Symptom Reports and Mortality in an Occupational Cohort

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The relationship between baseline symptom reports and subsequent mortality over a 24-year period was examined in a group of 1,224 white male nonsupervisory paper workers. Symptom reports were measured via the Cornell Medical Index, and the vital status of each participant was ascertained by reviewing company personnel records and death certificates. Analyses based on proportional hazards models suggest that symptom reports are predictive of mortality (RR = 1.24; P = .0002), independent of the participant's age and biologic risk status at intake. Analyses based upon age-specific and age-standardized mortality ratios confirm that the paper workers were subject to a "healthy worker effect" (standardized mortality ratio [SMR] = 0.66), and that the healthy worker effect is attenuated (SMR = 0.77) among those participants reporting ten or more symptoms at the beginning of the follow-up period.

In the general population, the proportion of symptoms brought to medical attention is low[1-3] and can be influenced by social, cultural, economic, and psychological factors.[4-8] In working populations, similar phenomena may be operating, but a variety of factors associated with employment in general may modify the number or type of symptoms reported. Both selective hiring and selective withdrawal from industry, which have been linked with the healthy worker effect,[9-13] may be responsible for a reduction in morbidity and symptom reports among workers. Alternatively, increased concern about the health effects of occupational exposures may increase the number of symptoms brought to medical attention. The present investigation was motivated by our interest in the meaning of self-reported symptoms in a working population. More specifically, we were interested in the empirical relationship between self-reported symptoms and subsequent all-cause mortality in an occupational cohort.

In 1972, Daly and Tyroler[14] reported that an association between Cornell Medical Index (CMI) responses and all-cause mortality generally persisted when four biologic risk factors were controlled one at a time. However, the authors did not consider the statistical variation in the data, or model the relationship between CMI scores and mortality while controlling for multiple biologic risk factors simultaneously.[15-16]

The present investigation includes a reanalysis of Daly and Tyroler's data based up 11 years of follow-up and 118 deaths[14] and a new analysis of the original cohort based on 24 years of follow-up and 383 deaths. Following Daly and Tyroler, it was hypothesized that there would be a positive association between the number of symptoms reported at intake and subsequent all-cause mortality. Moreover, assuming that there is a negative association between the number of symptoms reported and a worker's overall health status,[18] it was expected that the healthy worker effect would be smaller among workers who report more symptoms at intake.

Methods

The cohort of 1224 nonsupervisory white male pulp and paper workers in this investigation represents 93% of the age eligible (attained age > 35 years) workers employed at the study plant during the 3-month examination period ending in September 1959.[14, 17] Participants' blood pressure, height, weight were recorded, and responses to 195 symptom/behavioral items consti-

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0096-1736/86/2809-849$02.00/0

tuting the Cornell Medical Index[18] were obtained under standard conditions. Serum cholesterol values were based on the method of Abell et al.[19] The CMI scores used in the present (and the previous) investigation are formed by adding the number of positive responses, ignoring item content. Thus, the possible range of CMI scores is 0 to 195.

We attempted to ascertain the vital status of each cohort member as of Oct 1, 1983 by contacting the company personnel office and the offices of vital statistics in eight states. When a personnel record indicated that a death had occurred, we attempted to confirm that fact and date of death by obtaining a death certificate. Persons whose vital status as of October 1983 could not be ascertained were considered lost to follow-up (and alive) as of last date of "contact" or follow-up.

Descriptive and analytical statistics for this report were obtained from the Statistical Analysis System.[20] The relationship between CMI scores and mortality was characterized by fitting discrete proportional hazards models[21] to the data. The fit between the data and the final model was evaluated by comparing the observed number of deaths with that predicted from the model by 24-year risk category. The healthy worker effect in the cohort was estimated by comparing the mortality experience of cohort with the pertinent mortality experience of the general US population.

