BEHAVIORAL HEALTH INTERVENTIONS: WHAT WORKS AND WHY?

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Some interventions must ultimately be implemented to reduce racial, ethnic, and socioeconomic disparities in health. Designing such interventions is difficult, however, since not all interventions are equally efficacious. This paper reviews what is known about the success and failure of behavioral interventions and why some interventions succeed.

By behavioral interventions, I mean interventions designed to affect the actions that individuals take with regard to their health. The typical medical intervention is a clinical trial of a particular drug, surgery, or device. In the trial, doctors provide different services to different people, and then evaluate the outcomes. Variation in patient behavior is generally shunned; a strong emphasis is placed on making sure that patients do exactly what is expected from them. With behavioral interventions, in contrast, patient behavior is the key and the goal is to change it.

Behavioral interventions can be implemented at three levels.[1] The first are *individual* interventions. These interventions encourage people who are at high risk for a particular disease to do something about it. Examples are programs to encourage smokers to quit, or programs to encourage people at high risk for heart disease to take steps to reduce their risk. These steps involve lifestyle changes (eating well and exercising) and medical changes (regular testing of blood pressure and cholesterol). In both cases, though, the actions taken are controlled by the individual.

The most important individual intervention is the the Multiple Risk Factor Intervention

[1] Given the limits of this paper, my discussion is necessarily brief. For a more complete discussion of many interventions, see Powell (2001).

Trial (MRFIT) conducted in the 1970s. MRFIT was designed to induce men at high risk for heart disease to lower their blood pressure and cholesterol, and to stop smoking. It enrolled over 12,000 men in a trial lasting 6 years. The men received counseling and help with behavior modification. As I discuss below, the trial was only partly successful. Risk factors changed by more in the treatment group than in the control group, but the impact was less than was hypothesized. Further, mortality outcomes for the treatment group improved only slightly more than did outcomes for the control group.

The relative failure of individual interventions was interpreted by many as evidence on the importance of environmental factors in health. Individuals are products of their environment, the theory went, and thus one cannot change the individual without changing the community they live in. This led to a second type of intervention *community* interventions, designed to change behavior by modifying environments. Several community-level interventions were implemented in the 1980s. The setting was again risk for cardiovascular disease. These interventions used mass media, population screening, and community organizations to convey messages of healthy behavior. The results of these trials were very disappointing. Risk factors and health outcomes did not improve any more rapidly in the intervention sites than in the control sites.

While community-level behavioral interventions were not very successful, legislative changes at the community level were more successful. Governments that tax cigarettes uniformly find that the consumption of cigarettes drops. Restrictions on where people are allowed to smoke also lower consumption. Public policies can have large effects on health behavior, but it is different policies than just encouragement.

Beyond the community level, the third level of health intervention is at the national level.

The Federal government or private groups often convey health information to people, with the goal of encouraging behavioral change. These national interventions have a much more successful record than do community interventions. The campaign launched by the Surgeon General in 1964 to warn people of the harms of tobacco has been an important part of a nearly 50 percent decline in smoking. The anti-drunk driving movement pushed by Mothers Against Drunk Driving and the designated driver campaign over the past two decades has reduced the share of traffic fatalities involving drunk drivers by over a third. And information about the danger of high cholesterol has led to sustained reductions in red meat, eggs, and high-fat dairy products.

Determining why the national interventions had salient effects while individual and community level interventions had no effect or a much smaller effect is difficult. I do not have a definitive answer but I discuss several theories. The first is a theory of *permeability*. People would prefer not to change their behavior. Inertia is strong, and changing behaviors requires major changes in thinking and action. Health messages are easier to ignore when the intervention is small; there is no pressing need to respond to each such impulse. But when information permeates widely, it is difficult to continue on the old path without contemplation. Doing nothing becomes a choice in itself that individuals must make. At such moments, people may be more willing to undertake large changes in behavior.

The second theory is one of externalities. Many of the national interventions justified individual action by noting that people doing the activities were hurting others in addition to themselves. Examples of these externalities include the movement against drunk driving (drunk driving kills children) and the argument against smoking (passive smoking has adverse health

consequences). It is possible that highlighting these external consequences induces more behavioral change than simply stressing the benefits of behavioral change to individuals.

The third theory is of peer effects. People may judge appropriate behavior on the basis of what others are doing, as well as their own utility from the activity under question. Thus, changes in the share of people who engage in a certain behavior, for example smoking, may affect the decision of even heavy smokers to quit.

I present these theories but do not offer direct evidence for or against them. I do highlight several other theories that do not seem supported by the data. Some speculate that individual and community interventions do not work as well as national interventions because they are not implemented for a long enough period of time. But many national interventions achieve large behavioral changes within a shorter period of time than typical individual and community level interventions. Similarly, the nature of the information provided does not seem to be so important. National intervention campaigns have succeeded when their message is positive (you should help yourself by quitting smoking) or negative (you are evil if you drive while drunk). The salience of the information may be more important than the positive or negative way the information is conveyed.

This paper is structured as follows. The next section briefly outlines the nature of behavioral interventions. The following three sections consider evidence on the effectiveness of individual, community, and national level health interventions. The final section concludes by discussing the common features of successful interventions.

I. The Nature of Behavioral Interventions

Health behavior encompasses many facets, and so behavioral interventions are broad as well. To introduce the subject, it is helpful to consider a particular example. Much of the literature focuses on cardiovascular disease, so I do the same.

The process of cardiovascular disease begins with risk factors attributes of individuals that make them more likely to have a serious medical event. Some risk factors are exogenous to the individual, such as a family history of heart disease. Other risk factors are under the control of the person. These factors include hypertension, high cholesterol, smoking, obesity, and diabetes. People with elevated risk factors are more likely to suffer a serious adverse event than people at lower risk. The most common such events are heart attacks and strokes. Death is a common outcome of an acute event. For those who survive the acute event, there is then a subsequent period where risk remains high.

The classic medical intervention is in the treatment of people with a heart attack. There are a range of possible therapies, from medications to balloon angioplasty to coronary artery bypass surgery. The relative efficacy of these therapies has been evaluated in clinical trials. Similarly, clinical trials have examined which medications are most effective in managing hypertension, high levels of cholesterol, and diabetes given the frequency of usage and dosage strength. Some of these changes increase racial and ethnic disparities in health, while others diminish disparities. Given the large number of such innovations, I do not survey it here.

Behavioral interventions are targeted to all of the other factors. A simple intervention would be encouraging people to stop smoking. A more complex intervention would target

people with several risk factors and encourage a variety of behavioral changes eliminating cigarette smoking; lowering consumption of fatty foods; reducing overall calories; exercising more; visiting physicians for hypertension and cholesterol screening; and adhering to medication guidelines. Behavioral interventions are not independent of medical care; indeed, receipt of medical care is a key part of such interventions. But the idea is to change the actions of people rather than to act on them passively.

There are other interventions that bridge medical and behavioral factors. For example, physicians may not order the appropriate tests for measuring cholesterol, or may not prescribe the correct medications for reducing it. Some recent interventions have targeted physician behavior to correct these limitations. There are no definitive studies on physician interventions (Powell, 2001), however, so I do not consider such programs in any depth.

Individual behaviors might be modified in several ways. One possibility is to target high risk individuals and encourage behavioral changes among this group. This is the right strategy if individuals are autonomous actors. But it might fail for two reasons (see Rose, 1992). First, if there is a continuum of risk in the population, people with very elevated risk factors may not be the major source of adverse outcomes. A lot of people with a small excess risk could produce more adverse health outcomes than a few people with very substantial risk. Second, there may be peer effects that link high levels of risk to the average level of risk. If the behavior of outliers is influenced by the vast majority of the middle, targeting community-wide behavior could be more effective than trying to treat only the very high risk. For example, a number of epidemiologists have argued that it would be more effective to reduce rates of high cholesterol by encouraging better eating and exercise patterns throughout society, allowing for the feedback of

that strategy on the high risk population, than by seeking out and treating those with very elevated cholesterol levels.

For this reason, a second possibility is to target particular communities and modify the environments people live in. Community-level interventions rely on changes in the environment and on peer pressure to control the risk of disease. National interventions have both individual and community features. They convey information to particular people and encourage community-wide involvement. In the next sections of the paper, I evaluate individual, community, and national interventions.

Before doing so, however, I present a little more information on cardiovascular disease mortality. Figure 1 shows cardiovascular disease mortality over time for different racial and ethnic groups. Since 1950, cardiovascular disease mortality has declined across the board. Among white males, for example, mortality fell by 52 percent. For both men and women, the racial gradient in cardiovascular disease mortality increased. The relative change was largest for men. Compared to the 52 percent decline in cardiovascular disease mortality among whites, mortality for blacks declined by only 36 percent. The changes are close for women 54 percent for whites compared to 46 percent for blacks. The increased racial gradient in mortality suggests the importance of understanding how interventions affect particular racial and gender groups. I focus on this in the analysis.

