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Personalizing Mortality Prediction With Psychosocial Questionnaire Data

Benjamin P. Chapman, PhD, MPH[1], **Alexander Weiss, PhD**[2], **Kevin Fiscella, MD, MPH**[3], **Peter Muennig, MD, MPH**[4], **Ichiro Kawachi, MD, PhD**[5], and **Paul Duberstein, PhD**[1]

[1]Department of Psychiatry, University of Rochester Medical Center

[2]Department of Psychology, University of Edinburgh

[3]Departments of Family Medicine and Community and Preventive Medicine, University of Rochester Medical Center

[4]Department of Health Policy and Management, Mailman School of Public Health, Columbia University

[5]Harvard School of Public Health, Division of Social Epidemiology

Abstract

Background—Predicting risk of premature death is one of the most basic tasks in medicine and public health, but has proven difficult over the long term even with the best prognostic models. One popular strategy has been to improve prognostic models with candidate genes and other novel biomarkers. However, the gains in predictive power have been modest and the costs have been high, leading to a demand for cost-effective alternatives. We conducted a proof-of-principle investigation to examine whether simple, cheap, and non-invasive paper-and-pencil measures of social class and personality phenotype could improve the performance of one of the most widely used prediction models for all-cause mortality, the Charlson Comorbidity Index (CCI).

Methods—We used data from baseline and 25-year mortality follow-up of the UK Health and Lifestyle Study cohort. In a subset of the cohort, we first identified five psychosocial factors highly predictive of mortality: income, education, Type A personality, communalism (preference for the company of others), and "lie" scale (a measure of denial, putatively associated with ill-

Corresponding Author: Benjamin P. Chapman, PhD, MPH, Department of Psychiatry, University of Rochester Medical Center, 300 Crittenden, Rochester, NY 14624, Phone: 585-275-9431, Fax: 585-273-1082, Ben_chapman@urmc.rochester.edu.
Benjamin P. Chapman, PhD, MPH, Department of Psychiatry, University of Rochester Medical Center, 300 Crittenden, Rochester, NY 14624, Phone: 585-275-9431, Fax: 585-273-1082, Ben_chapman@urmc.rochester.edu
Alexander Weiss, PhD, Senior Lecturer, Department of Psychology, School of Philosophy, Psychology and Language Sciences, The University of Edinburgh, Dugald Stewart Building, 3 Charles Street, Edinburgh EH8 9AD, Phone: +44 (0)131 651 3083, Fax: +44 (0)131 651 3190, alex.weiss@ed.ac.uk
Kevin Fiscella, MD, MPH, Professor, Departments of Family Medicine and Community and Preventive Medicine, University of Rochester Medical Center, Family Medicine Research Programs, 1381 South Avenue, Rochester, NY 14620, Phone: (585) 436-3040, Fax: (585) 473-2245, kevin_fiscella@urmc.rochester.edu
Peter Muennig, MD, MPH, Associate Professor, Department of Health Policy and Management, Mailman School of Public Health, Columbia University, 600 W 168th St., 6th Floor, New York, NY 10032, Phone: 212-342-3170, Fax: 212-305-3405, pm124(@)columbia.edu
Ichiro Kawachi, MD, PhD, Professor, Harvard School of Public Health, Division of Social Epidemiology, 677 Huntington Avenue, Kresge Building 7th Floor, Boston, Massachusetts 02115, Phone: 617-432-3915, ikawachi@hsph.harvard.edu
Paul Duberstein, PhD, Professor, Department of Psychiatry, University of Rochester Medical Center, 300 Crittenden, Rochester, NY 14642, Phone: (585) 275-6742, Fax: 585-273-1082, paul_duberstein@urmc.rochester.edu

health). We then examined the predictive performance of the Charlson CCI with and without these measures in a validation subsample.

