Does Harboring Hostility Hurt? Associations Between Hostility and

1998). As of 1990, COPD was the sixth most common cause of death worldwide; it has been projected to become the third leading cause of death by the year 2020 (Murray & Lopez, 1997). Several risk factors have been identified, most notably smoking (Senior & Anthonisen, 1998). Others include occupational exposures, indoor air pollution, childhood infections, prenatal exposures, airway reactivity, and genetic predisposition (Calverley & Walker, 2003; Senior & Anthonisen, 1998). To date, these risk factors do not adequately explain individual differences in who develops COPD.

Level of pulmonary function is used as a reliable risk marker of COPD (Senior & Anthonisen, 1998). Pulmonary function reaches a peak and then plateaus during young adulthood (Apostol et al.,

young adults, taking account of a range of potential confounders. Because maximal pulmonary capacity is attained in young adulthood and implicated in the subsequent developmental course of pulmonary function, young adulthood is an important period in which to study the relationships between hostility and pulmonary function. Exposures and behaviors during this time lay the groundwork for adult hostility and later life health outcomes (Houston & Vavak, 1991).

We hypothesized an inverse relationship between hostility and pulmonary function; that is, with higher hostility, we expected to see poorer pulmonary function. We expected this relationship to persist even when accounting for standard control variables such as age and height, as well as current socioeconomic status (SES), smoking status, and asthma. We hypothesized that the pattern of this association would be similar for all racial/ethnic and gender groups: Black women, White women, Black men, and White men. However, given previous reports that Blacks report more hostility than do Whites and that men report more hostility than do women, we expected absolute levels of hostility to vary across these groups.

Method

The CARDIA study was designed to assess evolution of cardiovascular risk factors in young adults. The human studies committees of the Brigham and Women's Hospital and each of the CARDIA sites approved the study. Details about study design and recruitment are available elsewhere (Friedman et al., 1988).

The study was conducted in four urban centers in the United States: Minneapolis, MN; Birmingham, AL; Chicago, IL; and Oakland, CA. The following participants were included: those who self-identified as Black or as White (U.S. Census Bureau category), with a permanent address in the target area, free of long-term disease or disability, and not pregnant at baseline. All data for the current analyses were from baseline (1985–1986). Fifty percent of eligible individuals, ages 18–30 years, took part in the study, resulting in a total sample of 5,115 participants (2,787 women and 2,328 men) approximately balanced within each center across gender, race/ethnicity, and SES. The inclusion criterion for the current analyses was having complete assessments on each measure used for this study, yielding a final sample of 4,629.

Measure

Hostility. Hostility was measured using the Cook–Medley Questionnaire, which is derived from a subset of items from the Minnesota Multiphasic Personality Inventory (Cook & Medley, 1954) and has been used in other epidemiological studies (Miller et al., 1996). Respondents answered true or false to each of the 50 items; scale scores could range from 0 to 50, with higher scores indicating greater hostility. We used hostility measured as a continuous variable and checked goodness-of-fit using mean pulmonary function according to quintiles of hostility. Hostility was deemed missing if one or more of the items used to compute it were missing. Hostility score was missing for 199 participants, who showed no statistically significant differences compared with the rest of the sample on any of the remaining predictor variables: age, height, current SES operationalized by current education, smoking, and asthma.

Other correlates of pulmonary function. Participant race/ethnicity, gender, age, current SES, smoking status, and asthma were ascertained by an interviewer-administered questionnaire. Current SES was indexed by number of years of education the participant had completed at the time of the interview. Following previous research (Apostol et al., 2002), asthma was defined in two levels. The first was self-reported asthma symptoms without asthma diagnosis, namely shortness of breath "when hurrying on the level or walking up a slight hill" in conjunction with wheezing (either apart from colds or most days or nights). The second asthma level was self-reported doctor or nurse diagnosis of asthma or the participant receiving asthma medication (medicine containers checked), regardless of symptoms. Height was measured without shoes to the nearest 0.5 cm.

