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LETTERS TO THE EDITOR

Health expectancy: an indicator for change?

SIR – The article by Barendregt *et al* about the suitability of health expectancy as an indicator for change in population health requires a response.[1] As far as the authors describe the differences between the three available methods of calculation, we consider this publication a nice and useful illustration of the complexity of health expectancy calculations. However, their conclusion that the multistate approach is the only acceptable method of studying change and that Sullivan's cross sectional method produces incorrect results is based on one extreme and unrealistic example. As a participant of the meetings of the Network on Health Expectancy and the Disability Process (REVES), Barendregt can be expected to know that the REVES network has put much effort into debating the differences between the existing calculation methods. A number of papers presented at the sixth REVES meeting in Montpellier in October 1992 analysed the differences between the multistate and the Sullivan methods and presented more realistic examples.[2,3] We wonder why Barendregt *et al* do not refer to these REVES papers.

Barendregt *et al* do not explain sufficiently the concept of health expectancy, nor do they mention the different aims researchers might have in calculating this measure. Health expectancy is a general term that refers to the entire class of indicators expressed in terms of life expectancy in a given state of health (however defined).[4,5] Until now, health expectancy has most frequently been used as a public health indicator – that is, a yardstick for the (total) state of health of a population at a certain point in time. This population oriented use differs from the use as a predictor of the number of years an individual can expect to live in good health – the only application Barendregt *et al* seem to acknowledge.

As a public health indicator, a "Sullivan" health expectancy reflects the healthy years that a hypothetical individual can expect to live when current patterns of prevalence apply during an entire lifetime. Similarly, a multistate health expectancy reflects the hypothetical healthy years when current patterns of incidence apply for a lifetime. Neither assumption – constant prevalence rates or constant incidence rates – is realistic. Here Barendregt *et al* seem to be biased in favour of the multistate approach. Where the authors comment fully on the weaknesses of prevalence as a cumulative measure of present and past events, they forget to discuss the plausibility of stable incidences (which, by the way, like prevalence data, also reflect past conditions of living). They construct a hypothetical example – based on a sudden and very large change in survival rates, stable incidence rates, and varying prevalence rates – and thus make sure that in this example the multistate method performs well in predicting individual future health, while the Sullivan method fails.

The failure of the Sullivan method in extreme circumstances such as these is already well known from a previous analysis.[4] However, this study also made comparisons of the Sullivan and multistate method with French mortality and disability data under more realistic scenarios, typical of those which are actually likely to occur in populations. The conclusion of this study is that for realistic scenarios with moderate and long term trends in incidence and mortality rates, the difference between the estimates produced by the two methods is small and that Sullivan's method is acceptable for monitoring trends in health expectancies for populations.[4]

Barendregt *et al* claim that, unlike the Sullivan method, the multistate method "allows for one or more disease states including, when applicable, a 'cured' state" and that it can encompass patients who are cured or have intermittent disease free periods. It is well known, however, that the Sullivan method can also be used to calculate the expectation of years of life in any number of disease or health states, including, if desired, a "cured" state which is distinct from the disease free state. The Sullivan method also takes into account intermittent disease free periods, since these contribute to a lower measured average prevalence of disease.

Of course, every researcher dreams of perfect longitudinal databases that would not only facilitate the study of health status but also of the dynamics underlying the health and disability process. The reality to date, however, is that longitudinal databases are only available in a few countries and for restricted age groups (elderly). So, for years to come, Sullivan's method will be the most common method used worldwide. The consensus of the REVES network is that this method provides a useful indicator as long as its limitations are understood. Of course, it would be preferable if all calculations were made with the multistate method, but this will occur naturally if and when period data estimates become available.[5]

In conclusion, the argument – which has already occupied much time and effort – over which is the "right" measure seems unfruitful. What we really want to understand are differences across time and place in the population health structures and the outlook for individual lifes. If we had data to compute time series with both methods, simulations already carried out suggest that the answers would not differ greatly and that the Sullivan method is quite adequate to monitor long term trends in population health. Giving only half the story to policy makers is inappropriate.[2]

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1 Barendregt JJ, Bonneux L, Maas PJ van der. Health expectancy: an indicator for change? *J Epidemiol Community Health* 1994;48:482–7.
2 Crimmins EM, Saito Y, Hayward MD. Sullivan and multistate methods of estimating active life expectancy: two methods, two answers. In: Robine J-M, Mathers CD, Bone MR, Romieux I. *Calculation of health expectancies: harmonization, consensus achieved and future perspectives.* Paris/London: Colloque INSERM/John Libbey Eurotext Ltd, 1993;155–60. Vol 226.
3 Mathers CD, Robine J-M. Health expectancy indicators: a review of the work of REVES to date. In: Robine J-M, Mathers CD, Bone MR, Romieux I. *Calculation of health expectancies: harmonization, consensus achieved and future perspectives.* Paris/London: Colloque INSERM/John Libbey Eurotext Ltd, 1993;1–21. Vol 226.
4 Robine J-M, Mathers CD. Measuring the compression or expansion of morbidity through changes in health expectancy. In: Robine J-M, Mathers CD, Bone MR, Romieux I. *Calculation of health expectancies: harmonization, consensus achieved and future perspectives.* Paris/London: Colloque INSERM/John Libbey Eurotext Ltd, 1993;269–82. Vol 226.
5 Robine JM, Romieu I, Cambois E, Water HPA van de, Boshuizen HC, Jagger C. *Contribution of the Network on Health Expectancy and the Disability Process to World Health.* Montpellier: INSERM, 1995.

