Fertility

Proctor also comments on our reference to Martin Gumpert, who intimated that the Nazi campaigns to increase fertility were failing. Gumpert managed to escape from Germany before 1939 and did much to advertise the level of misery in Hitler's state. His book *Hoi! Hunger* was an attempt to demonstrate that a popular contemporary view—that the Nazis had improved health in Germany—was incorrect. There was widespread poverty outside of Germany and appears to have been an effective intervention. When discussing the fertility campaign Gumpert was referring to the later stage of the “battle for births”, rather than its early days. As Proctor points out there was an increase in the birth rate and marriage rate immediately following the imposition of the Nazi rule. The birth rate increased from 15.1/1000 in 1932 to 18.9/1000 in 1936.14 But, this should be seen against the decline which preceded it. From a rate of 35.6/1000 in 1900 the fertility rate declined (to 31.6/1000 in 1910, 26.8/1000 in 1914, 20.3/1000 in 1917, and 17.5/1000 in 1918) and by the early 1930s it had reached an all-time low.15 Seen in this light the “success” of the pronatalism campaign was modest.16

Interest-free marriage loans were offered from 1933, when many partners had passed the tests of political and eugenic reliability. Family allowances, with one-off payments at the birth of each child, were followed by the introduction of recurrent grants, intended with each child, and propaganda intended to encourage working women to return to the home to raise children.17 Increasing legal sanctions against abortion were imposed, culminating in the death penalty in 1933. Many were being introduced for habitual assistance at abortions.18 In the light of these activities, the last of which would increase the birth rate by far rather than winning the propaganda war, the cessation of the death penalty in any sustained rise in fertility can be seen as the basis for Gumpert’s consideration that German mothers “had gone on strike”. Gumpert commented cynically on many of the details of Nazi social policy. He considered that attempts to claim that poor health was due to bad lifestyle was serving as a smoke screen, to cover up for the genuine decline in Germany which was due to the iniquities of Nazi policies. Thus he considered the then campaigns to reduce fat consumption during a period of hunger were particularly invidious, stating, “there emerges today health administration hyenas who proclaim to the public that butter is poison.” 19

Lack of support

The anti-smoking campaigns in Nazi Germany, extensive as they appear, did not engage the unquestioning support which might have been expected for activities seen to be fully in line with the Führer’s wishes. When the Deutscher Bund Zur Bekämpfung der Tabakgelenw, a president it received a stream of letters from potential candidates stating that they would not take up the posts. The reasons given included the invited individual admitting to be “a passionate smoker”. Even letters from the feared Nazi labour chief, Fritz Sauckel, received the same response. When Karl Astel, rector of Jena University and head of the Institute for the Struggle against the Dangers of Tobacco, attempted to have the bodies of all deceased smokers sent to the Jena Pathology Institute for investigation of the damage that smoking had induced, public opposition required the withdrawal of the proclamation within two months of it being issued. The military were also opposed to restrictions on tobacco and this may have led to a lessening of the degree to which anti-smoking legislation was enforced.20

Legacies of Nazism

The legacies of Nazism in contemporary Germany are complex and contradictory.21 A motivated systematic rejection of the Nazi period can be seen in everything from functionalist architecture, the spact and apolitical nature of universities and television, the desire for press freedom even when it produces the embarrassment that is *Illud* (Europe's biggest selling newspaper, which can on occasions make the English *Sun* read like *New Left Review*), and consensus Government, through to the more extreme and obvious counter-reaction to the Nazi past by the Baader-Meinhof Group and Red Army Faction,22 or the alternative living situations in squats of many German cities of the Aussenring and Sponti. Some commentators consider through it all an intense sense of that through it all an intense sense of formality remains.23 With these contradictions, the direct translation of policies enacted during the Nazi period into what has happened in Germany since the war is problematic, but then again so is simply ignoring history.114

Estimating life expectancy using an age–cohort model: a critique

Sir—In a recent article published in this journal, Lee and Hsieh1 proposed using the age–cohort model for studying on smoking and Schneider2 to estimate cohort mortality and cohort life expectancy at birth. They applied it to estimate the cohort life expectancy in Taiwan. The model is a multiplicative Poisson regression model with an intercept term, an age effect term, and a cohort effect term. It does not include an interaction term. As such, the model assumes a constant age pattern of mortality across cohorts.1

I have great reservations about this implicit assumption. Child and adult mortality are subject to different factors. On one hand, during the epidemiologic transition, communicable diseases were replaced by chronic diseases, which are more common than communicable diseases. Besides, most public health measures in developing countries after the second world war focused on improving maternal and child health. Therefore, the relationship between mortality generally declined faster than adult mortality. On the other hand, however, we expect that in countries whose mortality level is already very low, further mortality declines will be concentrated in older ages because of the law of diminishing marginal returns. So there is no reason to presume a constant age pattern of mortality.

