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DID EARLY DETECTION AND TREATMENT CONTRIBUTE TO THE DECLINE IN ISCHEMIC HEART DISEASE MORTALITY? PROSPECTIVE EVIDENCE FROM THE ALAMEDA COUNTY STUDY

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Cohn, B. A. (Human Population Laboratory, California Public Health Foundation, Berkeley, CA 94704), G. A. Kaplan, and R. D. Cohen. Did early detection and treatment contribute to the decline in ischemic heart disease mortality? Prospective evidence from the Alameda County Study. *Am J Epidemiol* 1988;127:1143–54.

In a previous study, the authors reported a 45 per cent decline in ischemic heart disease mortality between cohorts selected to be representative of Alameda County, California, in 1965 and 1974. The decline could not be explained by baseline differences in the distribution of many of the known ischemic heart disease risk factors available for analysis in this cohort. This study reports the results of further analyses which evaluated the hypothesis that early detection and improved treatment contributed to the decline. In multiple logistic analyses adjusted for age, sex, and race, those who reported heart trouble at baseline had an ischemic heart disease mortality decline 2.5 times greater than those who did not ($p = 0.01$). Those who used preventive health services had an ischemic heart disease mortality decline 2.2 times greater than those who did not ($p = 0.03$). These interactions were independent of each other and were not explained by adjustment for physical activity, smoking, social connections, or body mass index. There was an increase in the prevalence of self-reported heart trouble between 1965 and 1974, especially among younger age groups. These results are consistent with the hypothesis that early detection and treatment contributed to the decline in ischemic heart disease mortality observed in the Alameda County Study.

cardiovascular diseases; coronary disease; longitudinal studies; mortality

The decline in ischemic heart disease mortality observed since the late 1960s may be due to primary prevention and/or to improved medical care (1–3). In a previous analysis (4), we reported that the 45 per cent decline in ischemic heart disease mortality between the 1965 and 1974 cohorts of the Alameda County Study was not explained by reduced prevalence of many conventional risk factors, including cigarette smoking, inactivity during leisure time, underweight or overweight, social isolation, and report of hypertension (blood pressure and lipid measures were unavailable). The present paper examines the hypothesis that early detection and treatment contributed to the decline in ischemic heart disease mortality.

The evidence to examine this hypothesis in these data is indirect since information about severity of baseline disease, timing of diagnosis, and treatment is not available. However, if improvements in detection and

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treatment of ischemic heart disease have occurred in this population, they would be manifested by differential declines for subgroups.

A greater decline for those who reported heart trouble at baseline than for those who did not would suggest that either secondary prevention had an important role in the decline, or earlier detection had resulted in reduced severity of disease among those reporting heart trouble in the later cohort. A greater decline for those who used preventive care than for those who did not would suggest that earlier detection and/or treatment contributed to the ischemic heart disease mortality decline. In addition, a secular increase in the prevalence of self-reported heart trouble, especially among younger age groups, would be consistent with earlier detection and greater public awareness of heart disease symptoms. Since mortality declines in any of these subgroups could be explained by changes in life-style, improvements in medical interventions, or both, we also examined the role of secular changes in smoking, physical activity, weight, and social networks in explaining the differential declines observed in ischemic heart disease mortality.

In summary, improved detection and treatment should lead to the following predictions: 1) the decline in risk of ischemic heart disease mortality should be greater among those who reported heart trouble than among those who did not; 2) the decline in risk should also be greater among persons who used preventive medical services than among those who did not use them; and 3) the prevalence of heart trouble should be higher in the 1974 cohort than in the 1965 cohort, especially at younger ages.

Materials and methods

In 1965 and 1974, cohorts representative of Alameda County, California, were selected by the Human Population Laboratory of the California Department of Health Services. The study population has been described elsewhere (4, 5). The 1965 cohort consists of 6,928 persons: 3,158 males and 3,770 females. The 1974 cohort consists of 3,119 persons: 1,409 males and 1,710 females. The present analyses are restricted to white participants and black participants aged 40 years and over (n = 3,751 for the 1965 cohort, n = 1,551 for the 1974 cohort, or 5,302 for both cohorts). Persons with missing data on variables were excluded (categories are not mutually exclusive): high blood pressure (n = 16), chest pain (n = 22), heart trouble (n = 11), self-assessed physical activity (n = 30), marital status (n = 2), body mass index (n = 52), and utilization of preventive care (n = 53), resulting in a sample size of 5,104 for analyses using all variables and slightly larger samples for analyses using subsets of these variables.

