LETTERS TO THE EDITOR

How good is Sullivan’s method for monitoring changes in population health expectancies?

Sir—In an interesting paper Colin Mathers and Jean-Marie Robine ask whether Sullivan’s method is capable of monitoring changes in health expectancies.1 Sullivan’s method combines cross-sectional prevalence of, for example, disability with period mortality figures to calculate a disability-free life expectancy. As several articles have pointed out, the use of cross-sectional prevalence poses a problem because prevalence, as a stock variable, reacts only slowly to changes in the flow variables incidence, recovery, and survival that govern it.2 The combination of life expectancy (calculated with the flow variable mortality) and the stock variable prevalence (to calculate disabled years) may therefore result in spurious trends in the health expectancy indicator.3

In an elaborate simulation experiment, Mathers and Robine compare results from simulations with multi-state life table-based estimates of disability-free life expectancy with that of Sullivan’s method. They formulate a “number of simple but reasonably realistic scenarios” to determine the circumstances under which the much less demanding Sullivan’s method provides reasonable estimates, and conclude that it “seems to be a quite acceptable method for monitoring relatively smooth long term trends in health expectancies at the population level in non-stationary populations”.

In this letter we argue that: 1. their approach ignores the aspect which caused most trouble in a simulation we undertook; 2. the example of the improvement in survival which we used, is anything but “hypothetical”, and 3. the question of “compression and expansion of morbidity”, a main application for trends in health expectancy, requires the estimation of changes in life expectancy with disability, which is much more sensitive to the Sullivan problem than the disability free life expectancy Mathers and Robine look at.

Firstly, these authors’ scenarios are not nearly as simple as they may seem. The scenarios change the incidence of disability, while keeping recovery rates constant. Naturally, this changes the prevalence of disability, and since the disabled in the simulations have a relative risk of 3.66 of dying, this would change mortality and life expectancy too, if all else remains equal. In order to keep the estimated life expectancies at the observed values, Mathers and Robine use two equations (they would be numbered “3” and “4”, if the text were a paper) to divide total mortality between disabled and non-disabled. But, as they observe, this makes mortality of disabled and non-disabled a function of disability prevalence, and hence, when in the scenarios disability incidence is manipulated, these mortalities change too. So outflow from the disabled state is not constant, as the constant recovery rates would suggest.

Keeping the life expectancy unchanged, no matter what happens with disability prevalence, artificially removes one source of problem with the Sullivan method. We have published the results of a simulation where survival after myocardial infarction improves as a consequence of the adoption of thrombolysis. Such an improvement in survival cuts into the period mortality immediately after MI, and hence changes life expectancy immediately, while the prevalence of post-infarction patients takes time to adjust. This combination produced a long period of spuriously declining life expectancy.

Mathers and Robine say that our “hypothetical example” was based on “a sudden and very large change in survival rates”. We readily admit that the change was large and sudden. However, as we pointed out earlier, the 25% decline in in-hospital death after myocardial infarction due to the adoption of thrombolysis that we used was, if anything, conservative. Recent work has shown a probably threshold effect of thrombolysis that caused the sudden large drop in cardiac death that has been observed in The Netherlands.4 Several other studies report similar observations. In Minnesota heart study was a 25% decline in coronary heart disease mortality and a 40% decline in in-hospital mortality between 1985 and 1990.5

Mathers and Robine agree that the Sullivan method does not cope very well with such changes. Moreover, the changes cannot be dismissed as “hypothetical”. And it is not just cardiovascular disease where epidemiology changes in an “unpredictable way”. For example,6 Nevertheless, for the purpose of the analysis of Mathers and Robine’s paper, making health expectancy estimates on an annual basis, the Sullivan method seems adequate. Indeed, one might like all other indicators to be as close to the “real” values as Sullivan’s method does in scenarios 2-4. Even in scenario 5, with sudden changes in disability incidence, it performs reasonably well.7 Then however,7 Problem 2, which originates from the use of health expectancy estimates to assess whether compression or expansion of morbidity is occurring. If, for example, life expectancy is estimated at 75 years and a sudden compression of disability free life expectancy is 63 years, while the real value is 64 years, nobody will complain about the Sullivan estimates being wrong: a deviation of 1.6% is not worth mentioning. But the next step becomes a bit awkward. To look at compression and expansion, life expectancy with disability is calculated as life expectancy minus disability free life expectancy = 75 - 63 = 12 years. Now the deviation is 9%. Still not a huge problem and a static estimate. The real problem comes when in a estimate a few years later the prevalence has reached its equilibrium value, and the Sullivan method reproduces the real value of 64 years. Looking for an answer to the compression and expansion question results in the conclusion that disability has compressed by 8.3% between the two measurements. A compression from 12 to 11 years with disability over a period of a few years is considerable, in particular when inferences on further developments are made based on this. Our point is that such changes could very well be artifacts produced by the Sullivan method, and that estimates based on this method should therefore not be used for conclusions on compression versus expansion of morbidity, as has been done.8


Reply

We are pleased that Barendregt et al support the major conclusion of our paper, that the Sullivan method is adequate for monitoring the changes in which transition rates and mortality rates are evolving without sudden and substantial change. We also agree with Barendregt et al that the Sullivan method will not cope well in situations where disability incidence rates have undergone a sudden change. We presented such a “sudden change” scenario at the population level in figure 8 of our paper, and Barendregt et al have presented a simulation of a “sudden change” scenario for myocardial infarction patients involving a rapid 25% decline in in-hospital death rates.7

Clearly, we are in substantial agreement on the weaknesses and limitations of Sullivan’s method to monitor changes in population health expectancies. What then about the specific criticisms of our simulations raised in the letter above?

Firstly, Barendregt et al argue that we keep life expectancies “unchanged” by forcing mortality rates of disabled people to depend on disability prevalence, which removes one source of problem with Sullivan’s method. We deliberately chose simulations based on real long term trends in life expectancy for French males and their observed disability prevalence change.9 We asked how Sullivan’s method and the multi-state method would compare under scenarios where the 1982 distribution was the result of
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 and women 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.

We intended no implication as to the plausibility of the scenario of Barendregt et al by describing it as “hypothetical disease example” or that was described by themselves used.1 We have no disagreement with their scenarios or its results, but repeat that such a simulation is not relevant to the question of how effective 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 also 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. 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 causes mortality rates (the relevant rates for use of 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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Figure 1 Trends in mortality from all causes and ischaemic heart disease, males and females, The Netherlands, 1952-94.

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 trial and the Canadian study of cardiovascular disease, have shown no significant effect of smoking, others, including studies in The Netherlands1 and America,2 have reported that the prevalence of Alzheimer’s disease was significantly lower in smokers than non-smokers. It has even been suggested that the mode of action might be via nicotine.3 Paradoxically, Brenner et al concluded that the protective effect was most marked at low dose exposure to cigarettes, but disappeared in those with the highest packs year exposure. van Duijn and Hofman, however, found an inverse relation between the number of cigarettes smoked and the risk of Alzheimer’s disease in women.4 Moreover, the Canadian study5 also confirmed the findings of other workers that the odds ratios for suffering 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,6 we reported findings based on 5319 respondents in the nationwide health and lifestyle follow-up survey during 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.

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

Sir—You published an interesting paper by Poulter et al7 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 medical information obtained from “true” controls and their proxy respondents was evaluated thoroughly. Categorical data were evaluated using Kappa statistics. Continuous data were evaluated by calculating the prevalence of each condition performed by means of the intraclass correlation coefficient (ICC). Nevertheless, detailed