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Breathing Easy: A Prospective Study of Optimism and Pulmonary Function in the Normative Aging Study

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ABSTRACT

Although there is good evidence that emotions are associated with chronic airways obstruction, evidence for the influence of psychological factors on the level and decline of pulmonary function is sparse. Optimism has been linked to enhanced well-being, whereas pessimism has been identified as a risk factor for poor physical health. This investigation examines prospectively the effects of optimism versus pessimism on pulmonary function. Data are from the Veterans Administration Normative Aging Study, an ongoing cohort of older men. In 1986, 670 men completed the revised Minnesota Multiphasic Personality Inventory from which we derived the bipolar Revised Optimism–Pessimism Scale. During an average of 8 years of follow-up, an average of 3 pulmonary function exams were obtained. Men with a more optimistic explanatory style had significantly higher levels of forced expiratory volume in 1 sec (FEV₁) and forced vital capacity (both $p < .01$). Interactions between time and optimism suggested that rate of decline in FEV₁ over time was slower in men with a more optimistic explanatory style relative to men who were more pessimistic. These data are the first to link optimism with higher levels of pulmonary function and slower rate of pulmo-

nary function decline in older men, a protective effect that is independent of smoking.

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INTRODUCTION

The influence of emotion on the respiratory function is well known from everyday life. Sudden cessation of breathing in anxiety is referred to in such expressions as “breath-taking.”

Sighing is a common expression of despair. Crying is another complex expressive phenomenon in which the expiratory phase of respiration is involved. Because of this close correlation between emotional tension and the respiratory functions, it is probable that in most diseases of the respiratory organs, psychological factors play an important role (1, p. 133).

Although there is good evidence that emotions are associated with chronic airways obstruction, evidence implicating psychological factors in the growth and decline of pulmonary function is sparse (2). Recent epidemiological research has focused on identifying and characterizing determinants of level and rate of decline in pulmonary function (3,4). There has been a great deal of interest in identifying the “rapid decliner,” that is, the individual in whom pulmonary function declines at an accelerated rate. Longitudinal data have indicated that accelerated decline of pulmonary function may be a marker of an individual’s increased susceptibility to developing chronic obstructive pulmonary disease (COPD), the fourth leading cause of death and the third most important cause of morbidity in the United States (5,6). However, as yet there is a paucity of data about host factors that predispose certain individuals to decline at more rapid rates. Epidemiologic studies have suggested a role for smoking, occupational exposures, familial factors (genetic or possibly prenatal influences), childhood illnesses, and air pollution. These factors, however, account for only a portion of risk, suggesting that level and rate of decline in adult lung function is

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influenced by, as yet undefined, environmental, psychological, behavioral, or biologic factors.

A sense of optimism has been linked to enhanced well-being (7), whereas pessimism has been identified as a risk factor for poor physical health (8). An optimistic explanatory style is characterized by the belief that the future will be pleasant because one can control important outcomes (9). A pessimistic explanatory style has been linked to a sense of hopelessness, and it is marked by the view that problems are permanent and reflect one's shortcomings (10). Some investigators have argued that optimism and pessimism are related to the ways in which people routinely explain events in their lives (10). Individuals with a pessimistic explanatory style generally explain bad events as being caused by themselves (internal), because of chronic factors (stable), and generalizable to other situations (global), but good events are interpreted as external, transient, and coming from specific causes. By contrast, individuals with an optimistic explanatory style are likely to explain bad events as related to external, transient, and specific causes, and make opposite attributions for good events. Because pessimistic individuals expect bad events to occur consistently, they feel hopeless about changing the future (11). Despite the common belief that positive thinking (such as an optimistic explanatory style) may enhance health outcomes, few studies have tested this hypothesis in the context of pulmonary function. However, optimism and pessimism have been linked to a number of other health outcomes. For example, one study found a moderately protective effect of an optimistic explanatory style on all-cause mortality, although analyses in this study were not broken down by cause of death (12). In another study assessing pessimistic explanatory style at age 25, men (Harvard University graduates) had significantly poorer health or were more likely to have died when they were assessed 20 to 35 years later (13).

