This, first issue of the *International Journal of Epidemiology* (*IJE*) for 2015 is a lively, diverse and stimulating, if occasionally contentious, read. Undoubtedly the same will be said of the next two issues for this year; April and June: special themed issues on recent advances in the fields of epigenetic epidemiology and Mendelian randomization respectively. Although these issues are closed for submissions, a special themed issue on another recent development, metabolic phenotyping in epidemiology, is open for submissions to the 30th June 2015—see the *IJE* website (http://ije.oxfordjournals.org/) for details; and further information about a special themed issue on life course epidemiology and ageing, open for submissions to the 31st July 2015, can be found in this issue of the journal.1

Although February is a general issue, many of its contributions could easily coalesce around the theme ‘the association between evidence and policy’. Two organizations for which this association is their raison d’être are the Cochrane Collaboration and the Campbell Collaboration.

### The evidence evaluators

The Cochrane Collaboration, founded in the UK in 1993, is named after Archie Cochrane (1909–1988), a Scottish epidemiologist and vociferous advocate of the use of randomized controlled trials as a means of informing healthcare practice (Photo 1). Since it was set up, Cochrane has grown to include collaborators from over 120 countries. Its aim is to use systematic reviews of randomized controlled trials to evaluate healthcare interventions unbiased by commercial and other conflicts of interest. Over the past two decades, thousands of Cochrane reviews, 525 on cancer alone, have been made available free of charge via the Cochrane Library to inform the choices made by health professionals, patients, policy makers and others according to the principles of evidence-based medicine.

The Campbell Collaboration, founded in 2000, is named after Donald T Campbell, an American social psychologist and advocate of the view that governments should develop an experimental approach to social reform (Photo 2). Like Cochrane, with which it cooperates, the Campbell Collaboration undertakes systematic reviews. However, whereas Cochrane’s focus is health, Campbell’s focus is social policy, including education and justice outcomes. One of Campbell’s earliest and most influential systematic reviews in the justice field is an evaluation of the ‘Scared Straight’ programmes in the USA. Scared Straight is designed to deter participants from future offending through visits to prisons and interaction with adult inmates. First published in 2002 and most recently updated in June 2012, the review, by Anthony Petrosino and colleagues, established that participation in the Scared Straight programme is linked to higher, not lower, rates of offending.2

### Let the punishment fit the evidence

Petrosino’s findings would come as no surprise to John Palmer, author of the classic reprint in this issue of the *IJE*—Smoking, caning, and delinquency in a secondary modern school—or to Archie Cochrane who suggested and encouraged the study.3 Palmer worked with Cochrane in the Medical Research Council Epidemiological Research Unit in Cardiff from 1962 to 1965. In one of the secondary schools in the locality, the headmaster kept a record of every boy he had caned for smoking. During the school year 1961–62 this punishment was administered to 23 boys. As these boys were in the 2nd, 3rd and 4th forms, roughly covering those aged 12–13, 13–14 and 14–15
years, respectively, Palmer included all 147 boys from these forms in his study. He questioned the boys about their smoking habits in September 1961 and at the time of interview, November 1962. These data showed that smoking among the boys who were caned increased, whereas among those who were not caned it decreased. In May 1964, as part of a separate, much larger study of juvenile offences, Palmer examined police records to determine the prevalence of delinquency. This study included 146 of the 147 boys in his original study of smoking and caning. Although prevalence of smoking was high, at 26%, and the boys with a police record were heavier smokers, he found no evidence of an association between being caned at school for smoking and delinquency. Discussing the limitations of his study, Palmer suggests that randomized controlled trials are required to determine the reformative value of punishments like caning. In a paper published 2 years later, Palmer reported on a randomized controlled trial he conducted to compare the relative effectiveness of a verbal reprimand vs detention or extra work for the prevention of late arrival at school. Although the trial was too small to provide firm evidence, Palmer used it to emphasize points made in his earlier paper, that research using randomized controlled trials of the effectiveness of punishments in schools and in the penal system is both feasible and desirable.4 This point was reiterated by Petrosino and colleagues 50 years later: ‘Agencies that permit such programs [Scared Straight], however, must rigorously evaluate them not only to ensure that they are doing what they purport to do (prevent crime)—but at the very least they do not cause more harm than good to the very citizens they pledge to protect’.2

