## Semantic priming in schizophrenia

**Sir:** In their study on the effects of semantic priming on control subjects and subjects with schizophrenia (Weisbrod *et al*, 1998), the authors suggested that differences in performance relate to the presence or absence of thought disorder. However, the group of subjects defined as thought-disordered differed from other people with schizophrenia in that they had significantly less schooling, and also significantly higher Brief Psychiatric Rating Scale (BPRS) scores ($P$=0.027, not $P$=0.27 as they inadvertently stated). Thus, the differences they detect may well simply be manifestations of greater severity of illness, and may have nothing to do with thought disorder *per se*.

There are also grounds for concern regarding the instrument they have chosen to measure thought disorder. Subjects were defined as thought-disordered or not according to whether they scored higher than 3 on item 4 of the BPRS. This is the cut-off which distinguishes 'moderate' from 'mild' thought disorder, and might require the assessor to decide, on the basis of a single interview, whether the subject's degree of speech incomprehensibility is best described as: "occasional irrelevant statements, infrequent use of neologisms, or moderate loosening of associations", rather than "frequently vague, but the interview is able to progress smoothly; occasional loosening of associations". As Marder (1995) writes of this scale, "Without anchor points the definitions of the items can be vague and subject to different interpretations". Another disadvantage of the BPRS is that the reliance on a single item means that no attempt can be made to characterise different types of thought disorder, which may well be due to different underlying mechanisms rather than just due to aberrant association. Other instruments are far more satisfactory for this purpose. For example the Scale for Assessment of Thought, Language and Communication (Andreasen, 1979) has demonstrable reliability and uses multiple ratings to assess different aspects of thought disorder.

It is important that future studies are methodologically sound or else this one will join the many studies on thought disorder which have failed to be replicated.

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**Weisbrod, M., Maier, S., Harig, S., *et al* (1998)** Lateralised semantic and indirect semantic priming effects in people with schizophrenia. *British Journal of Psychiatry*, **172**, 142–146.

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**Author's reply:** Bhandari & Curtis correctly notice a misprint of a $P$ value. In the text we refer to the correct lower $P$ value and report significant differences between the BPRS scores of the two patient groups. This difference, however, was accounted for by the increased thought disorder ratings in the group with thought disorder. Hence, the general pathology of these patients is greater, but not their pathology as regards symptoms other than thought disorder (non-significant $t$-test). In our view, this state of affairs justifies the attribution of differences in dependent variables to the measure of thought disorder.

Moreover, if both groups had had the same BPRS sum score, one could have argued that – as thought disorder items reached high scores in the group with thought disorder – their remaining symptoms must have been lower. This would warrant the conclusion that the decreased general psychopathology of the patients with thought disorder is the cause of the increased priming effect. This conclusion, however, is ruled out by the higher general BPRS score in the thought-disordered group.

Generally speaking, the argument of non-specific differences is a serious one and has to be addressed in research on schizophrenia (cf. Chapman & Chapman, 1978). However, if the results of an experimental procedure indicate that the worse-off patients perform comparatively better (i.e. show larger priming effects) then the argument is no longer valid. In other words, if the difference between patients with and without thought disorder were such that the patients with thought disorder had lower semantic priming, then the argument would hold. Since the patients with thought disorder have larger priming effects, these effects cannot be accounted for by a more pronounced general deficit.

Bhandari & Curtis furthermore raise concerns about the way thought disorder was assessed in this study. In previous studies on priming effects in schizophrenia we had used other measures, among them the one they propose. However, these measures lump different kinds of symptoms together and are in no way indicators of the specific kind of loose associations we scrutinise in our work. Empirically, these measures came out worse than well-informed clinical judgement when used to form patient subgroups.

In sum, the proof, here, is in the pudding. We have reliably measured semantic and indirect semantic priming effects in people with schizophrenia and compared subgroups with and without thought disorder. The effects and their specific lateralised pattern do not allow their attribution to a general deficit.

Finally, we want to emphasise that the increased semantic and indirect semantic priming effects in thought-disordered people with schizophrenia have been replicated several times in several languages using different methods (see Spitzer, 1997). We currently use event-related potentials in conjunction with semantic priming paradigms to clarify further the nature of formal thought disorder within the framework of cognitive neuroscience, and we aim at the exact characterisation of the involved dysfunctional cognitive processes in space and time.

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**Spitzer, M. (1997)** A cognitive neuroscience view of schizophrenic thought disorder. *Schizophrenia Bulletin*, **23**, 29–50.

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## Are first-rank symptoms encryption errors?

**Sir:** Cognitive psychology has borrowed heavily from computer science, having incorporated notions such as memory 'encoding' and 'retrieval', articulatory 'loops' and visual 'scratch pads', and the 'global workspace' of consciousness, into accounts of human cognition. One problem facing cognitive neuropsychiatry is how cognitive accounts of psychotic symptoms might be implemented at the level of neurobiology. Accounts which treat subjective phenomena (quales) as analogous to 'software' properties, in contrast to neurological 'hardware', may perpetuate a form of dualism. How might quales and aberrant neurophysiology be reconciled?