Mortality was ascertained for both cohorts using a computer-matching linkage with the California Death Registry to obtain the death certificates of persons who had died in California or who had died outside the state with notification to California. The process is described elsewhere (6, 7). Persons not known to be dead are considered alive. Loss to follow-up was estimated from a 1974 follow-up of the 1965 cohort and is approximately 4 per cent over nine years. Ischemic heart disease death was coded according to the *International Classification of Diseases*, Eighth Revision, codes 410–414, for both cohorts to ensure comparability. There were 218 ischemic heart disease deaths (5.8 per cent) in the 1965 cohort and 72 ischemic heart disease deaths in the 1974 cohort (4.6 per cent) during their respective nine-year follow-up periods.

Risk factor data were collected by self-administered questionnaires. The questions were identical in 1965 and 1974. The report by Kaplan et al. (4) describes the method of measurement and coding for all variables except utilization of preventive care. In the present study, utilization of preventive care was dichotomized: never-

users were coded as 0; ever-users were coded as 1.

The use of self-reported data raises issues of validity. However, some investigators have found that self-reported data for smoking (8), height, and weight (9) have adequate validity. Also, self-reports of heart trouble, chest pain, and high blood pressure have been shown to predict subsequent ischemic heart disease mortality in the Alameda County Study cohort (4, 10). Self-reported smoking, height, weight, the leisure activity score, and the social network index have also demonstrated predictive validity in the Alameda County Study (5).

Analyses

We examined the hypothesis that the decline in ischemic heart disease mortality between the 1965 and 1974 cohorts differed within subgroups. This hypothesis, which in statistical terms is an interaction hypothesis, was examined using stratified analyses and logistic regression. Two parallel sets of analyses were performed: the first examined the interaction between reported heart trouble and cohort membership; the second examined the interaction between use of preventive medical services and cohort membership. The analysis plan is described in terms of the first set of analyses.

To quantify the decline in mortality between cohorts, age- and sex-adjusted nine-year risks of ischemic heart disease mortality were calculated for each cohort for those reporting or not reporting heart trouble. Adjustment was by the direct method using the age and sex distribution for both cohorts combined.

The decline in mortality was measured by the cohort relative risk: the ratio of nine-year ischemic heart disease mortality risks for the two cohorts. The ratio of the 1965 adjusted risk to that for 1974 corresponds to a secular decline in risk when relative risk is greater than one. The decline can also be expressed as a percentage of the risk in the 1965 cohort as: per cent decline = $100 \times (1965 \text{ risk} - 1974 \text{ risk})/1965 \text{ risk}$. This formula can be written in terms of relative risk as: per cent decline = $100 \times (\text{relative risk} - 1)/\text{relative risk}$.

Since the per cent decline is a one-to-one function of the relative risk, confidence intervals for the per cent decline are obtainable from those for the relative risk. Finally, the relative risk can be easily modeled, which facilitates the comparison of the declines between subgroups while controlling for other variables.

Logistic regression analysis was used to estimate the per cent decline in mortality while adjusting for multiple confounding variables. In these models, cohort membership was represented by a binary variable (1 = 1965 cohort, 0 = 1974 cohort). The approximate relative risk (odds ratio) estimated from the regression coefficient for cohort membership was used to calculate the per cent decline in the formula given above. The odds ratio was considered to be a satisfactory approximation to the risk ratio because of the low risk of ischemic heart disease mortality.

To compare the declines between subgroups, e.g., between those reporting and not reporting heart trouble, logistic models were fit containing the binary variable for cohort membership, a binary variable for heart trouble (1 = heart trouble, 0 = no heart trouble), and their product (cohort × heart trouble). The models also contained other relevant adjustment variables.

Inclusion of the product term in the model allows one to estimate the decline in mortality separately for each subgroup. The ratio of the subgroup specific cohort relative risks is estimated by exponentiating the coefficient for the product term. If this ratio is one, it implies equality of the subgroups in terms of both cohort relative risks and per cent declines. Consequently, subgroup equality can be assessed by hypothesis testing or interval estimation applied to this regression coefficient. Note that the exponentiated value of the interaction coefficient is equal to the ratio of the

cohort relative risks for subgroups but not to the ratio of per cent declines. However, equality of cohort relative risks implies equality of per cent declines.

RESULTS

Differential decline by report of heart trouble at baseline

Table 1 presents age- and sex-specific nine-year ischemic heart disease mortality risks by cohort and heart trouble. Among those with heart trouble, all age and sex groups had a decline in nine-year mortality between the 1965 and 1974 cohorts. Among those without heart trouble, all but two groups (females under age 60 and females aged 70 and over) showed a decline. The age- and sex-adjusted decline for those with heart trouble was 55 per cent (95 per cent confidence interval (CI) = 21–74 per cent). Those without heart trouble had a 19 per cent decline (95 per cent CI = 9 per cent increase to 39 per cent decline).

