Eur J Epidemiol (2009) 24:157–159
DOI 10.1007/s10654-009-9324-0

COMMENTARY

Medical demography and epidemiology: dizygotic twins

Luc Gustaaf Antoon Bonneux

Received: 23 February 2009 / Accepted: 24 February 2009 / Published online: 17 March 2009

Abstract Demography is the statistical study of populations, epidemiology of health of populations. Fascinating challenges for future research, shared by epidemiology and demography, are to be found in the human life course, how the life course is transmitted over generations, how the healthy life is shaped by dynamics within the life course and how the life course is affected by the social and economic environment. The life course begins when two future parents meet. Then social disadvantage or privilege is beginning and diverse rates of ageing are being fixed. Health in utero and at young age will co-determine health at middle and old age, among others mediated by a greater intelligence and a better education. Particularly better education compresses cognitive disability at the end of life, building cognitive reserves that increase brain plasticity and enhance resistance to damage. While with extended life spans may come extended life spans with disability, the twin demography and epidemiology may show how to lighten the burden of senescence at the end of life.

Keywords Demography · Epidemiology · Cognitive epidemiology · Ageing · Life expectancy, disability · Social status · Life table analysis

Demography is the statistical study of populations, studying changes in these populations in response to birth, migration, aging and death. Central to modern demography is the study of the human life course: how we are born, grow up, find a partner, settle down, get children, raise them, become a grandparent, age and die. While demography is small in academia, it is arguably the most multidisciplinary human science of all, drawing from anthropology, economics, sociology, biology and, last but not least, medicine and epidemiology. Demography and epidemiology join at many places, but particularly at the beginning and the end of the life course: fertility, birth, aging and death. Fascinating challenges for future research, shared by both epidemiology and demography, are to be found in the human life course, how the life course is transmitted over generations and how the healthy life is shaped by dynamics within the life course and how the life course is affected by the social and economic environment [1, 2].

Health and ill health are transmitted over generations through genetic, social and cultural mechanisms [3, 4]. Social status is as heritable as money or hypercholesterolemia. Social epidemiologists assume extrinsic conditions causing ill health, but the tenacity and ubiquity of these relationships between socio economic status, education, disease and mortality in very different societies defy that simple theory [5]. This does not mean that extrinsic conditions play no role. In complex systems, many processes interact. Indeed, it has been shown, also in this journal, that a social "good" or "bad" environment will promote good health or disease in its inhabitants [6, 7]. But there is more to it. In the European Union, the largest differences in health between social classes are to be found in the former socialist states, while in the rest of the EU there is no obvious correlation between socio economic differences in mortality and inequality in society [8]. Somewhat surprisingly, the lowest mortality differentials between social classes have been found in the Mediterranean countries, where cardiovascular mortality is lowest [9]. The sharp

L. G. A. Bonneux (✉)
Netherlands Interdisciplinary Demographic Institute,
NIDI, P.O. Box 11650, 2502 AR Den Haag, The Netherlands
e-mail: bonneux@nidi.nl

decline in total mortality has been dominated by decreasing cardiovascular mortality. The risk of cardiovascular mortality is enhanced by psychosocial stress, caused by poverty and social disadvantage [10]. The message may be important, suggesting that lowering cardiovascular mortality in all classes will decrease at least absolute mortality differences between social classes.

The rates of aging are higher in those who are disadvantaged. At the same chronological age since birth, people of lower socio economic classes have an older biological age, closer to death. The life course begins even before conception, when two future parents meet. Then social disadvantage or privilege is beginning, diverse rates of ageing are becoming fixed. In some cultures, if you are a boy or a girl can already determine if you are born or aborted [11]. Increased paternal age at conception predicts mortality in adulthood [12]. If your father is of a lower social class, you are more likely to be obese [13]. Students with no siblings experienced higher respiratory mortality [14]. The Journal extensively documented how health at middle and old age, and particularly cardiovascular disease, is strongly determined by health in utero and at young age [15–20].

The history of the human species has been determined by selection for increasing brainpower, to cope with the increasing demands of social life in large societies [21]. There is a direct evolutionary link between longevity and intelligence. Optimal use of brainpower needs a long youth to learn, a long parenthood to support the children and additional help from grandparents, creating a potential evolutionary role for the elderly. Even in the society of today, the more intelligent children with the better education will live a longer life, more protected against coronary heart disease and stroke later in life [22, 23].

Some of the effects of innate intelligence are direct, increasing life expectancy free of cognitive dysfunctioning and decreasing life expectancy with cognitive disability [24]. In developed societies, higher levels of innate intelligence lead to a higher education, a better trained intelligence and to a greater likelihood of maintaining that trained intelligence at old age. That supplies a reserve of cognitive skills and capacities, allowing to cope better with brain damage [24]. The upshot of this cognitive reserve theory is that existing cognitive reserves and acquired compensatory mechanisms are able to postpone the incidence of clinical dementia to a more advanced age and a more severe stage of brain damage, leading to a worse prognosis and shorter duration of clinical dementia in those that are more highly educated. Better education means better cognitive health for life. Improved education of younger cohorts may be an important driver of future life extension, and even of the gender mortality gap, as girls get increasingly better education than before.

