

Correspondence

Correspondents should note that space is limited and shorter letters have a greater chance of publication. The Editors reserve the right to cut letters and also to eliminate multitudinous references. Please try to be concise, strictly relevant and interesting to the reader, and check the accuracy of all references in Journal style.

CANCER AND DEPRESSION

DEAR SIR,

I read with interest Drs. Brown and Paraskevas article on cancer and depression (*Journal*, September 1982, **141**, 227–32). The association between depression and cancer is indeed strong and the suggestion of a possible immunologic link between the two entities is certainly appealing. The authors propose that antibodies against tumor-related proteins, because of cross-reactivity with CNS proteins that are conceivably identical to serotonin receptors, may interfere with the brain activity of serotonin and thus lead to depressive symptoms.

Although this is a reasonable hypothesis, there are conceivably other, more direct ways in which immune factors can provide the necessary link between depression and cancer. Preliminary data from our own laboratory (Kronfol *et al.*, 1982) as well as others (Linn *et al.*, 1982) suggest that depression may be associated with an impairment in cell-mediated immunity, elements of which are said to protect the organism against cancer (Penn, 1981). Since depressive symptoms usually antedate clinical manifestations of cancer, we therefore propose that depression may impair host defense mechanisms, thus allowing neoplastic cells to proliferate and spread out of control in certain predisposed individuals. The etiology of cancer is complex and still eludes our complete understanding.

Genetic predisposition, environmental factors, pharmacologic agents, hormones, diet and life style have all been suggested as possible contributing factors. When severe depression precedes cancer, the depressive illness may present an additional risk.

ZIAD KRONFOL

*University of Iowa College of Medicine, Iowa City,
Iowa 52242*

References

- KRONFOL, Z., SILVA, J., GREDEN, J., DEMBINSKI S. & CARROLL, B. J. (1982) Cell-mediated immunity in melancholia. *Psychosomatic Medicine*, **44**, 304.
LINN, B., LINN, M., & JENSEN, J. (1982) Degree of depression and immune responsiveness. *Psychosomatic Medicine*, **44**, 128–9.

PENN, I. (1981) Depressed immunity and the development of cancer. *Clinical and Experimental Immunology*, **46**, 459–74.

PARANOIA AND DYSMORPHOPHOBIA

DEAR SIR,

I would like to reply to a comment made by Alastair Munro in his recent paper "Paranoia Re-visited" (*Journal*, October 1982, **141**, 344–49). He states "Many authors fail to differentiate between neurotic and psychotic disorders with rather similar complaints. For example dysmorphophobia should be by definition a non-psychotic illness but is often used to describe delusions of misshapeness (Hay, 1970)".

This view can only result from a basic misunderstanding of my paper. In that article I resurrected the term "dysmorphophobia" which had been used in the 19th century (Morselli, 1886) to describe those patients who present with "a fear of being mishapen" when in fact objectively they have no cause for a complaint. I remarked that dysmorphophobia is a symptom not a diagnosis or illness and was at pains to point out that after investigation the eventual diagnosis could vary from a sensitive personality development to an attenuated schizophrenic illness or even occasionally to affective disorder. In other words dysmorphophobia is non-specific as a symptom and can occur in a variety of different psychiatric syndromes.

We appear to be in danger of getting lost in a semantic quibble. If we choose to restrict the term dysmorphophobia to those patients whose symptom is purely personality based, there is nothing inherently wrong with that, but we would be using the term as diagnosis. Monosymptomatic hypochondriacal psychosis can be used to describe those patients whose complaint is delusional and there are certainly advantages in this somewhat cumbersome label in that it avoids using the word schizophrenia. What we do not know is how many of those patients, either the very sensitive personalities or those with monosymptomatic hypochondriacal psychosis, become schizophrenic with time. This question can only be settled by long term studies. I am at present following up the group of

dysmorphophobics seen in the 1960's. To date the numbers are small, but at least some of the patients who had monosymptomatic delusions 20 years ago have become more floridly ill with time and the diagnosis now is that of a schizophrenic illness.