Table 2 presents results of the logistic analyses which examined the interaction between the decline and self-report of heart trouble. There was a sizable interaction in all of these models. With adjustment for age, sex, and race, the cohort relative risk for ischemic heart disease mortality among those reporting heart trouble was 2.5 times that for those who did not report it (95 per cent CI = 1.3–5.2). In percentage terms, those who reported heart trouble had a 72 per cent decline in risk (95 per cent CI = 46–85 per cent) compared with a 27 per cent decline for those not reporting heart trouble (95 per cent CI = 2 per cent increase to 48 per cent decline). The magnitude of the interaction was not substantially changed by adjustment for current and past smoking, physical activity, chest pain, high blood pressure, body mass index, social connections, or use of preventive health care. Simultaneous adjustment for all of these risk factors reduced the ratio of cohort relative risks to 1.9 (95 per cent CI = 0.9–3.9).

Differential decline by use of preventive health services

Table 3 presents age- and sex-specific nine-year ischemic heart disease mortality risks by cohort and use of preventive health services. Among those who used preventive health services, all but one age and sex group (women under age 60) had a decline in nine-year mortality between the cohorts. Among those not reporting use of preventive care, only two groups (females under age 60 and males aged 70 and over) had a decline in mortality risk. The age- and sex-adjusted decline for users of preventive services was 41 per cent (95 per cent CI = 20–56 per cent). Nonusers of preventive health services had a 29 per cent increase in mortality risk between cohorts after ad-

TABLE 1

Age-, sex-specific nine-year ischemic heart disease mortality risk by self-report of heart trouble at baseline for the 1965 and 1974 cohorts: Alameda County Study

Sex and age (years)	With heart trouble				No heart trouble			
	1965 cohort		1974 cohort		1965 cohort		1974 cohort	
	%	No. at risk	%	No. at risk	%	No. at risk	%	No. at risk
Males								
<60	11.6	43	7.3	41	2.4	1,129	1.5	411
60–69	38.5	39	4.2	24	7.7	273	7.2	110
70+	47.1	34	25.0	12	25.8	178	16.9	89
Females								
<60*	0.0	32	0.0	26	0.5	1,304	0.8	497
60–69	23.4	47	12.9	31	5.6	324	2.9	137
70+	25.8	62	11.1	45	13.4	277	14.3	126

* Stratum excluded from calculation of age-, sex-adjusted ischemic heart disease mortality decline for those with heart trouble (see text).

TABLE 2

Effect of adjustment for risk factors on the interaction between cohort membership and self-reported heart trouble for predicting nine-year ischemic heart disease (IHD) mortality: Alameda County Study, 1965 and 1974 cohorts

Adjustment variables	Interaction coefficient (p)	Ratio of IHD mortality decline* in those with heart trouble compared to those without	95% confidence interval
Age, sex, race only†	0.94 (0.009)	2.55	1.25–5.17
Plus current smoking and former smoking	0.95 (0.009)	2.58	1.27–5.25
Plus high blood pressure	0.92 (0.01)	2.50	1.23–5.12
Plus chest pain	0.85 (0.02)	2.34	1.15–4.74
Plus body mass index	0.87 (0.02)	2.39	1.17–4.88
Plus leisure-time physical activity	0.96 (0.008)	2.62	1.29–5.33
Plus self-assessed physical activity	0.94 (0.01)	2.56	1.26–5.23
Plus preventive health care use	0.78 (0.03)	2.12	1.03–4.35
Plus social connections‡	0.90 (0.01)	2.47	1.21–5.02
All of the above variables	0.63 (0.09)	1.88	0.90–3.92

* Relative risk for cohort membership (1965/1974) for those who reported heart trouble divided by relative risk for cohort membership (1965/1974) for those who did not report heart trouble.

† Each of the following variables or sets of variables were added to the logistic model containing age, sex, and race, one at a time.

‡ This step consisted of adjusting for marital status, social group membership, church group membership, and social isolation simultaneously.

TABLE 3

Age-, sex-specific nine-year ischemic heart disease mortality risk by use of preventive health services at baseline for the 1965 and 1974 cohorts: Alameda County Study

Sex and age (years)	Users of preventive care				Nonusers of preventive care			
	1965 cohort		1974 cohort		1965 cohort		1974 cohort	
	%	No. at risk	%	No. at risk	%	No. at risk	%	No. at risk
Males								
<60	3.0	1,015	1.3	379	1.3	153	5.6	72
60–69	11.6	277	6.2	113	9.7	31	10.0	20
70+	27.2	180	15.6	77	41.9	31	30.0	20
Females								
<60	0.4	1,195	0.8	453	1.1	131	0.7	70
60–69	7.3	327	4.1	148	9.8	41	11.1	18
70+	16.3	277	9.9	132	13.0	54	25.7	35

justment for age and sex. However, the 95 per cent confidence interval for this value was very broad, ranging from a 21 per cent decline to a 113 per cent increase.