Expanding knowledge over the last 2 decades of important interactions among psychological, neural, endocrine, and immune processes that may be involved in inflammatory diseases has provided impetus for research on the role of psychological states in lung function. At the same time, evolving research demonstrates the interrelations between immune and inflammatory processes and chronic lung disease. Airway inflammation is recognized as a central process in the pathogenesis of COPD (14). Inflammatory changes in the airways are related to clinical airflow obstruction in patients with COPD (15) and to inflammatory cell infiltration in airway walls (16). The presence of proinflammatory cytokines in induced sputum have been demonstrated in COPD patients (17,18). Thus, emerging evidence that psychological factors influence immune and inflammatory processes (19) suggests their possible role in lung function decline and, ultimately, chronic lung disease.

Psychological factors may invoke physiologic responses, which in turn may influence immune processes thought to play a role in chronic airway inflammation in COPD. For example, in a study of pessimism and immune function, a pessimistic explanatory style was found to be associated with lowered immunocompetence, caused in part by increased percentages of T suppressor cells (CD8) relative to T helper cells (20). In this study,

more pessimistic older (but healthy) individuals were found to have lower CD4/CD8. Notably, increased numbers of T suppressor cells have been demonstrated in the lungs (16) and peripheral blood (21) in patients with COPD. Moreover, these studies have found decreased CD4/CD8 ratio to be associated with decreased lung function. In a study of quality of life in patients with chronic nonspecific lung disease, Kaptein and colleagues (22) found that optimism was associated with an increased postbronchodilator percentage predicted forced expiratory volume in 1 sec (FEV₁). Given the cross-sectional nature of these studies, such findings are suggestive but inconclusive in terms of whether optimism may actually influence lung function.

In the present investigation, we prospectively examined the effects of an optimistic versus pessimistic explanatory style on pulmonary function over time in the Normative Aging Study (NAS), a longitudinal study of 2,280 community-dwelling men. We hypothesized that relative to a pessimistic explanatory style, an optimistic one would be associated with higher levels of pulmonary function at baseline and a decelerated rate of decline in lung function. The prospective relation of these potential risk factors to level and rate of decline of pulmonary function has not been examined previously.

METHOD

The NAS is a longitudinal study of aging established by the Veterans Administration in 1961 (23). The study cohort consists of 2,280 community-dwelling men from the Greater Boston area, age 21 to 80 years at the time of entry. Volunteers were screened at entry, according to health criteria, and were free of any known chronic medical conditions at the start of follow-up (23). Because men with known chronic conditions (including asthma, chronic bronchitis, and chronic sinusitis) were excluded at baseline, the cohort originally comprised healthy individuals.

Assessment of Optimistic Versus Pessimistic Explanatory Style

We assessed optimism and pessimism in the NAS using the Revised Optimism–Pessimism Scale (PSM–R), developed and validated by Malinchoe, Olford, and Colligan (24). This bipolar scale measures explanatory style on a continuum from optimistic to pessimistic by using 263 items selected from the revised Minnesota Multiphasic Personality Inventory (MMPI–2). Based on explanatory style theory (25), the scale was developed using the Content Analysis of Verbatim Explanations (CAVE) technique, one of the two primary ways in which explanatory style may be measured (26). The CAVE technique may be used to evaluate the content of spontaneous written or verbal explanations to determine explanatory style, and it has been demonstrated to be valid and reliable (27,28). Based on an assumption that MMPI statements endorsed as true are equivalent to spontaneous discourse, expert raters first classified which MMPI items were appropriate for evaluating causal explanation (24). Subsequently, this pool of items was submitted to three independent raters who evaluated each item for scores on internality, stability, and globality. Finally, a composite weight of the three scores

was derived for each item, and the items (and their associated weights) were combined to derive optimism–pessimism scores, which were then normed. Further details about this scale have been described elsewhere (24). Scale scores in the present sample ranged from 14.9 to 75.7, and the internal consistency reliability of the scale was 0.86. Prior research has suggested that this scale has a high test–retest reliability of 0.90 (29). A high score on this scale indicates a pessimistic explanatory style, and a low score indicates an optimistic one. Optimism–pessimism scores were analyzed as a continuous variable. For some analyses, we also categorized optimism–pessimism scores into tertiles based on the distribution of scores in this cohort.

The MMPI–2 was administered by mail to all active cohort members ($N = 1,881$) in 1986 (30). A total of 1,550 men responded (82.4% response rate), of whom 1,472 provided complete and valid questionnaire data (95% of those responding). Comparison of men included in the study with those who were excluded (based on whether they completed the questionnaire) indicated that the nonresponders were somewhat younger and had better pulmonary function (higher levels of FEV₁) but did not differ on likelihood of smoking or level of education. Men were included in the present study if they had a pulmonary exam within 1 year of completing the MMPI–2 and had at least one follow-up exam. This resulted in a study population of 670 men.

