The inverse hazard law: Blood pressure, sexual harassment, racial discrimination, workplace abuse and occupational exposures in US low-income black, white and Latino workers ☆

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ABSTRACT

Research on societal determinants of health suggests the existence of an "inverse hazard law," which we define as: "The accumulation of health hazards tends to vary inversely with the power and resources of the populations affected." Yet, little empirical research has systematically investigated this topic, including in relation to workplace exposures. We accordingly designed the *United for Health* study (Greater Boston Area, Massachusetts, 2003–2004) to investigate the joint distribution and health implications of workplace occupational hazards (dust, fumes, chemical, noise, ergonomic strain) and social hazards (racial discrimination, sexual harassment, workplace abuse). Focusing on blood pressure as our health outcome, we found that among the 1202 low-income multi-racial/ethnic working class participants in our cohort – of whom 40% lived below the US poverty line – 79% reported exposure to at least one social hazard and 82% to at least one high-exposure occupational hazard. Only sexual harassment, the least common social hazard, was associated with elevated systolic blood pressure (SBP) among the women workers. By contrast, no statistically significant associations were detectable between the other additional highly prevalent social and occupational hazards and SBP; we did, however, find suggestive evidence of an association between SBP and response to unfair treatment, implying that in a context of high exposure, differential susceptibility to the exposure matters. These results interestingly contrast to our prior findings for this same cohort, in which we found associations between self-reported experiences of racial discrimination and two other health outcomes: psychological distress and cigarette smoking. Likely explanations for these contrasting findings include: (a) the differential etiologic periods and pathways involving somatic health, mental health, and health behaviors, and (b) the high prevalence

of adverse exposures, limiting the ability to detect significant associations. As clarified by the "inverse hazard law," to understand health inequities, research is needed that contrasts exposures and health status population-wide, not just among those most inequitably exposed.

In 1971, Julian Tudor Hart famously penned the "inverse care law": "The availability of good medical care tends to vary inversely with the need for the population served" (Hart, 1971). Research on societal determinants of health likewise suggests the existence of an "inverse hazard law," which we define as: "The accumulation of health hazards tends to vary inversely with the power and resources of the populations affected." Clustered together and embodied conjointly (Krieger, 2004), these health hazards include economic deprivation, discrimination, and hazardous living and working conditions, harming the health of societal groups exposed to – as compared to those who are buffered from, and often benefit from – these inequities (Krieger, 2004; Levy & Sidel, 2006).

As a case in point, research indicates that in the United States low- compared to high-wage workers are more likely to be exposed to occupational hazards, have less job autonomy, have less or no coverage for health insurance, fewer resources to live a healthy life, and are more likely to be persons of color and women, reflecting past and present patterns of racial/ethnic and gender discrimination (Baron & Dorsey, 2006). Yet, to date, scant research has focused on the combined health impact of social and occupational hazards at work (Lipscomb, Loomis, McDonald, Argue, & Wing, 2006; Quinn, 2003).

We accordingly designed the *United for Health* study to investigate the joint distribution and health implications of occupational (dust, fumes, chemical, noise, ergonomic strain) and social (racial discrimination, sexual harassment, workplace abuse) hazards in a US population of predominantly low-wage white, black, and Latino women and men unionized workers. In prior publications, we have reported on this cohort's high levels of exposures to economic deprivation and both social (Krieger et al., 2006) and occupational (Quinn et al., 2007) hazards; a sub-sample analysis documented associations between racial discrimination and both cigarette smoking and psychological distress (Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005). In this paper, we analyze associations between the workers' blood pressure and their exposure to social and occupational hazards. Fig. 1 presents our conceptual model. It is premised on an ecosocial analysis of embodying inequality in relation to pathways, levels, and lifecourse (Krieger, 1994, 2004), with the specific content informed by research on social, physical, occupational, behavioral, and anthropometric determinants of blood pressure, their societal distribution, and social disparities in blood pressure (Baron & Dorsey, 2006; Huang & Ghio, 2006; Kasl, 1996; Krieger, 1999; Lundberg, 2002; Pickering, 2001; Talbott, Gibson, Burks, Engberg, & McHugh, 1999; Wyatt et al., 2003).

Study population and methods

Study population and protocol: the United for Health cohort

As described in detail in our prior publications (Barbeau, Hartman, Quinn, Stoddard, & Krieger, 2007; Krieger et al., 2005, 2006; Quinn et al., 2007), we recruited participants in the *United for Health* study from the rosters of union members employed in 14 worksites located in the greater Boston area in Massachusetts and variously engaged in meat processing, electrical light manufacturing, retail grocery stores, and school bus driving. The unions and management had no access to the study data and no role in the preparation, review, or approval of the study's scientific papers. The study incentive was either a 1-h paid work-release plus a $25 pre-paid grocery card (handed out after the survey was completed) or, if paid work-release was not an option, a $50 pre-paid grocery card. All participants received an informed consent reference sheet and provided verbal informed consent. Conduct of the study was approved by the Dana-Farber Cancer Institute's Office for the Protection of Research Subjects, the Human Subjects Committee of the Harvard School of Public Health, and the Institutional Review Board of the University of Massachusetts.

Recruitment took place between March 2003 and August 2004. Union members at each worksite were sent an introductory letter by the union. Study staff then screened, recruited, and administered the survey to the workers on-site. The 40–45 min survey was administered (either in English or Spanish) in a private room, followed by a 15-min health check. For the survey, we used audio-computer assisted self-interviewing (ACASI), both to improve likelihood of obtaining sensitive information and to enable persons with low literacy to respond (Office of Applied Studies, 2001). The Spanish version of the survey was translated from English and then back-translated to ensure accuracy. One or more of the interview staff were bilingual in English and Spanish and were available to answer participants' questions.