Results

Table 1 describes the vital status of the study cohort in 1983 according to company personnel records. Average baseline (1959) characteristics of the study cohort are presented in Table 2. Comparisons between the data summarized in Table 2 and data from Evans County,[22] Framingham,[23] or a national probability sample[24-25] indicate that the paper workers' blood pressure, serum cholesterol, and obesity levels at baseline were typical for their age and calendar period. The average observed CMI total score (mean = 12.9) is similar to the average (mean = 11.6) for ostensibly healthy men (N = 345; mean age = 50.1) reported by Costa and McCrae.[26]

Fig. 1 presents the distribution of CMI total scores at baseline. Inspection of the distribution reveals the positive skew, and that 90% of the participants had CMI scores of less than 27 or that the prevalence of scores of 30 or greater was less than 10%. Abramson[18] has suggested that among ostensibly healthy persons, the prevalence of total scores of 30 or greater varies from 0% to 12%. Higher prevalences of total scores of 30 or greater are expected[18] among nonpsychiatric patients (12% to 26%) and emotionally disturbed patients (40% to 79%).

Table 3 shows baseline characteristics of the participants by their vital status in 1983. Inspection of Table 3 reveals the consistency of the data. In particular, comparisons across working individuals, retired/disabled individuals, and decedents reveals gradients in age and each of the remaining characteristics. The difference between median CMI total scores for decedents *v* survivors (13.0 *v* 9.0) can be viewed as a crude test of the hypothesized association between CMI total scores and subsequent mortality.

TABLE 1
Vital Status of Study Cohort in 1983

Status*	N	%
Alive	803	65.6
(Working)	(139)	(11.4)
(Retired/disabled)	(643)	(52.5)
(Employment status unknown)	(21)	(1.7)
Dead†	383	31.3
Withdrawn (alive)‡	36	2.9
Lost§	2	0.2
Total	**1,224**	**100.0**

* According to company personnel records.
† Date of death known; death certificates were obtained for 376 decedents.
‡ Date withdrawn alive known.
§ Date withdrawn equals last day of follow-up.

TABLE 2
Characteristics of Study Cohort* at Baseline (1959)

Characteristic	Mean (Median)	SD	Range
Age in 1959 (AGE)	46.7 (45.5)	8.0	35–68
Systolic pressure (SBP)	136.0 (130)	19.3	100–260
Diastolic pressure (DBP)	82.5 (80)	11.6	54–130
Quetelet's Index (QUET)†	3.6 (3.5)	0.5	2–6
Serum cholesterol level (CHOL)	235.1 (232)	43.5	126–460
Symptom reports (Cornell Medical Index [CMI])	12.9 (10)	11.4	0–76

* N = 1,224.
† QUET = weight/(height)^2 (100); lbs/in^2.

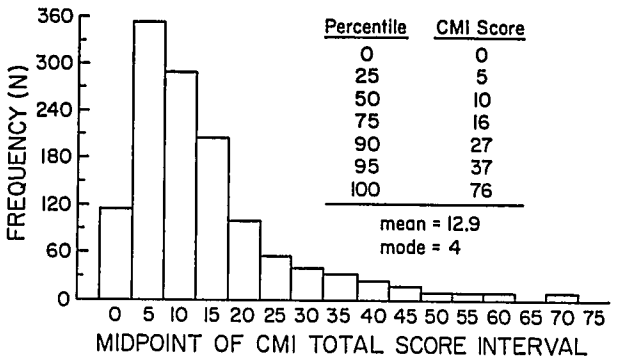

Fig. 1. Distribution of symptom/behavioral responses (Cornell Medical Index scores) at baseline, N = 1224.

TABLE 3
Baseline Characteristics of Cohort by Vital Status (1983)

	Alive	Working	Retired/ Disabled	Dead	Withdrawn
AGE*	44.7	37.1	46.5	51.5	41.3
SBP	133.0	131.3	133.5	142.6	132.7
DBP	80.8	79.4	81.1	86.2	82.9
QUET	3.5	3.5	3.5	3.6	3.5
CHOL	232.6	227.2	233.2	240.9	227.6
CMI†	9.0	8.0	9.0	13.0	10.5
N	803	139	643	383	38

* Abbreviations are explained in Table 2.
† Median level reported because of skewed distribution.

