Assessing the Possibility of Publication Bias; Failsafe N

	<u>K</u>	Failsafe <u>N</u>	Critical value
<u>Studies that include PF</u>			
Semantic Fluency	30	4522	160
Premorbid IQ	23	535	125
VIQ	15	573	85
FSIQ	13	967	75
Digit Symbol	18	2669	100
BNT	8	119	50
WCST CC	40	7470	210
WCST PE	43	7627	225
Stroop interference	20	1535	110
<u>Studies that include SF</u>			
Phonemic Fluency	30	3262	160
Premorbid IQ	16	131	90
VIQ	4	29	30
FSIQ	9	440	55
Digit Symbol	5	195	35
BNT	6	31	40
WCST CC	13	896	75
WCST PE	14	796	80
Stroop interference	13	717	75

Appendix A

Studies Included in Quantitative Review

Study	Schizophrenia group;			Control	Effect Size		Study manipulation of schizophrenia group;
	N	BPRS	Duration (years)	N	Phonemic	Semantic	
Allen & Frith (1983)	11		20.4	11		.65	Positive symptoms only
Allen & Frith (1983)	12		25.2	11*		.78	Negative symptoms only
Allen et al. (1993)	20		Chronic	10		.95	
Aloia et al. (1996)	28			32		.57	
Arango et al. (1999)	85	36.2	14.8	36	.33	.55	
Artiges et al. (2000)	14		Chronic	14	.64		
Ashton et al. (1999)	39		FE	39	.28		
Basso et al. (1997)	24		10.4	20	.41		Adolescent onset (before age 21)
Basso et al. (1997)	20		8.1	20*	.34		Adult onset (25 or older)
Beatty et al. (1993)	13		12.3	20	.63	.71	
Blanchard & Neale (1994)	28	43.9	Chronic	15	.46		
Brazo et al. (2002)	12		12.8	35	.59	.55	Deficit
Brazo et al. (2002)	9		10.6	35*	.46	.49	Disorganised
Brazo et al. (2002)	14		9.4	35*	.37	.36	Positive
Buchanan et al. (1994)	18		15.3	30	.52		Deficit
Buchanan et al. (1994)	21		12.4	30*	.32		Non-deficit
Chaikelson & Schwartzman (1983)	21		16.2	21	.36		Younger
Chaikelson & Schwartzman (1983)	21		34.1	21	.09		Older
Chan et al. (1999)	40	10.1	6.3	40		.52	
Chen et al. (2000)	21	26.7	17.1	11		.70	
Chen et al. (2000)	23		9.3	26		.70	
Crawford et al. (1993)	48		10.5	48	.55		
Crowe (1996)	21		> or = 5	23	.47		
Danion et al. (2001)	48	31.8	10.6	24		.50	
DeLisi et al. (1997)	41		2.8	26	.36		
Dollfus et al. (2002)	17			17	.61	.45	
Egan et al. (2001)	120			43	.39	.42	

Appendix A (continued)

Studies Included in Quantitative Review

Study	Schizophrenia group;			Control	Effect Size		Study manipulation of schizophrenia group;
	<u>N</u>	BPRS	Duration (years)	<u>N</u>	Phonemic	Semantic	
Elvevag et al. (2001)	13			15	.48	.53	
Elvevag et al. (2002)	24			24		.62	
Evangeli & Broks (2000)	12		15.8	12	.28		
Faber & Reichstein (1981)	14			28		.40	Thought-disordered
Faber & Reichstein (1981)	10			28*		.30	Non-thought-disordered
Feinstein et al. (1998)	23		17.1	11	.32	.43	
Fossati et al. (1999)	14		11.2	20	.44	.60	
Franke et al. (1993)	57			32	.47		
Franke et al. (1993)	30			30	.39		
Friedman et al. (1995)	20	21.1	14.5	24	.40		
Gard et al. (1999)	20	47.7		20	.53		
Glahn et al. (2000)	62	29.7	6.9	62	.53		
Goldberg et al. (1993)	23		10.3	14	.30		
Gourovitch et al. (1996)	23			22	.43	.75	
Gruzelier et al. (1988)	26		2.5	29	.08	-.04	
Gureje et al. (1994)	40		3.1	53	.31	.24	
Hagger et al. (1993)	36	41.9	13.6	26	.45	.65	
Hanes (1998)	14		7.3	24		.39	
Hanes et al. (1996)	20		7.3	26		.55	
Hawkins et al. (1997)	46			26	.44		
Hoff et al. (1992)	32		FE	25	.31		First-episode
Hoff et al. (1992)	26		6.8	25*	.53		Chronic
Hutton et al. (1998)	25		FE	25	.40	.56	
Ismail et al. (2000)	34			75	.47		Low minor physical anomalies
Ismail et al. (2000)	26			75*	.60		High minor physical anomalies
Johnson & Crockett (1982)	16		< or = 5	16	.09		
Jonsson (1998)	10			10	.61		
Joyce et al. (1996)	49		12.0	24	.39	.39	

