EDITOR'S CHOICE

How do we know, what do we know and what can knowledge do? From John Brownlee to translational medicine

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At our recent annual associate editors meeting we discussed whether the IJE should follow most other general medical and leading epidemiology journals in releasing press releases to publicise particular papers. One associate editor (not a natural media tart or self-publicist) is enthusiastic about press releases, and thinks that the IJE may not be helping authors’ careers and their institutional standing in not routinely engaging in promotion of the material appearing in its pages. Receiving coverage in the popular press certainly increases the citations in the scientific literature that a paper receives, as demonstrated by the study showing that scientific papers covered in the New York Times were cited considerably more often in the subsequent 10 years than papers not covered in the New York Times.1 The particularly elegant aspect of this study was that during a 3 month strike at the New York Times the paper was not released to the public, but an “edition of record” was still produced by the journalists; receiving coverage in the newspaper during this period was not associated with increased citations, indicating that it was not simply that the New York Times reported on the papers that would in any case have been cited more often.

Our unease with “press-release epidemiology” reflects the fact that it is rarely the case that any paper that we (or, let’s face it, other epidemiology journals) publish definitively demonstrate things that are of concern to the public, while often raising issues that, with popular press coverage, could represent yet more scare-mongering. Our reticence in this regard might adversely affect our impact factor, and while we obviously think the papers concerned deserve publication in a scientific journal, could we envisage headlines reading “gene for cardiac arrest found”,2 “green tea stops stroke”,3 “fish makes you live longer”,4 “get a sun-tan to avoid cancer”,5 “tea, fish and fruit keep you cancer-free”,6 “losing weight can be fatal”7 and “hot and cold both kill”8 in the newspapers? A concern with how epidemiological methods can be improved to strengthen inference is seen in several papers in this issue,9–11 but the fact that we have clearly expressed disagreement in commentaries regarding the appropriateness of approaches in two cases9,12,13 demonstrates the dialectical nature of epidemiological discourse. Probably rather too much unfinished epidemiological business already appears in news stories, without us throwing our oar in.

One example of the long gestation of epidemiological knowledge appears in our historical reprint, a translation of Kristian Feyer Andvord’s 1930 work on the generation effect in tuberculosis.14 Andvord pointed out that the experience of TB among successive birth cohorts was compatible with primary infection occurring in early life and mortality at later ages reflecting reactivation of earlier infection; the “once infected, always infected” hypothesis.15 Oyvind Næss and colleagues point out how Andvord’s identification of what are now called “cohort effects” in tuberculosis reflected his thinking about the aetiology of the disease in general, something that he had developed over several decades.16 His work on this issue perhaps became more widely known because he published one paper on it in German, whereas most of his work was published only in Norwegian. Our commentators15–18 demonstrate that there is still debate regarding the relative roles of re-activation and re-infection in accounting for the population dynamics of TB.

Næss and colleagues rightly point out that the purpose of reflecting on now-historical epidemiological contributions is not simply to right apparent wrongs with respect to credit and priority. They show this with respect to Andvord’s complex ideas regarding tuberculosis, some of which would now be accepted and some of which would now appear eccentric. The same certainly applies to the work of the medical statistician John Brownlee, for whom priority in describing cohort or generation effects in tuberculosis—back in 1916—has been claimed.19,20 Brownlee’s paper does clearly contain these ideas, and also identifies the mid-19th century as representing the high-water mark with respect to TB infection in early life, something which later data analyses supported. However it is a paper from which it is difficult to draw straightforward conclusions, and in
the discussion that followed it a Major Butler commented that “Dr Brownlee had spoken in a language only his own, and was difficult to follow. He had produced a minimum amount of arithmetic in the curves of the diagrams, and it was difficult to understand this or that construction he put on these curves”.

John Brownlee, originally a medical practitioner, became founding director of the statistics department of the UK Medical Research Committee (later Council; MRC) in 1914. He therefore held a key position in integrating statistical with clinical and biomedical thought. He was perhaps not constitutionally suited to this role, however, and many seemed to agree with Major Butler. In a letter to Sir Walter Fletcher, the head of the MRC, the Cambridge statistician George Udny Yule likened Brownlee to “an odd sort of fly which you often see in a garden on a hot day in summer: it seems to be nearly still, hovering over a rose-bush say, for seconds together, when suddenly flick! and the blessed fly is in quite a different position, and you can’t think how in Hell it ever got there. I know that my mind sometimes seems to work like that, but Brownlee’s is much worse. Place-names in Aberdeenshire and on the Black Sea, the occurrence of elephants in Scotland, the whisky-drinking habits of one of his mother’s trustees, the price of Turner drawings, the existence of areas where illegitimacy seems to be the normal thing in Scotland...you wouldn’t think, would you? that so many subjects could arise really naturally out of a short conversation on the work of the Anthropometric Standards Committee?”. On hearing that Brownlee might leave his post Fletcher declared this “a bright ray of light for the new year”.

We will endeavour to reprint one of Brownlee’s papers in the *IJE* at some stage, but clearly he had a problem with translation—translating between statistical and other forms (clinical, public health) of reasoning. “Translation” is certainly the buzz word *du jour* in medical science, with the need to move across levels of knowledge-generation to practical implementations that improve health being seen as key. Evaluation of how research impacts health is demanded by funding bodies and governmental agencies alike. And rightly so. As Karin Michels points out, population strategies for prevention, even when they seem to be based on sound scientific evidence, can prove problematic. She discusses compulsory folic acid fortification, and Jennifer Dowd and Allison Aiello demonstrate that even the apparently simple question “did compulsory folic acid fortification reduce disparities?” is problematic; it depends whether you consider absolute or relative differences to be more important. In terms of moving from science to policy, however, pragmatic decisions need to be made, something brought home clearly in the important paper from New York City Commissioner of Health Thomas Frieden and colleagues, which documents how public health policy making can be made in an early 21st century metropolis.

“Translational medicine” has become an obligatory point of passage in talking about health and medicine, but we have to admit here at the *IJE* that we are somewhat uncertain as to its implications for epidemiology. Therefore our associate editor, Lyle Palmer will be convening a themed issue on “what is translational epidemiology?” to allow debate of this issue. We welcome expressions of interest or submissions on this topic.


**References**


12 Willett: Commentary: Flawed study designs are not salvaged by large samples. Int J Epidemiol 2008;37:987–89.


