Effects of a Community-wide Health Education Program on Cardiovascular Disease Morbidity and Mortality

The Stanford Five-City Project

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The authors examined changes in morbidity and mortality from 1979 through 1992 during the Stanford Five-City Project, a comprehensive community health education study conducted in northern California. The intervention (1980–1986), a multiple risk factor strategy delivered through multiple educational methods, targeted all residents in two treatment communities. Potentially fatal and nonfatal myocardial infarction and stroke events were identified from death certificates and hospital records. Clinical information was abstracted from hospital charts and coroner records; for fatal events, it was collected from attending physicians and next of kin. Standard diagnostic criteria were used to classify all events, without knowledge of the city of origin. All first definite events were analyzed; denominators were estimated from 1980 and 1990 US Census figures. Mixed model regression analyses were used in statistical comparisons. Over the full 14 years of the study, the combined-event rate declined about 3% per year in all five cities. However, during the first 7-year period (1979–1985), no significant trends were found in any of the cities; during the late period (1986–1992), significant downward trends were found in all except one city. The change in trends between periods was slightly but not significantly greater in the treatment cities. It is most likely that some influence affecting all cities, not the intervention, accounted for the observed change. Am J Epidemiol 2000;152:316–23.

cardiovascular diseases; death certificates; epidemiologic methods; health education; medical records; mortality; myocardial infarction; program evaluation

Epidemic cardiovascular disease (CVD), the major cause of death and disability in industrialized countries, is largely attributable to lifestyle behaviors, including diet, exercise, and cigarette smoking, both directly and as mediated by blood pressure and plasma lipoprotein levels. The importance of CVD to public health and its link with lifestyle behaviors led pioneers in CVD control to address the problem on a community level. The two earliest community-based CVD studies were the Stanford Three-Community Study in California (1) and the North Karelia Project in Finland (2–4), both initiated in 1972. The Three-Community Study targeted CVD risk factors through a health promotion program that used mass media supplemented by individual and group education for high-risk persons in one town and mass media alone in a second town; a third town served as a control. Findings showed a significant reduction in overall cardiovascular risk in the two treatment towns, compared with the control town, in cohorts that were surveyed annually in each town at baseline and for three follow-up visits.

The Three-Community Study generated the Stanford Five-City Project, a larger-scale and longer California field trial designed to test whether a comprehensive program of community organization and health education could produce favorable changes in CVD risk factor prevalence, morbidity, and mortality (5). A 6-year education intervention (1980–1986) targeted all residents in two treatment communities and involved a multiple risk factor strategy delivered through multiple educational methods (6). The overall changes in risk factors (knowledge about CVD, blood pressure, smoking, cholesterol) have been reported elsewhere and were positive in both treatment and control towns, with the exception of obesity (7). The changes in knowledge about CVD and in systolic and diastolic blood pressure in the treatment cities significantly exceeded those in control cities in the cross-sectional surveys (two-tailed \( p < 0.05 \)); in repeated-measures cohorts, smoking and cholesterol changes also favored the treatment cities. Other papers provide more detailed analyses of these risk factor changes (8–12) and their maintenance (13).

As the North Karelia Project did, the Five-City Project included monitoring of trends in CVD rates and risk factors. The hypothesis was that the Five-City Project education program would significantly reduce myocardial infarction and stroke rates in the two treatment communities relative to the
three control cities. CVD endpoint results have been reported for the North Karelia Project (14) and another major US community CVD trial, the Minnesota Heart Health Project (15); the purpose of this paper is to report these results for the Five-City Project.

MATERIALS AND METHODS

The design and methods of the Five-City Project (5) and the morbidity and mortality surveillance system (16) have been described previously and are summarized here. The two treatment cities (1980 total population) were Monterey (43,400) and Salinas (80,500), and the three control cities were Modesto (132,400), San Luis Obispo (34,300), and Santa Maria (39,700); for simplicity, these cities are numbered 1 to 5, respectively, in the tables of this paper. No population risk factor surveys were conducted in Santa Maria. Treatment and control cities were selected from all northern California cities by using criteria for size, distance from Stanford, and media markets. Random assignment of communities to treatment and control conditions was precluded by constraints on city selection, particularly the requirement that the broadcast media in treatment cities not reach control cities. The originally planned duration of morbidity and mortality monitoring (1979–1986) was extended to 1992 when it became apparent that the impact of the gradual risk factor changes induced by the intervention would not occur, if at all, until after the intervention ended (1986).

