a long term decline or increase in the disability incidence rates. Over the 20th century, French male mortality rates declined substantially, and we would argue that it is quite implausible to assume that the mortality rates of disabled men would not have declined along with those of non-disabled men. In the French male population, life expectancy has not remained unchanged, and it is not reasonable to expect outflows from the disabled state to remain unchanged. We made the arbitrary assumption that the relative risk of death for disabled versus non-disabled people remained constant at 3.66 in the published simulations. We also carried out simulations where the relative risk ranged from 1 to 10 and also varied with age. We found that the results were not highly sensitive to the value assumed and did not affect our conclusions.

In response to criticism of the constant relative risk assumption, we have carried out additional simulations in which we assume that the ratio of the mortality risk for disabled male to the average mortality risk for all males remains constant at its value in the year 1945, and so is independent of changes in disability prevalence. This had very little effect on the estimated health expectancies under any of the scenarios and confirms our published comment that the results are not highly sensitive to assumptions about the mortality rates for disabled people.

Figure 1 Trends in mortality from all causes and ischaemic heart disease, males and females, The Netherlands, 1952-94.

We intended no implication as to the plausibility of the scenario of Barendregt *et al* by describing it as a "hypothetical example"; that was the description they themselves used.² We have no disagreement with their scenario or its results, but repeat that such a simulation is not relevant to the question of how adequate Sullivan's method is for monitoring at the whole of population level. Thrombolytic therapy may well have been introduced in a three year period in The Netherlands, and may well have had a major effect on survival rates and a detectable effect on cardiovascular mortality at population level. Despite this, in-hospital or post-hospital mortality after myocardial infarction does not account for most cardiovascular deaths (in Australia around 80% of myocardial infarction deaths occur outside hospital).

Figure 1 shows long term trends in mortality rates for The Netherlands for all causes mortality and ischaemic heart disease mortality.³ Rates are age standardised using the European standard population and five year averages are shown prior to 1985. It is clear that the impact of thrombolytic therapy has not caused any sudden change in all cause mortality rates (the relevant rates for use with Sullivan's method) at the population level,

and that Sullivan's method would be entirely adequate for monitoring long term trends in Dutch health expectancies. Very few changes in treatment practice would result in such dramatic changes in transition rates as the "hypothetical example" of Barendregt *et al*, and it is very unlikely that sudden changes in all causes mortality or disability transition rates at the population level will result from new medical interventions.

Finally, to the question of monitoring compression or expansion of morbidity. The example in the letter above is based on the assumption that compression ceases, allowing the disability prevalence in the population to reach its equilibrium value. We have no disagreement with the conclusion that in such a case the Sullivan's method would give a spurious compression—it is another example of the limitation of Sullivan's method when there are sudden changes in transition rates. In a more realistic example, where disability incidence and prevalence evolve smoothly, Sullivan's method will give a reasonably good indication of whether compression or expansion is occurring. In our published scenarios, Sullivan's method provides quite accurate estimates of the degree to which compression or expansion is occurring.

In conclusion, we do not believe we are in disagreement over the usefulness and limitations of Sullivan's method or that the example of Barendregt *et al* contradicts our conclusion that when population health is evolving reasonably smoothly, Sullivan's method is acceptable.

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Smoking and Alzheimer's disease: an alternative hypothesis

Sir—Many studies have been carried out identifying risk and protective factors for Alzheimer's disease, often with conflicting results. This particularly applies to smoking. While some, such as the case-control studies from the MRC elderly hypertensive trial¹ and the Canadian study of health and aging,² have shown no significant effect of smoking, others, including studies in The Netherlands³ and America,⁴ have reported that the prevalence of Alzheimer's disease was significantly lower in smokers than non-smokers. It has been suggested that the mode of action might be via nicotine.⁵ Paradoxically, Brenner *et al*⁶ also found that the protective effect was most marked at low dose exposure to cigarettes, but disappeared in those with the highest pack years exposure. van Duijn and Hofman, however, found an inverse relation between the number of cigarettes smoked and the reduction in Alzheimer's disease cases. The Canadian study² also confirmed the findings of other workers that the odds ratios for suf-

fering from Alzheimer's disease were lower in subjects with a history of arthritis or of non-steroidal anti-inflammatory (NSAIDs) drug use.

Could these observations be linked? In a paper published in the *European Journal of Public Health* in September,⁷ we reported findings based on 5319 respondents in the nationwide health and lifestyle follow-up survey of 1991-92. This showed that women under 55 with a smoking history were more likely to report having suffered from arthritis/rheumatism (OR 1.88 (95% CI 1.39, 2.54)) as were men aged under 65 who were current smokers (1.49 (1.0, 2.22)). Furthermore, smoking was found to be associated with currently taking NSAIDs or drugs prescribed for analgesia (some of which have anti-inflammatory properties). Analysis by logistic regression (age adjusted) of those aged 25-80 showed that men who were current smokers (2.11 (1.07, 4.17)) and women with a smoking history (1.69 (1.11, 2.57)) were more likely to be taking NSAIDs, and all smoking groups were significantly more likely than lifetime non-smokers to be taking NSAIDs and/or analgesics. There was also a dose effect, with those smoking 15 or more cigarettes a day the most likely to be taking these medications.

We hypothesise that the links between both arthritis and smoking, and NSAID/analgesic medication and smoking, suggest that the apparent protective effect of smoking against Alzheimer's disease could be due in part to the anti-inflammatory effects of these drugs. Those with a smoking history were more likely than those who had never smoked regularly to be taking these medications.

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The evaluation of agreement on continuous variables by the intraclass correlation coefficient

Sir—You published an interesting paper by Poulter *et al*¹ on the reliability of data derived from proxy respondents in an international case-control study of cardiovascular disease and oral contraceptives. The agreement between the information obtained from "true" controls and their proxy respondents was evaluated thoroughly. Categorical data were evaluated using Kappa statistics. Comparisons between continuous variables were performed by means of the intraclass correlation coefficient (ICC). Nevertheless, detailed