Evidence and public health policy

An editorial on the Campbell Collaboration in the Cochrane Library compares Cochrane and Campbell to the boy with his finger in the dyke against a rising tide of ineffective, or unproven, interventions.5 Based on rough calculations, John Bridgeland and Peter Orszag, officials in the Bush and Obama administrations, respectively, have suggested that less than US$1 out of every US$100 spent by the US government is backed by even the most basic evidence that the money is being spent effectively—decisions being based largely on good intentions, inertia, hunches, partisan politics and personal relationships.6

Policies most certainly based on good intentions are analysed and discussed in the section on Public Health Policy in this issue of the journal.7,8 In the wake of decisions by the United States Preventive Services Taskforce to retract recommendations in favour of breast cancer screening for women aged 40–49 and prostate cancer screening in healthy men, Nazmus Saquib and colleagues have used the Taskforce, Cochrane and PubMed databases as sources of high quality evidence to determine whether screening for nine major diseases, mostly cancers, in asymptomatic adults actually saves lives.7 There is ongoing debate as to whether cause-specific or all-cause mortality is the most appropriate metric for the evaluation of screening programmes; Saquib and colleagues report both, where available, for 45 individual randomized controlled trials and 8 meta-analyses. Their comprehensive analyses provide evidence of a reduction in mortality in only 30% of the disease-specific mortality estimates and 10% of the all-cause mortality estimates from the randomized controlled trials they evaluated. In the case of disease-specific mortality, findings from the individual randomized controlled trials are backed by evidence from meta-analyses, but none of the six meta-analyses that included estimates of all-cause mortality produced any evidence of reduction.7 Given that almost all screening procedures have some iatrogenic effects, commentaries on the paper emphasize the importance of demonstrable benefit if screening is to be applied to large proportions of the general population. Peter Gøtzsche, a well-known critic of screening and general health checks,9–11 laments the lack of consideration given
to the harmful by-products of screening. He argues that quantification of this harm should be the main focus in screening trials, and that the benefits of screening be measured in terms of all-cause mortality. While conceding that death is an important outcome, Paul Shekelle makes the point that it is not the only outcome. Even in the absence of effects on mortality, patients may value screening tests that decrease the risk of serious morbidity. Consequently, Shekelle argues for the inclusion of serious morbidity and patient preferences in evaluations of screening—outcomes currently more noted for their absence. Paul Taylor, who elsewhere has presented a detailed and fascinating review of the debate on breast cancer screening, is also keen not to throw the baby out with the bath water. At the end of his article on the breast cancer debate, he concludes that ‘The question of whether or not breast screening should continue will be determined by politicians’ assessments of what the public wants’. Disappointing though this may be, it is unlikely to deter the Cochrane and Campbell Collaborations whose mission it is to turn the tide.

In their review of vitamin A policies, John Mason and colleagues assert that the remarkably slow decline in vitamin A deficiency in low- and middle-income countries is due mainly to the failure to apply scientific evidence to policy. The authors document how one policy, the administration of 6-monthly, high-dose vitamin A capsules, has come to dominate the approach to treatment. The policy was based on evidence from clinical trials in the late 1980s and early 1990s which showed a 23% reduction in all-cause mortality among children aged 1–6 years. However, the current effectiveness of this policy, estimated to cover more than 80% of 1–5-year-olds in low- and middle-income countries, has been brought into question recently by findings from the only field evaluation to date, which showed no association with mortality. The authors provide a number of potential explanations for the apparent change in efficacy of the treatment and make the case for a phased move towards frequent intakes of vitamin A in physiological doses, such as via the fortification of staple foods: a recommendation first made in 1987. According to Mason and colleagues, the evidence that vitamin A deficiency, redefined by the World Health Organization in 2002 as low serum retinol, can be alleviated by this policy, but suggest that these should include a third arm in which regular, low-dose vitamin A supplements are provided. The drawback of this approach is that it is likely to be another 10–20 years before the results of such trials become available. In the meantime, in another letter to the editor, Mary Schooling and Heidi Jones express concerns that high doses of vitamin A in infancy could have long-term effects on susceptibility to chronic diseases in adulthood.