When this study was conducted, Salinas, Modesto, and Santa Maria were agricultural service communities with few large, nonagricultural employers (with the exception of a large winery in Modesto and of Vandenberg Air Force Base near Santa Maria). Monterey and San Luis Obispo were smaller cities, and tourism was a larger component of the local economy. About 20 percent of the Salinas and Santa Maria populations was Mexican American in 1980, and this proportion increased (absolutely and relatively) during the decade. Most other residents were White and non-Hispanic.

Morbidity and mortality surveillance system

Although mortality rates can be obtained directly from routine vital statistics, their precision and accuracy may not be sufficient to test the impact of community intervention, particularly over a short time and in moderate-sized cities. Also, routine vital statistics might be biased by the intervention program, for example, by altering physician reporting on death certificates. Restricting evaluation to fatal events would provide insufficient power, but there was no routine reporting of nonfatal CVD events. The Five-City Project therefore included a community surveillance system designed to validate fatal CVD events and count nonfatal events; this system was modeled on the World Health Organization myocardial infarction registries (17) and work from the Framingham Study (18). Events occurring in residents of the five cities who were aged 30–74 years at the time of the event were included.

Selection of events and criteria. Only nonfatal events for which residents were hospitalized (myocardial infarction and acute stroke) were deemed practical for inclusion; total mortality, fatal myocardial infarction and stroke, and other fatal coronary heart disease (mainly out-of-hospital sudden death) were included as fatal events. We recognized that a substantial but unknown proportion of nonfatal myocardial infarctions would be clinically unrecognized and therefore uncounted, but we assumed that this proportion would be similar for all cities. Likewise, nonfatal events occurring away from home would be missed but were also likely to be similar between cities. Fatal events that occur in travelers are reported to the county of residence and therefore were included. The criteria were designed to maximize reliability by minimizing the need for clinical judgment, manual calculations, or the use of implicit assumptions and to rely on data usually found in a hospital chart (19).

Criteria were established for both definite events (specific criteria) and possible events (sensitive criteria) to enable detection of diagnostic drift and other threats to the validity of the process (20); only definite events are reported in this paper since our goal was to examine the main Five-City Project hypothesis. In brief, definite myocardial infarction was diagnosed in the presence of evolving electrocardiographic changes (new Q waves or evolving ischemic ST-T wave changes) or, in their absence, a combination of either less-definite electrocardiographic changes or documented, prolonged chest pain (>20 minutes) plus abnormal cardiac enzymes. Definite stroke was diagnosed when a physician documented the rapid onset (<48 hours) of a new, distinct neurologic deficit or in the presence of a new brain lesion on computerized tomography interpreted by the radiologist as a definite stroke. Autopsy data were obtained when available and when documentation of new cerebral or myocardial infarction was sufficient to include the event as definite. Sudden (<1 hour) and rapid (1–24 hours) deaths occurring out of hospital were included as definite coronary deaths when information from the next of kin and the regular physician indicated either a history of coronary disease or was otherwise consistent with coronary disease as the only apparent cause of death.

