

Depression in Women

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Depression in treated samples shows an approximately 2 : 1 female predominance. The sex ratio is not due simply to more help-seeking behaviour in women, for it applies equally in studies of community prevalence. Differential acknowledgement and direction of distress may be a partial explanation, supported by male predominance of alcoholism and completed suicide, and by a possible recent increase in depression in young men. Biological mechanisms acting through hormonal effects on the brain are plausible, but hard to test. Epidemiological studies indicate that much of the excess occurs in married women aged 25–45 years with children. This strongly suggests social causation and highlights the vulnerable situation of young mothers. The full explanations for the sex difference are not yet clear, but are likely to combine factors related to expression of distress, biology and social situation.

Depression is the most prevalent disorder in psychiatry. It is also a condition in which illness shades imperceptibly through subclinical distress to a normal mood which is part of universal human experience. Defining thresholds for the disorder and trying to think separately about disorder and normal mood has therefore been important, even if abrupt distinctions are artificial. Community prevalence studies indicate that 5% of the population satisfy the criteria of the Present State Examination (PSE; Wing *et al*, 1974) or DSM–III (American Psychiatric Association, 1980) criteria for defined psychiatric depression in a six month period.

Perhaps the most striking fact about depression is its differential sex incidence, with more women affected than men. This paper examines in detail the differential sex ratio and considers alternative explanations for it. It is not possible to point to one single explanation, or indeed to many firm conclusions, but in my view the evidence is gradually making up a consistent story.

Sex ratios

Table 1 shows sex ratios in treated cases of depression in some representative studies in Western cultures between 1942 and 1973, reviewed by Weissman & Klerman (1977). There are many other studies, and more recent ones, but they also show a female predominance. The average female : male ratio in these studies was around 2.1 : 1 and all showed female predominance. In the literature as a whole there are some exceptions, but not many.

A number of broad explanations for this difference have been advanced. The first suggests that perhaps it is entirely an artefact: women may simply seek help more for the depression which both sexes have equally. A second obvious set of explanations has to

Table 1
Sex ratios in some studies of treated depression (from Weissman & Kerman, 1977)

Study	female : male ratio
<i>USA</i>	
Cooper <i>et al</i> , 1942	2.0
Wechsler, 1961	2.5
Gardner <i>et al</i> , 1963	2.1
Rosen <i>et al</i> , 1964	1.5
Duvall <i>et al</i> , 1966	2.3
Tarnower & Humphries, 1969	2.4
Lehmann, 1971	1.7
Paykel <i>et al</i> , 1970	3.0
Pederson <i>et al</i> , 1972	1.5
Cannon & Redick, 1973	2.1
<i>Europe</i>	
Kielholz, 1959	1.5
Essen-Moller & Hagnell, 1961	1.8
Juel-Nielsen <i>et al</i> , 1961	3.0
Odegaard, 1961	1.4
Adelstein <i>et al</i> , 1964	1.9
Grewel, 1967	2.6
Lehmann, 1971 (England)	1.6–1.9
Mean	2.1

do with biological make-up: there may be genetic differences due to the different chromosomal make-up of the sexes, or female sex hormones may have an effect in some way. A more popular line of explanation lies in social effects of life stress, of social vulnerability factors and absence of support, and of women's role in society more widely. A fourth explanation is that the two sexes may differ in the way distress gets acknowledged and directed: into depression in females, in other ways and disorders in males. This explanation lies somewhere between and involves aspects of all the other three.

The remainder of this paper will examine detailed evidence for each of these broad explanatory hypotheses.

Help-seeking behaviour

Conclusive answers from the literature can only be given to the first explanation: the differential sex ratio is not simply due to differences in help-seeking behaviour. There are some well documented differences in the extent to which the two sexes tend to use medical services in general. Most studies of attendance at doctors for physical or psychiatric complaints, show women attending more. For instance, Hinkle *et al* (1960), in their study of telephone employees in New York aged over 20 years, found women had more visits to the doctor and were away from work for health reasons more frequently than men. These differences were almost all accounted for by minor illnesses: men had more life-threatening illnesses and had higher death rates. Higher consulting rates do not necessarily mean a greater readiness to consult: they might simply mean more illness. Kessler *et al* (1981) analysed data for psychiatric symptoms from four large-scale community surveys. Women did report more psychiatric distress but also showed a greater readiness to consult at the same level of morbidity: this appeared to be due to a greater readiness to translate non-specific feelings of distress into a recognition that they had an emotional problem.