Among the 2323 union members on the list we were given by the unions, 1776 stated that they met study eligibility criteria for age (25–64 years old) and length of employment (at least 2 months). Of these, 1282 (72%) completed the survey, of whom 80 had an age that was either unknown or outside the eligible age range, yielding an analytic sample of 1202 age-eligible workers.

Sociodemographic measures: individual-level and worksite

We obtained self-reported data on race/ethnicity, gender, sexuality, age, nativity, and current and childhood socioeconomic position. All persons who self-identified as
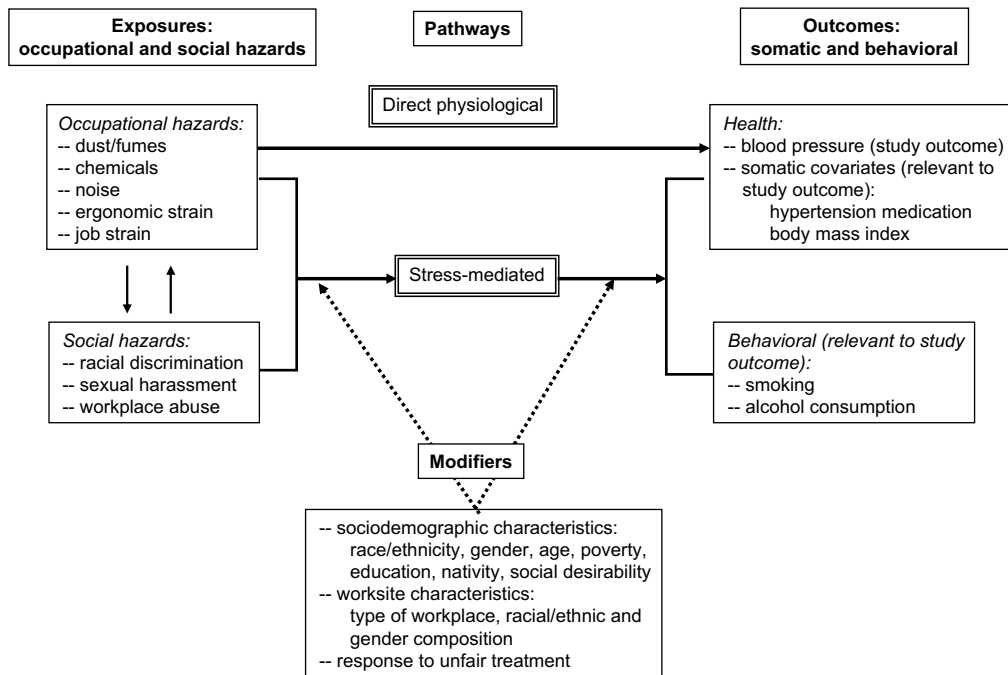
Fig. 1. Conceptual model for blood pressures analyses: United for Health study (Boston, MA, 2003–2004).

being Latino or Hispanic were categorized as such; the "white" and "black" designations refer only to non-Hispanic white and black populations. We obtained data on the highest level of education attained by the respondents, their partners (if applicable), and their parents, and assessed income in relation to household poverty level, taking into account the number and age of persons in the household; in 2003, the US federal poverty line for a household of 2 adults and 2 children equaled $18,660 (US Census Bureau, 2004). At the worksite level, we obtained data on the gender and racial/ethnic composition of the participants' coworkers and also worksite type (manufacturing, retail, transportation).

Social hazard measures

We included self-report measures for three social hazards: workplace abuse, sexual harassment, and racial discrimination (Krieger et al., 2006). To control for how self-presentation might affect these responses, we used a 5-item validated social desirability scale (Hays & the RAND Corporation, 1989).

The measures of workplace abuse (reduced 8-item version of the Generalized Workplace Abuse (GWA) instrument (Richman et al., 1999)) and sexual harassment (5 items from two validated instruments (Fitzgerald, Gelfand, & Drasgow, 1995; Richman et al., 1999)) referred to events in the past 12 months. Examples of workplace abuse included being yelled or sworn at; the sexual harassment instrument focused on sexual coercion, unwanted sexual attention, and gender-based hostility. Summary scores (recommended for analysis as continuous variables (Richman et al., 1999)) equaled the sum of items reported

(workplace abuse: "never" = 0, "once" = 1, "more than once" = 2, score range = 0–16; sexual harassment: "no" = 0, "yes" = 1, score range = 0–5).

To measure racial discrimination, we used the validated 9-item "Experiences of Discrimination" (EOD) questionnaire (Krieger et al., 2005, based on Krieger, 1990; Krieger & Sidney, 1996), which employs a timeframe of "ever," reflecting how exposure can start in childhood and can have a cumulative as well as contemporaneous adverse impact on health (Krieger, 1999). We employed the more commonly used measure of number of domains (0, 1–2, 3+) in which experiences of racial discrimination were reported (range: 0–9), rather than the frequency score, since the two measures yielded comparable findings.

Occupational hazard measures

Self-report data on workplace occupational hazards were based on 12-month recall period, using a 3-point scale corresponding to low, moderate, or high exposure, except for chemical and dust exposure, which used a 4-point scale to maintain comparability to prior studies (Quinn et al., 2007). Measures pertained to: (1) airborne dust, fumes, and chemicals (American Thoracic society questionnaire, European Community Respiratory Health Survey, 1993; Ferris, 1978); (2) noise (<3, 3–6, or >6 h per shift spent in a noisy environment, assessed using Occupational Safety and Health Administration (American National Standards Institute, 1991) and World Health Organization (2001) guidelines); (3) ergonomic strain (<1, 1–4, >4 h per shift involving strained postures in the shoulder, neck, and back or repetitive hand motions, assessed using the Washington State (2000) Ergonomics

Rule, plus self-report on the heaviest objects lifted at work); and (4) job strain (adapted from the Karasek Job Content Questionnaire for demand-control (Karasek, 1985), with scores dichotomized at the national median value). High exposure equaled the top level for all occupational hazards except for: (a) dust and chemicals, for which we used the top two levels, and (b) job strain, where "high strain" equaled "high demand/low control" and other combinations were categorized as "low strain."

