

Are Vacations Good for Your Health? The 9-Year Mortality Experience After the Multiple Risk Factor Intervention Trial

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Objective: The objective of this study was to determine the risk for various causes of posttrial death associated with vacation frequency during the Multiple Risk Factor Intervention Trial (MRFIT). **Methods:** Middle-aged men at high risk for coronary heart disease (CHD) were recruited for the MRFIT. As part of the questionnaires administered during the first five annual visits, men were asked whether they had had a vacation during the past year. For trial survivors ($N = 12,338$), the frequency of these annual vacations during the trial were used in a prospective analysis of posttrial all-cause and cause-specific mortality during the 9-year follow-up period. **Results:** The relative risk (RR) associated with more annual vacations during the trial was 0.83 (95% confidence interval [CI], 0.71–0.97) for all-cause mortality during the 9-year follow-up period. For cause of death, the RRs were 0.71 (95% CI, 0.58–0.89) and 0.98 (95% CI, 0.78–1.23) for cardiovascular and noncardiovascular causes, respectively. The RR was 0.68 (95% CI, 0.53–0.88) for CHD (including acute myocardial infarction). These associations remained when statistical adjustments were made for possible confounding variables, including baseline characteristics (eg, income), MRFIT group assignment, and occurrence of a nonfatal cardiovascular event during the trial. **Conclusions:** The frequency of annual vacations by middle-aged men at high risk for CHD is associated with a reduced risk of all-cause mortality and, more specifically, mortality attributed to CHD. Vacationing may be good for your health. **Key words:** coronary heart disease, vacations, respite, restorative behaviors, Multiple Risk Factor Intervention Trial.

CHD = coronary heart disease; CI = confidence interval; DBP = diastolic blood pressure; ICD-9 = *International Classification of Diseases*, ninth revision; MI = myocardial infarction; MRFIT = Multiple Risk Factor Intervention Trial; RR = relative risk; SES = socioeconomic status; SI = special intervention; UC = usual care.

Psychological stress is positively associated with morbidity and mortality for a variety of diseases, including atherosclerosis (1), MI (2, 3), metabolic syndrome (4, 5), infectious disease (6, 7), and AIDS (8, 9). Stress is thought to influence disease risk through a number of pathways, including alterations in health behaviors such as smoking (10), alcohol consumption (11), compliance with medications (12), and emotional states (13). More recently, it has been recognized that normal restorative activities, such as sleep (14), exercise (15), and other leisure time activities, might influence disease risk. The present study examined whether frequent annual vacations, a common form of respite, serves a health protective function.

The Framingham Heart Study found an association between infrequent vacationing and increased inci-

dence of MI or death due to coronary causes during a 20-year follow-up of women participants (16). Because coronary death was not analyzed separately in this study (there were only 10 coronary deaths), this increased risk associated with infrequent vacationing does not necessarily represent an increased risk of mortality. In another study, men who developed psychosomatic illnesses were less likely to take vacations than men who never developed such illnesses (17).

Although taking regular annual vacations may serve a protective function, it is important to consider alternative explanations. For example, higher SES may produce both lower morbidity and mortality (18) and more frequent annual vacations, thereby producing a spurious association between frequent annual vacations and physical health. Along similar lines, poor health (eg, nonfatal MIs) may produce both higher rates of subsequent mortality (19) and prevent annual vacations. It is important to consider these alternative explanations when analyzing the effects of vacationing annually on mortality.

The purpose of this study was to evaluate 9-year posttrial mortality and cause of death among MRFIT participants as a function of the number of annual vacations assessed during 5 years of the trial. This study includes statistical controls for nonfatal health-related events and SES.

METHODS

Design of the MRFIT

A total of 361,662 men aged 35 to 57 years were initially screened at 22 clinical centers in 18 US cities for MRFIT. A total of 12,866 of these men were enrolled in the trial. At the time of study entry, these men were in the upper 10% to 15% of the risk score distribution, which was derived from the Framingham Heart Study data. Exclusion criteria included clinical evidence of CHD, based on history,

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physical examination, or electrocardiography (at rest); a serum cholesterol level ≥ 9.05 mmol/liter (350 mg/dl); DBP readings ≥ 115 mm Hg; or a body weight at least 50% greater than the standard weight for height (20–22). After exclusion, the remaining participants were randomly assigned to an SI ($N = 6428$) or UC ($N = 6438$) group. Participants in the SI group received dietary instructions designed to alter eating patterns to reduce the intake of saturated fats and to reduce blood cholesterol levels, a smoking cessation program, and stepped-care drug therapy for hypertension. Details of the multifactor intervention program are described elsewhere (23, 24). In our analyses, we included survivors of the trial ($N = 12,338$).