Table 4 presents results of the logistic analyses which examined the interaction between the decline and use of preventive health services. There was a sizable inter-

TABLE 4

Effect of adjustment for risk factors on the interaction between cohort membership and use of preventive health services for predicting nine-year ischemic heart disease (IHD) mortality: Alameda County Study, 1965 and 1974 cohorts

Adjustment variables	Interaction coefficient (*p*)	Ratio of IHD mortality decline* in preventive care users compared to nonusers	95% confidence interval
Age, sex, race only†	0.78 (0.03)	2.18	1.06–4.43
Plus current smoking and former smoking	0.79 (0.03)	2.20	1.07–4.50
Plus heart trouble	0.85 (0.02)	2.34	1.14–4.78
Plus high blood pressure	0.74 (0.04)	2.10	1.02–4.35
Plus chest pain	0.81 (0.03)	2.25	1.10–4.59
Plus body mass index	0.86 (0.02)	2.33	1.15–4.76
Plus leisure-time physical activity	0.77 (0.04)	2.16	1.05–4.42
Plus self-assessed physical activity	0.81 (0.03)	2.24	1.09–4.59
Plus social connections‡	0.78 (0.03)	2.17	1.06–4.47
All of the above variables	0.89 (0.02)	2.44	1.17–5.10

* Relative risk for cohort membership (1965/1974) for those who used preventive health care services divided by relative risk for cohort membership (1965/1974) for those who did not use preventive health care services.

† Each of the following variables or sets of variables were added to the logistic model containing age, sex, and race, one at a time.

‡ This step consisted of adjusting for marital status, social group membership, church group membership, and social isolation simultaneously.

action in all of these models. With adjustment for age, sex, and race, the cohort relative risk for ischemic heart disease mortality among those who used preventive health services was 2.2 times that for those who did not use them (95 per cent CI = 1.1–4.4). In percentage terms, it was estimated that those who used preventive health services had a 49 per cent decline in risk (95 per cent CI = 21–64 per cent) compared with an 11 per cent increase for those not using them (95 per cent CI = 40 per cent decline to 107 per cent increase). The magnitude of the interaction was not substantially changed by adjustment for current and past smoking, physical activity, chest pain, high blood pressure, body mass index, social connections, or heart trouble. Simultaneous adjustment for all of these risk factors increased the ratio of cohort relative risks slightly, to 2.4.

The interactions between cohort membership and use of preventive health services and heart trouble were independent of each other. In a model containing all ischemic heart disease risk factors (tables 2, 4) and both interactions, the ratio of cohort relative risks was 2.4 (95 per cent CI = 1.2–5.0) for preventive health care use and 1.8 (95 per cent CI = 0.9–3.8) for heart trouble.

Prevalence of self-reported heart trouble

Table 5 presents the prevalence of self-reported heart trouble by age, sex, and cohort. For all but one group (males aged 70–79), the 1974 cohort had a higher preva-

lence of heart trouble than the 1965 cohort.

A logistic regression model was used to predict prevalence of self-reported heart trouble from age, sex, race, cohort, current smoking status, high blood pressure, physician visits within the last year, and the following interactions: age and cohort, age and sex, age and preventive care, preventive care and sex, preventive care and cohort, and a three-way term for age, cohort, and preventive care. The age × sex ($p = 0.005$) and preventive care × sex interactions ($p = 0.006$) were required in the model to provide the best fit to the data.

Current smoking, high blood pressure, and physician visits were strongly associated with reported heart trouble (table 6). There was an interaction ($p = 0.05$) between age, preventive health care use, and cohort membership. For both those who did and did not use preventive health services, the secular increase in heart trouble prevalence was largest at younger ages. However, the rate of decline with age was greater for those who did not use preventive health care. Among those aged 60 and over, there was essentially no estimated secular increase in heart trouble prevalence for either preventive care users or nonusers in contrast to increases for those under age 60.

