1982, 140, 174-80) that the GHQ is "unsuitable as a screening instrument for mental illness in the community" therefore deserves close scrutiny.

The major criticisms of the conclusion of Benjamin et al come under two headings. First, their study was of a small biased sample, and second, they only examined the validity of the 60 item GHQ.

The first feature, that of the biased sample, is an important one because it restricts the appropriateness of generalizing the findings of Benjamin et al. There is agreement on the need to revalidate the GHQ when used in different settings or in populations with different characteristics. So at best their conclusion has to be confined to GHQ use on women aged 40–49 who are still able to pass through a 'natural' menopause. To make any more general statement on the validity of the GHQ is bad science. Such general conclusions can only be reached from a consideration of many validation studies of the GHQ, most of which support its continuing use. Specifically, with non-consulting samples the GHQ provides a high validity research tool.

Some versions of the GHQ are demonstrably better and this differential validity is overlooked by Benjamin et al, who only consider the GHQ-60. And why "invent" a new 15 item version without assessing the merits of already validated shorter versions with their chosen sample, namely the GHQ-30, GHQ-20, GHQ-12 and GHQ-28? A recently completed study (Banks, 1983) has shown how the validity of the GHQ-30, GHQ-28 and GHQ-12 vary considerably within the same sample. In particular, attention should be drawn to the 28 item GHQ which had a sensitivity of 100 per cent, a specificity of 84.5 and overall misclassification rate of 15 per cent using a cutting score of 5/6.

It is important that clinicians and research workers receive a fair account of the GHQ, and that they understand it is composed of a family of instruments with much better psychometric, screening and validation properties than Benjamin *et al* would have us believe.

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ARE AUTISM AND ANOREXIA NERVOSA RELATED?

DEAR SIR,

I have recently come across 3 cases of males with

infantile autism who had female first-cousins with anorexia nervosa. In 2 of these cases the cousins were on the maternal side of the family. I would like to draw readers' attention to this observation and ask if any have noticed a correlation between the rare syndromes of autism and anorexia nervosa.

Two further points are worth mentioning in this context. First, there is now some evidence for a 'biochemical subgroup' of autism showing a particular chromatographic profile with regard to urinary excretion of substances giving absorbancy at 280 nm (Gillberg et al, 1982). This chromatographic pattern is now referred to as 'pattern A'. 'Pattern A' is not seen in normal children, but sometimes in childhood psychosis cases other than infantile autism. Also, it has been found in cases with anorexia nervosa (Trygstad et al, 1980). This latter point is of particular interest with regard to a hypothesis linking autism and anorexia nervosa. Second, the obsessive insistence on sameness seen in autistic children, is sometimes a striking phenomenon in anorexia nervosa too. Also, anorectic patients quite often show aloofness and problems of social relationships. Is there a possibility that a common biochemical disturbance may interact with other factors (brain damage, starvation, cultural factors) to cause autism in young boys and anorexia nervosa in prepubertal girls?

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FAMILY HISTORY STUDY OF ANOREXIA NERVOSA AND BULIMIA

DEAR SIR,

We regret to report that a number of numerical errors appeared in Table II in our recent article "Family History Study of Anorexia Nervosa and Bulimia" (*Journal*, February 1983, 142, 133–8). The corrected table is published below.

In addition, the last paragraph of the methods