Case identification and investigation. Fatal events were located by regularly reviewing county vital statistics records; deaths were not investigated further if the death certificate diagnoses clearly indicated a noncardiovascular cause. All other deaths were investigated by collecting additional medical information. Information on events for which residents were hospitalized came from the medical record; for out-of-hospital deaths, the staff interviewed the next of kin and obtained a completed one-page questionnaire from the decedent’s regular physician. Coroner records and autopsy results were copied for all deaths. Nonfatal events were identified through a regular review of computerized hospital discharge data, which were searched for relevant diagnoses (International Classification of Diseases, Ninth Revisions, Clinical Modification (ICD-9-CM) codes 410–414, 431–438, and 786.5) in age-eligible patients. For all events for which residents were hospitalized, an abstract of the history, physical examination, laboratory data, consultations, and other pertinent information was prepared by using standard forms; electrocardiograms and radiology reports were photocopied.
**Validation.** Information on each event was returned to Stanford, where individual and city identifiers were removed and all data were entered into a database. Electrocardiograms were sent to the Division of Epidemiology at the University of Minnesota (Minneapolis), where they were interpreted according to the Minnesota electrocardiogram code (21). These masked clinical data, including the electrocardiogram codes, were then reviewed independently by two research nurses, who determined the presence or absence of each component of the criteria by using their judgment when needed (e.g., whether or not the physical findings were sufficient to document a stroke). When the two analyses produced different endpoints, the case was reviewed and adjudicated by a panel of physicians.

This validation process was streamlined after about 4 years of data collection. In collaboration with two other US community CVD prevention projects, we examined the predictive value of certain elements of the process to validate myocardial infarction (22, 23). We showed that events that received an ICD-9-CM discharge code of 410 and for which residents had abnormal enzymes were essentially always coded as definite events after full validation. Similarly, events that did not receive an ICD-9-CM code of 410–414 and for which residents had normal enzymes were never correctly coded as definite events. We therefore instituted a screening step whereby events in these two categories were validated automatically and were not examined further, saving considerable effort. To minimize any potential for bias, this system was applied retroactively to events that had already been validated.

**Quality control.** Research staff performance was monitored through biennial review of a standard set of 20 charts circulated to all investigators, plus re-review of a randomly selected 5 percent of charts reviewed previously. In addition, validators were monitored annually through blind resubmission of events validated previously.

**Population estimates.** The geographic areas included in the study were defined by census tracts rather than city boundaries. Census data were obtained for 1980 and 1990, and the intercensal midyear population by 5-year age groups was estimated by linear interpolation. The 1991 and 1992 population data were estimated by linear extrapolation from the previous 10 years.

**Education program**

The intervention conducted in the treatment cities was a 6-year multifactor risk reduction education program that was coordinated, comprehensive, and community-wide (6, 24–32). Risk factor interventions had multiple target audiences and used multiple communication channels and settings (including newspapers, television and radio, mass-distributed print media, classes, contests, and correspondence courses). The program promoted reducing plasma cholesterol levels through a change in diet; reducing blood pressure through regular blood pressure checks, reduced salt intake, reduced weight, increased exercise, and full adherence to antihypertensive medication regimens; reducing obesity through increased exercise and reduced dietary energy intake; and increasing both moderate and vigorous physical activity. Program planning included setting general and specific goals for each year of the campaign (e.g., reducing meat intake, promoting the adoption of one new low-fat recipe per month, reaching 50 percent of smokers with a self-help quit kit). Data from baseline population surveys in the treatment communities were used to develop an overview of knowledge, attitudes, and behavior for the target audience. This audience was segmented by age, ethnicity, socioeconomic status, overall cardiovascular risk, media use, organization membership, and motivation to change behavior (including diet) (33). Formative evaluation with these audience subgroups was used to refine educational strategies, programs, and materials (29). Social learning theory (34) guided the development of educational materials.

On average, each adult in the two treatment cities was exposed to about 5 hours per year of Five-City Project educational messages (7). About 7 percent of this exposure was provided by classes, lectures, or workshops; 34 percent by television and radio; 41 percent by booklets and kits; and 18 percent by newspapers and newsletters (7). Additional details on the educational program are available in other publications (8–10, 12, 35).

**Statistical methods**

For the analyses presented in this paper, we included only those events classified as definite. However, persons might have experienced more than one definite event (e.g., a nonfatal event one year and a fatal event in another). Therefore, to increase the independence of the observations made in this paper, we included in the analyses only the first definite event recorded for each person. This first event in the database could have been a first or a recurrent event for that person, however.

