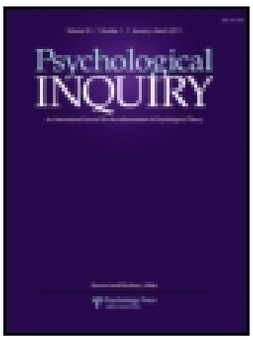

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Neuroticism, Anxiety, and Depression

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Barlow's analysis of emotional disorders presents a valuable contribution to a complex subject but leaves out important evidence on genetic and environmental relationships. It also fails to note certain psychological factors—like conditioning—that may largely contribute to the observed effects. Finally, it fails to note certain important extensions of the model to areas like cancer and coronary heart disease (CHD). My commentary is concerned with all three areas, not necessarily in a critical spirit.

1. The relationship between anxiety and depression forms an important part of Barlow's review, but it neglects the important contribution made by Eaves, N. G. Martin, Heath, and Kendler (1987), Jardine, N. G. Martin, and Henderson (1984), and Kendler, Heath, N. G. Martin, and Eaves (1986). This literature is reviewed, summarized, and extended in Eaves, H. J. Eysenck, and N. G. Martin (1989), which should be consulted as our final statement of results and conclusion. The material studied was the Bedford, Foulds, and Sheffield (1976) Personality Disturbance Scale (DSSI), which, in the form used, consisted of two 7-item subscales measuring, respectively, Anxiety and Depression. Also used was the Eysenck Personality Questionnaire (EPQ; H. J. Eysenck & S. B. G. Eysenck, 1975), particularly the Neuroticism scale. The population used was the Australian Twin Study group; responses of 3,798 adult twin pairs were available for analysis. Analyses were performed both for scales and for single items.

The scale analysis showed that the correlation between the traits (neuroticism, anxiety, depression) was comparable to the reliability of measurement: "Therefore, a very high proportion of the reliable variation in anxiety and depression symptoms is due to the same common factor measured by the [Neuroticism] scale of the EPQ" (Eaves et al., 1989, p. 305). The genetic analysis shows quite high E_W values (specific or within-family environmental variance), but this is only apparent. When we interpret the large environmental specifics, we have to remember that these are comparable to the error variances assessed by repeated measures. If we subtract measurement error from the estimates of specific variance, then we are left with virtually no trait-specific environmental effects. Thus, our model for the relationship of neuroticism to symptoms of anxiety and depression in a nonclinical population is comparatively straightforward. Variation in self-report symptoms is therefore no different from that of other measures of personality. Furthermore, the phenotypic correlations between Neuroticism and the symptom scales are high, indicating that the same factors contribute to variation in all three scales. Anxiety and Depression scores are highly correlated with each other and with Neuroticism scores. The genetic analysis of trait covariation gives strong support to the view that the same genetic effects that contribute to symptoms of anxiety also contribute to mild symptoms of depression. There is virtually no specific genetic variance in either trait. In addition, all that these symptoms have in common with one another genetically is also shared with the Neuroticism scores derived from the EPQ.

A similar view seems true for the effects of the environment. Short-term changes apart—which contribute to specific environmental variation—virtually all the environmental variation in Neuroticism and scores on the DSSI Anxiety and Depression subscales has a general effect on all scales. Long-term environmental effects contribute to all traits simultaneously. That Neuroticism, Anxiety, and Depression are not completely correlated is probably due to short-term fluctuations rather than to an underlying difference in the genetic basis of the traits. Insofar as neuroticism is a "trait" measure and anxiety and depression symptoms, as recorded, are "state" measures, we expect that the specific variation in anxiety and depression would be due largely to short-term fluctuations.

In general, our analysis shows that neuroticism, as found previously, is highly heritable in both sexes. Anxiety and depression are far more influenced by environmental effects, some of which precipitate the expression of depressive symptoms without affecting anxiety. It seems that the specific environmental effects found for the scores on the Anxiety and Depression subscales are caused by short-term environmental effects:

> One major implication of the model is that genetically "vulnerable" individuals may develop symptoms of anxiety or depression at different times in their life as a function of the particular kind of environmental stress that happens to be operating at the time of follow-up. (Eaves et al., 1989, p. 313)

At the level of item analysis, and of particular interest for Barlow's argument, we have the finding that, for the "panic" item, analysis in terms of V_A (additive genetic variance) and E_W (within-family environmental variance) gives a fairly good fit to the data, whereas a significant improvement results from the addition of a dominance parameter to the model. It is not suggested that our data contradict Barlow's model; it is merely proposed that consideration of the very detailed and complex data here very briefly and inadequately presented would extend and improve his model.

2. Barlow's discussion of fear and anxiety rightly emphasizes their essential difference but neglects to discuss the theory that anxiety is produced by a process of Pavlovian conditioning—Pavlovian B conditioning, to be precise (H. J. Eysenck, 1976, 1979). This fairly obvious hypothesis that anxiety is a learned kind of fear was already proposed by Cicero 2,000 years ago in his *Tusculan Disputations*. It does not seem right to abandon such a venerable theory, which in any case has been well translated into modern concepts and has received considerable support (H. J. Eysenck & I. Martin, 1987). Barlow may not approve of the theory, but it not only explains the relation between fear and anxiety but also suggests the origins of neurosis, its genetic basis, and its

relation to personality (H. J. Eysenck & M. W. Eysenck, 1985).

The theory not only explains the origins of anxiety, depression, and neurosis; it also suggests biological roots that may form the link between heredity and behavior (H. J. Eysenck & Kelley, 1987). It also explains its adaptive function, which no doubt accounts for its genetic survival; it foreshadows danger and helps the organism to avoid that danger. Again, it is not suggested that what is said here in any sense invalidates Barlow's model; it merely makes it more specific and brings it into line with a large body of data that has not been covered.

3. A final proposal suggests an extension of the model to take into account psychosomatic illnesses—in particular, cancer and CHD. There is now a great deal of evidence that suggests a close relation between suppressed anxiety and cancer and between suppressed anger and CHD (H. J. Eysenck, 1985, 1988a, 1988b, 1990; Grossarth-Maticek, H. J. Eysenck, & Vetter, 1988). Depression and feelings of hopelessness–helplessness, in particular, are closely related predictively to the genesis of cancer, and CHD too is linked with suppression of feelings and ineffective reactions to stress—although not exactly as proposed by adherents of the "Type A" personality theory (H. J. Eysenck, 1990).

Exploration of the rich and important areas relating to personality, anxiety, depression, stress, and disease would allow a further extension of the model toward which we are all working and some of whose properties Barlow has sketched in his article. The more that related features can be taken into account, the less likely are we to miss some important aspects or to misconstrue some vital clues. Factually oriented efforts to construct such a model, like Barlow's, are certainly to be welcomed; by their refinement, improvement, and extension, we may finally arrive at a better understanding of these exceedingly complex relationships. Particularly welcome is Barlow's recognition of our biosocial nature (H. J. Eysenck, 1980a, 1980b), placing equal emphasis on social and biological, genetic and environmental, psychological and physiological-hormonal factors in explaining behavior and cognition. Such a shift from purely environmental "empty organisms" theories has been long overdue!

Note

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