Pulmonary function. Assessments of FEV_1 and FVC were obtained with a Collins Survey 8-L water-sealed spirometer and the Eagle II Microprocessor (Warren E. Collins, Inc., Braintree, MA) while participants were standing and wearing nose clips. FEV_1 is an indicator of upper airway pulmonary obstruction, and FVC is an indicator of total lung volume. Pulmonary function data were acceptable if at least three reproducible (within 10% of each other) tests of FEV_1 and FVC were performed out of up to five attempts, in accordance with American Thoracic Society standards for pulmonary function. Of the 5,115 CARDIA participants, 4,861 (95%) yielded acceptable data for FEV_1 and FVC. Percent predicted equations—derived from large population-based samples—are used to determine how close the observed data are to values expected for a given individual. Using the equations set forth by Hankinson and colleagues (Hankinson, Odencrantz, & Fedan, 1999), we calculated corresponding percent predicted values as the observed pulmonary function value multiplied by 100 and then divided by the individually predicted pulmonary function value. Although the percent predicted values are designed to remove associations with race/ethnicity, gender, age, and height, we found some residual confounding when we adjusted race/ethnicity- and gender-specific models for age, age squared, and height. We therefore included these variables in the reduced and full models (see below).

Analyse

The analyses are in two parts, performed using SAS Version 9. All analyses were run stratified by race/ethnicity and gender (Black women, White women, Black men, and White men). First, we computed descriptive statistics for hostility and pulmonary function, as well as other contributors to pulmonary function. Second, using multiple linear regression models, we examined the associations between hostility as a continuous variable and pulmonary function. Hostility was scaled in 1 standard deviation (*SD*) units to enhance interpretability. We ran two versions of each model: reduced (Model 1) and fully adjusted controlling for known potential confounders (Model 2). The reduced model used hostility as the independent variable of interest and used age, age squared, and height as covariates. The fully adjusted model used the variables in the reduced model and further included current SES, smoking status, and asthma as covariates. We evaluated effects on percent predicted FEV_1 and FVC in separate models.

Results

Table 1 presents descriptive statistics on hostility, pulmonary function, and contributors to pulmonary function, stratified by race/ethnicity and gender group. Using a 2 (race/ethnicity)

1 *SD* increase in hostility score. Although the levels of hostility in this sample are higher among Blacks than Whites and among men

Table 3
Parameter Estimates for Hostility and Covariates Predicting Pulmonary Function

Predictors	FEV_1 % predicted				FVC % predicted			
	Model 1		Model 2		Model 1		Model 2	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
				Black women (n = 1,293)				
Intercept	105.69***	17.43	104.43***	17.27	119.64***	17.06	119.29***	17.05
Hostility[a]	−1.10**	0.36	−0.83*	0.37	−0.85*	0.35	−0.78*	0.37
Age	0.95	1.25	1.01	1.26	−0.19	1.22	−0.07	1.24
Age squared	−0.02	0.03	−0.02	0.03	0.01	0.03	−0.00	0.03
Height	−0.11*	0.05	−0.10†	0.05	−0.10†	0.05	−0.09†	0.05
Current SES			−0.02	0.21			−0.10	0.21
Smoking status								
Ever smoker			1.08	1.28			2.48*	1.26
Current smoker			−1.03	0.82			0.39	0.81
Asthma								
Symptoms			−5.14***	1.51			−3.42*	1.49
Diagnosis or medications			−4.99***	1.21			−1.49	1.19
				White women (n = 1,187)				
Intercept	42.97*	17.57	45.57**	17.45	50.11**	18.03	51.82**	18.09
Hostility[a]	−0.69*	0.31	−0.63*	0.31	−0.47	0.31	−0.54†	0.32
Age	3.24*	1.28	3.12*	1.30	2.75*	1.32	2.66*	1.35
Age squared	−0.06*	0.03	−0.06*	0.03	−0.05†	0.03	−0.05†	0.03
Height	0.09†	0.05	0.09†	0.05	0.09†	0.05	0.10†	0.05
Current SES			−0.00	0.15			−0.10	0.16
Smoking status								
Ever smoker			1.78*	0.80			2.10*	0.83
Current smoker			−1.41†	0.76			0.74	0.79
Asthma								
Symptoms			−2.70†	1.45			−1.31	1.50
Diagnosis or medications			−4.27***	1.15			−0.12	1.20
				Black men (n = 1,054)				
Intercept	74.02***	19.93	77.10***	19.71	52.98**	19.08	54.99**	19.13
Hostility[a]	−0.93*	0.40	−0.74†	0.40	−0.71†	0.37	−0.66†	0.39
Age	2.09	1.45	1.86	1.45	3.70**	1.39	3.45*	1.41
Age squared	−0.03	0.03	−0.03	0.03	−0.06*	0.03	−0.06*	0.03
Height	−0.03	0.06	−0.05	0.06	−0.01	0.05	−0.02	0.05
Current SES			0.29	0.23			0.20	0.23
Smoking status								
Ever smoker			0.67	1.38			1.71	1.34
Current smoker			0.14	0.88			1.22	0.85
Asthma								
Symptoms			−2.87	2.41			−2.77	2.34
Diagnosis or medications			−7.13***	1.25			−0.75	1.21
				White men (n = 1,095)				
Intercept	75.80***	18.99	82.64***	18.93	50.86**	17.72	51.67**	17.87
Hostility[a]	−0.48	0.33	−0.31	0.33	−0.31	0.31	−0.24	0.31
Age	1.45	1.42	0.86	1.44	2.69*	1.33	2.67*	1.36
Age squared	−0.03	0.03	−0.02	0.03	−0.05†	0.03	−0.05†	0.03
Height	0.03	0.05	0.02	0.05	0.08†	0.04	0.07†	0.05
Current SES			0.17	0.15			0.02	0.14
Smoking status								
Ever smoker			1.85*	0.94			2.01*	0.89
Current smoker			−0.92	0.83			0.10	0.79
Asthma								
Symptoms			−2.76	1.80			−3.22†	1.70
Diagnosis or medications			−5.41***	1.18			−0.26	1.11

