"Closing the gap" by 2030: aspiration versus reality in Indigenous health

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ABSTRACT

- The goal of "closing the gap" in life expectancy between Indigenous and non-Indigenous people by 2030 is probably unattainable.
- Despite our best efforts, it is implausible that, within 21 years, preventive strategies, social or medical, will extinguish all excess expression and risk of chronic disease, the greatest contributor to excess Indigenous deaths.
- Developing systems to supply optimal primary care, as we currently know it, will take time. In addition, we have an incomplete understanding of the nature of excess risk, and lack remedies to totally contain it. Furthermore, vertical imprinting of excess risk will take some generations to ameliorate.
- To avoid failure by specifying unattainable goals, emphasis should be given to process measures that will lead to better outcomes.
- It is self-evident that sustained change requires better education, nutrition, employment opportunities and infrastructure.
- Within the health system, access to good quality, integrated primary care, needs-based health services funding, and an urgent and intensified focus on areas with the highest mortality rates, are top priorities.

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Most Australians support the Labor Government's resolve to improve the health and welfare of Indigenous Australians. This includes an explicit intent to "close the gaps" in health status and life expectancy between Indigenous and non-Indigenous Australians by the year 2030.[1] Notwithstanding the recent downward trend in Indigenous mortality rates,[2] the goal of closing the life expectancy gap by 2030 — whether this means a convergence in life expectancy in those alive in 2030, or in those born in 2030 — is probably unattainable. It implies that, by 2030, Indigenous people will have the same level of risk and disease as non-Indigenous people, or that interventions will be able to eliminate the expression of all excess risk. Both these scenarios are implausible. ABC journalist Kerry O'Brien expressed doubt about the life expectancy time line soon after the announcement,[3] and Professor Jon Altman and colleagues from the Centre for Aboriginal Economic Policy Research, Australian National University, consider that complete convergence of socioeconomic outcomes within the stated time frame is also unlikely.[4]

The life expectancy of non-Indigenous Australians is the second longest in the world, and is still increasing.[2] The accrual of the last additional 25 years of life expectancy occurred over 100 years, against a background that included containment of many serious infectious diseases.[2] The average gap between non-Indigenous and Indigenous life expectancy is said to be about 17 years, although Indigenous mortality rates, which vary greatly by region, are vastly worse in some areas.[2,5,6] Maori still have a life-expectancy gap of more than 8 years relative to other New Zealanders,[7] although their empowerment is more advanced and advocacy has been more forceful than for Indigenous Australians, and service delivery is less impeded by great distances. The life expectancy of American Indians is still 2.4 years less than that of the general United States population, despite huge improvements in health as a result of socioeconomic advancement and more than 80 years of federally funded and accountable health care through the Indian Health Service and its precursor within the Bureau of Indian Affairs.[8]

Why the goal is probably unattainable

Socioeconomic factors are fundamental determinants of health.[1,9] Australian governments are seeking to remedy the deficiencies in education, employment opportunities, nutrition, and community infrastructure that are central to disadvantage in remote and marginalised Indigenous communities. However, even the best programs will take time to implement, and more time to improve health status. Beyond these measures, any concerted effort to improve the health of Indigenous Australians must also consider clinical profiles, risk status, health interventions and health services.

An infant who survives to the age of 1 year, or a child who survives to 15 years, is an infant or childhood death prevented. Indigenous infant and childhood death rates will probably continue to fall, but this will not translate in the same measure to improved childhood health, due to higher risks of congenital abnormalities and compromised intrauterine development, which might take generations to extinguish, as well as persisting environmental disadvantage. Furthermore, prevention of some infant and childhood deaths creates a more susceptible population of adults.

Most excess deaths in Indigenous adults are due to external causes and to chronic disease.[2] Deaths from external causes should fall with the promised multisector improvements, although normalisation by 2030 seems unlikely, given the long lead-time of social change. However, elimination by 2030 of excess deaths due to chronic diseases (eg, cardiovascular disease, diabetes, chronic lung disease and renal disease) is probably impossible, because of the high proportions of people living today with disease or accentuated risk, the higher risk that will be imprinted on those yet unborn, and the current limitations of primary and secondary prevention.

Early diagnosis and treatment of people with chronic disease (ie, secondary prevention) reduces complications and delays death, but it does not restore life expectancy to normal levels.[10] Even if optimal treatment were extended to all, Indigenous people would experience disadvantage in proportion to their greater burden of disease. Furthermore, optimal treatment does not reach everyone in need, due to poor access to treatment, a vast deficiency in health services capacity,[9] and lack of a needs-based payment system. Finally, the nature of the accentuated risk of chronic disease is not sufficiently understood, or sufficiently under our control, for the risk to be extinguished or totally suppressed by the target date.