Measurement of Annual Vacations

Vacationing was assessed by an item on a checklist of life events ("Within the last 12 months, have you experienced a vacation?"). The vacation item was not included on the checklist used at baseline but was included on the checklist used at the first, second, third, fourth, and fifth annual examinations. This checklist did not include an option for indicating that an event had not occurred; as a result, a missing response on items on this questionnaire did not enable differentiation of missing data from intentional indications that an event had not occurred. However, the life events checklist was administered along with a number of other questionnaires; therefore, we treated data as missing for participants with known missing data for five forced-choice questions that either directly preceded or followed (depending on year) the life events questionnaire. Missing data increased steadily from year 1 ($N = 695$, 5.4%) to year 5 ($N = 1550$, 12.0%) because of participant death or attrition. Mean vacationing was computed for all valid responses and reflects the proportion of years the participant reported vacationing for those years with valid data.

Risk Factor Assessment

The following risk factors were considered: age at study entry; MRFIT group assignment (SI or UC); education, measured on a scale from 1 (eighth grade or less) to 9 (graduate or professional degree); total family income, measured on a scale from 1 ($< \$4200$) to 9 ($\geq \$35,000$); DBP, defined as the average of two random-zero manometer readings; serum cholesterol concentration; and cigarette smoking, determined by self-report. To index cumulative risk factor burden, we computed trial means for blood pressure, serum cholesterol, and smoking. For smoking, the average corresponds to the proportion of assessments when smoking was reported. Additional details about these assessments are published elsewhere (25, 26).

Morbidity and Mortality Ascertainment

A nonfatal cardiovascular event during the trial was defined as angina (Rose questionnaire), intermittent claudication (Rose questionnaire), congestive heart failure, peripheral arterial occlusive disease, stroke, left ventricular hypertrophy (electrocardiography), impaired renal function, accelerated hypertension, coronary artery bypass surgery, serial electrocardiographic evidence of MI, or definite clinical MI (determined by coding criteria of electrocardiographic tracings and/or physician inspection of hospital records) (27).

Since February 1982, vital status has been ascertained by matching identifying information reported by participants at the time of enrollment with the National Death Index. The latest search of the National Death Index was for all deaths through December 1990 and is considered to be essentially 100% complete (28). To determine

cause of death, death certificates were collected and coded independently by two nosologists using ICD-9 codes (29). Disagreements between the two nosologists were adjudicated by a third nosologist. Cause-specific mortality categories (and corresponding ICD-9 codes) considered in the present study were selected by the MRFIT Mortality and Morbidity Committee and are reported elsewhere (27).

Statistical Analyses

All analyses included trial survivors and considered mortality during the 9-year follow-up period. Posttrial mortality was analyzed using Cox proportional hazard regression equations and 95% CIs for the RRs associated with mean vacationing during the trial. In these analyses, proportion of years with report of vacation was treated as a continuous variable (ranging from 0 to 1.00). To control for possible confounding variables, additional analyses included age, MRFIT group assignment, DBP, serum cholesterol, education, family income, and cigarette smoking as covariates. Because the occurrence of nonfatal cardiovascular events (ie, morbidity) might both interfere with vacationing and predict future cardiovascular mortality, the occurrence of a nonfatal cardiovascular event during the trial was included as an additional covariate.

For analysis of differences in participants' trial characteristics as a function of annual vacationing, vacationing frequency was dichotomized into those reporting vacationing $\leq 60\%$ of the years surveyed (low-frequency group, $N = 6745$) and those reporting vacationing for $> 60\%$ of the years surveyed (high-frequency group, $N = 5822$) in these analyses. This dichotomous variable (low vs. high vacationing frequency) was used in analyses of variance for tests of continuous variables (blood pressure, age, educational attainment, smoking, and cholesterol) and a $2 \times 2 \chi^2$ analyses of group proportions for tests of categorical variables (ie, study group and occurrence of nonfatal cardiovascular events during the trial).

