Population Health and the Hardcore Smoker: Geoffrey Rose Revisited

MICHAEL O. CHAITON¹, JOANNA E. COHEN¹,², and JOHN FRANK¹,³,⁴,⁵

¹Department of Public Health Sciences, University of Toronto, Toronto, Canada
²Ontario Tobacco Research Unit, Toronto, Canada
³CIHR – Institute of Population and Public Health, Toronto, Canada
⁴Institute for Work and Health, Toronto, Canada
⁵Canadian Academy of Health Sciences, Toronto, Canada

Correspondence: Michael Chaiton, T421-c/o OTRU, 33 Russell St., Toronto, Ontario, M5S 2S1, Canada. E-mail: Michael.Chaiton@utoronto.ca

ABSTRACT

The “hardening hypothesis” suggests that as smoking prevalence decreases, lighter smokers will quit first, leaving more “hardcore” smokers in the population. At a population level, however, the weight of evidence suggests that no hardening is occurring. By understanding the lessons from Geoffrey Rose’s model of population-level risk factor change, we argue that the hardening of the smoking population is not inevitable. The Rose model predicts that the effect of policy interventions, and changes in social norms, can shift the population-level risk distribution for continuing to be a smoker, making it more likely that all smokers will quit. This analysis also suggests that further reductions in smoking prevalence will not come without further changes in the underlying – and largely cultural – root causes of smoking in a population.


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Despite substantial reductions in smoking over the past 50 years in some countries in North America, Australasia, and Europe, tobacco is still widely used, remains the greatest cause of death and disease worldwide, and is a risk factor for six of the eight leading causes of death in the world (1). The World Health Organization has estimated that 100 million people have died of tobacco-related diseases during the 20th century and that if trends continue, another
500 million may die by the end of this century (1). Clearly, more efforts are required to continue to lower the prevalence of smoking in places where smoking rates are increasing such as China or sub-Saharan Africa (1) as well as countries such as Canada where almost one in five people continues to smoke (2).

McKinlay's population health model of prevention suggests that there are three levels of interventions: upstream (macro-level policies), midstream (community-level programs), and downstream (individual policies) (3). To date, successful interventions for reducing tobacco use have been upstream or midstream policies, particularly the following population-level strategies: cigarette tax increases, restrictions on smoking in public places and workplaces, restrictions on tobacco advertising, and mass media campaigns (4,5). However, in addition to having different demographic profiles, those who smoke today are hypothesized to be qualitatively different than previous smokers with respect to their motivation and ability to quit smoking.

The “hardening” theory, involving “hardcore” or “recalcitrant” smokers, goes as follows: given that societal pressures to quit smoking have been increasing over the last 50 years, many smokers who found it relatively easy to quit have done so already, leaving behind proportionately more “hardcore” smokers in the population – smokers who are less likely to respond to population-level tobacco control strategies, presumably due to their more severe dependence (6). This theory predicts that the population of hardcore smokers will continue to increase, relative to other smokers.

If the hardening hypothesis is correct, the concern is that previously effective population strategies will no longer be successful, thus requiring future tobacco control efforts to focus on inherently more intensive (and potentially more expensive) individual approaches to prevention, and to cessation in particular (4,6). The hardening hypothesis has been lent credibility by reports from clinicians, who struggle to help apparently increasingly challenging smokers, and from a meta-analysis by Irvin, Hendricks, and Brandon who found that smokers in clinical trials were becoming less likely to quit smoking than in the past (7).

Assume that a hardening population has practical implications that are already being seen in the literature. For instance, the promotion of products by public health authorities such as smokeless
tobacco, which have a measurable risk to health, can be more easily justified in a “hardcore” or high-risk population (8). Some other proposed “high-risk” strategies include the use of potential reduced exposure products (9), long-term use of pharmaceutical nicotine (e.g. Nicotine gum) (10), or the promotion of smokeless tobacco (8). If the target population of smokers is more at risk of continuing smoking and suffering harms caused by smoking, because they are more addicted and less likely to quit, this changes the risk/reward balance for future interventions.

It may be, however, that clinical experiences are misleading when trying to understand the overall picture of smoking at the population level, and that the findings presented by Irvin, Hendricks, and Brandon are mostly the result of the well-known “volunteer” selection bias affecting trial participants, which itself may have been shifting over time. In fact, at a population level, the current weight of evidence suggests that no hardening is occurring, a comprehensive National Cancer Institute monograph concluded (11–16). When followed over time, decreasing smoking prevalence in populations is consistently associated with decreased levels of dependence, more quit attempts, and more successful quit attempts (17).

