

EDITORIAL

Sex and depression¹

Sex differences in rates of depressive disorder have not been convincingly explained, and this reflects a more general failure of research to provide a comprehensive aetiological account of depression. The difference can be used as a probe for evaluating the research base of integrative models of depressive disorder (e.g. Akiskal & McKinney, 1975). It is particularly likely to be illuminating if the causes of the sex difference do not overlap completely the causes of depression itself. While there have been many reviews in the area (Weissman & Klerman, 1977; Kessler & McRae, 1981; Wolk & Weissman, 1995; Bebbington, 1996), this point has not been adequately expressed.

Several lines of investigation are necessary for assessing the relative importance of social, psychological and biological influences: the epidemiological study of macrosocial variables and of age effects; temperament, personality, and attributional and coping styles; the experience of psychosocial adversity; and the possibility of increased susceptibility to some forms of stress in women. Both the tendency to affiliation and the requirement for social support may differ by sex. The particular strains of the roles available to women may increase their risk of depression. Possible genetic explanations of the sex difference are of special relevance because of their implications for biological differences. The latter can also be studied directly: hormonal theories in particular must be evaluated.

EPIDEMIOLOGICAL SUPPORT FOR A SEX DIFFERENCE

Community surveys are the best source for assessing sex differences in depressive disorder. (Bebbington, 1994; Kessler *et al.* 1993; Meltzer *et al.* 1995; Wolk & Weissman, 1995). The sex ratio is maintained in all Western societies, and in most Non-western studies as well. Some authors have suggested that it is declining (Srole & Fischer, 1980), but recent cohort studies do not reveal much change (Wolk & Weissman, 1995).

HIGH RISK GROUPS

If higher rates of depression in women were due, for instance, to a universal biological vulnerability, the sex ratio ought to be unaffected by sociodemographic attributes. This is certainly not true, for example, of the effect of marital status, since sex ratios typically differ in the single, the married and the post-marital. Two situations would be of interest: where more women than men occupy a social category with high rates of depression, and where women in a given category seem to be at a special disadvantage. In the second case, the reason for the disadvantage then has to be explained.

It has been argued that being married brings more stress for women than for men (Weissman & Klerman, 1977; Paykel & Rowan, 1979; Weissman *et al.* 1984). Many studies have found that young married women looking after small children are particularly at risk (Baldwin, 1971; Grad de Alarcon *et al.* 1975; Moss & Plewis, 1977; Richman, 1974, 1977; Brown & Harris, 1978; Bebbington *et al.* 1981 *a*, 1984; Ensel, 1982). However, marital status has different associations with effective disorder in different cultures. Married women are at low risk of disorder in Mediterranean countries (Mavreas *et al.* 1986; Vázquez-Barquero *et al.* 1987), in rural New Zealand (Romans-Clarkson *et al.* 1988) and in British orthodox Jews (Loewenthal *et al.* 1995). These societies all accord a high

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value to the home-making role. This suggests not only that social variables are important in determining the sex ratio for depression, but that the association with relatively simple sociodemographic factors is itself affected by more subtle sociocultural influences.

In most Western societies women are still less likely to be employed than men. Employment generally has beneficial effects on psychological health: it brings interest, income, fulfilment, social contacts and status, and provides structure and a sense of control (Jahoda, 1982; Krause & Geyer-Pestello, 1985). The availability of these benefits is likely to differ both among women, and between men and women. The advantages of employment are weaker in married women (Roberts & O'Keefe, 1981; Roberts *et al.* 1982; Warr & Parry, 1982), more so if they have children (McGee *et al.* 1983; Parry, 1986), most so when the children are of pre-school age (Haw, 1995). Full-time employment is particularly demanding (Cleary & Mechanic, 1983; Elliott & Huppert, 1991). The most likely explanation for these findings is role conflict and overload. Thus, part of the excess of depressive disorders in women may be related both to their reduced involvement in employment and to the particular strains they are exposed to if they do work.

Overall, the results that emerge from the epidemiological study of macrosocial variables are suggestive. They are also inconsistent, probably because they are imperfect markers of variables much more intimately and consistently related to the individual experience of depression, that is, subjects' immediate personal circumstances and their psychological attributes. These may explain both the existence of socially defined high risk groups and why high risk has a different social distribution in different localities and cultures.

