situation we are faced with two alternatives; either we can say, as J. D. Frank (1961) used to do, that the placebo effect is *only one* of "a number of features common to all types of psychotherapy which probably contribute more to their efficacy than the characteristics that differentiate them"; or we can say, as Frank now does (Frank, 1983) that the placebo *is* psychotherapy. On the former view we are then faced with the problem of disentangling "placebo" from "psychotherapy" effects. In 1961 Frank attributed the effects of placebo to the "alleviation of anxiety and arousal of hope." Yet a decade later his pupil I. D. Yalom (1970) listed "instillation of hope" as one of the operative mechanisms in the process of group psychotherapy. And what of, say, suggestion? A hypnotherapist would regard this as the "specific" ingredient of his treatment, whereas a psycho-analyst or a behaviour therapist would see it as a "non-specific" or "placebo" ingredient of theirs. It is clear that the categorisation of such mechanisms into one or other pigeon-hole is purely arbitrary, and depends upon the point of view of the therapist concerned.

If, on the other hand, we adopt the latter alternative and say with Frank that the placebo *is* psychotherapy (and I agree with this) then a number of problems are solved. In the first place the need to distinguish "placebo" from "psychotherapy" is removed. Secondly, the behaviour therapist can henceforward use patients treated with general "non-specific" psychotherapy as his "placebo controls" against his supposedly specific mode of treatment. This is, of course, what Sloane *et al* (1975) did ten years ago though they did not then regard their "psychotherapy" patients as "placebo controls" against his supposedly specific mode of treatment. Thirdly, if, as seems likely, those especially efficacious factors which are common to all forms of psychotherapy are centred in the personal relationship between therapist and patient (or between person and person), then we can equate "psychotherapy effects" with "therapeutic relationship" effects. Furthermore, if "placebos" produce beneficial psychological effects (and the presumption that they do provides the need to control for them), and if, as Prioleau *et al* (1983) have recently concluded, psychotherapy effects are approximately "equivalent" to placebo effects then we are left with the approximate equation: placebo = beneficial psychological effects = psychotherapy = therapeutic relationship, and therefore placebo = therapeutic relationship.

It may be that, because "placebos" have unfortunate associations with inert pills, we tend to underestimate their value. On this view, rather than deploring the fact that psychotherapy is *no better than* placebo as Eysenck (1983) does, we should on the contrary welcome the evidence that the effects of therapeutic relationships are *at least equivalent* to those of placebos—and the former are infinitely more meaningful than inert pills. Nor need psychotherapy be unduly expensive in departments such as that at St Mary Abbots where the total psychotherapeutic effort is pooled amongst the various members of the multidisciplinary team.

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Irritability

DEAR SIR,

Snaith and Taylor (*Journal*, August 1978, **147**, 127–136) raise some very important issues as to psychiatric research on irritability. Their tentative conclusions are that "outwardly expressed irritability is an independent mood disorder and not merely one which is symptomatic of states of depression or anxiety" and that its finding in post-natal mood disorder indicates a state rather than a personality trait.

An often overlooked issue in self-rating scales is the psychometric distinction between the measurement of a trait (a long standing disposition) and distress (a temporary and changeable state) (Kellner, 1971). The responses to items in a personality inventory should be stable over time, but responses to items in a distress scale should change over time and measure changes in the clinical state of a patient (Kellner, 1971). Many scales consist of a mixture of trait and state variables. An unfortunately common example of this confusion is the Minnesota Multiphasic Personality Inventory, unreliable both in

terms of personality configuration (because of lack of stability over time) and state determination (because variability over time is not treated as a source of error) (Gleser, 1975). Also the measurement of irritability is hindered by difficulties in discriminating its trait and state dimensions. Results may largely depend on the type of inventory employed. Readily available indications come from the relationship of hostility and irritability to depression, reviewed in detail elsewhere (Fava *et al*, 1982; Fava *et al*, in press). Although the evidence as to a direct correlation between hostility and depressed mood is conflicting—and also Snaith and Taylor observed a lack of correlation—several reliable studies suggest that hostility in depressed patients decreases with improvement in mood disturbances. Interestingly, in a study (Fava *et al*, 1982) that was performed in Italy, it was found that depressives displayed more hostility yet not less friendliness than a normal control group. These data were replicated in a more recent work in the United States (Fava *et al*, in press), where hostility decreased in melancholic patients after treatment with amitriptyline to such an extent that—upon recovery—there were no significant differences between patients and controls, whereas such differences were striking during the illness. In both studies the results were obtained by using the hostility and friendliness scales of the Symptom Questionnaire (SQ), a self-rating scale of psychological distress (Kellner, in press). The hostility-irritability scale is based on factor analyses and consists of 17 items such as "angry", "feeling of hate" and "hostile", and the six item scale for friendliness was constructed from antonyms with items such as "feeling friendly" or "feeling kind to people". In several published studies the SQ was found to fulfil the psychometric requirements for a pure state or distress measurement: the hostility scale significantly decreases upon pharmacological treatment and not with placebo or upon completion of prenatal diagnostic procedures, and discriminates between patients and controls (Kellner, in press). Its test-retest correlation in normals is very high ($r = 0{\cdot}93$) (Fava *et al*, in press), indicating a high consistency of response in subjects whose state remains unchanged.

Each individual may have his own irritability threshold. Anger and hostility may be a personality trait, as discussed by Snaith and Taylor. In some cases, however, affective disturbances such as depression may lower this threshold and irritable mood may ensue. Behavioural scientists ought to be aware of the psychometric distinction between state and trait and of the limitations of their psychological instruments when studying the clinical aspects of irritability and hostility.

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Plasma Amino Acids, Downs Syndrome and Dementia

DEAR SIR,

Patients suffering from senile dementia (Alzheimer's disease), have been shown to exhibit a reduction in the ratios of both tryptophan and tyrosine to the sums of the larger neutral amino acids (Shaw *et al*, 1982) competing with them for transport across the blood brain barrier (Lajtha, 1974). This is likely to alter the relative amounts of these amino acids supplied to brain tissue, and thus to change the pattern of synthesis of proteins in the central nervous system.

Identification of patients undergoing the changes of early dementia of Alzheimer type is an uncertain exercise, and instead of this we have studied the nutritional status of individuals with Downs syndrome, a condition which is almost invariably associated with the pathological changes of Alzheimers disease (Burger & Vogel, 1973) if sometimes not the cognitive deficits (Hewitt *et al*, 1985).

There were significant differences between the plasma amino acid patterns of a group of 19 patients with Downs syndrome compared with an age and sex matched group of individuals with subnormality from other causes. Most of the latter had had brain damage at birth, and any with inborn errors of metabolism or who were receiving anticonvulsant drugs were excluded.

The findings in Down's syndrome included significantly higher plasma concentrations of cystine (1.6 ± 0.2 v 0.8 ± 0.2, mean $\pm$S.E.M., uMol.L^{-1}, $P < 0.002$), leucine (125 ± 4 v 112 ± 4, $P < 0.02$), phenylalanine (56 ± 2 v 51 ± 2, $P < 0.05$) and isoleucine (71 ± 2 v 64 ± 2, $P < 0.05$) and the ratios of leucine, of phenylalanine and of isoleucine to the