Number of cases of tuberculosis and annual incidence by ethnic origin and area of residence, Newcastle upon Tyne, 1990-4

	White population		Ethnic minorities		All cases	
	No of cases	Annual incidence/100 000	No of cases	Annual incidence/100 000	No of cases	Annual incidence/100 000
Inner city	46	13·8	26	132·8	72	20·4
Outer city	43	4·7	30	90·9	73	7·7
Whole city	89	7·1	56	106·5	145	11·2

ing of the possible reasons behind this increase in incidence, case finding and effective treatment are unlikely to reverse the trend. We therefore welcome the contribution by N Bhatti and colleagues for two reasons.[1] Firstly, they show that the increase in tuberculosis is not due to an improvement in the notification system but reflects a true increase in the disease. Secondly, they produce evidence challenging the suggestion that the increase in tuberculosis is due to imported cases among immigrants and refugees; they believe that it is more likely to be the result of changes in the socioeconomic conditions of the inner cities.

In Newcastle upon Tyne, from 1990 to 1994, 145 cases of previously undiagnosed tuberculosis in city residents were identified from the notification registers and laboratory and hospital records. The table shows the number of cases and the annual incidence by ethnic origin and by residence in the eight deprived wards of the inner city and elsewhere. The relative risk of tuberculosis for ethnic minorities living in the inner city compared with the outer city is 1·5 (95% confidence interval 0·9 to 2·5, P=0·15). For the white population the relative risk is 2·9 (1·9 to 4·4, P<0·001). The Mantel-Haenszel procedure combines the evidence from both subsets. When the confounding influence of ethnic origin is taken into account the weighted relative risk is 2·2 (1·6 to 3·0).

These figures show that in Newcastle there is a significant association between the incidence of tuberculosis and residence in deprived inner city wards, irrespective of ethnic origin. Although the incidence of tuberculosis in the ethnic minority community is 15 times that in the white population, the association with socioeconomic deprivation at ward level is much stronger in the white population.

Bhatti and colleagues conclude that a reversal in the increasing incidence of tuberculosis is unlikely without an improvement in income for some sectors of the population. Although we do not disagree with this opinion, the more practical response is to target measures to control tuberculosis at high risk populations. Until now such action has been limited to recent immigrants and close relatives of people with active disease. Should we consider screening programmes in deprived inner city areas?

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Risk factors for diabetes in men

Risk factors are closely linked with socioeconomic status

EDITOR,—The association between risk factors and diabetes described by Eric B Rimm and colleagues[1] is unlikely to be simple, and both smoking and obesity are linked to socioeconomic status. Our studies on diabetic subjects in Glasgow[2] and Middlesbrough[3] have shown that low socioeconomic status is associated with a high prevalence of smoking and higher body mass indices. The increased smoking and obesity in low socioeconomic groups remain unexplained, although intrauterine, genetic, and psychological factors have been proposed. Ivan J Perry and colleagues include socioeconomic status in their study by including manual occupation as a variable, but this alone did not predict diabetes.[4]

We suggest that measures of socioeconomic status based on areas[5,6] could be used, combined with measures of smoking, obesity, and physical activity. We are performing a study to investigate these factors. It may then be possible to identify high risk areas and populations in which screening for diabetes will be beneficial. Because of the suggestion that diabetes is undiagnosed in up to 1% of the population and the difficulties associated with screening, perhaps resources should be focused on areas of low socioeconomic status in an effort to uncover the hidden burden of diabetes.

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Japanese study confirms findings

EDITOR,—Eric B Rimm and colleagues' findings[1] are in line with those of a large cohort study conducted in Japan. The lifestyles of 265 118 adults aged 40 and over (122 261 men; 142 857 women) in six prefectures (Miyagi, Aichi, Osaka, Hyogo, Okayama, and Kagoshima) were studied. Household interviews were conducted by public health nurses in 1965, and the subjects were followed up for 17 years.[2] Diabetes mellitus was recorded in the death certificates of 1678 subjects (846 men, 832 women)—as the main cause of death in 946 (454 men and 492 women) and as a contributory cause of death in 732 (392 men and 340 women).