RESULTS

Characteristics of Participants Taking Frequent and Infrequent Vacations

For the five assessments of vacationing in the past year, 12.8% of participants in MRFIT reported never taking an annual vacation, 10.1% reported 1 annual vacation, 11.4% reported 2 annual vacations, 14.4% reported 3 annual vacations, 18.2% reported 4 annual vacations, and 25.6% reported 5 annual vacations. The remaining 7.6% had missing data; therefore, frequency of annual vacations for these participants was the proportion of years in which the participant took a vacation for those years with valid data. The differences in characteristics of participants taking frequent and infrequent annual vacations are shown in Table 1. The large sample in MRFIT provides power to detect rather small effects. Those taking frequent annual vacations during the trial were significantly younger at study entry, were less likely to be in the SI group, were less likely to report cigarette smoking, were more educated, had a higher family income, were less likely to experience a nonfatal cardiovascular event during the trial, and had a higher serum cholesterol level during the trial.

TABLE 1. Characteristics of Participants in MRFIT Taking Infrequent and Frequent Annual Vacations During the Trial

Participant Characteristics	Annual Vacationing		p
	Infrequent (N = 6745)	Frequent (N = 5822)	
Age at entry into study, years	46.57	46.21	.001
Study group, % in SI group	51.49	48.29	<.001
Smoking, %†	48.66	43.45	<.001
DBP, mm Hg ^a	86.36	86.21	.189
Serum cholesterol, mg/dl ^a	239.16	241.48	<.001
Education ^b	5.14	5.70	<.001
Total family income ^c	6.21	6.85	<.001
Nonfatal cardiovascular event during trial, %	24.02	19.34	<.001

^a Values are trial means.

^b Education was measured using a nine-point scale (1 = eighth grade or less, 9 = graduate or professional degree).

^c Total family income was measured using a nine-point scale (1 = <\$4200, 9 = ≥\$35,000).

Frequency of Annual Vacations During the Trial and Posttrial Mortality

The median length of follow-up for survivors of the trial through December 1990 was approximately 9 years (8.8 years; range = 7.8 to 10.1 years). During this time, 770 cardiovascular and 743 noncardiovascular deaths occurred. The RRs associated with more frequent annual vacations were 0.68 (95% CI, 0.59–0.79) for all-cause mortality, 0.61 (95% CI, 0.50–0.75) for cardiovascular mortality, and 0.77 (95% CI, 0.62–0.96) for noncardiovascular mortality. As shown in Table 2, after the addition of covariates to control for potential confounding factors, the RRs associated with more frequent annual vacations were 0.83 (95% CI, 0.71–0.97) for all-cause mortality, 0.71 (95% CI, 0.58–0.89) for cardiovascular mortality, and 0.98 (95% CI, 0.78–1.23) for noncardiovascular mortality.

In addition to analyses of all cardiovascular and all noncardiovascular deaths, specific causes of death within each category were considered. For cardiovascular mortality, the frequency of annual vacations was associated with a significant RR, 0.68 (95% CI, 0.53–0.88), for CHD. Within the CHD category, cause of death was further categorized into acute MI (ICD-9 code 410) and other ischemic (coronary) heart disease (ICD-9 codes 411–414 and 429.2). The RRs associated with more frequent annual vacations were marginally significant for acute MI (RR = 0.70, 95% CI, 0.49–1.01) and significant for other ischemic (coronary) heart disease (RR = 0.66, 95% CI, 0.46–0.94). The frequency of annual vacations was not associated with significant RRs for any other specific cause of cardiovascular mortality or noncardiovascular mortality.

TABLE 2. Cause of Death, Number of Deaths, and RR (with 95% CI) for Deaths Through 9 Years of Post-trial Follow-Up Associated With Frequency of Annual Vacations During the Trial

Cause of Death	Deaths (N)	RR (95% CI) ^a	p
All causes	1443	0.83 (0.71–0.97)	.018
All cardiovascular causes	745	0.71 (0.58–0.89)	.002
CHD	540	0.68 (0.53–0.88)	.003
Acute MI	262	0.70 (0.49–1.01)	.058
Other ischemic CHD	278	0.66 (0.46–0.94)	.020
Cardiac dysrhythmias	27	0.72 (0.23–2.23)	.571
Hypertensive heart disease	14	^b	
Other hypertensive	7	^b	
Cerebrovascular	61	0.86 (0.41–1.82)	.699
Other cardiovascular disease	97	0.76 (0.42–1.38)	.361
All noncardiovascular causes	696	0.98 (0.78–1.23)	.868
Neoplastic	464	1.16 (0.88–1.53)	.294
Lip, oral cavity, and pharynx	10	^b	
Digestive organs and peritoneum	120	1.25 (0.72–2.18)	.415
Colorectal	47	1.00 (0.42–2.40)	.996
Other gastrointestinal	73	1.45 (0.71–2.95)	.305
Respiratory and intrathoracic organs	187	1.15 (0.74–1.77)	.541
Lung	178	1.07 (0.69–1.68)	.751
Other neoplasms	147	1.12 (0.68–1.82)	.663
Respiratory	41	1.16 (0.45–2.95)	.760
Digestive system	48	0.54 (0.23–1.25)	.152
Accidents	57	0.65 (0.30–1.42)	.282
Other noncardiovascular disease	86	0.71 (0.38–1.34)	.292

