

# The Brave New World of Lives Sacrificed and Saved, Deaths Attributed and Avoided

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**Abstract:** Attributable risks are routinely estimated in “risk-factor” epidemiology. Often, these risks are interpreted as the numbers of deaths caused by (or numbers of lives lost by) exposure to the factor of interest. It is then often surmised that removal of exposure will avoid deaths and save lives. This reasoning leads to confusion because of 2 underlying assumptions. One is that removal of the exposure will reduce permanently the annual number of deaths by the number attributed to the factor. In reality, deaths are merely postponed and lives are merely prolonged; estimating the effect of exposure on life expectancy is both more straightforward and of greater public health interest. The other misleading premise is that the deaths attributed to a certain risk factor can be identified. While such identification may be possible for certain immediate external causes of death (eg, accidents), it is not usually feasible for deaths attributed to factors that merely contribute to development of chronic disease and ultimately death. For such exposures, it is more reasonable to suggest that they contribute to death in all who are exposed – more so in some people than in others. Again, it is more appropriate to calculate the average loss of life expectancy associated with exposure from follow-up studies; the years of life lost by individuals who are exposed then varies around this average. The “real age” concept popularized in lifestyle Web sites and television programs may be more useful than calculations of the numbers of attributable deaths for communicating individual as well as public health risks associated with common environmental, occupational and lifestyle risk factors.

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When Aldous Huxley wrote *Brave New World* in 1932, he envisaged a world inhabited by healthy people looking and feeling young forever—that is, until they, somewhere in their 60s, entered the Park Lane Hospital for the Dying for a brief, accelerated but comfortable aging and dying process.<sup>1</sup>

Together with many other preoccupations, epidemiology would surely have vanished from Huxley’s World. After all, with health, well-being, and death programmed to the point where everybody’s experience is equivalent to that of the whole group, there would be no need to study their distributions or determinants.

Few people would have much sympathy for the means used to create the “Brave New World,” with its eugenics, totalitarianism, and mind control. But some of the ends seem familiar—a long healthy life, morbidity that is compressed into a late and short last phase of life, and compassionate end-of-life care. It is interesting to look at some of the ramifications of risk-factor epidemiology in the light of Huxley’s book, and in particular to consider the tendency (or is it the temptation?) to interpret, at the level of the individual, results and concepts based on group differences.

Let’s take the example of air pollution. Cohort studies<sup>2,3</sup> have documented that people living in urban areas with high concentrations of particulate matter in the atmosphere have a lower life expectancy than subjects breathing clean air. Such effects are expressed as relative, which in this context is the proportional change in mortality hazards (ie, age-specific death rates) per unit of fine particles. Higher relative risks mean “extra” deaths in the more polluted communities.

However, these cohort studies make no attempt to identify which individual deaths in the more polluted areas are attributable to air pollution—nor could they. We know that the excess mortality is focused among certain causes of death (cardiorespiratory and lung cancer). We also know that relative risks are somewhat higher in some subgroups, notably those with lower educational status<sup>3</sup>—a finding widely understood as reflecting increased susceptibility among poorer people. Even so, more precise identification of those affected by air pollution remains elusive, precisely because the role of pollution is best understood as increasing the risks among many.

Although the effects can be described reasonably reliably for the population or community as a whole, we cannot say how this translates into effects on specific individuals. For example, we cannot say whether a given excess risk of lung cancer is due to new cases in persons who otherwise would not have been affected, or to an acceleration of the disease among those who were going to get lung cancer anyway. Relative risks do not distinguish between the two.

Increasingly, policies to protect health by better control of air pollution are informed by health impact assessments. These assessments estimate the mortality and morbidity due to current levels of air pollution, and the public health benefits that might accrue by reducing air pollution in various ways and to varying extents. These health impact assessments link the relative risks from cohort studies with estimates of the size of the exposed population and the extent of ambient pollution experienced by that population, to estimate the numbers of deaths (or the change in life expectancy) that can be “attributed” to the exposure.<sup>4</sup>

