Feeling Vigorous and the Risks of All-Cause Mortality, Ischemic Heart Disease, and Diabetes: A 20-Year Follow-up of Healthy Employees

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Objective: To investigate prospectively the effects of vigor at work on the end points of mortality and the prevalence of ischemic heart disease (IHD) and diabetes. **Methods:** We tested the hypothesized beneficial effects of feeling vigorous at work at baseline on the risks of all-cause mortality, IHD, and diabetes during a 20-year follow-up. Participants were healthy employees (n = 968) who underwent a routine health check at baseline. We calculated the risk of all-cause mortality, IHD, and diabetes, with days as the time scale, using the Cox proportional hazard model. In our analyses, we predicted the above end points by baseline vigor, age, gender, and educational level, adjusting for the physiological risk factors of total cholesterol, glucose, and body mass index, the behavioral risk factors of smoking, alcohol intake, and physical activity, and the psychological risk factors of depressive and anxiety symptoms. **Results:** As hypothesized, we found that, after the above adjustments, baseline vigor decreased the risk of follow-up mortality by 26% (hazard ratio, 0.74; 95% confidence interval, 0.58–0.95) and the risk of diabetes by 17% (hazard ratio, 0.83; 95% confidence interval, 0.68–0.98). However, vigor did not have a significant effect on the risk of IHD. **Conclusions:** Independently of physiological, behavioral, and psychological risk factors, feeling vigorous at work protected the participants from diabetes and reduced their risk of mortality. **Key words:** vigor, positive affect, all-cause mortality, ischemic heart disease, diabetes, prospective design.

IHD = ischemic heart disease; **CHS** = Clalit Health Services; **BMI** = body mass index; **SES** = socioeconomic status.

INTRODUCTION

Vigor, a positive affect experienced at work, refers to individuals' feelings that they possess physical strength, emotional energy, and cognitive liveliness and has been defined primarily as a mood state (1). The term "positive affect" refers to any pleasurable engagement with the environment eliciting feelings, such as joy, vigor, happiness, and contentment (2); by "work-based affect," we mean an affect evoked by work-based targets (3,4). The scientific study of vigor is important because there is evidence suggesting that most people want to feel vigorous or energetic and view it as a significant dimension of their affective experiences (4). In comparison with other types of positive affects, vigor has been investigated in the emerging area of positive psychology to a limited extent only (5). Although it was assessed in relation to self-rated health (6), vigor has hardly been the focus of any study relating it to mortality and morbidity. Moreover, positive affects were hardly related to the prevalence of diabetes.

We follow the Differential Emotion theory (7), which argues that each basic human affect has distinct neurophysiologic, phenomenological, physiognomic, and motivational properties, because there is evidence supporting it with regard to positive affects (8). We focus on vigor because it represents a type of adaptive propensities that facilitate responses to experienced challenges and opportunities; at work, feeling vigorous is probably a necessary precursor to employees' motivational processes in organizations (1). As evident from qualitative (9) and quantitative (10) reviews, most past studies on the associations between positive affect and physical health used global measures of positive affect. For example, of the 25 past prospective studies that used psychological well-being to predict mortality, Chida and Steptoe (10) identified eight studies that used a specific positive affect: happiness (four studies); vitality (three studies); and energy (one study). Vitality is tangentially related to vigor because it is defined to include a sense of positive energy; however, it also includes positive well-being (11); therefore, it probably represents both positive affect and well-being.

In the current study, we focus on three end points: all-cause mortality; ischemic heart disease (IHD); and diabetes. The focus on IHD is due to the fact that it is a major component of cardiovascular disease—in turn, a principal cause of death in Israel (12) and in most Western countries (13). IHD is associated with multiple physiological, psychological, and sociodemographic risk factors that often interact in complex causal paths (14,15). Empirical evidence suggested that there are marked associations between positive psychological states and reduced cardiovascular disease risk (16), but vigor as a positive affect at work has yet to be investigated in this context. We focus on diabetes because it has been reported as growing in prevalence globally (17) and in Israel (18), and because of its well-documented risk for mortality in the Western world (17).