^a In all Cox proportional hazard models, the following characteristics were included as covariates: age, study group (SI vs. UC), educational attainment, income, occurrence of a nonfatal cardiovascular event during the trial, smoking, DBP, and serum cholesterol (the later three values were trial averages). Missing covariate values resulted in a subject being dropped from the analysis.

^b Insufficient number of events for calculation of RR.

The effect of morbidity on both vacationing frequency and subsequent mortality is a particular concern when considering the effects of the frequency of annual vacations on subsequent mortality. Therefore, we conducted additional analyses using only the frequency of annual vacations assessed at the first, second, and third annual examinations to predict mortality in survivors of the trial. Using this approach, a 4-year gap is placed between the assessment of the frequency of annual vacations and the assessment of mortality, making it unlikely that any observed association between vacationing frequency and mortality would be a spurious association created by their common association with morbidity. In these analyses and with the addition of covariates to control for potential confounding factors, the RRs associated with more frequent annual vacations were 0.86 (95% CI, 0.76–1.01, *p* = .079) for all-cause mortality and 0.76 (95% CI, 0.60–0.95, *p* = .018) for CHD mortality.

DISCUSSION

More frequent annual vacations during the MRFIT was associated with a significant reduction in the risk of death during the 9-year posttrial period. The specific cause of death most strongly associated with vacationing frequency was CHD. This association persisted with the addition of statistical controls for baseline characteristics.

Although frequent vacationing may have a direct protective effect on health, it is important to consider alternative explanations for the observed associations. First, it is possible that morbidity produces both less frequent vacationing and an increased risk of death, thereby producing a spurious association between the frequency of annual vacations and mortality. In other words, those who are ill are both unable to take a vacation and more likely to die. The continued association of annual vacation frequency and CHD mortality in the context of statistical controls for nonfatal cardiovascular events and when using the frequency of annual vacations assessed during the first 3 years of the trial to predict mortality 4 years later (during the posttrial period) does not support this alternative explanation.

The positive association between SES and health is well documented (18), and in the current sample, lower SES was associated with less frequent vacationing. Therefore, another possibility is that those of lower SES are both unable to take a vacation and more likely to die. A continued significant association between vacationing frequency and CHD mortality in the context of a statistical control for participants' educational attainment and income does not support this alternative explanation.

There are a few possible mechanisms through which vacationing might have direct protective effects on health. First, vacations may reduce stress by removing ongoing stressors (eg, avoidance). The health benefits of stress reduction are well documented (30, 31). Furthermore, the current pattern of findings (ie, stronger effects with CHD relative to other causes of death, eg, cancer) is consistent with research demonstrating stronger stress and disease associations for CHD relative to cancer (32). Second, vacations may reduce stress by removing potential stressors and anticipated threats, providing a period of "signaled safety" (33). Anticipated threats are known to have adverse effects as great as (34), if not greater than (35), the threat itself. Finally, annual vacations may provide a unique opportunity for behaviors having restorative effects on anabolic physiological processes, such as social contact with family and friends (36–38) and physical activity

(15), in the context of reduction of stress-initiated catabolic effects.