TABLE 5

Prevalence of self-reported heart trouble by age, sex, and cohort: Alameda County Study, 1965 and 1974 cohorts

Sex and age (years)	1965		1974	
	%	No. at risk	%	No. at risk
Males				
40–49	2.9	699	4.2	217
50–59	4.9	474	13.6	235
60–69	12.5	313	17.9	135
70–79	16.9	155	9.6	73
80+	13.8	58	17.8	28
Females				
40–49	1.6	775	4.8	252
50–59	3.6	563	5.2	272
60–69	12.7	374	18.5	168
70–79	18.2	258	25.9	116
80+	18.3	82	27.2	55

DISCUSSION

The significant interactions observed in this study can be interpreted from two different perspectives: 1) there were different declines for subgroups, and 2) there were secular changes in risk associated with subgroup membership. Figure 1 illustrates these two perspectives by graphing risk as estimated by the interaction models. Decline differences for subgroups are seen by comparing slopes for subgroups. In addition, secular differences in the strength of associations between risk factors and mortality are seen by comparing vertical distances between points for the 1965 and 1974 cohorts. These data support the hypothesis that the association between report of heart trouble and ischemic heart disease mortality declined between the

TABLE 6

Correlates of self-reported heart trouble prevalence in the 1965 and 1974 cohorts as estimated by the logistic model: Alameda County Study*

Correlates of self-reported heart trouble	Prevalence odds ratio	95% confidence interval
Current smoking (vs. never smoking)	1.39	1.07–1.82
Former smoking (vs. never smoking)	1.20	0.89–1.62
High blood pressure (vs. none)	2.90	2.31–3.64
Physician visit in past year (vs. none)	8.19	4.80–13.97

* Results reported for multivariate model adjusted for age, sex, race, preventive health care use, and the following interactions: age × sex, age × cohort, age × preventive care, preventive care × sex, preventive care × cohort, age × preventive care × cohort. There was a three-way interaction between age, use of preventive care, and cohort: coefficient = 0.048, $p = 0.05$. Details for coding independent variables in this model are found in Kaplan et al. (4).

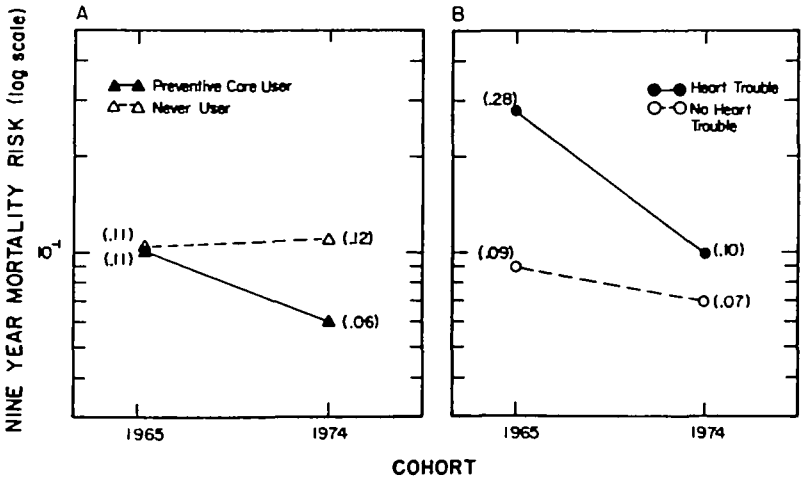

FIGURE 1. A) Nine-year ischemic heart disease mortality risk in the 1965 and 1974 cohorts of the Alameda County Study by preventive care use estimated by a logistic model containing terms for age, sex, race, cohort, preventive care use, and cohort × preventive care use evaluated for white males age 60. The 95% confidence interval (CI) for the preventive care relative risk in the 1974 cohort excludes 1. The 95% CI for the decline in risk among preventive care users excludes 0. The interaction between the decline and preventive care use was significant ($p = 0.03$). B) Nine-year ischemic heart disease mortality risk in the 1965 and 1974 cohorts of the Alameda County Study by report of heart trouble estimated by a logistic model containing terms for age, sex, race, cohort, heart trouble, and cohort × heart trouble evaluated for white males age 60. The 95% CI for the heart trouble relative risk in the 1965 cohort excludes 1. The 95% CI for the decline in risk among those with heart trouble excludes 0. The interaction between the decline and heart trouble was significant ($p = 0.01$).

1965 and 1974 cohorts. For the same period, the protective effect of preventive health care utilization may have increased.

There are several possible explanations for the decline patterns observed in this study. First, the interactions observed could have been due to differences in lifestyle among subgroups. This does not appear to be a plausible explanation in these data. Adjustment for current smoking, former smoking, physical activity, body mass index, or social connections did not explain the interaction between cohort membership and either heart trouble or preventive health care utilization. It is possible that other ischemic heart disease risk factors not measured in this study, including blood pressure readings and/or lipoprotein levels, could explain the decline patterns observed. However, correlates of these two factors, body mass index and exercise, did not explain the observed decline patterns even though they predicted ischemic heart disease mortality as expected (4). Differences in baseline health, measured as report of high blood pressure, chest pain, or heart trouble, also did not explain the interaction between cohort and use of preventive health services.