Measurement of Other Pulmonary Function Risk Factors

Every 3 to 5 years, participants in the NAS are seen for a comprehensive examination that includes a medical history and physical examination, electrocardiogram, chest radiograph, blood and urine tests, and spirometry. Before the examination, participants are instructed to refrain from eating or drinking after midnight and to refrain from smoking after 8:00 p.m. of the previous night. Cigarette smoking status (current, former, never) is ascertained by a trained interviewer. Current smokers are defined as men who smoke one or more cigarettes per day. Weight and height are measured with participants wearing only socks and underpants.

Assessment of Pulmonary Function

At baseline, this study included measures of pulmonary function obtained within 1 year of the 1986 survey. Included participants were followed from 2 to 10.5 years, with a mean follow-up period of 8.24 years. The mean number of spirometric tests was 3.57, with a range from 2 to 5. Forced vital capacity (FVC) maneuvers were performed in the standing position without nose clip, using an 8-L water-filled spirometer (Warren E. Collins, Inc., Braintree, MA). Acceptability of the spirograms was judged according to American Thoracic Society standards (31). Spirometry was repeated, up to a maximum of eight spirograms, until at least three acceptable tests were obtained from each participant among which at least two were reproducible (FEV₁ and FVC within 5%). Predicted values for FEV₁ and FVC were calculated using regression equations relating each spirometric index to age and height among 215 asymptomatic lifetime nonsmokers in the NAS cohort.

Data Analysis

Data were examined using both analysis of variance (ANOVA) techniques and hierarchical linear modeling (HLM; also known as random effects modeling) using repeated measures analysis in the Statistical Analysis System (PROC MIXED; SAS Institute, Cary, NC). ANOVA techniques were used to give us a basic sense of whether rate of decline differed depending on level of optimism. For these ANOVA analyses, we included only those participants for whom we had information on four exams ($n = 457$). We first obtained the slope for FEV₁ and FVC over time (rate of change in pulmonary function) for each individual participant. We then calculated an average slope for each tertile of optimism–pessimism. Finally, using one-way ANOVA, we examined whether the mean slopes differed across the optimism–pessimism tertiles. Differences in the slopes may suggest differences in rate of change depending on levels of optimism. One problem with this analysis, however, is that it does not take advantage of the continuous nature of the measure of optimism–pessimism, and it does not allow us to make full use of the pulmonary function data available to us.

To take full advantage of the longitudinal richness of our data, we subsequently used HLM to estimate parameters for the effect of optimism on pulmonary function, using continuous variables for both indexes. HLM is a method that allows one to examine how people change over time and how that change might be related to other variables (32). Multiple observations at different times are formally viewed as nested within the individual. Each model has two levels: (a) specifying the model for individual time paths—a within-subject model, and (b) specifying the model to examine whether differences in amount of change over time can be accounted for by group membership—a between-subjects model (32). Once the variance in individual intercepts and slopes has been examined, a conditional model predicts intercept and slope terms using group as a predictor variable. In longitudinal multilevel models, missing values of the dependent variable do not pose any particular problem because it is not necessary for each individual to have the same number of observations from which to calculate change over time (for further description, see 33). Thus, data from the full sample of 670 participants were used. The HLM technique provides some additional advantages: (a) Models can control for the effects of potentially confounding independent variables, and (b) models can control for the relation between rate of decline in pulmonary function and baseline levels of pulmonary function. Thus, using this mixed model regression method, we were able to consider the effect of optimism on pulmonary function over time and account for the correlation between levels of pulmonary function measured in the same individual across exams (33). In addition, we were able to control for the effects of smoking within these models. The covariance structure for the pulmonary function data was specified using a compound symmetry model. A number of types of covariance structures were examined and compared (including first order autoregressive and unstructured), and selection of the best fitting model was done using Akaike's Information Criterion for non-nested models and

TABLE 1
Distribution of Pulmonary Function Risk Factors and Pulmonary Function, According to Level of Optimism—Pessimism at Baseline

	Level of Optimism—Pessimism		
	Optimistic	Neutral (Balanced)	Pessimistic
Range of Optimism—Pessimism Score	14.9–41.2	41.3–50.6	50.7–75.7
Risk factor			
Number	227	215	228
Age (years)	61.8	61.7	62.6
Height (in)	68.6	68.7	68.6
Current smokers (%)	12	9	17
Former smokers (%)	56	57	53
Mean pulmonary function			
FEV ₁	3200.4	3154.2	2986.9
SD ^a	605.7	648.8	662.8
FVC	4088.6	4033.0	3852.8
SD ^a	738.5	735.2	782.6