However, for specific depressive symptoms and anxiety the female predominance in treated samples is found to an equal or greater extent in community samples. It is useful to make a distinction in these between studies using questionnaires, which report symptoms, but not necessarily of sufficient severity

Table 2

Sex ratios of point prevalence of depressive symptoms in some community samples (from Boyd & Weissman, 1982)

Study	Female : male ratio
Martin <i>et al</i> , 1957	2.4
Warheit <i>et al</i> , 1973	1.8
Mellinger <i>et al</i> , 1974	1.8
Blumenthal, 1975	2.7
Comstock & Helsing, 1976	
Missouri	1.4
Maryland	1.8
Weissman & Myers, 1978a	
1967 survey	1.3
1969 survey	1.8
Mean	1.9

Table 3

Prevalence rates for depressive disorders in recent community surveys employing psychiatric definitions for major depression – PSE/ID/CATEGO and DSM-III/RDC

	Rates per 100		
	Men	Women	Total
<i>PSE/ID/CATEGO (1 month)</i>			
Henderson <i>et al</i> , 1979	2.6	6.7	4.1
Bebbington <i>et al</i> , 1981	4.8	9.0	7.0
Dean <i>et al</i> , 1983		5.9	
Mavreas <i>et al</i> , 1978	4.3	10.1	7.4
<i>DSM-III/RDC (6 month)</i>			
Weissman & Myers, 1978b	3.2	5.2	4.3
Dean <i>et al</i> , 1983		7.0	
Myers <i>et al</i> , 1984	1.7	4.0	3.0
Canino <i>et al</i> , 1987	2.4	3.3	3.0
Mean	3.2	6.4	4.8

to reach the level of disorder, and studies employing definitions for psychiatric disorder, which usually give lower rates.

With regard to depressive symptoms, Boyd & Weissman (1982) reviewed the point prevalence in some community studies. These gave relatively high rates of 13%–20% overall. Table 2 summarises the sex ratios. From these data a mean sex ratio of 1.9 can be calculated, which is virtually the same as that for treated depression.

Studies employing psychiatric definitions give lower total rates, as might be expected from more stringent criteria. Here, the recent large-scale epidemiological studies employing DSM-III/Research Diagnostic Criteria (RDC; Spitzer *et al*, 1978) or PSE criteria, including the US Epidemiological Catchment Area (ECA) studies, provide the best evidence. Table 3 summarises six-month prevalences for major depression in some of these studies. Calculating average sex ratios gives a figure of 2.0 – again virtually identical to that for treated depression.

The female predominance of depression is therefore not an artefact of treatment seeking. In the US ECA studies, women with psychiatric disorders were a little more likely to consult a doctor than men, although, if they sought help, men were more likely to turn to a specialist. The overall difference was small, around 10%–20% (Shapiro *et al*, 1984), and this was for disorder irrespective of diagnosis. There is relatively little direct evidence specific to depression of clinical intensity as to who seeks help.

Biological causes

Given that the difference is real, what is the likely explanation? Here we are on less certain grounds

regarding conclusions. Part of the problem has been the absence of tough-minded research directly aimed at resolving the questions; part is that the right kinds of studies, comparing the two sexes, are not easy.

Biological explanations have centred around genes or hormones. Regarding the former, there is clear evidence from twin studies of a genetic element in affective disorders. This is most marked for bipolar disorder but fairly clear, although less in magnitude, for unipolar psychotic depression (McGuffin & Katz, 1986). For unipolar neurotic depression, although there is also a familial element, twin studies are far less conclusive as to whether its origin is genetic or environmental. Even for bipolar disorder, the genetic evidence now suggests multiple genes rather than single ones.

If a disorder is X-linked, with a gene on the X chromosome, differential sex incidence will be found. X-linked dominant disorders will be more common in women. An X-linked recessive condition, such as haemophilia, almost solely occurs in men but can be transmitted to offspring only via women.

There is some evidence of X linkage in bipolar disorder. This evidence depended initially on linkage studies using phenotypic markers, but there are now more recent studies using markers localised to the distal portion of the long arm of the X chromosome which show X linkage (Mendlewicz *et al*, 1987). If it does occur, it is only in some families rather than in all. However, this is unlikely to explain the sex incidence. The biggest problem lies in the sex incidence of bipolar disorder, which is approximately equal. Here there are some good studies of incidence, which, where possible, is more illuminating than prevalence since it is not affected by differential prognosis. In some Danish annual incidence studies quoted by Boyd & Weissman (1982) the female : male ratios were 1.1, 1.3, 0.5, 1.3, averaging, in this unrepresentative series, at 0.9. These studies are not exhaustive but others, including prevalence figures in the American ECA studies, show similar trends. There is no good evidence for X linkage in unipolar disorders.