We expected baseline vigor to predict lower risks of follow-up all-cause mortality, IHD, and diabetes. The rationale for our expectations includes both theoretical arguments and empirical evidence. From a theoretical viewpoint, the broaden-and-build model of positive emotions, frequently used in the area of positive affect and health, proposes that positive affects, such as happiness, joy, pride, and love, have health-protecting physiological effects (19,20). The enhancing effects of positive affects on physical health and longevity are supported by an accumulating body of evidence (9,10,21). Recent research suggested that possible pathways linking vigor with improved physical health could include reduced levels of inflammatory processes in the body (22), and probably also lower levels of ambulatory heart rate and blood

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pressure, and lower salivary cortisol (23). Vigor may influence physical health by encouraging exercising to enhance physical fitness. Several reviews (24,25) found vigor and physical activity to be closely interrelated, predicting each other over time. Vigor, like other positive affects, accentuates the positivity of self-cognitions and prospectively predicts self-reported health (6). Self-reported health was found in several meta-analytic studies to be a unique predictor of longevity (6). Additionally, recent studies (26) found that positive affect predicted lower risk of mortality in diabetic patients. However, there is little prior work on the role of positive affects in the onset of diabetes (27).

METHODS

Participants

Using a unique 9-digit national identification number assigned at birth or immigration to each of the country's residents, we combined two data sets. The first data set was derived from the periodic health examinations and responses to a questionnaire completed by 1,042 participants, all continuously enrolled in Clalit Health Services (CHS)—the country's largest health maintenance organization—from baseline to follow-up or death. The participants underwent these comprehensive health examinations at one of the country's largest screening centers (the Mor Institute) during 1988. Examinees arriving at Mor during baseline, who were no longer gainfully employed (e.g., retirees or the unemployed), were not asked to participate in the study because many measures in the study's questionnaire—including the measure of vigor—were employment-contextualized. The second data set consisted of the electronic medical records of all participants made available to the study by CHS. We excluded from the study 41 participants because key sociodemographic variables (including age and gender) between the two data sets did not match. In this study, we sought evidence of the effects of vigor on morbidity and mortality, and not vice versa. Additionally, we sought to reduce the possibility that poorer initial health drives the relationships between vigor and the criteria under investigation. For these reasons, we also excluded from the study 33 respondents with either self-reported or were diagnosed by a Mor physician to have cancer ($n = 6$), diabetes ($n = 12$), or any category of cardiovascular disease ($n = 15$) as of 1988. After the above exclusions, the number of study participants was 968.

Uniform examination procedures and standardized measurement techniques were used at Mor throughout the baseline period. Employers who send their employees for periodic health examination at the Mor Institute include some of the country's largest firms in finance, insurance, public utilities, health care, and manufacturing. Other investigators, who have systematically compared the sociodemographic characteristics of samples that they had drawn from the Mor Institute's database with the general Jewish population of the country, concluded that the examinees were representative of the adult Jewish workforce (28).

At baseline, the respondents' mean age was 41.70 years (range, 18–73 yers). They reported working, on the average, 8.8 hours per day. About one third (33%) of the respondents were female. About 82% of the respondents were married; and of these, 80% had children (on the average, two children). The sample, when compared with the Jewish labor force (29), was biased in favor of employees with managerial jobs (20% versus 5.3%) and employees holding academic degrees (27% in the sample as compared with 16% in the labor force). The sample, thus, overrepresented managerial and highly educated employees, which is indicative of the fact that some employers offer periodic health examinations to their senior employees as a fringe benefit.

Measures

Study Outcomes

All data on mortality and morbidity were obtained from CHS automated databases that include data on chronic disease diagnoses, hospitalization discharge data, dispensed community prescriptions, and physicians' office visits, in addition to sociodemographic data (18). In Israel, mortality data were retrieved from the Israel National Population Registry, which maintains records for all citizens and permanent residents. Death records are routed routinely from the National Population Registry to CHS via the National Insurance Institute, usually within a month from the date of death. In the CHS, death records are linked with the personal medical files of the relevant enrollees via their identification numbers. However, as the cause of death could not be retrieved from death notifications, we refer only to all-cause mortality (30).