Note FEV₁ = forced expiratory volume in 1 sec; FVC = forced vital capacity
^aDifferences significant at $p < .01$.

maximum likelihood ratio tests for nested models. The compound symmetry covariance structure was found to be the best fit for the data (data not shown). This is a structure that specifies constant variance and constant covariance. Other research has also suggested that a compound symmetry covariance structure is adequate when there are a limited number of time points (34). As we were primarily interested in the between-group effects of these models (i.e., more vs. less optimistic people), we present only the data for fixed effects. To determine whether optimism influenced the rate of pulmonary function decline in the mixed regression models, we created interaction terms for optimism and time (all variables were centered). Because the dependent variable in this longitudinal analysis, change in pulmonary function, was not normally distributed, data were normalized by taking the logarithm of FEV₁ and FVC. This better satisfies the linearity and normality assumptions inherent in the models (35). These analyses report the effect of a 1-unit change in optimism on pulmonary function unless otherwise stated.

RESULTS

The mean age of the study population was 62 years ($SD = 6.9$ years; range = 45–89 years). The mean optimism–pessimism score was 46.4 ($SD = 10.8$; range = 14.9–75.7), which is close to the mean value ($M = 50$, $SD = 10$) in normative reference samples (24). Optimism–pessimism was normally distributed in this sample, although with a somewhat more limited range than has been found in normative samples. Tertiles were created based on the distribution of scores in this sample, with cut points 14.9 to 41.2, 41.3 to 50.6, and 50.7 to 75.7. Because the HLM models take advantage of data from 670 participants, we report the distribution of pulmonary function risk factors by level of optimism–pessimism for the full sample. Few differences emerged (see Table 1). We adjusted for all variables shown in Table 1 in the hierarchical linear models. Table 1 also

shows mean FEV₁ and FVC across tertiles of optimism–pessimism at baseline. The most optimistic men were significantly more likely to have high levels of FEV₁ and FVC at baseline relative to less optimistic men (all $ps < .01$). Examination of pulmonary function levels for both FEV₁ and FVC across tertiles of optimism over each exam also indicated that more optimistic individuals had higher levels of pulmonary function compared with more pessimistic individuals at every exam over the follow-up period (see Figure 1). We also examined whether optimism–pessimism was related to the punctuality of the medical assessment, to determine whether optimistic men came in sooner for their physical exam relative to more pessimistic men. If this were true, such differences might influence any differences we saw in pulmonary function. We found no relation between amount of time between exams and optimism–pessimism. However, to be conservative, we included this as a control variable in subsequent analyses.

Our primary research question concerned whether the rate of decline in pulmonary function was slower for more optimistic versus more pessimistic men. As a crude measure of this, we compared the slope means across levels of optimism–pes-