Some limitations of the current study should be noted. First, vacationing frequency was assessed using a single question about "a vacation" within the past year. Therefore, we have no information about the quantity or length of vacations within each year nor information about the quality of these vacations. Such information might enable a description of the type and pattern of vacationing that have health-protective effects. Second, the MRFIT included the question about annual vacations as a measure of attention to the questionnaires. Therefore, it is possible that the generally hasty or less than fully compliant participants were both at greater risk of CHD death and reported few annual vacations as a consequence of low adherence. Finally, vacationing frequency may serve as a marker of other activities or personality characteristics that are, in turn, associated with the reduced risk of mortality. For example, those taking annual vacations may also engage in more health-promoting leisure-time activities. Leisure-time physical activity was measured in MRFIT using a questionnaire assessment of various physical activities. This questionnaire has been validated against treadmill exercise performance in MRFIT (39). Furthermore, leisure-time physical activity measured with this questionnaire had a modest inverse relationship with CHD and overall mortality during MRFIT (15). However, the inclusion of leisure-time physical activity as a covariate in the current analyses did not alter the significant risk reduction in CHD mortality associated with vacationing (RR = 0.69, $p = .006$ with all covariates, including leisure-time physical activity).

In conclusion, more frequent annual vacations during the MRFIT seemed to exert a direct positive effect on mortality during the 9-year posttrial period. Although the specific mechanism of this association remains unknown, these findings suggest the importance of considering the health benefits of restorative behaviors, such as vacationing. Vacations may not only be enjoyable but also health promoting.

