Commentary: The message is rarely simple: the J-curve and beyond

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In their article in this issue of the International Journal of Epidemiology, Adams and White take on the important topic of population-based prevention strategies. Their central thesis is that when the relation between exposure and average risk of an outcome is J-shaped, a population-based prevention strategy has the obvious potential to harm a group of individuals by pushing them into a higher risk level through a reduction in exposure. In their paper, the authors focus mainly on the outcome of all-cause morbidity/mortality.

In their first paragraph, the authors invoke the name and work of Geoffrey Rose, and note that his reasoning has been so persuasive that the population approach has been described as one of the ‘absolute truths’ of preventive medicine. In this era of market individualism and consumer-driven medicine, I believe this description of Rose’s impact is, unfortunately, highly debatable in the US, but this is not the central point here. More relevant to Adams and White’s work, I would suggest that whenever one hears the language of ‘absolute truth,’ it is usually necessary to return to the original texts themselves, to see what was actually argued and advocated. In his book The Strategy of Preventive Medicine, Rose concluded (p. 14) that preventive medicine must embrace both the ‘high-risk’ and population strategy of prevention, but, of the two, the power to bring about sustained reductions in disease burden resided with the population strategy. Rose was not one-sided in his thinking, and in fact laid out four schematic models of exposure–disease associations, with the last one being the J-shaped model that Adams and White focus on. He noted that this model was different from the others, and that it demonstrated graphically the oft-expressed wisdom that moderation, rather than extremism, promotes health (pp. 17–18).

Adams and White make their point well about the potential dangers involved in shifting the entire population downward in exposure level when the relation between exposure and outcome is J-shaped rather than monotonically positive. Rose makes this point himself, noting that (p. 106) in any widespread change, some individuals will be hurt. Because a change in exposure is good on average does not mean, obviously, that every single individual will benefit if he or she experiences a change in exposure—an average benefit can be accompanied by individual harm. (As example of this, there is the concern that folic acid may mask the relatively rare condition of pernicious anaemia, thus allowing further neurological deterioration from the disease than would otherwise occur.)

Adams and White state that when exposure–outcome curves are J-shaped, some type of ‘informed consent’ perhaps should be required from those at high risk of being negatively affected by population interventions, so that they understand the risks involved and have the opportunity to ‘opt out.’ There is no logical reason to restrict such ‘informed consent’ procedures to the J-curve situation, though it may take more thought and effort to identify the individuals that will be harmed by an intervention when the exposure–outcome curves are monotonic.

Another important point concerning the inherent complexities in going from average risk to individual risk is that specific information on specific individuals often makes ‘averages’ irrelevant. A woman at high risk of breast or ovarian cancer due to genetic susceptibility, or a man at high risk of cardiovascular disease because of familial hyperlipidaemia, or an individual who has witnessed alcoholism ravage family members may all be far less concerned about morbidity or mortality in general than with a specific outcome. The authors note that the usual one-disease-at-a-time focus has real limitations when considering population interventions, and this is undoubtedly true when considering the general concept of population health. However, the focus of Adams and White is on individuals, more precisely those individuals who should be singled out of population interventions because they stand to lose. It is precisely at this individual level where concern about specific diseases to the exclusion of others makes the most sense, because at the individual level, there is usually a wealth of information that is not considered or conveyed in the averages making up an empirical curve relating exposure to all-cause mortality. If the goal, as Adams and White imply, is to identify those individuals who stand to lose by being exposed to an intervention (including those interventions of verbal encouragement or pressure to change), personal information must be considered by knowledgeable professionals and discussed with the individual before any discriminating judgement about potential personal harm can be made. Otherwise, we run the risk we are trying to avoid in the first place: we risk doing harm because we fail to consider the
complexities of applying epidemiological averages to the individual level. For instance, if we wish to protect all ‘modest’ or ‘moderate’ drinkers from abstaining from alcohol because of the J-curve relating alcohol consumption to total mortality, we can cause the mortality risk to increase among moderate-drinking women who are predisposed to breast cancer. All this is not to say that Adams’ and White’s central point is mistaken; rather, the issue is more complex than they propose, I believe.