AGE, SEX AND DEPRESSION

In assessing the origins of sex differences in depression we must consider the major effect of age on prevalence and incidence. In prepubertal children there is, if anything, a male predominance in depressive symptoms and disorders (Anderson *et al.* 1987; Kashani & Carlson, 1987; Fleming & Offord, 1990), while during adolescence the female:male (F:M) ratio approaches the 2:1 value seen in adults (Rutter *et al.* 1976; Cohen *et al.* 1993). This change in ratio appears to be specific to depression (Rutter, 1991; Angold & Rutter, 1992).

In adulthood, the sex ratio tends to decline with age (Bebbington, 1988). The interesting question is whether this can be related to the timing of the menopause. Jorm's (1987) meta-analysis suggested that the sex ratio was much reduced at the ends of the life span, but was maintained for some time after the menopausal years. However, the recent British National Survey of Psychiatric Morbidity (Melzer *et al.* 1995; Bebbington *et al.* 1998) suggests that after age 55, the sex ratio changes, due mainly to a fall in female rates. Considerable weight should be placed on these findings because of the size and representativeness of the sample.

Thus, it remains possible that the high F:M ratio is restricted to the period of female fertility and declines afterwards due to a reduction in female rates. This might have hormonal origins: certainly Bebbington *et al.* (1998) found it to be independent of changes in marital status, child-care arrangements and employment.

Linking the hormonal changes of puberty with increases in female depressive disorder requires, as a first step, direct hormonal measurement in relation to mood. However, the effect of sex hormone levels is trivial beside the impact of social factors (Brooks-Gunn & Warren, 1989), and relates more to depressed mood than to formal depressive disorder. Moreover, several studies have found that controlling for age eliminates the contribution of pubertal stage to the adolescent increase in rates of depressive disorders in girls (Brooks-Gunn & Warren, 1989; Paikoff *et al.* 1991; Angold & Rutter, 1992). Despite one well conducted exception (Patton *et al.* 1996), the evidence favours the importance of being in a pubertal cohort over the personal experience of puberty: this is easier to explain in social terms.

Thus, the relationship between the sex difference and age suggests important changes around the time of puberty. While there is probably a genuine post-menopausal fall in rates in women, this does not seem closely related to the timing of the actual hormonal changes. The thrust of the evidence

is, therefore, in favour of regarding these as times of social and psychological transition rather than hormonally regulated.

SEX HORMONES AND DEPRESSION

What about more direct evidence linking hormone status to depressive disorder? These have some face validity: oestradiol and progesterone seem to modulate neurotransmitter and neuroendocrine systems, including those involving monoamines, and there are transitions in women's lives characterized by hormonal shifts that are also associated with mood disturbance.

I have reviewed elsewhere the extremely complicated evidence in this area (Bebbington, 1996). The real difficulty in attributing the sex difference to female sex hormones arises from our access to a more plausible neuroendocrine hypothesis involving glucocorticoids. This offers an explanation for a range of other neurohumoral phenomena and a mechanism whereby extrinsic stress may result in the features of depressive disorder (Dinan, 1994; Checkley, 1996). It links over-activation of the hypothalamo-pituitary axis and the associated hypercortisolism with the changes in the central monoaminergic pathways thought to underlie depression and the actions of antidepressants. These changes will probably turn out to be the major hormonal concomitants of depressive disorder. Unfortunately, for our purpose, they cannot explain the sex difference: specifically, cortisol responses are not found to be increased before menstruation, while the function of the HPA axis in general does not differ by sex in the required manner (Allen & Pitts, 1984; Anseau *et al.* 1987; Maes *et al.* 1989a; Hunt *et al.* 1989).

NO ROLE FOR GENETICS

The failure to find a convincing sex-related biological mechanism for depression that would account for the sex difference has its parallel in genetic studies. The model with the most potential in this area is one based on multiple threshold liability. This assumes that the familial liability to a disorder is continuously distributed, comprising both genetic factors and familial-environmental effects. Depression in women can be conceived as a broad form of disorder with a lower threshold than the narrow male form. Under this model the relatives of male probands will be more frequently affected, because in them the loading of familial factors will be greater (Carter, 1969). In practice, the relatives of male depressives are not at higher risk than the relatives of female depressives (Merikangas *et al.* 1985; Kupfer *et al.* 1989), so it seems that the sex ratio must be explained in terms of extra-familial influences. This still allows for genetic effects in the transmission of depression, merely averring that they do not cause the sex difference.