Note. Predictors in Model 1 are hostility, age, age squared, and height. Predictors in Model 2 include those in Model 1 and current SES, participant's smoking status (reference group is never smoker), and asthma (reference group is no asthma; comparison groups are [a] self-reported asthma symptoms without asthma diagnosis, namely shortness of breath "when hurrying on the level or walking up a slight hill" in conjunction with wheezing [either apart from colds or most days or nights] and [b] self-reported doctor or nurse diagnosis or taking of asthma medications [medicine containers checked], regardless of symptoms). FEV_1 = forced expiratory volume in 1 s; FVC = forced vitality capacity; SES = socioeconomic status.

[a] Hostility is scaled to represent the effect of a 1 standard deviation (*SD*) change on the outcome pulmonary function variable; thus, slopes are in units of percent predicted per standard deviation.

Standard deviations are listed in Table 1 and are approximately 8 units on the Cook–Medley scale for each race/ethnicity and gender group.

† $< .10$. * $< .05$. ** $< .01$. *** $< .001$.

We controlled for smoking; however, smoking itself may be on the causal pathway between hostility and pulmonary function. It could be that individuals with high levels of hostility are more likely to smoke as a form of coping with emotional distress (Siegler et al., 2003). Further, individuals higher on hostility may begin smoking at an earlier age and smoke in heavier doses. If smoking attenuates the relationship between hostility and pulmonary function, then controlling for smoking would result in an underestimate of the hostility–pulmonary function association. Given that our final models included smoking, this suggests that our analyses may be particularly conservative.

We cannot make causal claims, of course, with cross-sectional data. It could be that lower pulmonary function causes greater hostility. However, this seems unlikely. Although there is variability among participants in this sample, they have relatively normal levels of pulmonary function expected for this age range (cf. Wang et al., 2004). Another explanation for the findings may be that some third variable we did not account for contributes to both hostility and pulmonary function. For example, environmental toxins may both increase hostility and decrease pulmonary function. We were not able to investigate this hypothesis in the current study. Moreover, findings are consistent with other research that finds a role for hostility in the development of major health outcomes (Miller et al., 1996). Importantly, one advantage of using pulmonary function as an index of health is that it can be objectively and reliably measured over a wide range of values. Thus, it is less susceptible to the problem of restricted range, also referred to as disease-based spectrum bias (Miller et al., 1996), that has been found in research on the effects of hostility on congenital heart disease. That is, research on hostility and congenital heart disease that examines associations in high-risk populations may show attenuated relationships between hostility and disease because, by definition, there is less between-subjects variability in the outcome compared to a less restricted population.

This study is the first to make a detailed examination of the inverse link between hostility and pulmonary function; further, it examines this link in relation to race/ethnicity and gender. It appears that harboring hostility hurts, insofar as it is associated with lowered pulmonary function. More research is needed to establish whether hostility is prospectively associated with change in pulmonary function or is associated with pulmonary function at other points in the life course, especially during older adulthood. As well, the possible influence of social status on personality functioning and in turn pulmonary health deserves further exploration.

References

Apostol, G. G., Jacobs, D. R., Jr., Tsai, A. W., Crow, R. S., Williams, O. D., Townsend, M. C., & Beckett, W. S. (2002). Early life factors contribute to the decrease in lung function between ages 18 and 40: The Coronary Artery Risk Development in Young Adults Study. *American J. rnal f Re ira r and Cri ical Care Medicine, 166,* 166–172.