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REFERENCES

1. Rozanski A, Blumenthal JA, Kaplan J. Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation* 1999;99:2192–217.

2. Kamarck T, Jennings JR. Biobehavioral factors in sudden death. *Psychol Bull* 1991;109:42-75.
3. Karasek RA, Theorell T, Schwartz JE, Schnall PL, Pieper CF, Michela JL. Job characteristics in relation to the prevalence of myocardial infarction in the US Health Examination Survey (HES) and the Health and Nutrition Examination Survey (HANES). *Am J Public Health* 1988;78:910-8.
4. Räikkönen K, Keltikangas-Järvinen L, Hautanen A, Adlercreutz H. Neuroendocrine mechanisms in chronic perceived stress: associations with the metabolic syndrome. *Endocrinol Metab* 1997;4:247-54.
5. Räikkönen K, Keltikangas-Järvinen L, Adlercreutz H, Hautanen A. Psychosocial stress and the insulin resistance syndrome. *Metabolism* 1996;45:1533-8.
6. Cohen S, Williamson GM. Stress and infectious disease in humans. *Psychol Bull* 1991;109:5-24.
7. Cohen S, Tyrrell DAJ, Smith AP. Psychological stress and susceptibility to the common cold. *N Engl J Med* 1991;325:606-12.
8. Leserman J, Jackson ED, Petitto JM, Golden RN, Silva SG, Perkins DO, Cai J, Folds JD, Evans DL. Progression to AIDS: the effects of stress, depressive symptoms, and social support. *Psychosom Med* 1999;61:397-406.
9. Evans DL, Leserman J, Perkins DO, Stern RA, Murphy C, Zheng B, Gettes D, Longmate JA, Silva SG, van der Horst CM, Hall CD, Folds JD. Severe life stress as a predictor of early disease progression in HIV infection. *Am J Psychiatry* 1997;154:630-4.
10. Cohen S, Lichenstein E. Perceived stress, quitting smoking, and smoking relapse. *Health Psychol* 1990;9:466-78.
11. Jennison KM. The impact of stressful life events and social support on drinking among older adults: a general population survey. *Int J Aging Hum Dev* 1992;35:99-123.
12. Coons SJ, Sheahan SL, Martin SS, Hendricks J, Robbins CA, Johnson JA. Predictors of medication noncompliance in a sample of older adults. *Clin Ther* 1994;16:110-7.
13. Eckenrode J. Impact of chronic and acute stressors on daily reports of mood. *J Pers Soc Psychol* 1984;46:907-18.
14. Kripke DF, Ancoli-Israel S, Mason W, Messin S. Sleep related mortality and morbidity in the aged. In: Chase M, Weitzman E, editors. *Sleep disorders: basic and clinical research*. New York: SP Medical & Scientific Books; 1983. p. 415-29.
15. Leon AS, Connett J, Jacobs DR Jr, Rauramaa R. Leisure-time physical activity levels and risk of coronary heart disease and death: the Multiple Risk Factor Intervention Trial. *JAMA* 1987;258:2388-95.
16. Eaker ED, Pinsky J, Castelli WP. Myocardial infarction and coronary death among women: psychosocial predictors from a 20-year follow-up of women in the Framingham Study. *Am J Epidemiol* 1992;135:854-64.
17. Vaillant GE. Natural history of male psychological health. IV. What kinds of men do not get psychosomatic illness? *Psychosom Med* 1978;40:420-31.
18. Adler NE, Boyce T, Chesney MA, Cohen S, Folkman S, Kahn RL, Syme SL. Socioeconomic status and health: the challenge of the gradient. *Am Psychol* 1994;49:15-24.
19. The Multiple Risk Factor Intervention Trial Research Group. Mortality after 16 years for participants randomized to the Multiple Risk Factor Intervention Trial. *Circulation* 1996;94:946-51.
20. Multiple Risk Factor Intervention Trial Research Group. Statistical design considerations in the NHLI Multiple Risk Factor Intervention Trial. *J Chronic Dis* 1977;30:261-75.
21. Sherwin R, Kaelber CT, Kezdi P, Kjelsberg MO, Thomas HE Jr. The Multiple Risk Factor Intervention Trial (MRFIT). II. The development of the protocol. *Prev Med* 1981;10:402-25.
22. Neaton JD, Grimm RH Jr, Cutler JA. Recruitment of participants for the Multiple Risk Factor Intervention Trial (MRFIT). *Controlled Clin Trials* 1987;8:41S-53S.
23. Benfari RC. Multiple Risk Factor Intervention Trial (MRFIT). III. The model for intervention. *Prev Med* 1981;10:426-42.
24. Caggiula AW, Christakis G, Farrand M, Hulley SB, Johnson R, Lasser NL, Stamler J, Widdowson G. Multiple Risk Factor Intervention Trial (MRFIT). IV. Intervention on blood lipids. *Prev Med* 1981;10:443-75.
25. Multiple Risk Factor Intervention Trial Research Group. Multiple Risk Factor Intervention Trial: risk factor changes and mortality results. *JAMA* 1982;248:1465-77.
26. Multiple Risk Factor Intervention Trial Research Group. Baseline rest electrocardiographic abnormalities, antihypertensive treatment, and mortality in the Multiple Risk Factor Intervention Trial. *Am J Cardiol* 1985;55:1-15.
27. Multiple Risk Factor Intervention Trial Research Group. Mortality after 16 years for participants randomized to the Multiple Risk Factor Intervention Trial. *Circulation* 1996;94:946-51.
28. Wentworth D, Neaton J, Rasmussen W. An evaluation of the Social Security Administration master beneficiary record file and the National Death Index in the ascertainment of vital status. *Am J Public Health* 1983;73:1270-4.
29. Anonymous. *International Classification of Diseases, North American Clinical Modification*. Ann Arbor (MI): Edwards; 1981;1.
30. McCubbin JA, Wilson JF, Bruehl S, Ibarra P, Carlson CR, Norton JA, Colclough GW. Relaxation training and opioid inhibition of blood pressure response to stress. *J Consult Clin Psychol* 1996;64:593-601.
31. Dusseldorp E, van-Elderen T, Maes S, Meulman J, Kraaij V. A meta-analysis of psychoeducational programs for coronary heart disease patients. *Health Psychol* 1999;18:506-19.
32. Rosengren A, Tibblin G, Wilhelmsen L. Self-perceived psychological stress and incidence of coronary artery disease in middle-aged men. *Am J Cardiol* 1991;68:1171-5.
33. Weinberg J, Levine S. Psychobiology of coping in animals: the effects of predictability. In: Levine S, Ursin H, editors. *Coping and health. NATO conference series III: human factors*. New York: Plenum; 1980. p. 39-60.
34. Spacapan S, Cohen S. Effects and aftereffects of stressor expectations. *J Pers Soc Psychol* 1983;45:1243-54.
35. Nomikos MS, Opton EM, Averill JR, Lazarus RS. Surprise versus suspense in the production of stress reaction. *J Pers Soc Psychol* 1968;8:204-8.
36. House JS, Landis KR, Umberson D. Social relationships and health. *Science* 1988;241:540-4.
37. Kaplan GA, Cohn BA, Cohen RD, Guralnik J. The decline in ischemic heart disease mortality: prospective evidence from the Alameda County study. *Am J Epidemiol* 1988;127:1131-42.
38. Orth-Gomer K, Johnson JV. Social network interaction and mortality: a six year follow-up study of a random sample of the Swedish population. *J Chronic Dis* 1987;40:949-57.
39. Leon AS, Jacobs DR Jr, DeBacker G, Taylor HL. Relationship of physical characteristics and life habits to treadmill exercise capacity. *Am J Epidemiol* 1981;113:653-60.