The diagnosis and date of diagnosis of IHD and diabetes were retrieved from the (fully digitized) electronic medical record of each participant in the CHS. All of CHS's primary care physicians use a single type of an electronic medical record connected to a centralized data warehouse, a fact that makes the CHS data set unique in its completeness. The diagnoses of the two end points of IHD and diabetes were based on the CHS chronic diseases register. Each diagnosis in the chronic diseases register was based on a special algorithm developed for this diagnosis by the CHS (18). The diagnosis of each chronic disease is essentially a two-stage process. In the first stage, an initial diagnosis is determined. In the case of the two chronic diseases under study, IHD and diabetes, it is based on the International Statistical Classification of Diseases and Related Health Problems (ICD) codes appearing in the discharge letters post hospitalization, for the diagnosis of IHD, and on reports of primary care physicians cross-validated against medication use files and serum analysis of HbA1c for the diagnosis of diabetes. Subsequently, during the second stage, a chronic disease diagnosis may be modified or changed by the relevant primary care physician based on new indications, such as echocardiographic reports, laboratory tests, and imaging studies. Diagnostic categories in the chronic diseases register were used in many past studies, including studies of diabetic patients in the CHS (31,32).

Study's Predictors and Control Variables

The respondent's score on each of the indices was obtained by computing the mean of his or her responses to the items in the index. Index reliability was gauged by the α coefficient (i.e., Cronbach's internal consistency reliability). In the following description of each index, we include examples of the items it contains. Mean and standard deviation values for the indices are presented in Table 1.

Vigor was assessed based on four items ($\alpha = 0.85$), which requested respondents to indicate how often they felt energetic, vigorous, alert, and active on their job, on a 7-point scale ranging from 1 being almost never to 7 being almost always. The above four items represent the subscale of vigor in the short form of the Profile of Mood States (33), used in many studies (24) and validated as a distinct factor (34). We subsequently expanded this four-item measure of vigor to include additional items representing each of vigor's major facets and several studies (4,6) reported on aspects of the expanded meaure's construct validity. We refer to studies using the subsequently expanded version of the vigor scale because we found that it correlated ($r = .90$) with the four-item version used in the current study. Age, sex, and educational level were gauged based on the computerized questionnaire as validated by the respondents' personal medical files. A well-established finding in the scientific literature is the inverse association between socioeconomic status (SES) and health: the higher the SES, the lower the prevalence and occurrence of health problems, illness, disease, and death (35). Following this evidence, we used as a control variable a proxy measure of SES, namely, educational attainment. Educational level ranged from 1 (primary education or less) to 5 (at least first academic degree or above).

From the computerized questionnaire, we also obtained the set of control variables referring to health behavior and negative affective states. The Smoking index was constructed based on the number of cigarettes smoked per day and ranged from 1 (not smoking) to 5 (smoking >1 pack—20 cigarettes—per day). The Exercise index was based on the self-reported number of weekly hours engaged in sport activities and ranged from 1 (<1 hour) to 5 (>5 hours). Consumption of alcoholic drinks was assessed by the frequency of drinking per week and ranged from 1 (none) to 2 (very infrequently or only on Fridays) to 5 (drinking on a daily basis). The body mass index (BMI) used in the study was measured by the respondent's weight (in kg) divided by his/her height (in m^2). Fasting blood samples were drawn from the respondents in the morning, on arrival at the Mor Institute. The levels of total

