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

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section should indicate the number of patients in the bipolar disorder and schizophrenia comparison groups as 40 and 46 respectively. The third sentence of that paragraph should read: "Portions of the first two of these comparison groups have been described in a previous report (Pope *et al*, 1980)."

None of these changes alters any aspects of the discussion or conclusions of this paper.

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TABLE II

Morbid risk for major affective disorder and number of probands with a family history of major affective disorder among diagnostic groups*

Diagnosis	Number of probands	Number of first-degree relatives	Cases of major affective disorder	Morbid risk for major affective disorder in relatives	Number of probands with family history of major affective disorder
Anorexia nervosa	14	70	11	28%	7
Bulimia	55	251	41	27%	30
Anorexia nervosa and bulimia	20	99	18	30%	10
Total anorexia nervosa	34	169	29	29%	17
Total bulimia	75	350	59	28%	40
Total eating disorders	89	420	70	28%	47
Bipolar disorder	40	242	22	19%	17
Schizophrenia	46	223	4	3%	4
Borderline personality disorder	15	78	1	3%	1

Significance		P**
Morbid risk for affective disorder in first-degree relatives		
Differences between subgroups of eating disorders		all NS†
Eating disorders subgroups	vs. bipolar disorder	all NS
Eating disorders subgroups	vs. schizophrenia	all <0.001
Eating disorders subgroups	vs. borderline personality disorder	all < .01
Percentage of probands with a family history of affective disorder		
Differences between subgroups of eating disorders		all NS
Eating disorders subgroups	vs. bipolar disorder	all NS
Eating disorders subgroups	vs. schizophrenia	all < .005
Anorexia nervosa	vs. borderline personality disorder	<0.03
Bulimia	vs. borderline personality disorder	<0.002
Anorexia nervosa and bulimia	vs. borderline personality disorder	<0.02
Total eating disorders	vs. borderline personality disorder	<0.001

* Major affective disorder = bipolar disorder and major depression.
** Fisher's exact test, two-tailed.
† P >0.05. Not significant.

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