Physical health exam

After participants sat quietly for 5 min, we used the Dinamap 8100 automated blood pressure recorder (Whincup, Bruce, Book, & Shaper, 1992) to get 3 readings each for systolic and diastolic blood pressure; in our analyses, we used the average of the 3 measures. We next asked participants to remove their shoes and measured their height to the nearest half-inch and weight to the nearest pound and used these data to calculate body mass index (BMI = weight (kg)/height (m^2)). We also collected participants' self-report data on: having ever been told by a physician they had hypertension; current use of anti-hypertensive medication; and time (in hours and minutes) since their last cigarette, caffeinated beverage (coffee, tea, or soda), alcoholic beverage (beer, wine, or liquor), and food consumption. Virtually all current smokers (as categorized based on self-report survey data, using questions from 1997 NHIS Sample Adult Core Questionnaire (National Center for Health Statistics, 2007)) reported having smoked in the past 24 h.

Statistical analysis

Statistical analysis of the data, conducted using SAS (SAS, 2001), was premised on our conceptual model (Fig. 1). We first described the distribution of the outcome, exposures, and covariates, overall and by race/ethnicity and gender, in the analytic dataset and also examined correlations between the social and occupational hazards. Although most variables had little missing data (usually under 1% to at most 5%), for a few key covariates (e.g., adult poverty, educational level, and place of birth for Latinos), it equaled approximately 10%; for a handful of others, e.g., concerning childhood socioeconomic conditions, it exceeded 20%. To avoid problems posed by potentially invalid data, we restricted our analyses to key variables for which the overall percent missing did not exceed 10%. Even so, among the remaining participants, 307 (26%) were missing data on at least one key variable. To avoid bias and enhance statistical power, we employed multiple imputation (including on the individual items used to create summary scores for both the social and occupational hazards), using the Amelia II program (King, Honaker, Joseph, & Scheve, 2001). We created 20 multiply imputed datasets based on an imputation model containing all variables from Table 1 and conducted all subsequent analyses (including combining results across imputations) in SAS.

The multivariable linear regression analyses investigated the associations between blood pressure (separately for systolic (SBP) and diastolic (DBP)) and the social and occupational hazards, controlling for relevant covariates; analogous logistic regression analyses used hypertension as the outcome (SBP ≥ 140 mm Hg and/or DBP ≥ 90 mm Hg and/or taking hypertension medication (Chobanian et al., 2003)). Based on bivariate analyses of each variable in relation to blood pressure, our series of multivariable analyses included: (1) only the sociodemographic variables; (2) only the social hazards (singly, then all 3 together); (3) only the occupational hazard summary score (since bivariate analyses of the individual occupational hazards showed the summary score to be an appropriate exposure measure); and (4) all covariates. We report results only for the "all covariate" model, since it most concisely and efficiently summarized the key findings. In these models, the beta coefficient can be interpreted as the difference in systolic blood pressure (mm Hg) across comparison groups: for the categorical variables, the contrast is the comparison group vs. the specified referent group; for the continuous variables, the difference refers to the change in systolic blood pressure per unit change of the continuous predictor variable.

We conducted analyses first for the total population, controlling for race/ethnicity and gender, and second, stratified by race/ethnicity and gender. Given evidence of considerable of effect modification by race/ethnicity but less by gender (affecting solely sexual harassment and workplace abuse), we present results separately for the white, black and Latino workers, with models including an interaction term for gender with both sexual harassment and workplace abuse. We do not report blood pressure results for the 121 workers of other race/ethnicity (2 American Indian, 28 Asian, 1 South Asian, 1 Native Hawaiian, 77 "two or more races," and 12 "other") because small numbers precluded meaningful analysis. Given similar findings for SBP, DBP, and hypertension, we report only the SBP results. Initial analyses used a mixed model approach to account for clustering of workers within workplaces. Because results showed that, conditional on the covariates, there was negligible worksite clustering, we present results based on fixed effect models, ignoring worksite.

Results

Table 1 shows the distribution of the outcome and selected exposures and key covariates among the 1202 members of the *United for Health* cohort, overall and by race/ethnicity, prior to imputing missing values. As presented in more detail in Supplemental Table 1 and in our previous papers (Barbeau et al., 2007; Krieger et al., 2005, 2006), the findings reveal high levels of economic deprivation and of exposure to social and occupational hazards (79% to at least 1 social hazard, 82% to at least 1 occupational hazard) among this cohort of predominantly low-income multi-racial/ethnic working class women and men, of whom fully 40% were below the US poverty line (24% of whites vs. 40–50% among the black, Latino, and other workers of color). Among all workers, the average systolic blood pressure equaled 134.8 mm Hg and about 21% of the workers were hypertensive (ranging from 6% among

Table 1

Distribution of blood pressure and selected aspects of sociodemographic and worksite characteristics, social and occupational hazards, and additional covariates, overall and by race/ethnicity and gender[a]: *United for Health* study, Boston, MA (USA), 2003–2004

