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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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