Having recently returned from a scientific meeting at Arusha, near Kilimanjaro in Tanzania my attention was attracted to a letter in this issue on male circumcision and risk of HIV infection.1 One of the Ugandan participants at our meeting had spoken about the depressing experience of the Rakai research group’s thoughtful and well-coordinated attempts to turn their trial findings2 into national policy. Despite consistent findings of a halving in HIV incidence from three recent trials of circumcision among heterosexual men in sub-Saharan Africa, there does not seem to be much appetite for translating these findings into health policy. The social scientists participating in the meeting were, of course, not at all surprised by this policy inaction and were somewhat surprised by the naivety of trialists in expecting any direct linkage between evidence and action. Our correspondents state that two decades of observational epidemiology have shown the beneficial effect of male circumcision, although there was some debate about the consistency of the observational findings at our meeting in Arusha. I was prompted to check this out. The authors of a systematic review and meta-analysis published in 2000 were sufficiently impressed by the evidence to conclude that: ‘…consideration should be given to the acceptability and feasibility of providing safe services for male circumcision…’.3 However, a subsequent systematic review (adding additional data) was much more impressed with the heterogeneity of effects, concluding that: ‘conducting a meta-analysis was inappropriate’.4 Our correspondents make the important point that the trial findings cannot be generalized to men who have sex with men, and present a systematic review of the two relevant observational studies showing a (consistent) halving of HIV risk, in line with the trial findings among heterosexual African men.

Our Tanzanian meeting, set up by the Wellcome Trust, was about prospective cohort studies and how to make best use of the resources invested in them. Most large cohort studies are located in affluent countries, raising the question of how best to mount large-scale studies in developing countries where resources for long-term funding are difficult to obtain. In this issue, one of our cohort profiles focuses on a new Thai cohort of over 87,000 participants recruited from Thai Open University students.5 This study was supported under a unique collaborative co-funding scheme between Wellcome Trust, Australian National Health & Medical Research Council and New Zealand Health Research Council, which aimed to promote research collaborations between partners in the South East Asian and Western Pacific regions. The Thai study is examining the ‘health transition’—shifts in social and cultural patterns of behaviour, improved child and adult survival prospects and associated epidemiological changes. Making use of the Open University was the stroke of genius in this study that assured its funding. The benefits are of a ready-made nationally based sampling frame of all enrolled students, a wide age-range of participants (nearly half are over the age of 30, range 15–87) and a means of following them up through the Open University for the several years that most students take to complete their degrees. I am sure it was hoped that participation rates would be enhanced by making completion of the 20-page questionnaires a condition of progression through the Open University. Presumably this was not possible, but a respectable 44% participation rate was achieved. The investigators have processed 10 tonnes of questionnaires and have created a clean database. The first follow-up in 2008 will be the real test of the utility of using this approach.

Large cohorts are needed to study the effects of genetic variants on common disease, and UK BioBank is now moving into its data collection phase. Collecting blood in large-scale studies is a major undertaking—and I suspect the Thai cohort may wish they had considered the collection of blood (or at least saliva) in their participants—requiring careful protocols. In this issue, Paul Elliot and Tim Peakman explain how they went about blood sample handling and storage6 and an IJE supplement giving details of all the relevant protocols accompanies the print version of the IJE and is available at http://ije.oxfordjournals.org. Anyone contemplating setting up even modest sized cohorts would do well to see how cutting edge technology can be brought to bear on practical problems.

Socio-economic differences in blood levels of C-reactive protein (CRP), an acute phase inflammatory marker, are large and confound associations between C-reactive protein and coronary heart disease.7 Several studies have shown life-course socio-economic position is associated with increased

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coronary heart disease risk and, if CRP is causative, an interesting question is at what age does the socio-economic patterning of CRP levels begin? Gimeno and colleagues have used the Young Finns cohort study to examine this question and show that CRP levels measured in childhood (ages 3–9) and adolescence (age 12–18) are not socially patterned but this appears to examined in adulthood (ages 24–39). Casas and Hingorani, commenting on this paper, note that as genetic factors may contribute to the social patterning of adult mortality, it is possible that the CRP gene might be involved. Gimeno and colleagues did not find any evidence of association between socio-economic position and different CRP polymorphisms. This lack of association also indicates that assortative mating by socioeconomic position does not explain social variation in CRP levels.

Some hypotheses are simply too seductive to die. Does personality predict mortality? Ask investigators using data from the GAZEL French prospective study. The 1980s saw the Type A personality as a coronary risk predictor fail to replicate only to be replaced by the ‘hostility’ component, which has also met the same fate. This study examined the effects of 10 different personality traits—ranging from ‘neurotic hostility’ to ‘CHD-prone’, ‘cancer-prone’ etc. Perhaps unsurprisingly with so many exposures, a few findings survived a fairly modest attempt at adjustment for confounders (age, sex, educational attainment and marital status). ‘Neurotic hostility’, ‘ambivalent’ and ‘anti-social’ personalities were all associated with all-cause mortality which, I think, makes it difficult to come up with a coherent theory of causality. ‘CHD-prone’ personality was indeed associated with cardiovascular mortality (only 72 events for analysis) but no personality type was associated with cancer mortality, not even ‘cancer-prone’. The most plausible findings were that ‘anti-social’ and ‘neurotic hostility’ personality types were associated with mortality from external causes. Some people just have personalities that make their lives dangerous.

Finally, chronic diseases in developing countries have fallen off the global health agenda owing to staggering amounts of money, and therefore policy, health services and research interest, being focused on HIV, malaria, TB and child survival. These priorities are totally understandable but the rise in chronic diseases is attributable, in large measure, to success in reducing infant mortality and the control of many infectious diseases. Communication about the rise in chronic diseases is couched in terms of burden, problem and challenge—which of course it is—rather than as a consequence of the successful organisation of society. But these negative words may be the undoing of the recent call to action mounted by the Lancet. In a detailed editorial, I explain my personal views on calls to action (overused) and, nonetheless, my support for taking actions to prevent chronic diseases.

**Conflict of interest:** None declared.

**References**