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 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 save for 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 their scenario as "hypothetical example"; that was the description they themselves used. We have no disagreement with their scenario or its results, but repeat that such a simulation is not relevant to the question of how deep in Sullivan's model 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. 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 methods.

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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JEAN-MARIE ROBINE
Equipe INSERM Demographie et Santé, Montpellier, France

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 health and aging, 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 been suggested that the mode of action might be via nicotine.3 Paradoxically, Brenner et al4 also found that the protective effect was most noticeable 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 risk of Alzheimer's disease.5 The Canadian study6 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,7 we reported findings based on 5319 respondents in the nationwide health and lifestyle follow-up survey in The Netherlands 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 Poulet et al8 on the reliability of data derived from proxy respondents in an international case-control study of cardiovascular disease and use of 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 the intraclass correlation coefficient (ICC). Nevertheless, detailed
study of the paper has revealed that the refer-
ence on which the calculation of the concord-
ance between the continuous variables con-
sidered in the study is based contains a possible error.

The ICC is the proportion of total variabil-
ity accounted for by the variability among sub-
jects. A high ICC means that not much of the
variability is due to variability in measure-
ment on different occasions (proxy versus
true control, in this case) or, what amounts to
the same thing, that the agreement between
each subject can easily be calculated from
an analysis of variance (ANOVA), as shown in
the example in table 1 (formula 1). As the
ANOVA may be somewhat burdensome to
carry out, Deyo et al offered a simple method
for calculating the ICC (see formula 2 in
table 1).

The calculation of the ICC by Poulter et al
seems to be based on a commonly cited review article on statistics of concordance,4
which unfortunately omitted n in the de-
nominator of the original equation for ICC
proposed by Bartko.1 Results based on this
mistaken formula underestimate the ob-
served agreement as shown in table 1
(formula 3).

Formulas 1 to 3 assume a random effects
model. Another model must nevertheless be
considered in making inferences about the
factors affecting the measurements—the
fixed effects model.7 In the random effects
model, the two raters implied in the measure-
ment are assumed to be a sample from a
larger population of raters. In the fixed effect
model, the two raters are the only raters about
which inferences will be made. Although the
calculation of ICC assuming a fixed effects
model is also based on the ANOVA table,
the formula is different (see formula 4 in table 1).

Prie to et al have also proposed a simpler
method for calculating the ICC in this case
(see formula 5 in table 1).

The calculations of Poulter et al on the
ICC assume a random effects model. Since
proxy and "true" controls are the only raters
about which inferences were made, we
believe that it would be more correct to
assume a fixed effects model. Nevertheless,
more information concerning the selection of
cases and "true" and proxy controls would
be necessary to strengthen this hypothesis.

Since important differences in results may
be obtained, future studies that consider the
agreement of continuous variables must be
extremely careful in selecting formulas to
estimate the appropriate ICC.

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ALFONSO CASADO
JORDI ALONSO
Health Services Research Unit, Institut Municipal
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E-08003 Barcelona, Spain

1 Poulter NR, Chang CL, Farley TMM, Marmot
MG. Reliability of data from proxy respondents
in an international case-control study of
cardiovascular disease and oral contraceptives.

2 Fleiss JL. The design and analysis of clinical
experiments. New York: John Wiley & Sons,

3 Deyo RA, Diehr P, Patrick DL. Reproducibility
and responsiveness of health status measures.

Table 1 Estimation of agreement between two measures (A and B) through the intraclass correlation coefficient (ICC). Hypothetical data concerning two measures in 10 patients

<table>
<thead>
<tr>
<th>Subject</th>
<th>Measure A</th>
<th>Measure B</th>
<th>Difference (B-A)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>75</td>
<td>80</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>74</td>
<td>84</td>
<td>10</td>
</tr>
<tr>
<td>3</td>
<td>76</td>
<td>81</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>79</td>
<td>83</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>82</td>
<td>92</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>83</td>
<td>88</td>
<td>5</td>
</tr>
<tr>
<td>7</td>
<td>85</td>
<td>90</td>
<td>5</td>
</tr>
<tr>
<td>8</td>
<td>87</td>
<td>92</td>
<td>5</td>
</tr>
<tr>
<td>9</td>
<td>87</td>
<td>92</td>
<td>5</td>
</tr>
<tr>
<td>10</td>
<td>88</td>
<td>93</td>
<td>5</td>
</tr>
<tr>
<td>Mean</td>
<td>81.6</td>
<td>87.5</td>
<td>5.9</td>
</tr>
<tr>
<td>SD</td>
<td>28.1</td>
<td>25.4</td>
<td>4.8</td>
</tr>
</tbody>
</table>

ANOVA table on preceding data

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>DF</th>
<th>SS</th>
<th>MS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between subjects (S)</td>
<td>9</td>
<td>459.45</td>
<td>51.05</td>
</tr>
<tr>
<td>Within patients (Measurements) (M)</td>
<td>1</td>
<td>174.05</td>
<td>174.05</td>
</tr>
<tr>
<td>Residual (R)</td>
<td>9</td>
<td>21.45</td>
<td>2.38</td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
<td>654.95</td>
<td></td>
</tr>
</tbody>
</table>

Formulas for the calculation of the ICC

Random effects

ICC Bartko8

\[ n(MS_x-MS_y) = \frac{10-(51.05-2.38)}{51.05+2.38} = 0.55 \]  
(formula 1)

ICC Deyo et al9

\[ SD_x^2 + SD_y^2 + (n-1)MS_x = (28.1+4.8) = 0.55 \]  
(formula 2)

Icc Kramer and
Feinstein10

\[ MS_x + MS_y = \frac{10.51-2.38}{51.05+2.174} = 0.12 \]  
(formula 3)

Fixed effects

ICC Fleiss6

\[ n(MS_x-MS_y) = \frac{10-(51.05-2.38)}{51.05+2.38} = 0.69 \]  
(formula 4)

ICC Prie to et al7

\[ SD_x^2 + SD_y^2 + (n-1)MS_x = (28.1+4.8) = 0.69 \]  
(formula 5)

1 Poulter NR, Chang CL, Farley TMM, Marmot
MG. Reliability of data from proxy respondents
in an international case-control study of
cardiovascular disease and oral contraceptives.