Characteristics	Total[a]	Black[a]		Latino[a]		White[a]		Other race/ethnicity[a]	
	All (N = 1202)	Men (N = 308)	Women (N = 158)	Men (N = 152)	Women (N = 115)	Men (N = 182)	Women (N = 102)	Men (N = 73)	Women (N = 45)
Blood pressure									
Systolic blood pressure: mean (SD) (mm Hg)	134.8 (17.9)	138.5 (18.0)	134.6 (18.7)	132.7 (15.4)	125.7 (19.6)	138.1 (16.4)	129.1 (16.9)	137.1 (17.8)	131.8 (15.8)
Diastolic blood pressure: mean (SD) (mm Hg)	78.6 (12.9)	82.5 (12.9)	77.8 (11.7)	76.9 (11.8)	70.4 (14.1)	81.9 (12.3)	73.6 (11.9)	79.8 (10.0)	74.4 (13.1)
Missing (%)	4.8	0.7	2.5	16.5	15.7	0.6	1.0	4.1	2.2
Hypertensive (%)	20.9	27.3	22.2	9.9	6.1	28.0	19.6	17.8	15.6
Missing (%)	4.7	0.7	1.9	16.5	15.7	0.6	1.0	2.7	2.2
Hypertensive medication (%): yes	17.4	21.1	24.7	7.9	4.4	15.9	12.8	26.0	22.2
Sociodemographic and worksite characteristics									
Age (%)									
24–44 years	49.3	38.3	55.1	64.5	68.7	39.6	45.1	48.0	62.2
45–64 years	50.7	61.7	44.9	35.5	31.3	60.4	54.9	52.1	37.8
Nativity (%): born in US state or territory	47.8	27.0	67.7	27.0	24.4	86.8	92.3	27.4	44.4
Poverty level (household) (%): <100% poverty	40.4	46.8	48.7	46.7	56.5	21.4	29.4	32.9	48.9
Education (respondent) (%): <12th grade	22.8	20.1	15.2	35.5	36.5	20.3	20.6	12.3	28.9
Type of worksite (%)									
Manufacturing	29.0	2.3	2.5	73.0	70.4	40.7	25.5	23.3	15.6
Retail	32.6	18.5	54.4	16.5	25.2	41.2	64.7	21.9	55.6
Transportation	38.4	79.2	43.0	10.5	4.4	18.1	9.8	54.8	28.9
Worksite gender composition of coworkers (%)									
Mostly men	36.2	55.5	36.1	21.7	5.2	45.1	19.6	45.2	20.0
Mostly women	13.3	6.5	12.7	19.7	25.2	10.4	11.8	17.8	13.3
About even	50.0	37.7	50.0	58.6	69.6	44.5	68.6	35.6	66.7
Worksite racial/ethnic composition of coworkers (%)									
Mostly same race/ethnicity	25.6	30.5	24.1	27.6	32.2	21.4	9.8	34.2	22.2
Mostly different race/ethnicity	46.7	43.5	50.6	39.5	33.0	50.0	58.8	49.3	53.3
About even	27.4	26.0	24.7	32.9	33.9	28.6	31.4	16.4	24.4
Social hazards									
Reported at least 1 type of workplace abuse (%)	70.1	66.6	71.5	66.4	62.6	84.1	80.4	65.8	66.7
Workplace abuse summary score: mean (SD)	4.9 (4.0)	3.2 (3.9)	3.9 (3.8)	3.8 (4.1)	3.1 (3.8)	5.3 (4.3)	4.4 (3.5)	4.3 (4.6)	3.6 (3.8)
Reported at least 1 type of sexual harassment (%)	23.1	25.3	27.2	21.0	17.4	19.2	19.6	24.7	33.3
Sexual harassment summary score: mean (SD)	0.4 (0.9)	0.4 (0.9)	0.5 (1.0)	0.4 (0.9)	0.3 (0.9)	0.3 (0.6)	0.3 (0.6)	0.6 (1.4)	0.5 (0.8)
Racial discrimination: situations mentioned (%): 3+	28.4	46.4	35.4	28.9	13.9	10.4	8.8	39.7	22.2
Racial discrimination summary score: mean (SD)	1.9 (2.4)	3.0 (2.9)	2.4 (2.5)	1.8 (2.3)	1.0 (1.6)	0.8 (1.4)	0.8 (1.4)	2.5 (2.7)	1.7 (2.0)

Occupational hazards: high exposure (%)								
Dust	31.5	26.3	30.4	33.6	20.0	38.5	47.1	32.9	24.4
Chemicals	22.3	17.0	26.6	27.6	15.7	32.4	31.4	21.9	17.8
Noise	29.7	24.4	15.8	46.1	44.4	33.0	31.4	34.3	8.9
Ergonomic strain: shoulder	25.2	32.5	22.8	28.3	28.7	15.4	23.5	26.0	22.2
Ergonomic strain: neck	39.2	34.1	32.9	47.4	52.2	39.0	47.1	34.3	35.6
Ergonomic strain: back	33.2	29.9	22.2	46.7	40.0	36.3	40.2	28.8	33.3
Ergonomic strain: hand repetition	30.2	20.5	26.6	36.8	33.9	38.5	47.1	19.2	33.3
Ergonomic strain: heavy lifting	20.2	10.1	14.6	31.6	17.4	33.0	31.4	12.3	24.4
Combined occupational exposures: high exposure (%)								
0	16.1	22.1	19.6	6.6	12.2	11.0	11.8	19.2	28.9
1–2	35.4	36.7	34.8	32.9	33.0	38.5	32.4	38.4	31.1
3–4	26.6	21.4	25.3	32.2	33.0	30.2	32.4	23.3	24.4
5+	13.5	8.4	8.9	21.7	13.9	18.1	23.5	9.6	11.1
Job strain (%): high exposure	31.3	26.3	36.1	19.1	27.0	39.0	43.1	30.1	44.4
Additional covariates								
Response to unfair treatment								
Act/talk	42.1	41.6	48.7	37.5	34.8	41.2	54.9	43.8	48.9
Act/quiet	10.7	9.7	7.6	13.2	13.9	12.1	9.8	5.5	11.1
Accept/talk	23.6	24.7	27.2	21.7	20.9	23.6	22.6	26.0	26.7
Accept/quiet	17.6	19.8	12.0	19.1	23.5	20.9	9.8	19.2	11.1
Social desirability: mean (SD)	37.2 (31.8)	38.5 (30.7)	51.1 (34.1)	35.7 (31.0)	34.0 (30.0)	28.0 (29.9)	41.0 (32.5)	28.1 (29.4)	36.8 (30.0)
Body mass index (kg/m^2): mean (SD)	29.4 (6.1)	28.3 (4.6)	32.9 (8.5)	28.3 (4.9)	28.9 (5.8)	29.7 (6.0)	29.1 (7.2)	28.4 (4.4)	28.8 (6.4)
Hypertensive medication (%): yes	17.4	21.1	24.7	7.9	4.4	15.9	12.8	26.0	22.2
Smoking (%): current	22.7	16.2	16.5	21.1	15.7	32.4	50.0	19.2	17.8
Alcohol consumption in past 24 h (%): yes	10.2	8.1	8.2	5.9	1.7	27.5	11.8	6.9	0.0

Note: column percent are based on distributions including "missing" as a category; for detailed distributions, include percent missing, see Supplemental Table 1.