All age and sex adjustment was made by using the age and sex structure of the pooled 5-city population in 1990 and the direct method of adjustment. All rates were expressed per 100,000 persons.

The unit of analysis throughout was the city- and year-specific adjusted rate for the sexes pooled. There were 70 rates: five cities by 14 years. To assess the trends in rates over time or to assess the trends in rates for the treatment or control communities separately, we used the mixed models procedure (PROC MIXED) in the Statistical Analysis System (version 6.12; SAS Institute, Inc., Cary, North Carolina). We entered into the model 28 rates (two cities by 14 years) for the two treatment cities and 42 rates (three cities by 14 years) for the control cities. Cities were considered a random effect. After examining the distribution of rates and their correlation structure, we specified a compound symmetric covariance structure by city, thus taking into account the year-to-year correlation differences between cities. To test the null hypothesis that there was no difference between treatment and control, the mixed procedure was again used, with city nested within treatment and a compound symmetric covariance structure by city. The model for fixed effects included treatment and year (continuous) and the treatment-by-year cross product.

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Trends were contrasted between the first 7 years (“early”; during the intervention) and the second 7 years (“late”; after the intervention) of the study by including a period variable in the model. The mixed procedure was used once again with the fixed effects: treatment, year, period, and their cross products.

RESULTS

Continuous quality control monitoring showed high levels of comparability (>90 percent agreement within and between abstractors) for all data collection elements at every measurement. From 1979 through 1992, we recorded 35,468 events in 24,200 persons. About half of these events (17,516) were validated fully, while the remaining were validated on the basis of ICD-9-CM discharge code and cardiac enzyme results alone. Of this total, 9,479 first definite events occurred in different persons (i.e., the first event for each person was counted). About twice as many events occurred in men as in women (6,239 vs. 3,240). In subsequent analyses, we combined men and women, since the Five-City Project hypotheses were not sex specific (and there was no reason to expect the intervention to affect event rates in men and women differently). Coronary disease deaths, nonfatal myocardial infarction, and stroke each accounted for about one-third of the events.

Table 1 displays the baseline (1979) event rates by city and by event type; sudden death was combined with other fatal coronary disease events. The population size shown is the 1980 US Census number of persons aged 30–74 years in each city, and this number was used in the denominator for these rates. The rates were quite variable, but there were no statistically significant differences among the cities. The combined event rate (fatal and nonfatal myocardial infarction and stroke plus fatal coronary heart disease) was chosen a priori as the main outcome variable for these analyses (5).

Trends in definite event rates could be affected by changes in documentation of clinical events over time. For example, if physicians gradually order more tests and record more elements of the history of each event, events previously classified as possible could be reclassified as definite. Therefore, we next examined changes in documentation of important variables over time. The proportion of all coronary events that included a record of chest pain at baseline was high (94–99 percent) and did not change appreciably over time in any of the cities. The proportion of these events that included at least two electrocardiograms and available information on cardiac enzymes was lower at baseline (64–86 percent and 76–86 percent, respectively). Both rates increased over time in all cities, but only the increases in documentation of enzymes were significant. For none of these three elements of the diagnostic criteria was the change significantly different between treatment and control cities. Thus, the definite coronary event trends may be biased slightly upward over time, but the comparison between treatment and control cities is valid. Documentation of the neurologic examination in stroke patients was also fairly high at baseline (85–95 percent) and rose only slightly over time. However, there was a marked and significant increase in the number of patients examined by computerized tomography of the brain (beginning below 60 percent and ending above 90 percent in all cities). Again, there were no significant among-city differences in the increases in either of these two variables. Thus, in general, these trends should bias the stroke rates upward over time but equivalently across cities, so that treatment-control comparisons are valid.