Factors shaping accentuated risk

Genetic influences, epigenetic modifiers, past events in an individual's life course, and current circumstances all shape risk. Most studies in Indigenous people have addressed only a limited number of current features and exposures (eg, current body size, smoking, drinking, diabetes), but not issues like intrauterine environments, infant and childhood growth patterns, psychosocial stress and past

infections, while studies of genetic and epigenetic factors have not yet begun. Moreover, most studies are cross-sectional. The few longitudinal surveys reported are intensely regional,[11-15] but there is great variation nationwide in the characteristics, exposures and genotypes of people identifying as Indigenous.

Some risk factors, such as smoking, might be modified in an individual's lifetime, but other risk factors, such as a past burden of infection, cannot. Furthermore, extrapolations from statistical associations to estimates of potential benefits of risk factor modification can be hazardous. For example, although more than 60% of chronic diseases in Indigenous people are purported to be associated with overweight,[16] the predicted body mass indices or waist circumferences at which rates of diabetes in remote-living Aboriginal people would be equivalent to those of non-Indigenous Australians are too low to be feasible population targets.[17] This is because diabetes, like other chronic diseases, is not caused by a single risk factor, but is multideterminant.[18,19]

Prenatal exposures are also important. Historically, Aboriginal infants in remote areas have had low birthweights, due in part to the young age and poor nutrition of their mothers, and to inadequate antenatal care. Today, high rates of maternal smoking and drinking predict ongoing intrauterine growth restriction, and poor perinatal outcomes and childhood health. Intrauterine growth restriction predicts accentuated susceptibility to chronic disease and premature death.[20-26] Many communities now have families with three generations with fetal alcohol spectrum disorder: this is manifested by dysmorphic features in extreme cases, but is associated with neurocognitive impairment more widely, which, in turn, compounds societal dysfunction and deaths from external causes. Fetal alcohol exposure also compromises vascular, cardiac, lung and renal development, which is probably fuelling the chronic disease epidemic.[22,24] A major impact of the current pandemic of ganja (cannabis) use[26,27] on fetal development and postnatal health must also be anticipated.

Even if exposures during an index pregnancy are later modified (eg, cessation of maternal smoking), there is only partial mitigation of risk to fetuses in subsequent pregnancies.[28] The health of offspring is also influenced by paternal experiences, and, more remotely, by maternal and paternal ancestors, through epigenetic modifiers of gene expression.[29,30] Thus, ancestors can influence intrauterine growth, postnatal body habitus, risk of the metabolic syndrome, diabetes, cardiovascular disease and life expectancy of their descendants; stress, smoking and poor nutrition in the ancestors are among the precipitants described. In one study, against a prior background of food scarcity, food sufficiency during the slow-growing period of prepubertal boys conferred high diabetes and cardiovascular risk and a reduced life expectancy on their grandsons.[31] The tumultuous changes that Indigenous people have experienced in the past century probably have health consequences for those alive today, who, in turn, could imprint legacies of their own continued transition onto their descendants for many years beyond 2030.

Some risk factors flow from historical events more directly. The potentiation of chronic disease expression through past therapeutic and public health triumphs is now becoming clear. For example, better survival of children with pneumonia/bronchiectasis results in more adults with chronic lung disease; and extended survival in adults with infections potentiates expression of the accentuated susceptibility to cardiovascular, diabetes and renal disease that a high burden of infection confers. Dramatic reductions in Aboriginal infant mortality were achieved by better care of sick infants between the 1960s and 1990, so that large cohorts of lower birthweight infants have now survived to adult life, where, as proposed by Barker,[25] and more recently demonstrated in the remote Indigenous setting, they are at accentuated risk of chronic disease.[26,32,33] With a persisting, although reduced, difference between Indigenous and non-Indigenous birthweights in some areas today, that effect will operate for several more generations.

Interventions that totally suppress expression of accentuated risk are not yet available, although altered behaviours, exercise and better nutrition will be of some benefit. Pharmacological preventive measures, including treatments like the polypill,[34,35] might surprise with their efficacy, but are not currently widely applied or recommended, and any effect will have a long lead-time.

A focus on shorter-term process measures

It will probably take several generations for Indigenous people's health to approximate that of non-Indigenous Australians. Rather than specify an unrealistic time line for aspirational goals, it would be better to focus on shorter-term process measures. Such process measures have been carefully considered, as outlined in background documents,[1,9] and include:

- ensuring adequate primary care health services and health infrastructure by 2018;
- developing (and implementing) a comprehensive long-term plan of action;
- targeting that plan of action (and its implementation and resources) to need;
- building the evidence base and supporting what works;
- measuring, monitoring and reporting outcomes; and
- implementing adequately funded campaigns to contain problem drinking and smoking.