[a] Data on the 1202 workers include the 38 with data missing on race/ethnicity and the 29 with data missing on gender. The data shown by race/ethnicity and gender do not include: among the 480 total black workers, the 14 missing data on gender; among the 274 total Latino workers, the 7 missing data on gender; among the 289 white workers, the 5 missing data on gender; and among the 121 workers belonging to other racial/ethnic groups, the 3 missing data on gender.

Latinas to 28% among white men), with a similar proportion taking medication for hypertension.

Table 2 presents Pearson correlations between the social and occupational hazards. Statistically significant correlations ($p < 0.05$) were especially evident between: (a) racial discrimination and workplace abuse, among the workers of color; (b) racial discrimination and sexual harassment, among the women workers; and (c) workplace abuse and high exposure to occupational hazards, among all workers. Only among black men was the correlation significant between high exposure to occupational hazards and racial discrimination. In all cases, the significant correlations ranged between 0.2 and 0.5 (indicating an association, but not co-linearity), with correlations above 0.35 occurring only among workers of color.

Table 3 presents the analytic results for associations between SBP and the specified variables, overall and by race/ethnicity. Key findings for the total population are as follows. First, as expected, adjusting for covariates notably altered the bivariate results, including slightly increasing the difference in SBP among blacks compared to whites (by 1.3 points), further decreasing the SBP of women compared to men (by 2.5 points), and reducing the Latinos' initially statistically significant 5.3 point lower SBP compared to whites to 1.0, a non-significant difference. Covariates

significantly associated with elevated SBP in the multivariable analyses thus included: race/ethnicity (but only for black vs. white comparisons (3.5 mm Hg, 95% CI 0.3, 6.7)); gender (men higher than women by 8.8 mm Hg, 95% CI 5.7, 11.8); age (by year, 0.4 mm Hg, 95% CI 0.3, 0.5); BMI (by unit, 0.7, 95% CI 0.5, 0.8); hypertension medication (7.4 mm Hg, 95% CI 4.7, 10.0); and alcohol (5.1 mm Hg; 95% CI 1.9, 8.4). The interaction of sexual harassment and gender was statistically significant (higher for women, by 2.8 mm Hg, 95% CI 0.6, 5.0). A protective effect for workers in transportation compared to retail (by 3.4 mm Hg, 95% CI 0.5, 6.4) was also evident. Weaker associations with elevated SBP occurred for lower vs. higher education, and for responding to unfair treatment by accepting it as a fact of life and keeping it to oneself, vs. taking action and talking to others. No significant associations were evident for poverty, racial discrimination, workplace abuse, occupational hazards, social desirability, nativity, job control, co-worker gender, or co-worker race/ethnicity. Given the lack of a main effect for the occupational hazards, we did not model the interaction of social and occupational hazards on SBP.

Analyses stratified by race/ethnicity yielded similar results, albeit with three differences. First, the interaction of sexual harassment and gender was strongest in the white population (6.3 mm Hg, 95% CI 0.8, 11.8) but similarly

Table 2
Pearson correlations between the social and occupational hazards,[a] by racial/ethnic-gender group: ρ (*p*-value*), United for Health study (Boston, MA, 2003–2004)

Hazard	Race/ethnicity	Gender	Workplace abuse	Sexual harassment	Racial discrimination	High exposure to occupational hazards
Workplace abuse	Black	Men	1.00			
		Women	1.00			
	Latino	Men	1.00			
		Women	1.00			
	White	Men	1.00			
		Women	1.00			
	Additional race/ethnicity	Men	1.00			
		Women	1.00			
Sexual harassment	Black	Men	**0.17** (**<0.01**)	1.00		
		Women	**0.24** (**<0.01**)	1.00		
	Latino	Men	0.07 (0.43)	1.00		
		Women	**0.26 (0.01)**	1.00		
	White	Men	0.06 (0.43)	1.00		
		Women	0.12 (0. 23)	1.00		
	Additional race/ethnicity	Men	−0.07 (0.56)	1.00		
		Women	0.05 (0.77)	1.00		
Racial discrimination	Black	Men	**0.36** (**<0.01**)	0.07 (0.24)	1.00	
		Women	**0.38** (**<0.01**)	**0.28** (**<0.01**)	1.00	
	Latino	Men	**0.44** (**<0.01**)	0.10 (0.24)	1.00	
		Women	**0.49** (**<0.01**)	**0.39** (**<0.01**)	1.00	
	White	Men	**0.20 (0.01)**	**0.28** (**<0.01**)	1.00	
		Women	0.08 (0.44)	**0.35** (**<0.01**)	1.00	
	Additional race/ethnicity	Men	**0.52** (**<0.01**)	**0.32 (0.01)**	1.00	
		Women	**0.46** (**<0.01**)	**0.33 (0.03)**	1.00	
High exposure to occupational hazards	Black	Men	**0.26** (**<0.01**)	0.05 (0.40)	**0.35** (**<0.01**)	1.00
		Women	**0.19 (0.02)**	0.03 (0.75)	0.13 (0.12)	1.00
	Latino	Men	**0.27** (**<0.01**)	−0.02 (0.77)	**0.16 (0.04)**	1.00
		Women	**0.22 (0.02)**	0.00 (0.96)	0.13 (0.15)	1.00
	White	Men	**0.29** (**<0.01**)	−0.09 (0.24)	0.11 (0.16)	1.00
		Women	0.07 (0.51)	0.15 (0.14)	0.11 (0.27)	1.00
	Additional race/ethnicity	Men	**0.38** (**<0.01**)	0.04 (0.76)	0.22 (0.06)	1.00
		Women	0.30 (0.05)	0.10 (0.51)	0.24 (0.11)	1.00

*Correlations with $p < 0.05$ noted in bold.
[a] The correlations are based on the summary score for each of the social hazards and the composite high-exposure occupational hazard score.