II. Individual Interventions

The most important individual interventions in health behavior were conducted in the 1970s. Knowledge of cardiovascular disease risk factors solidified in the 1960s. Results from the Framingham Heart Study and other research efforts demonstrated the importance of several risk factors for cardiovascular disease: hypertension (or high blood pressure); high cholesterol; obesity; smoking; and diabetes. The natural policy goal was to intervene to change these risk factors. In the 1970s, experiments were designed to do just this. The most important of these interventions was the Multiple Risk Factor Intervention Trial (MRFIT) (MRFIT, 1982, 1990, 1997; Gotto, 1997).

The MRFIT was initiated in 1972. Over 350,000 men aged 35 to 57 were screened to produce a sample of about 12,000 men at high risk for coronary heart disease. The screening focused on blood pressure, cholesterol, and smoking status. The final sample was 12,866.

Eligible individuals were divided into two groups. Members of the control, or usual care group, were examined once a year for medical history, physical examination, and laboratory results. The results of the screening and lab exams were conveyed to their primary care physicians, but no other intervention was undertaken. Members of the treatment, or special intervention group, received several interventions. Smokers were counseled by physicians to quit smoking. All intervention members were invited to attend weekly discussion groups addressing control of risk factors. After an intensive initial phase, participants in the intervention group were seen every four months, where they received individual counseling from a team of behavioral scientists, nutritionists, nurses, physicians, and general health counselors. The

intervention lasted for six years.

The MRFIT investigators expected significant reductions in all three risk factors. It was hypothesized that cholesterol would decline by 10 percent for men with elevated levels (220 mg/dL), diastolic blood pressure would decline by 10 percent for those with high levels (95 mm Hg) and that somking would decline from 20 to 40 percent depending on the initial level smoked (Sherwin et al., 1981). If achieved, these changes would translate into a 27 percent reduced chance of coronary heart disease mortality.

Table 1 shows the results the trial actually produced. For each of the three risk factors, there were improvements in risk factors for the intervention group. Blood pressure declined by 12 percent, smoking fell nearly in half, and cholesterol was lower by 5 percent.[2]

But there were also favorable changes in risk factors in the control group. Aside from smoking, where some reduction was expected in the control group, these risk factor changes in the control group were not expected. As a result, the net change in risk factor control for the intervention group was below expectations. Cigarette smoking declined by more than the forecast amount, but the decline in blood pressure was only 75 percent of expected levels, and the decline in cholesterol was only half of expected levels. These differential changes between the treatment and control group were statistically significant, but were smaller than expected. The behavioral intervention worked, but not to the extent forecast.

Before moving on to the mortality outcomes, I note the racial homogeneity of the MRFIT results. Figure 2 shows the relative change in risk factors for whites and blacks in the

[2] Note that these are averages over the entire population of men enrolled in the trial, so they are not readily comparable to the goals for men at high risk on any particular dimension.

intervention group compared to the treatment group (Connett and Stamler, 1984). For each of blood pressure, cholesterol, and smoking status, changes were similar for blacks and whites indeed, if anything a bit larger for blacks than whites. Since blacks are more likely to be hypertensive than whites, this part of the intervention reduces racial disparities in health.

The ultimate end-point for the study was mortality. The mortality effects are shown in the next row of Table 1. These changes are even smaller. Coronary heart disease mortality was only 7 percent lower in the treatment group than in the control group, and overall mortality was slightly higher. Neither estimate is statistically significant.

There are two disappointments in this trial the lower than expected effect of interventions on risk factors, and the small translation between risk factor changes and mortality. The second issue has been investigated more extensively than the first. The leading hypothesis is put forward is that risk factor reduction did not translate into large net mortality improvements because one of the antihypertensive medications used was actually harmful to some men. For men with ECG abnormalities at baseline, use of hydrochlorothiazide (a type of diuretic) was associated with increased mortality. On the basis of this evidence, in the fifth year of the intervention, a decision was made to replace use of hydrochlorothiazide with chlorthalidone (a different diuretic).

In a follow-up several years after the intervention was completed and 10½ years after the trial began, the differences in mortality between the treatment and control groups were larger (11 percent for coronary heart disease mortality, 8 percent for total mortality) but still not statistically significant (one-tailed p=.12 and .10). This change was consistent with an adverse effect of the antihypertensive medication. The same conclusion was reached at a 16 year evaluation published

late in the 1990s. Mortality was lower for the treatment group compared to the control group (11 percent for coronary heart disease mortality, 6 percent for total mortality), although again the results were not statistically significant.

Perhaps more important for this paper is the fact that the behavioral interventions had less impact than expected. There is mixed evidence on the extent of behavioral change. Smoking cessation was more successfully accomplished than was expected. Hypertension control (largely through medication) was close to expectations, while cholesterol reduction (largely through weight reduction) was farther away. The social component of the experiment was not a failure, but it was not a complete success either.

There are several possible explanations for this mixed record. A first explanation is that the 6 year trial was not long enough to effect significant behavioral changes. Without continuing the experiment longer, it is impossible to test this theory. I suspect the theory is incorrect, however. If this theory were correct, the change in risk factors between the treatment and control groups should be increasing over time, as more treatment group members adopt healthier lifestyles. In fact, however, the risk factor change is relatively constant from year 1 to year 6 (MRFIT, 1982).

A second theory is the effect of background changes. In the study design, it was assumed that there would be no major change in risk factors in the control group, other than a modest reduction in smoking. In fact, large changes occurred in all three of the risk factors. It is possible that even the modest intervention for the control group annual risk factor measurement and referral to a doctor for care led to changes in behavior for this group. But a comparison of those in the control group with those at high risk but not in the trial suggests this is not the case

(Luepker, Grimm, and Taylor, 1984). Rather, the control group improved because the population as a whole was improving and the treatment had some impact above that, but not an enormous amount.

The background changes are not hard to divine. Over this time period, there was a great deal of public attention focused on the dangers of hypertension and smoking, and some focus on cholesterol as well. The issue is why the intensive intervention was not even more successful.

One possibility is that the background knowledge dissemination was close to mimicking what the treatment group received. Thus, there might have been little additional information from the intervention. This explanation is not very satisfying, though. One of the premises of the MRFIT trial was the information itself was not enough. Just telling people to quit smoking or exercise more, it was assumed, would not be sufficient to induce smoking cessation or greater physical activity.

A more refined version of this theory is that only a certain number of people are susceptible to change, and that some of this susceptible population in the control group was reached through general information. The remaining group of susceptible people in the treatment group might thus be smaller. In hindsight, however, this theory seems incorrect as well. After the MRFIT trial ended, there were continued reductions in blood pressure, cholesterol, and cigarette smoking in the population as a whole (although there was some reversion among the intervention group [Cutler et al., 1991]). There is no evidence in the aggregate data that society had reached the endpoint of behavioral changes to improve cardiovascular disease health by the end of the MRFIT trial or indeed that we have reached it now.

A third explanation is that the trial was unsuccessful because the behavioral intervention

was poorly designed. There are two possible avenues of this explanation. First, the focus on individual behavior leads to a fear of blaming the victim. If people are told high risk factors are their fault, they may resist change to avoid admitting responsibility. In this theory, one needs more positive messages than negative ones. A second issue is that the intervention focused on individual change but ignored the environment the person lives in. Eating better is difficult if one's family and friends do not change their eating patterns. Smoking cessation is harder when a person's co-workers and family continue to smoke. In this theory, the focus should be on community-level interventions, rather than individual-level interventions.[3]

This latter argument was convincing to many. The failure of the MRFIT to achieve risk reduction on the scale hypothesized led to a series of community-level interventions to reduce cardiovascular disease risk. I describe these community-level interventions in the next section. As a prelude, however, I note that the community-level interventions were not very successful either. Thus, this interpretation is probably not right.

From today's perspective, it is not clear why the MRFIT trial failed to have the impact on behavior that was hypothesized. In the last section of the paper, I suggest it may have to do with the degree to which the MRFIT information forced the men to re-evaluate their lives, or to consider the external effects of their actions.

[3] There is some evidence that women whose husbands were in the MRFIT were more likely to change their risk factors than were women with husbands who were not enrolled see Sexton et al., 1986.

III. Community-Level Interventions

The successor to individual-level interventions were community-level interventions, designed to change the environment as a whole. I discuss these interventions in two strands. The first are community-level experiments designed to encourage better health behaviors. The second are public policy interventions such as taxation and regulation that affect what people are allowed to do or the price they pay for doing things.

A. Community-Level Health Promotion

The implication some people drew from the MRFIT trial was that individual interventions are not enough. Peoples actions cannot be separated from the environment they live in. Changing individual behaviors thus requires changing the environment as a whole. The logical implication of this finding is that trials need to be undertaken at the community level, rather than at the individual level.

This conclusion was acted on in the 1980s. Three community interventions were sponsored in that decade, again with the goal of reducing cardiovascular disease risk.[4] The three interventions were the Stanford Five City Project (Farquhar et al., 1990), the Minnesota Heart Health Program (Luepker et al., 1994, 1996), and the Pawtucket Heart Health Program (Carleton et al., 1995).