In general, it has been found, as would be expected, that when smokers are followed longitudinally, less addicted smokers are more likely to quit (11,18–20). When measures of dependence are tracked in cross-sectional “snapshots,” however, the percentage of smokers in the population with high levels of nicotine dependence has shown declines or is stable (11,12,21). Nicotine dependence in these studies is measured using a number of metrics such as number of cigarettes per day, or one of the nicotine dependence scales such as the Fagerstrom Tolerance Questionnaire. In cross-sectional examinations of smoking status in the COMMIT study, for instance, the number of cigarettes smoked per day fell over time (15). The massive US Cancer Prevention Study also reported lower average cigarettes per day, and fewer people smoking large numbers of cigarettes, as the prevalence of smoking fell (11). In California, which has shown some of the biggest declines in smoking prevalence in the world, and thus should show the greatest hardening, the smoking population is arguably softening. The proportion of light smokers (<15 cigarettes per day) in California increased from
44% in 1990 to 60% in 1999; smokers reporting serious past-year quit attempts rose from 49% in 1990 to 62% in 1999; as well, the percentages of attempting quitters who succeeded (24%) and of so-called “hardcore” smokers who reported never expecting to quit (10%) remained similar in 1990 and 1999 (13). Such observations would be unexpected under the hardening hypothesis, where one would expect the less dependent smokers to quit, leaving a higher proportion of persistent smokers with high levels of nicotine dependence. Remaining smokers, according to the hardening hypothesis, should be more dependent smokers, with less desire and ability to quit.

How can one explain this apparent paradox that the percentage of smokers with high levels of nicotine dependence in the population is not increasing even though less dependent smokers are more likely to quit? While there are numerous models of behavior change in populations, there are few theories that predict what happens to the distribution of a population after upstream changes. Burns (14) explained the lack of observed hardening as an “interaction with the environment,” suggesting that the relative risk of quitting depends on “environmental characteristics” that promote or detract from attempting or succeeding in a quit attempt.

In fact, this sort of epidemiological phenomenon, where individual-level characteristics are not predictive of population-level changes in prevalence until one accounts for the effect of population-level environmental factors, has been described previously in another context by Geoffrey Rose, the father of modern chronic disease epidemiology. We propose that Rose’s model of population health can succinctly explain the apparent paradox, whereby declines in smoking prevalence are not accompanied by increases in the proportion of hardcore smokers.

Rose was an influential British cardiovascular epidemiologist, who argued that an awareness of the reasons why individual patients get sick can be insufficient for understanding the health of entire populations (22). He maintained that it can sometimes be more enlightening to have an appreciation of the reasons why a population has a particular distribution of risk (23,24). Rose’s hypothesis has been shown to be an illuminating model of population-level changes in risk factor distributions over time, and of differences between entire populations’ distributions of chronic disease risk factors (25–29).
Recent changes in blood pressure distribution over time in Britain followed exactly the kind of “whole-curve shifts” hypothesized by Rose – reduction in population averages were seen, alongside corresponding increases in the proportion of people with low blood pressure, equal to decreases in the proportion of those at “high levels” (25). If changes in blood pressure distribution in a population were due to increased or more effective clinical diagnosis and treatment, then one would expect the curve to be “dented in” only at the high-risk end of the blood pressure spectrum, where individual patient diagnosis and treatments would necessarily be focussed. Instead, the distribution of Britain’s blood pressures over time – measured in a careful longitudinal study – shows no “indenting” of the right side of the distribution at all, but only a clear shift to the left (downwards) of the entire distribution. This suggests that changes in the social determinants of blood pressure (such as nutrition) affected the whole population, including the high-risk end of the spectrum.

The hardening hypothesis for smoking can be compared to the “indentation” hypothesis for blood pressure, in terms of changes in population distribution of high-risk subgroups. While it does not work on all levels, the blood pressure analogy is an example of a model of change in health behaviors and outcomes. The hardening hypothesis suggests that we should currently expect reductions in smoking prevalence to be accompanied by increases in recalcitrance or hardening, as measured by such items as smokers’ levels of nicotine dependence, unwillingness to quit smoking, or likelihood of quitting smoking. Specifically, one should expect to observe an “indenting” over time of the left-hand side of the hypothetical distribution of “hardcore smokers” among smokers (Figure 1), precisely because the fewer hardcore smokers have quit. But what if the entire distribution of that risk has actually shifted downwards, due to profound cultural changes in recent decades? Such a “softening” in persistent smokers’ levels of hardcore-ness would then be consistent with reduced smoking prevalence, as suggested by Rose’s model of population-level distribution shifts. To put it another way, the reasons why an individual continues smoking may not explain why a population has a particular distribution of smoking.

In applying Rose’s thinking to the apparent hardening paradox, we are not suggesting that clinical detection and treatment of
high-risk individuals has been unimportant. (A test of that hypothesis would require more rigorous analysis and other study designs.) Indeed, aggressive clinical management of the individual smoker is necessary, useful, and arguably ethically mandated (30) for primary care practitioners, and can have great success in individual cases. Rather, we suggest that failure to apply Rose’s thinking to the smoking problem has led to the identification of a “false paradox.”

