PostScript

LETTERS

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Siberian live birth sex ratios and the SPrOO hypothesis

Melnikov and Grech found a highly significant seasonal pattern of the sex ratio (SR) at birth in western Siberia, namely, a peak in the second and a trough in the fourth quarter of the year.[1] This peak and trough are in line with the seasonal preovulatory overripeness ovopathy (SPrOO) hypothesis, which states (1) an approach to gender equity at the peak of the ''ovulatory'' seasons, (2) preferential fertilisation of non-optimally matured oocytes by Y-bearing sperm during the transitional stages between them, and (3) SR reversal because of excess of male biased fetal loss during the ''anovulatory'' seasons.[2] The mentioned peak would correspond, just like in non-human mammals with the breakdown of the ''ovulatory'' season in spring; the trough with SR reversal during the ''anovulatory'' season in winter.

The authors wonder about the annual secular trend in a fall and then rise of the SR with the turning point in early 1980s, being different from the continuous decline in industrial countries over the past half century. We have argued that the rise in SRs before the first world war in Finland[3] and in many other countries concurred with continuous improvement in living conditions, education, and reproductive hygiene, and, thus, a decrease of conceptopathology rate, and in turn, increase of male surviving fetuses.[4] The fall of the SRs after the turning point around the second world war was interpreted as consequence of further amelioration of the ovulatory and conception pattern, reflected by the concurrent decrease in pregnancy wastage. The same reasoning accounts for the initial very low and then increasing and again decreasing SRs in developmental countries that are in demographic transition going hand in hand with amelioration of socioeconomic conditions. This may be compared with the rise and fall in socially upward family conditions.[5] The odds for delivering a male child increases (while pregnancy loss diminishes) when the socioeconomic level increases from low to moderate up to a plateau and then decreases (despite continuation of decreasing pregnancy wastage) when this level increases further from moderate to higher.

The relatively low SRs in western Siberia, when compared with other countries in west Europe, may also be related to higher rates of conceptopathology because of the extreme climatic variations and inherent stronger seasonal variation in reproduction further away from the equator. This suggests higher rates of male biased pregnancy loss as the underlying mechanism. The SR increase in the early 1980s would mean that this rate is diminishing and that further progress in socioeconomic conditions will result in still higher SRs and ultimately in a decrease, in analogy with those in west European countries.

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Siberian pros and cons of the SPrOO hypothesis

I thank Dr Jongbloet for thoughtful response to our article. According to the SPrOO hypothesis, human females have a fundamental seasonal variation in ovulatory pattern, one of several factors explaining differences in sex ratio (SR). The hypothesis presumes inherent ovulatory and anovulatory seasons and suggests that secondary SR varies over the year from the femininity, or an approach to gender equality, coinciding with the zenith of birth frequency, to the excess of male births concentrating at the beginning and the end of this optimum. The question is what are these seasons in Siberia?

An analysis of our data by month yielded the seasonal pattern for SR variation ($p<0.1$, Edwards' test). According to the hypothesis, the birth optimum should occur in February–March when the small trough in SR is observed. This period of gender equality corresponds to the ''ovulatory'' season in May–June. This trough in turn was preceded by a sharp peak in SR occurring in January and followed by the major peak in April–June. These two peaks might seem to reflect ''transitional stages''.

What are the arguments against the hypothesis?

Huntington's 1938 report of the seasonal variation in both the SR and the total number of births in seven countries, based on analysis of about 52×10^6 births, showed an *inverse* relation between the SR and the number of births.[1] Just the contrary is apparent in Siberia. These two curves agree closely at least during the first half of the year. In other words, the total number of births and the number of male births vary correspondingly. This direct relation between SR and birth number seems to be a characteristic feature of the Siberian population.

An analysis of 1989 Novosibirsk census data shows that January born males and females comprise 20.8% and 24.2%, respectively, of men and women aged more than 80 years, whereas the expected proportions according to the uniform distribution would be 100/12 = 8.3%. This means that January as a month of birth and April as a month of conception are strong predictors of longevity in Siberia. However, in accordance with the hypothesis, April ought to be a month of conceptopathology, associated with ''preferential fertilisation of non-optimally matured oocytes by Y-bearing sperm'' and would consequently not seem to be associated with the surprisingly long span of life seen in this cohort.