A significant dose-response relation was observed in men between the number of cigarettes smoked daily and the relative risk of diabetes, adjusted for age, the risk for non-smokers, and for those who smoked 1-9, 10-19, 20-29, 30-39, and >40 cigarettes daily being 1·00, 0·69, 1·16, 1·14, 1·81, and 2·70 respectively (χ^2 for trend=11·9, P=0·0004). A similar trend was observed in women, the relative risk adjusted for age in non-smokers and those who smoked 1-9, 10-19, and ≥20 cigarettes daily being 1·00, 1·03, 1·26, and 1·39 respectively. In men, diabetes mellitus was the fifth ranking disease related to smoking (except for cancer) in terms of risk for those smoking ≥40 cigarettes daily, after arterial embolism and thrombosis (relative risk 19·2), aortic aneurysm (relative risk 3·9), gastric ulcer (relative risk 3·6), and emphysema (relative risk 3·2).

These results are in line with those of other large cohort or cross sectional studies reported.[3-5] There are also negative reports in the literature, but apparently most of these studied did not consider that the risk might vary with the number of cigarettes smoked. As shown above, a slightly lower risk of diabetes was found among light smokers in the Japanese study; this was probably due to the anti-obesity effect of light smoking, as shown by the national nutrition survey in Japan in 22 019 men in 1986-92: the proportion of men who were obese (≥40 mm subcutaneous fat) was 14·9%, 11·3%, 9·7%, 12·2%, 14·3%, and 17·1% in non-smokers and those who smoked 1-9, 10-19, 20-29, 30-39, and ≥40 cigarettes daily respectively.

Thus people should be strongly advised to stop smoking to prevent the occurrence as well as the progression of diabetes mellitus.

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Breast feeding of adopted infants

EDITOR,—Tony Waterston gives advice on how to induce lactation when a woman wishes to breast feed a baby adopted from birth.[1] I am surprised that the only pharmacological method mentioned is stimulation of the let down reflex with oxytocin nasal spray. Although the exact method of inducing lactation in the study that Waterston quotes was not specified, at least 54% of the nursing mothers had to give supplementary feeds.[2] This may well be adequate in the industrialised world, where good hygiene and a good nutritional quality of artificial feeds are likely. For developing countries, however, successful lactation without the need for supplementation is crucial for the survival of the adopted infant. There, drugs that cause hyperprolactinaemia due to antidopaminergic effects, such as metoclopramide and phenothiazines, can be used to maximise the volume of milk produced.[3]

In Papua New Guinea the following method is advocated.[4] If the woman is nulliparous or has never lactated the breast tissue is primed with ethinyloestradiol 50 μg three times a day for one week or (more simply) depot medroxyprogesterone acetate 100 mg intramuscularly once. Women who have lactated in the past or already have some milk omit this phase and start immediately to take metoclopramide 10 mg four times a day or chlorpromazine 25 mg four times a day, or both. On the rare occasions that this does not produce satisfactory results within two weeks methyldopa 125 mg four times a day is added. Supplementary feeds are rarely given and not encouraged. Frequent suckling is advocated. During the interim period until lactation is adequate the child may be

additionally breast fed by a lactating relative or the child's natural mother.

In my experience in Papua New Guinea this method worked well in all cases in which the adoptive mother was motivated and received the necessary support from her family. Side effects of the drugs used were rarely complained of, and only once was it necessary to add methyldopa in the sequence as described above.

Clearly, in industrialised countries, where induction of lactation is not considered to be essential for the survival of the infant, the relative importance of side effects and concerns regarding excretion of the drugs into the milk are greater. Nevertheless, at least some of the aspects of the above method could well be adopted in industrialised countries.

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Author's reply

EDITOR,—Padraig Kramer's recommendations on the use of oestrogens, metoclopramide, and chlorpromazine to induce lactation are helpful, and I too have used chlorpromazine for this purpose, in Africa. Opinion seems to be divided over the drugs' value in Western countries, and drugs other than nasal oxytocin (not now available in Britain) are little used in Britain, though they are used in the United States. All the drugs mentioned boost prolactin concentrations but are less effective in increasing the output of milk and may be of mainly psychological benefit. Other methods seem to be equally effective in helping relactation and have fewer side effects. Documented trials, however, are scanty. The drugs mentioned are not licensed for use in inducing lactation in Britain.

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Accuracy of local data on skin cancer

EDITOR,—The paper by Clive Richards and colleagues on underregistration of melanoma in the South Western Cancer Registry[1] was published alongside our paper reporting trends in registrations of melanoma in the Oxford registry.[2] This naturally raised doubts in the mind of some readers about the validity of our findings. In particular, Dafydd L Roberts questions whether an artefact due to underreporting could have exaggerated the apparent decline in melanoma in women from 1988 to 1992.[3]

Apart from the fact that sex specific artefact is hard to explain, we can respond to Roberts, and other similarly sceptical readers, on several counts. Throughout the period of our study the Oxford Cancer Registry routinely received reports from pathology laboratories, except for one of the eight districts. The paper by Richards and colleagues shows the value of using pathology records to identify melanomas, if indeed this has ever been in doubt. From 1992, the reporting process in this region has been largely automated by use of systematised nomenclature of medicine (SNOMED) codes to identify potentially registrable cancers.[4] Thus a process similar to that used by Richards and colleagues to validate the South Western statistics has already been adopted for routine registration.