The change in autopsy rates was more complicated. For the first 5 years of the study, the autopsy rates in the two treatment cities, which were served by a single medical examiner, were substantially higher than those in the three comparison cities (60 percent vs. 25–50 percent). Autopsy rates fell significantly in all five cities, averaging 1.5–3.5 percentage points per year, but the decline was greater in the treatment cities ($p = 0.002$ in the mixed model analysis) and occurred fairly suddenly in about 1984. After that year, autopsy rates were fairly similar and tended to fall slowly over time in all five cities. The effect of a decline in the autopsy rate on definite event rates is difficult to predict because autopsy data are not required for all definite events; however, such a decline could shift some events from the definite to the possible category, artificially lowering the definite-event mortality rate. Such a shift could have produced a greater decline in the definite-event mortality rate in the treatment cities studied. We will return to this issue after comparing the trends in event rates.

**TABLE 1.** Baseline event rates (standard errors) of cardiovascular disease* in 1979, Stanford Five-City Project, northern California

<table>
<thead>
<tr>
<th>City†</th>
<th>Population‡</th>
<th>Fatal CHD§</th>
<th>Fatal MI§</th>
<th>Nonfatal MI</th>
<th>Fatal stroke</th>
<th>Nonfatal stroke</th>
<th>Combined</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (Treatment)</td>
<td>19,386</td>
<td>85.0 (19.8)</td>
<td>55.8 (16.4)</td>
<td>148.9 (26.4)</td>
<td>33.7 (12.9)</td>
<td>21.9 (10.0)</td>
<td>345.2 (40.1)</td>
</tr>
<tr>
<td>2 (Treatment)</td>
<td>32,501</td>
<td>104.9 (17.2)</td>
<td>44.6 (10.9)</td>
<td>158.8 (21.4)</td>
<td>25.0 (6.5)</td>
<td>74.8 (15.3)</td>
<td>408.1 (34.1)</td>
</tr>
<tr>
<td>3 (Control)</td>
<td>63,414</td>
<td>140.0 (14.5)</td>
<td>39.3 (7.6)</td>
<td>194.5 (17.3)</td>
<td>33.5 (7.1)</td>
<td>69.3 (10.4)</td>
<td>476.6 (25.8)</td>
</tr>
<tr>
<td>4 (Control)</td>
<td>12,037</td>
<td>120.0 (28.3)</td>
<td>18.6 (10.8)</td>
<td>62.7 (19.9)</td>
<td>0</td>
<td>12.9 (9.2)</td>
<td>214.2 (37.3)</td>
</tr>
<tr>
<td>5 (Control)</td>
<td>27,157</td>
<td>110.2 (20.2)</td>
<td>42.0 (13.0)</td>
<td>190.4 (27.4)</td>
<td>21.4 (9.8)</td>
<td>43.1 (12.7)</td>
<td>407.2 (39.6)</td>
</tr>
<tr>
<td>Treatment</td>
<td>51,887</td>
<td>96.7 (12.9)</td>
<td>48.7 (9.1)</td>
<td>154.4 (16.5)</td>
<td>28.2 (7.1)</td>
<td>54.7 (10.2)</td>
<td>382.7 (25.9)</td>
</tr>
<tr>
<td>Control</td>
<td>102,608</td>
<td>130.4 (11.0)</td>
<td>37.6 (6.0)</td>
<td>176.4 (13.0)</td>
<td>25.6 (4.9)</td>
<td>55.5 (7.3)</td>
<td>425.5 (20.0)</td>
</tr>
</tbody>
</table>

* Per 100,000 persons.
† 1, Monterey; 2, Salinas; 3, Modesto; 4, San Luis Obispo; 5, Santa Maria.
‡ Total population aged 30–74 years according to the 1980 US Census.
§ CHD, coronary heart disease; MI, myocardial infarction.
For the remainder of the analyses, we combined all first definite events to calculate a single age- and sex-adjusted event rate for each year in each city, following the original study hypothesis. First, we examined trends over the entire 14 years of the study (table 2 and figure 1). There were strong and significant declines in all five cities, and no difference in rate trends existed over time between treatment and control when the entire 14-year period was considered.