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[4] I focus on the trials in the United States. There was another trial in Finland that is not discussed. The conclusions are similar, however.

Table 2 describes these trials and the individual results. Each trial had one or more treatment cities matched with control cities (2 treatment and 2 control cities in the Stanford Five City Study; 3 treatment and 3 control cities in the Minnesota Heart Health Study; and 1 treatment and 1 control city in the Pawtucket Heart Health Program). The interventions began in the early 1980s and lasted for 5 to 7 years. Data collection began before the intervention and continued for a short period of time afterwards.

While the goals of the experiments were similar to reduce coronary heart disease risk the interventions differed somewhat across sites. The Stanford Five City Project focused on mass media (TV, radio, and newspapers) and direct education (classes; pamphlets and kits; newspapers and letters). Treatment cities received continual exposure to cardiovascular disease education campaigns, along with four to five separate risk factor education campaigns per year. In addition, there were school-based programs for children. The researchers estimated that each adult in the treatment cities was exposed to 527 educational episodes over the five year period of the trial, or about 26 hours per adult.

The Minnesota Heart Health Program also used mass media to provide risk factor messages and establish awareness of the program. In addition, health professionals were involved in encouraging healthier behavior. Finally, risk factor screening and individual education were carried out. About 60 percent of adult residents received on-site measurement, education, and counseling; about 30 percent participated in face-to-face intervention programs. The messages stressed self-management and included changes in behaviors, the meaning of those behaviors, and the environmental cues that supported those behaviors. The experiment itself lasted about 5 years.

The Pawtucket (Rhode Island) Heart Health Program focused on community involvement in behavioral change rather than mass media. Schools, religious and social organizations, large employers, and city government were recruited to encourage behavioral change. The focus of these interventions was to promote awareness and agenda setting, and train people in skills needed to change behaviors and sustain those change. Particular emphasis was placed on nutrition, blood pressure, and weight programs. In addition, grocery stores labeled low-fat foods, exercise courses were installed in the community, restaurant menus highlighted heart healthy foods, and nutrition programs were available in public libraries. It is estimated that the 70,000 people in Pawtucket had over 110,000 contacts with the program. People particularly liked the nutrition, blood pressure, and weight programs.

In each case, the interventions were more than just the dissemination of knowledge. To be sure, knowledge dissemination was important. But each of the studies also stressed messages from social learning theory people had to learn how to take actions for themselves, and what the impact of those actions would be. Further, there was emphasis on using the medical system appropriately for example to screen and treat hypertension and high cholesterol. People were not just advised and then left on their own.

In all cases where the data were measured, awareness of cardiovascular disease risk rose in the treatment cities compared to the control cities. In the Stanford Five City Project and the Minnesota Heart Health Program, for example, knowledge of coronary heart disease risk factors rose significantly more in the treatment group than in the intervention group. Thus, the programs achieved their first goal of making people aware of disease risk.

But the other goals were nowhere near as successful. In each of the sites, there were

positive changes in risk factors for the treatment cities, but also for the control cities. The differential change in risk factors was much smaller, and generally statistically insignificant. There were some successes: blood pressure and obesity declined mildly more in the treatment cities than the control cities in the Stanford Five City Project; physical activity increased more in the treatment cities in the Minnesota Heart Health Program; and BMI increased less in the treatment city in the Pawtucket Heart Health Program.

But these successes need to be contrasted with the much greater failures of the interventions. There were no differential changes in smoking in the treatment cities compared to the control cities, cholesterol was generally unaffected, and blood pressure was mostly unaffected. Obesity did not change significantly.

The samples involved in each case were small, sine the unit of analysis is the community rather than the individual. But even pooling the data does not suggest large intervention effects. Winkleby, Feldman, and Murray (1997) estimate that smoking rates fell an average of -.3 percent per year in the treatment cities compared to the control cities (p=.54), diastolic blood pressure fell by -.1 mm HG per year (p=.68), and cholesterol rose by .23 mg/dL per year (p=.66).[5] Overall mortality risk was only negligably affected. This matches the empirical results. The Minnesota study did not find significantly different trends in outcomes between the treatment and control cities. The Stanford study found some changes in outcomes, but only for selected people and for a limited period of time.[6]

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[5] These results are for men. Changes for women are very similar.

[6] The Stanford study found evidence of significant health changes using a cohort sample but not a cross-section sample. Effects were also larger in the 2 to 4 year interval rather than the 6 year interval.

Thus, the overall conclusion from the community-level studies is that the interventions were largely ineffective in modifying cardiovascular disease risk. This conclusion is particularly important in light of the very substantial cost of running community-level interventions. The Stanford Five City Project, for example, cost $4 per person per year (in 1980s dollars).

Once again, it is important to note that the control cities had changes in behavior as well. Indeed, the improvement in the risk factor profile in both treatment and control cities was large; only the differential between the two was small.

There is no consensus for why the community-level interventions fared so poorly. It is possible that the community-level interventions failed because they were not carried on long enough to have a significant effect on health behaviors. This seems unlikely, however. In the Stanford Five City Project, the effect on health behaviors was greatest after two to four years, and then declined by year six. In Minnesota, the same pattern was observed in health knowledge and those behaviors that were statistically significantly different in the treatment cities. The time period examined was when the program had its maximal effect; the impact was actually declining by the end.

Further, it is not a case of lack of effort. As best as can be told, the message did get out. Knowledge of cardiovascular disease risk improved when it was measured, and people interacted with the program in the intended ways. Rather, the knowledge did not produce appropriate action.

A third explanation is that the community is not the right level to target. People may take social cues from areas larger than just their local community. In each of the sites, careful attention was directed to this issue. The communities chosen were relatively homogenous and

stable. They were not immediate subsets of a larger metropolitan area, where other messages might conflict. Thus, while the contamination explanation cannot be discounted, it is not likely.

A final explanation is that the programs were not large enough to have the intended effect. While the interventions cost several million dollars each, they did not fully saturate the communities. It may be that the effects of the nutrition and obesity messages were drowned out by the enormous volume of food advertizing on TV and radio. The national level data on eating behaviors presented below suggests that larger interventions may well have bigger effects than smaller interventions. If so, this argues that only major changes in policy will affect racial and ethnic disparities in health.

B. Public Policy Interventions

In addition to community-level behavioral interventions, public policy changes have been enacted to influence health behaviors as well. The most important public policy intervention for health is in the area of cigarette smoking. I thus focus on this example.

Public policy affects smoking in several ways. A first mechanism is through taxation. Along with the Federal government, most state governments tax cigarettes. These taxes are almost uniformly passed through into prices (Evans, Ringel, and Stech, 1999) and thus affect the cost of cigarettes for smokers. Governments also spend money on anti-tobacco advertizing, with the goal of counteracting the advertizing done by cigarette companies and encouraging people to quit the habit. Finally, the public sector regulates who can smoke and where smoking can occur. Cigarettes are not allowed to be sold to minors (although this is frequently violated), and smoking is now prohibited in many buildings and public spaces.

A vast literature has evaluated the impact of these public policies on smoking behavior. Chaloupka and Warner (2000) and the Surgeon General (2000) review this evidence in detail; I summarize it briefly. The most research has focused on the impact of cigarette taxes on utilization. The methodology for measuring the price effects of cigarettes is straightforward. Different states raise tobacco prices at different times. As a result, one can compare cigarette usage before and after the tax increase, differentiating between treatment and similar control states.

The results of these studies uniformly show large demand responses to price increases. A consensus estimate is that the elasticity of demand for cigarettes is about -0.4 every 10 percent increase in price reduces consumption by 4 percent. Further, the poor seem to be more affected by prices than the rich. Gruber and Kosygi (2002) estimate that the cigarette price elasticity for the poor is greater than -1 in absolute value; the price elasticity for the rich is much smaller. The finding that cigarette taxes discourage utilization is not in very much dispute.

Other public policies also affect cigarette consumption. For example, broadcast advertisements of cigarette ads were effectively banned in 1971. The ban seemed to reduce consumption, but the effect of this ban is modest. Many authors have found a small impact of the advertising ban on consumption, although others have not. Even the studies finding an effect estimate it to be relatively minor. In part, this may be attributable to the many other ways that cigarette companies can advertise their products, for example through newspapers, magazines, and direct promotion.

Somewhat more effective is anti-tobacco advertising. Such advertising was conducted at the Federal level in the 1960s, and has more recently been the province of state governments. In

each case, evidence suggests relatively sizeable impacts of anti-tobacco messages on consumption. For example, California spent $26 million in the early 1990s on an anti-tobacco media campaign. Hu et al. (1995) estimate that smoking declined by 8 packs per person in response.

Finally, public policies that regulate access to cigarettes and appropriate places for cigarette smoking seem to affect consumption as well. In recent years, many governments have adopted smoking bans in particular areas, including elevators, public transportation, government buildings, restaurants, shopping malls, and private workplaces. Most of the economic studies of these restrictions find large impacts on consumption, particularly as the regulations become more comprehensive. Workplace smoking bans, for example, are estimated to reduce the share of workers smoking by 5 percent and overall cigarette consumption by 10 percent (Evans, Farrelly, and Montgomery, 1999).

