instead of a daughter, than is the case with the parents of heterosexual women, was confirmed by the findings." Again, the hypothesis only stated that lesbians would *report* the phenomenon to a greater degree. We do not know that the parents of homosexual women wanted a son more frequently.

I stress these points for the following reasons. The hurried professional, flooded by a sea of medical literature, finds it impossible to read all the articles he would like. Much reading is reduced to summaries of papers, or to abstracts which quote statements from the original paper succinctly summarizing the results. Furthermore, full-length papers frequently refer to the past related publications of other authors also by quoting one or two of the results. Thus it behoves all of us who publish to be ever scrupulous in the wording of our material. Dr. Bene's study was a carefully conducted one in an area which demands sophisticated research. Undoubtedly, it will be widely cited. Thus, I consider it important to emphasize its potential for overinterpretation.

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DEPRESSION: PSYCHOTIC/NEUROTIC; ENDOGENOUS-EXOGENOUS

DEAR SIR,

Your correspondent (Foulds, *Journal*, November 1965) begins his letter with a misunderstanding of my report. He states that "Mendels (. . .) seems to regard it (viz. the separation of endogenous and neurotic depression) as neither possible nor useful."

As far as the usefulness of the separation is concerned, I wrote (*Journal*, 1965, p. 683), "We found a striking difference in response to ECT between the groups of patients designated as endogenous and reactive". Further on, in discussing the symptomatological overlap I stated ". . . the response to treatment was significantly associated with a small difference in balance between the two groups of symptoms, in spite of the marked overlap"; and, at the risk of overstating the point, I wrote, in conclusion, "Using symptomatic definitions of endogenous and reactive, a clear-cut difference in response to ECT of two syndromes was demonstrated."

With regard to Foulds's contention that I claimed that the separation was not possible, it appears that he has been selective in his interpretation of the paper. One of the major points made is that (using the single criterion of response to ECT) these are two distinct syndromes. I emphasized the need to consider the overall clinical picture, rather than reach conclusions based on the presence or absence of individual clinical features. While this would seem axiomatic, the fact is that there are many published reports in this field which do emphasize the importance of the individual signs and symptoms (references in the original articles).

Foulds's next point (paragraph 2) also quotes the paper out of context. To correct the erroneous impression he creates, I again quote from the paper. In the course of discussing some of the implications of the results, I stated 'This lends support *to the concept* that there is always an endogenous element to a depressive illness, and that the reactive element is more variable. Furthermore, the response to ECT is *perhaps* related most intimately to the extent of the reactive components . . . " In the conclusion I reiterated that the results and conclusions were based on defined symptomatic definitions of the two depressive syndromes (i.e. that this was not meant as an exhaustive study of the problem with final conclusions); I stated that "an endogenous component" *appears* to be present in *most* of the patients; the diagnosis as well as the response to ECT is *more closely related* to the "reactive features" present. To my mind this does not support the dogmatic interpretations made by Foulds.

The typing errors in Table II, while unfortunate, were not crucial. The Table should have read: A/E, Steady, Course, 10; B/E "Adequate Premorbid Personality" 17; B/R "Adequate Premorbid Personality" 10. This criterion does not significantly alter the distribution of the symptoms. Chi Squared analysis bears out most of the original interpretations as well as the general argument (viz. the apparent "dominance" of "over reactive" and "endogenous" symptoms in the context of this experiment). Chi Squared analysis, using the figures in Column B as Foulds has done (it is much more arbitrary than Column A) and ignoring the dichotomized personality and course variables, reveals that certain "reactive" features are highly significant in their distribution (neurotic traits in adulthood p ·001; precipitating factors, p ·001; and emotional liability, p ·005). Two of the six "endogenous" factors shows less significant variation (diurnal variations (worse a.m.), p. ·05; and psychomotor retardation, p ·01). Foulds also ignores the supporting findings in the first paper: that when the factors were considered individually, emotional liability, precipitating factors, neurotic traits in early life, and inadequate premorbid personality (as a single factor) were significantly associated with poor response to

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treatment. Not one "endogenous" factor showed a significant association.

Using Column A (which is a more realistic and conservative estimate than Column B), diurnal variation is no longer significant, and psychomotor retardation is thus the only one of the six "endogenous" factors to be significant. However, the three "reactive features" which were significant in Column B remain so.

Perhaps the most significant aspect of Table II is the large number of "mixed" (really undiagnosable, according to our criteria) cases. When the more conservative method of distributing the patients was used, 32 per cent. of the patients did not fall into either the "reactive" or "endogenous" group.

To turn to Foulds's claim that the use of "adequate personality and steady course under endogenous, and their opposites under reactive", is inadmissible: These factors were originally studied as continuous variables, in which case the extremes might have validity. Furthermore, using "course of illness" as an example, if fluctuating course as reactive feature were removed, this would create a bias in the direction of making the diagnosis of "endogenous". To remove both is to ignore what may be significant components of the syndrome.

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DEAR SIR,

Professor Fish (*Journal*, January, 1966) says that I make the erroneous assumption that reactive and endogenous depressives are equivalent to my neurotic and psychotic depressives. But I criticized Carney, Roth and Garside for using terms from two different universes of discourse (endogenous and neurotic)! As the two dimensions (endogenous-exogenous and psychotic-neurotic) are used by psychiatrists, they are very far from being orthogonal. When I have been wanting to dichotomize depressives into psychotic and neurotic and some wayward psychiatrists have written endogenous or reactive, I have asked them to use psychotic: neurotic. Almost invariably endogenous and psychotic have been associated, and so have reactive and neurotic. I dislike endogenous: exogenous because it is an aetiological classification (without adequate basis and with less likelihood of inter-judge agreement than presence or absence of delusions) amidst surrounding phenomenological classes.

With regard to sleep, my more general point was that clinicians often confirm their hunches because they so arrange the situation that there is no possibility of disconfirmation. I could have made this point better had I said paranoid rather than reactive depressive.

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DEAR SIR,

Recent correspondence in the *Journal* on the nature of depressive illness is rather disturbing: it is especially a ground for despondency that controversy remains after so many years' discussion, although this is one of the occasions when clinical experience and more academic studies appear to be in agreement. One is bound to ask just what fundamental advances have been made in psychiatry for which administrators and the pharmaceutical industry are not responsible.

It is a part of human experience that some suffer changes in mood for which they can find no explanation, while others suffer from a change in mood for which an environmental cause is only too clear. Those who experience both types of mood change at one time or another can distinguish them not only by the presence or absence of an environmental cause, but also in the quality of the mood change. When they suffer reactive depression they have suffered a stress which they are, at least temporarily, unable to withstand; they lie awake thinking of the problem at night, and then sleep through the alarm clock; they forget the problem temporarily at a party and feel happier until they are again reminded of it.

These are also the symptoms of a neurotic depression, and when one moves from normal experience to experience of disease one finds neurotic depression affecting one sort of person, who experiences one set of symptoms and shows one type of response to treatment; and endogenous depression affecting another type of person, with different symptoms and a different response to treatment: and none of these differences looks like a merely different point along the same line. If neurotic depression and endogenous depression were merely quantitatively different one would have to place the endogenous depression at the more severe end of the scale; and yet we can find mild depressions which share the basic symptomatology of severe endogenous depression, which are milder than other depressions which share the symptomatology of a non-pathological reactive

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