Atmaca, M., Kuloglu, M., Tezcan, E., Gecici, O., & Ustundag, B. (2002). Serum cholesterol and leptin levels in patients with borderline personality disorder. *Ne. r ch bi l g , 45,* 167–171.

Butovskaya, M. L., Boyko, E. Y., Selverova, N. B., & Ermakova, I. V. (2005). The hormonal basis of reconciliation in humans. *J. rnal f Ph i l gical An hr l g and A lied H. man Science, 24,* 333–337.

Calverley, P., & Walker, P. (2003). Chronic obstructive pulmonary disease. *Lance , 362,* 1053–1061.

Cohen, S., & Rodriguez, M. S. (1995). Pathways linking affective disturbances and physical disorders. *Heal h P ch l g , 14,* 374–380.

Cook, W., & Medley, D. (1954). Proposed Hostility and Pharisaic–Virtue scales for the MMPI. *J. rnal f A lied P ch l g , 38,* 414–418.

Cooper, M., Frone, M., Russell, M., & Mudar, P. (1995). Drinking to regulate positive and negative emotions: A motivational model of alcohol use. *J. rnal f Pe r nali and S cial P ch l g , 69,* 990–1005.

Crockett, A., Cranston, J., Moss, J., & Alpers, J. (2002). The impact of anxiety, depression and living alone in chronic obstructive pulmonary disease. *Q. ali f Life Re ea rch, 11,* 309–316.

Donaldson, G. C., Seemungal, T. A., Patel, I. S., Bhowmik, A., Wilkinson, T. M., Hurst, J. R., et al. (2005). Airway and systemic inflammation and decline in lung function in patients with COPD. *Che , 128,* 1995–2004.

Everson, S. A., Kauhanen, J., Kaplan, G. A, Goldberg, D. E., Julkunen, J., Tuomilehto, J., & Salonen, J. T. (1997). Hostility and increased risk of mortality and acute myocardial infarction: The mediating role of behavioral risk factors. *American J. rnal f E idemi l g , 146,* 142–152.

Friedman, G. D., Cutter, G. R., Donahue, R. P., Hughes, G. H., Hulley, S. B., Jacobs, D. R., Jr., et al. (1988). CARDIA: Study design, recruitment, and some characteristics of the examined subjects. *J. rnal f Clinical E idemi l g , 41,* 1105–1116.

Geronimus, A. T. (2001). Understanding and eliminating racial inequalities in women's health in the United States: The role of the weathering conceptual framework. *J. rnal f he American Medical W men' A cia i n, 56,* 133–136.

Hankinson, J. L., Odencrantz, J. R., & Fedan, K. B. (1999). Spirometric reference values from a sample of the general U.S. population. *American J. rnal f Re ira r and Cri ical Care Medicine, 159,* 179–187.

Higgins, M., Keller, -316(ggin1-316(gginu8(D.,)-3eller,)-232lercctherithe)-4tt(ggin1-L14.7

J., & Nissinen, A. (2001). Smoking cessation, decline in pulmonary function and total mortality: A 30 year follow up study among the Finnish cohorts of the Seven Countries Study. *Th ra , 56,* 703–707.

Pollitt, R. A., Daniel, M., Kaufman, J. S., Lynch, J. W., Salonen, J. T., & Kaplan, G. A. (2005). Mediation and modification of the association between helplessness, hostility, and progression of carotid atherosclerosis. *J . rnal f Beha i ral Medicine, 28,* 53–64.

Ritz, T., & Steptoe, A. (2000). Emotion and pulmonary function in asthma: Reactivity in the field and relationship with laboratory induction of emotion. *P ch ma ic Medicine, 62,* 808–815.

Scherwitz, L., Perkins, L., Chesney, M., & Hughes, G. (1991). Cook–Medley Hostility Scale and subsets: Relationship to demographic and psychosocial characteristics in young adults in the CARDIA study. *P ch ma ic Medicine, 53,* 36–49.

Senior, R. M., & Anthonisen, N. R. (1998). Chronic obstructive pulmonary disease (COPD). *Ame ican J . rnal f Re ira r and C ri ical Ca e Medicine, 157,* S139–S147.

Sidanius, J., & Pratto, F. (1999). *S cial d minance: An in e rg r . he r f cial hie ra ch and re i n.* New York: Cambridge University Press.

Siegler, I. C., Costa, P. T., Brummett, B. H., Helms, M. J., Barefoot, J. C.,