In their study of the incidence of bladder cancer among men at a large German and Schneider's2 justified their application of the model graphically by showing that the cohort curves of logitnormalized, age specific incidence rates, were “quasi–parallel” to one another. However, the determinants of a single disease are not as diverse as those of all–cause mortality; the study of adenocarcinoma, for example, is more complex than that of a vascular disease. The quasi–parallel is unlikely to occur in the context of Lee and Hsieh's application.3

Figure 1 plots some of the data which appears in Lee and Hsieh's paper. Not surprisingly, for the cohorts born earlier this century, the mortality curves are reasonably parallel in adulthood and old age. The two

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Figure 1 Age specific mortality rates in selected cohorts in Taiwan. The 1941 curve is close to the 1951 curve. It is excluded to avoid visual confusion. The legends indicate the mid-points of the five year cohorts.

post war cohorts have an almost identical mortality level in the age range 15–29, but the 1961 cohort has much better child mortality level. Due to the lack of an interaction term, the model would overestimate child mortality in the 1961 cohort and underestimate its adult mortality level in relation to the 1951 cohort’s. Note that child mortality is more heavily weighted in the calculation of life expectancy since it affects the number of person-years lived in all subsequent age intervals. In contrast adult and old age mortality affect fewer intervals. Therefore, the estimates of life expectancy at birth based on the age-cohort model may be biased. We should not read too much into figure 1 because the data are scanty. Nor should we overlook it because it is consistent with our epidemiological knowledge.

Obviously the age–cohort model cannot accommodate an interaction term because of the missing data for some cells. Therefore, its use should be limited to the age ranges and/or cohort ranges for which we expect the assumption of constant age pattern to hold. For instance, we can apply it to the estimation of life expectancy at age 60 with reasonable confidence. Of course, the assumption must be empirically checked whenever possible.

When the 1951 cohort was compared with the 1941 cohort, the relative risk of death in 1951 was significantly higher than in 1941. This is consistent with the assumption that the 1951 cohort was exposed to the effects of the 1951 pandemic. However, the results are not statistically significant for the 1961 cohort compared with the 1951 cohort. This may be due to the small size of the 1961 cohort or to the lack of an adequate interaction term in the model.

The results of this study suggest that the age–cohort model is a useful tool for estimating life expectancy in populations with incomplete data. However, further research is needed to validate the results and to refine the model for use in populations with more complete data.

Tuberculosis among homeless people at a temporary shelter in London

SIR—Each year the charity Crisis sets up temporary Christmas shelters, not for “the homeless” but for homeless people. Kumar et al use the former term on 23 occasions in their report of a chest x ray screening programme.1

Issues of concern were the high prevalence of tuberculosis and suboptimal management of known and presumed cases due to patient “absence”. These problems have been previously documented in a very similar group of homeless people in London during the mid 1980s.2 In the earlier survey, there was a notable association between tuberculosis and alcohol (ab)use,3 which may have an impact on treatment compliance. Data from the Crisis Open Christmas4 could perhaps be used to ascertain whether there was an association between “loss to follow up” and accommodation status of “no fixed abode” or self reported “regular alcohol consumption”.

The documented health care needs of single homeless, homeless and roofless people are multiple.4 The prevalence of mental illness noted by Kumar et al is low by comparison with other survey data,5 may have been significantly underestimated by selection or reporting bias.

A comparably wide range of accessible and acceptable health care provision therefore seems appropriate and is, indeed, a feature of most dedicated services for homeless people.6 Recommendations to improve current provision, based on specialist outreach respiratory/tuberculosis services, will have limited capacity to address important and potentially confounding endemicity.

The most effective means of delivering comprehensive health care to homeless people, including case finding and management of those with tuberculosis and their contacts, needs formal evaluation. However, the views of professionals in primary and community health services and in the voluntary sector, who best understand the motivating factors and influential social networks of works of homeless people, should not be overlooked. Indeed, the suggestion of patient held records was recently raised in The Big Issue.7

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10 The Big Issue 1996;170:25.

Reply

Dr Cheung points out correctly that the age–cohort model we used to estimate cohort life expectancy in Taiwan has an implicit assumption of constant age pattern across cohorts. Dr Cheung also explains why such an assumption is unlikely to be met, especially in a population under epidemiologic transition. I fully agree with his views.

In the context of age-period–cohort analysis, the problem of non-parallelism in age patterns has been explicitly addressed.23 Unfortunately, the proposed methodologies are a bit too complex for the present purpose. Instead, simple addition of an interaction term to the age–cohort model is not feasible because of unavoidable missing cells from age–by-cohort cross tabulation. Instead of resorting to models with interaction terms, I feel that a practical solution to this problem may be to perform a “separate analysis”. For example, we can break down the data by age into, say, “childhood”, “adolescence” and “elderly” group, each modelled separately, and then the results combined. By this method, different segments of the population assume different cohort trends. Alternatively, we can break down the all-cause data into their component causes, modelled separately and then combined—an approach suggested in our paper.1 Clearly, such an approach pays due respect to the different roles of communicable and non-communicable diseases during the epidemiologic transition. However, the benefits we gain from these more “sophisticated” approaches must be weighed against what we can infer from “crude” but “simple” age–cohort modelling as shown in our paper.1

Finally, Dr Cheung has stated that the assumption of parallelism is not met in our data after the 1961 cohort. I wish to point out that we also refute the results after 1961, though for a different reason.

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