EDITORIAL

The promise and challenges of population strategies of prevention

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In public health, we are concerned with improving the health of populations. When considering prevention strategies, we have to decide between a population strategy and a high-risk strategy.¹ The population strategy aims to shift the distribution of a risk factor in the entire population whereas the high-risk strategy targets individuals in the population who are at highest risk of disease and might benefit most from prevention. A population-wide prevention strategy is often easier to implement as high-risk individuals do not have to be identified and targeted and may be more successful because it does not require behaviour modification and can be maintained indefinitely if appropriate. If a risk factor is well-established as causing disease, is difficult to control or modify on an individual level, and a shift of its distribution in the population does not move some people who are at low risk pre-intervention to a high risk post-intervention, a population strategy is the method of choice.

The elimination of artificial trans fatty acids from the food supply is a perfect example of a population strategy—and long overdue.² The detrimental effects of trans fat on human health are well established,³ avoidance of foods with trans fat is difficult for most individuals because few read and understand food labels and the hardened vegetable oils are hidden in many prepared dishes, and there is no known disadvantage to health from eliminating trans fats altogether. Trans fatty acids result from partial hydrogenation of vegetable oils and are popular among food manufacturers because they are inexpensive, increase shelf life time of processed foods and make fried and baked products such as French fries and cookies crispy. However, trans fatty acids increase low-density lipoprotein and lower high-density lipoprotein thereby promoting arteriosclerosis even at low levels of intake.

The New York City Department of Public Health is exemplary in the United States in being proactive about banning the use of the artery-clogging fats in restaurants, as described in this issue of IJE.⁴ As of July 1, 2008, restaurants are required to clear artificial trans fat from all their menu items. A similar ban was announced on July 25, 2008, for the state of California when Governor Arnold Schwarzenegger signed a bill to phase out trans fats in restaurants and retail baked goods by 2011. The beauty of a ban of the bad oils is that the consumer does not have to worry about their intake, hence difficult to accomplish behavioural changes are not required. Of course, this only applies to meals consumed in restaurants, while foods sold in manufacturer-sealed packages are exempt. Optimally, the hardened fats should be eliminated from the entire food supply. Can this be accomplished? Denmark set the standard making it illegal for any food to have >2% trans fat—offenders may face jail charges. The ban that went into effect in January 2004 has forced food manufacturers to be creative. A few years ago, McDonald’s claimed they could not reduce the excessively high trans fat content of their French fries without compromising their taste; but in Denmark—and now also in New York City—McDonald’s French fries are just as crispy with <0.5 g of fat per serving. There is no good reason to add an artificial disease-promoting substance to our food supply. Manufacturers and food suppliers must realize that by adding partially hydrogenated oils or vegetable shortening to their products they promote premature disease and death among their customers—not an easy moral burden to carry. Governments may have to take action to protect people’s well-being if the quest for profit trumps responsibility on the suppliers’ side.

But there is no free lunch. The required listing of trans fat contents on food labels has lead some manufacturers to replace them with naturally hard tropical oils, such as palm, palm kernel or coconut oil. Palm oil is one of the most widely produced edible oils. While these tropical oils do not contain artificial trans fatty acids, they are naturally very high in saturated

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fats which raise low-density lipoprotein levels. Luckily, the substitution of artificially hardened fat with tropical oils does not seem common practice.\(^5\)

New York City Health Commissioner Dr Thomas Frieden and Major Michael Bloomberg have to be commended for implementing a progressive and innovative public health programme in the largest and most challenging city in the United States.\(^4\) Besides the trans fat ban, Frieden and Bloomberg support legislation that would require chain restaurants to provide information on the caloric content of their foods on menu and menu boards with the ultimate goal of battling the obesity epidemic. Recognizing the shift of the burden of disease to chronic illnesses, Frieden and Bloomberg have implemented other bold approaches since taking office in January 2002; these include an aggressive increase in cigarette tax, which has reduced smoking among New York City public high school students by half, introduction of a diabetes registry that provides surveillance and improved patient care and free distribution of nicotine patches and gums.

Another population-wide prevention programme is the folic acid fortification of flour, cereal, pasta, rice and other grain products adopted by the United States government in 1998. The goal of this programme is the reduction of the incidence of neural tube defects in children of folate deficient mothers. While this intervention is geared to ensure sufficient preconceptual folate levels among women of reproductive age, the entire population is exposed to increased folic acid supplementation including men, children and the elderly. The question arises whether the disease risk of any of these groups is affected by shifting the distribution of folate status in the population to the right. Folate maintains cell renewal, is necessary for DNA synthesis and replication and reduces homocysteine levels and may thereby lower the incidence of cardiovascular disease.\(^6\) Folate also counters the effect of alcohol on breast cancer incidence.\(^7,8\) Based on these beneficial effects and its low toxicity, folic acid was embraced as a risk-free fortification programme. More recent randomized trial evidence suggests that folic acid supplementation may increase tumourigenesis among individuals with precursors of cancer,\(^9,10\) and also if given at very high doses.\(^11–13\) Animal studies support that dose and timing of folic acid intervention determine its effect: very high folic acid supplementation and intervention after early lesions may promote rather than suppress colorectal carcinogenesis.\(^14,15\) The essential role of folate in nucleotide synthesis, which aids growth and proliferation may provide a mechanistic explanation for any tumour-promoting effect. Furthermore, de novo methylation of promoter CpG islands of tumour suppressor genes may contribute to tumour progression,\(^16\) but the role of folate in these epigenetic modifications is not well understood.

However, no upwards trend in colorectal cancer mortality has occurred since folic acid fortification was introduced in the United States, in the contrary, mortality has decreased steadily over the past years. Any promoting effect on established tumours should have been reflected in an increase in mortality by now.

Dowd and Aiello report in this issue of IJE that while folate levels in the population have increased since the adoption of folic acid fortification in the United States, this increase has been larger among higher income socioeconomical classes and non-hispanic whites than among lower income classes and non-hispanic blacks.\(^17\) Dowd and Aiello suggest that this difference may be mainly due to differences in supplement use rather than consumption of fortified foods—a reasonable assumption since consumption of breads, pasta and rice is not likely to differ substantially by socioeconomical class. But just how much folate is best? The United States Public Health Service recommends a daily allowance of at least 400\(\mu\)g of folic acid intake per day. However, neither the optimal amount of folic acid intake nor the optimal blood level of folate are known. While the incidence of neural tube defects has decreased by \(~25\%\), since implementation of the fortification programme in the United States,\(^18\) it remains unresolved whether factors other than folate deficiency may account for the remaining cases. Dowd and Aiello propose a high-risk approach to folic acid fortification-targeting subpopulations with particularly low folate levels. Indeed, if folate deficiencies persist this may be a better strategy than shifting the entire population further to the right, since exposing individuals at high levels to even higher levels may be too much of a good thing. Additionally, high folate levels can mask a B12 deficiency, which can lead to irreversible neurological damage, a concern in particular among the elderly. Hopefully, additional data from population-based studies and mechanistic insights will inform the optimal prevention strategy.

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References
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