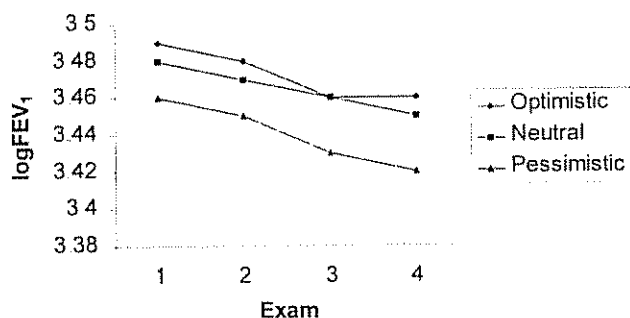


FIGURE 1 FEV₁ by Optimism–Pessimism over Time

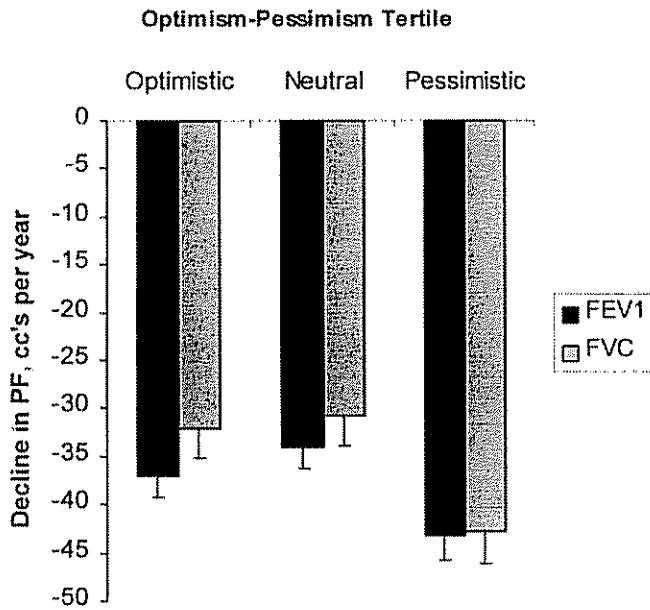


FIGURE 2 Mean slopes for FEV₁ and FVC across tertiles of Optimism-Pessimism

simism. For FEV₁, the overall *F* test was significant, $F(2, 454) = 3.95, p < .05$, suggesting that the mean slopes or rate of decline differed across optimism-pessimism tertiles. Post hoc contrast tests suggested that the middle and pessimistic tertiles were significantly different ($p < .01$), but the optimistic and pessimistic tertiles were marginally different ($p = .05$). The optimistic and middle tertiles were not significantly different from one another. These analyses suggest that more pessimistic people have a faster rate of decline in FEV₁ over time relative to more optimistic people. For example, pessimistic individuals declined approximately 43 cc/year versus more optimistic individuals who declined 37 cc. This difference in

rate of decline is comparable to that found between male current smokers versus never smokers in other studies (differences of 5–7 cc/year) (36). Results were somewhat stronger but otherwise identical for FVC, $F(2, 454) = 4.15, p < .05$. However, there was greater variance associated with the FVC estimates than with the FEV₁. Figure 2 illustrates these relations.

We subsequently used hierarchical linear models to examine both levels and rate of decline in pulmonary function according to optimism (using optimism-pessimism as a continuous variable). These analyses included men who had at least one follow-up pulmonary function exam after the baseline exam. The models include a main effect for optimism-pessimism score, which adjusts for differences in pulmonary function at the initial exam between men with a more optimistic or pessimistic explanatory style. There was an inverse association between the optimism-pessimism score (lower scores indicating an optimistic explanatory style) and pulmonary function, suggesting that men with a more optimistic explanatory style had significantly higher levels of FEV₁ ($\beta = -9.8E-4, p < .01$) and FVC ($\beta = -7.5E-4, p < .01$) when age, height, and time from exam were adjusted (see Model 1, Tables 2 and 3). Parameter estimates did not change substantially after adding smoking status to the models (see Model 2, Tables 2 and 3). As expected, age, height, years, and smoking were all significantly related to both FEV₁ and FVC.

Using an interaction term where both optimism and time were considered as continuous variables, we were able to test more formally whether more optimistic individuals evidenced a slower rate of decline than more pessimistic individuals. A significant interaction term ($\beta = -3.5E-5, p = .03$) suggested that FEV₁ declined faster in pessimists than in optimists, and this finding remained strong after controls for smoking were included in the models (Table 2). Findings were in a similar direction for FVC, although the interaction term did not reach statistical significance, perhaps because of the greater variance in these estimates, noted earlier (see Table 3).

TABLE 2
Fixed Effects of Optimism-Pessimism on FEV₁ at Baseline and Over Time

Effects	Model 1		Model 2	
	Coefficient	SE	Coefficient	SE
Initial status (intercept)	3.13**	0.096	3.17**	0.095
Optimism-Pessimism	-9.8E-4**	3.4E-4	-9.8E-4**	3.3E-4
Optimism-Pessimism × Years	-3.5E-5*	1.7E-5	-3.2E-5*	1.7E-5
Years ^a	-5.3E-3**	1.9E-4	-5.2E-3**	1.9E-4
Age	-4.5E-3**	5.3E-4	-4.8E-3**	5.3E-4
Height	9.9E-3**	1.2E-3	9.9E-3**	1.2E-3
Current smoker ^b			-2.9E-2**	8.1E-3
Former smoker ^c			-2.9E-2**	7.1E-3
N ^d	2391		2383	

Note. Lower optimism-pessimism numbers indicate higher levels of optimism. FEV₁ = forced expiratory volume in 1 sec.

^aYears = time between assessment of optimism-pessimism and pulmonary exams. ^bEffects are relative to never smokers (1 = current smoker, 0 = otherwise).