Results—Across 5, 10, 15, 20, and 25-year time horizons, the psychosocially augmented CCI showed substantially better discrimination (AUCs (95% CI) from .83 (.81, .85) to .84 (.83 .86)) than the CCI (AUCs from .74 (.71, .76) to .77 (.76 to .79)). These translated into net reclassification improvements from 27% (23%, 31%) to 35% (32%, 38%) of survivors and from 23% (17%, 30%) to 34% (17%, 30%) of decedents; and 23%–42% reductions in the Number Needed to Screen. Calibration improved at all time horizons except 25 years, where it was decreased.

Conclusion—Widespread attempts to improve prognostic models might consider not only novel biomarkers, but also psychosocial questionnaire measures.

Keywords

Charlson Comorbidity Index; Prognostic Models; Personality Phenotype; Socioeconomic Status; Health and Lifestyle Study

Introduction

Survival in clinical settings can sometimes be predicted based on clinical judgment, and/or over relatively short time periods of time[1]. In other cases mortality statistics for a particular disease may be of help (e.g., five year survival probabilities for a particular type of cancer). However, achieving accurate prediction of survival over longer time periods, in patients who are not acutely ill or elderly, has often proven challenging[2,3].

Existing models for both clinical prediction and risk adjustment are based primarily on clinical and demographic factors such as presence of chronic diseases, age, and gender. One approach to improving prognostic models of all kinds is to leverage the predictive power of genetic data—a central goal in “personalized medicine” [4–6]. Other biological markers connected to death, such inflammatory cytokines, have also been used to achieve better survival prediction[7]. While incorporating these factors often produces improvements in prediction models, an increasing emphasis on the tradeoff between prediction gains and cost has emerged as an important issue[8,9]. One argument is that many novel biomarkers are expensive and involve tests not reimbursed by payers, but improve models only modestly[9]. A theoretical cost function has been proposed to evaluate CE, but requires placing a dollar amount on false negatives, false positives, and true positives in addition to the costs of the added marker themselves[10]. Clearly, the price of false positives or negatives varies according to the situation, and even then exact costs would be subject to debate. As a general principle however, the Cochrane Prognosis Group has suggested searching for lower cost alternatives to improve prognostic models[11].

Here, we raise the possibility that a paper-and-pencil questionnaire of psychosocial factors associated with health and longevity--while not entirely without cost--is on average likely to be less costly than a novel biomarker (as well as less invasive). Thus, fixing other costs and the false negative rate, virtually any improvement in true positives (i.e., discrimination and reclassification) may compare favorably with that of a more expensive prognostic factor.

The “burden of proof” then becomes showing that such questionnaire measures achieve even minimal gains in prediction.

Personality phenotype (relatively stable patterns of behavior, cognition, and affect[12]), cognitive ability[13], and social support[14,15] are frequently assessed by questionnaire measures. Cognitive ability and personality affect health and mortality via daily lifestyle practices, health literacy and decision-making, coping behaviors, and neuro-endocrine responses to chronic stress. Socioeconomic position (SEP) is thought to influence mortality risk via environmental exposures, material resources, access to care, health literacy, and socio-cultural health norms and attitudes[16,17]. The addition of education and income to prognostic models for cardiovascular disease has recently been studied, with encouraging results [18–21].

A small questionnaire covering highly predictive psychosocial health risks might thus be a cheap, brief, and non-invasive adjunct for predictive models. Moreover, patient questionnaires are becoming increasingly common with the data-capture capacity of electronic health records. Such information is thus increasingly feasible to collect for both clinical prediction and administrative risk adjustment models alike. We explored the extent to which long-term survival prediction over 5, 10, 15, 20, and 25-year time horizons could be improved with a brief battery of psychosocial questionnaire items. We focused on a widely studied prognostic model for mortality: the Charlson Comorbidity Index (CCI), which involves age, sex, and disease profile[22,23]. Specifically, our goal was to first identify strong psychosocial prognostic factors, then use these variables to construct a psychosocially-augmented version of the CCI, and finally compare its predictive performance to the traditional CCI across a comprehensive set of criteria.