As it is extremely unlikely that biological differences between women would be unaffected by intrafamilial (specifically genetic) factors, the failure of genetic explanations inevitable moves the focus of interest onto the physical and social environment.

PSYCHOLOGICAL EXPLANATIONS

The psychological characteristics associated with depression might serve as an explanation if it could be demonstrated that they are more typical of normal women than men. In fact there has been surprising little research into sex differences in ordinary cognitive style, and when differences are demonstrated, they are not always in the right direction. Moreover, the attitudes and attributions supposed to contribute to the causation of depression may merely be symptoms of it (e.g. Lewinsohn *et al.* 1981; Haaga *et al.* 1991). The grail is to find markers of attributional style that can be detected during periods of well-being (Power, 1990); this underlying style might then be activated in appropriate circumstances. One example is the 'interacting cognitive subsystems' postulated by Teasdale & Barnard (1993; Teasdale, 1995), whereby specific maladaptive attitudes can be reactivated by specific experiences. While this has considerable intuitive appeal and leads to testable hypotheses, no work relevant to sex differences has been carried out. Moreover, as the dysfunctional

systems are held to be the residues of prior depressive states, the theory explains recurrences rather than initial episodes.

A number of authors have tried to link styles of coping with sex differences (Billings & Moos, 1984; Fondicaro & Moos, 1987; Bruder-Mattson & Hovanitz, 1990), but with little success. Nolen-Hoeksema (1987, 1990) has argued that the development of depressive disorder is crucially determined by individuals' responses to an initial lowering of mood. Ruminative styles, more often seen in women (Nolen-Hoeksema *et al.* 1994), are particularly likely to result in an increasing spiral of depression, while the more active styles of coping typical of men appear to abort it.

The two most elaborate psychological formulations of depression are those of Abramson and her colleagues (1978, 1989, 1993) and of Beck *et al.* (1980). Scrutiny of these models suggests they might be rather good at explaining sex differences in depression. However, little specific research has been done, and what there is inconclusive and confusing (Calicchia & Pardine, 1984; Hirschfeld *et al.* 1984; Martin *et al.* 1984; Sowa & Lustman, 1984). The link between cognitions and early social experience and socialization is a fruitful topic for investigation, but again the quantity and quality of research has been inadequate.

ADVERSITY AND VULNERABILITY

Women may be subject to more, or more upsetting, life events than men. Some researchers have found this (Brown & Birley, 1968; Bebbington *et al.* 1981*b*, 1991), others have not (Uhlenhuth & Paykel, 1973*a, b*; Uhlenhuth *et al.* 1974; Thoits, 1982; Perugi *et al.* 1990). Women may be exposed to more chronic problems than men (Radloff, 1975; Pearlin & Johnson, 1977; Pearlin & Lieberman, 1977). Bebbington and his colleagues (1991) found that, although recent adversity was indeed commoner in women, it could not account adequately for their excessive frequency of minor affective disorder.

Early hardships and misfortunes may be more serious candidates for explaining high female rates of depression. The direct effects of physical abuse are quite clear during childhood, and suggest a link with adult depressive disorder, since they lead to depression, low self-esteem, hopelessness, and an external locus of control (Allen & Tarnowski, 1989). Roesler & McKenzie (1994) state that abuse in childhood leads to adult depression, that sexual abuse is worse in this respect than physical abuse and that forced sexual abuse is the worst of the lot. Sexual abuse is commoner in females (Nuttall & Jackson, 1994), and might go a considerable way to explaining the adult sex difference in depressive disorders. Bifulco and her colleagues (1991) found major effects of sexual abuse on rates of depression in their working class London women. Kuyken & Brewin (1994) reported that, of 35 depressed women who had been abused in childhood, 30 had experienced 'flashbacks' of the abuse in the previous week. Abuse induces the sorts of cognitive changes that would be expected to mediate the link with adult depression (Rose *et al.* 1994; Brewin, 1996). This is an under-researched area as an explanation of sex differences in the experience of depression. It may be very important.