By the middle of the 1980s, it was evident to investigators that any risk reductions in the treatment cities were occurring slowly and that it would be unrealistic to expect an impact on morbidity and mortality by the end of the intervention, in 1986. Surveillance was therefore extended, and we hypothesized that trends in the late period, following the intervention (1986–1992), compared with those in the early period, during the intervention (1979–1985), would be more favorable in the treatment than the control cities. This hypothesis is examined in table 3 and figure 2.

Table 3 looks at the city-specific, treatment, and control trends for the two periods. In the early period, none of the cities showed significant trends; in the late period, all cities except Salinas experienced significant downward trends. The treatment and control results reflect these trends—not significant in the early period and significantly downward in the late period. When period was taken into account in the mixed model regression, the three-way interaction of year by treatment by period, although not significant (\( p = 0.09 \)), suggested some difference between treatment and control regarding change over time. Figure 2 shows that in the treatment communities throughout the early, intervention period, the combined-event rate did not decrease and in fact rose slightly, although not significantly. On the other hand, the rate in the control communities decreased during intervention, and this trend accelerated after intervention ended. Thus, the change in slope direction between periods was more dramatic for the treatment than for the control cities, although the difference was not statistically significant.

Could even this suggestion of a greater change in the treatment cities be due to the greater decrease in autopsy rates in those cities? To examine this question, we analyzed only nonfatal definite events and found trends that were more nearly flat for all five cities, no period effect, and no evidence of a treatment-control difference. Another way to examine this question was to include possible fatal events, since it is reasonable to suppose that the absence of an autopsy would disallow a definite code but not remove the death entirely from

<table>
<thead>
<tr>
<th>City†</th>
<th>No. of events</th>
<th>Beta‡ (SE§)</th>
<th>95% CI§</th>
<th>% Change per year¶</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (Treatment)</td>
<td>965</td>
<td>-10.74 (2.53)</td>
<td>-16.24, -5.24</td>
<td>-3.95</td>
</tr>
<tr>
<td>2 (Treatment)</td>
<td>1,939</td>
<td>-12.76 (3.17)</td>
<td>-19.67, -5.85</td>
<td>-3.48</td>
</tr>
<tr>
<td>3 (Control)</td>
<td>4,185</td>
<td>-13.72 (1.44)</td>
<td>-16.86, -10.59</td>
<td>-3.46</td>
</tr>
<tr>
<td>4 (Control)</td>
<td>618</td>
<td>-12.13 (3.76)</td>
<td>-20.32, -3.93</td>
<td>-3.09</td>
</tr>
<tr>
<td>5 (Control)</td>
<td>1,772</td>
<td>-12.50 (1.78)</td>
<td>-16.39, -8.62</td>
<td>-3.81</td>
</tr>
<tr>
<td>Treatment</td>
<td>2,904</td>
<td>-11.75 (2.00)</td>
<td>-15.86, -7.64</td>
<td>-3.48</td>
</tr>
<tr>
<td>Control</td>
<td>6,575</td>
<td>-12.78 (1.43)</td>
<td>-15.67, -9.88</td>
<td>-3.09</td>
</tr>
</tbody>
</table>

* Fatal and nonfatal definite coronary disease and stroke.
† 1, Monterey; 2, Salinas; 3, Modesto; 4, San Luis Obispo; 5, Santa Maria.
‡ Rate of change in annual age- and sex-adjusted event rate; for each city, a separate regression model was fitted to calculate the beta; for the combined treatment and control cities, the mixed-effects model was used (refer to Materials and Methods in the text).
§ SE, standard error; CI, confidence interval; both calculated from the simple regression models for each city and from the mixed-effect regression model for the combined treatment and control cities.
¶ Calculated from log(rate).
FIGURE 2. Combined-event rate of cardiovascular disease in treatment (T, solid lines) and control (C, dashed lines) cities, Stanford Five-City Project, northern California, during two periods: while intervention was occurring (early period, 1979–1985; left) and following intervention (late period, 1986–1992; right). The lines were fitted by simple linear regression; mixed model regression analysis indicated a significant period effect (the rate of decline was significantly greater in the late period than the early period). Rates per 100,000 persons.