The uniform availability of quality primary care within 9 years should be non-negotiable. Within those primary care structures, regular health checks, preventive measures and good treatment are more likely to occur. Mechanisms of delivery can be flexible, and include mobile health services, interactive web technologies, and telemedicine. In "hardship" placements at least, strong incentives will be required to recruit and retain a stable, robust, well trained and enthusiastic workforce. In some areas, this will be a largely non-Indigenous workforce until the great deficit in Indigenous capacity is remediated.[9] Rational, single-core funding is needed to accommodate different situations and different burdens of disease. Thus, a community in which people have a 14-fold greater burden of multiple, chronic disease morbidities will need more per-capita funding for good outcomes than one with a sixfold greater burden.[26,36] For greatest impact, interventions must be fast-forwarded in areas with the highest death rates. The widely quoted threefold increase in mortality in Indigenous people nationwide looks trivial in relation to rates in some remote Northern Territory and Western Australian communities.[5,6] In disaster regions, intermediate-term emergency response teams could supply primary health services and health service training while maintenance capacity is being developed. Smoking, alcohol and ganja use, and nutrition must be tackled head-on. Major advertising companies, food franchises and national public health instruments should be engaged to develop campaigns for an Indigenous audience, and those campaigns must be sustained and adapted until they succeed.

The costs will be high, but the response to public health catastrophes in Australia should not be metered by budgetary

considerations. Well targeted and resourced efforts, continually evaluated and adapted, will produce steady improvements in all important parameters of Indigenous health.

Competing interests

None identified.

