levels, that anosmia is due to low gonadotrophins and that failure of the temperature to fall at night is due to the absence of the hypothermic influence of dopamine.

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GENETICS OF MANIC-DEPRESSIVE DISEASE: ICELAND REVISITED

DEAR SIR,

Winokur *et al.* (1967, 1970), in an elegant series of studies based on family history data, have proposed a genetic theory for the aetiology of the bipolar group of manic-depressive disorders. These authors have further suggested that the transmission is through an X-linked dominant gene (Winokur *et al.*, 1969). In support of this hypothesis, they cite the well-substantiated fact that manic-depressive disease is much more common in females than in males. Other family history data on manic-depressive disorders have supported a genetic theory of transmission, but not Winokur's specific X-linked dominant gene hypothesis (Perris, 1966).

While it is true that there is an increased incidence of females over males in manic-depressive disorder, this greater incidence is also seen in *all* affective disorders regardless of specific diagnosis, as Winokur himself notes (Winokur *et al.*, 1969). This is illustrated by the figures for patients admitted with diagnoses of depressive disorders to the in-patient services of state and county mental hospitals in the United States during 1969 (Department of Health, Education and Welfare, Publication #HSM 72-9048, 1971). These H.E.W. data would suggest that the explanation for the approximately 2 : 1 ratio of females to males would have to be broader than Winokur's specific X-linked genetic hypothesis for bipolar depressions. There is additional evidence from an epidemiological study in Iceland (Helgason, 1964) which appears to be incompatible with an X-linked dominant hypothesis for manic-depressive illness.

Helgason's data consisted of 5,395 probands who were born in the years 1895–1897 and who were followed until 1957. Within the category of manic-depressive psychosis Helgason included patients who had had periods of elation or depression, or both, which occurred without known external precipitants. He also included as part of this group patients with the diagnosis of involutional depression. A total of 81 probands received a diagnosis of manic-depressive psychosis: of these, 51 had had depressions only 7 had had mania only, and 23 had had both mania and depression. The overall expectancy rate for manic-depressive disorder was 1·80 for males and 2·46 for females, giving a ratio which is compatible with an X-linked dominant hypothesis. Helgason was also concerned with migratory patterns and their relationship to the prevalence of mental disorders. Consequently, he divided his probands according to their place of residence (rural/urban) at the beginning (1910) and the end (1957) of the observation period. He then reanalysed his expectancy rates according to the place-of-residency variable (Table I).

The female/male ratio of the expectancy rate for developing manic-depressive disease for probands living in rural areas in 1910 was approximately 2 : 1 (3·77 for females, 1·88 for males). The female/male ratio of expectancy rate for probands living in urban areas in 1910, however, was approximately 2 : 3 (2·09 for females, 2·98 for males). A chi-square analysis (computed by the present authors) on the absolute numbers of manic-depressive females and males in rural and urban areas, allowing for differences in numbers of probands at risk in each area, is significant at the $p < \cdot 01$ level ($\chi^2 = 5 \cdot 12$). Further, when the expected numbers of manic-depressive males and females were calculated for rural and urban areas, using the ratio derived from an X-linked dominant gene hypothesis (1 male : 2 female at the population incidence of manic-depressive disorder), and compared with Helgason's observed number of cases in each category (rural and urban), they differed significantly ($\chi^2 = 11 \cdot 3$, $p < \cdot 01$, see Table

TABLE I

Expectancy rates for manic-depressive psychosis according to residence at the beginning of the observation period

	MALE			
	Rural (adjusted population at risk*—1,634·96		Urban (adjusted population at risk*—625·12)	
	No. of cases	Expectancy rate	No. of cases	Expectancy rate
Manic-depressive disease	20	1·88	13	2·98
Schizophrenia	11	0·68	3	0·72
	FEMALE			
	Rural (adjusted population at risk—1,557·08)		Urban (adjusted population at risk—732·84)	
	No. of cases	Expectancy rate	No. of cases	Expectancy rate
Manic-depressive disease	38	3·77	10	2·09
Schizophrenia	13	0·90	9	1·28

Manic-depressive disease female to male ratio rural *vs.* urban $\chi^2 = 5{\cdot}12$, $p < {\cdot}01$.

Schizophrenia female to male ratio rural *vs.* urban—N.S.

* Adjusted population at risk has been determined by the weighting method of E. Slater (1938) as cited by Helgason; weights are differentially calculated according to age of onset and manifestation period of various disorders.