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detectable impact on the combined-event rate trend is reinforced by the lack of any difference found in the linear component of the rate trends over the entire time period 1979–1992 (figure 1). Since some risk factor differences benefiting the treatment cities were observed, they were either insufficient to affect morbidity and mortality (given the precision of our measurement methods) or were countered by other, unmeasured factors.

Two other major community intervention studies have been conducted in the United States, in Minnesota (37) and Rhode Island (38). To date, only the Minnesota Heart Health Program has reported results for morbidity and mortality, and it also failed to find compelling evidence of an intervention effect (15). While numerous community trials have been conducted outside the United States, only the North Karelia Project monitored morbidity and mortality trends. This study began in 1972, when the intervention area had the highest rate of coronary disease in the world, in men. Coronary disease was also highly prevalent in the control area and in Finland as a whole. There have been strong declines in coronary disease rates in Finland since 1972. Although the limited number of units precludes having great confidence in causal inference, it does appear that the intervention program was associated with an acceleration of the decline in coronary disease rates (39). The remarkable decline in coronary disease in Finland over the 20 years following 1972 was associated with significant declines in risk factors, which prediction models indicate largely explains the decline in disease rates (40, 41).

The US community intervention studies were modeled to some extent on clinical trial experience; when these studies were designed in the mid-1970s, it was considered obligatory to include assessment of disease endpoints in addition to risk factor change. In retrospect, it was probably naive to think that these interventions could produce detectable changes in such endpoints (42). Even if the projected risk factor changes had been achieved during the planned 5- to 6-year interventions, they would have occurred toward the end of that time, before the full effect was in place, and there would then presumably be a delay between risk factor change and morbidity–mortality change (2 or 3 years, based on clinical trial results). In the mid-1970s, we also underestimated the strength of the secular trends in both risk factor change and coronary disease incidence in the United States as a whole and in the control cities in particular. We also clearly overestimated the ability of the education efforts to accelerate lifestyle and risk factor changes; many more sustained efforts are likely necessary (43). The North Karelia Project (39–41) results illustrate this point.

Another unanticipated problem was the possibility that highly effective clinical therapies would be introduced widely, including in the control areas, obscuring any effect of the risk factor change. The Stanford Five-City Project never intended to alter the medical and surgical treatment of coronary disease. While it still seems unlikely that we observed any intervention effect, the data presented here are consistent with an effect from some rapidly diffusing treatment innovation occurring in the mid-1980s.

The Stanford Five-City Project was not designed to explore secular trends in acute coronary care. After the study began, we became a participant in the World Health Organization MONICA Project, an international study to MONItor trends and determinants of Cardiovascular disease (44, 45), and began collecting limited data on such treatment. It is beyond the scope of this paper to explore these data fully, but the downturn in event rates observed appears to have occurred well before the rise in revascularization rates and to predate the widespread introduction of thrombolytic therapy. This observation supports the usefulness of CVD surveillance, which is not performed systematically in the United States.

Other limitations of this study include its limited generalizability because of the small number of cities selected, its quasi-experimental design, and possible confounding from differential secular trends in migration; unrecognized, nonfatal myocardial infarction; or other variables affecting the events included in the study. There are also other limitations to the accuracy and precision of the surveillance system and to the statistical modeling.

In summary, the Stanford Five-City Project documented a steepening in the decline of CVD morbidity and mortality that began relatively suddenly about the time that the education program ended in the two treatment cities, but it appears to have occurred similarly in the control cities. While the rate trends during the intervention were less favorable in both treatment cities than in the control cities, it is speculative to presume that this difference would have continued in the absence of the intervention. It is more likely that some influence affecting all cities, perhaps an innovation in the medical management of CVD, accounted for the observed change. Further exploration of this possibility is warranted. Public health policies concerning control of cardiovascular risk factors must rest on considerations other than the overall morbidity and mortality findings of US community intervention trials.

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REFERENCES
4. Salonen JT, Puska P, Kottke TE, et al. Changes in smoking, serum cholesterol and blood pressure levels during a commu-