Restrictions on places where people can smoke may affect cigarette consumption in two ways. First, it increases the price of cigarettes. People who must go outside to smoke effectively face a higher cost of cigarette consumption (although not in dollars). Second, it may increase the stigma associated with smoking, or reinforce in peoples minds the harms from smoking.

The distinction between price and non-price effects is important in designing public policy. While price increases are a good way to discourage smoking, price increases have distributional implications that need to be considered as well. Since lower income people smoke at much higher rates than higher income people, tax increases would be paid more by lower income people than by higher income people. Some have criticized this on distributional grounds (although the benefits of smoking cessation go to lower income people more than higher

income people as well). The very large effect of the workplace smoking bans, combined with the results from limiting tobacco advertising and sponsoring anti-tobacco advertising, suggest that non-price policies may be important to combine with price changes.

C. Summary

Overall, there is a mixed message about the impact of community-level interventions on health. Experimental programs to change community environments and encourage healthy behavior have generally been ineffective. But price and non-price factors undertaken by governments have had a bigger impact on behavior.

It is not clear how to explain the difference between these divergent findings. One hypothesis is that the community-level intervention trials were not large enough to add to the background information people were already seeing. The Surgeon General suggests this explanation in a recent report on smoking cessation (Surgeon General, 2000). Alternatively, the public programs may have had more prestige os plausibility than the private interventions. Understanding the difference between these responses has important implications for public policy.

IV. National Interventions

The third level of intervention is the nation as a whole. Many health interventions are conducted on everyone at the same time. This is valuable because the scale of the intervention is large. But it is more difficult to evaluate a national intervention than a local one. Accordingly,

our understanding of how and why national interventions work is limited.

In this section, I review three national interventions: information about the harms of tobacco; the movement against drunk driving; and information about appropriate dietary habits. These interventions were chosen because there is some evidence they were at least partly effective. Choice of these examples does not imply that all national level interventions were successful; some are not. But the hope is to learn from examples that do work.

A. Anti-Tobacco Information

The single most successful health intervention of the past half century has been the movement to reduce smoking. Where smoking was high and rising in the early 1960s, it is lower and continuing to fall today.

Figure 3 shows the average number of cigarettes consumed per person over the 20th century.[7] Cigarette smoking rose markedly in the first half of the century, from virtually nothing to over 4,000 cigarettes per adult. To some extent, the increase in smoking is artificially inflated hand-rolled cigarettes are missing from the total. But the increase is still impressive. Indeed, public policy encouraged cigarette consumption, for example by distributing cigarettes to soldiers in the World Wars.

Some information about the harms of smoking was available by mid-century. Cutler and Kadiyala (2002) present results from surveys showing that about 60 percent of people recognized the harmful effects of cigarettes in the 1950s and 1960s. But people were not greatly attuned to

[7] Cigarette consumption data are tabulated by the Centers for Disease Control and Prevention.

the issue. Many people responded to survey questions by asserting that they did not smoke enough to cause harm to themselves.

That perception ended with the landmark report of the Surgeon General in 1964. The Surgeon General s report showed that smoking caused disease, particularly cancers and likely respiratory disease as well (later strengthened). Further, even moderate amounts of smoking were harmful.

The Surgeon General s report was national news. It was highlighted in the popular press and widely disseminated. The message was clearly heard. By 1970, 90 percent of people reported that they believed smoking was harmful to health. More people recognized the link between smoking and specific ailments such as heart disease and cancer. And people recognized that even moderate smoking was harmful to health.

This knowledge provision was accompanied by an immediate decline in smoking. Between 1963 and 1970, the share of the population smoking fell by 7 percent. Ironically, the Surgeon General s report was not very expensive for the government to produce or disseminate. But the scale of the national reporting made it very significant for people.

Over time, the Surgeon General s report was followed by many similar messages, including subsequent reports of the Surgeon General and other organizations such as the American Heart Association and the National Institutes of Health. Smoking continued to decline. By 2000, the number of cigarettes smoked was at roughly half its 1964 level.

Price increases played some role in this smoking decline, but not much. Cigarette taxes were increased in the 1960s, with the new health information. But taxes were fixed in nominal terms in the 1970s and through the first part of the 1980s. Since inflation was high, the real

value of the cigarette tax eroded. In recent years, cigarette taxes have again increased, but this largely makes up for the inflationary erosion of previous decades. Real cigarette taxes today are close to their level in the early 1960s (Gruber, 2001).

Other public policies have affected smoking over this time period, but these too cannot explain all of the trend. Bans on broadcast advertising of cigarettes had a negative effect on consumption, but it was relatively minor. More recent bans on smoking in restaurants, work sites, and public places cannot explain much of the historical trend.

It is clear that a lot of the response in lower cigarette consumption was individual decisions to quit smoking. What community-level interventions could not do bring about large changes in smoking rates the national interventions were able to accomplish.

What is unclear is what factors are most important in this. To some extent, smoking reduction is a result of individuals making health decisions in light of new information. This is certainly true about the immediate response to the Surgeon General s report. But social factors or peer pressure may also play a role. People may find it more difficult to justify smoking now than they did in the past, even if they would like to smoke. No studies have attempted to differentiate the impact of information from that of social pressure if indeed, they can even be separated.

In thinking about racial and ethnic disparities in health, it is important to look at the composition of smoking in addition to the level. Figures 4 and 5 provide information on this. The data are from periodic years of the National Health Interview Survey, as tabulated by the Surgeon General (2000).

Figure 4 shows racial trends in the share of people who report smoking. Blacks and

whites smoke at relatively similar rates, with black rates being slightly higher.[8] Importantly, the trends have tracked each other over time. The information intervention in smoking has had little effect on racial disparities in health, if anything leveling them.

That is not true about socioeconomic differences. Figure 5 shows that smoking rates declined by much more for better educated groups than for less educated groups. In 1966, smoking rates were 6 percentage points lower for people with a college degree compared to high school dropouts.[9] By 1995, smoking rates were 19 percentage points lower for college graduates than for high school dropouts. Put another way, smoking declined by 60 percent for college graduates, compared to only 20 percent for high school dropouts. The decline in smoking has raised the socioeconomic disparity in health.

B. Anti-Drunk Driving Movement

Actions to reduce drunk driving represent a particularly notable chapter in health interventions. The drunk driving example is so salient because, like with smoking, a national intervention accomplished major behavioral changes that a community-level intervention had failed to do.

In the years after World War II, it became increasingly clear that drinking and driving presented a public health challenge. Road mileage increased as rising incomes allowed more

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[8] These rates are unadjusted for income. Adjusting for income, blacks smoke less than do whites.

[9] Indeed, the 6 percent differential is probably larger than the difference a few years earlier; when incomes were lower, smoking rates were higher among higher income people than among lower income people.

people to own a car. People began living farther from work. In addition, alcohol consumption increased. The result was a perceived high rate of drunk driving deaths actual data on drunk driving fatalities in this period are sparse.

The prosecution and rehabilitation of drunk drivers is under state control. All states had laws about drunk driving, but police were not trained to stop or test suspected drunk drivers, and the court system was poor at prosecuting them. Rehabilitation efforts were limited. Many drunk drivers got off with a warning or light fine. Thus, through the 1960s, drunk driving became an increasing problem. A sense took hold that something needed to be done, and in particular that better enforcement and coordination mechanism could substantially reduce the incidence of drunk driving.

Responding to this, the Federal Transportation Department established the Alcohol Safety Action Project (ASAP) in the 1970s (Gusfield, 1996; Voas, 1981). The ASAPs operated in 35 communities. There were numerous specific ASAP interventions, but two themes. The first was to improve the operation of the legal system in dealing with drunk drivers. Arrest procedures were streamlined, improved breath-testing devices were adopted, and mobile vans were deployed to catch drunk drivers. In addition, courts were trained to screen for problem drinkers. The second theme was to encourage rehabilitation of problem drinkers. Identified problems drinkers were provided education and treatment programs to reduce continued drunk driving.

The ASAP programs were in place from two to five years, depending on the community. The program was expensive, costing $88 million between 1970 and 1977 (in 1970s dollars). There is some debate about the effectiveness of the ASAP program, but most analysts believe the programs were not very successful. Some studies find positive effects of the program, others

find inconclusive effects, and still others find negative effects. Since the methodology is similar to the community-level cardiovascular disease interventions discussed above, I do not present details here. It is sufficient to note that the program was certainly not an enormous success. As of the late 1970s, it was relatively easy for a researcher to conclude that drunk driving was a stubborn social problem, immune to public intervention.

Beginning in the early 1980s, though, drunk driving began a dramatic decline. The initial spur for the decline was the formation of Mothers Against Drunk Driving (MADD) and similar grassroots programs. MADD was organized in 1980 by Candy Lightner, a mother in California whose 13 year old daughter was killed by a drunk driver. The driver had been arrested a few days before for driving under the influence of alcohol (one of many such arrests for that driver), but had been released. MADD reached national prominence in 1982, when a TV special about the Lightner case was aired. By 1984, there were several hundred MADD chapters around the country.