I suggest one tentative reconciliation: that first-rank symptoms of schizophrenia

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are analogous to disorders of neural encryption. Encryption is a coding system used over the Internet to ensure privacy of communication: ". . .[using] two different decoding keys, one to encipher a message and a different but related one to decipher [it]". "Deciphering requires a separate key, available only to the intended recipient of the message – or, rather, to the recipient's computer" (Gates, 1995, p. 108).

Might first-rank symptoms of schizophrenia be analogues of abnormal encryption code? A few strands of evidence are suggestive. First-rank symptoms of schizophrenia are not well mimicked by static, unifocal structural brain lesions (David, 1994); they are most closely mimicked by distributed disorders of cerebral function (phencyclidine and amphetamine psychoses; metachromatic leukodystrophy). Also, clinically effective antipsychotics induce specific changes in neural firing ('depolarisation block') which are predictive of clinical efficacy (Grace *et al*, 1997). These changes exhibit a temporal delay, as with clinical response (weeks post-initiation of pharmacotherapy).

First-rate symptoms may be the result of distributed disturbances in neural code. How might these be analogous to disordered encryption? If neural systems relied upon the accurate 'reading' of output from one brain region by another, then an encryption error might lead to: (a) a 'message' failing to reach its (neural) 'destination'; (b) being 'read' by another brain region; (c) being 'misinterpreted' as originating in a false location (the 'false sender'; giving rise to abnormal form); (d) being subject to misreading, message degradation (producing abnormal content); or (e) a failure of the 'message' to reach its original target location, and hence, a failure of modulation of one (target) brain region by another.

Any such theory must provide falsifiable hypotheses: that neuroimaging investigations of patients with (a) first rank symptoms of schizophrenia would reveal disordered functional connectivity (compared with other people with schizophrenia); and (b) that there would be a temporal relationship between abnormal quales and abnormal electroencephalogram signals in the 40 Hz or Gamma band range (that associated with subjective phenomena, in consciousness).

If conscious phenomena are codes (implying cognitive–neurophysiological monism), then the medium really is the message.

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## Lifetime risk of suicide in affective disorders

**Sir:** Inskip *et al* (1998) propose that we revise the lifetime risk of suicide in patients with affective disorders from 15% to 6%. We believe that their argument can be taken much further. The reduced estimate followed reanalysis of the results from 27 long-term outcome studies using more sophisticated computerised modelling techniques. The nature of the samples used for this modelling can also be questioned. We have identified the papers included from the references in Harris & Barraclough's (1997) original paper.

Four-fifths of the samples were restricted to in-patients and were therefore biased towards increased severity. None selected first-episode cases, and only 7% were first-admission cohorts. All these factors will inflate long-term mortality estimates as they progressively select cases of greater severity and greater risk of recurrence. Less than a quarter of the samples were series from defined catchment areas, increasing the likelihood of selection bias with difficult-to-treat patients referred to tertiary academic centres entering the meta-analysis. Several samples were recruited before drug treatments such as lithium and antidepressants were available and in a few cases even before the use of electroconvulsive therapy was widespread. Suicide rates in the general populations of the countries studied varied widely and are not stable over time (Diekstra, 1989).

We suggest that Inskip *et al*'s expectation of 6% long-term mortality from suicide should refer to patients from undefined catchment areas with two or more admissions for affective disorder. The long-term suicide rate for a first-admission catchment area cohort (which equates more accurately to the lifetime risk for severe disorder) is likely to be much lower than this. For example Sletten *et al* (1972) (included in Harris & Barraclough's (1997) paper) give numbers of suicides related to numbers of previous admissions for a mixed diagnostic sample; those patients with a single admission accounted for only 18% of suicides. Kessing *et al* (1998), in a large Danish case register study, describe a median time to re-admission of 12.8 years for 17 434 patients discharged after their first admission for unipolar depression. Only 38% had two or more admissions. There are differences between the two samples, but the suicide rate for a first-admission cohort could be several-fold less than for those on subsequent admissions, and therefore much lower than Inskip *et al*'s estimate of 6%.

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**Sletten, I. W., Brown, M. L., Evenson, R. C., *et al* (1972)** Suicide in mental hospital patients. *Diseases of the Nervous System*, **33**, 328–334.

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## Suicide, country of birth and coroners' verdicts

**Sir:** Neeleman *et al* (1997) refer to two of our publications on suicide rates among immigrant groups in England and Wales which were, unavoidably, restricted to suicide verdict deaths. However, in other, more recently published work (Raleigh & Balarajan, 1992; Raleigh, 1996) not cited in their paper, we have noted that suicide verdicts alone significantly underestimate the number of such deaths and, consistent with Department of Health policy on suicide monitoring (Department of Health, 1997), we have included open verdicts. We

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