G. G. HAY

Withington Hospital, West Didsbury,
Manchester M20 8LR

References

- HAY, G. G. (1970) Dymorphophobia. *British Journal of Psychiatry*, **116**, 399–406.
MORSELLI, E. (1886) Sulla dismorfofobia e sulla tafefobia *Boll Accad Med (Genova)* VI, 110–19.

COMMUNITY SCREENING FOR MENTAL ILLNESS

Benjamin, De Calmer and Haran (*Journal*, February 1982, **140**, 174–80) claim to have shown that the General Health Questionnaire (GHQ) is “unsuitable as a screening instrument for mental illness in the community” (p 174). Since the GHQ is one of the most widely used psychiatric screening questionnaires, it is important that their claim be rigorously examined. Two issues arising from their paper require consideration.

1. The respondents were all women aged between 40 and 49 (a demographic subgroup which comprises about 6 per cent of the population). The results cannot therefore be taken to apply to men or to young or old women. Indeed, the sample was not representative of 40–49 year old women, since membership was confined to those of caucasian origin, who “were still able to pass through a ‘natural’ menopause and who could cooperate with multiple investigations of physical, mental and social state”. From a random sample of 228, only 100, or 44 per cent, met these criteria. The sample is thus not representative of anything at all. The authors are, it seems, aware of this, and they defend themselves on p. 179 by observing that “validation studies of the GHQ–30 in a consulting setting do not appear to be affected by demographic variables”. Yet earlier in their paper (p. 174) they observe, as part of their reason for doing the study, that “it is questionable whether responders to such a questionnaire will behave in an identical manner regardless of how they are identified or the circumstances in which the questionnaire is presented”. The importance of the representative nature of a sample (assuming that one wishes, as the authors do, to generalise from it) is a cornerstone of epidemiological investigation, and cannot be dismissed as irrelevant.
2. Even if the sample were representative of the

population (or some definable subgroup), there remains the issue of whether Benjamin *et al*, have in fact shown the GHQ to be “unsuitable as a screening instrument”. Their principal reason for drawing this conclusion is the low sensitivity ($18/33 = 55$ per cent) found when GHQ was compared with the Clinical Interview Schedule (Table III, p. 176), primarily because the questionnaire tended to miss chronic cases. It is worth noting that the number of cases on which this finding is based (33) is relatively small, so that the 95 per cent confidence limits, 38 per cent and 72 per cent, are widely spaced.

Apart from this, sensitivity and specificity are measures of the *validity* of a questionnaire, which is not at all the same as its potential as a screening instrument. To assess this, the relevant indices are the *predictive values* (Galen and Gambino 1975). The positive predictive value (PPV) is the probability that a screened positive will be a “true case”: for Benjamin *et al*'s, data (Table III), the value is 0.78. The negative predictive value (NPV) is the probability that a screened negative will be normal, also 0.78 for the present data. Thus, 8 out of 10 high scorers will be cases, and 8 out of 10 low scorers will not be.

It may well be said that this is unacceptable. Whether, however, the modified GHQ (15 items, Likert scoring) does any better depends on the purpose to which the screening exercise is to be put. It can be calculated from Table VI of Benjamin *et al*, (inspection of which shows that the modified GHQ fails to identify 12 per cent of cases, and not 4 per cent as they claim) that the positive and negative predictive values are 54 per cent and 94 per cent respectively. Thus, if not missing cases were the prime consideration, then the modified version is better; if it were more important to identify *only* cases, then the original version is better, since only about half of the screened positives are cases when the modified version is used. In any case, it is highly unlikely that a questionnaire derived on so atypical a sample will have general validity.

It appears that further studies are required before the claim that “the GHQ is inappropriate as a screening instrument for mental illness in the community” can be substantiated.

PAUL WILLIAMS

*Institute of Psychiatry, Denmark Hill,
London SE5 8AH*

References

- GALEN, P. S. & GAMBINO, S. R. (1975) *Beyond Normality: the Predictive Value and Efficiency of Medical Diagnosis*. New York: Wiley.