be developed. Carr-Hill *et al* suggest that their findings could also be used to develop a formula to help health commissions allocate resources to general practices, but such a formula would be difficult to implement.

Firstly, the populations served by general practices (typically 2000-20 000 people) are much smaller than those of health commissions (typically around 500 000 people). Consequently, small differences in the distribution of patients with a high demand for care can have a dramatic effect on the workload of general practitioners.

Secondly, the only socioeconomic variables routinely available for general practices are proxy variables derived by linking patients' postcodes with census data,[3] and we do not know if these derived variables are accurate enough to be used to help allocate resources to general practices.[4]

Thirdly, the data in the morbidity survey have limitations (for example, the practices that participated were not a random sample), and these limitations need to be borne in mind when trying to generalise the findings to other general practices.[5] Hence, although this study may help to pave the way towards a more rational method of funding primary medical care services at health commission level, more work still needs to be done to develop fairer methods of funding primary care services at general practice level.

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Does the variation in the socioeconomic characteristics of an area affect mortality?

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See editorial, pp 999, 1004

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BMJ 1996;312:1013-4

Our research in England has shown that the more deprived an area the greater its incidence of premature mortality.[1] Wilkinson has argued that in the developed world income distribution is a more important predictor of life expectancy between countries than simply mean income.[2] We aimed to determine whether the risk of mortality in a geographical area was related to the degree of socioeconomic variation within that area as well as the average level of deprivation.

Methods and results

For each of the 8464 wards in England we obtained the Townsend deprivation index from the 1981 census[1] and directly standardised all cause mortality for 1981-5. Mortality under the age of 65 was used as an indicator of premature mortality. Male and female mortality rates were averaged for each ward. Twenty four wards were excluded because we could not compute the mortality rate, and two local authorities were excluded because each contained only one ward. The remaining 369 local authorities contained an average of 23 wards (6-47).

For each local authority we computed the median of the ward Townsend scores as a measure of overall deprivation and their interquartile range as a measure of variation in deprivation (correlation between the two measures 0·33). We also computed the average of the ward mortality rates. The local authorities were divided according to their quartile of deprivation and variation and the mean mortality for each group computed. We then constructed models in which mortality was regressed on quartile of variation within each quartile of deprivation. Because deprivation still varied between wards within a quartile of deprivation "fully adjusted" analyses also controlled for deprivation as a continuous variable in each model.

Mortality was strongly positively associated with average deprivation (table). The trend for mortality was 26 per 100 000 per quartile of deprivation (95% confidence interval 23 to 28, $P<0·001$). Mortality was also positively associated with variation: the average fully adjusted trend was 7 per 100 000 per quartile of variation (4 to 9, $P<0·001$). Although this effect appeared to be stronger in the middle quartiles of deprivation, the trends did not differ significantly ($P=0·09$ for heterogeneity).

Results were similar using mean and standard deviation, all age mortality, and male and female mortality separately; after ward mortality had been transformed by taking either the square root or the square; and after we had adjusted for the number of wards both as a continuous and a quartile variable.

Comment

Our results confirm a strong gradient in mortality related to deprivation, together with a positive association between degree of variation within an area and increased mortality ($P<0·001$). These results support

Mean mortality of local authorities by average deprivation and variation of deprivation. Values are mean mortality per 100 000 (and number of local authorities) for each quartile of variation and deprivation and trend in mortality

	Mortality				Trend in mortality per 100 000 per quartile of variation (95% confidence interval)	
	Least variable quartile (1·03-2·14)†	Second quartile (2·15-3·01)	Third quartile (3·03-4·13)	Most variable quartile (4·15-9·55)	Simple	Fully adjusted‡
Most affluent quartile (−4·87 to −2·00)*	249 (30)	254 (36)	253 (19)	261 (7)	3 (−3 to 9)	2 (−3 to 8)
Second quartile (−1·98 to −0·71)	256 (34)	273 (26)	270 (23)	298 (10)	11 (6 to 16)	11 (6 to 15)
Third quartile (−0·69 to 1·41)	272 (19)	278 (15)	292 (22)	302 (37)	10 (6 to 15)	8 (4 to 12)
Most deprived quartile (1·48 to 9·21)	358 (9)	330 (15)	334 (29)	342 (38)	−1 (−6 to 4)	4 (−1 to 9)

*Median value of Townsend deprivation score.
†Interquartile range of Townsend deprivation score.
‡Adjusted for residual differences in deprivation scores.

the hypothesis that variations in income contribute an additional effect on mortality over the effect of deprivation alone.

This analysis cannot show which wards in an area of greater inequality suffer higher mortality: all might, or only the most deprived. Alternatively, increased mortality for poor wards might not be balanced by decreased mortality for rich wards in the same area—that is, the relation may not be linear, but analyses on transformed data did not alter the effect of variation on mortality.