This difference between bipolar disorder and the other affective disorders in sex incidence is important and has not received enough comment. It reinforces the evidence that bipolar disorder is a separate disorder. It also appears more biological in origin than other forms of depression: perhaps this hints that the female predominance in unipolar disorders is more psychosocial in origin.

The more obvious biological possibility lies in the important hormonal differences between women and men, the former having exposure to oestrogens and

progestogens rather than predominantly to androgens, and to cycling of gonadotrophins and sex hormones. Here it is virtually impossible to make useful direct female : male comparisons at present – such comparisons show the obvious, but in most circumstances are totally confounded by other sex and gender differences.

Indirect inferences can be attempted from some possibly hormone-related phenomena. Pre-menstrual tension appears to be a real phenomenon, which includes depression among other feelings such as tension and irritability. Occasionally women show virtually a clinical depression pre-menstrually, particularly, in my clinical experience, when relapsing into a depressive episode. Unfortunately, however, the specific hormonal change responsible for pre-menstrual tension remains obscure.

The remaining lines of evidence are rather negative. Oral contraceptives were blamed for depression at one time: studies are at best equivocal and several placebo-controlled studies do not show any increase in depressed mood (Weissman & Slaby, 1973). The effect, if present, is a small one: clinical major depression rarely appears related to use of oral contraceptives.

Childbirth is undoubtedly associated with an increase in onsets of major psychosis and hospital admissions in the first post-partum month (Kendell *et al*, 1987), and is a time of massive hormonal change. Increased onset of bipolar disorder does seem to occur at this time, but many post-partum psychoses are not affective. Recent evidence indicates that such psychoses are not related to recent life stress, making hormonal aetiology more plausible (Martin *et al*, 1989; Brockington *et al*, 1990; Dowlatshahi & Paykel, 1990).

Milder post-partum depression at the general practice or subclinical level is common, but recent studies (Cox *et al*, 1989) making comparisons with age-matched non-postpartum women suggest that rates are not very dramatically raised: depression is common in married women in the childbearing era. Such milder depression appears to be highly related to life events and social stress (Paykel *et al*, 1980; Watson *et al*, 1984; Cooper & Stein, 1989).

However, the excess of depression in women is by no means confined to the period shortly after childbirth. Onsets at this time cannot explain more than a very small proportion of the total.

The fourth possibility, the biological menopause, does not make a contribution. At least three studies have failed to show any peak in major depressive disorder at the biological menopause (Winokur, 1973; McKinley & Jeffries, 1974; Hallstrom, 1973), although psychosocial changes and life events

occurring around this time in the life cycle may be important.

This leaves little solid to depend on. Nevertheless, it is hard to ignore the possible contribution of hormones. This need not be directly in adulthood. The developing male and female brains *in utero*, in the neonatal period and in infancy are exposed to different hormonal environments, resulting in such effects as later hormonal cycling. Differences in brain function related to level of emotionality or particular mood states cannot be ruled out, at least until brain function is better studied.

Social causes

Social causes have been the focus of more research. Stronger links are emerging, although the evidence is not as strong as some of the claims. Much of the research, for instance that on social vulnerability factors, has been confined to female samples. Before conclusions about their contribution to the sex ratio can be drawn, we need to have similar studies in men.

Social explanations have generally concerned life events, social support, or women's roles and status. Regarding the first, there is now a large and conclusive volume of research showing that clinical depressions are preceded by elevated rates of the more threatening classes of life events (Paykel & Cooper, 1991). Women do not appear to experience more life events than men. Studies in the community which have looked at the question suggest equal event rates, but that women react with higher symptom intensities to the same stress (Uhlenhuth & Paykel, 1973). Also the two sexes, when asked to rate the stressfulness of different events, weight them similarly (Paykel *et al*, 1971). The answer appears to lie in a greater vulnerability to the effects of life events, rather than more life-event stress. Such vulnerability might be genetic or environmental, biological or social.

Social vulnerability studies have laid particular emphasis on social support. They include the seminal studies of women by Brown and Harris (Brown & Harris, 1978; Brown & Prudo, 1981; Brown *et al*, 1986) implicating, most clearly, the absence of a confidant, and less consistently, the presence of young children at home, being of lower social class, not working, and early loss of mother. Although there has been much debate about these, the evidence from other studies seems quite good for the first two or three. For work outside the home there has been less replication, and studies suggest more complex effects. For instance, Parry (1986) found that, in the case of working-class women, working outside the home was associated with less depression where there

was good social support; where there was not good support, however, it was associated with more depression. Important factors seem to be whether a woman chooses to work or is driven to it by necessity and whether it leaves no gap at home or produces a situation where problems with growing children are worsened. All these need testing in men. The available evidence suggests that not working because of unemployment is generally very bad for them.

