

can technically reproduce the procedure. Furthermore, and most importantly, each sample was normalized to a control DNA sample to minimize the inter-assay variability. The inter-assay CV as determined from this control DNA sample was 2.4%. The major focus of our work was to introduce a well-controlled high-throughput genotyping method to obtain reliable estimates for relative telomere length with low amounts of DNA. This is very similar to the performance of a high-quality enzyme linked immunosorbent assay (ELISA) for measurement of standard biomarkers.⁵

With respect to the reliability of the qPCR approach in general, we would like to point to another very recent article on telomere attrition rates over 10 years, which had a very similar study design: Nordfjäll *et al.*⁶ investigated the dependency of the telomere attrition rate on the baseline telomere length and found a correlation coefficient nearly identical to ours ($r=0.752$ in Nordfjäll *et al.*⁶ versus $r=0.674$ in Ehrlénbach *et al.*²). This is another strong indication that the qPCR approach is highly reliable, valid and replicable. In addition, recent papers in high-ranking journals (e.g. *Lancet*³, *PLOS Genetics*⁶) on telomere dynamics used the qPCR approach instead of the TRF methodology for association studies, thus indicating that qPCR is becoming a state-of-the-art technology for inferring relative telomere length and associated phenotypic consequences.

We certainly respect that researchers like Dr Aviv passionately advance their view. However, 'raising the bar on telomere biology' does not necessarily mean sticking to old, but still valuable, methods,

but to take a step forward towards new technologies, thus enabling assessment of novel risk conditions in a large number of epidemiological studies.

References

- 1 Aviv A. Commentary: Raising the bar on telomere epidemiology. *Int J Epidemiol* 2009;**38**:1735–36.
- 2 Ehrlénbach S, Willeit P, Kiechl S *et al.* Influences on the reduction of relative telomere length over 10 years in the population-based Bruneck Study: introduction of a well-controlled high-throughput assay. *Int J Epidemiol* 2009;**38**:1725–34.
- 3 Brouillette SW, Moore JS, McMahon AD *et al.* Telomere length, risk of coronary heart disease, and statin treatment in the West of Scotland Primary Prevention Study: a nested case-control study. *Lancet* 2007;**369**:107–114.
- 4 Pfaffl MW. A new mathematical model for relative quantification in real-time RT-PCR. *Nucleic Acids Res* 2001;**29**:e45.
- 5 Kronenberg F, Lobentanz EM, König P, Utermann G, Dieplinger H. Effect of sample storage on the measurement of lipoprotein[a], apolipoproteins B and A-IV, total and high density lipoprotein cholesterol and triglycerides. *J Lipid Res* 1994;**35**:1318–28.
- 6 Nordfjäll K, Svenson U, Norrback KF, Adolfsson R, Lenner P, Roos G. The individual blood cell telomere attrition rate is telomere length dependent. *PLoS Genet* 2009;**5**:e1000375.

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Chronic disease prevention: the importance of calls to action

From PERVIZ ASARIA,¹ ROBERT BEAGLEHOLE,^{2*} DAN CHISHOLM,³ THOMAS A GAZIANO,⁴ RICHARD HORTON,⁵ STEVEN LEEDER,⁶ STEPHEN S LIM,⁷ COLIN MATHERS,⁸ SRINATH REDDY,⁹ KATHLEEN STRONG¹⁰ and JANET VOUTE¹¹

¹Department of Cardiology, Chelsea and Westminster Hospital, London, UK, ²University of Auckland, New Zealand, ³Department of Health Systems Financing, World Health Organization, Geneva, Switzerland, ⁴Brigham & Women's Hospital, Harvard Medical School, Boston, MA, USA, ⁵Editor, *The Lancet*, London, UK, ⁶The Australian Health Policy Institute, The University of Sydney, Sydney, Australia, ⁷Institute for Health Metrics and Evaluation, University of Washington, Seattle, WA, USA, ⁸Department of Epidemiology and Burden, World Health Organization, Geneva, Switzerland, ⁹Public Health Foundation of India, New Delhi, India, ¹⁰UNITAID, Geneva, Switzerland and ¹¹World Heart Federation, Geneva, Switzerland.