With respect to our study of melanomas, there were specific concerns about completeness in only two districts. Staff of the cancer registry therefore visited the relevant laboratories and reviewed all pathology reports for the periods in question, looking for melanomas. This laborious process added to the number of registrations of melanoma in those districts by about 10% but did not alter the secular trends. As a further test of completeness we obtained a list of patients with melanoma diagnosed in a single year (1992) from a comprehensive diagnostic register held by a dermatology department in one of the districts. Only two confirmed cases (5%) were not already registered, both in private patients (one male and one female).

It would have been helpful if Richards and colleagues had pointed out in their paper that the South Western registry was one of only three registries in England that were not using pathology reports routinely for registration in 1989.[5] Clearly, it would be wrong to generalise from their study to those cancer registries that do use such reports.

We are confident that the trend in incidence in women in Oxford is not an artefact, and a similar trend has now been reported by another registry (North Western). Why this trend occurred and whether it will be sustained remain open questions.

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Targets for glycated haemoglobin

Glycated haemoglobin targets are attainable

EDITOR,—Chris Butler and colleagues criticise the targets set for glycated haemoglobin concentration by various organisations and guidelines on the grounds that they are not based on normative data and seem to be largely unattainable in clinical practice.[1] The authors are incorrect on both counts.

The targets set by the British Diabetic Association and St Vincent Declaration have a common history, which can be most easily traced to the consensus guidelines of the European Non-Insulin Dependent Diabetes Mellitus Policy Group published in 1988.[2] Those guidelines, adopted by a consensus meeting of over 200 European doctors, were compared by the policy group's chairmen with local results. At the time (1988), using an assay of haemoglobin A_1 (normal concentration $<7{\cdot}5\%$), the Newcastle service was achieving results in the "good" or "acceptable" range ($<8{\cdot}8\%$) in 64% of people with non-insulin dependent diabetes and in 44% of those with insulin dependent diabetes. Results achieved in primary health care in Newcastle (non-insulin dependent diabetes: 62%) were similar.[3] Futhermore, in a progressive disorder such as non-insuin dependent diabetes the figures for satisfactory control will always be diluted by patients approaching the next step of intervention treatment.

Nevertheless, the group with "poor" control was subsequently split into two ("poor" and "very poor") by the joint audit working party of the Royal College of Physicians and British Diabetic Association, so that analyses of population distributions could be more informative.[4] In the working party's paper data from a hospital in southern England were reasonably evenly distributed among the four groups.

In 1993, under the auspices of the St Vincent Declaration initiative, the guidelines on non-insulin dependent diabetes were revised, and the new European Insulin Dependent Diabetes Mellitus Policy Group liaised with the initiative to produce identical cut off values.[5] At that time the categories were again reduced to three and the cut off values raised slightly, partly in recognition of the problem that assays of haemoglobin A_{1c} gave different (higher) distributions of patients for glycated haemoglobin than assays of haemoglobin A_1.

More importantly, even before the results of the diabetes control and complications trial were available it was evident that the relation between glycated haemoglobin and microvascular complications steepened considerably at a haemoglobin A_{1c} concentration of around $7{\cdot}5\%$.[6,7] With this assay (normal concentration $<6{\cdot}1\%$) 65% of people with non-insulin dependent diabetes and 39% of those with insulin dependent diabetes achieve satisfactory blood glucose control (good plus borderline control) in our service. Without such targets some of these people would be denied freedom from the risk of microvascular complications and the many with concentrations just above the cut off value would face much higher risks.

The guidelines accept that target concentrations are not achievable by everyone and emphasise that therapeutic targets must be adjusted for individual people in accordance with other factors, such as personal experience of risk of hypoglycaemia. At present, review of any person with diabetes with a haemoglobin A_{1c} concentration over $7{\cdot}5\%$ requires consideration of whether blood glucose control can be optimised further.

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Targets set reference points for clinic comparisons

EDITOR,—Chris Butler and colleagues show impressively how systematic collection and use of case based information can help identify problems in the search for improved quality of care in diabetes.[1] Pointing to the difficulties with setting targets in the treatment of diabetes and in its care, they argue that targets for haemoglobin A_1 concentration may be set more on an idealistic