Methods

Cohort

The Health and Lifestyle Survey (HALS) is a cohort study designed to examine behavioral factors associated with health outcomes in non-institutionalized adults 18 and over living in England, Scotland, or Wales[24]. The study consisted of a baseline interview by a visiting nurse about numerous aspects of lifestyle, conducted in 1984–1985. During this interview, nurses distributed an extensive psychosocial questionnaire with standard instructions, which subjects completed and returned by mail. Of the 9002 interviewed persons, 6550 returned usable psychosocial data (73%), with 6,474 having complete data on all variables in the analysis. A multivariate model revealed no differences between the analysis sample and broader sample in age or CCI scores, although women were slightly less likely to return the inventory, as were persons of lower social class, education, and household income (all P’s $< .05$). Inverse probability of selection weights based on these factors were developed for later use in sensitivity analysis. The study conformed to all ethical and review board codes in place at the time, and was run out of Cambridge University.

Measures

We selected candidate prognostic psychosocial factors from the broader HALS battery based on the following criteria: 1) The measure had to reflect a relatively stable circumstance or trait (since many transient factors might have less influence on long-term mortality risk); 2) the measure, if it was a multi-item scale, had to have sufficient reliability (e.g. composite internal consistency ≥ .70 in HALS, meaning that at least 70% of scale score variance reflected the psychosocial construct in question); 3) the measure had to be one that patients could complete themselves on paper or a computer screen (e.g., no health professional time or special equipment was required, which would pose an unnecessary challenge to implementation).

Socioeconomic Position

To assess socioeconomic position, we used household income, educational level, and Registrar General's Social Class (RGSC). The RGSC groups people into social classes based on occupation type: Class 1, professional; Class 2, managerial and technical; Class 3, skilled (both manual and non-manual); Class 4, partly-skilled; Class 5, unskilled[25]. Education level was coded according to the National Vocational Qualifications (NVQ) levels: 1 = 5th grade or less, no school-leaving qualifications; 2 = CSE, O Level, and equivalent qualifications; 3 = A Level and equivalent; 4 = guild, clerical, or commercial degree; 5 = degrees and professional qualifications. Weekly household income was measured using 12 brackets ranging from 20 pounds sterling or less per week, to 500 pounds sterling or greater (1985 currency rates). 18% with psychosocial and other data were missing household income bracket data, so we imputed bracket probability with ordinal logit models based on age, sex, home ownership, marital status and spouse's employment status (if married), RGSC, and education level, assigning each individual their most probable income bracket[26], to avoid complete-case analysis bias.

Psychosocial Factors

Personality was assessed with the Framingham Type A Behavior Pattern Inventory (TABP)[27], a 6 item questionnaire (Bentler's dimension-free composite reliability[28] of .71), measuring hard-driving, time pressured, work-obsessed tendencies thought to be linked to health risk. Other personality phenotypes were assessed by subscales on the Eysenck Personality Inventory (EPI) [29] and included a set of traits collectively grouped under the broad "Neuroticism" and "Extraversion" personality dimensions. The EPI also includes a "Lie" scale designed to assess dishonest responding. Social support was assessed with the 7-item Perceived Social Support Inventory (PSSI)[30], and Fluid cognitive ability (abstract reasoning unrelated to language), was assessed using a geometric and spatial reasoning test[31]. Scales were standardized to a mean of 0 and standard deviation (SD) of 1 so that higher scores reflected higher levels of the quality in question. Further details, including composite reliabilities, appear in the Supplementary Digital Content (SDC) Table 1. Finally, standard health behavioral questions were asked, including whether participants were current or former smokers, whether they had ever drank to excess for a period of a year or longer, and their height and weight was taken by nurse-interviewers. BMI was computed and included to reflect diet and exercise behavior.

CCI

We utilized scores from self-report version of the CCI[32,33] based on respondents' reports of health information to examining nurses during the first wave of interviews from 1984 to 1985. The CCI includes one entry for AIDS, which was not measured in the HALS baseline due to the newness of the disease at that time. This is unlikely to have effected CCI scores however, as only 17 known cases of AIDS existed in the UK at the end of 1983[34]. The CCI categories were asthma/respiratory disease, arthritis/rheumatism, ulcers/gastro-intestinal disease, stroke, diabetes, congestive heart failure/angina/coronary artery disease, kidney disease, liver disease, and cancer. The CCI scoring systems weights each condition with 1 or more points when present, then adds 1 additional point for every decade of age over 50.[32] Preliminary analyses revealed an extremely low number of liver disease cases, and when this component was eliminated, the CCI performed virtually identically. All-cause mortality was based on death records from the Registrar General's Office through 2009, and available for all subjects. Death at 5, 10, 15, 20, and 25 years was coded based on the time from the participant's age at the baseline interviews in 1984–1985.