Appendix A (continued)

Studies Included in Quantitative Review

Study	Schizophrenia group;			Control	Effect Size		Study manipulation of schizophrenia group;
	<u>N</u>	BPRS	Duration (years)	<u>N</u>	Phonemic	Semantic	
Kenny & Meltzer (1991)	15	42.3	10.9	14	.54	.68	
Kenny et al. (1997)	17		1.9	17	.18	.32	
Kiefer et al. (2002)	24	35.4	10.8	24	.36	.52	
Kolb & Wishaw (1983)	30		6.6	30	.56	.03	
Kuperberg et al. (1998)	10	43.7	11.0	10	.44	.50	Non thought-disordered
Kuperberg et al. (1998)	17	48.5	14.0	10*	.61	.65	Thought-disordered
Lafont et al. (1998)	26		2.7	26		.61	
Laurent et al. (2000)	23		9.7	34	.46	.61	
Leonard et al. (1999)	37			33	.39		
Lewis et al. (1992)	25		8.0	25	.58		
Livingston et al. (1998)	38		FE	38	.32		
Mahurin et al. (1998)	23	42.1	8.5	20	.56		Withdrawal retardation
Mahurin et al. (1998)	10	44.7	13.0	20*	.48		Conceptual disorganisation
Mahurin et al. (1998)	20	49.3	13.3	20*	.49		Reality distortion
McGrath et al. (1997)	36	37.6	8.0	20	.23		
Mellers et al. (2000)	22		17.5	24	.32		
Moelter et al. (2001)	38	45.3	6.9	47		.57	
Mohamed et al. (1999)	85		3.7	240	.29		
Morrison-Stewart et al. (1992)	20		9.1	30	.47		Medicated
Morrison-Stewart et al. (1992)	10		10.3	30*	.27		Off medication
Nathaniel-James et al. (1996)	25		15.6	25	.38		
Rains et al. (1995)	10			10	.58		
Riley et al. (2000)	40		FE	22	.58	.66	
Rizzo et al. (1996)	33	48.5	8.5	33	.58		
Robert et al. (1997)	22			22		.64	
Robert et al. (1998)	78			62	.46	.63	
Robertson & Taylor (1985)	61		10.6	41	.23	.29	

Appendix A (continued)

Studies Included in Quantitative Review

Study	Schizophrenia group;			Control	Effect Size		Study manipulation of schizophrenia group;
	<u>N</u>	BPRS	Duration (years)	<u>N</u>	Phonemic	Semantic	
Rossell et al. (1999)	46		11.3	31	.46	.53	Current delusions
Rossell et al. (1999)	28		10.6	31*	.34	.34	No current delusions
Sachdev et al. (1999)	27		7.2	34	.38		Late onset (> or = 50)
Sachdev et al. (1999)	30		36.2	34*	.25		Early onset (< or = 35)
Sagawa et al. (1991)	59	54.9	8.8	36	.62		
Sautter et al. (1997)	50		1.8	20	.09		Familial
Sautter et al. (1997)	46		1.5	20*	.07		Nonfamilial
Schuepbach et al. (2002)	15		FE	24	.44		Low SANS improvers
Schuepbach et al. (2002)	14		FE	24*	-.02		High SANS improvers
Shoqeirat & Mayes (1988)	16			16	.66		
Simms et al. (1996)	17	46.4		24	.63		Typical antipsychotics
Simms et al. (1996)	17	41.2		24*	.39		Atypical antipsychotics
Snitz et al. (1999)	42		12.1	54	.48		
Sumiyoshi et al. (2001)	23		10.0	33		.52	Earlier onset
Sumiyoshi et al. (2001)	20		6.4	33*		.49	Later onset
Thompson & Copolov (1998)	13			13	.09	.29	Non-hallucinators.
Thompson & Copolov (1998)	19			19	.59	.56	Hallucinators.
Verdoux et al. (1995)	17		10.6	17	.44		
Williamson et al. (1989)	10		10.4	20	.08		Off medication
Williamson et al. (1989)	10		8.9	20*	.39		On medication
Yurgelun-Todd et al. (1996)	12			11	.19		
Zakzanis et al. (2000)	24		15.2	35	.77		'Executive' subtype
Zakzanis et al. (2000)	18		14.6	35*	-.11		'Normative' subtype
Zakzanis et al. (2000)	25		19.7	35*	.83		'Global' subtype
Zakzanis et al. (2000)	20		16.2	35*	.82		'Executive motor' subtype

*Group already entered in table

FE = First-episode