TABLE 1. Predictors of Mortality and Morbidity, Stratified by Tertiles of Vigor

Vigor Tertile Predictor	1 (Lowest) (n = 290)	2 (n = 306)	3 (Highest) (n = 320)	Total (n = 916)	p (F Test)
Age	41.76 (9.25)	41.43 (9.00)	41.11 (9.92)	41.42 (9.40)	.70
Gender (1 = men)	0.61 (0.45)	0.68 (0.47)	0.72 (0.45)	0.67 (0.47)	.01
Educational level	3.24 (1.29)	3.35 (1.27)	3.38 (1.25)	3.33 (1.27)	.38
Total cholesterol	205.35 (41.80)	202.40 (38.21)	204.32 (40.0)	204.00 (39.9)	.66
Glucose	92.38 (17.59)	92.37 (18.30)	94.18 (14.85)	93.00 (16.9)	.30
Body mass index	25.15 (3.85)	25.33 (3.90)	25.28 (3.37)	25.25 (3.70)	.83
Alcohol consumption	2.51 (1.08)	2.63 (1.16)	2.59 (1.10)	2.58 (1.11)	.44
Smoking Index	1.65 (1.26)	1.47 (1.11)	1.49 (1.13)	1.53 (1.17)	.12
Exercise Index	1.97 (1.21)	2.08 (1.28)	2.20 (1.37)	2.08 (1.29)	.09
Depressive symptoms	0.45 (0.86)	0.24 (0.73)	0.11 (0.39)	0.26 (0.77)	.001
Anxiety symptoms	0.64 (1.02)	0.40 (0.85)	0.31 (0.65)	0.45 (0.86)	.001
Chronic disease diagnosis (1 = yes)	0.22 (0.41)	0.18 (0.35)	0.13 (0.34)	0.18 (0.38)	.02
Past hospitalizations (1 = yes)	0.33 (0.47)	0.31 (0.46)	0.28 (0.45)	0.31 (0.46)	.43

Data represented as mean (standard deviation).
We stratified vigor according to the respondents' mean value in the total score. <5.00 = low tertile; 5.00–6.00 = medium tertile; and >6.00 = high tertile.

cholesterol and triglycerides were determined, using the Coulter "S" Counter, calibrated daily using the 4C Standard of Coulter Electronics. Fasting glucose was determined with the glucose oxidase method, using an autoanalyzer (Beckman Instruments, Fullerton, California). Arterial blood pressure (mm Hg) was measured twice in the left arm, while sitting, after a 1-hour rest. The average of two independent measures was used.

In the current study, we followed the recommendation provided to researchers by Ryff et al. (36) to control for the effects of depression and anxiety when examining the cumulative effects of affective experiences on physical health. Depressive and anxiety symptoms were assessed based on the items of the Cornell Medical Index (37). Depressive symptoms were gauged based on responses to seven dichotomous items, requesting respondents to report yes (scale value of 1) if most of the time they recently felt sad, depressed, pessimistic, lonely, unsuccessful in life, lacking confidence in the future, and unable to enjoy daily activities and no (scale value of 0) otherwise (α = 0.70). Anxiety symptoms were gauged based on the respondents' indication that most of the time they had recently felt tensed, anxious, angry, worried, and moody (1 = yes; 0 = no). For the anxiety symptoms scale, α = 0.90.

Procedure

The ethics committees of CHS and Tel Aviv University Faculty of Management approved the study. All respondents provided their informed consent. Respondents voluntarily completed the study's baseline (T1) questionnaire at the Mor Institute, as they were awaiting their turn for the clinical examination. They (95% of those initially eligible) were promised confidentiality of their individual data. At that time, they also completed a computerized questionnaire, which included sociodemographic characteristics. The second stage of the comprehensive health examination consisted of blood tests, a physical examination by a physician, electrocardiographic recording, and respiratory measurement of the lung function. Also assessed during this stage were the respondents' weight, height, blood pressure, pulse rate, routine urinalysis, and vision and hearing functions. Measuring these physiological parameters at the center is fully automated and the results are fed directly into the computerized data bank.