It is also possible that women have a special susceptibility to life events (Bebbington *et al.* 1981*b*; Cooke & Hole, 1983), itself requiring explanation. Certain subgroups of women, for instance those caring for young children, may be especially prone to depressive responses to life events (Brown & Harris, 1978; Bebbington *et al.* 1984).

Particular events may have more impact on women, specifically those affecting close emotional ties. Kessler & McLeod (1984) and Turner & Avison (1989) developed the 'cost of caring' hypothesis: women care more for others and are more affected by events affecting others rather than themselves. Turner & Avison (1989) found that women were equally vulnerable to self-focused events, but more so than men to events affecting others.

The importance for women of events affecting intimate relationships brings us to a final vulnerable group, people with low social support (Alloway & Bebbington, 1987). These appear to be at particular risk of developing depression, at least partly because they are more vulnerable to the experience of psychosocial adversity. Women might be at high risk of depression because they have less access to social support, or because they are more vulnerable to its absence. In fact there

is no evidence whatever that women have reduced social support, as is only to be expected in view of their affiliative style. Turner & Marino (1994) claim that the epidemiology of poor social support is very similar to that of depression, with the exception of sex. In other words, the high rates of depression in women cannot be blamed on poor social support. Thus, women when depressed are likely to use more prosocial coping strategies than men (Hobfoll *et al.* 1994), but at the same time low social support is associated with a greater tendency toward (maladaptive) rumination (Nolen-Hoeksema *et al.* 1994).

ROLE INVOLVEMENT

The manner in which prevalence varies by gender, marital status and involvement in child care suggests that depression may be linked to the particular things that people do, and the cultural variation suggests that it is affected by the meaning they attach to what they do. This can be conceptualized in terms of roles, especially those that are influenced by sex. Most human behaviour can be defined as role-related, and roles have a central significance for self-definition and self-evaluation. Role attributes should thus be an important determinant of affective tone, and, further, of affective disorder. This hypothesis affords a conceptual link between social and attitudinal variables, and thus between social and cognitive theories of depression. It may also help our understanding of the differential impact of particular life events. Finally, it may offer a solution to the cultural plasticity of the macrosocial correlates of depression: a geographical difference in the value placed on individual roles may be reflected in differences in the sociodemographic characteristics of high risk.

Until recently, the treatment of roles in social psychiatry has been both superficial and speculative. Men and women differ in terms of the range and status of the roles they carry out, and they certainly differ in the satisfactions they obtain from them. A detailed and critical study of role-based behaviour promises a major step forward in understanding sex differences in the frequency of affective disorder. Important developments in this area have been reported by Power and his colleagues (Lam & Power, 1991; Champion & Power, 1995) and by Nazroo *et al.* (1997). I have set out elsewhere the aspects of roles that are likely to be significant (Bebbington, 1996).

CONCLUSION

Surprisingly, it is possible to draw some reasonably firm conclusions from this literature, although they may be subject to revision from new and better research. The determinants of the sex difference differ from those involved in the aetiology of depressive disorder, particularly with regard to genetic factors and poor social support. However, the failure of genetic factors to account for the difference is a major setback for explanations in terms of sex-related biological factors. There seems no doubt that biological factors are involved in the emergence of depressive disorder, it is just difficult to argue that they are responsible for the sex difference. This pushes us towards a consideration of the physical and social environment. For depressive disorder the evidence about the environment is much better for social factors, and a plausible case can be made for a developmental perspective involving the interaction of social experience and psychological attributes. However, the sex difference is considerable and if its determinants are predominantly social, this emphasizes their contribution to the aetiology of depression as a whole.

These conclusions are in line with my own prejudices. I am wary of attempts to identify a biological 'inferiority' underlying the female proneness to depression. Such attempts have a long history, and were important in the development of psychiatric thought in Victorian times (Oppenheim, 1991). However, this is an area of dispute where there seem to be no neutrals: one would like to think that the issue might be resolved empirically, but this requires results of such clarity that the need for interpretation is minimal. Our sole current certainty is that we have not reached this stage.

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