Analysis

Primary analyses used parametric survival models, which have advantages in precision and flexibility over the Cox model, as well as the capacity to provide predicted probabilities of survival[35]. The Weibull model reflected the best fit and was used in all analyses. Within the development sample, we examined the added predictive value for each potential prognostic factor, selecting those with the best performance across multiple criteria reflecting discrimination, reclassification, and calibration (details in SDC Table 2)[36]. We then added these to the standard CCI conditions to create a CCI-Psychosocial Risk (CCI-PSR) score. Weights were estimated in the development sample, and these were used to compute risk scores (cumulative hazards) in the validation sample at 5, 10, 15, 20, and 25 year follow-up, reflecting conventional CCI time horizons (5–10 year), as well as longer time points at regular increments.

Scores for both the CCI and CCI-PSR were compared with the Area Under the Receiver Operating Curve (AUC; true vs. false positives across all possible cutpoints of the risk score). The utility of the AUC alone in evaluating models has come into question: it is insensitive to improved classification, often unrelated to calibration, lacks an intuitive clinical or public health metric, and can have a high error rate in small samples[37]. Thus we also examined calibration with Cook's classification calibration for four risk strata, using expected survival at each time horizon from the Kaplan-Meier estimator[35]. Clinical significance was assessed via the Net Reclassification Index (NRI; percent with improved classification due to the addition of psychosocial data in the CCI-PSR); the Integrated Discrimination Improvement (IDI; the improvement in discrimination slope of the CCI-PSR over CCI); and Number Needed to Screen (NNS) to identify 100 decedents. The latter took the form of the inverse of the risk differences between those classified as decedents vs. survivors, according to the cut-point where sensitivity and specificity curves crossed. Secondary measures of performance included Net Benefit, or the improvement in true positive predictions, penalized for false positives; the mean-squared error of prediction, or Brier score; and NNS based on a cut-point maximizing sensitivity + specificity. Sensitivity

analyses examined whether common health behavioral factors (normal vs. overweight vs. obese; current vs. former vs. ever smoker; history of problem drinking) either a) improved the original CCI as much as selected psychosocial factors, or b) improved upon the CCI-PSR. Analyses were performed in Stata Version 13.

Results

Table 1 shows sample descriptive statistics, stratified by survival status. Weights estimated for the standard CCI conditions in the development sample are shown in the left hand portion of Table 2; the risk associated with decades of age increased over follow-up time ($P < .001$) and was treated as a time varying covariate.

Our evaluation of psychosocial factors revealed a clear distinction between strong and weaker predictors of survival (SDC Tables 2 and 3). Five prognostic factors improved the model substantially across all five prediction criteria: higher levels of income, education, and the EPI trait of "communalism" were associated with reduced mortality risk, while higher scores on the lie and Type A scales were associated with increased risk. Income's protective effect lessened with age ($P < .001$), while Type A risk increased with follow-up time ($P < .001$). The CCI-PSR model, including these five psychosocial factors, is shown in the right portion of Table 2.

Table 3 reports results of risk-score comparisons in the validation sample. Compared to the CCI, the CCI-PSR showed substantially better discrimination (AUC differences of .07 to .08) across all time horizons (all Ps < .001). With respect to the NRI, the CCI-PSR improved prediction in at least 60% of individuals at 5-, 10-, and 15-year time horizons, and in over 50% of people at 20 and 25-year follow-up. While all NRIs were significantly different from 0 (Ps < .001), they were not significantly different across varying lengths of follow-up, or when examined separately for decedents and survivors.