Two discrete proportional hazards models[21] linking baseline CMI total scores with subsequent mortality during 11 years of follow-up[14] are summarized in Table 4. In the first model AGE and CMI scores were the only independent variables, whereas in the second model systolic blood pressure (SBP), serum cholesterol level (CHOL), and Quetelet's Index (QUET) were included along with AGE and CMI. The two models based upon 11 years of follow-up yield very similar estimates of the mortality increase associated with given increase in CMI total score. Specifically, the model based upon AGE and CMI scores only yields a mortality rate ratio estimate of 1.21 ($P = .0643$) when a CMI total score differential of 15 positive responses is assumed. (A report of 15 additional symptoms would shift a worker from the first quartile, to the fourth quartile of the empirical CMI total score distribution.) The more complex (five independent variables) model yields a mortality rate ratio estimate of 1.23 ($P = .0446$) when a 15-symptom differential is assumed. The similarity between these two hazard ratio estimates (or the corresponding model parameters) suggests that the simpler two-variable model is an adequate description of the association between CMI total scores and subsequent mortality in the paper workers cohort.

Table 5 displays 2 variable (AGE and CMI) and five variable (AGE, CMI, SBP, CHOL, QUET) regression models based upon 24 years of follow-up. The two- and five-variable 24-year models yield mortality rate ratio estimates of 1.23 and 1.24 ($P = .0002$) when a 15-symptom differential is assumed. Thus, the accumulation of additional deaths and person-years subsequent to the 11-year follow-up did not appreciably change the estimate of the strength of the CMI total score-mortality association, but it did reduce the SE of the estimate by over 40%. In addition, the analysis of the data from 24 years of follow-up confirms the previous observation that the association between CMI total scores and all-cause mortality in the study cohort is largely independent of systolic blood pressure, serum cholesterol, and Quetelet's Index levels at baseline. (Note that the cholesterol coefficient also increases nearly threefold and approaches a nominal level of statistical significance with 24 years of follow-up).

Because the analyses summarized in Tables 4 and 5 are based upon discrete proportional hazards models, there was an interest in the overall fit between the data and the family of models employed. Table 6 presents observed and expected deaths by category of predicted 24-year mortality risk. The calculation of expected deaths and the assignment of observed survivors and decedents to a risk category was based upon a discrete proportional hazards model of follow-up time, baseline age, and baseline CMI total score (see Table 5). The methodology is very similar to that used previously to assess the fit between data and the logistic model[16] in prospective studies of ischemic heart disease.[27-28] Inspection of the distributions of observed and expected deaths in Table 6 suggests that the model employed is reasonable from the perspective of approximating the observed data. The fit of the model could be improved if

TABLE 4
Regression Models* Linking Baseline Symptom Reports (Cornell Medical Index) With Subsequent Mortality During 11 years of Follow-up†

Independent Variable	Parameter Estimate	SE	χ^2	P Value‡
	(−2 LOG LIK for no covariates = 1,666.3)			
AGE§	0.09558	0.01171	66.6	.0000
CMI	0.01244	0.00673	3.4	.0643
	(−2 LOG LIK for two-variable model = 1,584.9)			
AGE	0.07956	0.01224	42.2	.0000
CMI	0.01370	0.00682	4.0	.0446
SBP	0.01862	0.00388	23.0	.0000
CHOL	0.00077	0.00206	0.1	.7089
QUET	−0.13735	0.19375	0.5	.4784
	(−2 LOG LIK for five-variable model = 1,563.1)			

* Discrete proportional hazards model of time in follow-up.
† 118 deaths, 1,224 observations.
‡ H_0: $\beta = 0$.
§ Abbreviations are explained in Table 2.

TABLE 5
Regression Models* Linking Baseline Symptom Reports (Cornell Medical Index Scores) With Subsequent Mortality During 24 Years of Follow-up†

Independent Variable	Parameter Estimate	SE	χ^2	P Value‡
	(−2 LOG LIK for no covariates = 5,284.2)			
AGE§	0.08497	0.00649	171.5	.0000
CMI	0.01394	0.00381	13.4	.0002
	(−2 LOG LIK for two-variable model = 5,074.7)			
AGE	0.07654	0.00673	129.3	.0000
CMI	0.01420	0.00381	13.9	.0002
SBP	0.01163	0.00236	24.2	.0000
CHOL	0.00205	0.00117	3.1	.0789
QUET	0.10716	0.11451	0.9	.3494
	(−2 LOG LIK for five-variable model = 5,042.8)			

* Discrete proportional hazards model of time in follow-up.
† 383 deaths, 1,224 observations (see Table 1).
‡ H_0: $\beta = 0$.
§ Abbreviations are explained in Table 2.