Statistical Analyses

There were 52 missing cases as a result of missing data on the predictors. We tested if not responding to the items assessing vigor predicted any of our criteria. We did not find any evidence that no-responding to the vigor items was a significant predictor of either of the endpoints. Also, we tested the possibility that the status of nonresponding to any other predictor interacted with vigor in predicting any of our three criteria, again failing to find any significant effect. In addition to the above tests of possible bias due to missing data, we used logistic regressions to test systematically if any of our predictors and additional ones predict the dichotomous criterion of not fully responding on the questionnaire (score of 1) versus fully responding (score of 0). Only educational level was found to be a significant predictor. We controlled for educational level in all our analyses.

For the outcome of mortality, we used a Cox proportional hazard regression to estimate hazard ratios (HRs) and their 95% confidence intervals. For those who died and did not have IHD or diabetes, the event of death impedes the possible incidence of IHD and diabetes and, therefore, represents a competing risk. Consequently, for estimating the prevalence of IHD and diabetes, we used competing risk proportional hazards models. We used the program STCRREG in Stata 11 which implements the proportional hazard model of Fine and Gray (38) for the subdistribution of a competing risk, also known as the cumulative incidence function. We modeled the hazard of the subdistribution as a function of the above covariates. The time scale used in the Cox regressions was days of follow-up, from the date of arrival at the Mor Institute for the periodic health examination in 1988 to the date of occurrence of the specific outcome (i.e., diagnosis of IHD or diabetes or death) or October 1, 2008—whichever occurred first. Average follow-up was approximately 20 years. We used STATA because it allowed us to test the proportional hazard assumption of the Cox regressions used: we found that it was met in all three regressions. Unless otherwise noted, all predictors were used as continuous variables. The full multivariate model included the following baseline values hierarchically entered: age; gender (dichotomy, 1 = men); educational level; vigor; total cholesterol; glucose; BMI; alcohol consumption; smoking; depressive symptoms; anxiety symptoms; and self-reported diagnosis of a chronic disease (dichotomy, 1 = yes) and past hospitalization (dichotomy, 1 = yes).

The effects of vigor on health-related outcomes could be age- and gender-specific (6) Higher frequencies of positive affects are often found in older than younger adults (39). Similarly, a synthesis of the literature on positive well-being (40) found that women were more likely than men to report positive affects, such as happiness. Consequently, we used age and gender as predictors and systematically tested the possibility that the expected predictions formulated above are moderated by either of them. Consequently, we ran additional Cox regressions to systematically test the possibility that gender or age moderates the prediction of each outcome by vigor and also by the physiological and behavioral risk factors. These tests were conducted by including the interactive terms of the above predictors (centered to reduce multicollinearity) with age (centered) and gender in the Cox regressions predicting each outcome. All interactive terms were entered in the very last step and only if significant (using the stepwise version of the Cox regression). All main effects were entered hierarchically, following the a priori order explained above. The only significant interactive term found is reported

below. Following a reviewer's suggestion, we tested the above interactions using age groups (terciles and quintiles of age), instead of age, as a continuous variable, but as before we did not find any significant interactive term. However, it may be unreasonable to postulate that risk suddenly increases as an age category is crossed, and the results of the above tests clearly depend on the number and choice of the age categories. Therefore, we supplemented the above analyses by the use of fractional polynomials to estimate the effects of age on the risk of each of our outcome variables (41), using the Stata Fracpoly program (42). This Stata program compares the deviations of different models, each expressing a specific polynomial of age (ranging from −2 to + 3) with the simple first-degree linear representation of age (43). For all three models reported in Table 2, we found that the difference between the best fitting polynomial model (e.g., for IHD, −2.0, for diabetes, +2.3) did not differ significantly from the simple linear model with age, thus further cross-validating the results we obtained using categorical representations of age with terciles and quintiles.

In separate analyses, we tested if the additional potential control variables of baseline systolic and diastolic blood pressure have any effect on our results; because these two additional controls were insignificant in all runs and did not have any appreciable influence on the set of findings reported below they were not included in our tables.

RESULTS

Table 1 shows the percentage of respondents according to approximate terciles of vigor for the study's predictors. Very few significant differences among the three subgroups of vigor emerge. Aside from the expected gender difference in vigor, Table 1 provides evidence that the higher the level of vigor, the lower the level of depressive and anxiety symptoms and the lower the percentage of respondents reporting at baseline that they were diagnosed in the past to have any chronic disease.