Cook classification chi-squares tended to improve by 50% or more in the CCI-PSR compared to the conventional CCI, through 20-year follow-up. At 25-year follow-up, the CCI showed better calibration, however. Finally, fewer persons needed to be screened with the CCI-PSR at 5, 10, 15, 20, and 25 years to identify 100 decedents. SDC Table 4 shows that the CCI-PSR showed similar improvements in supplementary measures; health behaviors improved the traditional CCI slightly but significantly less than the psychosocial factors, and did not substantially improve the CCI-PSR (SDC Table 5); and that IPW estimates adjusting for sample selection yielded very similar model coefficients (SDC Table 6).

Conclusions

Our results from this proof-of-principle study indicated that inexpensive questionnaire measures of psychosocial risk factors substantially improved a common predictive model of mortality. These improvements were significant in not only statistical terms and metrics, but also in clinically relevant metrics. Three central issues warrant consideration.

First, the utility of new prognostic factors must be evaluated in the context of their costs[8,10]. Many promising candidate genes may indeed justify genotyping costs, and biomarkers often decrease in price with time. Nevertheless, information with predictive value is often readily available at minimal cost and without obtaining tissue or blood samples[11,38]. In this case, paper and pencil measures of socioeconomic status and personality phenotype (totaling 19 questions) provided non-trivial added predictive power for premature death, over relatively long time horizons.

The five psychosocial factors improved not only overall discrimination, but also reclassification in 50% to 60% of people. Calibration improved by roughly 50% up to 15-year follow-up times, and the NNS decreased by 23–53%. For comparison, recent reports in the prognostic model literature indicate that 7 common genetic variants increased the Gail Breast Cancer Model AUC by .02, while another panel of 10 increased it by .04[39]. Adding HDL to the Framingham model improved risk classification in roughly 12% of subjects[3]. C-reactive Protein improved a CVD risk model's calibration by roughly 42%.[40] In the context of these examples, present results suggest that psychosocial risk markers can, in principal, compare favorably with many biomedical markers proposed for addition to predictive models. Naturally, the exact degree of improvement required to justify an added prognostic factor depends on context.

Second, while age, gender, and chronic diseases are fundamental biological predictors of survival, numerous psychosocial risk factors for mortality have also been documented for some time.[12,17] In the present analysis, communalism, or the tendency to seek out the company of others, as well as Type A tendencies, were highly prognostic of delayed and premature death, respectively. Less is known about the third measure, the EPI Lie scale, for which higher scores were associated with increased mortality risk. While interpretations vary, some data suggest that it may reflect denial of negative feelings[41], a coping response hypothesized to engender neuroendocrine dysregulation[42] and unhealthy behavior[43]. Another possibility is that it reflects general dissembling that, over time, eventuates in indirect health consequences—for instance, by chronic misreporting to providers of symptoms, treatment adherence, or damaging health behaviors.

Third and finally, the CCI-PSR outperformed the CCI across all five time horizons and model-performance criteria, with one exception. Most prediction questions in medicine concern 5- or at most 10-year time horizons. These were arguably the spans over which psychosocial prognostic factors helped most. Note that psychosocial factors degraded model calibration at 25-year follow-up, however. Calibration decline in the presence of discrimination and other improvements sometimes occurs[40], and in this case hints at a shift over time in one or more psychosocial factors. Social mobility may change socioeconomic position, and personality traits evidence normative lifespan change and maturation, with some elements perhaps amenable to intervention[44]. Some have also suggested that personality phenotype can be "phenocopied"—that is, people may be taught to emulate certain traits like communalism, even if they are not naturally predisposed to them.[17] More broadly, the utility of psychosocial factors likely lays in their characterization of health risks that are largely invisible in clinical, demographic, or biomarker measurements.