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References

1 Close The Gap. Indigenous Health Equality Summit. Statement of intent. March 2008. http://www.hreoc.gov.au/social_justice/health/statement_intent.html (accessed Mar 2009).
2 Australian Institute of Health and Welfare. Australia's health 2008. Canberra: AIHW, May 2008. (AIHW Cat. No. AUS 99.) http://www.aihw.gov.au/publications/index.cfm/title/10585 (accessed Mar 2009).
3 Australian Broadcasting Corporation. The 7.30 report. Reporter: Kerry O'Brien. Kerry O'Brien discusses the Government's plans for Indigenous health equality with Health Minister Nicola Roxon. March 2008. http://www.abc.net.au/7.30/content/2007/s2196074.htm (accessed Mar 2009).
4 Altman J, Biddle N, Hunter B. The challenge of "closing the gaps" in Indigenous socioeconomic outcomes. Canberra: Centre for Aboriginal Economic Policy Research, Australian National University, 2008. http://www.anu.edu.au/caepr/ClosingTheGaps.pdf (accessed Mar 2009).
5 Australian Institute of Health and Welfare. Rural, regional and remote health: a study in mortality. Rural Health Series, No. 2. Canberra: AIHW, 2003. (AIHW Cat. No. PHE 45.)
6 Australian Institute of Heath and Welfare. Rural, regional and remote health: a study on mortality (2nd edition). Rural Health Series, No. 8. Canberra: AIHW, 2007. (AIHW Cat. No. PHE 95.) http://www.aihw.gov.au/publications/phe/rrrh-som-2/rrrh-som-2.pdf (accessed Mar 2009).
7 Maori Health. Statistics. Health status indicators. Life expectancy. http://www.maorihealth.govt.nz/moh.nsf/indexma/life-expectancy (accessed Mar 2009).
8 Indian Health Service (Grimm CW, Director). Facts on Indian health disparities. http://info.ihs.gov/Files/DisparitiesFacts-Jan2006.pdf (accessed Mar 2009).
9 CloseTheGap. Solutions to the Indigenous health crisis facing Australia. A policy briefing paper from the National Aboriginal Community Controlled Health Organisation and OXFAM Australia. April 2007. http://www.ahmrc.org.au/Downloads/CTG.pdf (accessed Mar 2009).
10 Daviglus ML, Lloyd Jones DM, Pirzada A. Preventing cardiovascular disease in the 21st century: therapeutic and preventive implications of current evidence. *Am J Cardiovasc Drugs* 2006; 6: 87-101.
11 Daniel M, Rowley KG, McDermott R, et al. Diabetes incidence in an Australian Aboriginal population: an 8-year follow-up study. *Diabetes Care* 1999; 22: 1993-1998.
12 Rowley KG, O'Dea K, Anderson I, et al. Lower than expected morbidity and mortality for an Australian Aboriginal population: 10-year follow-up in a decentralised community. *Med J Aust* 2008; 188: 283-287.
13 Hoy WE, Wang Z, VanBuynder P, et al. The natural history of renal disease in Australian Aborigines. Part 2. Albuminuria predicts natural death and renal failure. *Kidney Int* 2001; 60: 249-256.
14 Wang Z, Hoy WE. Is the Framingham coronary heart disease absolute risk function applicable to Aboriginal people? *Med J Aust* 2005; 182: 66-69.
15 Wang Z, Hoy WE. Albuminuria and incident coronary heart disease in Australian Aboriginal people. *Kidney Int* 2005; 68: 1289-1293.
16 Voss T, Barker B, Stanley L, Lopez AD. The burden of disease and injury in Aboriginal and Torres Strait Islander peoples. Summary report. Brisbane: School of Population Health, University of Queensland, 2007. http://www.uq.edu.au/bodce/docs/2MB-Indigenous-BoD-Summary-Report.pdf (accessed Mar 2009).
17 Kondalsamy-Chennakesavan S, Hoy WE, Wang Z, Shaw J. Quantifying the excess risk of type 2 diabetes by body habitus measurements among Australian Aborigines living in remote areas. *Diabetes Care* 2008; 31: 585-586.
18 Hoy WE, Mathews JD, McCredie DA, et al. The multidimensional nature of renal disease: rates and associations of albuminuria in an Australian Aboriginal community. *Kidney Int* 1998; 54: 1296–1304.
19 Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet* 2004; 364: 937-952.
20 Sayers S, Powers J. Risk factors for Aboriginal low birthweight, intrauterine growth retardation and preterm birth in the Darwin Health Region. *Aust N Z J Public Health* 1997; 21: 524-530.
21 Harris KR, Bucens IK. Prevalence of fetal alcohol syndrome in the Top End of the Northern Territory. *J Paediatr Child Health* 2003; 39: 528-533.
22 Odendaal HJ, Steyn DW, Elliott A, Burd L. Combined effects of cigarette smoking and alcohol consumption on perinatal outcomes. *Gynecol Obstet Invest* 2009; 67: 1-8.
23 Somm E, Schwitzgebel VM, Vauthay DM, et al. Prenatal nicotine exposure alters early pancreatic islet and adipose tissue development with consequences on the control of body weight and glucose metabolism later in life. *Endocrinology* 2008; 149: 6289-6299.
24 Gray SP, Kenna K, Bertram JF, et al. Repeated ethanol exposure during late gestation decreases nephron endowment in sheep. *Am J Physiol Regul Integr Comp Physiol* 2008; 295: R568-R574.
25 Barker DJ. Adult consequences of fetal growth restriction. *Clin Obstet Gynecol* 2006; 49: 270-283.
26 Hoy WE, Kondalsamy-Chennakesavan S, Smith J, et al. The Aboriginal Chronic Disease Outreach Program [Final report, Northern Territory]. http://www.healthinfonet.ecu.edu.au/uploads/resources/3289_aboriginal_chronic_disease_program.pdf (accessed Mar 2009).
27 Clough AR. Associations between tobacco and cannabis use in remote Indigenous populations in Northern Australia. *Addiction* 2005; 100: 346-353.
28 Mohsin M, Jalaludin B. Influence of previous pregnancy outcomes and continued smoking on subsequent pregnancy outcomes: an exploratory study in Australia. *BJOG* 2008; 115: 1428-1435.
29 Morgan DK, Whitelaw E. The case for transgenerational epigenetic inheritance in humans. *Mamm Genome* 2008; 19: 394-397.
30 Gluckman PD, Hanson MA. Metabolic disease: evolutionary, developmental and transgenerational influences. *Nestle Nutr Workshop Ser Pediatr Program* 2005; 55: 17-27.
31 Kaati G, Bygren LD, Edvinsson S. Cardiovascular and diabetes mortality determined by nutrition during parents' and grandparents' slow growth period. *Eur J Hum Genet* 2002; 10: 682-688.
32 Hoy WE, Rees M, Kile E, et al. Low birth weight and renal disease in Australian Aborigines. *Lancet* 1998; 352: 1826-1827.
33 Singh GR, Hoy WE. The association between birth weight and current blood pressure: a cross-sectional study in an Australian Aboriginal community. *Med J Aust* 2003; 179: 532-535.
34 Wald NJ, Law MR. A strategy to reduce cardiovascular disease by more than 80%. *BMJ* 2003; 326: 1419.
35 Sleight P, Pouleur H, Zannad F. Benefits, challenges and registerability of the polypill. *Eur Heart J* 2006; 27: 1651-1656.
36 Hoy WE, Kondalsamy-Chennakesavan S, Wang Z, et al, and the AusDiab Study Group. Quantifying the excess risk for proteinuria, hypertension and diabetes in Australian Aborigines: comparison of profiles in three remote communities in the Northern Territory with those in the AusDiab study. *Aust N Z J Public Health* 2007; 31: 177-183.

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Artist: Weaver Jack *(see page 527)*