Table 3
Association of systolic blood pressure with social and occupational hazards: United for Health study (Boston, MA, 2003–2004)

Variable		Systolic blood pressure (difference in mm Hg): $\hat{\beta}$ parameter estimate (95% confidence interval)									
		Total population (N = 1202)				White (N = 300)		Black (N = 493)		Latino (N = 286)	
		Bivariate		Multivariable[a]		Multivariable[a]		Multivariable[a]		Multivariable[a]	
Race/ethnicity	White (referent [ref])										
	Black	2.20	(−0.36, 4.76)	3.52	(0.34, 6.23)						
	Latino	−5.29	(−8.37, −2.22)	−1.05	(−4.57, 2.47)						
	Other	0.30	(−3.45, 4.04)	3.08	(−0.86, 7.03)						
Gender	Men (ref)										
	Women	−6.29	(−8.39, −4.19)	−8.75	(−11.78, −5.71)	−8.14	(−14.54, −1.74)	−8.76	(−13.91, −3.62)	−6.92	(−13.14, −0.70)
Poverty	No (≥100% poverty) (ref)										
	Yes (<100% poverty)	1.42	(−0.79, 3.63)	−0.68	(−2.81, 1.45)	0.80	(−3.64, 5.23)	−0.43	(−3.72, 2.87)	0.62	(−4.11, 5.35)
Education	<High school (HS)	1.36	(−1.18, 3.90)	1.83	(−0.56, 4.21)	5.53	(0.83, 10.22)	1.09	(−2.89, 5.07)	0.27	(−4.49, 5.02)
	≥HS, <4 yrs college (ref)										
	4+ yrs college	−0.43	(−3.94, 3.09)	0.12	(−3.10, 3.35)	−2.09	(−7.94, 3.75)	1.05	(−4.35, 6.44)	−0.08	(−7.75, 7.59)
Sexual harassment		0.22	(−0.91, 1.36)	−0.46	(−1.78, 0.87)	−5.35	(−8.86, −1.85)	0.20	(−1.87, 2.28)	1.14	(−2.22, 4.50)
Sexual harassment × gender				2.75	(0.56, 4.95)	6.30	(0.81, 11.79)	2.50	(−1.02, 6.01)	2.55	(−2.47, 7.57)
Racial discrimination	never (ref)										
	1–2 exposures	1.69	(−0.94, 4.32)	1.48	(−1.00, 3.97)	−0.61	(−4.85, 3.64)	3.82	(−0.63, 8.27)	1.49	(−3.56, 6.53)
	3+ exposures	0.16	(−2.25, 2.57)	−1.19	(−3.72, 1.35)	2.43	(−4.19, 9.06)	1.52	(−2.46, 5.50)	−3.74	(−9.73, 2.26)
Workplace abuse		−0.13	(−0.38, 0.12)	−0.16	(−0.47, 0.16)	0.16	(−0.39, 0.72)	−0.39	(−0.93, 0.16)	0.07	(−0.71, 0.86)
Workplace abuse × gender				0.24	(−0.26, 0.75)	−0.01	(−1.01, 0.99)	0.43	(−0.43, 1.30)	−0.32	(−1.47, 0.83)
Occupational hazards	0 exposures (ref)										
	1–2 exposures	−1.09	(−4.16, 1.97)	−0.76	(−3.63, 2.10)	1.74	(−4.67, 8.14)	−2.23	(−6.25, 1.79)	0.87	(−7.23, 8.96)
	3–4 exposures	−2.06	(−5.20, 1.09)	−0.77	(−3.77, 2.23)	1.32	(−5.15, 7.79)	−0.20	(−4.78, 4.38)	0.22	(−7.62, 8.06)
	5+ exposures	−2.58	(−6.30, 1.13)	−0.72	(−4.38, 2.93)	0.98	(−6.13, 8.09)	1.28	(−4.94, 7.50)	−4.00	(−12.53, 4.53)
Age		0.51	(0.41, 0.61)	0.42	(0.31, 0.53)	0.26	(0.05, 0.47)	0.58	(0.38, 0.79)	0.35	(0.09, 0.61)
Body mass index		0.63	(0.46, 0.80)	0.65	(0.49, 0.82)	0.88	(0.58, 1.18)	0.50	(0.24, 0.77)	0.58	(0.15, 1.01)
Hypertension medicine	No (ref)										
	Yes	11.65	(9.07, 14.24)	7.35	(4.71, 9.98)	3.26	(−1.92, 8.44)	10.26	(6.36, 14.15)	8.85	(0.52, 17.17)
Smoking	Never (ref)										
	Ex	3.21	(0.34, 6.07)	−1.48	(−4.28, 1.31)	0.10	(−5.00, 5.20)	−0.82	(−5.57, 3.93)	1.08	(−6.30, 8.47)
	Current	−0.38	(−2.89, 2.13)	−0.42	(−2.90, 2.07)	−0.97	(−4.91, 3.36)	0.51	(−4.04, 5.05)	−3.81	(−9.83, 2.21)
Alcohol	No (not in past 24 h) (ref)										
	Yes (in past 24 h)	4.82	(1.50, 8.14)	5.14	(1.91, 8.37)	4.02	(−0.36, 8.40)	2.18	(−3.99, 8.36)	7.60	(−2.09, 17.28)
Social desirability		0.01	(−0.02, 0.04)	0.01	(−0.03, 0.04)	0.08	(0.02, 0.14)	−0.03	(−0.08, 0.03)	0.03	(−0.04, 0.11)
Nativity	US born (ref)										
	Foreign born	−1.39	(−3.41, 0.63)	−0.29	(−2.19, 2.76)	0.06	(−5.81, 5.94)	2.24	(−2.10, 6.57)	−1.41	(−6.55, 3.73)
Job control	High (ref)										
	Low	0.36	(−1.90, 2.61)	0.91	(−1.20, 3.02)	−0.72	(−4.48, 3.05)	1.18	(−2.33, 4.69)	1.94	(−3.62, 7.49)
Workplace	Retail (ref)										
	Manufacturing	−1.61	(−4.16, 0.95)	−0.53	(−3.39, 2.34)	0.77	(−3.48, 5.02)	−3.16	(−13.91, 7.59)	2.96	(−2.70, 8.61)
	Transportation	4.11	(1.72, 6.49)	−3.43	(−6.37, −0.48)	2.52	(−3.57, 8.61)	−7.85	(−12.67, −3.03)	−1.22	(−10.25, 7.81)
Co-worker gender	About even (ref)										
	Mostly men	3.22	(0.94, 5.51)	1.06	(−1.35, 3.47)	0.90	(−3.54, 5.35)	1.48	(−2.27, 5.23)	0.45	(−5.81, 6.70)
	Mostly women	−1.54	(−4.77, 1.68)	−1.64	(−4.65, 1.36)	−4.20	(−10.10, 1.70)	−1.26	(−7.23, 4.70)	−1.29	(−6.89, 4.31)

