

clinician. I am sorry I got it wrong when I said it was an original idea.

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Portrayal of women in advertisements

SIR: The paper by Ashton (*Journal*, May 1991, 158 (suppl. 10), 30–35) on psychotropic drug prescribing for women, quotes an American study (Prather & Fidell, 1975) which showed that advertisements for psychoactive drugs show significantly more women. The same conclusion was reached by Penfold & Walker (1984) who also looked at North American journals.

In a retrospective study of our own, we have examined all advertisements for psychotropic drugs appearing in the *British Journal of Psychiatry* over the last 30 years and surprisingly find the situation to be different. We looked at the three categories of antidepressants, anxiolytics, and antipsychotics, and found the portrayal of gender within each group of advertisements to be in line with accepted prevalences of illness (e.g. Kendell & Zealley, 1988), i.e. there was no gender bias in numbers of women shown.

What we did find, however, was a marked difference in the portrayal of women. Whereas depressed or anxious men tended to be shown at work, or worrying about deadlines, women were more often portrayed in the home or with children. In 30 years, only one woman has been shown in a professional role – as a teacher in 1961! Similarly there has never been a female doctor portrayed – despite the fact that about one-third of members of the Royal College of Psychiatrists are women.

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PENFOLD, S. & WALKER, G. A. (1984) *Women and the Psychiatric Paradox*. Milton Keynes: Open University Press.

PRATHER, J. E. & FIDELL, L. S. (1975) Sex differences in the content and style of medical advertisements. *Social Science and Medicine*, 9, 23–26.

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Independent evaluation of new drugs

SIR: Healy (*Journal*, June 1991, 158, 737–742) is right to draw our attention to the weaknesses of the

Hamilton Rating Scale in the assessment of response to antidepressant medication, and to question the marketing policies of drug companies to which clinicians are exposed without the benefit of 'independent trials'. While dilating upon the link between dopamine and serotonin he does not consider the relationship between noradrenaline and serotonin. The implication is that theories of depression involving these two neurotransmitters are mutually exclusive. Close anatomical relationships with reciprocal connections have been demonstrated between the brainstem raphe nuclei and the locus coeruleus suggesting closely related systems (Descarries & Leger, 1978; Fuxe *et al*, 1978).

Sulser (1987) has described the complex relationship between the changes in noradrenergic receptors in response to antidepressant medication and the role of serotonin in these changes. In drug-free animals, lesions of the serotonin system produce an increase in the density of β -adrenoceptors. In the absence of serotonin, downregulation of the β -adrenoceptor in response to antidepressants does not occur. This downregulation is a common feature of many treatments for depression (Leonard, 1988). Acute reductions in serotonin may reverse antidepressant-induced downregulation. The mechanism of interaction between the two may be modulated by 'second' or 'third' messenger activated protein kinases (Sulser, 1987).

Leonard (1988) suggests that the adaptive changes in the serotonin and noradrenergic receptors in response to chronic antidepressant administration have a similar time course to the improvement seen clinically in the depressed patient.

Clearly the story is a complex one and much remains to be clarified. The marketing strategies of the drug companies exploit these areas of doubt. While agreeing with Healy that there is a need for 'independent' studies and critical evaluation of these, this cannot be achieved by ignoring the wealth of evidence that serotonin in conjunction with noradrenaline has an important role in the molecular biology of depression.

DESCARRIES, L. & LEGER, L. (1978) Serotonin nerve terminals in the adult rat. In *Interactions Between Putative Neurotransmitters in the Brain* (eds S. Garattini, J. F. Pujol & R. Samanin), pp. 355–367. New York: Raven Press.

FUXE, K., HOKFELT, T. & AGNALI, L. F. (1978) Mapping out central catecholamine neurones. In *Psychopharmacology: A Generation of Progress* (eds M. A. Liplan, A. DiMascio & K. F. Killam), pp. 67–94. New York: Raven Press.

LEONARD, B. A. (1988) Pharmacological effects of serotonin reuptake inhibitors. *Journal of Clinical Psychiatry*, 49 (suppl. 8), 12–17.

