Can dietary changes rapidly decrease cardiovascular mortality rates?

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Received 18 November 2010; revised 29 December 2010; accepted 1 February 2011; online publish-ahead-of-print 2 March 2011

The UK National Institute for Health and Clinical Excellence (NICE) recently published guidance on the prevention of cardiovascular disease (CVD) in whole populations.¹ The lukewarm government response demonstrated a surprising ignorance of the scale and rapidity of the potential benefits.

The long ‘incubation period’ paradigm for cardiovascular diseases

Most heart attacks and ischaemic strokes are caused by complicated atheroma, usually compounded by thrombosis suddenly reducing blood flow in a critical artery. Extensive evidence suggests that this atheroma silently builds up over decades. Thus, early atheroma streaks were seen in classical autopsy studies on Korean war casualties and teenage traffic fatalities.² Hence, the emerging paradigm describing the temporal relationship between risk factor change and the corresponding change in CVD mortality. The time scales for this paradigm are generally perceived in terms of decades. Thus, Rose thought the ‘incubation period’ was a decade or more,³ whereas Law et al.⁴ proposed a three-decade lag time to explain the paradoxically low French CVD rates.

Reversing the CVD process following reductions in major cardiovascular risk factors has therefore also been assumed to require decades. However, this is wrong.

Cardiovascular disease mortality can change quickly

Evidence from clinical trials

Evidence in individuals and in populations actually suggests that decreases in fatal and non-fatal CVD events can rapidly follow reductions in risk. Thus, individual patients in therapeutic randomized trials often demonstrate mortality reductions within 1 or 2 years of blood pressure or cholesterol lowering.⁵ Trials in diet and lifestyle interventions also demonstrate rapid and substantial changes in multiple risk factors, notably DASH, DASH-Sodium, OMNI-Heart, and PREMIER. Even more importantly, several randomized controlled trials showed that diet interventions could have relatively rapid effects on CVD outcomes.⁶

The rapid reduction in diabetes incidence achieved with lifestyle interventions is also relevant, meaning subsequent reductions in costly diabetic care, as well as in CVD events.⁷

Even more, population level legislation like the smoking ban in Scotland and elsewhere has resulted in dramatic reductions in admissions for acute myocardial infarctions.⁸

Evidence from natural experiments and policy interventions

The natural experiments and policy interventions observed in whole populations in Cuba,⁹ Mauritius,¹⁰ Finland,¹¹ and elsewhere suggest that reductions in major factors can be quickly followed by rapid changes in CVD mortality rates. Indeed, the first clues emerged from reports of wartime Europe. Dramatic brief reductions in coronary deaths rapidly followed food rationing in the UK, and more savage ‘hunger winters’ in Holland and Norway.¹² The recent trends observed in several central European and Baltic countries were also remarkable. Thus, after steady rises through the 1970s and 1980s, CVD mortality in Poland suddenly declined sharply in the early 1990s, immediately following the profound socio-economic changes experienced after the break-up of the Soviet Union in 1989. This mortality fall was consistently attributed to diet changes. Specifically, subsidies for meat and animal fats ended and consumption fell dramatically, along with substantial increases in vegetable oils and fresh fruit.¹³ The subsequent 26% decrease in CVD deaths between 1990 and 1994 was one of the fastest declines ever observed. It could not be dismissed as a fluke, because corresponding changes were also observed in other central European and Baltic countries,¹³ many of them having experienced a profound socio-economic transformation in...
the 1990s that allowed their admission to the European Union (Figure 1).

Conversely, mortality can also increase rapidly after adverse changes in diet and lifestyle. For example, the unfavourable lifestyle changes during the 1980s in the USSR were quickly followed by marked increases in total and cardiovascular mortality, probably reflecting dramatic fluctuations in alcohol consumption imposed on a background of chronically poor diet and persistently high smoking prevalence.14

These ‘natural experiments’ consistently suggest that changes in cardiovascular risk factors can be associated with fast changes in CVD mortality in whole populations. Kuulasmaa et al. subsequently analysed data from 27 MONICA populations. The model of trends in 10-year coronary event rates against risk scores and single risk factors showed a mediocre fit, but this was improved with a 4-year time lag for coronary events.15

The inflammatory and thrombotic milieu is a very dynamic environment. The different pathological processes leading to plaque development, destabilization, rupture, and occlusion also demonstrate different time scales.16 It is thus interesting to hypothesize that trends in the determinants of atherosclerosis build up and trends in event triggering factors might act in different time frames; some very short indeed.

Moreover, the emerging links between metabolism, inflammation, and thrombosis16 likewise suggest that changes in diet and lifestyle could also alter short-term disease determinants within very short time scales. For example, the observed short-term effects on C-reactive protein and other inflammatory markers may explain the substantial CVD risk associated with trans fat consumption.17

Implications for cardiovascular disease prevention strategies

Increasing evidence suggests that policy interventions may represent the most effective and cost-effective public health approaches. In other words, making healthier diet choices easier, what Brownell calls the ‘default option’.18 The powerful levers of legislative regulation taxation and subsidization have been effective in diverse populations. Improving the contents of products, for example, by increasing the content of ω-3 fats (re-formulation); controls on the marketing of junk foods (energy dense, nutrient poor, high in saturated fat, salt, and sugar); clear package labelling reinforced by supportive taxation and subsidies.1 Reducing or banning trans fats,19 and Health promotion and legislative efforts to reduce the salt content of industrial food, is also equally important.20 Equally important and perhaps politically more popular would be policies to increase the affordability and availability of healthier foods, particularly an increase in the intake of fruits and vegetables.

All of these measures also make strong economic sense. The evidence consistently suggests that population-based CVD prevention is actually cost-saving.1,21 The NICE recently commissioned detailed economic modelling for the England and Wales population of 50 million.1 Reducing population cardiovascular risk by even 1% would generate discounted savings of approximately £260 millions (400 million) per year, resulting in 50,000 fewer CVD deaths each year. Furthermore, the model was based on conservative assumptions and probably seriously underestimates the true
health benefits. It did not quantify additional benefits to existing CVD patients, nor the inevitable reductions in other chronic diseases. The additional contribution of high-risk medical strategies, targeting individuals without CVD but at higher risk, cannot be ignored. If fully implemented in the UK, following the recommendations of the 4th Joint European Societies Taskforce on CVD prevention might prevent about 700 fewer deaths annually within 5 years. However, implementing these population-wide strategies could be politically challenging. Fatalism, pessimism, cynicism, and complacency all support the status quo. Furthermore, some libertarian politicians might instead wish to emphasize individual responsibility. But dietary interventions in individuals typically achieve only modest and transient benefits. Other politicians advocate voluntary agreements with industry (slow and relatively ineffective). Policy makers thus need to overcome their natural reluctance for legislative action. Furthermore, interventions may become easier when a deteriorating situation demands it. For example, when the obesity epidemic continues in spite of well meaning but ineffective initiatives. Or when, after decades of decline, CVD mortality rates in young adults are seen to plateau in the USA, UK, and elsewhere. A large evidence base exists. We know what to do, but we need to find out how best to do it. We need to better understand the social, economic, and legislative environment in order to facilitate healthier choices and reach every single individual in any population. More research is therefore needed on how to achieve these policy goals, how choices are being made by individuals, and also a better understanding of the dynamics of the biological mechanisms related to diet and lifestyle. In conclusion, population-wide interventions aimed at the major dietary determinants of CVD could have large and rapid benefits. These disease reductions might even be visible within the electoral term of many politicians. As well as health benefits, such strategies could also produce economic gains.

References