(continued on next page)

Table 3 (continued)

Variable		Systolic blood pressure (difference in mm Hg); $\hat{\beta}$ parameter estimate (95% confidence interval)				
		Total population (N = 1202)		White (N = 300)	Black (N = 493)	Latino (N = 286)
		Bivariate	Multivariable[a]	Multivariable[a]	Multivariable[a]	Multivariable[a]
Co-worker race/ethnicity	Mostly same (ref)					
	Mostly different	1.58 (−0.93, 4.09)	0.81 (−1.56, 3.17)	−6.01 (−11.24, −0.78)	2.30 (−1.43, 6.03)	2.35 (−2.84, 7.54)
	About even	0.94 (−1.87, 3.75)	0.80 (−1.89, 3.49)	−6.71 (−12.31, −1.11)	3.16 (−1.31, 7.62)	2.40 (−3.28, 8.07)
Response to unfair treatment	Do something/talk (ref)					
	Do something/quiet	1.07 (−2.35, 4.48)	1.84 (−1.32, 4.99)	−0.32 (−6.44, 5.80)	1.28 (−4.54, 7.11)	4.52 (−2.13, 11.16)
	Accept/talk	1.12 (−1.44, 3.68)	−0.26 (−2.64, 2.12)	2.39 (−2.22, 7.00)	−2.70 (−6.62, 1.22)	2.25 (−3.38, 7.89)
	Accept/quiet	3.01 (0.07, 5.96)	2.34 (−0.33, 5.01)	3.54 (−1.67, 8.75)	0.55 (−4.04, 5.14)	2.64 (−3.21, 8.48)

[a] In multivariable models, all parameter estimates are mutually adjusted for all the other included variables.

weaker among the black (2.5 mm Hg, 95% CI −1.02, 6.01) and Latina (2.6 mm Hg, 95% CI −2.5, 7.8) populations. Second, the association of SBP with hypertension medication was much greater among the black and Latino compared to white workers (10.3 and 8.8 vs. 3.3 mm Hg). Third, only black workers exhibited the protective effect for transportation.

Discussion

Among the 1202 low-income multi-racial/ethnic working class participants in our cohort – of whom 40% lived below the US poverty line, 79% reported exposure to at least one social hazard and 82% at least one high-exposure occupational hazard – only sexual harassment, the least common social hazard, was associated with elevated SBP among the women workers. By contrast, no statistically significant associations were detectable between the other additional highly prevalent social and occupational hazards and SBP; we did, however, find suggestive evidence of an association between SBP and response to unfair treatment, implying that in a context of high exposure, differential susceptibility to the exposure matters. These results interestingly contrast to our prior findings for this same cohort, in which we found associations between self-reported experiences of racial discrimination and two other health outcomes: psychological distress and cigarette smoking (Krieger et al., 2005). Likely explanations for these contrasting findings include: (a) the differential etiologic periods and pathways involving somatic health, mental health, and health behaviors, and (b) the high prevalence of adverse exposures, limiting the ability to detect significant associations.

Study limitations

Before accepting our findings at face value, it is important to consider study limitations. The first is that despite our high response rate (72%), we ended up with a smaller analytic dataset than expected, due to management refusal to permit entry to certain workplaces where the union members worked, thereby reducing statistical power. Even so, we did find the expected strong and significant effects for major known risk factors for elevated blood pressure (e.g., gender, age, BMI, hypertension medication, and alcohol). Problems with high levels of missing data also meant we could not control for childhood socioeconomic position, known to influence adult blood pressure (Adair & Dahly, 2005; Lawlor & Smith, 2005), but we nevertheless were able to control for two related earlier life variables: country of birth and educational level (Krieger, Williams, & Moss, 1997). Our use of multiple imputation for the remaining key variables with lower levels of missing data in turn depends on Missing At Random (MAR) assumptions, i.e., that conditional on observed covariates, non-response to a specific question is not associated with the dependent variable (SBP). Although these assumptions cannot be tested in our dataset, our inclusion of multiple established correlates of SBP in our imputation model reduced the possibility of violating the MAR assumptions.