In addition to the interesting proposition of Schooling and Jones, the Letters section of this issue of IJE includes several contributions on the use and abuse of evidence. Igor Radun and colleagues reiterate and add to concerns about the dangers inherent in the involvement of commercial interests in research on road traffic injury prevention. Originally expressed in a paper by Gareth Davies and Ian Radun and colleagues reiterate and add to concerns about the dangers inherent in the involvement of commercial interests in research on road traffic injury prevention. Originally expressed in a paper by Gareth Davies and Ian Roberts in the October 2014 issue of IJE, such concerns were the subject of the journal’s first appearance on the social microblogging platform, Tumblr (Figure 1). Simultaneously, similar concerns about the involvement of vested interests are raised in an exchange of letters relating to a paper in the December 2014 issue of IJE on Agent Orange exposure and risk of death in Korean Veterans. As their measure of exposure to Agent Orange, the authors used an exposure opportunity index or E4 score. Developed by a team of researchers at Columbia University’s Mailman School of Public Health, led by Steven and Jeanne Stellman, the E4 score is designed to provide a rank-
ordering of exposures based on dates and specific locations in Vietnam. Michael Ginevan and colleagues, consultants to the Dow Chemical Company and Monsanto, the major suppliers of Agent Orange used in Vietnam, have taken issue with the E4 score on previous occasions and do so again in a letter to the Editor published in this issue of the journal. The nub of their concern appears to be that E4 scores are subject to random misclassification bias. In a letter of response, the Stellmans address the detailed methodological points raised by Ginevan and colleagues. However, the authors of the original paper, So-Yeon Ryu and Sang-Wook Yi, point out the more obvious
epidemiological implication that non-differential misclassification of an exposure will bias associations towards the null, meaning that their original findings are more likely to underestimate than overestimate the associations observed between Agent Orange and mortality.26

Evidence of cancer risk, policy and Professor Dimitrios Trichopoulos

Although high birthweight has been established as a risk factor for childhood leukaemia, the associations between birthweight and other childhood cancers is less clear, largely because few studies are of sufficient size to detect associations. In this issue of the *IJE*, Kate O’Neill and colleagues use two large independent datasets to examine the association between birthweight and childhood cancer. They show high birthweight to be associated with an increased risk of neuroblastoma, lymphoma, germ cell tumours, other malignant neoplasms/melanomas, specific subsets of leukaemia, tumours of the central nervous system, renal tumours and soft-tissue sarcomas. Low birthweight on the other hand is associated only with an increase in the risk of hepatic tumours. However, together these cancers and cancer subsets represent approximately half of all childhood cancers. US registry data for nearly 17,000 cases and 54,000 controls produced findings very similar to those observed for nearly 24,000 cases and 33,000 controls from UK registries. Analyses of the US data included adjustment for gestational age, maternal age, plurality, birth order and maternal race/ethnicity and the authors conclude that the apparent independent associations between birthweight and childhood cancers indicate the importance of intrauterine growth.27

O’Neill and colleagues’ results would have delighted Professor Dimitrios Trichopoulos, whose death on the 1 December 2014 we are saddened to note. Vincent L. Gregory Professor of Cancer Prevention and Professor of Epidemiology at Harvard School of Public Health, Dimitrios Trichopoulos will be remembered for his seminal contributions to cancer epidemiology. Perhaps his most consequential contribution is the documentation of the health effects of passive smoking. A case where evidence has been used to inform policy across the globe, Trichopoulos considered himself fortunate to have received recognition and to have seen the results of his work translated into policy in his lifetime. In addition to his work on passive smoking, Trichopoulos is also known for his early major work linking hepatitis B and C and smoking with hepatocellular carcinoma, and for the hypothesis that the origins of breast cancer can be traced back to exposures in utero; a finding he felt had captured less enthusiasm because implications for policy were less obvious.28