The relative risks for air pollution are small (on the order of 1.1–1.2 for dirty vs. clean air communities). Still, given that the number of exposed people is large, the numbers of “attributable” deaths can also be large. For instance, Künzli et al<sup>5</sup> estimated that air pollution causes 6% of annual mortality in Austria, France, and Switzerland, for a total of 40,000 annual deaths. A Dutch report<sup>6</sup> attributed 18,100, or more than 10% of some 130,000 annual deaths, to particles smaller than 10  $\mu\text{m}$ , and estimates for the European Commission’s Clean Air for Europe program suggested 200,000 “attributable” deaths annually across the 25 countries in the European Union at that time.<sup>7</sup> In their investigation of the worldwide impact of a broad range of external risk factors, Ezzati et al<sup>8</sup> attributed 40 million of a total of 56 million deaths per year to 6 main categories of risk factors (childhood and maternal undernutrition, other nutritional risks and physical inactivity, sexual and reproductive health risks, addictive substances, environmental risks, and occupational risks). (The authors cautioned that overlap among these risk factors would make this number of “attributable” deaths an overestimate).

“Attributable” deaths are estimated by assessing the mortality experience of populations under 2 scenarios—a baseline using current death rates, and an alternative using death rates that reflect reduced exposure. “Attributable” deaths are the differences in deaths (the “extra” deaths) between the 2 scenarios. These deaths are sometimes also labeled as “avoidable.”<sup>9</sup> The all-too-easy interpretation is that deaths attributed to factor X will be avoided when exposure to X ceases. Authors may or may not acknowledge explicitly that these are not “avoidable” deaths per se, but rather avoidable premature deaths. Death, in the end, is not avoidable. Death can merely be postponed; what can be influenced is not the fact of death, but its timing.

This has a number of important implications. One is technical. The concept of “avoidable” deaths was popularized some 30 years ago for the purpose of evaluating the benefits of health care for ailments such as appendicitis.<sup>10</sup> To avoid the complications of such calculations at more advanced ages, the estimates of “avoidable” deaths were usually constrained to younger people (below age 65, 70, or 75 years).<sup>11,12</sup> This convention is also carried over into assessments of other risk factors. As an example, an analysis of 40 years of follow-up of the famous Framingham heart study cohort documented dramatic joint effects on survival of smoking and obesity at baseline, and then estimated the numbers of “premature” deaths only as those occurring before age 70.<sup>13</sup>

However, most deaths attributed to external risk factors such as air pollution or smoking are not expected to occur at these younger ages. For this reason, most health impact assessments of air pollution consider all “attributable” deaths, without restriction by age.<sup>5,7,8,14</sup> Counting “attributable” deaths only up to a given age would have important implications for the results. In the Netherlands, for example, 81% of all deaths nowadays occur in subjects older than 65, 74% in subjects older than 70, and 59% in subjects older than 75. More discussion is needed of the trade-offs between underestimating the effects by narrowing the age range, and increasing the uncertainty by estimating attributable deaths in older people.

A second implication is in the very notion of “premature” death. This tacitly supposes that we have some idea of the age at which deaths stop being “premature” and become “mature.” In Huxley’s *Brave New World*, where individuals experience the group average, this was easy: they’re the sexagenarians in Park Lane Hospital (showing that in the 1932 United Kingdom, living to your 60s was not considered a bad deal). But what about our less brave and older world? At what age does death become “mature”? In the well-known Global Burden of Disease project, the human life expectancy was set at 82.5 years for women and 80 years for men.<sup>9</sup> This was based not on any medical or biologic reasoning, but rather on the highest observed life expectancy at the time (Japanese women). Ten years later, the World Health Organization World Health Report 2006 shows that Japanese women have a life expectancy of 86 years.

Life expectancy continues to increase around the globe, even in countries with the highest expectancies already. We would consider it a major public health crisis if our exposure to one of the many risk factors we throw into our health impact assessments today were to suggest a shortening of life expectancy even to that of our parents’ generation. Maybe this is just as well. Like poverty, life expectancy can be considered in absolute or relative terms, and it is no harm to have our sights raised regarding what is possible.

In relative terms, “premature” carries the simple connotation of “earlier than it would otherwise have been.” This concept is captured in the slightly awkward but informative phrase of “deaths brought forward.”<sup>15</sup> This term was introduced to refer to deaths “attributable” to short-term exposure to air pollution, ie, deaths triggered by higher air pollution on the same or on immediately preceding days. It is widely acknowledged that such deaths occur primarily in a subgroup with serious preexisting (though possibly undiagnosed) cardiorespiratory disease, and who therefore have a life expectancy far less than others of the same age. This further example of a “moving target” shows that the choice of reference group is crucial when estimating the size of life-expectancy gains and losses.