TABLE 6
Observed and Expected Number of Deaths by Predicted 24-Year Risk Category

Risk Cateogry*	No. At Risk	Observed Deaths (O)	Expected Deaths (E)	$\frac{(O-E)^2}{E}$
0.00–0.09	0	—	—	—
0.10–0.19	416	60	62.2	0.08
0.20–0.29	280	57	68.9	2.06
0.30–0.39	162	54	55.9	0.06
0.40–0.49	121	49	53.9	0.45
0.50–0.59	84	54	46.5	1.21
0.60–0.69	83	49	53.8	0.43
0.70–0.79	57	41	42.4	0.05
0.80–0.89	21	19	17.7	0.10
0.90–0.99	0	—	—	—
Total	**1,224**	**383**	**401.3**	**4.44**

* Based upon a discrete proportional hazards model of follow-up time, and Cornell Medical Index scores (see Table 5).
$\chi^2 = 4.44$ with (8-3-1) degrees of freedom; $.25 < P < .50$; H_0: model fits the data.

the variable SBP were included, but the identification of the "best fitting" model was not our objective.

Fig. 2 displays a plot of 24-year mortality risks *v* baseline CMI total scores by baseline age. The plotted points are based upon the hazards model of follow-up time, age, and CMI scores. A detailed examination of the predicted risk values summarized in Figure 2 reveals the monotonic (but nonlinear) effects of (age and) CMI scores upon risk. Among workers 40 years of age, successive 15-point CMI score increments (5 to 20, 20 to 35, 35 to 50, 50 to 65) yield risk ratios of 1.210, 1.204, 1.198, and 1.191, whereas among workers 60 years of age identical CMI score increments yield risk ratios of 1.128, 1.110, 1.091, and 1.092. All of these model-based risk ratio estimates are less than the overall model-based rate ratio estimate of 1.23 mentioned above. Thus, the plot of mortality risk *v* CMI scores by age underestimates the effect of CMI total scores upon the force of mortality (mortality rates), and illustrates the phenomenon that risk ratios approach unity when the event of interest (death) becomes less rare.[29-30]

Table 7 describes the mortality experience of the paper workers by CMI total score dichotomy (high scores were defined as scores $\geq$ 10) and 5-year age (age-at-death) groups, and compares the mortality experience in the cohort (O) with the mortality experience of white males in the US general population (E). Inspection of Table 7 reveals that within each age group the observed mortality rate (per 10^{-3} person-years) among paper workers is higher, and the healthy worker effect (O/E) is lower, in the half of the cohort with high CMI scores.

Discussion

Our principal research objective was to examine the relationship between self-reported symptoms and subsequent all-cause mortality in a working population. Our results are consistent with the hypothesis that there is a positive association between the number of reported symptoms at intake and subsequent mortality over a period of two decades, independent of the worker's age and biologic risk status at intake.

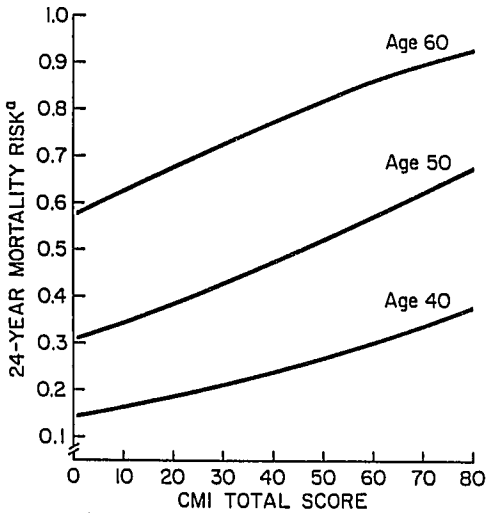

Fig. 2. Twenty-four year mortality risk *v* symptom/behavioral reports (Cornell Medical Index [CMI] scores) by baseline age. Risk was estimated from a discrete proportional hazards model of follow-up time, age, and CMI score.