An alternative explanation of the reduced association between self-reported heart trouble and ischemic heart disease mortality in the 1974 cohort is a secular increase in false positives among those reporting heart trouble. This remains a possible explanation which cannot be evaluated directly in these data. However, comparisons between self-reported data in the Health Interview Survey, 1972, and examination data from the Health Examination Survey, 1960–1962, suggest that self-report underestimates rather than overestimates disease prevalence (11).

Comparison of age- and sex-specific prevalence of definite and suspect coronary heart disease estimated by the Health Examination Survey, 1960–1962 (12), with self-reported prevalence of heart trouble in the 1965 Alameda County Study cohort suggests that self-reported heart trouble

may be an underestimate of actual prevalence of ischemic heart disease for the 1965 cohort, with the possible exception of the oldest age groups. Prevalence comparisons by age and sex for the Health Examination Survey compared with self-report for this study are shown in the Appendix. Of course, differences between national data and those for Alameda County may also be partly due to true differences in prevalence.

In previous reports for the Alameda County sample (4, 10), self-reported heart trouble, high blood pressure, and chest pain predicted ischemic heart disease mortality. In addition, the behavioral correlates of self-reported heart trouble prevalence (table 6) are consistent with known associations between ischemic heart disease morbidity and both cigarette smoking and high blood pressure. The high proportion of those reporting heart trouble who sought medical care specifically for that condition (98 per cent for the 1974 cohort, data not available for the 1965 cohort) also suggests that self-reported heart trouble may be a reasonable proxy for prevalent disease. The proportions of those persons who reported heart trouble and who sought medical care in this study are slightly higher than those reported for the Health Interview Study (11). In addition, prevalence of self-reported heart trouble by age in the 1974 cohort was close to that reported in the Health Interview Survey for 1972 (9.1 per 100 vs. 8.8 per 100 for ages 45–64 and 21.1 per 100 vs. 19.9 per 100 for ages 65 and over) (11).

Since there was a secular increase in heart trouble prevalence among younger persons in this sample, regardless of utilization of preventive care, it is plausible that improvements in routine screening, as well as earlier recognition of symptoms, contributed to earlier detection in the 1974 cohort. Existing studies of the epidemiology of ischemic heart disease during the 1960s documented substantial rates of silent myocardial infarction which went undetected in the absence of thorough screening (13, 14). During the late 1960s and early 1970s, there were widespread publicity campaigns to improve recognition of early warning signs of ischemic heart disease and urging prompt medical attention.

It is unlikely that the secular trends of increased prevalence of self-reported heart trouble in this study are completely explained by increases in the incidence of congenital, valvular, or hypertensive heart disease. Mortality from these forms of heart disease has been declining (2, 15). Since ischemic heart disease is proportionately the greatest component of self-reported heart trouble (11), it would be expected that increased prevalence of self-reported heart trouble would reflect increases in recognized ischemic heart disease prevalence.

It is not likely that the increased prevalence of self-reported heart trouble is due to a rise in the incidence of ischemic heart disease. And it is not likely that a rise in incidence would be observed during a period when mortality was declining. One population-based study (16) which has been able to examine secular changes in ischemic heart disease incidence for some manifestations reported a decline.

A combination of early detection and improved treatment may be a plausible explanation for the decline patterns observed in these data. Earlier detection would imply reduced severity of disease among those reporting heart trouble in the 1974 cohort. Earlier detection could have led to the observed reduction in relative risk associated with the report of heart trouble in the 1974 cohort, even in the absence of secular improvements in treatment.

However, since most persons who reported heart trouble in 1974 also sought medical care for the condition, it is possible that treatment played a role in the decline. In addition, the secular trend toward a stronger protective effect for preventive health care utilization supports the hypothesis that treatment, or at least direct medical intervention, may explain the decline pattern observed in these data. This hypothesis is further strengthened by the fail-

ure of many life-style risk factors to explain the observed declines in any of the subgroups.

Unfortunately, there are no direct measures of secular changes in treatment or in diagnostic techniques available in these data. However, there has been a large increase in the availability of specialists in cardiovascular disease treatment in California: from 1.0 per 100,000 population in 1966 to 3.6 per 100,000 in 1976 (17). Since this increase averages over both urban and rural areas, the increase in a highly urban area such as Alameda County may have been much greater. Other evidence suggests that, during the time period spanned by this study, there were substantial improvements in the diagnosis and treatment of both acute and chronic ischemic heart disease (3).

Evidence concerning the role of medical care in prolonging survival of ischemic heart disease patients comes mainly from the examination of secular trends in myocardial infarction survival. Some studies which have examined secular differences in in-hospital myocardial infarction survival suggest that there have been improvements in survival during the acute phase in-hospital (16, 18, 19). However, not all studies report improved survival for all patients in the acute phases of myocardial infarction (20, 21).