Together with our genes, we inherit from our parents and the society we are born in a complex cultural and social environment, that is increasingly favorable to a long and healthy life. That begs the research question, how long and how healthy? Aging societies, together with increasing life expectancy, predict increasing long term care needs. On average, a lower hazard of death goes together with a lower hazard of disability, but the duration of disability is dependent on the balance between both hazards. The main demographic tool of the twenty-first century is the multi state life table, modeling multiple outcomes in the context of the finite life course [25, 26]. If the hazard of disability and the hazard of death are both dependent of an underlying rate of aging, a lower rate of aging predicts a longer stay in the state of disability. A higher age at death is nearly always associated with a longer life in good health, but also with a prolonged duration of care dependence [27–29].

Life expectancy at birth of Japanese women exceeded 86 year in 2008. The residual life expectancy at age 65, describing mortality at old age, is now close to 24 year. If the mortality decline does not stop, the projected mean age at death of Japanese women from the baby boom cohort, today between 50 and 65, is not far from 95 years. This is hard to believe, but demographers showed that this continued mortality decline has always been stretching our imagination [30]. It is at least wise to foresee the possibility, as such an increased life expectancy will severely stretch pension plans and long term care needs.

Epidemiology and demography are dizygotic twins in population health sciences, sharing the healthy life course of humans as common strand [31]. It is a happy observation that we could illustrate this with a large spate of recent articles from the European Journal of Epidemiology. It will be the shared duty of epidemiology and demography to study how to optimize the healthy life span of people, and what will be the policy consequences of longer and healthier lives, but with increased durations of disability. We will not avoid ageing, but together we should be able to alleviate the burden of senescence further.

Acknowledgment The author wishes to acknowledge Frans J. Willekens and Joop J. de Beer for helpful comments on previous drafts. However, the opinions and errors stated herein are entirely his responsibility.

References

1. Ben-Shlomo Y, Kuh D. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. Int J Epidemiol. 2002;31(2):285–93. doi:10.1093/ije/31.2.285.
2. Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. J Epidemiol Community Health. 2003; 57(10):778–83. doi:10.1136/jech.57.10.778.

