matters arising

No evidence for heritability of social attitudes

EAVES and Eysenck[1] have concluded that six types of personality orientations, including both radicalism and neuroticism, have substantial genetic components. These conclusions, based on simple models, are presented with some qualifications but not without conviction and certainty. The reader is informed that: "a model invoking only environmental factors fails to provide an adequate description of the variation and covariation of the six traits"; that, although a larger study would be desirable, "we can, however, be reasonably confident that some kind of genetic mechanism has to be involved to account for the observations so far"; and that the correct interpretation of a table presenting exact heritability estimates for each trait, is that "about half the variation between individuals for the six traits has a genetic basis".

None of these conclusions is justified by the data. Eaves and Eysenck's two-parameter environmental model tests only whether dizygotic twins are as similar in their personality scores as monozygotic twins. The high χ^2 for this model indicates that the monozygotic twins are more similar than dizygotic twins. Eaves and Eysenck simply assume that this extra similarity is genetic. In a paper[2] on the heritability of IQ performance we showed that comparisons between monozygotic and dizygotic twins can only set upper limits to the heritability of traits because of the greater similarity of treatment of monozygotic twins. We estimated the magnitude of this treatment effect and showed it to be comparable with the presumed genetic effect.

Eaves and Eysenck make the same error in the present study. They have assumed that monozygotic twins, who look alike and are regularly confused for each other, receive the same treatment as dizygotic twins who are rarely confused[3] and they assume that the rejection of a primitive two-parameter environmental model means that no environmental model can be fitted to the data. Such procedures are inadequate. Comparisons between dizygotic twins of the same sex and dizygotic twins of the opposite sexes, for example, should be reported to see if the differences are comparable with monozygotic–dizygotic differences. If so, this would indicate that environmental effects could account for greater monozygotic similarity and indicate an absence of genetic effects. Even better would be to compare dizygotic twins who look alike[3] with monozygotic twins, to control for physical similarity.

The models used in this study and in IQ studies[4] have come to substitute for sensible experimental controls and for adequate tests of competing hypotheses. The conclusions are unwarranted and misleading. They reflect only the assumptions of the authors and assume the very results they are trying to prove.

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Specificity in initiation of mRNA translation

LODISH[1] proposes a model for the regulation of mRNA translation in which changes in specificity may actually be provoked by a nonspecific change in a kinetic parameter of protein synthesis. I had developed a similar idea earlier[2,3] in the discussion of ribosomal discrimination at the elongation level[4] whereas Lodish discusses the initiation step. The mathematics of Lodish's paper seem to be inaccurate, in addition to the acknowledged simplifications.

Equation (1) applies to infinite chains only. Equation (3) cannot be correct; this can be checked by solving the steady-state equations in a simple case (for example, a messenger three codons in length where $L=2$). Combining these two leads to equation (5), which predicts negative rates of protein synthesis when initiatable ribosomes are in large excess ($K_e/K_1R^* < 1$). Lodish does not claim that his treatment is valid for such extreme cases. The presence of the approximations casts doubt on the claim that the treatment takes into account the effect of hindrance to translocation of a ribosome as a result of the presence of an adjacent ribosome. When this effect is negligible, the correct rate equation can be established very simply. Consider an initial situation in which a ribosome has just moved from the Lth to the $(L+1)$th codon. The mRNA becomes initiatable. The average time which elapses before there is a collision with a ribosome is, by definition of the kinetic parameters of Lodish's model, $1/R^*K_1$. Completion of initiation then takes a certain time ε, and the L successive translocations take, by definition, a time L/K_e. The mRNA is now initiatable again and a new cycle begins. Thus, the average time (that is the reciprocal of the turn-over number) it takes for a mRNA molecule to produce a protein is:

$$t = 1/R^*K_1 + \varepsilon + L/K_e \qquad (1)$$

When ε is negligible, equation (1) can be written as:

$$QL/mK_e = L/tK_e = K_1/(K_1 + K_e/R^*L) \qquad (2)$$

This kinetic treatment of specificity in