The association between variation and mortality appears to be least in the most affluent and most deprived areas, although the result of a heterogeneity test was not significant. These findings deserve further investigation as some evidence exists that community solidarity may have a beneficial effect on all residents.[3]

Studies have produced contradictory results on whether area characteristics have a truly independent effect on mortality.[4,5] Although our analysis is based on areas, not individuals, it suggests that the characteristics of individuals are insufficient to account fully for differences between areas, as individuals in more variable areas appear to have worse mortality than their counterparts in more homogeneous areas.

Funding: None.
Conflict of interest: None.

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(Accepted 23 November 1995)

Effect of inadvertent intradermal administration of high dose percutaneous BCG vaccine

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BMJ 1996;312:1014

In the United Kingdom it is recommended that all children receive BCG vaccination to prevent development of tuberculosis.[1,2] The standard route of administration is intradermal. In neonates a 10 times more potent percutaneous preparation is used, a small volume being injected to a depth of 2 mm with a modified Heaf gun. The two preparations have similar packaging and labelling and so may easily be confused.[3] We report the outcome of inadvertent intradermal administration of the stronger percutaneous product and we describe the relation between the development of skin lesions and the dose of vaccine.

Subjects, methods, and results

Thirty two school children aged 11-14 received 0·1 ml of BCG vaccine intradermally at a routine school immunisation session. Nineteen received the preparation for percutaneous administration, which contains 50-250 million colony forming units per ml. Thirteen received the correct preparation, which contains 8-26 million colony forming units per ml (Evans Medical, Leatherhead, England). The error occurred because of the similar packaging and labelling of the two products. All parents and children were informed of the error and all agreed to a period of close observation. All observations were performed by the same observer (MMM). The study was not blind since a component of the consultation was to discuss any concerns with children and parents.

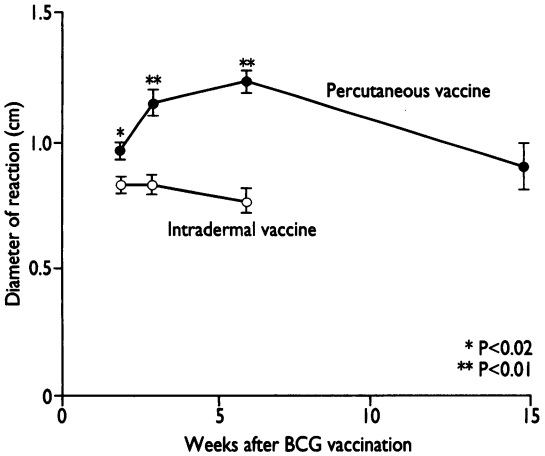

Fig 1—*Mean (SE) diameters of skin lesions after intradermal BCG vaccine and percutaneous vaccine given intradermally*

The area of induration increased progressively and was significantly greater in the group who had received the percutaneous vaccine intradermally (fig 1). Mean diameters were 0·96 cm (range 0·8-1·3, $P<0·02$ by Student's t test) at two weeks, 1·15 cm (0·9-1·5, $P<0·01$) at three weeks, and 1·23 cm (1·0-1·6, $P<0·01$) at six weeks; the mean diameter had decreased to 0·91 (0·6-2·2) cm by 15 weeks. By contrast, the mean diameter of the induration in those receiving the correct intradermal vaccine was 0·83 cm (0·7-0·9) at two weeks, 0·83 cm (0·6-1·1) at three weeks, and 0·77 cm (0·5-1·1) at six weeks.

The sequence of symptoms was itchiness, soreness, and discharge. These were similar in both groups, although discharge was more common at six weeks in the group inadvertently given the percutaneous vaccine (nine out of 15 children *v* one out of 10 children). Ten of the 15 children given the percutaneous vaccine were examined at 28 weeks. They complained of no symptoms, and mature scars had formed (mean diameter 1·2 cm (range 0·8-2·0)).

Comment

This study emphasises the requirement for clear packaging and labelling of drugs. If subjects inadvertently receive the percutaneous BCG vaccine intradermally they may, however, be reassured that the lesion will resolve over a few months without treatment. Although the lesions were sometimes painful, the induration was larger and discharge more common after six weeks when the high dose preparation was given intradermally. The lesions had started to resolve at 15 weeks.

The results also suggest a clear dose-response relation in vivo between the size of the inflammatory response and the dose of vaccine. We do not know whether there is a relation between the size of the inflammatory response and the protective effect of BCG or the size of the scar.

Funding: Parkside Health (Community Trust).
Conflict of interest: None.

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(Accepted 1 November 1995)