The third set of social explanations concerns the psychosocial disadvantages of women's roles and status. Such hypotheses suggest that particularly the housewife role is associated with low social status. Social discrimination makes it difficult to achieve mastery by direct action and assertion, inequities lead to legal and economic helplessness, dependency on others, chronically low self-esteem, and low aspirations. Learned helplessness is induced, starting from childhood socialisation, self-images and expectations. Again, Brown's work has contributed in relation to depression by a recent focus on low self-esteem and its antecedents (Brown *et al*, 1986): again the way forward would also seem to lie in comparative studies of the two sexes.

Epidemiological studies

Epidemiological studies are starting to provide some answers concerning the interactions of sex with age, marital status and having children.

Jorm (1987) carried out combined analyses of a large number of studies of sex and age in depression. He looked at sex ratio with age and found a curvilinear relationship. The female predominance is absent in childhood, most marked in middle age, and weak in old age. In a complicated regression analysis looking at rates of depression rather than just ratios, there was a high rate for women in their 20s which declined slowly as they got older. The rise in rate reached its peak well after puberty in females, and the decline started well before the menopause and showed no acceleration then: these are difficult to explain on endocrine hypotheses.

For marital status there is a somewhat consistent trend, as was pointed out in the 1970s by Gove, an American sociologist (e.g. Gove & Tudor, 1973). He noted that high rates of many mental illnesses for women are particularly accounted for by married women: single women have lower rates, although for those divorced, separated and widowed, rates are often high. For men the ratio is reversed: those who are single have higher rates than those who are married. For men marriage appears to be protective, for women detrimental. This result was also found

in the Camberwell survey (Bebbington *et al*, 1981), although not all studies support it (Romans-Clarkson *et al*, 1988). Bebbington (1987), reviewing first admission statistics for depression, found lower rates for the married than the single in both sexes, but again the effect was much more marked in men than in women.

With regard to age, sex and marital status, there are some old findings which have never achieved the prominence they deserve. Grad de Alarcon and colleagues set up a case register to compare services in Chichester and Salisbury, around 1960. Later, they reported specific referral rates for psychiatric treatment by age, sex, marital status and diagnosis (Grad de Alarcon *et al*, 1975). For depression there was the expected excess of women over men in middle age, and this was found for married women in particular. They also found an excess of neurotic depression for married women aged 25–44 years. There was in fact a later peak for psychotic depression, in unmarried women aged 35–65 years, but this was smaller and less convincing. Other studies, not altogether consistently, suggest similar results.

The last piece of evidence concerns having children. Gater *et al* (1989) found that first admission rates for affective psychosis for the North West Region were higher in women. Detailed breakdown of the results showed the excess was accounted for by women who had had one or more children. Marital status and parity are closely associated, but looking respectively at unmarried women with children and those married but without children, the excess appeared to be accounted for by having children. Unfortunately, these latter conclusions were of necessity based on rather low numbers. Once you have a child, of course, you have the social consequences for a long time. Bebbington *et al* (1991), using data for prevalence of minor affective illness, obtained similar findings, although in their data it was less clear whether having children rather than marital status was responsible.

These findings point in a consistent direction. There is a particular peak of depression in women aged 20–40 years who are married and have children. It is difficult to reconcile these findings with any endocrine hypothesis, easy to do so with hypotheses of social causation pointing to the particular problems of young mothers in developed Western societies, where families are nuclear and geographically mobile, extended family support is uncommon, and a woman with children is particularly dependent on the quality of relationships with her partner. In our own studies of life events and depression, marital arguments and break-up have

been among the most common antecedent events (Paykel, 1974).

Differential acknowledgement and direction of distress

A final possible explanation for the differential rates lies in a difference in acknowledgement and direction of distress. It is commonplace that women in our society cry more readily than men. It is accepted as normal that a woman should weep on receiving very bad news but it is regarded as dubious for men to do so. This is usually explained as a matter of social acceptance in our culture, and undoubtedly much of it is. Some of it could be biological: higher emotionality might be a concomitant of greater sensitivity in interpersonal relationships and perceptiveness of the needs of others, something which might have biological value in differentiated childrearing.