Table 2 presents the results of the tests of our major hypotheses. For each outcome, we report on the number of events and the number of missing values (Table 2, last row). In the competing risk Cox regression with IHD as the criterion, there were 45 respondents who passed away and did not have a diagnosis of IHD. In the competing risk Cox regression with diabetes as the criterion, there were 46 respondents who passed away and did not have a diagnosis of diabetes. As evident from Table 2, there was strong support for our expectations regarding the protective impact of baseline vigor on the risk of all-cause mortality and diabetes, independently of age, education, gender, and the physiological, behavioral, and psychological covariates controlled for in our analyses. We found that, after the above adjustments, baseline vigor decreased the risk of follow-up mortality by 26% and of the incidence of diabetes by 17%. However, we failed to find support for the protective effect of baseline vigor on the risk of IHD.

In Table 2, we report our findings obtained when all control variables were forced to enter as predictors, regardless of their significance. To test the possibility of overadjustment bias (44), we used Cox proportional hazard regressions in a stepwise manner, entering control variables only if they were significant. We obtained the same set of results reported in Table 2, with only minor increases in the HR of the significant

TABLE 2. Multivariate Cox Regression Model Predicting Mortality by the Job Demand-Control-Support Model, Covariates, and Control Variables

Predictor	Model 1. Mortality		Model 2. Ischemic Heart Disease		Model 3. Diabetes	
	HR	95% CI Lower–Upper	HR	95% CI Lower–Upper	HR	95% CI Lower–Upper
Sociodemographic predictors						
Age	1.08*	1.04–1.11	1.05*	1.03–1.07	1.02	0.99–1.03
Gender (0 = women; 1 = men)	1.32	0.68–2.57	2.14*	1.25–3.66	1.03	0.63–1.68
Educational level	0.92	0.75–1.11	0.87*	0.75–0.99	1.01	0.88–1.18
Vigor	0.74*	0.59–0.98	0.98	0.82–1.18	0.83*	0.67–0.98
Physiological risk factors						
Total cholesterol	1.01*	1.00–1.02	1.01*	1.00–1.02	1.00	0.99–1.01
Glucose	1.02*	1.01–1.03	1.01*	1.00–1.02	1.03*	1.02–1.04
Body mass index	1.02	0.94–1.11	1.03	0.98–1.07	1.16*	1.10–1.21
Behavioral risk factors						
Alcohol consumption	1.27*	1.03–1.57	1.05	0.89–1.25	1.02	0.85–1.22
Smoking Index	0.94	0.73–1.21	1.10	0.96–1.26	1.14*	1.01–1.29
Exercise Index	0.77*	0.63–0.95	0.99	0.85–1.16	0.75*	0.63–0.90
Depressive symptoms	0.99	0.77–1.28	0.96	0.78–1.18	0.89	0.68–1.18
Anxiety symptoms	0.79	0.58–1.07	1.11	0.88–1.39	1.00	0.78–1.26
Self-reported medical history						
Chronic disease diagnosis (1 = yes)	1.08	0.63–1.87	1.42	0.90–2.18	1.99*	1.31–3.08
Past hospitalization (1 = yes)	1.57	0.87–2.86	1.46	0.93–2.32	1.05	0.62–1.78
Model N, Wald χ^2	n = 916; χ^2 = 155 (p<.001; df = 14)		N = 916; χ^2 = 140 (p<.001; df = 14)		n = 916; χ^2 = 283 (p<.001; df = 14)	
Number of events, missing values	Events = 59; MV = 52		Events = 126; MV = 52		Events = 109; n = 870; MV = 52	

* $p < .05$.
HR = hazard ratio; CI = confidence interval; MV = missing values.