In their earlier report Daly and Tyroler[14] state that the relative (mortality) risk of elevated CMI scores (highest tertile *v* lowest two tertiles) was 1.41 for the entire cohort and ranged from 1.05 to 1.72 for subgroups with different numbers of biologic risk factors present (residing in the upper tertile of blood pressure, cholesterol, or Quetelet Index values). In this investigation we used proportional hazards models to obtain an estimate of the mortality rate ratio (1.24) associated with a report of 15 additional symptoms while adjusting for the potential confounding effects of systolic blood pressure, serum cholesterol level, and Quetelet's Index. Our analyses suggest that these three extraneous variables produced very little confounding bias. Thus, the difference between our rate ratio estimate and the estimate reported by Daly and Tyroler is largely attributable to different analytic procedures. Daly and Tyroler compared the mortality experience of paper workers in the highest tertile of the CMI score distribution with the mortality experience of workers in the lower two tertiles of the CMI score distribution. The proportional hazards model estimates the impact of each additional symptom report upon the mortality rate. If CMI scores approximate an interval score then the use of each worker's actual score instead of a CMI score dichotomy (high/low) should yield a better estimate of the effect of CMI scores upon mortality.

The similarity of the mortality rate ratio estimates from the 11-year (1.21) an 24-year follow-up (1.23) models of AGE and CMI scores is intriguing given the expectation that CMI scores might lose some of their ability to predict death when the follow-up period is doubled. The uniformity of the CMI score-mortality relationship during follow-up time is consistent with the proposition that self-reported symptoms represent truly important long-term prognostic information. Alternatively, it is possible that CMI scores are only predictive of early death from certain causes, and that subsequent deaths from other causes were evenly distributed with respect to CMI scores. Cause-specific analyses of these data could address this issue.

A possible source of selection bias[30] in our study is the 38 persons who were withdrawn alive during, or at the end of the follow-up period (see Table 3). These 38 workers had a mean age of 41 years at baseline, median CMI total score of 10.5 (at baseline), and experienced an unknown number of events (deaths) prior to the end of follow-up. However, it is likely that the 38 workers withdrawn alive did not seriously bias our estimate of the association between CMI scores and subsequent mortality away from the null value ($RR = 1.0$) because their CMI scores are lower than the scores of the decedents.

Information bias[30] could have been introduced through measurement errors with respect to the various

TABLE 7
Mortality* by Symptom Report (Cornell Medical Index [CMI] Score) Dichotomy and Age

Age at Death	High CMI Scores†			Low CMI Scores‡			All CMI Scores		
	Observed O§	Expected (E)‖	(O/E)	O	(E)	(O/E)	O	(E)	(O/E)
40–44	3.2	4.1	(.78)	1.7	4.1	(.41)	2.3	4.1	(.56)
45–49	6.0	6.8	(.88)	2.7	6.8	(.40)	4.2	6.8	(.62)
50–54	8.5	10.9	(.78)	4.9	10.8	(.45)	6.6	10.8	(.61)
55–59	14.2	16.8	(.85)	7.5	16.5	(.45)	10.8	16.6	(.65)
60–64	21.2	25.7	(.82)	15.7	25.4	(.62)	18.6	25.6	(.73)
65–69	32.7	38.0	(.86)	22.7	37.4	(.61)	28.3	37.7	(.75)
70–74	26.2	56.0	(.47)	20.6	55.7	(.37)	23.9	55.9	(.43)
75–79	69.3	83.0	(.83)	50.5	83.2	(.61)	62.3	83.1	(.75)
80–84	92.8	118.4	(.78)	67.0	118.4	(.57)	82.6	118.5	(.70)
85–89	252.9	182.8	(1.38)	97.8	182.2	(.54)	165.4	182.5	(.91)
Adjusted	17.9¶	23.2	(.77)	12.1¶	23.2	(.52)	15.2¶	32.2¶	(.66)

* Rates per 10^{-3} person years; estimates obtained from Monson's program.
† High CMI scores ≥ 10.
‡ Low CMI scores < 10.
§ Observed (in cohort) age-specific rates are not adjusted for date of death.
‖ US white male age-specific rates adjusted for date of death.
¶ Adjusted for age and date of death by weighting the matrix of age- and period-specific rates by the person-year distribution in the entire cohort.

baseline characteristics or with respect to the fact or date of death. However, standardized data collection procedures were employed at the initial examination, and these procedures yielded very consistent data (see Table 3). Moreover, death certificates were obtained for 376 (98%) decedents thus confirming the fact and date of death. Therefore, it is also likely that information bias did not seriously compromise our results.

