a percentage of all low income households (n = 704 066). Cumulative proportions for the total number of sectors and the total population contained within those sectors were also calculated.

On the basis of these results, if 20% of the most deprived sectors in Scotland (1 205 833/4 998 202 (24%) of the population) were targeted, 41% of unemployed people and 34% of low income households would be “captured” (figure). By targeting 254 postcode sectors (1 501 569 (30%) of the population), resources could be directed to 48% of unemployed people and 40% of low income households. If 55% of the postcode sectors are targeted (62% of the population), 80% of unemployed people and 74% of low income households are captured, but even then, 20% and 26%, respectively, are excluded. Modest improvements in capture rates (2-6%) were achieved when the analysis was repeated using census enumeration districts (data not shown).

Comment
This analysis reaffirms Townsend’s argument that the selective targeting of resources on an area basis would miss more deprived people than it would include. On the basis of Carstairs scores, more than 60% of the population in Scotland would need to be targeted to include 74% of low income households. The poor sensitivity of an area based approach means that the group of people to whom resources are directed includes people who are not poor. There are higher concentrations of poverty in some areas; however, the current increase in area based initiatives ignores the wide spatial distribution of deprived people. Only a small proportion of government spending is directed towards area initiatives, but their high profile implies that deprivation is a problem only within certain areas. However, deprived areas can include people who are not deprived and vice versa. Debate continues about whether the health experience of poor people in deprived areas is worse than that encountered by other poor people.5 Targeting deprived areas may have merits, but a greater emphasis on national strategies is the key to dealing with poverty and improving the health of the population.

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a clinic at the Northern General Hospital, Sheffield, and 392 (95% of those interviewed) attended, where stereoscopic photos of both fundi were taken. Photographs were graded by one observer (NFH), who was unaware of the participants’ drug history, against standard images using the Wisconsin age related maculopathy grading system. We excluded 12 participants who had non-age related degenerative macular changes and one participant who was taking part in a trial of statins. The analyses that follow are therefore based on 379 participants.

Of the 379 subjects, 27 (7%) reported taking statins and 77 (20%) had some evidence of macular degeneration. Age related macular degeneration was more common among the participants who did not take statins (see table 1: 76/352 (22%) of participants who did not take statins showed signs of macular degeneration, compared with only 1/27 (4%) of participants taking statins (P=0.02, Fisher’s exact test). This is equivalent to an odds ratio for macular degeneration among participants who took statins of 0.14 (95% confidence interval 0.02 to 0.83) compared with those who did not.

A history of coronary artery bypass grafting or angioplasty was associated with macular degeneration. Eight of the 77 participants with macular degeneration (10%) had undergone coronary angioplasty or bypass grafting compared with 13 of the 302 participants (4%) without macular degeneration (P=0.05, Fisher’s exact test). Not surprisingly, people who had undergone coronary angioplasty or bypass grafting were more likely to have taken statins than those who had not (6/22 (27%) compared with 21/389 (5%) respectively). In a logistic regression model—after adjustment for age, sex, smoking, and history of coronary angioplasty or bypass grafting—the odds ratio for macular degeneration (early or late) among participants taking statins was 0.09 (0.01 to 0.79) compared with those who did not take the drug.

Comment

In this survey of men and women aged 66-75 those who took statins had an elevenfold the risk of age related macular degeneration (after adjustment for coronary artery disease and smoking) compared with those not taking the drug. The confidence intervals are wide, however, giving an imprecise estimate of the reduced risk. Bias could lead to this association if people with macular degeneration and taking statins were less likely to participate, or people without macular degeneration and not taking statins were more likely to participate, but this seems unlikely.

We suggest three mechanisms that could link statin use with lower risk of macular degeneration. Firstly, statins might prevent the accumulation of basal linear deposit in Bruch’s membrane, which occurs with higher concentrations of plasma cholesterol. Secondly, antioxidant properties of statins might protect the outer retina from oxidative damage. Thirdly, simvastatin inhibits endothelial cell apoptosis and preserves ischaemic vasculature, perhaps maintaining a competent vascular supply to the macula.

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