grounds that it did not include young and middle-aged widows as we reported. But Maddison himself later described the subjects as 'young and middle-aged' (Maddison, 1968). Perhaps the confusion lies in the fact that it was the deceased *husbands* who were reported as being older (i.e. over 45).

Parkes cites Lundin's Swedish study as added evidence for his position even though that study included persons up to 65 years of age. But Lundin's study is especially vulnerable to the criticisms we have raised. We seriously question, for example, whether social insurance claims are a valid measure of morbidity.

Lundin reported that relatives of 32 sudden-death patients showed a marked increase in morbidity following the death while relatives of 55 controls who suffered a more prolonged death did not. However, Lundin also reported that the prolonged-loss group showed higher-than-normal morbidity both *before* and *after* the loss. This raises the question: Does the fact that their condition did not worsen after the death necessarily indicate 'good outcome?' We think not.

We recognize that a sudden and untimely death can be a catastrophic event for a survivor and may lead to pathogenic grief. But so may a prolonged and stress-filled anticipated loss, even for a young survivor. As we observed, the important point for the clinician is that 'outcome' is not determined simply by the presence of 'forewarning', but rather by the manner in which the 'forewarning' is experienced.

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GUIDED MOURNING FOR MORBID GRIEF

DEAR SIR,

In the article 'Guided Mourning for Morbid Grief' (*Journal*, March 1981, **138**, 185–93) the concept of phobic avoidance concerning the loss of a loved person does not fit comfortably in the process of mourning. An apt comparison would be with a phantom limb: the victim may try helplessly to avoid the non-existent limb and a lay therapist may be tempted to treat with desensitization.

In the present study the time factor and general supportive measures may conceivably have played a crucial rôle in the healing process: therefore, guided mourning may not, in fact, have had any therapeutic effect, while the directive avoidance therapy given to the control group may well have had a positive anti-therapeutic effect. It is indeed likely that neither therapists nor patients in the control group believed in the efficacy of the treatment. Hence the modest significant difference between the two groups in favour of guided mourning.

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SEX DIFFERENCE IN SEASONAL VARIATIONS IN SUICIDE RATE

DEAR SIR,

I think I might be able to throw some light on the finding of Meares *et al* (*Journal*, April 1981, **138**, 321–5) that whereas for men there is a single peak in suicide rate in April/May, for women there are two peaks, one in March/April and one in October/November.

I am at the moment publishing a hypothesis (Skutsch, 1981) about the causes of manic/depression which I believe to be due to a disorder of dopamine metabolism such that dopamine is too low in manics and too high in depressives.

As a corollary I have, in the same paper, suggested that other types of depression might well prove to be due to high levels of dopamine. Thus Euvraard *et al* (1980) have shown (in women) that oestrogen stimulates prolactin release by inhibiting the output of dopamine. (Dopamine, of course, exerts an inhibitory control over prolactin release). Valsik (1965) has demonstrated in a large group of European girls that menarche occurs least often in March and in October (the effect being more marked in March). Menarche is caused by rising oestrogen levels so one can probably deduce that oestrogen levels are unusually low (and therefore that dopamine levels are exceptionally high) in March and in October. So far as I know nobody has demonstrated that there *is* a circannual rhythm of basal oestrogen levels in adult women, but Reinberg *et al* (1978) *have* shown (in a group of young European men) that there is a circannual rhythm of testosterone in which there is a trough in May and a peak in November. As oestrogen in men is derived from peripheral conversion of testosterone one can probably safely assume that oestrogen is also at its lowest

in May. Thus the oestrogen levels in both sexes seem very likely to be at their lowest (so that dopamine will be at its highest) at precisely the times when suicides are commonest in the respective sexes.

Incidentally the idea that depression is caused by high levels of dopamine resulting from low levels of oestrogen would explain the type of depression which follows gonadectomy, contraceptive pill-withdrawal and the menopause. It might even explain puerperal depression because during pregnancy both oestrogen and prolactin are at astronomical levels while at parturition oestrogen falls amost to zero.

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SCHIZOPHRENIA: BEHAVIORAL VARIABILITY AND EVOLUTIONARY PERSISTENCE

DEAR SIR,

Schizophrenia has probably existed as a disease entity for at least 3,000 years and possibly has afflicted mankind since his origins (Lewis, 1966). A number of hypotheses have been advanced to account for the persistence of a schizophrenia gene or genes positing both physiological (e.g. Huxley *et al*, 1964) and behavioral (e.g. Hammer and Zubin, 1968; Jarvik and Chadwick, 1974; Kuttner *et al*, 1967) advantages for nonpsychotic relatives. Among these hypotheses is the proposal that creativity and schizoprenia might be genetically linked, and that creativity exhibited by nonpsychotic carriers of the genes provides an adaptive advantage (e.g. Claridge, 1972; Hammer and Zubin, 1968; Heston, 1966; Karlsson, 1968). However, the process underlying the similarity between creativity and schizophrenic thought has yet to be conclusively demonstrated, and a specific hypothesis of the adaptive advantage of this process is lacking.

Sengel and Lovallo (1980) have recently proposed that extreme variability in memory retrieval processes is the functional deficit which underlies thought disorder in non-paranoid schizophrenia. Further, it is postulated that variability of memory retrieval increases along a continuum from normal behavior to creativity to schizophrenia. The characteristic most frequently seen to demonstrate the similarity between creative and schizophrenic thinking is the tendency to emit unusual associations (Hasenfus and Magaro, 1976). With the Sengel and Lovallo (1980) model, the concept of variable memory retrieval provides a process that accounts for the associative substitutions and intrusions that characterize creative and schizophrenic thinking. Moreover, this model leads to a hypothesis of the adaptive advantage of the schizophrenia-creativity polymorphism.

An analogy can be drawn between genetic variability, adaptation, and fitness, on the one hand, and behavioural variability, adaptation, and fitness on the other hand. The postulated increased variability of retrieval enhances the potential for adaptive responses to changing environmental contingencies. Thus, this greater variability provides a large 'pool' for selection. In other words, the creative individual generates more low probability responses, among which may be the adaptive solution to a new evolutionary problem (e.g. Campbell, 1974). Variable memory retrieval can be seen as producing the novelty selection operates on. At its most extreme, this variability manifests itself as psychosis, and the psychotic carrier of the schizophrenia-creativity polymorphism loses his adaptive advantage because he lacks the structure to integrate feedback and preserve, or retain, a successful behavior. The selection of successful variations will lead to a favourable fitness differential, ensuring preservation of the schizophrenia-creativity polymorphism and the concomitant risk of schizophrenia.

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