An understanding of study limitations is crucial in interpreting our results. First, our goal was not to endorse a particular set of psychosocial prognostic factors, but instead to conduct a proof-of-principle investigation. Our findings are limited to all-cause mortality and this set of psychosocial measures, in this sample. Future work might consider “macro” level predictors of health, such as neighborhood poverty. Measures of SEP and personality phenotype will be of differential value depending on the model, population, and outcome. We also examined psychosocial factors at a single time point, and the way they change over time (i.e., worsening Type A tendencies, upward socioeconomic mobility) may have prognostic significance. Relatedly, a very large number of predictive models are in existence, and are continually being updated. Our study focused on the CCI which, while popular, may not be the strongest comorbidity index currently available. For instance, recent work has focused on the Elixhauser[45], which may outperform the CCI[22,46]. The next step in this avenue of research is thus not only to consider a wider range of psychosocial prognostic factors, but other comorbidity indices. Risk models for particular outcomes, such as cardiovascular events, have the advantage of greater focus. Finally, we do not intend to suggest that SEP or personality phenotype should be examined *instead of* genotype or other biomarkers. We merely suggest that psychosocial questionnaire measures may also be worthwhile to consider in formal risk prediction. Clinically, models based on low-cost and/or non-invasive sources of information may also be useful in serial screening, serving as an initial tool to “rule in” those at risk for an outcome. More expensive/invasive models, presuming they improve final classification, can then be saved only for those identified with elevated risk at the first stage.

The advancing scope of electronic medical records (EMRs) offers the possibility to capture psychosocial data for use in predictive algorithms enacted by clinical decision support software. However, implementing such assessment in health care settings is a major, long-term endeavor which must be approached through a series of steps, similar to the roll-out of PROMIS[47]. Determining whether psychosocial data successfully enhances model performance is the natural first step. Many translational steps are needed before psychosocially augmented prediction models might be used in practice. Even then, if prognostic models are run in the background via clinical decision support software--requiring no work on the part of the clinician--clinicians may or may not use their results. As well, there should be no doubt that psychosocial data collected for clinical use is private health information, guarded carefully against parties without the patient’s best interests in mind.

Methodologically, researchers with a vested interest in the predictive power of a particular variable (e.g., a particular biomarker, trait, or SES indicator) may selectively report data that cast their variable in the most favorable light. By averaging across multiple criteria (discrimination, calibration, reclassification improvement, clinical, statistical significance), multiple time horizons (5, 10, 15, 20, 25 years), and using a split sample development-validation strategy, we mitigated that type of bias. Nevertheless, clinical and policy decisions affect many constituencies, and these stakeholders will rarely agree on which outcome or predictor variables ought to be prioritized. For example, clinicians and patients may be more interested in clinical metrics over the shorter term, while governments may be

more interested in public health metrics over the longer term. Moreover, to counteract the well-funded constituency interested in biomarker data (biomedical industry), there is a need to identify stakeholders with a vested interest in collecting psychosocial data that are noninvasive, inexpensive, and unlikely to be tainted by the prospect of profit. Given these imperatives, present findings warrant further consideration of psychosocial factors in the medical prediction literature.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Descriptive Statistics by Survival Status

	Survivors, N = 4,548	Deceased, N = 1,926
Baseline Variable	**Mean (SD) or Percent (N)**	**Mean (SD) or N (%)**
Age at baseline (years)	38.29 (12.70)	63.14 (12.48)
Sex		
Male	41% (1,879)	52% (999)
Female	59% (2,669)	48% (927)
CCI Comorbidities[a]		
Chronic Asthma / Emphysema	3% (116)	5% (102)
Arthritis / Rheumatism	3% (134)	11% (204)
Cancer	<1% (17)	1% (25)
Diabetes	1% (30)	3% (51)
GI Disease	2% (104)	4% (81)
Heart Disease	1% (58)	11% (207)
Kidney Disease	<1% (16)	1% (12)
Liver Disease	<1% (3)	<1% (4)
Stroke	<1% (14)	3% (52)
Social Circumstances[b]		
Registrar General Social Class		
I	6% (292)	4% (83)
II	25% (1,124)	21% (399)
III	47% (2,157)	50% (958)
IV	15% (684)	18% (347)
V	4% (202)	6% (114)
Other	2% (100)	1% (16)
Education Level		
≤5th grade, no school-leaving qualifications	42% (1,896)	70% (1,348)
CSE, O Level, and equivalent	18% (823)	7% (142)
A Level and equivalent	6% (278)	3% (54)
Guild, clerical, or commercial degree	14% (643)	6% (111)
Degree or professional qualifications	20% (902)	14% (271)
Weekly Household Income (pounds)	161.35 (92.36)	107.18 (79.45)
Perceived Social Support Score [c]	.05 (.89)	-.05 (1.12)
Phenotypic Traits [c]		
Rumination	-.06 (.96)	.12 (1.06)
Mood Lability	.10 (.99)	-.24 (.98)
Oversensitivity	.07 (1.00)	-.16 (.98)
Nervous Tension	-.04 (.97)	.07 (1.05)