II). It is not possible, therefore, to explain Helgason's difference in sex ratio according to place of residence entirely by an X-linked dominant genetic hypothesis. While it is true that the Helgason data include both bipolar and unipolar groups of affective disorders, the preponderance of females to males is a general phenomenon of all affective disorders.

If the X-linked dominant theory cannot alone account for the increased prevalence of affective disorders in females over males, what other explanations can be given? A recent publication by an advocate of the Woman's Liberation Movement has suggested that the increased prevalence of all mental disorders in women is due to her culturally determined role (Chesler, 1972). While it is much too sweeping a generalization to conclude that all mental disturbances are culturally determined, there is considerable evidence to suggest that socio-cultural factors do play a role in the prevalence of affective disorders. Kendall (1970), for example, concluded that the incidence of depressive disorders varies as a function of the way aggressive impulses are handled by individual societies. Further support for Kendall's hypothesis comes from a study of the incidence of affective disorders in Belfast (Lyons, 1972), which showed that the incidence of manic-depressive disorders in males declined in the riot-torn part of the city.

If the sex-linked difference in the prevalence of affective disorders is in large measure due to the cultural concomitants of the 'woman's role', one would expect that as the female's role varies so also will the prevalence of manic-depressive disorders. While there is as yet no direct evidence on this point, some

TABLE II

Comparison of observed with expected lifetime incidence of manic-depressive disorders occurring in probands living in rural and urban areas in Iceland in 1910

	*Observed rate of m dep. disease/1,000 of pop at risk	Expected rate of m dep. disease/1,000 of pop. at risk according to X-linked hypothesis
URBAN		
Male ..	20·8	11·46
Female ..	13·6	22·92
RURAL		
Male ..	12·2	12·1
Female ..	24·1	24·2

Observed *vs.* expected values—Chi square = 11·3, d.f. = 1, $p < {\cdot}01$.

* Observed rate calculated from data given in Table I, provided by Helgason (personal communication, 1973).

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indirect support comes from Craig and Pitts (1968), who calculated the suicide rates of male and female physicians for the years May 1965 to May 1967, and found them approximately equal (males: 38·3/100,000, females: 40·5/100,000). This finding is in striking contrast to the suicide rates in the general population, which are much higher for males than females. Suicide rates, according to Craig and Pitts, can be used to give an indication of the prevalence of affective disorders, since a constant 15 per cent of deaths of individuals with affective disorders are due to suicide. However, 25 per cent of the deaths of males in the general population with affective disorders are due to suicide, as compared with only 10 per cent of deaths of females in the general population with affective disorders. As the female physicians' suicide rate calculated by Craig and Pitts is very significantly ($p < \cdot00001$) higher than the suicide rate of the general population of white U.S. females (11·4/100,000) over the same time period, they conclude, using this rate as an index of depression, that the incidence of affective disorders in female physicians is very high, certainly much higher than the incidence of affective disorder in male physicians. An equally probable alternative hypothesis from the socio-cultural perspective can be made. That is, social role may affect not only the prevalence of depressive disorders but also the behavioural expression of this depression, e.g. suicides. Thus similar suicide rates in male and female physicians may reflect either similar prevalence rates of depressive disorders within this sub-population or similar behavioural expressions within this sub-population.

No final conclusions can be drawn until epidemiological studies of the prevalence of depressive disorders can be carried out in various cultural segments of our society where the 'woman's role' has become similar to the man's, e.g. as in occupational categories. It is, however, probably premature to settle on any specific explanation, genetic or otherwise, for the apparent sex-related differences in diagnostic subcategories of affective disorders.

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ECONOMICS OF REHABILITATION

DEAR SIR,

Mr. Cheadle and Dr. Morgan (*Journal*, August, pp. 193–201), have made a most valuable contribution to the discussion of rehabilitation. At a time when the scrutiny of costs is becoming more and more rigorous throughout the public services it is at least equally important to examine and measure the benefits achieved. The meticulous research which is evident in this paper is an excellent example of the process of probing into the realities of health care which will be essential for the new Health Authorities in establishing their policies.

The paper is notable for its insistence upon hard facts; indeed it is most gratifying that the Department of Health and Social Security was able to be so helpful in providing information about National Insurance contributions, Sick Benefit and Unemployment Benefit. On the other hand, it was to be expected, and readily understood, that details of income tax could not be obtained. The authors, therefore, resorted to an estimate of earnings from which they calculated tax contributions.

All this is admirably done, but it is arguable that