This concerns normal mood but might carry over to pathological disorder. Some other problems are more common in men, particularly alcoholism and crime. Crime does not bear any great relationship to mood disorder, and in most cases it is not psychiatric. Alcoholism does have a relationship to mood disorder. Winokur (1979) described 'depressive-spectrum' disease in which women tend to have depression, while male family members have alcoholism and antisocial personality. It could be that men disguise their depression by drinking, or, at a deeper level, distress is directed into other disorders. Men have a higher rate of suicide than women, so perhaps the depression is there. There might be other explanations for this difference, however: more women unsuccessfully attempt suicide, and the choice of method may determine outcome, since men tend to use violent methods of high lethality, women to use less lethal overdoses.

It might therefore be that, irrespective of help seeking, women are more prepared to acknowledge their depression and to report it in surveys. Briscoe (1982) found women more willing to acknowledge feelings, both positive and negative. Angst & Dobler-Mikola (1984), in a community survey in a Zurich canton, found one-year prevalences for depression to be higher in women than men, but little difference between the sexes for prevalence in the last three months. They suggested the most likely explanation to be that men tended to forget. This seems more relevant to identification of mild cases in the community than to severe treated disorder.

A valuable approach in confirming this lies in studies of other cultures where such factors might operate differently. The Old Order Amish of

Pennsylvania, a fundamentalist religious group, are a subculture in the US who lead a 19th-century life, with strong prohibitions on drink and antisocial behaviour. They appear to have high rates of affective disorder. Among them unipolar disorder, as well as bipolar, shows an approximately equal sex incidence (Egeland & Hotstetter, 1983).

More distant cultures would be informative. Weissman & Klerman (1977) reviewed a number of studies from Guinea, India and Papua, where the Western sex ratio appeared to be reversed to a male predominance. Unfortunately they were all studies of treated samples. In many of these cultures women lead a life which is relatively enclosed in the family, and they might not have access to help-seeking to the same extent as men. More community studies are needed, and are starting to appear. Such studies will have to contend with methodological difficulties of non-Western ways of expressing depression, and also to look at factors which might change the rates, such as better extended family support.

A final piece of evidence is provided by Western temporal trends. There is suggestive evidence that reported Western rates of depression, as detected in community surveys, are increasing, particularly for young men, and that the sex ratios are becoming more equal. The ECA studies in the US have been inferred to show this (Klerman, 1988), as has a similar study in Christchurch, New Zealand (Joyce *et al*, 1990). Care is needed in interpretation: the conclusion depends on reported lifetime rates being higher in the young than would be expected from lifetime rates in the old, but the instrument, the Diagnostic Interview Schedule, may well be unreliable for lifetime recollection (Parker, 1987), and the elderly might simply forget.

More persuasively, Murphy (1986) has reviewed four community studies, two in the USA, one in Canada and one in Sweden, repeated 10–20 years apart. They showed a trend for sex ratios to equalise. There have been considerable societal changes towards less gender-role differentiation, especially in Scandinavia, which might both change the acknowledgement and the social stress. Perhaps men are starting to acknowledge their depression. Sex ratios for depression appear to be more equal in University students (Wilhelm & Parker, 1989), and Jenkins (1985) found no major sex differences in rates in civil service executive officers.

Conclusions

The explanations for a differential sex incidence of depression are still only tentative. The most plausible conclusion is that there are multiple factors at work in the same direction.

Help-seeking behaviour may make a small contribution to treated rates, but only a small one. Biological factors cannot be discounted, at least not those based more on the effects of hormonal environment on brain biology than on an abnormal gene on the X chromosome. There is a particular vulnerability to depression in married women aged 20–40 years with children. This strongly suggests a social explanation. Differential acknowledgement may also be important, and may be changing. More research is needed which, in illuminating this specific problem, is likely in addition to reveal much that is of central importance for the genesis of depression in general.