In contrast to findings for the acute phase of myocardial infarction, several studies which have examined secular trends in long-term myocardial infarction survival reported no improvements in long-term myocardial infarction survival (18, 19, 22). These studies compared time periods ranging from the 1960s to early and mid-1970s, with only one study (22) including data beyond 1975. It is possible that these studies failed to find improvements in long-term survival because the time periods studied preceded widespread use of beta-blocking agents at appropriate doses for long-term patient management (3).

There is little available evidence on secular changes in the prognosis of angina pectoris. A study of the Mayo Clinic population did report improvement in survival after initial diagnosis of angina between the periods 1965–1969 and 1970–1975 (18).

The ischemic heart disease survival benefits of coronary artery bypass surgery (23–26) and hypertensive treatment (27) have been described for some subgroups. It is difficult to ascertain the contribution of these treatments as assessed by intervention trials to the observed ischemic heart disease decline at the population level since this depends upon access to and utilization of medical care. However, these innovations may have contributed to the decline observed in these data. Interestingly, there was no interaction between cohort and report of hypertension in the present study.

Because of the intensive program to detect and treat hypertension begun after 1965, the report of "high blood pressure within the last 12 months" in this study may have different clinical meaning in 1965 than in 1974. For example, more of those who report not having high blood pressure in 1974 may be controlled hypertensives, resulting in a very high-risk group among those reporting hypertension within the last 12 months in 1974. This would obscure a positive interaction between cohort and self-reported hypertension and may account for our failure to detect an interaction.

Overall response rates for these two cohorts were relatively high: 86 per cent for the 1965 cohort and 80 per cent for the 1974 cohort. However, those who are seriously ill may tend to be nonrespondents. This bias would have reduced the mortality rates in those reporting heart trouble at baseline since only healthier persons would be included in the sample. However, the observed interaction between cohort and report of heart trouble could not be explained by this nonresponse bias unless this bias was greater in the 1974 cohort than in the 1965 cohort. We have no evidence to suggest this occurred, but it is possible.

Since the findings for a representative sample of Alameda County, California, may

not necessarily reflect patterns for other populations, it would be desirable to examine other long-term cohort studies for consistency with the findings presented here. A recent report from the Framingham Study (28) is consistent with the secular increase in cardiovascular disease prevalence reported here and with the hypothesis that improved survival among cases rather than reduction in incidence may explain the decline in ischemic heart disease mortality.

It is of interest to consider the special advantages that this data set provides for the examination of the role of early detection and/or treatment in the decline of ischemic heart disease mortality. Changes in the efficacy of treatment, or advantages of early diagnosis, are applicable only to those who seek treatment and who have access to treatment. In the absence of an acute, life-threatening event, the self-perception of illness or the routine utilization of preventive care services begins the process of treatment. For this reason, changes in the mortality rates associated with self-reported illness and use of preventive care services in a population sample are pertinent to understanding secular changes in mortality.

This report suggests that both self-report of heart trouble and utilization of preventive health services are important discriminators in predicting the magnitude of the decline in ischemic heart disease mortality, independent of many life-style risk factors. Perhaps other data can provide a means to examine the specific nature of secular changes in medical practice, medical utilization, or other risk factors not measured in this study that could explain the patterns of the decline in ischemic heart disease mortality observed. The results of this study suggest that future studies of the causes of the ischemic heart disease mortality decline should examine the following factors: 1) effects of early detection on survival; 2) secular changes in the perception of early ischemic heart disease symptoms and subsequent access to early or improved treatment; 3) the relative contribution of early detection versus improved treatment to the ischemic heart disease mortality decline; and 4) life-style changes that result from earlier detection which also influence subsequent ischemic heart disease mortality risk.