An argument that seems to be implicit in Adams and White’s writing, as evidenced by their use of the language of ‘informed consent’ and ‘protection’ of individuals, is that ‘population intervention’ necessarily connotes ‘coercive’ intervention, or that individuals are somehow passive absorbers of directives to change—i.e. that individuals in a certain exposure group can merely ‘be’ moved to a different exposure level. There are examples of this in the history of public health interventions (e.g. fluoridation of the water supply, removal of lead from gasoline, recent fortification of many commercial flour goods with folic acid). However, each of the exposure examples discussed by the authors in their paper fall into the broad category of ‘lifestyle’ exposures—alcohol, use of pharmacological agents to lower blood pressure, and body weight. In democratic societies, especially those like the US which place a high value on individualism and ‘consumer choice’, there are usually different types of approaches used by public health professionals to effect population-wide shifts in ‘lifestyle’ risk factor distributions. One type might work through economic incentives—e.g. attempts at taxes on cigarettes and ‘fast food,’ or lower health insurance costs for non-obese and non-smoking individuals. Another type of strategy might work through the law—e.g. laws about minimum drinking age, seatbelt usage, and smoking in certain venues. Probably the most common type of population health intervention for lifestyle factors in modern society today is mass communication/mass education, however. Indeed, such communication nearly always accompanies other types of interventions for lifestyle factors. These communication interventions are both formal and scientifically sanctioned (e.g. the US’s National Cholesterol Education Program, sponsored by the National Heart, Lung, and Blood Institute; HIV/AIDS prevention messages sponsored by the Centers for Disease Control and Prevention, as well as local and regional health departments) and informal and unsanctioned (e.g. advertisements from food industries and companies exhorting individuals to eat or drink more of their product, whether it be milk, walnuts, ketchup, red meat, fruits, low-carb or low-fat frozen meals).

Whether exposure–outcome relations are monotonic or not, it is incumbent upon those of us in public health to work to ensure that any communication the public hears is as simple, but no more simple, than it needs to be. This means not only working to improve communication between medical and public health researchers and the public, but also, ideally, taking it as our scientific responsibility to work against simplistic, misleading, or even false communications that emanate from any source, including commercial sources. (This is a huge challenge, and it is unlikely that scientists can compete on an equal footing with corporate advertising and entertainment for public attention, but the importance of the task of disseminating accurate health information is not diminished by the probability of partial failure!)

When the relations between exposure and risk are complex, individuals should not be given overly simplistic messages. For instance, individuals need to hear, consistently, that there is an optimal range of body weight and that it is indeed possible to be too thin. Physicians should know that pharmacological lowering of blood pressure beyond a certain level can be detrimental to patients’ health. Individuals should understand that scientific data show that a modest amount of alcohol consumption is probably better than abstinence when it comes to total mortality risk (though the total mortality risk here is mainly driven by coronary heart disease risk). At the same time they should realize that epidemiological studies can say little about the psychological and social distress that alcohol may bring to an individual’s life, even if it is longer in quantity. Indeed, with the chief exceptions of environmental pollutants (e.g. asbestos, PCBs, mercury) and infectious agents (anthrax, tuberculosis) it is likely that most curves relating exposure to all-cause mortality are J-shaped, or at least not monotonic, and that the age-old advice of ‘moderation in everything’ applies far more widely than epidemiologists have yet ‘discovered.’

Prevention, at its heart, is all about communication—communication between researchers and their audiences of policymakers, physicians, and the public, communication between clinicians and patients, essentially communication between any two or more parties (including, today, commercial interests) that have a stake in either the exposure or the outcome. As researchers, we cannot give overly simplistic messages to individuals, and it is not up to us, or even to policymakers, to decide, autocratically, what acceptable risk/benefit trade-offs are for the public, when substantial risks do exist. Simplistic communication on our part engenders a self-fulfilling prophecy—we deem the public incapable of understanding complex information and of participating in societal debate about risks, so often all we provide is simple ‘soundbites,’ and then this is all they have to repeat back to us. The authors’ points about J-curves and the obvious potential dangers posed to certain individuals because of mass intervention can really be subsumed under the more general problem of communicating complex information about health and disease to policymakers, physicians, and individuals in a society already overwhelmed by information overload. This issue goes far beyond the J-curve.

References

1 Adams J, White M. When the population approach to prevention puts the health of individuals at risk. Int J Epidemiol 2005;34:40–43.