References

- ADELSTEIN, A. M., DOWNHAM, D. Y., STEIN, Z., *et al* (1964) The epidemiology of mental illness in an English city: inceptions recognised by Salford Psychiatric Services. *Social Psychiatry*, **3**, 445–468.
- AMERICAN PSYCHIATRIC ASSOCIATION (1980) *Diagnostic and Statistical Manual for Mental Disorders* (3rd edn) (DSM-III). Washington, DC: APA.
- ANGST, J. & DOBLER-MIKOLA, A. (1984) The definition of depression. *Journal of Psychiatric Research*, **18**, 401–406.
- BEBBINGTON, P. E. (1987) Marital status and depression: a study of English national admission statistics. *Acta Psychiatrica Scandinavica*, **75**, 640–650.
- , HURRY, J., TENNANT, C., *et al* (1981) Epidemiology of mental disorders in Camberwell. *Psychological Medicine*, **11**, 561–579.
- , DEAN, C., DER, G., *et al* (1991) Gender, parity and the prevalence of minor affective disorder. *British Journal of Psychiatry*, **158**, 40–45.
- BLUMENTHAL, M. D. (1975) Measuring depressive symptomatology in a general population. *Archives of General Psychiatry*, **32**, 971–978.
- BOYD, J. H. & WEISSMAN, M. M. (1982) Epidemiology. In *Handbook of Affective Disorders*, (ed. E. S. Paykel) Edinburgh: Churchill Livingstone.
- BRISCOE, M. (1982) Sex differences in psychological well-being. In *Psychological Medicine* (monograph suppl. 1). Cambridge: Cambridge University Press.
- BROCKINGTON, I. F., MARTIN, C., BROWN, G. W., *et al* (1990) Stress and puerperal psychosis. *British Journal of Psychiatry*, **157**, 319–326.
- BROWN, G. W. & HARRIS, T. O. (1978) *Social Origins of Depression. A Study of Psychiatric Disorder in Women*. London: Tavistock.
- & PRUDO, R. (1981) Psychiatric disorder in a rural and an urban population: 1. Aetiology of depression. *Psychological Medicine*, **11**, 581–599.
- , ANDREWS, B., HARRIS, T., *et al* (1986) Social support, self-esteem and depression. *Psychological Medicine*, **16**, 813–831.
- CANINO, G. J., BIRD, H. R., SHROUT, P. E., *et al* (1987) The prevalence of specific psychiatric disorders in Puerto Rico. *Archives of General Psychiatry*, **44**, 727–735.
- CANNON, M. & REDICK, R. (1973) *Differential Utilisation of Psychiatric Facilities by Men and Women: US 1970*. Statistical note 81, June 1973. Washington DC: Surveys and Reports Section, US Department of Health, Education and Welfare.
- COMSTOCK, G. W. & HELSING, K. J. (1976) Symptoms of depression in two communities. *Psychological Medicine*, **6**, 551–563.