References

1. Stern MP. The recent decline in ischemic heart disease mortality. Ann Intern Med 1979;91:630–40.
2. Levy RI. The decline in cardiovascular disease mortality. Annu Rev Public Health 1981;2:49–70.
3. Goldman L, Cook EF. The decline in ischemic heart disease mortality rates. Ann Intern Med 1984;101:825–36.
4. Kaplan GA, Cohn BA, Cohen RD, et al. The decline in ischemic heart disease mortality: prospective evidence from the Alameda County Study. Am J Epidemiol 1988;127:1131–42.
5. Berkman LF, Breslow L. Health and ways of living: the Alameda County Study. New York: Oxford University Press, 1983.
6. Belloc NB, Arellano MG. Computer record linkage on a survey population. Health Serv Rep 1973;88:344–50.
7. Arellano MG, Petersen GR, Pettiti DB, et al. The California Automated Mortality Linkage System (CAMLIS). Am J Public Health 1984;74:1324–30.
8. Petitti DB, Friedman GD, Kahn W. Accuracy of information on smoking habits provided on self-administered research questionnaires. Am J Public Health 1981;71:308–11.
9. Stewart AL. The reliability and validity of self-reported weight and height. J Chronic Dis 1982; 35:295–309.
10. Kaplan GA, Kotler PL. Self-reports predictive of mortality from ischemic heart disease: a nine-year follow-up of the Human Population Laboratory cohort. J Chronic Dis 1985;38:195–201.
11. US Department of Health, Education, and Welfare. Prevalence of chronic circulatory conditions. Washington, DC: GPO, 1972. (DHEW publication no. (HRA)75-1521).
12. National Center for Health Statistics. Coronary heart disease in adults: United States 1960–1962. Washington DC: National Center for Health Statistics, 1965. (Vital and health statistics. Series 11, no. 10).
13. Roseman RH, Friedman M, Jenkins CD, et al. Clinically unrecognized myocardial infarction in the Western Collaborative Group Study. Am J Cardiol 1967;19:776–82.
14. Kannel WB, Abbott RD. Incidence and prognosis of unrecognized myocardial infarction. N Engl J Med 1984;311:1144–7.
15. Rosenberg HM, Klebba AJ. Trends in cardiovascular mortality with a focus on ischemic heart disease: United States, 1950–1976. Washington DC: GPO, 1979:109–14. (NIH publication no. 79-1610).
16. Gillum RF, Folsom A, Luepker RV, et al. Sudden death and acute myocardial infarction in a met-

ropolitan area, 1970–1980, the Minnesota Heart survey. N Engl J Med 1983;309:1353–8.
17. Goodman LJ. Physician distribution and medical licensure in the U.S., 1976. Chicago: American Medical Association, 1977.
18. Elveback LR, Connolly DC, Kurland LT. Coronary heart disease in residents of Rochester, Minnesota. II. Mortality, incidence, and survivorship (1950–1975). Mayo Clinic Proceedings 1981;56: 665–72.
19. Goldberg RJ, Szklo M, Tonascia JA, et al. Time trends in prognosis of patients with myocardial infarction: a population-based study. Johns Hopkins Med J 1979;144:73–80.
20. Goldberg RJ, Kennedy HL, Gore JM. Absence of secular changes in the prognosis of patients with an initial myocardial infarction. Clin Cardiol 1982;5:469–75.
21. Goldman L, Cook F, Hashimoto B, et al. Evidence that hospital care for acute myocardial infarction has not contributed to the decline in coronary mortality between 1973–1974 and 1978–1979. Circulation 1982;65:936–42.
22. Weinblatt E, Goldberg JD, Ruberman W, et al. Mortality after first myocardial infarction. JAMA 1982;247:1576–81.
23. Takaro T, Hultgren HN, Detre KM, et al. The Veterans Administration cooperative randomized study of surgery for coronary occlusive disease: II. Subgroup with left main lesions. Circulation 1976;54(suppl III):107–17.
24. Takaro T, Hultgren HN, Detre KM, et al. The Veterans Administration cooperative study of stable angina: current status. Circulation 1982;65 (suppl II):60–7.
25. European Coronary Surgery Study Group. Prospective randomized study of coronary bypass study in stable angina pectoris: a progress report on survival. Circulation 1982;65(suppl II):67–71.
26. CASS principal investigators and their associates. Coronary Artery Surgery Study (CASS): a randomized trial of coronary artery bypass surgery: survival data. Circulation 1983;68:939–50.
27. Stamler J, Stamler R. Intervention for the prevention and control of hypertension and atherosclerotic diseases: United States and international experience. Am J Med 1984;76(2A):13–36.
28. Sytkowski PA, Kannel WB, Wolf PA, et al. Mechanism of the decline in cardiovascular disease mortality: the Framingham Study. (Abstract). Am J Epidemiol 1987;126:741.

APPENDIX

*Comparison of self-reported heart trouble prevalence in the Alameda County Study 1965 cohort with heart trouble prevalence estimated by the National Health Examination Survey (HES) 1960–1962**

Age (years)	Prevalence per 100 (no. at risk)			
	Males		Females	
	Alameda County	HES	Alameda County	HES
45–54	4.6 (609)	6.9 (693)	2.7 (662)	4.1 (433)
55–64	8.7 (366)	14.1 (1,060)	6.4 (454)	9.9 (807)
65–74	14.5 (234)	16.8 (837)	15.9 (314)	14.3 (886)
75–79	18.9 (53)	13.0 (185)	20.8 (120)	11.9 (172)

* HES data from reference 12, table 2, p. 16. Prevalence of definite and suspect coronary heart disease.