	Survivors, N = 4,548	Deceased, N = 1,926
Baseline Variable	**Mean (SD) or Percent (N)**	**Mean (SD) or N (%)**
Social Dynamism	.08 (.98)	-.18 (1.02)
Impulsivity	.06 (1.01)	-.16 (.96)
Communalism	.11 (.97)	-.25 (1.03)
Jocularity	.110(1.04)	-.25 (.85)
Haste	.03 (1.00)	-.06 (.99)
Social Dependency	-.05 (.96)	.09 (1.07)
"Lie" scale	-.20 (.92)	.46 (1.02)
Type A Tendencies	.10 (.98)	-.24 (1.00)
Fluid IQ	.12 (.90)	-.10 (1.03)

Notes: Survival over 25-year follow-up period. M = Mean, SD = Standard deviation

[a] Charlson Comorbidity Index (CCI) self-report conditions.

[b] "Other" category includes students, armed services, and those never employed;.

[c] social support scale and phenotypic traits in z-score metric; Mean=0, SD=1.

Higher scores indicate higher value of property being assessed for psychosocial measurements. All values simple, unadjusted means. Type A relationship to mortality reverses (i.e., higher scores indicate higher mortality risk) with demographic adjustment and grows in size over time (see Table 2).

Table 2

Components and Weights of Charlson Comorbidity Index Traditional and Augmented Versions.

	Charlson Comorbidity Index (CCI)			Charlson Comorbidity and Psychosocial Risk Index (CCPRI)		
Risk Factor	**HR**	**95% CI**	**P**	**HR**	**95% CI**	**P**
Male (vs. Female)	1.57	(1.39, 1.78)	<.001	1.86	(1.64, 2.12)	<.001
Decades of Age Over 50 [a]	3.14	(2.62, 3.76)	<.001	2.59	(2.15, 3.11)	<.001
Chronic Asthma/Emphysema	1.62	(1.24, 2.11)	<.001	1.41	(1.08, 1.84)	0.012
Arthritis/Rheumatism	1.78	(1.45, 2.18)	<.001	1.60	(1.30, 1.96)	<.001
Cancer	3.06	(1.74, 5.36)	<.001	3.07	(1.73, 5.43)	<.001
Diabetes	1.47	(1.01, 2.14)	0.044	1.71	(1.18, 2.49)	0.005
Gastro-Intestinal Disease	1.18	(0.86, 1.63)	0.300	1.13	(0.82, 1.55)	0.469
Heart Disease	2.70	(2.17, 3.35)	<.001	2.27	(1.83, 2.80)	<.001
Kidney Disease	1.07	(0.39, 2.97)	0.891	0.70	(0.25, 1.97)	0.498
Stroke	2.36	(1.62, 3.44)	<.001	2.02	(1.38, 2.94)	<.001
£100s of weekly income [b]				0.40	(0.33, 0.48)	<.001
Education Level [c]				0.88	(0.84, 0.93)	<.001
Type A, 1 SD [d]				1.43	(1.11, 1.85)	.006
Communalism, 1 SD				0.89	(0.84, 0.95)	<.001
Lie Scale, 1 SD				1.29	(1.21,1.38)	<.001

Notes: Development sample, N = 3,236. Final weights for traditional Charlson Comorbidity Index and for CCI and CCI-PSR, fitted to all 25 years of mortality follow-up data. HR= Hazard Ratio, 95% CI = 95% Confidence Interval, SD = Standard Deviation. Estimates from parametric survival model.