- COOPER, M., LEMKAU, P. & TIETZE, C. (1942) Complaint of nervousness and the psychoneuroses: an epidemiological viewpoint. *American Journal of Orthopsychiatry*, **12**, 214–223.
- COOPER, P. J. & STEIN, A. (1989) Life events and postnatal depression: the Oxford study. In *Current Approaches: Childbirth as a Life Event* (eds J. L. Cox, E. S. Paykel & M. L. Page). Southampton: Duphar Medical Relations.
- COX, J. L., PAYKEL, E. S. & PAGE, M. L. (eds) (1989) *Current Approaches: Childbirth as a Life Event*. Southampton: Duphar Medical Relations.
- DEAN, C., SURTEES, P. G. & SASHIDHARAN, S. P. (1983) Comparison of research diagnostic systems in an Edinburgh community sample. *British Journal of Psychiatry*, **142**, 247–256.
- DOWLATSHAHI, D. & PAYKEL, E. S. (1990) Life events and social stress in puerperal psychoses: absence of effect. *Psychological Medicine*, **20**, 655–662.
- DUVAL, H. J., KRAMER, M. & LOCKE, B. Z. (1966) Psychoneuroses among first admissions to psychiatric facilities in Ohio, 1958–1961. *Community Mental Health Journal*, **2**, 237–243.
- EGELAND, J. A. & HOSTETTER, A. M. (1983) Amish study. I: Affective disorders among the Amish, 1976–1980. *American Journal of Psychiatry*, **140**, 56–61.
- ESSEN-MOLLER, E. & HAGNELL, O. (1961) The frequency and risk of depression within a rural population in Scandinavia. *Acta Psychiatrica Scandinavica*, **162**, (suppl.), 28–32.
- GARDNER, E. A., BAHN, A. K., MILES, H. C., *et al* (1963) All psychiatric experience in a community. *Archives of General Psychiatry*, **9**, 365–378.
- GATER, R. A., DEAN, C. & MORRIS, J. (1989) The contribution of childbearing to the sex difference in first admission rates for affective psychosis. *Psychological Medicine*, **19**, 719–724.
- GOVE, W. R. & TUDOR, J. R. (1973) Adult sex roles and mental illness. *American Journal of Sociology*, **78**, 812–835.
- GRAD DE ALARCON, J., SAINSBURY, P. & COSTAIN, W. R. (1975) Incidence of referred mental illness in Chichester and Salisbury. *Psychological Medicine*, **5**, 32–54.
- GREWEL, F. (1967) Psychiatric differences in Ashkenazim and Sephardim. *Psychiatry, Neurology and Neurochir*, **70**, 339–347.
- HALLSTROM, T. (1973) *Mental Disorder and Sexuality in the Climacteric*. Goteberg, Sweden: Ordadius Biktryckeri AB.
- HENDERSON, S., DUNCAN-JONES, P., BYRNE, D. G., *et al* (1979) Psychiatric disorder in Canberra. A standardised study of prevalence. *Acta Psychiatrica Scandinavica*, **60**, 355–374.
- HINCKLE, L. E., REDMONT, R., PLUMMER, N., *et al* (1960) II. An explanation of the relation between symptoms, disability, and serious illness in two homogeneous groups of men and women. *Journal of Public Health*, **50**, 1327–1336.
- JENKINS, R. (1985) Sex differences in minor psychiatric morbidity. In *Psychological Medicine* (monograph suppl. 7). Cambridge: Cambridge University Press.
- JORM, A. F. (1987) Sex and age differences in depression: a quantitative synthesis of published research. *Australian and New Zealand Journal of Psychiatry*, **21**, 46–53.
- JOYCE, P. R., OAKLEY-BROWNE, M. A., WELLS, J. E., *et al* (1990) Birth cohort trends in major depression: increasing rates and earlier onset in New Zealand. *Journal of Affective Disorders*, **18**, 83–89.
- JUEL-NIELSEN, N., BILLE, M., FLYGENRING, J., *et al* (1961) Frequency of depressive states within geographically delimited population groups. *Acta Psychiatrica Scandinavica*, **162**, 69–80.
- KENDELL, R. E., CHALMERS, J. C. & PLATZ, C. (1987) Epidemiology of puerperal psychoses. *British Journal of Psychiatry*, **150**, 662–673.
- KESSLER, R. C., BROWN, R. L. & BROMAN, C. L. (1981) Sex differences in psychiatric help-seeking: evidence from four large-scale surveys. *Journal of Health and Social Behavior*, **22**, 49–64.
- KIELHOLZ, P. (1959) Drug treatment of depressive states. *Canadian Psychiatric Association Journal*, **4S**, 129–137.
- KLERMAN, G. L. (1988) The current age of youthful melancholia. Evidence for increase in depression among adolescents and young adults. *British Journal of Psychiatry*, **152**, 4–14.
- LEHMANN, H. E. (1971) The epidemiology of depressive disorders. In *Depression in the 70s* (ed. R. R. Fieve). The Hague: Excerpta Medica.
- MARTIN, C. J., BROWN, G. W., GOLDBERG, D. P., *et al* (1989) Psychosocial stress and puerperal depression. *Journal of Affective Disorders*, **16**, 283–293.
- MARTIN, F. M., BROTHERTON, J. H. F. & CHAVE, S. P. W. (1957) Incidence of neurosis in a new housing estate. *British Journal of Preventive and Social Medicine*, **11**, 196–148.
- MAVREAS, V. G., BEIS, A., MOUYIAS, A., *et al* (1978) Psychiatric disorders in Athens. A community study. *Social Psychiatry*, **21**, 172–181.
- MCGUFFIN, P. & KATZ, R. (1986) Nature, nurture and affective disorder. In *The Biology of Depression* (ed. J. F. W. Deakin). London: Royal College of Psychiatrists/Gaskell.
- McKINLEY, S. M. & JEFFRIES, M. (1974) The menopausal syndrome. *British Journal of Preventive and Social Medicine*, **28**, 108–115.
- MELLINGER, G. D., BALTER, M. B., PARRY, H. J., *et al* (1974) An overview of psychotherapeutic drug use in the United States. In *Drug Use: Epidemiological and Sociological Approaches*, (eds E. Josephson & E. E. Carrol), pp. 333–336. New York: Hemisphere Publishing.
- MENDELWICZ, J., SIMON, P., SEVY, S., *et al* (1987) Polymorphic DNA markers on X chromosome and manic-depression. *Lancet*, **i**, 1230–1231.
- MURPHY, J. M. (1986) Trends in depression and anxiety: men and women. *Acta Psychiatrica Scandinavica*, **73**, 113–127.
- MYERS, J. K., WEISSMAN, M. M., TISCHLER, G. L., *et al* (1984) Six-month prevalence of psychiatric disorders in three communities: 1980–1982. *Archives of General Psychiatry*, **41**, 959–967.
- ODEGAARD, O. (1961) The epidemiology of depressive psychoses. *Acta Psychiatrica Scandinavica*, **162**, 33–38.
- PARKER, G. (1987) Are lifetime prevalence estimates in the ECA study accurate? *Psychological Medicine*, **17**, 275–282.
- PARRY, G. (1986) Paid employment, life events, social support and mental health in working class mothers. *Journal of Health and Social Behaviour*, **27**, 193–208.
- PAYKEL, E. S. (1974) Recent life events and clinical depression. In *Life Stress and Illness* (eds E. K. E. Gunderson & R. H. Rahe). Springfield, Illinois: Charles C. Thomas.
- & COOPER, Z. (1991) Life events and social stress. In *Handbook of Affective Disorders* (2nd edn) (ed. E. S. Paykel). Edinburgh: Churchill Livingstone (in press).
- , EMMS, E. M., FLETCHER, J., *et al* (1980) Life events and social support in puerperal depression. *British Journal of Psychiatry*, **136**, 339–346.
- , KLERMAN, G. L. & PRUSOFF, B. A. (1970) Treatment setting and clinical depression. *Archives of General Psychiatry*, **22**, 11–21.
- , PRUSOFF, B. A. & UHLENHUTH, E. H. (1971) Scaling of life events. *Archives of General Psychiatry*, **25**, 340–347.
- PEDERSEN, A. M., BARRY, D. J. & BABIGIAN, H. M. (1972) Epidemiological considerations of psychotic depression. *Archives of General Psychiatry*, **27**, 193–197.
- ROMANS-CLARKSON, S. E., WALTON, V. A., HERBISON, G. P., *et al* (1988) Marriage, motherhood and psychiatric morbidity in New Zealand. *Psychological Medicine*, **18**, 983–990.
- ROSEN, B. F., BAHN, A. K. & KRAMER, M. (1964) Demographic and diagnostic characteristics of psychiatric clinic outpatients in the USA, 1961. *American Journal of Orthopsychiatry*, **34**, 445–468.
- SHAPIRO, S., SKINNER, E. A., KESSLER, L. G., *et al* (1984) Utilization of health and mental health services. Three epidemiologic catchment area sites. *Archives of General Psychiatry*, **41**, 971–978.

