for females to have higher rates when the presence of suicidal intent is required. If this were the case, it might help to explain how Isometsä & Lönnqvist found a higher proportion of female suicides with previous suicide attempts.

Unfortunately, the issue of definition in suicidology continues to provoke controversy. The lack of standardisation limits our ability to make comparisons and generalisations based on the research findings of others, whether from the same jurisdiction or not.

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Post-abortion mania

I was interested to read the report by Dr Mahe and his colleagues, describing a woman who suffered from five episodes of puerperal mania and two of post-abortion psychosis, one after a therapeutic abortion and one after a spontaneous abortion. This clinical observation is a valuable contribution to the literature.

The association of acute psychosis with abortion in women susceptible to puerperal psychosis has previously been noted in nine reports, starting with Esquirol in 1819. Some of the terminations were carried out in order to prevent a puerperal psychosis! This literature is summarised in my book *Motherhood & Mental Health*, pages 91–93. There is evidence, especially from Denmark (David, 1985), that abortion is a greater risk factor than a full-term pregnancy.

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Cognitive effects of antipsychotics in schizophrenia and relationship to quality of life

In his overview on cognitive effects of antipsychotics in schizophrenia Sharma (1999) stresses a relationship between cognitive function in schizophrenia and quality of life as an outcome measure. I think that Sharma's use of the concept 'quality of life' has to be clarified to prevent a number of rather common biases. He quotes two studies that are said to support a relationship between cognitive function in schizophrenia and quality of life (Davidson & Keefe, 1995; Green, 1996). The term quality of life is not operationalised in the first study. In the second study, which is in fact an overview of other studies, it is reported by Heinrichs' Quality of Life Scale (Heinrichs *et al*, 1984). Like most other instruments which have been used to detect the effect of atypical neuroleptics on quality of life in schizophrenia (Priebe *et al*, 1999) the Quality of Life Scale (subtitled "An instrument for rating the schizophrenia deficit syndrome") assesses clinical judgements of negative symptoms of schizophrenia rather than subjective appraisals of quality of life made by the patient. As it seems reasonable to assume at least a moderate relationship of negative symptoms and cognitive functions in schizophrenia, it is not surprising that a relationship is found between cognitive functioning and quality of life when the quality of life measures seem to be confounded to a considerable extent by psychiatric symptomatology.

We think that it is necessary to make a distinction between quality of life as an evaluation criterion for illness-related phenomena (negative symptoms), and quality of life as a subjective assessment by the patient as a "subjective evaluation of oneself and one's social and material world" (Orley *et al*, 1998) – that is, subjective quality of life, not as a disease but as a generic concept. Since there are some studies that show that cognitive functioning in schizophrenia may predict social outcome, and since objective social outcome is moderately (although surprisingly weakly) associated with generic subjective quality of life, some association between cognitive functioning and subjective quality of life is conceivable, but has not yet been supported by empirical evidence.

In a validation study of a German short form of the Lancashire Quality of Life Profile (Kaiser *et al*, 1999), equivalent to the English short form of the instrument MANSA (see Priebe *et al*, 1999), we did not find any significant correlation between any of the categories of the Wisconsin Card Sorting Test (WCST; Heaton *et al*, 1993) (number of categories, perserverative errors and responses, etc.) and the mean value of all satisfactions ratings, satisfaction with life as a whole and with satisfaction with mental health in a carefully selected sample of out-patients with DSM–III–R schizophrenia (American Psychiatric Association, 1987; n=36; mean age=47 years; mean illness duration=19 years). Our conclusion so far is that whether or not subjective quality of life is related to cognitive deficits in schizophrenia (in attention or memory, besides deficits in executive functioning, which are seen on a variety of tasks, most notably the WCST) remains unclear and so far is only a hypothesis, although it is widespread as an advertising slogan for atypical antipsychotic medication.