Reply

We are pleased to hear that Van de Water *et al* consider our article "a nice and useful illustration of the complexity of health expectancy calculations". We agree that the Sullivan method for the calculation of health expectancies provides a useful indicator, as long as its limitations are understood. The agreement ends though with the understanding of those limitations and, in particular, the consequences that should be drawn from them.

But first a matter of simple misunderstanding. When we said that the multistate method allows for various disease states, including cure, we were not implying that the Sullivan method does not, only that the double decrement method does not.

We do not understand the distinction Van de Water *et al* make between the population oriented and individual oriented use of health expectancy. As with the life expectancy estimator, it can be used on the population level as an indicator of public health, and be interpreted as the number of years an individual may expect to live in good health. Both uses are valid, and do not require different estimation procedures.

We also fail to see the point Van de Water *et al* are making about the realism of constant incidence and prevalence rates. We are certainly not claiming that incidence rates (including survival and cure rates) are more likely to be constant through time. On the contrary, prevalence, as a stock variable, tends to be more stable. Indeed, we are arguing that it is the relative volatility of flow variables (like survival and incidence rates), as compared to prevalence, that is causing bias in the Sullivan based trend estimates.

Nor have observed changes in incidence and survival rates been trivial. The effectivity of thrombolytic treatment is well known (our assumption of a 25% decrease in acute in-hospital deaths from myocardial infarction is conservative, if anything), as is its rapid introduction. Other major causes of disability and mortality that have recently seen rapid changes in The Netherlands are stroke (a 30% decline in mortality during the 1980s), accident mortality (down 20%), and hip fracture incidence (up 25%).

The "extreme" circumstances that make the Sullivan method fail thus seem to be far more common than Van de Water *et al* are willing to acknowledge. Of course, some causes seem to have remained fairly constant, like dementia and arthritis, but in a trend analysis, where you look at *differences* between levels, it is the causes which change that matter, not those which remain stable.

Van de Water *et al* contend that the problems with the Sullivan method and trend analysis have been well known for a long time.

It is true that these have been a recurrent, and clearly as yet unresolved, point of debate at the meetings of the REVES network, but in the peer reviewed international literature the problem has only been mentioned, albeit unsufficiently and not quite correctly explained, by Robine and Ritchie.[1] Other researchers apparently prefer to ignore it. In a comprehensive overview of the Dutch population health status, aimed squarely at policy makers, an interesting section, written by Van de Water, Boshuizen, and Perenboom, is devoted to health expectancy and its trends in The Netherlands.[2] Although the trend analysis is based on the Sullivan method, no mention is made that there might be a problem with the results. This can hardly be considered as giving the full story to policy makers.

And lastly, "pooh-poohing" the problems of health expectancy trend estimation from cross sectional data is a self defeating strategy. What policy maker worth his salt is going to endorse the large chunks of taxpayers' money needed for longitudinal studies when the researchers themselves say cheap cross sectional data will do fine? If the policy makers take the word of Van de Water *et al* for it, we will never be able to find out how wrong we are.

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1 Robine JM, Ritchie K. Healthy life expectancy: evaluation of global indicator of change in population health. *BMJ* 1991;**302**:457–60.
2 RIVM. *Volksgezondheid toekomst verkenning: de gezondheidstoestand van de Nederlandse bevolking in de periode 1950–2010.* Den Haag: Sdu Uitgeverij, 1993.

Socioeconomic factors and injuries

Sɪʀ – The conclusions stated in the article by Petridou *et al*[1] seem a little confusing. Their multiple logistic regression-derived odds ratio estimate for paternal schooling is 0·66 (95% CI=0·44, 0·99). It is then concluded that low socioeconomic status (SES), as reflected by paternal education, increases the risk for school related injuries. An odds ratio of less than 1, however, suggests there is a protective effect against school injuries for paternal schooling. Unfortunately, since the authors did not provide sufficient information on how paternal education was defined in this study, their conclusion seems to contradict their data. It is likely that the authors' data reflect a protective effect against injuries for some degree of paternal schooling, but the reader is left to infer just what this may be.