A third implication of the fact that death can only be postponed is that removal of exposure from a specific cohort produces only temporary reductions in the absolute number of deaths per year.<sup>16,17</sup> Of course, a reduction in risk that leads to lower annual death rates and an increased life expectancy will, over time, increase the size of the population surviving at a given age. But for this very reason, there will come a time when a cohort with cleaner air and better survival will experience more deaths at advanced ages than a cohort breathing “dirty” air, simply because more people in the “clean air” cohort have survived to older age.

An added complication is that factors contributing to chronic disease development and early death usually do not operate discretely (producing death in some but not in others), but rather increase the risk of death in everyone exposed. Attributable fraction calculations, though based on the risk differences between 2 groups, easily lend themselves to the interpretation that some of the exposed die of their exposure but the rest do not. This dichotomy doesn’t make much sense from a biologic point of view, and it seems more logical to express effects of exposure in terms of “rate advancement periods,” which estimate by how many months or years the survival curve among the exposed is “advanced” or brought forward compared with the survival curve among the unexposed.<sup>18</sup>

In keeping with this line of reasoning, health gains from the reduction of multiple external risk factors were recently estimated in terms of increases in (healthy) life expectancy rather than reductions in numbers of deaths.<sup>19</sup> Similarly, we have for some years now<sup>20–22</sup> proposed and used changes in life expectancy rather than estimates of “attributable deaths” in estimating the mortality impacts of longer-term exposure to air pollution. This approach is also championed by Rabl,<sup>16</sup> and is increasingly used either on its own or in parallel with estimates of “attributable” deaths.<sup>7,14</sup>

These thoughts lead to some conclusions. First of all, when it comes to the risk of dying from chronic disease at an advanced age, the term “avoidable” deaths is an unlucky one, even when “avoidable” is put between quotation marks. “Postponable” is more like it. Similarly, lives are not “sacri-

ficed” or “saved”—they’re cut short or prolonged. Second, the concept of “avoidable” or “postponable” deaths may have meaning at the individual level for patients whose lives are prolonged by a specific medical procedure, but is not meaningful for subjects exposed to some risk factor that decreases their average life expectancy as a group. Removal or reduction of exposure to a given risk factor will increase life expectancy by some calculable amount, but we do not know how that gain in life expectancy is distributed across individuals. It leads to some reduction in risk in possibly all (formerly) exposed subjects, and variable postponement of death in an unquantifiable number of them. It is impossible to say how many deaths are “avoided” or “postponed” because the numbers of deaths attributed to such exposures refer to group differences, and are not linked to identifiable patients.

It can be useful to calculate reductions in the numbers of deaths in populations, for example when age-specific death rates are reduced through improved air quality—but there are difficulties in interpreting such numbers as “attributable” deaths. In particular, it is misleading to take the average gain in life expectancy over the whole population, and attribute it to the “deaths avoided,” thereby inferring an estimate of “live years saved” per death “avoided”. The arithmetic is easy but the interpretation is not.

So perhaps we should try to use more appropriate terminology when communicating the public health threats related to external risk factors, and the benefits expected from their reduction. Body counts have the potential to seriously mislead, whereas risks and benefits expressed as changes in life expectancy are more straightforward. Maybe we can learn something here from popular Internet and television questionnaire-based programs called “your true age” that help individuals translate their own set of “risk” factors into an estimate of how much different their “true” age is from the calendar one.

This is not all that new. Sir Joseph Bazalgette was chief engineer of the reconstruction of the London sewers in the 19th century. At his death in 1891, his obituary in the London Times ended thus: “Of the great sewer that runs below Londoners know, as a rule, nothing; though the Registrar General could tell them that its existence has added some 20 years to their chance of life.”<sup>23</sup>

## REFERENCES

1. Huxley A. *Brave New World*. London, UK: Granada Publishing; 1932.
2. Dockery DW, Pope CA 3rd, Xu X, et al. An association between air pollution and mortality in six U.S. cities. *N Engl J Med*. 1993;329:1753–1759.
3. Pope CA 3rd, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA*. 2002;287:1132–1141.
4. Sanderson EG, Hurley JF, eds. Air pollution and risks to human health—an AIRNET report. 2004. Available at: <http://airnet.iras.uu.nl/>. Accessed March 7, 2007.
5. Künzli N, Kaiser R, Medina S, et al. Public-health impact of outdoor and traffic-related air pollution: a European assessment. *Lancet*. 2000;356:795–801.