- SPITZER, R. L., ENDICOTT, J. & ROBINS, E. (1978) Research Diagnostic Criteria: rationale and reliability. *Archives of General Psychiatry*, **35**, 773–782.
- TARNOWER, S. M. & HUMPHRIES, M. (1969) Depression: a recurring, genetic illness more common in females. *Diseases of the Nervous System*, **30**, 601–604.
- UHLENHUTH, E. H. & PAYKEL, E. S. (1973) Symptom intensity and life events. *Archives of General Psychiatry*, **28**, 473–477.
- WARHEIT, G. J., HOLZER III, C. E. & SCHWAB, J. J. (1973) An analysis of social class and racial differences in depressive symptomatology: a community study. *Journal of Health and Social Behaviour*, **14**, 291–299.
- WATSON, J. P., ELLIOTT, S. A., RUGG, A. J., *et al* (1984) Psychiatric disorder in pregnancy and the first postnatal year. *British Journal of Psychiatry*, **144**, 453–462.
- WECHSLER, H. (1961) Community growth, depressive disorders, and suicide. *American Journal of Sociology*, **67**, 9–16.
- WEISSMAN, M. M. & KLERMAN, G. L. (1977) Sex differences and the epidemiology of depression. *Archives of General Psychiatry*, **34**, 98–111.
- & MYERS, J. K. (1978a) Rates and risks of depressive symptoms in a United States urban community. *Acta Psychiatrica Scandinavica*, **57**, 219–231.
- & —— (1978b) Affective disorders in a US urban community. The use of Research Diagnostic Criteria in an epidemiological survey. *Archives of General Psychiatry*, **35**, 1304–1311.
- & SLABY, A. E. (1973) Oral contraceptives and psychiatric disturbance: evidence from research. *British Journal of Psychiatry*, **123**, 513–518.
- WILHELM, K. & PARKER, G. (1989) Is sex necessarily a risk factor to depression? *Psychological Medicine*, **19**, 401–413.
- WING, J. K. & STURT, E. (1978) *The PSE – ID – CATEGO System: Supplementary Manual*. London: MRC Social Psychiatry Unit.
- , COOPER, J. E. & SARTORIUS, N. (1974) *Measurement and Classification of Psychiatric Symptoms: an Instruction Manual for the PSE and Catego Program*. London: Cambridge University Press.
- WINOKUR, G. (1973) Depression in the menopause. *American Journal of Psychiatry*, **130**, 92–93.
- (1979) Unipolar depression. Is it divisible into autonomous subtypes? *Archives of General Psychiatry*, **36**, 47–52.

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