In this context, clinical trials of oestrogen therapy in climacteric depression should be judged on the precision of the psychiatric criteria employed. Established diagnostic procedures for depressive illness should be used, for example, those outlined in DSM–III–R (American Psychiatric Association, 1987). Furthermore, the severity of psychiatric morbidity and its response to any form of treatment needs to be assessed by standard psychiatric rating scales. At least in one study, which utilised the Beck Depression Rating Scale, no significant difference was demonstrated between oestrogen (Premarin) and placebo (Campbell, 1976).

On the other hand, Studd *et al* quote one of their own papers (Montgomery *et al*, 1987) on a placebo-controlled study of the beneficial effect of oestradiol implants on depression, in a group of women attending their menopausal clinic. However, the measuring instrument for depression that they used was the SRD-30 (self-rating scale of distress; Kellner & Sheffield, 1973), which is little known to psychopharmacology research workers. Whether or not it represents a valid measure of depressive illness is debatable, since the following items which would be considered indispensable in most psychiatric depression rating scales are missing: suicidal tendencies, retardation, loss of libido, somatic gastric symptoms, weight loss, hypochondria, insight, and diurnal variation (although many believe the last named to be a diagnostic criterion rather than a measure of severity). These are all items to be found in the universally accepted Hamilton Rating Scale for Depression (HRSD), which is that most frequently employed internationally for the measurement of severity of depressive illness.

The relief of menopausal misery has been revolutionised by the introduction of HRT, but it is not a universal panacea. When depression occurs at the menopause, whether idiopathic or iatrogenic, specific antidepressant drug therapy should not be spurned. As Studd *et al* (1990) have so rightly emphasised, there is a considerable need for properly controlled clinical trials with both hormones and antidepressants, or even a combination of both.

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SIR: The importance of temperamental differences in gender-mediated clinical features of depressive illness is supported by women's less active, more ruminative responses (*Journal*, December 1990, **157**, 835–841), linked to dysfunction of the right frontal lobe in which the metabolic rate is higher in females (Friedman & Jankovic, 1990). The role of gender-related cerebral asymmetries was supported by fluvoxamine and fluoxetine-induced apathy and indifference in a predominantly female sample (4 of 5) with panic or depression (Hoehn-Saric *et al*, 1990), and by fluoxetine-induced bradycardia accompanied by faintness or syncope in two women (Ellison *et al*, 1990) which may have been due to serotonergic-mediated inhibition of dopamine lateralised to the right hemisphere (Friedman & Jankovic, 1990). This hypothesis was supported by suppression of the right hemisphere decreasing motor-exploratory aspects of directed attention (Spiers *et al*, 1990), and by unilateral cerebral inactivation producing differential left/right heart-rate responses (Zamrini *et al*, 1990).

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Racism and psychiatry

SIR: Dolan & Evans (*Journal*, December 1990, **157**, 936–937) wilfully misunderstand our comments on racism and psychiatry (Lewis *et al*, *Journal*, 1990, **157**, 410–415). Unlike the correspondents, we as