predictors. We also tested vigor as the only predictor of our three health outcomes. The only change relative to the results reported in Table 2 was that vigor, when used as the only predictor, was an only marginally significant predictor of all-cause mortality (HR, 0.81; $p = .06$; 95% confidence interval, 0.64–1.01). Therefore, we entered each predictor in a separate step sequentially to find the predictor which, when controlled for in the Cox regression analysis, led to vigor becoming a significant predictor. We found that it was anxiety symptoms; when adjusted for in our analyses, vigor became a significant predictor of all-cause mortality as reported in Table 2. We also tested the possibility that the effect of vigor in predicting the risk of all-cause mortality is attenuated by the diagnosed postbaseline IHD and diabetes, modeling the two additional controls as dummy variables. The results reported in Table 2 for all-cause mortality did not change: Vigor remained a significant predictor of mortality. However, the two additional control variables clearly violated the assumption of proportional hazard underlying our use of Cox regressions; therefore, we did not include them in Table 2.

As explained above, we systematically tested the possibility that vigor and also the physiological and behavioral risk factors interact with gender and age in the prediction of the study's end points. All except one of the interactive terms tested turned out not to be significant. The only exception is the interaction of baseline glucose levels and gender in the prediction of the risk of diabetes at follow-up. We plotted this interaction and found that higher (above mean) levels of baseline glucose were significantly more powerful in predicting the risk of diabetes for women than for men (this plot is available from the first author on request). We did not include this interaction in Table 2 because it was found in an explorative analysis and because it was unrelated to the study's objectives.

DISCUSSION

We investigated whether vigor, representing moderate intensity positive affect, influenced the risk of mortality and the prevalence of IHD and diabetes in a cohort of initially healthy employees followed up for about 20 years. Past qualitative (9) and quantitative (10) reviews established these linkages for positive affects in general, but vigor has hardly been investigated as a predictor of the above outcomes. In testing our major hypotheses, we tested vigor after including as predictors age, gender, and educational level. We controlled for the possible confounding effects of several physiological and behavioral risk factors, including smoking, drinking alcoholic beverages, and exercise behavior. We also controlled for depressive and anxiety symptoms, representing negative affective states whose predictive powers relative to the major health outcomes that we used as criteria have been well documented.

We found considerable support for our major hypotheses in that baseline vigor decreased the risk of dying and predicted lower diabetes risk over a 20-year period. We consider it significant that the predictive effects of vigor on mortality and on the prevalence of diabetes were maintained after we controlled for the potential detrimental influence of depressive and anxiety symptoms on these health outcomes. However, in contrast to our expectation, vigor was not found to exert a significant protective effect for IHD risk. Our finding that baseline vigor predicted higher probability of surviving is supported by previous studies that found vitality—a construct with a strong component of vigor and energy—to have an analogous effect (9). As indicated above, positive affect or vigor has hardly been investigated as predictors of diabetes incidence. We argue that our findings contribute to the ongoing debate about whether positive affect and negative affect represent different ends of a single dimension (45) or whether they are independent of each other (46). Our study provides evidence that positive affect and negative affect tend to function relatively independently in predicting important health outcomes, such as mortality and the risk of diabetes.

Study's Limitations

Before discussing our results, it is appropriate to note some of the limitations of the study. First, the sample overrepresents employees with higher than average SES. SES is known to be a strong predictor of each of our criteria (35) and was only partly controlled in our analysis by using educational level. However, we argue that this limitation acted against our hypotheses, and that the major effects found would probably be stronger had we investigated a sample of persons more representative of the lower SES strata. As argued by other researchers, it is plausible that positive affects buffer the effects of work and life stresses more powerfully for those who lack coping resources, such as high SES (47,48). Second, we do not have any data on the extent to which employees who were eligible for the periodic health examination declined their employer's offer to undergo such a check-up. It is well known that preventive health behaviors, like going for a health check-up, vary among different categories of employees (49). Third, all our predictors were based on baseline measures.