[a] Risk for each decade of age over 50 for 5-year mortality; risk increased with follow-up time (P < .001) and is respectively at 10, 15, 20, and 25 years (HR (95% CI)): 4.24 (2.97, 6.07) 6.95 (4.08, 11.84); 11.39 (5.60, 23.14); 18.65 (7.69, 45.23), all P's < .001 in augmented model. Similar increases observed in standard CCI.

[b] 1985 income for household. Protective effect of income at age 50 and below; effects vary across age (P<.001), and are at ages 60, 70, and 80, respectively: .52 (.47, .59), P < .001; .67 (.61, .75), P < .001; . 88 (.76, 1.01), P = .077.

[c] Estimate for education corresponds to a 1-level move upward in the following scale: a) < 5th grade; b) CSE, O level, or equivalent; c) A level or equivalent; d) guild, clerical, or commercial degree; e) professional degree.

[d] Type A risk for mortality by 5 year follow-up; effect changes over time (P < .001), and is at 10, 15, 20, and 25 year follow-up respectively: 2.43 (1.46, 4.03), P = .001; 4.12 (1.92, 8.81), P < .001); 6.99 (2.53, 19.28), P < .001; 11.86 (3.33, 42.22), P < .001

Table 3

Comparative Predictive Power of Two Charlson Comorbidity Indices Over Varying Time Horizons

Performance Criterion	**Discrimination**	**Reclassification**		**Calibration**		**Clinical**	**Public Health**	
Time horizon / predictive model	**Area Under Curve**	**Net Reclassification Improvement**	**Integrated Discrimination Improvement**	**Classification Calibration χ^2**	**Sensitivity**	**Specificity**	**# Needed to Screen (NNS) to Detect100 Decedents**	**% Change, NNS**
5 Year Follow-up (5.2% deceased)								
Charlson Comorbidity Index	.75 (.71, .79)			28.36	.69	.66	1360 (1077, 1787)	
Charlson Comorbidity and Psychosocial Risk Index	.83 (.81, .86)	.69 (.54, .85)	.04 (.02, .06)	7.18	.70	.78	787 (652, 965)	↓42.2%
10 Year Follow-up (11.6% deceased)								
Charlson Comorbidity Index	.74 (.71, .76)			84.00	.68	.69	609 (525, 722)	
Charlson Comorbidity and Psychosocial Risk Index	.83 (.81, .85)	.67 (.57, .78)	.06 (.04, .08)	19.71	.74	.78	384 (343, 435)	↓ 37.0%
15 Year Follow-up (17.8% deceased)								
Charlson Comorbidity Index	.74 (.72, .77)			111.03	.70	.65	488 (430, 564)	
Charlson Comorbidity and Psychosocial Risk Index	.83 (.82, .85)	.61 (.52, .70)	.07 (.06, .08)	43.10	.74	.78	281 (2.58, 3.09)	↓ 42.4%
20 Year Follow-up (24.3% deceased)								
Charlson Comorbidity Index	.76 (.74, .78)			127.20	.68	.69	349 (316, 391)	
Charlson Comorbidity and Psychosocial Risk Index	.84 (.82, .86)	.57 (.48, .64)	.08 (.06, .09)	72.17	.74	.79	227 (212, 246)	↓ 36.0%
25 Year Follow-up (29.2% deceased)								
Charlson Comorbidity Index	.77 (.76, .79)			88.20	.71	.71	281 (259, 309)	
Charlson Comorbidity and Psychosocial Risk Index	.84 (.83, .86)	.51 (.43, .58)	.08 (.06, .09)	101.04	.76	.79	217 (195, 222)	↓22.8%

Notes: All results from validation sample, N = 3237. Higher values of risk classification calibration χ^2 indicate worse fit, or worse calibration. Net Reclassification Improvement is the proportion of persons receiving an improved risk score, adjusted for those whose risk score worsens with the addition of psychosocial factors to the prediction model; Integrated Discrimination Improvement is the improvement in discrimination slopes from adding psychosocial factors. Clinical and public health measures based on risk score cut point where sensitivity and specificity curves cross in development subsample.