Misclassification of exposure could also have affected the study results. Despite our use of validated instruments,

data on the social and occupational hazards may have been more subject to misclassification than the data on key major risk factors (i.e., self-reported gender, age, and hypertensive medication, and measured BMI). Research on self-report vs. observed occupational hazards at work, for example, has found that workers can both over- and under-estimate their exposures (Ikin, Fritschi, & Sim, 2002; Spielholz, Silverstein, Morgan, Checkoway, & Kaufman, 2001). Other research indicates that racial discrimination and sexual harassment are more likely to be under- than over-reported, likely due to well-documented psychological tendencies to present oneself positively and to distance oneself cognitively from negative attributes (Hearn & Parkin, 2005; Hodson & Esses, 2002; Krieger, 1999). These types of misclassification would likely attenuate estimation of the association, if any existed, between the study exposures and the study outcome. Nevertheless, the prior associations we detected in this cohort between self-reported experiences of racial discrimination and both psychological distress and cigarette smoking imply that differences in etiologic pathways, and not simply sample size and misclassification, might be at issue.

Interpretation: implications of the "inverse trouble law" for studying health inequities

Sustained higher blood pressure does not occur instantaneously; it instead reflects the accumulation of exposures over time, from *in utero* onwards, which operate via anthropometric as well as psychosocial pathways (Adair & Dahly, 2005; Lawlor & Smith, 2005; Pickering, 2001). The cross-sectional association of SBP with concurrent social hazards is thus likely to differ from that of outcomes whose etiologic pathways are chiefly psychosocial and for which the etiologic period can be quite short, e.g., psychological distress and health behaviors.

Also germane are problems posed by the high and relatively limited range of exposure to both poverty and the social and occupational hazards, as well as the correlations between these hazards. As famously argued by Rose (1985), if everyone smokes, smoking would be unrelated to disease, even though it would be responsible for the rates of smoking-related disease; only factors affecting susceptibility to smoking would be detected. It is likely for this reason that we found no association between poverty and SBP – not because poverty is irrelevant, but rather because of the narrow income range among our cohort of low-income workers (Krieger, 2007). Further underscoring this point, whereas associations between job strain and hypertension have been found in studies examining workers across a wide range of occupations and income levels (Kasl, 1996; Pickering, 2001), job strain was unrelated to hypertension in a study of urban bus drivers, all of whom worked under high-stress conditions and had fairly similar incomes (Albright, Winkleby, Ragland, Fisher, & Syme, 1992). Research on racial discrimination and blood pressure among African Americans has likewise shown evidence of associations when exposure and socioeconomic position vary sufficiently (Krieger & Sidney, 1996; Paradies, 2006). By contrast, within a population with uniformly high levels of self-reported exposure to racial

discrimination, blood pressure was unrelated to racial discrimination but was associated with response to unfair treatment (Davis, Liu, Quarells, & Din-Dzietham, 2005). These results, like those of our study, imply the exposure mattered, because if exposure was irrelevant, susceptibility would be too.

Consequently, our finding of an association between sexual harassment and SBP may have been because sexual harassment was the only hazard whose overall prevalence was below 25%, with the effect strongest among the white women (who were a group less likely to be exposed also to racial discrimination). To our knowledge, this association has not previously been reported, despite the plausibility of sexual harassment (Richman et al., 1999) triggering the same kinds of physiological reactions to stress hypothesized to link experiences of racial discrimination to adverse cardiovascular outcomes (Krieger, 1999; Wyatt et al., 2003). Future research should test if our results can be replicated.

One final noteworthy finding was our observation of much smaller racial/ethnic disparity in blood pressure and hypertension than is typically reported. For example, a recent study based on the 2003–2004 US National Health and Nutrition Examination Study found that in analyses controlling for age, gender, education, and BMI, the odds for being hypertensive were 1.6 times higher among non-Hispanic blacks (95% CI 1.3, 2.0) and 0.7 times lower among Mexican Americans (95% CI 0.4, 1.1), compared to non-Hispanic whites (Ong, Cheung, Man, Lau, & Lam, 2007). Yet, in our study, we observed no racial/ethnic (or nativity) differences in blood pressure or hypertension when controlling for these same covariates, even as adjustment for these covariates, as expected, did reduce the observed racial/ethnic disparities. Potential explanations might include racial/ethnic patterns of job segregation, whereby the occupations represented in our study were what might be considered relatively good jobs for the black and Latino workers, but less good jobs, comparatively, for the white workers (Baillargeon, 2001; Baron & Dorsey, 2006). Combined with the "healthy worker" effect, the net result would be to reduce the likelihood of racial/ethnic disparities in blood pressure within the *United for Health* cohort. Even so, the much stronger association we detected between blood pressure and hypertensive medication among the black and Latino compared to white population attests to racial/ethnic disparities in blood pressure control, as also observed in the general population (Ong et al., 2007).

In summary, the "inverse hazard law" means trouble: for the people affected and for research on what causes their ills. As our data indicate, the highest correlations between the occupational and social hazards occurred among those sociodemographic groups with the least societal power and resources to contest them, an inverse relationship as perverse as the one that inspired Tudor Hart to delineate the "inverse care law". A crucial – and under-appreciated – corollary of the "inverse hazard law" is that detecting the health consequences of exposures disproportionately concentrated or uniquely occurring among populations most burdened by economic and social injustice and poor health is unlikely to be achieved by investigations that focus solely on these populations (Krieger,

2007). To understand health inequities, research instead is needed that contrasts exposures and health status population-wide, not just among those most inequitably exposed. That said, to do this research right, we need clear understanding of the exposures at issue. Studies like *United for Health* play a useful role in exposing the poorly documented independent and joint distributions of social and occupational hazards and poor health among understudied low-income working class populations – and hence what needs to be addressed to eliminate health inequities.

Appendix. Supplementary data

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.socscimed. 2008.09.039.

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