SULSER, F. (1987) Serotonin-norepinephrine receptor interactions in the brain: implications for pharmacology and pathophysiology

of affective disorders. *Journal of Clinical Psychiatry*, 48 (suppl. 3) 12–17.

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Winnicott's contribution

SIR: I read Dr Wardle's comprehensive article "Twentieth-century influences on the development in Britain of services for child and adolescent psychiatry" (*Journal*, July 1991, 159, 53), with interest. It seems extraordinary that he omits Dr Donald Winnicott's enormous contribution to the understanding of children. Winnicott qualified as a consultant paediatrician in 1923 and later became a psychoanalyst and a child psychiatrist. He was appointed psychiatric consultant to the Government Evacuation Scheme in the county of Oxford in 1940 and worked with children evacuated during the war. While Klein was concerned solely with the internal world of the child, Winnicott, along with Bowlby, recognised the significance of the early mother-child relationship on the development of the child's personality. He developed, among other things, the concepts of the facilitating environment, the use of child's play in treatment, and the transitional phenomena. His radio broadcasts and popular writings helped to make these developing ideas accessible to the general public. Dr Wardle's account would be incomplete without the acknowledgement of Winnicott's contribution.

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Incidence rates of schizophrenia

SIR: Recent papers concerning unexplained variations in incidence rates of schizophrenia by Kendell & Adams (*Journal*, June 1991, 158, 758–763) and by season of birth according to genetic risk by O'Callaghan *et al* (*Journal*, June 1991, 158, 764–769) provide an element of support for the theoretical consideration of the role of light as an aetiological factor in the genesis of the disorder. This was discussed in correspondence (Quested, *Journal*, November 1990, 157, 782), and then proposed as a short paper at the Spring 1991 Quarterly meeting of the Royal College of Psychiatrists. O'Callaghan *et al* found that a winter birth excess was confined to those

at low genetic risk for the condition, thus strengthening the evidence for the aetiological role of an environmental factor. Kendell & Adams report the interesting finding of a correlation between fluctuations in the month of birth of a sample of schizophrenics and temperature variations from the mean in the third month of gestation. The most interesting aspect of their results is that the influence inverted according to whether the births occurred in Spring or Autumn. Increased incidence rates were seen in births occurring in a phase of increasing day-length following a fall in temperature six months previously, while higher rates were seen for births occurring during a phase of decreasing day-length following an increase in temperature six months previously. While this is difficult to understand in terms of the viral hypothesis which the study was testing, the finding is highly relevant when considered in terms of biological rhythms under the influence of photoperiod and is actually predicted by the theory referred to above. Animal studies of the effect of photoperiodic fluctuations on neurodevelopment have revealed the positive correlation of post-natal day-length with cerebral mass and density in males (Dark, 1987) and the relevance of the prenatal maternal photoperiod to both somatic and neurodevelopment in males and females (Lee, 1988). One of the fundamental purposes of the transfer of photoperiodic information between mother and offspring in animals is to prepare the foetus for the expected season of birth so that appropriate patterns of development can occur. Variations in the maternal photoperiod have been shown to set up different patterns of growth even though the encountered light:dark ratio at birth may be constant (Horton, 1990).

In the present context of the understanding of schizophrenia as a neurodevelopmental disorder, it is possible that variations in photoperiod could mediate the establishment of inappropriate development patterns. The model is well placed to explain present queries in schizophrenia research such as the increased incidence in winter births, especially in those with no family history, and rates in second-generation Afro-Caribbeans, as well as urban/rural differences.

It is likely that discordance between the signal communicating expected season of birth and actual post-natal photoperiod could interact with asymmetrical cerebral development to cause the differences seen in neuropathological studies of schizophrenia. Kendell & Adam's paper suggests the sensitive period for the transfer of seasonal phase information is the third gestational month, in humans, and the "influence which varies consistently with season and temperature" referred to in the abstract is, in all likelihood, light itself.