Although there are many implications of Rose’s model, one conclusion suggests that while focusing on individual patients with high levels of risk can have benefit for the patient, this approach does
not address the underlying reasons for the elevated risk-factor prevalence in a population, and thus cannot prevent the incidence of new cases. Such an approach will not address the “causes of the causes.” (31) A change in “upstream cultural and behavioral forces,”(30) on the other hand, addresses the underlying causes of the risk factor’s incidence and will have much larger and more permanent effects on population outcomes, reducing risk for the many (largely young) persons with early or low-risk factor levels – in this case, the tendency to start and/or to continue smoking – thus addressing the root causes of the problem, in an “upstream” fashion.

It seems likely to us that the range of upstream policies and programs for tobacco control implemented in recent decades, such as reductions in the accessibility of tobacco products, measures tackling the environmental cues for smoking, and well-designed campaigns to reduce the social acceptability of smoking, have increased the probability of many smokers – at all levels of dependency – successfully quitting, reduced their chances of relapse, and made it less likely for younger people to start smoking in the first place. Some of these measures may not have had large individual effects, but a small reduction in the chance that an individual might start to smoke, and an increase in the chance that a smoker might quit, spread over the entire population, have likely led to substantial population-level effects.

A reduction in the prevalence of current smoking may well reflect the fact that everyone has become less likely to smoke, including the “hardcore,” because the underlying reasons for smoking, and not smoking, in society, are being addressed (24). It may well be that some “hardcore” smokers are less likely to quit, over time, but that smokers in general are becoming more likely to quit and new smokers entering or re-entering the smoking population are less likely to be hardcore than in the past. Open cohort analyses, and more detailed examination of the natural history of smoking behavior over the life course, are needed to determine whether this is indeed occurring.

Although a number of population-level interventions have already been implemented widely in North America, Australia, parts of Europe, as well as new approaches in Thailand, South Africa, and Brazil, we have not had prolonged experience with many of them (1). For the relatively new interventions, our experience with their
long-term impacts is necessarily limited. We hypothesize that a continual increase in the “dose” of these population-level interventions, particularly as the Framework Convention on Tobacco Control is implemented around the world, will result in continued declines in smoking prevalence, and more “softening” among persistent smokers. Merely maintaining the status quo with these initiatives, while redirecting effort and resources into clinical treatment, will not have equivalent impact on smoking overall, because the latter “high-risk” approaches have inherently limited ability to change the underlying structural determinants of smoking initiation and continuation.

Despite current evidence to the contrary, the hardening hypothesis remains a compelling model for many researchers and clinicians trying to anticipate the effects of declining smoking prevalence. The hardening model is intuitive, and presents a much-welcomed alternative to renewed calls for just “more of the same” in tobacco control. We suggest, however, that there is no necessary scientific reason to believe that reductions in prevalence will necessarily make continued smokers more resistant to quitting. The apparent obviousness of the hardening hypothesis may be a result of the so-called “atomistic” fallacy of inferring population outcomes from individual-level data (32,33). To understand how, and how much, various population-level factors – including various broad tobacco control policies – affect prevalence and dependency, more research is surely needed. Studies such as the International Tobacco Control Four Country Survey (now expanded to more countries) may start to address some of these issues with a powerful research design that can compare the effect of interventions from jurisdiction to jurisdiction (34).

We agree that it is not enough to argue for “more of the same” in tobacco control, if some root causes of smoking are being neglected in current population-based interventions. In some subpopulations, the prevalence of smoking is still very high, suggesting that there is more to be done in changing the underlying causes of smoking – both starting to smoke, and failing to quit – in these subpopulations. New threats, such as changes to cigarette design that may make cigarettes more addictive, or novel methods of marketing new tobacco products, may change current trends, reversing declining prevalence in some countries.
We must and can do more to intervene against the underlying causes of smoking, whether these are at the individual or the population level. The evidence does not yet suggest that a hardening of the population. Further, we suggest an alternate health behavior model, like the one put forward by Geoffrey Rose. It suggests that hardening will not necessarily ever happen, and that consequently, the burden of proof must be placed on advocates of high-risk interventions to demonstrate that the marginal yield per unit cost of individual-based efforts is competitive with the efficiency of population interventions. A “balanced portfolio” of such interventions must surely include policies, programs, and practices targeted to both levels.

ABOUT THE AUTHORS

Michael Chaiton is a 4th-year Ph.D. candidate in Epidemiology at the University of Toronto and was introduced to tobacco control in 2000, working for Physicians for a Smoke Free Canada and the International Non-Governmental Coalition Against Tobacco.

Joanna Cohen is Director of Research and Training at the Ontario Tobacco Research Unit and an associate professor in the Department of Public Health Sciences at the University of Toronto. Trained in epidemiology and health policy, her research interests focus on the factors that affect the adoption and implementation of public health policies and on evaluating the beneficial effects and the unintended consequences of such policies.

John Frank is the Scientific Director of Canadian Institutes of Health Research, Institute of Population and Public Health, as well as a Professor in Public Health Sciences at the University of Toronto, and a Fellow of the Canadian Academy of Health Sciences.

REFERENCES


