Outcome of antenatal Down's syndrome screening. Values are numbers (percentages; 95% confidence intervals) unless otherwise indicated

	All women	White and other	Indian Asians
Total eligible for screening	9217	7562	1655
Uptake of serum screening	7953 (86·3)	6765 (89·5)	1188 (71·8)
Women screened positive	382 (4·8)	236 (3·5)	146 (12·3)
Relative risk			3·5 (2·9 to 4·3)
Amniocentesis uptake	179 (48)	142 (60)	37 (27)
χ_1^2			43·9 (P<0·001)
Detection rate for Down's syndrome	5/14 (36)	5/12 (42; 13 to 72)	0/2 (0; 0 to 84)
Positive predictive value	1:76 (5/382)	1:47	0:146
No of cases of Down's syndrome:			
Identified antenatally	6*	6*	0
Identified postnatally	12†	9†	3†
Rate per 1000 births	1·8‡	1·8‡	1·8

*Includes one case of Down's syndrome identified by primary amniocentesis; details available from the authors on request.
†Includes the Down's syndrome births missed by serum screening and those occurring in unscreened women.
‡After a 23% reduction in antenatally diagnosed and terminated cases to allow for the increased fetal death rate in pregnancies with Down's syndrome diagnosed by amniocentesis but not ending in a termination.[1]

the medians of the biochemical markers used in the United Kingdom are derived from largely white populations and extrapolated to other populations on the assumption that "there is no reason to believe that they cannot be generally applied."[1] Yet racial differences have been found in the age specific rates for Down's syndrome[4] and in the medians of the biochemical markers.[5]

This study suffers from the disadvantage that it is retrospective and confined to one health district. Nevertheless, it highlights the need for outcome data on serum screening to be collected by ethnic group, so that problems experienced by specific groups can be recognised and addressed. To date none of the published studies on serum screening has looked at the outcome by ethnic group. Ethnic minorities form a significant proportion of the obstetric population of some health districts, and to justify serum screening in these groups it must be based on validated risk estimations and be shown to be effective.

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Genetics versus environment in inflammatory bowel disease: results of a British twin study

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A genetic component to the cause of inflammatory bowel disease has been inferred from the increased risk among first degree relatives (5-20% cumulative incidence).[1] The only previous study of inflammatory bowel disease in twins used the Swedish twin registry and a register of hospital inpatients to identify 80 twin pairs.[2] The aim of our study was to determine the levels of concordance for inflammatory bowel disease in British twin pairs.

Subjects, methods, and results

The National Association for Colitis and Crohn's Disease is a patient support group with about 16 000 members with inflammatory bowel disease. All members were asked to complete and return a prepaid postcard if they had inflammatory bowel disease and were born as one of a twin pair. Those replying were sent a follow up questionnaire to obtain details of the member's diagnosis and twin. Zygosity was determined with a validated questionnaire.[3]

A report of inflammatory bowel disease in a proband's twin was confirmed by contacting the twin. All self reported diagnoses of inflammatory bowel disease were confirmed by contacting the patient's hospital physician or surgeon. Concordance rates for inflammatory bowel disease were compared by using Fisher's exact and Mantel-Haenszel χ^2 tests.

Completed questionnaires were obtained for 150 twin pairs in which at least one member had inflammatory bowel disease. For six twin pairs it was not possible to determine zygosity; these were excluded from analysis. The mean age of the probands was 32 (SD 13) years, the mean duration of their illness was 10 (9) years, and the mean age until which the twins had lived in the same house was 20 (3) years. In total, 15 pairs of twins were concordant for inflammatory bowel disease.

In 14 cases the reported diagnosis of inflammatory bowel disease in a proband's twin was confirmed; in one case the twin had died. The nature of the inflammatory bowel disease in concordant twin pairs was the same in all pairs. We were able to validate the diagnosis of inflammatory bowel disease in 149/159 patients (94%); in only one case was the diagnosis refuted. In four cases the diagnosis of Crohn's disease was changed to ulcerative colitis and vice versa in

Concordance for inflammatory bowel disease in twin pairs

	Identical twin		Non-identical twin	
Proband's diagnosis	**Disease**	**No disease**	**Disease**	**No disease**
Crohn's disease	5	20	3	43
Ulcerative colitis	6	32	1	33
Total	11	52	4	76

three cases. For 10 patients it was not possible to contact their doctor, their notes were lost, or their doctor did not reply to our inquiries.