2 Fleiss JL. The design and analysis of clinical
experiments. New York: John Wiley & Sons,

3 Deyo RA, Diehr P, Patrick DL. Reproducibility
and responsiveness of health status measures.

Table 1 Comparison of ICC estimates using random and fixed effects models (comparisons relate to data shown in reference 2)

<table>
<thead>
<tr>
<th>Subject</th>
<th>Random effects</th>
<th>Fixed effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Husband</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Weight</td>
<td>0.90</td>
<td>0.89</td>
</tr>
<tr>
<td>Height</td>
<td>0.80</td>
<td>0.79</td>
</tr>
<tr>
<td>Duration current OC</td>
<td>0.98</td>
<td>0.98</td>
</tr>
<tr>
<td>Duration last OC</td>
<td>0.79</td>
<td>0.80</td>
</tr>
<tr>
<td>Mother</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Weight</td>
<td>0.99</td>
<td>0.96</td>
</tr>
<tr>
<td>Height</td>
<td>0.90</td>
<td>0.89</td>
</tr>
<tr>
<td>Duration current OC</td>
<td>0.52</td>
<td>0.63</td>
</tr>
<tr>
<td>Duration last OC</td>
<td>0.35</td>
<td>0.25</td>
</tr>
<tr>
<td>Sister</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Weight</td>
<td>0.96</td>
<td>0.97</td>
</tr>
<tr>
<td>Height</td>
<td>0.87</td>
<td>0.86</td>
</tr>
<tr>
<td>Duration current OC</td>
<td>0.03</td>
<td>0.47</td>
</tr>
<tr>
<td>Duration last OC</td>
<td>0.89</td>
<td>0.93</td>
</tr>
<tr>
<td>All subjects</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Weight</td>
<td>0.93</td>
<td>0.92</td>
</tr>
<tr>
<td>Height</td>
<td>0.83</td>
<td>0.82</td>
</tr>
<tr>
<td>Duration current OC</td>
<td>0.94</td>
<td>0.91</td>
</tr>
<tr>
<td>Duration last OC</td>
<td>0.77</td>
<td>0.75</td>
</tr>
</tbody>
</table>

OG = oral contraception.
NOTICES

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Information for Authors

6th International Symposium on Epidemiology and Occupational Risks, 22-24 April 1996, Graz, Austria.
International conference on the role and importance of epidemiological studies in the prevention of and compensation for occupational accidents and diseases. Further information: Allgemeine Unfallversicherungsanstalt, Office for International Relations and Conferences, Adalbert-Stifter-Strasse 65, A-1200 Vienna. Tel: +43 1 33111 537. Fax: +43 1 33111 469. email: presse@auva.or.at.

BOOK REVIEWS


Responding to the needs of older people probably means the biggest challenge facing health and social care, to quote the author himself. This report examines the process of joint commissioning of services by health and local authorities in five contrasting local areas. Much of the work of the project has been recorded in a series of previous King’s Fund papers; the present report draws out the key lessons.

Monitoring the process of service development is a slippery task, especially when a majority of agencies is involved. As this report acknowledges, significant changes will take time, often longer than an individual researcher is able to follow them. So here we have an account of work in process, rather than nearly defined historical episodes.

Poxtton presents grounds for modest optimism. Achievements differ in the various localities, but rigorous evaluation of planning initiatives is seldom easy, and short-term perspectives may be misleading. The qualities of vision and passion, here identified as essential, are particularly difficult to pin down.

One complication to the success of joint commissioning which the report neglects relatively is that of the different occupational settings of the participants. GPs often point out that social services departments have high staff turnover; their own positions are relatively stable, while allowing them considerable scope in deciding the margins of their job. In a primary care led NHS, their contribution to processes like joint commissioning will be increasingly influential.

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Populations are ageing in almost every country, and this book begins to fill a large void in the literature of health in the elderly. There are 45 chapters with an impressive list of contributors. Two thirds are from the UK and the rest from eight different countries. Many are established authorities on their chosen subject. As Margot Jefferys says in her editorial preface, this should become a standard reference book.

Chapter subjects range well beyond consideration of individual diseases, to encompass, for example, health economics, community care, migration and ethnicity, and genitourinary. Its scope aims (and generally succeeds) in being truly international. The editors have succeeded in keeping contributors to a uniform chapter length of about nine pages. They have been less successful in achieving a universal standard of up to date
ness. However, given the explosion of knowledge in the past decade, and the ease with which it can be retrieved, this is a serious point. There are dangers ahead too—volumes like this age quickly in matters of detail.

Nevertheless, Epidemiology in Old Age is a splendid achievement. The price is too steep for individuals, but medical libraries will find it of great value. It is extremely well produced with a vivid purple cover. Specialists will find their particular concerns examined in more detail elsewhere, but for those who want to venture into new aspects of geriatric epidemiology, their search begins here.

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

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*J Epidemiol Community Health* 1997 51: 579-581
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