
Chapter 29

CONCLUSIONS AND DIRECTIONS FOR FUTURE RESEARCH

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The analyses of risk factors within a common analytical framework and using comparable methods as outlined in these volumes has ensured greater consistency and comparability in evaluating and using scientific evidence on major risks to health. At the same time, data and knowledge gaps identified in the analyses of these risks illustrate key areas of scientific enquiry necessary to better inform policies and programs that aim to prevent disease by reducing risk factor exposure. The principal findings were discussed in individual risk factor chapters, as well as in those that presented summary results for individual risk factors (chapter 26) and for the joint effects of multiple risks (chapter 27). In this chapter, we use these findings to describe broadly how the analyses might affect public health practice as well as research on risk factors.

The analyses of the selected risk factors considered in this work, based on comprehensive reviews of available evidence on exposure and hazards, suggest that a small number of risks, such as childhood and maternal underweight and unsafe sex, accounted for a very large contribution to global loss of healthy life. Further, several risks, such as high blood pressure, tobacco and alcohol, have relative prominence in regions at all stages of development. While reducing all of the above risks to their theoretical minima may not be possible using current interventions, the results illustrate that disease prevention by addressing known distal and proximal risk factors can provide substantial, and under-appreciated, public health gains.

Treatment of established disease will always have a role in public health, especially in the case of diseases such as tuberculosis where treatment contributes to prevention. At the same time, the current devotion of a disproportionately small share of resources to prevention by reducing exposure to major known risk factors, through personal and non-personal interventions, should be reconsidered in a more systematic way in the light of this evidence.

Beyond their total hazard, the distributions of risks in a population have major implications for prevention strategies. Risk typically increases along a continuum of exposures. Dichotomous labels such as “hypertensive” and “normotensive” are therefore not a description of the health consequences of risks, but rather an operational convenience. In fact, the “deviant minority” (e.g. hypertensives) who are considered to be at high risk are only part of a risk continuum, rather than a distinct group, leading to one of the most fundamental axioms in disease prevention across risk factors: “a large number of people exposed to a small risk may generate many more cases than a small number exposed to high risk”. Rose (1992) pointed out that wherever this axiom applies (see chapter 26 for possible exceptions), a preventive strategy focusing on high-risk individuals will deal only with the margin of the problem and will not have any impact on the large proportion of disease occurring in the large proportion of people who are at moderate risk.

While a high-risk approach may be more appropriate to the individuals and their physicians at any point in time, treating prevention as managing individual, high-risk crises can only have a limited effect at the population level and over long time periods. This is particularly relevant in the context of efforts to improve global health by addressing multiple diseases and risk factors, many of which exhibit continuous associations with disease outcomes. Focusing on high-risk individuals does not alter the underlying causes of illness, relies on having adequate discriminative ability to predict future disease, and requires continued and expensive screening for new high-risk individuals. In contrast, population-based strategies that seek to shift the whole distribution of risk factors have the potential to control population incidence. Such strategies aim to make healthy behaviours and reduced exposures the social norm and thus lower risk in the entire population.

Our exploration of the joint contributions of multiple risk factors suggests that 20 leading risks contributed to considerable loss of healthy life in different regions of the world. In particular, for some of the leading global diseases (e.g. acute lower respiratory infections, diarrhoea, lung cancer, ischaemic heart disease and stroke), substantial proportions were attributable to these selected risk factors. Removing the leading 20 risk factors from among those studied here would not only have resulted in a 9.3-year (17%) gain in global healthy life expectancy in 2000, but also accounted for some of the interregional healthy life expectancy (HALE) differences. The analysis showed that even populations with currently high healthy life expectancy (e.g. developed regions of the Western Pacific and Europe) could further benefit considerably from risk reduction. These results provide a guide for achieving potential gains in (healthy) life expectancy that have been estimated statistically from past trends (Oeppen and Vaupel 2002; Riley 2001) through disease prevention by reducing known risks. The results for multiple risk factors further emphasize that for more effective and affordable implementation of a

prevention paradigm, policies, programmes and scientific research should acknowledge and take advantage of the interactive and correlated role of major risks to health, across and within causality layers. This could be an important step in addressing health inequalities, many of which may arise from concentration of major risks among specific socioeconomic groups.

Health research has at times focused on topics which, while scientifically intriguing, have not always been motivated by broader population health consequences (Anonymous 2001; Gross et al. 1999; Horton 2003; Willet 2002). The collation of evidence on exposure and hazard for different risks in this book, and the existing data gaps have illustrated data and monitoring needs for better quantification of important risks.

Research needs include more detailed and better quality data on exposure to most risks, using exposure variables that capture the full distribution of hazards in the population. Important examples include detailed data on alcohol consumption volume and patterns, dietary and biological markers for micronutrients, and better indicators for physical activity, indoor air pollution and occupational risks, all of which were quantified using indirect measures with limited resolution.

Assumptions and extrapolations were also needed in quantifying risk factor–disease relationships because of gaps in knowledge about the impact of some important global risk factors, particularly in developing countries. Examples include limited quantitative assessment of the hazards of specific sexual behaviours for HIV/AIDS or other sexually transmitted diseases, alcohol drinking patterns (Puddey et al. 1999) or exposure to indoor smoke from solid fuels (Ezzati and Kammen 2002). Equally important are detailed exposure data for risks that have been traditionally studied in developed countries, but have global importance and require more detailed data and hazard quantification in developing regions (e.g. alcohol and obesity).

The limited evidence on the effects of multiple risk factors and risk factor interactions also points to important gaps in research on multi-risk and stratified hazards as also discussed in chapters 27 and 28. Including multiple causes in epidemiological research and risk assessment would allow estimating the benefits of reduction in combinations of distal and proximal exposures using multiple interventions (e.g. using education and economic tools to: i) promote physical activity or healthier diet coupled with screening and lowering cholesterol; and ii) address overall childhood nutrition and environment instead of a focus on individual components). In such research, risk factor groups should be selected based on both biological relationships and socioeconomic factors that affect multiple diseases, as discussed in chapter 27. Examples include those risk factors that are affected by the same policies and distal socioeconomic factors (e.g. malnutrition, unsafe water, sanitation and hygiene, indoor smoke from solid fuels and rural development policies) or affect the same group of diseases (e.g. all of the above for child-

hood infectious diseases; smoking, diet and physical activity for cardiovascular diseases). Once risk factors have been selected, the emphasis on reducing confounding should be matched by equally important enquiry into independent and mediated hazard sizes that are stratified based on the levels of other risks.

This is a substantial research agenda, and one for which some progress has been made over the past decades. Yet public health policy needs demand that much greater priority is given to research that more reliably and relevantly identifies the potential for prevention in all countries, including information on exposures and risks among subpopulations, especially among the least well off. Such research will undoubtedly provide a more compelling basis for the massive increase in preventive efforts worldwide that is required if the potential for health gains identified in this book is to be realized.

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