3. Bonneux L. From evidence based bioethics to evidence based social policies. Eur J Epidemiol. 2007;22(8):483–5. doi:10.1007/s10654-007-9155-9.
4. Gravseth HM, Bjerkedal T, Irgens LM, Aalen OO, Selmer R, Kristensen P. Life course determinants for early disability pension: a follow-up of Norwegian men and women born 1967–1976. Eur J Epidemiol. 2007;22(8):533–43. doi:10.1007/s10654-007-9139-9.
5. Deary I. Why do intelligent people live longer? Nature. 2008; 456(7219):175–6. doi:10.1038/456175a.
6. Dragano N, Hoffmann B, Stang A, Moebus S, Verde PE, Weyers S, et al. Subclinical coronary atherosclerosis and neighbourhood deprivation in an urban region. Eur J Epidemiol. 2009;24(1):25–35. doi:10.1007/s10654-008-9292-9.
7. Li X, Sundquist S, Johansson SE. Effects of neighbourhood and individual factors on injury risk in the entire Swedish population: a 12-month multilevel follow-up study. Eur J Epidemiol. 2008;23(3):191–203. doi:10.1007/s10654-007-9219-x.
8. Mackenbach JP, Stirbu I, Roskam AJ, Schaap MM, Menvielle G, Leinsalu M, et al. Socioeconomic inequalities in health in 22 European countries. N Engl J Med. 2008;358(23):2468–81. doi:10.1056/NEJMsa0707519.
9. Wong A. Incident solar radiation and coronary heart disease mortality rates in Europe. Eur J Epidemiol. 2008;23(9):609–14. doi:10.1007/s10654-008-9274-y.
10. Brunner E, Marmot M. Social organization, stress, and health. In: Marmot W, Wilkinson RG, editors. Social determinants of health. 2nd ed. Oxford: Oxford; 1999. p. 17–43.
11. Auger N, Daniel M, Moore S. Sex ratio patterns according to Asian ethnicity in Quebec, 1981–2004. Eur J Epidemiol. 2009; 24(1):17–24. doi:10.1007/s10654-008-9307-6.
12. Zhu JL, Vestergaard M, Madsen KM, Olsen J. Paternal age and mortality in children. Eur J Epidemiol. 2008;23(7):443–7. doi:10.1007/s10654-008-9253-3.
13. Heraclides A, Witte D, Brunner EJ. The association between father's social class and adult obesity is not explained by educational attainment and an unhealthy lifestyle in adulthood. Eur J Epidemiol. 2008;23(8):573–9. doi:10.1007/s10654-008-9245-3.
14. Galobardes B, McCarron P, Jeffreys M, Davey Smith G. Association between number of siblings and cause-specific mortality in the Glasgow alumni cohort study. Eur J Epidemiol. 2008; 23(2):89–93. doi:10.1007/s10654-007-9210-6.
15. Koupil I, Shestov DB, Sparen P, Plavinskaja S, Parfenova N, Vagero D. Blood pressure, hypertension and mortality from circulatory disease in men and women who survived the siege of Leningrad. Eur J Epidemiol. 2007;22(4):223–34. doi:10.1007/s10654-007-9113-6.
16. Stein AD, Zybert PA, van der Pal-de Bruin K, Lumey LH. Exposure to famine during gestation, size at birth, and blood pressure at age 59 y: evidence from the Dutch Famine. Eur J Epidemiol. 2006; 21(10):759–65. doi:10.1007/s10654-006-9065-2.
17. Jaddoe VW, van Duijn CM, van der Heijden AJ, Mackenbach JP, Moll HA, Steegers EA, et al. The generation R study: design and cohort update until the age of 4 years. Eur J Epidemiol. 2008; 23(12):801–11. doi:10.1007/s10654-008-9309-4.
18. Jaddoe VW. Fetal nutritional origins of adult diseases: challenges for epidemiological research. Eur J Epidemiol. 2008;23(12):767–71. doi:10.1007/s10654-008-9304-9.
19. Osler M, Lund R, Kriegbaum M, Andersen AM. The influence of birth weight and body mass in early adulthood on early coronary heart disease risk among Danish men born in 1953. Eur J Epidemiol. 2009;24(1):57–61. doi:10.1007/s10654-008-9301-z.
20. Lawlor DA, Cooper AR, Bain C, Davey Smith G, Irwin A, Riddoch C, et al. Associations of birth size and duration of breast feeding with cardiorespiratory fitness in childhood: findings from the Avon longitudinal study of parents and children (ALSPAC). Eur J Epidemiol. 2008;23(6):411–22. doi:10.1007/s10654-008-9259-x.
21. Kaplan HS, Robson AJ. The emergence of humans: the coevolution of intelligence and longevity with intergenerational transfers. Proc Natl Acad Sci USA. 2002;99(15):10221. doi:10.1073/pnas.152502899.
22. Deary IJ, Batty GD, Pattie A, Gale CR. More intelligent, more dependable children live longer: a 55-year longitudinal study of a representative sample of the Scottish nation. Psychol Sci. 2008; 19(9):874–80. doi:10.1111/j.1467-9280.2008.02171.x.
23. Lawlor DA, Batty GD, Clark H, McIntyre S, Leon DA. Association of childhood intelligence with risk of coronary heart disease and stroke: findings from the Aberdeen Children of the 1950s cohort study. Eur J Epidemiol. 2008;23(10):695–706. doi:10.1007/s10654-008-9281-z.
24. Fratiglioni L, Wang HX. Brain reserve hypothesis in dementia. J Alzheimers Dis. 2007;12(1):11–22.
25. Andersen PK, Keiding N. Multi-state models for event history analysis. Stat Methods Med Res. 2002;11(2):91–115. doi:10.1191/0962280202SM276ra.
26. Putter H, Fiocco M, Geskus RB. Tutorial in biostatistics: competing risks and multi-state models. Stat Med. 2007;26(11):2389–430. doi:10.1002/sim.2712.
27. Mamun AA, Peeters A, Barendregt J, Willekens F, Nusselder W, Bonneux L. Smoking decreases the duration of life lived with and without cardiovascular disease: a life course analysis of the Framingham Heart Study. Eur Heart J. 2004;25(5):409–15. doi:10.1016/j.ehj.2003.12.015.
28. Peeters A, Mamun AA, Willekens F, Bonneux L. A cardiovascular life history. A life course analysis of the original Framingham Heart Study cohort. Eur Heart J. 2002;23(6):458–66. doi:10.1053/euhj.2001.2838.
29. Reuser M, Bonneux LG, Willekens FJ. Smoking kills, obesity disables: a multistate approach of the US health and retirement survey. Obesity (Silver Spring). 2009. doi:10.1038/oby.2008.640.
30. Oeppen J, Vaupel JW. Demography. Broken limits to life expectancy. Science. 2002;296(5570):1029–31. doi:10.1126/science.1069675.
31. Stolk RP, Rosmalen JG, Postma DS, de Boer RA, Navis G, Slaets JP, et al. Universal risk factors for multifactorial diseases: LifeLines: a three-generation population-based study. Eur J Epidemiol. 2008;23(1):67–74. doi:10.1007/s10654-007-9204-4.