In summary, while I agree that some aspects of the Siberian data are ''in line with'' the SPrOO hypothesis, I do not find this concept to be the most satisfying explanation of our findings. While the hypothesis has been supported by animal studies, its reliance on mechanisms such as ovopathy and differential pregnancy loss makes it difficult to establish (or refute) on the basis of studies such as our own. We remain persuaded that A Lerchl's hypothesis of the temperature dependence of SR better explains our peak of total and male births in January.[2]

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Ethnicity and epidemiological research: not so black and white

The analysis by Ahern *et al* of risk factors for preterm birth among African American and white women in San Francisco[1] concluded that pregnant African American smokers are more prone to preterm delivery than white pregnant smokers. This conclusion is misleading. Firstly, the evidence of interaction between smoking and ethnicity was unconvincing—the difference in the odds ratios

(ORs) was modest, and confidence intervals (CI) overlapped considerably (African American women: OR 1.77, 95% CI 1.12 to 2.79; white women: OR 1.25, 95% CI 1.01 to 1.55). Secondly, the authors did not consider residual confounding by factors such as maternal infection and previous preterm birth, which differ by ethnic group.[2 3] Their assumption that the smoking-preterm birth association is linear seems biologically unlikely and problematic, as African American women in their study population smoked more than white women.

Such analyses raise a more fundamental issue: the limitations of using ethnicity/race in epidemiological studies of causality. There is no consensus as to what "ethnicity" means. To some, "ethnicity" describes cultural differences between populations definable by phenotype, while "race" signifies differences strictly under genetic influence. This separation is imperfect—"ethnic groups" differ in genetic mix, culture, and socioeconomic situation. The "catch all" quality of ethnicity makes its use in epidemiological research attractive, but is an obstacle to causal inference. Ethnic groups have qualitative and contextual differences that do not make them directly comparable. Ethnic differences in biological effects will be difficult to uncover, as cultural, social, and economic factors that act as causal intermediates are too numerous and divergent to be adequately controlled.

Finally, ethnicity is not a singular exposure that can be turned on and off. The observation of higher preterm birth rates among black than among white women smokers is not simply the answer to the question "what would rates of preterm birth among white women who smoke be, if they were black?" Social inequity and deprivation, however, are amenable to change in a way that ethnicity is not. In the paper by Ahern *et al*, African American women delivering preterm clearly were more often single, working class, receiving public insurance, and had lower level of education. Yet their analysis stratified by ethnic group does little to explain these great disparities between African American and white women, concentrating instead on variations within ethnic groups. An analysis of their data comparing preterm birth between social strata rather than ethnicity would shed some light on the role of ethnicity in research: useful tool or epidemiological distraction.

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Authors' reply

In their letter, Tam *et al* present several critiques of our analysis of smoking and preterm delivery in African American and white women and then broadly criticise the use of race and ethnicity as factors in epidemiological research. We address each issue in turn.

Firstly, Tam *et al* object to the fact that the odds ratios (ORs) for the effects of smoking on preterm delivery for African American women and white women in our analysis were only modestly different and that their confidence intervals overlap. However, even a small difference in risk can translate to a very large difference in the number of people affected on the population level. In addition, the ORs presented in our paper were for 10 cigarettes per day. The ORs for a pack of 20 cigarettes per day were 3.13 for African American women and 1.55 for white women, a more substantial difference. More importantly, the key difference worth examining is that of the parameter estimates on the log odds scale. The change in the log odds of preterm delivery for every cigarette smoked per day was 0.057 for African American women, while it is only 0.022 among white women—a difference of more than twofold. Ultimately, while there was some overlap in the confidence intervals presented, this observation is not equivalent to a statistical test of the difference between the values. Such a test was not used because of the stratified nature of our sample; we chose a stratified sample for methodological reasons that we address below. We believe the interpretation of point estimates is still appropriate in our study.