*Corresponding author. University of Auckland, New Zealand. E-mail: r.beaglehole@auckland.ac.nz

We welcome the stimulating editorial by Shah Ebrahim on our call to action to scale up the prevention and control of chronic diseases.¹ It is an important contribution to efforts to ensure that chronic diseases receive the global and national attention commensurate with their enormous health and economic burdens, especially in low- and middle-income countries.²

Our call for action is 2-fold: an urgent call to implement policies where we already have the evidence on intervention cost-effectiveness (salt reduction, tobacco control and a multiple drug regime for managing high cardiovascular risk); and a similar call to collect rigorous evidence on other beneficial interventions with the potential for making a difference in populations. Regarding the latter, the editorial confirms

our assessment that the absolute effect size of many interventions such as community-based health promotion and changes in saturated and trans-fat intake needs to be confirmed. This is why we called for more rigorous evaluations of these interventions instead of their outright scaling up—an important distinction. Urgent operational research is needed now to identify the best ways to implement effective interventions in communities.

However, there is much in the editorial with which we agree. Population growth and ageing are key driving forces of the chronic disease pandemics. The point is clearly made in Figure 2 of the first paper in the *Lancet* chronic disease series.² We note that for the age group of >70 years, the proportion of deaths due to chronic diseases is projected to rise from 87.5% in 2005 to 90.9% in 2030. We agree too that the engagement of clinicians in the chronic disease response will be critical, especially as the health systems strengthening agenda and the reinvigoration of primary health care gather momentum.

Ebrahim questions the affordability of the interventions proposed in the series^{3,4} by contrasting the projected investment needed to implement the selected interventions across 23 low- and middle-income countries (\$58 billion over 10 years) with the economic productivity gains associated with reducing chronic disease deaths annually by an additional 2% (an estimated \$8 billion over 10 years). However, these estimates are extremely conservative because the major share of the benefits of increased chronic disease investment comes in the form of averted or 'delayed' deaths (an estimated 32 million deaths over 10 years). Since we consider health to have intrinsic value to society, we did not quantify these societal benefits in dollar terms, but we would only need to agree that each death averted/life saved is worth at least \$2,000—not hard when the value of life has been put at as much as 100 times GDP per capita—for the health system investment to pay itself back.

Further, the view that it is especially difficult to make reallocations in countries that only commit 2–3% of GDP to health services does not mean it cannot be

done or should not be attempted. Indeed, the case of HIV/AIDS shows well what level of budgetary reallocation can and have been achieved in resource-poor countries. In fact, it is imperative that allocative decisions in such countries are made using the best evidence available to maximize health. Furthermore, countries can and have substantially increased health spending. Reforms in Mexico will increase public spending on health by a full percentage point of gross domestic product over 7 years.⁵ The 2008–09 budget in India has committed to increasing health spending by 15%.⁶

Finally, on the importance of calls to action, *The Lancet's* experience is that these can help transform the global policy landscape provided they are based on sound data, trigger new collaborations based on trust and confidence and are followed up with events that publicly track and report on country progress with agreed indicators.⁷

References

- 1 Ebrahim S. Chronic diseases and calls to action. *Int J Epidemiol* 2008;**37**:225–30.
- 2 Abegunde DO, Mathers CD, Adam T *et al*. The burden and costs of chronic diseases in low-income and middle-income countries. *Lancet* 2007;**370**:1929–38.
- 3 Asaria P, Chisholm D, Mathers C *et al*. Population-wide interventions to prevent chronic diseases. *Lancet* 2007;**370**:2044–53.
- 4 Lim SS, Gaziano TA, Gakidou E *et al*. Preventing chronic disease in high-risk individuals: health impact and costs. *Lancet* 2007;**370**:2054–62.
- 5 Frenk J. Bridging the divide: global lessons from evidence-based health policy in Mexico. *Lancet* 2006;**368**:954–61.
- 6 The 2008–09 budget in India. Available at <http://india.budget.nic.in> (Accessed February 5, 2008).
- 7 Horton R. The coming decade for global action on child health. *Lancet* 2006;**367**:3–5.

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Chronic diseases and calls to action

From G. DE BACKER^{1*} and M KORNITZER²

¹Department Public Health, Ghent University, Ghent, Belgium and ²Prof. em. Brussels Free University, Brussels, Belgium.

*Corresponding author: University Hospital, DePintelaan 185, Ghent B 9000, Belgium. E-mail: guy.debacker@ugent.be

Within his lengthy editorial 'Chronic diseases and calls to action'¹ devoted to chronic diseases prevention and health promotion in low- and middle-income

countries. Shah Ebrahim questions the scientific evidence for the population strategy in the prevention of coronary heart disease within a rather provocative