6. Knol AB, Staatsen BAM. *Trends in the Environmental Burden of Disease in the Netherlands, 1980–2020*. Bilthoven, The Netherlands: National Institute of Public Health and the Environment; 2005. RIVM report 500029001.
7. Amann M, Bertok I, Cofala J, et al. Baseline scenarios for the Clean Air for Europe (CAFE) programme. IIASA. 2005. Available at: [http://www.iiasa.ac.at/rains/CAFE\\_files/Cafe-Lot1\\_FINAL\(Oct\).pdf](http://www.iiasa.ac.at/rains/CAFE_files/Cafe-Lot1_FINAL(Oct).pdf).
8. Ezzati M, Lopez AD, Rodgers A, et al. Selected major risk factors and global and regional burden of disease. *Lancet*. 2002;360:1347–1360.
9. Murray CJL, Lopez AD. *The Global Burden of Disease*. Cambridge, MA: Harvard University Press; 1996.
10. Rutstein DD, Berenberg W, Chalmers TC, et al. Measuring the quality of medical care. A clinical method. *N Engl J Med*. 1976;294:582–588.
11. Holland WW. Commentary: should we not go further than descriptions of avoidable mortality? *Int J Epidemiol*. 2003;32:447–448.
12. Treurniet HF, Boshuizen HC, Harteloh PP. Avoidable mortality in Europe (1980–1997): a comparison of trends. *J Epidemiol Community Health*. 2004;58:290–295.
13. Peeters A, Barendregt JJ, Willekens F, et al. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. *Ann Intern Med*. 2003;138:24–32.
14. Boldo E, Medina S, LeTertre A, et al. Apehis: health impact assessment of long-term exposure to PM(2.5) in 23 European cities. *Eur J Epidemiol*. 2006;21:449–458.
15. Department of Health. *Committee on the Medical Effects of Air Pollutants (COMEAP). Quantification of the Effects of Air Pollution on Health in the United Kingdom*. London: The Stationery Office; 1998.
16. Rabl A. Analysis of air pollution mortality in terms of life expectancy changes: relation between time series, intervention and cohort studies. *Environ Health*. 2006;5:1.
17. Miller BG, Hurley JF. Comparing estimated risks for air pollution with risks from other health effects. Edinburgh, UK: Institute of Occupational Medicine; 2006. Research report TM/06/01. Available at: [http://www.iom-world.org/pubs/IOM\\_TM0601.pdf](http://www.iom-world.org/pubs/IOM_TM0601.pdf). Accessed March 7, 2007.
18. Brenner H, Gefeller O, Greenland S. Risk and rate advancement periods as measures of exposure impact on the occurrence of chronic diseases. *Epidemiology*. 1993;4:229–236.
19. Ezzati M, Hoorn SV, Rodgers A, et al. Estimates of global and regional potential health gains from reducing multiple major risk factors. *Lancet*. 2003;362:271–280.
20. Brunekreef B. Air pollution and life expectancy: is there a relation? *Occup Environ Med*. 1997;54:781–784.
21. Holland M, Berry J, Forster D, eds. *ExternE, 1999: Externalities of Energy, Vol. 7: Methodology 1998 Update*. Luxembourg: Office for Official Publications of the European Communities; 1999:chap 8. Available at: <http://www.externe.info/>. Accessed March 7, 2007.
22. Miller BG. Life-table methods for predicting and quantifying long-term impacts on mortality. In: *Quantification of Health Effects of Exposure to Air Pollution. Report on a WHO Working Group, Bilthoven, Netherlands, 20–22 November 2000*. Copenhagen, Denmark: WHO Regional Office for Europe.
23. Halliday S. *The Great Stink of London—Sir Joseph Bazalgette and the Cleansing of the Victorian Metropolis*. Stroud, Gloucestershire: Sutton Publishing; 1999.