MADD focused on the passage and enforcement of more severe DUI laws. Legally acceptable blood-alcohol levels were lowered, for example, and mandatory penalties for drunk driving were enacted. The legal age for alcohol purchase was increased.

There are no national data on the share of people driving with blood-alcohol levels above acceptable levels. Thus, it is impossible to know about trends in the share of people driving drunk. But data on crash fatality victims are available since 1982. The beginning of the data in 1982 is unfortunate; one would like to measure the trend in drunk driving prior to the MADD experience. But it was only with the increased prominence given to drunk driving by MADD that accurate statistics began to be kept. Thus, this is all the information that is available.

The data on the share of fatalities to drunk drivers, presented in figure 6,[10] show a marked decline in the share of fatalities to people who were drunk. The share was 30 percent in 1982 and declined to 25 percent by 1987. The decline in the share of drunk driving fatalities was immediate. There was no startup period during which the program was without effect.

Around 1987 and 1988, drunk driving fatalities seemed to have plateaued. The share was falling only slightly compared to previous years. Around that time, a second campaign was launched, the designated driver campaign (DeJong and Winston, 1998). The goal here was to have at least one non-drinker to drive. This program worked as well. Beginning in the late 1980s, the share of deaths to drunk drivers began another major descent. The share is now 17 percent.

Ironically, the experience of the past two decades, MADD in particlar, violates a central tenet of many public health campaigns. It is frequently stressed in sociology writings that policies should avoid blaming people for their mistakes. The idea is that people respond poorly to being blamed for health problems. Since the early 1980s, however, drunk drivers have been stigmatized in exactly that way. Yet even with this blame, there have been large health improvements.

The contrast between the ASAP programs and the MADD experience is also striking. Both actions focused largely on legal responses to drunk driving. Both targeted police and courts as natural enforcement agents. But one was successful, while the other was not. It is not entirely clear what accounts for the difference. Certainly, the MADD experience drew far more media

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[10] The data are from the United States Department of Transportation, National Highway Safety Administration (2001).

attention than the ASAP programs. The scale of the intervention may matter a great deal. The deterrent effect of the intervention may also be enhanced by the publicness of the intervention. Laws passed in response to drunk driving concerns were much more noticeable in this era than were the changes brought about by the ASAP program. Whether these or other aspects account for the difference is response is not known.

C. Dietary Change

The final intervention to study is perhaps the most complex changes in diet. Heart disease and many other conditions are affected by the overall amount of caloric intake and the type of calories consumed. Excessive overall caloric intake leads to obesity, diabetes, and hypertension, all leading risk factors for cardiovascular disease. Excessive fat intake, given the level of calories consumed, leads to high cholesterol and atherosclerosis. For some years, the message to American consumers has been two-fold: reduce the overall level of calories, and decrease the share of fat in the diet.

The response to these messages has been mixed. Changes in the fat composition of the diet have been exemplary. This response is best seen since the early 1980s. While it has been known for some time (since at least the 1950s) that high cholesterol leads to heart disease, clinical trials did not show the efficacy of cholesterol-intervention programs until 1984. The trial, termed the Lipid Research Clinics Coronary Primary Prevention Trial (LRC-CPPT) showed conclusively that cholesterol control significantly reduced mortality risk. The LRC-CPPT was major news. It was covered in newspapers and magazines often on the cover and received attention on the evening news.

The public clearly heeded the message. Figure 7 shows food issues that are of most concern to consumers.[11] Beginning in the early 1980s, concern about the fat and cholesterol content of food increased from about 10 percent of the population to nearly half. In the 1990s, public health officials stressed the importance of fat intake over cholesterol intake in explaining high cholesterol. Consumer concern mirrored this changing information.

Action followed concern. The consumption of red meat and eggs fell markedly over this period, as consumers shifted into lower fat foods such as chicken and salads (figure 8). Within these categories, lower-fat items were increasingly purchased at the expense of higher fat items. Coupled with these dietary changes were medical interventions such as increased cholesterol screening and use of anti-cholesterol medication.

Figure 9 shows average levels of cholesterol over time. Accurate cholesterol levels require blood samples from a large share of the population. Standard population surveys are not good enough. The only viable data are from the National Health and Nutrition Examination Surveys (NHANES). The data presented here are from the early 1970s (1971-74) and the late 1980s and early 1990s (1988-94). A more recent NHANES was conducted in the late 1990s, but these data have not yet been publicly released.

Overall, the share of people with high cholesterol fell from 28 percent to 19 percent, a change of about 30 percent. Importantly, the change was common across racial groups. Indeed, high cholesterol rates for blacks declined by more than did rates for whites, while starting from nearly the same base. The change was also relatively similar by education groups. People with less than a high school education and those with a college degree had the largest declines. There

[11] These data are from surveys conducted by the Food Marketing Institute.

was no substantial change in the SES gradient of high cholesterol.

At the same time as cholesterol was falling, though, the overall level of caloric intake has increased. Food available for consumption in the United States increased by 500 calories per person between 1970 and 1994. Obesity increased as well. Medically, obesity is often defined as having a body mass index (BMI, or weight in kilograms divided by height in meters squared) of 30 or greater. That is the definition I use.

Figure 10 shows the trend in obesity. The share of people who are obese rose by over 10 percentage points between the early 1970s and the late 1980s. Other data show that obesity continued to increase throughout the 1990s. Blacks are more obese than whites. Somewhat surprisingly, though, obesity increased by more for whites than blacks. Increases were relatively similar by socioeconomic status. The more educated are less obese than the less educated, but the increase in obesity was relatively similar across education groups. In this case, the worsening of health status did not increase the racial or socioeconomic disparities in health.

D. Summary

Many national health interventions have had a large impact on health behaviors. With the exception of obesity, most health behaviors have improved over time, and public health interventions are a key part of this improvement. In the case of smoking cessation, the health improvement was greater for better educated people. That is not the case with the reduction in high cholesterol or the increase in obesity, however. A lot of changes either narrowed, or left unaffected, the racial, ethnic and socioeconomic measures of health.

V. Implications

What makes for a successful behavioral intervention? Making sense of the various facts is not straightforward. There may not be one theory that explains them all. In this section, I propose some empirical regularities and suggest one possible interpretation.

There are some basics that seem obviously to be true. Clearly, the message conveyed to people has to be simple. The harm in each of the national interventions is clear drunk driving kills children; smoking causes lung cancer. The solution is also clear: don t drive while drunk; stop smoking. People deal with simple messages far better than complex messages.

Beyond that, the situation is murkier. I start with some theories that can be rejected. One theory common in social psychology is that information provision is not enough. People learn new information, the theory goes, but do not act on it readily. One has to change the environment as well. The evidence is not greatly supportive of this theory. While new information does not always lead to behavioral change, it often does. A good part of the decline in smoking, and certainly the initial fall, is a result of increased public knowledge about the damage from smoking. Changes in cholesterol result to a significant extent from the same factors. Information by itself can significantly change behavior.

A second rejected theory is that the form of the message is very important. In particular, negative messages that blame people for their health problems will be less successful than messages that work with people in a positive way. But this theory too is incomplete. The anti-drunk driving campaign brings this out most prominently. The subtext of this intervention was telling drunk drivers that they were evil people who killed innocent children. They deserved

punishment (or possibly reward if they had a non-drinking driver). People responded to this antagonistic message by limiting their drunk driving.

A third theory is that behavioral experiments need to be carried out for a long time to have any effect. Clinical trials may simply not be long enough. But many of the behavioral experiments that have been conducted lasted for five to seven years. That is a long period of time by the standards of many successful interventions. Within six years of the Surgeon General s report on the harms of smoking, for example, cigarette consumption fell by a significant amount. Drunk driving rates changed in that time frame as well, as did food purchasing habits.

While some theories are clearly false, there are two other theories that might explain these effects. The first is a theory of *permeation*. The reason why national information intervention have a greater impact than community interventions may be a result of the fact that national information permeates more widely and deeply in peoples minds. I start from the premise that change is hard; people always prefer to continue on their current path. The key for interventions to succeed is that they force people to take some action. People can continue to do what they were doing, but if the information permeates widely enough, doing nothing becomes a choice that individuals have to rationalize. Once it becomes impossible to continue in the current path without making an explicit decision, people may be more likely to change to new paths.

In this sense, information interventions may be similar to taxes or regulatory interventions. When taxes on cigarettes are raised, people cannot smoke to the extent they formerly could without formally giving up some other consumption. When smoking is banned in buildings, people have to walk outside to smoke. Similarly, when the information about smoking

becomes so clear as to obliterate any doubt about its harms, people cannot continue to smoke without consciously deciding to sacrifice their health.

The national cholesterol intervention seems to fit the same pattern. It was impossible to miss the news about the harms from cholesterol. People had to act on it cut out foods high in fat or cholesterol, visit the doctor, etc. or consciously recognize that they were not going to do so. As a result, more people changed their behavior.