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Author's reply: I thank Dr Kaiser for drawing attention to the relationship between cognitive function and quality of life in schizophrenia. Dr Kaiser does not find any relationship with a single test that he uses to assess cognitive function (the WCST) and a German short form of the Lancashire Quality of Life Profile and thus goes on to suggest that there is no relationship. However, there is evidence that such a relationship does exist (Addington & Addington, 1999). Dr Kaiser feels that Heinrichs' Quality of Life Scale (QLS) is an assessment of clinical judgements of negative symptoms but a detailed look at the scale reveals that the four sub-scales of the QLS do indeed measure interpersonal relationships, instrumental role functioning and common objects and activities, among others. It is true that this is an interviewer rating scale and it would be better to have subjective ratings of quality of life. We have indeed carried out such a study and our (as yet unpublished) results show an association between quality of life, as measured by the Lancashire Quality of Life Profile that Dr Kaiser refers to, and measures of cognitive flexibility, verbal ability and verbal memory. Perhaps if Dr Kaiser had used more than one test to assess cognitive functioning in his patients, he may have found an association, as the WCST measures only one aspect of cognitive function. Meanwhile, his point of using subjective measures of quality of life is well taken.

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Antidepressant response reversed by interferon

A 31-year-old woman with depression lost her previously good response to antidepressants (paroxetine plus trazodone) when treated with interferon alpha. We suggest this occurred as a result of the anti-serotonergic actions of interferons.

The single woman was referred by her general practitioner in September 1996 with several years' history of melancholic depression. At presentation she satisfied full clinical criteria for non-psychotic major depressive disorder. Her medical history included ongoing problems with complex partial and generalised epileptic seizures, seronegative arthritis, irritable bowel syndrome and migraine. A paternal uncle suffered from schizophrenia but there was no other family history of psychiatric illness. She had been a university student but discontinued her studies as a result of her depression.

At presentation her daily medication included dothiepin 150 mg, carbamazepine 500 mg, lamotrigine 50 mg, mebeverine 405 mg, plus sumatriptan 50 mg and dihydrocodeine–paracetamol as required. Her antidepressant was changed from dothiepin to the selective serotonin reuptake inhibitor (SSRI) paroxetine, increased to 50 mg daily, and trazodone 50 mg at night. There followed a dramatic and sustained improvement in her mood and other depressive symptoms.

In June 1997 she was diagnosed as having essential thrombocythaemia with a platelet count of 1400×10^9/l. Although distressed by the diagnosis, no return of her depressive symptoms was seen. Following unsuccessful treatment with dipyridamole, interferon alpha was prescribed by her haematologist. She was given 3 million units, stabilised at three times weekly after her platelet count fell to $700–800 \times 10^9$/l. She experienced the usual flu-like symptoms, and after three months noted the recurrence of depression with a similar profile and severity as that seen prior to treatment with paroxetine.

After six months of disabling depression, refractory to paroxetine plus increased doses of trazodone and cognitive therapy, she was admitted to hospital in May 1998. She was reviewed by her haematologist who discontinued interferon as a result of the depression and started hydroxyurea 1000 mg daily. After two weeks this was changed to anagrelide 500 µg twice daily (prescribed on a named-patient basis) together with atenolol 50 mg daily to reduce associated migraines. Her platelet count was around 400×10^9/l. She had 11 bilateral electroconvulsive therapy (ECT) treatments, administered twice weekly, and had a good response. At the end of treatment she described her mood as being 90% back to normal. She continued on paroxetine 50 mg daily and trazodone 150 mg at night and has remained psychiatrically well to date.

Interferon alpha is associated with a risk of depression, in some cases requiring discontinuation of treatment (McDonald *et al*, 1987). Treatment of interferon-induced depression has yet to be evaluated by controlled trials, but case reports have shown the benefits of antidepressants (Goldman, 1994).

A notable feature of this case is that interferon alpha appeared to reverse a pre-existing antidepressant response to paroxetine. This may be understood in terms of interferon's capacity to impair serotonin synthesis by inducing enzymes that degrade the serotonin precursor tryptophan (Werner-Felmayer *et al*, 1989). Previous research has demonstrated that dietary tryptophan depletion can strikingly reverse the antidepressant effect of SSRIs (Delgado *et al*, 1991).

The clinical improvement seen in the present case following hospitalisation may be related to ECT, interferon discontinuation, or both. Although discontinuation often gradually relieves interferon-induced depression, the rapidity and extent of response in the present case suggests at least some effect of the ECT. In contrast to the SSRIs, the antidepressant response to ECT appears resilient to tryptophan depletion (Cassidy *et al*, 1997). We therefore suggest that ECT is more likely than SSRIs to be effective in interferon-induced major depression.

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