Next, the authors suggest our effect estimates may be subject to residual confounding. This clearly may be the case with any observational study. However, in this particular instance, the factors that Tam *et al* discuss as omissions on our part are potentially on the causal pathway between our exposures and outcomes of interest, and thus, should not be adjusted for in our models. If prior cigarette smoking contributed to an earlier preterm delivery, adjusting for prior preterm delivery might have washed out the very effect we were interested in studying. The issue of maternal infections was explicitly discussed in our paper as a factor that may interact with smoking in its relation to preterm delivery. While we were unable to examine this in our analysis, as we lacked data on maternal infections, we hope that this work and our discussion will encourage others to look at this question.

Tam *et al* also question the linearity of the relation between smoking and preterm delivery. In our analysis, we found no evidence of non-linearity in the relation between smoking and preterm delivery. The fact that African American women smoked more on average does not affect the validity of the risk estimates, as cigarette smoking was modelled as cigarettes per day and the overall range of values was similar for African American and white women.

Finally, Tam *et al* criticise our decision to examine effects of neighbourhood and individual risk factors for preterm delivery within, rather than across, racial groups. Our decision to do so was inspired by the lack of such prior research and the fact that an expert conference on the problem of racial disparities in preterm delivery had called for just such an analysis.[1]

We agree with Tam *et al* that race encompasses social, economic, and cultural factors. Of course this makes the study of race complex. However, complexity has never before been a reason to abandon the study of an issue. In the context of the United States, the history of racial oppression and strife makes this a crucial issue to study in the hope of understanding its effects on people's lives. The differences in the rates of preterm delivery between African American and white women are independent of socioeconomic status[2 3] making racial differences even more important to understand. The complexity of the question, far from scaring us away, should draw us in. While race is not amenable to change, there may be underlying reasons for a racial disparity in health that may in fact be amenable to change. A differential effect of smoking on preterm delivery by race, suggested in our analysis, may be one such underlying factor contributing to racial disparities. We hope our analysis will lead to studies that help us intervene on one aspect of this important health problem and cause of racial disparities in health.

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Spending more, feeling worse: medical care expenditures and self rated health

International comparisons show that the United States spends more on health care than other industrialised nations.[1] According to 2000 data, the United States led the way in per capita healthcare spending at $4631, more than double the Organization for Economic Cooperation and Development (OECD) median of $1983 (in purchasing power parities based on the US dollar).[2]

Despite massive medical care expenditures, the US lags behind its industrialised counterparts in major indicators of population health. For example, researchers have reported that the US has lower life expectancy at birth and higher maternal and infant mortality.[3 4] Equally important, cross national studies consistently show that US citizens are less satisfied with their healthcare system than Canadians or Europeans.[2]

Another key indicator of population health, not previously used in cross national comparisons, is self rated health (SRH). As a broad measure of health status, SRH focuses on a person's subjective perceptions of their