The focus on the degree to which information permeates is not to deny that the message being conveyed is important. One of the features of all of the successful health information interventions is that their prescriptions are simple: one should not smoke; high cholesterol should be managed; drunk driving is bad. The simplicity of the message is clearly a key to its success.[12] But the simplicity of the message is not enough. It has to impact so deeply that people cannot ignore it.

A second theory has to do with externalities. One of the hallmark of many interventions is that they stress the harm that people do to others, not just to themselves. Drunk driving was stigmatized because innocent people (frequently children) were killed by it. Cigarette smoking came in for additional scorn when studies linked second-hand smoke to poor health (a subject which is still controversial). People may respond more to the idea that they are hurting others than to the harm they cause themselves. External effects also allow people not engaging in the activity a safer route on which to base negative stigma on those who do.

A third theory is of peer effects. People may decide what is appropriate behavior on the

[12] Indeed, it is possible that the lack of a simple prescriptive message is the key to why we have not been able to reduce obesity to date.

basis of what others are doing, in addition to their own utility from an activity. If more people engage in health promoting practices, people who would otherwise not engage in those practices might decide to as well. This is often referred to as a tipping point phenomenon, since it could be that small changes in the behavior of the average person could induce large changes in behavior even amongst those far away from the average. The tipping point model is similar to the theory of population epidemiology proposed by Geoffrey Rose (1992). It could help explain why national interventions seem to be more effective than community-level or individual interventions, since they result in more changes among the average person.

These theories may or may not be right. Understanding why some health interventions success and others fail, though, is essential to doing something about the issue of racial and ethnic disparities in health, as well as many other health problems.

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Table 1: Effects of the MRFIT on Risk Factors and Mortality

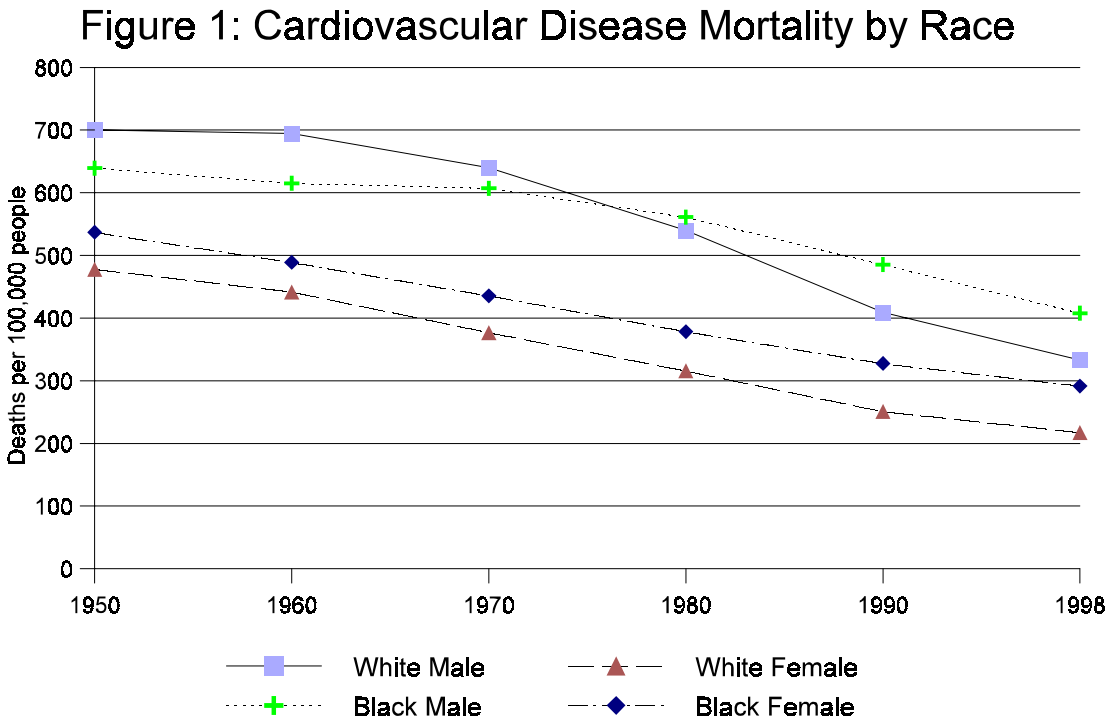
Measure	Experimental Results			Percent of Hypothesized Effect
	Intervention Group	Control Group	Difference-in-Difference	
Diastolic blood pressure	-12%	-8%	-4%	75%
Smoking quite rate	-46	-29	-17	145
Serum cholesterol	-5	-3	-2	50
CHD mortality*	17.9	19.3	-7	26
Overall mortality*	41.2	40.4	2	
10 year CHD mortality*	31.4	35.1	-11	
10 year overall mortality*	77.2	83.4	-8	
16 year CHD mortality*	57.6	64.7	-11	
16 year overall mortality*	154.2	163.1	-6	

Note: Difference-in-difference is the percent change for the intervention group less the percent change for the control group. In the mortality rate row, the difference-in-difference is the percent reduction in mortality rate. Differential changes in blood pressure, cigarette smoking, and cholesterol were statistically significant; mortality rate was not.
* Deaths are per 1,000 people.
Source: Data are from Multiple Risk Factor Intervention Trial Research Group (1982, 1990, 1996).

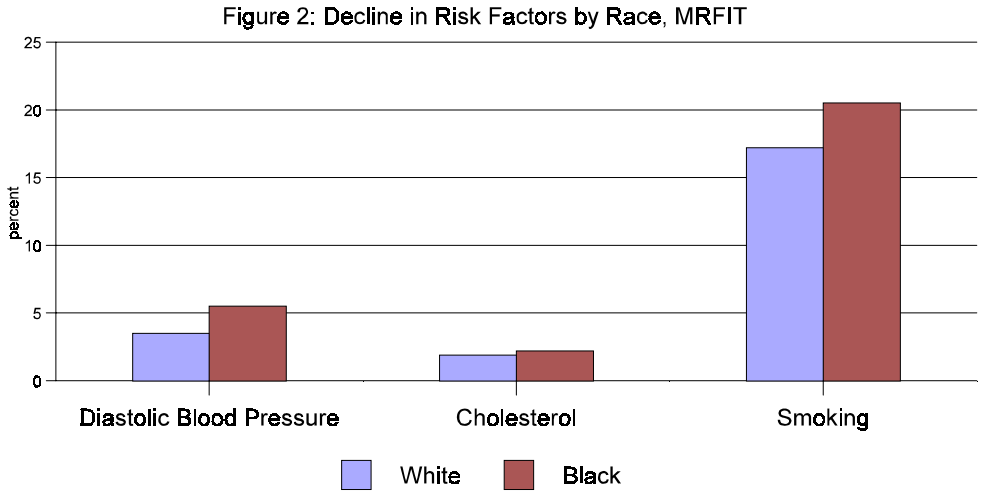
Table 2: Results of Community Level Cardiovascular Disease Intervention Trials

Trial	Years	Intervention	Results
Stanford Five-City Project	1980-87 (6 years of education)	o 2 treatment and 2 control cities o Interventions through media and direct education o General education and 4 to 5 risk factor education campaigns per year o Focus on dietary change, physical activity, and medication usage	o Increase in knowledge of CHD risk factors in treatment cities o Positive changes in risk factors for treatment and control cities o Modest differential reduction in blood pressure and obesity in treatment cities in some years o No differential effect on cholesterol, smoking in treatment cities o No differential effect on mortality in treatment cities
Minnesota Heart Health Program	1980-90 (5 years of education)	o 3 treatment and control cities o Advocated hypertension prevention and control, healthy eating, non-smoking, and regular exercise o Used community leaders, mass media, and health professionals o Population screening for risk factors	o Significant exposure to program by 3rd year, declining after 5 years o Positive changes in risk factors and health outcomes for treatment and control cities o No differential change in cholesterol, smoking, blood pressure, or BMI o Modest increase in physical activity in treatment cities o No differential effect on cardiovascular disease morbidity or mortality
Pawtucket Heart Health Program	1981-93 (7 years of intervention)	o 1 treatment and 1 control city o Advocated control of cholesterol and blood pressure, reduced smoking and obesity, and increased physical activity o Used community organizations, individual intervention, and community change (eg menu labeling) o No mass media	o Positive changes in risk factors and health outcomes for treatment and control cities o No differential change in risk factors across cities with the exception of BMI

Sources: Stanford Five City Project Farquhar, Fortmann, Flora, et al. (1990); Minnesota Heart Health Program Luepker, Murray, Jacobs, et al. (1994); Luepker, Rastam, Hannan, et al. (1996); Pawtucket Heart Health Program Carleton, Lasater, Assaf et al. (1995).

