How can we expect the general public to have a rational and informed approach to people with schizophrenia when learned journals accept advertisements that promote a product through negative stereotyping? Perhaps our willingness to allow this to happen is in accord with work in the field which suggests that health professionals may have even more negative attitudes to mental disorder than the general public (Jorm *et al*, 1999). A public campaign to combat stigma is undoubtedly important, but perhaps we should be prepared to examine our own beliefs about serious mental illness as a prelude to changing attitudes in society at large.

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Serum cholesterol and parasuicide

Garland *et al* (2000) reignited the various controversies on the role of cholesterol in psychiatric disorders. The methodology used was similar to those in previous studies (Asberg *et al*, 1976) which did not control for the substances used in parasuicide. This may affect the levels of the chemical or metabolites being researched. Garland *et al* (2000) did not mention the methods used in those parasuicides and whether they would have affected serum cholesterol.

Engelberg (1992) and Block & Edwards (1987) held contrasting views on the relationship between cholesterol and serotonin uptake. The work by Heron *et al* (1980) used to support the hyposerotonergic function caused by low cholesterol appeared flawed. The serotonin site labelled by Heron *et al* (1980) is not the uptake site (Hawton *et al*, 1993), and therefore changes in brain serotonin content cannot be explained on the basis of their data. Furthermore, the serotonin stored within brain cells is not accumulated from blood but synthesised *in situ* from L-tryptophan.

Plasma cholesterol is in a dynamic state, entering the blood complexed with lipoproteins that keep it in solution and leaving the blood as tissues take up cholesterol. High-density lipoprotein (HDL)-cholesterol that transports circulating cholesterol to the liver for clearance plays a crucial role. Excess HDL can result from excess alcohol (Parkes *et al*, 1989). This increases the amount of cholesterol transported peripherally, causing low serum cholesterol. Alcohol, drugs and poisons are usually involved in parasuicides (Asberg *et al*, 1976) and low cholesterol level may therefore be due to ethanol misuse or poisoning. It is unlikely that cholesterol would provide the needed answers to parasuicide. It would only reduce this complex human behaviour to a 'matter to mind' paradigm.

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Transcranial magnetic stimulation: asymmetrical excitability and depression

Maeda *et al* (2000) have succeeded in demonstrating the interhemispheric asymmetry of motor cortical excitability in major depression, using transcranial magnetic stimulation (TMS). This is an important finding that raises questions not only about the pathophysiology of major depression, but also about the state or trait nature of the results.

In discussing possible explanations for this functional asymmetry the authors consider the activity of inhibitory interneurons between cortical output cells, as proposed by Wasserman *et al* (1996), but it is not clear whether this mechanism is thought to act within the hemisphere being stimulated. The role of transcallosal inhibitory mechanisms has been demonstrated in schizophrenia (Davey *et al*, 1997; Boroojerdi *et al*, 1999) and is likely to be relevant to understanding asymmetrical motor thresholds in depression. In support of this view, Menkes *et al* (1999) hypothesised that depression is associated with decreased left hemisphere excitability with respect to the right hemisphere. They successfully showed that inhibitory low-frequency repetitive TMS applied to the right frontal lobe produced a significant antidepressant effect, in contrast to exciting the left frontal lobe by means of fast-frequency repetitive TMS, the antidepressant effects of which have been known for some years.

Furthermore, Maeda *et al* report mean motor thresholds in the depression group of 41.13% for the left hemisphere and 37.63% for the right hemisphere, and in the healthy group of 48.29% for the left hemisphere and 52.7% for the right hemisphere. This gives a mean motor threshold of 39.38% for the depression group and 50.50% for the controls, which suggests important differences in both absolute threshold and laterality between the groups. Any changes to either of these parameters in subjects recovered from depression, and possibly in their first-degree relatives, not only promises new insights into the pathophysiology of depression, but also may provide clues about the most elusive object, a biological marker for depression.

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