Fourth, an important additional reservation concerns the coarseness of our measures of depressive and anxiety symptoms that were found to be insignificant predictors in our analyses. Both probably did not represent all important symptoms of depression and anxiety (50) and were based on aggregating dichotomous items, thereby resulting in skewed distributions. In the same vein, the fact that we obtained only sporadic significant results for the behavioral risk factors of smoking, alcohol intake, and physical activity probably indicates that we did not cover important dimensions of these health behaviors in our measures. For example, smoking did not cover the total number of years of exposure to smoking, alcohol intake was assessed by frequency of drinking and not by the amount of alcohol habitually consumed, and our measure of physical exercise did not cover the number of hours of strenuous physical activity during and after work. Therefore, it is very likely that there is residual confounding regarding these control variables.

Fifth, using ICD codes for diagnosing IHD is known to be problematic because of biased hospital coding, incomplete

hospital coding, and errors caused by redundant codes or by hospital idiosyncrasies (51). As we noted, CHS' physicians have the ability and authority to modify indications of chronic diseases in CHS' chronic disease registry based on evidence they received. Still, the inaccuracies associated with the fact that the initial diagnosis of IHD was largely based on ICD codes appearing in hospitals' discharge letters could be a possible bias in the diagnosis of IHD in our study. Sixth, although vigor was found (6) to be relatively stable across periods of 2 or 3 years, with stability coefficients of 0.64, we cannot claim that it remained stable across the 20 years of our follow-up. Finally, prospective epidemiological studies cannot confirm causality, because—as suggested above—there may be residual confounders. Also, there is the possibility of unmeasured third factors influencing both vigor and risk of mortality or diabetes, such as upbringing or personality factors.

IHD

What could possibly explain our failure to support the expectation regarding the effect of vigor on IHD? A large-scale prospective study—part of Whitehall II Study (52)—used a five-item measure of positive affect in general to predict the risk of coronary heart disease among participants followed up over 12 years and failed to find support for the expected effect. Our study reached the same result, using a specific positive affect, vigor. It could be that vigor's effects on IHD are not direct but occur by buffering the system from the negative effects of exposure to stressors, as suggested in a recent review of the area (53). It could also be that vigor's influence on IHD is fully mediated by coping resources that it helps to create. We could not test these hypotheses in the current study because we did not assess at baseline our respondents' exposure to work-related stresses or their coping resources.

Potential Mechanisms Linking Vigor with Health End Points

Based on past research, we suggested above that the mechanisms linking vigor with the study's health end points may include its beneficial effects on inflammation biomarkers and self-rated health: However, these variables were not assessed in the current study. A possible clue to one of the mechanisms that may explain the effects of vigor on the risk of mortality was described above: Adjusting the main effects of vigor on risk of mortality for anxiety symptoms increased the significance of vigor as a predictor. However, we need to be cautious in interpreting our finding that the effect of vigor on mortality became significant only after controlling anxiety symptoms because this effect was marginally significant ($p = .06$) before we controlled for the two negative affects of depressive and anxiety symptoms.

Yet another possible mechanism that could link vigor with mortality concerns exercise behavior. In our study, the measure of exercise behavior was found to be positively associated with vigor. Several meta-analytic studies have found exercise behavior and vigor to be closely associated (24,54,55). For example, it has been found that a major reason people engage in physical activity is to experience vigor (56,57). Therefore, it could be that there is a reciprocal relationship between vigor and exercise behavior which unfolds over time and has the potential to explain the effects of vigor on mortality.

Regarding the effects of vigor on the risk of diabetes, Table 2 provides some clues as to the possible mechanisms. From Table 2, it is evident that body weight (as assessed by the BMI) increased and exercise behavior decreased the risk of diabetes. As we indicated above, vigor and exercise behavior were found in meta-analytic studies to be closely interrelated (25). Therefore, it is possible that the effects of vigor on diabetes risk operate, in part, through exercise behavior. Interestingly, the negative affective counterpart of vigor, burnout, was found in a prospective study to predict the onset of diabetes (58); future research may investigate how burnout and vigor interrelate in influencing the prevalence of diabetes. An additional area of study is the possibility that vigor operates as a stress buffer (9), influencing intensity of and recovery from chronic stressors at work.

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