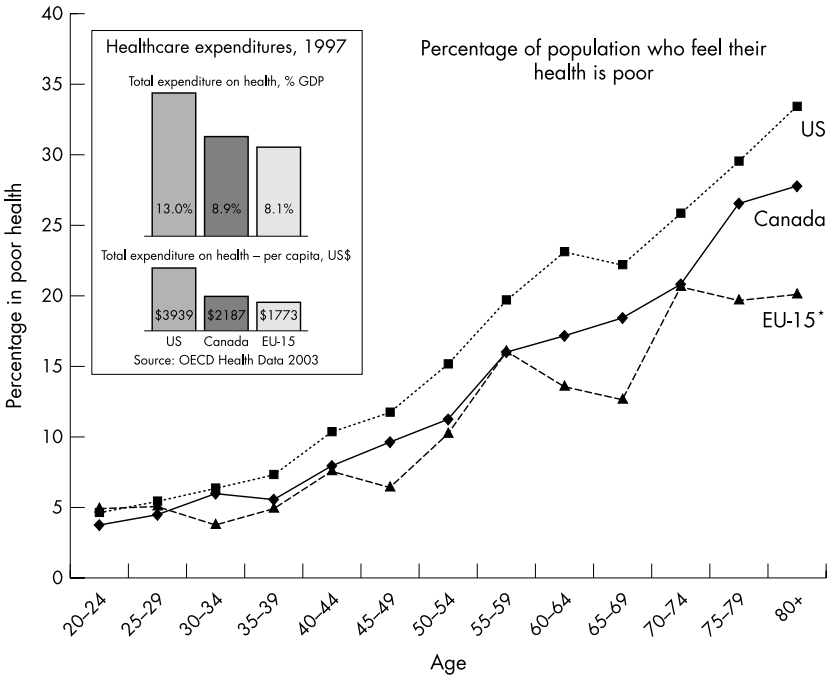

Figure 1 National healthcare expenditures and self rated health. Data sources: National Population Health Survey 1996–97 (Canada), National Health Interview Survey 1996 (US), and European Commission Eurobarometer 1996 (EU-15). *Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Luxembourg, Netherlands, Portugal, Spain, Sweden, and United Kingdom.

health. This popular single item measure on both sides of the Atlantic is related to mortality and to rates of recovery from episodes of illness across the life span.[5] Drawing on comparable datasets, we asked whether the prevalence of poor self rated health is higher in the US than in Canada or the European Union.

Data (weighted) were obtained from the 1996/97 Canadian National Population Health Survey—NPHS (n = 66 435), the 1996 US National Health Interview Survey—NHIS (n = 63 402), and the 1996 European Commission Eurobarometer (n = 16 235). Age specific self rated health was assessed using the NPHS and NHIS questions "In general would you say your health is excellent, very good, good, fair, or poor?" In the Eurobarometer, self rated health was assessed with "Over the last 12 months, would you say your health has on the whole been very good, good, fair, bad or very bad?" In our analysis, "poor health" refers to responses of "fair" or "poor" in the NPHS and NHIS and to "bad" or "very bad" in the Eurobarometer. Data for national healthcare expenditures were obtained from the OECD (http://www.oecd.org/health/healthdata).

Figure 1 shows that the US, Canada, and the European Union (EU-15) differ in terms of the prevalence of poor SRH. While outspending Canada on a per capita basis by 1.8 to 1 and the EU-15 by 2.2 to 1,[6] the US had higher prevalences of poor self rated health across the age span.

Overall, Canadians and Europeans spend about half of what Americans spend on health care yet feel better in nearly all age categories. As an approximate significance test for differences in average SRH, we computed t values pooling the standard errors across studies. The results indicated there were no significant differences between samples at the youngest age groups, but for middle age and older groups, beginning with age 30, Americans had significantly ($p<0.05$) higher prevalence of poor SRH. Interestingly, disparities in SRH seem to increase with advancing age, underscoring the need for better care and more complete coverage across the entire life span.

Our study confirms and complements earlier research indicating that the expensive and technologically advanced healthcare system in the US does not yield population health outcomes (including SRH) comparable to those in countries with much lower spending whether measured per capita or as a percentage of gross domestic product (GDP).[2 3 7] Unequal and uncoordinated provision of care along with other inefficiencies in the US health system[7] may explain why Americans spend more but feel worse.

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BOOK REVIEWS

Reducing the burden of headache

Edited by J Olesen, T J Steiner, R B Lipton. Oxford University Press, Oxford, 2003. (Pp 425; £85.00). ISBN 0-19-851589-8

Although not specifically stated this book represents the proceedings of the 11th international headache research seminar held in Copenhagen in March 2002. This 11th volume in the series *Frontiers in Headache Research* aims to bring the reader up to date on studies of the burden of headache. All the books in the series follow a similar structure: "Each topic is introduced by one or more overview chapters by leading experts. Thereafter, data from new studies are presented in short articles. Finally, each topic closes with a summary of discussion by the chairpersons."