Concordance rates are shown in the table. There was no difference in the levels of concordance between those with Crohn's disease and those with ulcerative colitis after zygosity was controlled for (P=0·64). Identical twins (11/63, 17%) of probands were significantly more likely to develop inflammatory bowel disease than non-identical twins (4/80, 5%); relative risk 3·49 (95% confidence interval 1·17 to 10·45; P=0·03). There was no difference between concordant and non-concordant twins in age, duration of disease, and age until which twins had lived in the same house. In concordant twins the mean period between diagnoses was five (SD 5) years.

Comment

As there is no twin registry in the United Kingdom it was necessary to obtain twin pairs by soliciting volunteers. This has the potential methodological problem known as the "rule of two thirds."[4] Two thirds of typical volunteer twin panels are monozygotic, which is the inverse of the normal ratio of two dizygotic pairs for each monozygotic pair. If the propensity to volunteer is associated with disease concordance then bias will result. In our study there was a preponderance of dizygotic twins (57%), which would suggest that this potential problem was avoided to a considerable degree; there was a similar proportion in the Swedish study (57·5%).[2]

In most cases zygosity in this study was determined from answers from one member of a twin pair; in all 14 twin pairs, when answers were obtained from both members the replies concurred with regard to zygosity. The error rate in determining zygosity has been shown to be similar whether one or both twins respond to the questions posed.[5]

In summary, identical twins are significantly more likely to be concordant for inflammatory bowel disease than non-identical twins. Nevertheless, even in identical twins the concordance was only 17%, which suggests that non-genetic—that is, environmental—causes are more important in the development of Crohn's disease and ulcerative colitis. This register of British twins with inflammatory bowel disease will be a valuable resource for future research.

We thank all those members of the National Association for Colitis and Crohn's Disease and their twins who participated in this study.

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Correction

Estimating sample sizes for binary, ordered categorical, and continuous outcomes in two group comparisons

Editorial errors occurred in this paper by Campbell *et al* (28 October, p 1145). In formula 5 the denominator should have read: $(\log OR)^2\bar{p}(1-\bar{p})$ [not $\log (OR)^2$]. On p 1147 in the second column, in the worked example for ordered categorical data, the formula should read: $C_{A1}+OR(1-C_{A1})$ [not $C_{a1}+OR(1-C_{a1})$]. Also, all logarithms are natural logarithms.

THE PERSON WHO MOST INFLUENCED ME

Miss Reynolds, biology mistress extraordinaire

Having chosen biology as my main sixth form subject, I looked forward with some trepidation to being taught by Miss Reynolds, the senior biology mistress, who had a reputation for expecting high standards and not tolerating any nonsense. But I knew that she was a good teacher; how good I was soon to find out.

I had little idea what career I wanted to follow. Indeed, I think most of us at that time looked forward only to continuing the educative process in some rather vague and comforting way. Whatever talents I had were fairly equally distributed between the arts and sciences, so perhaps it was not surprising that the choice was made for me by my remarkable biology mistress. Not that she dictated a career; the choice seemed to become obvious and inevitable.

Miss Reynolds had some quite outstanding gifts. She had gained a first class honours degree in biology, taken a PhD, and could easily have had a university career, but fortunately for us had chosen to teach at secondary level. Her enthusiasm for biology was manifest in every aspect of her teaching. Her course notes balanced the didactic with the thought provoking. Her planning of lessons was meticulous, so that for both O and A levels we were able to complete large curriculums.

Miss Reynolds never pushed her subject at us, but by her demeanour there seemed to be no other topic during those long, summer afternoons. Although a stickler for the use of scientific language appropriate to sixth formers, she was not without a sense of the ridiculous. During one question and answer session on the skin she asked the class to list its functions. One rather incredulous boy blurted out "Well, it keeps the meat in. . . ." Whereupon in her italic writing, she wrote "No 1. Keeps the meat in."

Miss Reynolds was an outstanding technical artist. She was one of those rare individuals who could draw a perfect circle with two confident sweeps of the arm. She would come in early before each lesson and produce minor works of art ranging from amoeba to liver cells. Such was our reverence for these drawings that they would frequently remain on the board for days until with a shrug of the shoulders and a slight laugh she would remove them.

I have had many wistful thoughts of Miss Reynolds since my sixth form days. Her influence on young scientific minds within my year alone was manifest by the high number of A and B grades achieved; she was and will remain one of the unsung heroes and heroines of a sometimes beleaguered but noble profession.—N S BABER *is a director of clinical pharmacology at Glaxo*