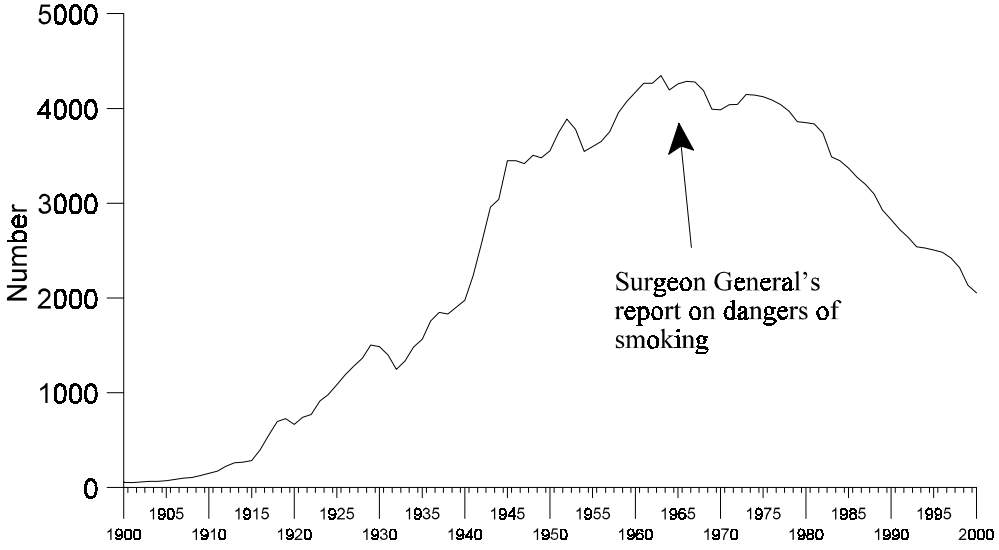


Source: U.S. Department of Health and Human Services (2001).



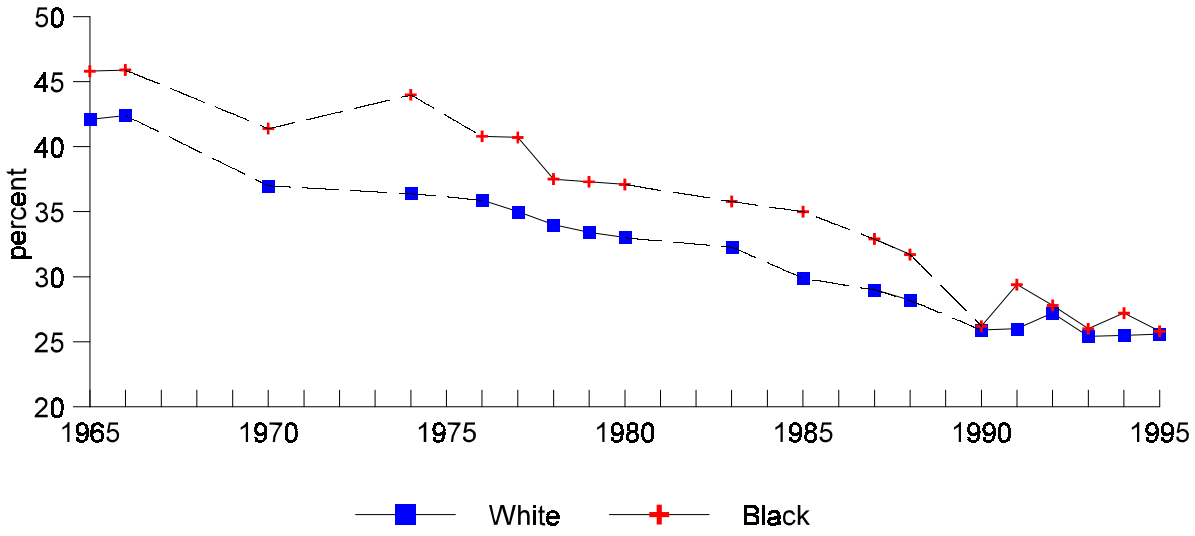
Source: Multiple Risk Factor Intervention Trial Research Group (1982).

Figure 3: Average Number of Cigarettes Smoked per Adult



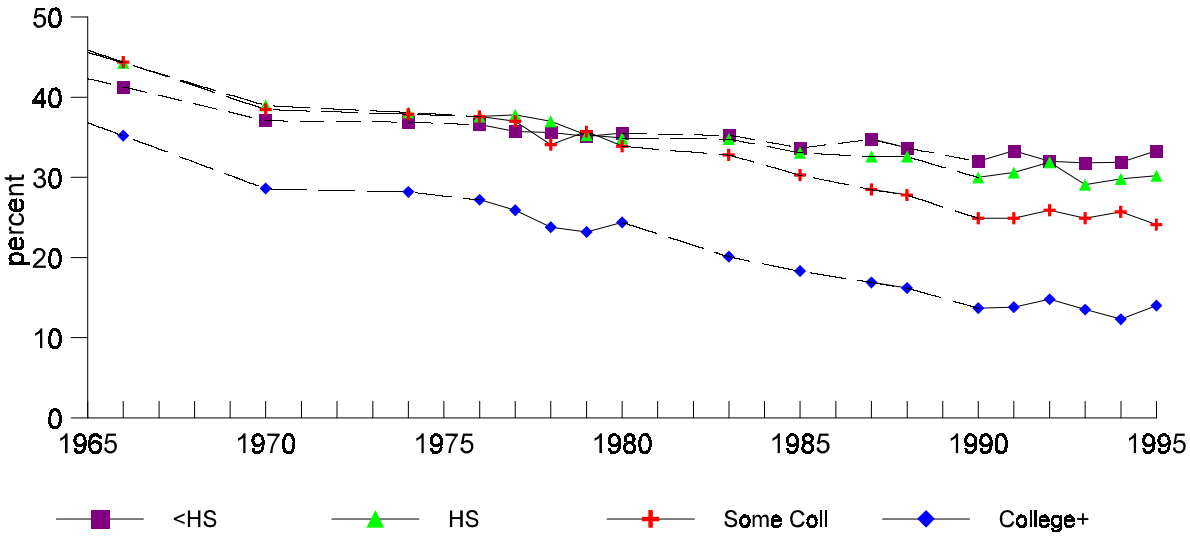
Source: U. S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Figure 4: Smoking Rates by Race



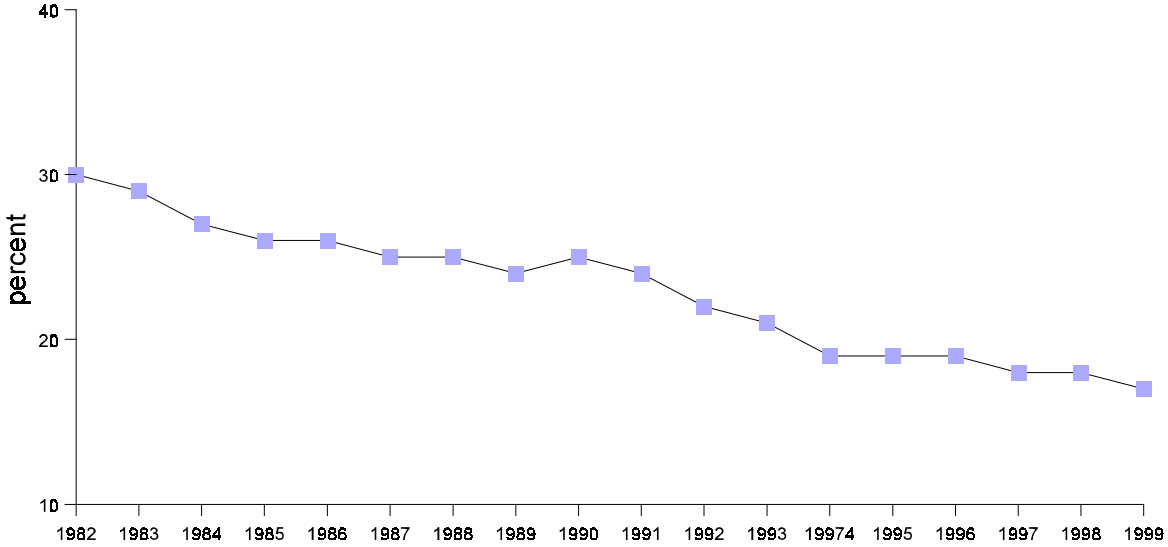
Source: U. S. Department of Health and Human Services (1998).