Certainly, Trichopoulos’s 1990 *Lancet* paper,29 the first to propose that increased concentrations of estrogens in pregnancy increase the probability of future occurrence of breast cancer in daughters, contributed to the early origins thesis originated by David Barker and colleagues.30 Trichopoulos’s interest in the early origins of breast cancer continued and in 2003 he co-authored a paper in a themed issue of *IJE* on cancer. Using data for Chinese women in China and Caucasian American women in the USA, the paper showed the birthweight difference between China and the USA to be fully explained by height, pre-pregnancy body mass index and weight gain during pregnancy. These findings led the authors to hypothesize that babies in China may have lower birthweight because their mothers’ anthropometry imposes constraints on the growth of the fetus. When young Chinese women migrate to the USA they tend to grow taller and heavier so that their babies reach, on the average, a higher birthweight. The authors speculated that recurrence of this process over successive generations could explain why Chinese Americans eventually tend to have birth and adult anthropometric characteristics, as well as breast cancer rates, comparable to those of Caucasian Americans.31

Inequality—evidence and policy

Much will be written in 2015 about the success or otherwise of the Millennium Development Goals and about the post-2015 agenda for sustainable development. This issue’s interesting and challenging editorial by Alan Lopez is our first contribution to this debate.32 In the draft United Nations Synthesis on the Post-2015 Agenda, the Secretary General, Dr Margaret Chan, describes 2015 as ‘the time for global action. During this single year we have the unequivocal opportunity and responsibility to adopt sustainable development, to restructure the global financial system in line with our needs, and to respond finally and urgently to the challenge of human-induced climate change.’33 Achievement by 2030 of the sustainable development goals listed in Table 1 of the report (Box 1) will be no mean feat.

Point 67 of the report indicates that Goal 1 is in pole position because:

Eradicating poverty by 2030 is the overarching objective of the sustainable development agenda. We live in a world of plenty, and in a moment of enormous scientific promise. And yet, for hundreds and hundreds of millions across the globe, this is also an age of gnawing deprivation. The defining challenge of our time is to close the gap between our determination to ensure a life of dignity for all on the one hand, and the reality of persisting poverty and deepening inequality on the other.33
Someone who has repeatedly expressed concern about deepening inequality is the social geographer Danny Dorling, Halford Mackinder Professor of Geography at the University of Oxford. In his most recent book on the topic, *Inequality and the 1%*, Dorling discusses the increasing gap between the 1% of the population at the top of the income distribution, who have manipulated the corridors of power and the legal system to increase their hegemony on wealth, and what Dorling reports as the increasingly equal 99%. He documents the adverse repercussions for the 99% of these divisive moves in terms of access to housing, health and education. In the book and in the many talks he has given on the topic it seems clear Dorling believes it is time for action. Principally it seems that the 10% immediately below the 1%, who perceive themselves to be increasingly disenfranchised in comparison with the upper echelon, are to be the main actors. Dorling urges them to look down instead of up and make common cause with the remainder of the 99%, but provides little evidence of the potential effectiveness of this strategy. Generally reviews of *Inequality and the 1%* have been positive, although the *Financial Times* noted ‘His new [book] reads like the work of a writer in a hurry’. Clearly he sees the book as setting out an agenda that will do nothing but increase inequality by restricting access to education. Concerned that *G is for Genes* and its theories will be taken seriously in policy circles, Dorling attacks its 11 recommendations, accusing the authors of using poor quality research and ‘because I say so’ arguments to bolster their views. No fan of the way the education system is currently run, the review reads as if written by a man on a mission and in a hurry. The authors of *G is for Genes* declined our offer to respond to the review, one suggesting that we ‘prepare to be embarrassed’. However, even reviewers more sympathetic than Dorling have remarked on the absence of evidence in support of Asbury and Plomin’s ‘big idea’—a unique curriculum for every child delivered by automated teaching, key workers and schools the size of universities. The review in the *Economist*, for one, concludes ‘Automated teaching. Meddling bureaucrats. Giant schools. It might work. But it is unscientific to assert that it would.’

**References**

2. Petrosino A, Turpin-Petrosino C, Hollis-Peel ME, Lavenberg JG. Scared straight and other juvenile awareness programs for


15. Taylor P. Commentary: Tempering expectations on screening: what is the most authoritative advice we can give, given the data that we have? Int J Epidemiol 2015;44:280–82.