This volume of 68 chapters is divided into six sections. In the preface the editors state that migraine, according to WHO, is number 20 of all diseases with regard to years lived with disability. They also state that migraine has more impact on quality of life than many other diseases generally considered to be more serious. In section I and II, headache related disability and quality of life are discussed in more detail. Section III deals with family burden, comorbidities, and healthcare utilisation for headache. Section IV discusses economic aspects of headache, whereas the two last sections describe and analyse guidelines and intervention aimed at improving health care for headache.

The studies presented at the 2002 Copenhagen meeting, and subsequently published in this volume, are described comparatively briefly and with variable quality. The most interested readers will probably need to search for more detailed information from

those studies also published elsewhere. However, an interesting summary discussion highlights key points at the end of each section. From my point of view, this book should be on the reading list of everyone involved in headache research, and may also be of interest to epidemiologists as well as primary healthcare workers.

K Hagen

The making of a global controversy

Edited by M W Bauer and G Gaskell. Cambridge University Press, Cambridge, 2002. (Pp 411; $75.00). ISBN 0-52177-317-2

Experts in different social and scientific fields have been coordinated by editors Martin W Bauer and George Gaskell with the aim to analyse, from different angles, one of the more important and dynamic socioeconomic phenomenon of the recent years: the biotechnology. As the editors expose in the volume presentation: ''*This book takes up themes explored at a conference at the Science Museum London, in 1993, which was convened to explore the structures and functions of resistance in the development of new technologies*''. The volume holds the main thesis of the conference, which could be resumed as follow: ''*resistance is not a problem of the public opinion rather it is a signal that acts as a catalyst for organisational and institutional learning*''.

The book comprises a total of 13 chapters gathered in five parts, which include the results of a four year international research project conducted between 1996 and 1999 and called ''Biotechnology and the European public''. In part I, named ''The framing of a new technology: 1973–1996'', the authors go over bioethics debate and biotechnology regulation during the past 25 years in Europe, throughout five contributions. Part II, deals with ''Public representation in 1996''; it includes four chapters about traditional blue and green resistance, the structure of public perceptions, and the image of the genetic engineering such as a way of nature manipulation. Part III highlights ''The watershed years 1996–97'' describing two emblematic examples: the modified soya and the cloned Dolly. In part IV, and under the attractive title of ''The transatlantic puzzle'', the authors analyse how genetically modified seed, food, and pharmaceuticals became a reality, in which great companies like Mosanto emerged from USA, although when extended to Europe, they found a controversial socio-political atmosphere. Finally, part V includes a unique chapter dealing with the biotechnology movement that integrates all the book contents under the highline ''Towards a social theory of new technology''.

In short, the book attempts to highlight a social representation about biotechnology and public opinion in occidental countries during the past decade. Appropriately, its contents could be resumed as ''*the making of a global controversy*''.

M A Peinado

CORRECTION

doi: 10.1136/jech.2003.011296corr1

Two typographical errors in the equations occurred in this paper by Drs Choi and Pak (2003;**57**:831–5).

Wrong equation	Correct equation
$C_{t\cdot i}$	$C_{t\cdot i} = R_0^t$.
$D_{t\cdot i.d} = C_{t\cdot i}$ X F.	$D_{t\cdot i+d} = C_{t\cdot i}$ X F.

The complete set of correct equations is therefore:

PREDICTING THE NUMBER OF SARS CASES

Mathematically, the predicted number of incident cases on day t·i, that is, $C_{t\cdot i}$, where t is time expressed in the number of incubation periods, is

$$C_{t\cdot i} = R_0^t.$$

The predicted total number of cases (C) is

$$C = \Sigma C_{t\cdot i}.$$

PREDICTING THE NUMBER OF SARS DEATHS

Mathematically, the predicted number of deaths on day t·i+d, that is, $D_{t\cdot i+d}$, where t is time expressed in the number of incubation periods, is

$$D_{t\cdot i+d} = C_{t\cdot i} \text{ X F}.$$

The predicted total number of deaths (D) is

$$D = \Sigma D_{t\cdot i+d}$$