Despite the fact that our analyses indicate that the association between CMI total scores and subsequent mortality is indepedent of age, systolic blood pressure, serum cholesterol, and body mass there is the theoretical possibility of confounding bias[30] introduced by extraneous factors which were not measured and thus not modeled. However, the relationship between CMI scores and mortality in the paper workers appears to be remarkably robust. We added a smoking status variable (yes/no) to the 24-year five variable proportional hazards model and obtained the same estimate of the CMI score effect upon mortality. The issue of possible confounding bias presupposes that there is a hypothesized causal relationship between self-reported symptoms and mortality, and the issue of causality hinges upon the nature of the phenomena measured by the CMI.

The Cornell Medical Index was originally developed as an adjunct to medical interviews used to identify unfit army inductees.[31–32] Inasmuch as CMI items are organized by content area [eight anatomic sections (A-H), fatigability (I), frequency of illness (J), miscellaneous disease (K), habits (L), six mood/feeling sections (M-R)], it was assumed that a clinician would conduct a more efficient or accurate interview and examination if he first reviewed patient CMI questionnaire responses prior to initiating the clinical interview and examination. Abramson[18] suggests that the CMI is a poor predictor of specific morbidities, a fair predictor of overall (physical and emotional) health, and a good predictor of emotional health. Studies subsequent to Abramson's review[18] have exploited the somatic[33] and the psychological[26] interpretations of CMI item responses.

In 1980, Weaver et al[34] examined the relationship between CMI scores and prevalent disease, incident disease, and mortality in a cohort of 1820 high socioeconomic status Taiwanese males. The highest mean CMI scores were among those workers (N = 20) with prevalent disease at entry who died during the subsequent 2-year period, followed closely by survivors with prevalent disease at entry (N = 604). The next lowest CMI scores were among workers with incident disease (N = 459). The lowest CMI scores were among survivors who remained disease free (N = 507) during the follow-up period. Weaver et al[34] concluded that the CMI is a predictor of both prevalent and incident disease, and measures the respondent's general health status.

Our results are consistent with previous investigations[18] which suggest that the CMI measures a respondent's general health status by ascertaining the number of perceived symptoms. In theory, these perceived symptoms may be indicative of prevalent clinical or subclinical disease or a general susceptibility to disease. In fact, some of the CMI items also inquire about a history of specific disease (items 124 to 138). Positive responses to these history items may truly indicate prevalent disease, or affirmative responses may be false positives from the perspective of a thorough physical examination and may tap a more general willingness to report health problems or a general susceptibility to disease. Subsequent analyses of our data will address these issues.

The participants in this study were long-term employees at the time of the baseline examination; all participants were assured that their CMI responses would not be placed in their personnel files. Thus, it is likely that the CMI scores analyzed in this report were not biased by repeated attempts to respond in a socially desirable manner. Respondents may minimize their current symptoms if the CMI, or another symptom checklist, is administered during a preemployment examination. Further research is needed to address the relationship between self-reported symptoms and mortality in other

occupational group and in less selected community samples.

Given current interest in "health risk" or "health hazard" appraisal there is a need for a better understanding of the relationship between self-reported symptoms and long-term risk of disease and death. Data sets like ours could be used to identify individual questionnaire items that are predictive of mortality. However, the list of candidate items should be tailored to the population and scientific issues under consideration.

Acknowledgments

This investigation was supported by research grant 1 RO3 OHO1724-01 from the Centers for Disease Control, National Institute for Occupational Safety and Health.

The assistance of persons affiliated with the company personnel department, and offices of vital statistics in several states is gratefully acknowledged. The authors are indebted to Phyllis H. Johnson for her administrative, word processing, and diplomatic talents.

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