These results, along with their reported significance for school injuries to children from single parent homes, are contradictory to our case-control study of Ghanaian childhood burns[2] and our prospective study of the incidence and determinants of all-cause injury in adolescents in the United States.[3] There are other examples of discrepancies in the literature for both SES as an injury risk factor[4 5] and in the reported incidence of injury in developing and developed countries.[6] Comparisons of injury data are most often impeded by two factors: a lack of a consistent case or attribute definition and variations in case ascertainment rate. We have been advocating the use of standardised terminology in injury research including demographic descriptors to permit valid comparisons of injury research. With the growing interest in risk factors for injuries in childhood and adolescence, including school injuries, it is important to present concise and thorough information as a guide to researchers so that comparisons can be made across studies.

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1 Petridou E, Kouri N, Trichopoulos D, Revinthi K, Skalkidis Y, Tong D. School injuries in Athens: socioeconomic and family risk factors. *J Epidemiol Community Health* 1994;**48**:490–491.
2 Forjuoh SN, Guyer B, Strobino DM, Keyl PM, Diener-West M, Smith GS. Risk factors for childhood burns: a case-control study of Ghanaian children. *J Epidemiol Community Health* 1995;**49**:189–93.
3 Anderson R, Dearwater SR, Olsen T, Aaron DJ, Kriska AM, LaPorte RE. The role of socioeconomic status and injury morbidity risk in adolescents. *Arch Pediatr Adolesc Med* 1994; **148**:245–9.
4 Pless IB, Verreault R, Tenina S. A case-control study of pedestrian and bicyclist injuries in childhood. *Am J Public Health* 1989;79:995–8.
5 van Rijn OJL, Bouter LM, Kester ADM, Knipshield PG, Meertens RM. Aetiology of burn injuries among children aged 0–4 years: results of a case-control study. *Burns* 1991;17:213–19.
6 Chiu W, Dearwater SR, McCarty DJ, Songer TJ, LaPorte RE. Establishment of accurate rates for head and spinal cord injuries in developing and developed countries: a capture–recapture approach. *J Trauma* 1993;35:206–211.

Reply

Sɪʀ – We appreciate the interest of Drs Forjuoh and Dearwater on our paper,[1] but we are mystified by their concern over our results concerning socioeconomic class as reflected in paternal education. Our data show that an increased paternal schooling by 3 years, that is *higher* socioeconomic status, is associated with significantly *reduced* risk for school injuries by 34% (odds ratio 0·66; 95% confidence interval 0·44, 0·99). Obviously, *lower* socioeconomic status *increases* this risk, which is exactly what we have reported. We find it hard to further simplify the expression "3 more years of paternal schooling".

We agree with Forjuoh and Dearwater that there are discrepancies in the literature concerning risk factors for childhood injuries but editorial policies of the *Journal* with respect to short reports did not allow as to expand on this issue. We also agree with Forjuoh and and Dearwater that standardised terminology is needed in this as in any other field. Whether their approach, ours, or that of another group should be the basis of an eventual consensus cannot be ascertained at the present time.

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1 Petridou E, Kouri N, Trichopoulos D, Revinthi K, Skalkidis, Y, Tong D. School injuries in Athens: socioeconomic and family risk factors. *J Epidemiol Community Health* 1994;**48**:490–491.

NOTICE

The 11th International Conference on Pharmacoepidemiology (ICPE), sponsored by the International Society for Pharmacoepidemiology (ISPE), will be held from Sunday, August 27, 1995 to Wednesday, August 30, 1995, in Montréal, Canada. The conference focusses on global public health issues, including a session devoted to issues from developing countries. For further information, contact Dr Stanley A Edlavitch at the ISPE office, University of Kansas Medical Center, Department of Preventive Medicine, Robinson 4040, 3901 Rainbow Blvd, Kansas City, Kansas 66160–7313. Tel: 913-588-2790; fax: 913-588-2791; e-mail: ISPE@UKANVM.CC.UKANS.EDU.

BOOK REVIEWS

Physical Activity and Health. Society for the Study of Human Biology Symposium 34. Ed N G Norgan. (Pp 262; price not stated.) Cambridge: Cambridge University Press, 1992. ISBN: 0-521-41551-9.

This is an up to date and wide ranging account of the key issues of the biology of physical activity and health. Its six chapters are presented by different contributors and cover comparative and temporal activity in humans, the concept and methodology issues associated with activity, exercise, health, and fitness (as well as their inter-relationships) and an overview of current and future lifestyles.

Metabolic rates, speeds, and geographical ranges of activity are compared with those of animals. People are neither remarkably active nor remarkably inactive for mammals of our own size. The problems associated with health measurements are discussed. It is argued that value judgements are implicit in the definition of health. The best that can be achieved is to make the value judgements used explicit so that those with other value systems can interpret the data. An account is given of both the Allied Dunbar fitness survey and the Welsh heart health survey. Studying childhood activity shows that the percentage of body fat in the early teenage period seems to be the most important coronary disease indicator in predicting risk levels. This indicates a need for increased activity and weight reduction. Retirement from full time employment can potentially result in a reduction in activity resulting in a vicious circle of declining function and further reduction in activity. In general, it seems that older people are not very active and become less so prematurely. It is confirmed that exercise seems to play an important role in the prevention of weight gain.