Figure 5: Smoking Rates by Education



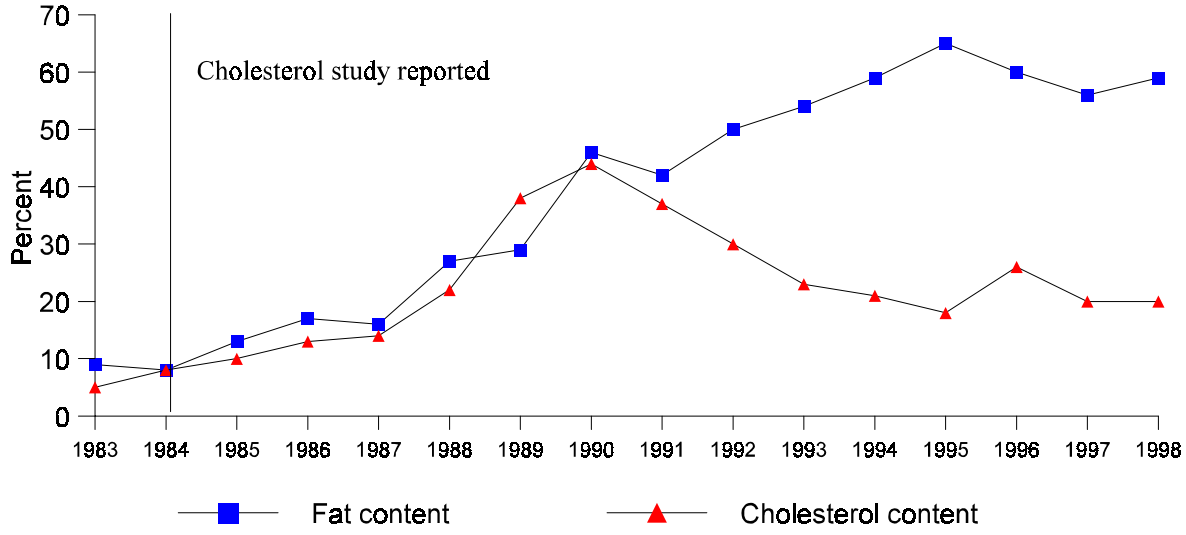
Source: U. S. Department of Health and Human Services (1998).

Figure 6: Share of Drivers in Fatal Crashes with BAC>=.10



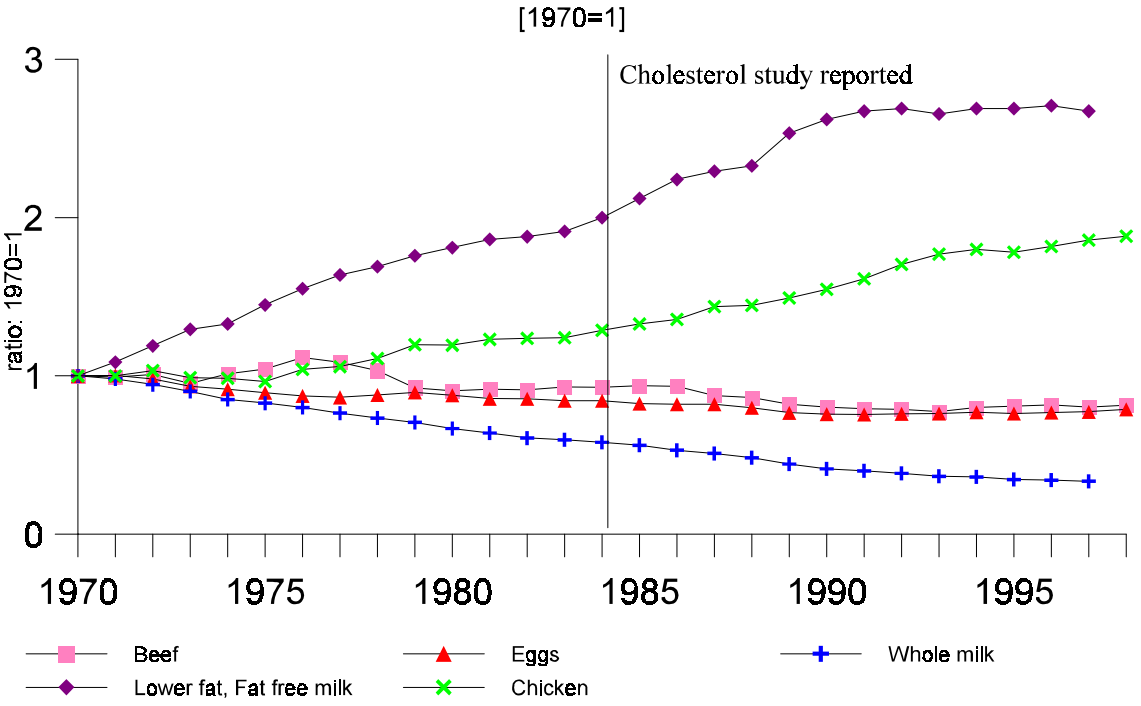
Source: U. S. Department of Transportation (2001).

Figure 7: Nutritional Issues that Most Concern Consumers

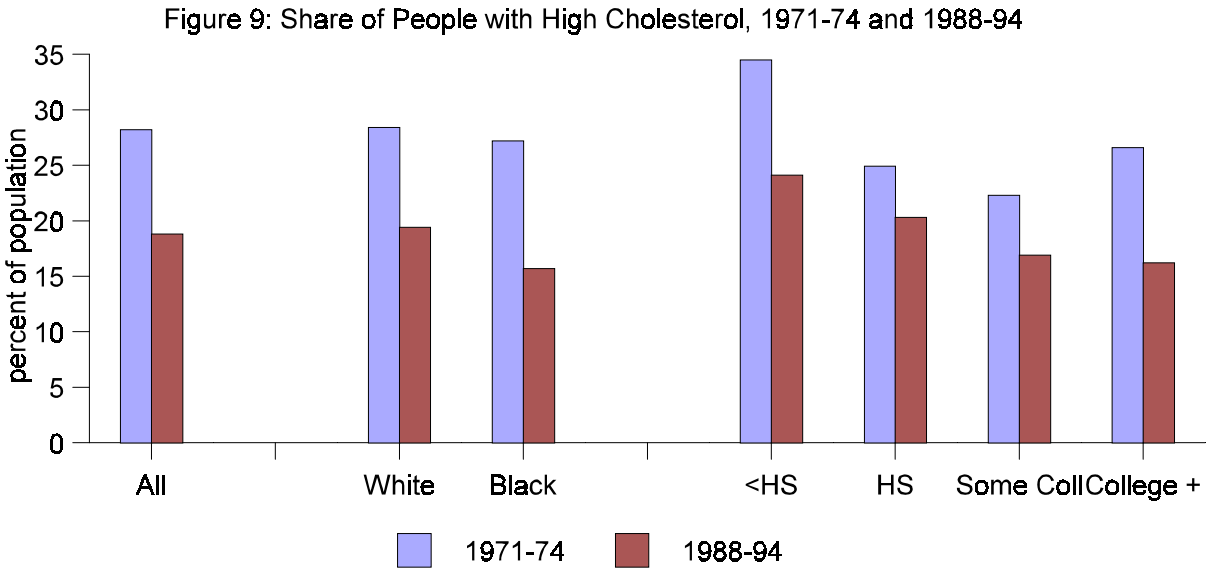


Source: Food Marketing Institute.

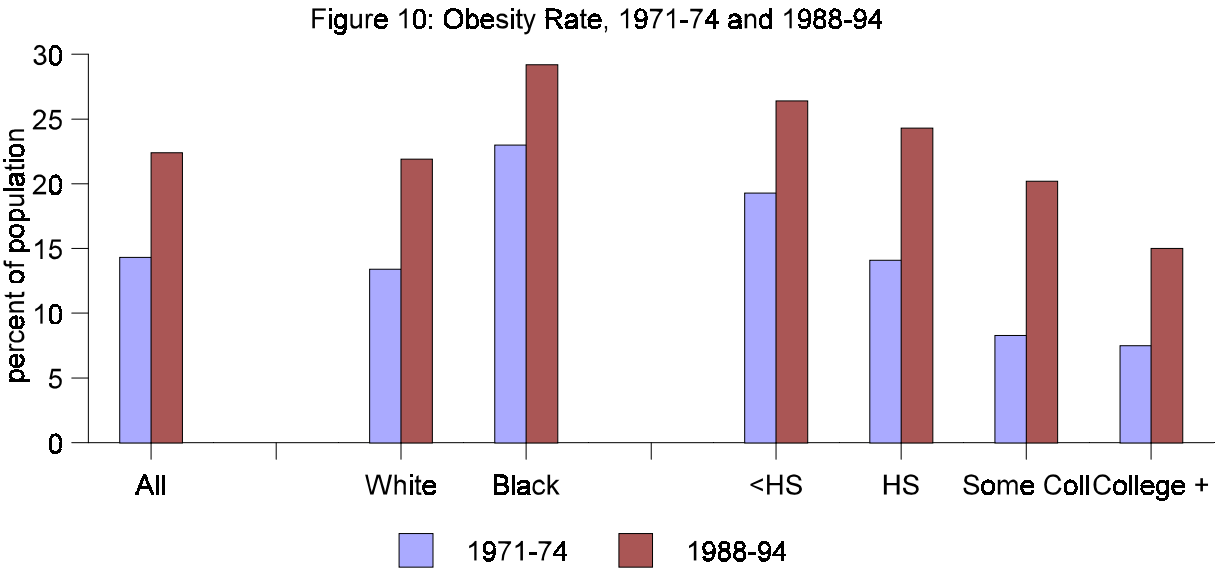
Figure 8: Trends in Food Consumption



Source: U. S. Department of Agriculture (1999).



Source: Author's calculations from National Health and Nutrition Examination Surveys.



Source: Author's calculations from National Health and Nutrition Examination Surveys.