^cEffects are relative to never smokers (1 = former smoker, 0 = otherwise). ^dThese are the number of observations (multiple exams for each individual) used in the analysis. Eight observations were dropped in analyses for Model 2 because of missing information on covariates.

* $p < .05$, ** $p < .01$.

TABLE 3
Fixed Effects of Optimism–Pessimism on FVC at Baseline and Over Time

Effects	Model 1		Model 2	
	Coefficient	SE	Coefficient	SE
Initial status (intercept)	2.98**	0.08	3.02**	0.08
Optimism–Pessimism	-7.5E-4**	2.7E-4	-7.5E-4**	2.7E-4
Optimism–Pessimism × Years	-2.2E-5	1.7E-5	-2.0E-5	1.7E-5
Years ^a	-3.4E-3**	1.9E-4	-3.3E-3**	1.9E-4
Age	-3.7E-3**	4.2E-4	-3.9E-3**	4.2E-4
Height	1.3E-2**	1.0E-3	1.3E-2**	9.9E-4
Current smoker ^b			-1.9E-2**	6.8E-3
Former smoker ^c			-1.7E-2**	5.8E-3
N ^d	2391		2383	

Note. Lower optimism–pessimism numbers indicate higher levels of optimism. FEV = forced vital capacity.

^aYears = time between assessment of optimism–pessimism and pulmonary exams. ^bEffects are relative to never smokers (1 = current smoker, 0 = otherwise).

^cEffects are relative to never smokers (1 = former smoker, 0 = otherwise). ^dThese are the number of observations (multiple exams for each individual) used in the analysis. Eight observations were dropped in analyses for Model 2 because of missing information on covariates.

* $p < .05$. ** $p < .01$.

DISCUSSION

These prospective data are the first to link an optimistic explanatory style with higher levels of pulmonary function in older men. The protective effect appears independent of cigarette smoking, and findings are generally consistent across both FEV₁ and FVC outcomes, although findings for FEV₁ are somewhat stronger. At every exam, spanning a period of 10 years, more optimistic men had better lung function than more pessimistic men. Moreover, when accounting for baseline pulmonary function, men with higher levels of optimism exhibited a slower rate of pulmonary function decline over time, relative to more pessimistic men. Taken together, these findings add support to the notion that the effects of optimism on pulmonary function accumulate over time.

The PSM–R scale is designed to evaluate optimism and pessimism on a continuum so that with each 1-point decrease in the scale, individuals are considered to be more optimistic, with individuals scoring in the middle of the range exhibiting some combination of optimism and pessimism. Given the bipolar nature of the PSM–R scale and its monotonic relation with pulmonary function, our findings apply equally to decreased pulmonary function with greater pessimism. However, it is interesting to note that our sample was somewhat more optimistic than normative samples. For example, the mean score on optimism–pessimism was 46 relative to 50 in reference samples. More important, fully 66% of the sample scored below 50, the score that Malincho and colleagues (24) suggested indicates an optimistic explanatory style. Thus, failure to find strong differences between the “optimistic” and “neutral” tertiles is not surprising, given that both groups reported high levels of optimism. Moreover, the explanatory style measure may range from 0 to 100; however, the highest score in our sample was 75, suggesting a somewhat more limited range within this sample. In addition, it is also of note that the pessimistic group was the most heterogeneous with the greatest amount of variance for both FEV₁ and

FVC. It may be that we have simply identified a threshold at which pulmonary function is affected. However, it is also possible that we did not have sufficient range to detect dose-response effects and that our findings are even somewhat conservative.

We cannot fully rule out the possibility that there is a third factor leading to optimism, high pulmonary function, and decreased rates of pulmonary function decline. Or it may be that higher levels of pulmonary function lead to higher levels of optimism. However, several factors argue against the explanation that pulmonary function drives optimism. First, the longitudinal finding of an effect of optimism on rate of decline, controlling for initial levels of pulmonary function, suggests that level of optimism influences changes in lung function. Moreover, although we measure explanatory style at only a single time point in this study, the PSM–R was designed as a trait measure (24) and has been demonstrated to be stable across a long span of time (37). This suggests that the PSM–R measure obtained in 1986 is likely to be representative of explanatory style throughout our follow-up period.

Psychologists have suggested that optimism may allow individuals to mobilize highly effective coping resources (psychological, social, behavioral) when confronted with adversity (7,38). Thus, explanatory style affects an individual's ability to adapt to a myriad of environmental demands, and it appears to be stable across time and situations (8,37). This ability to adapt may influence both direct and indirect pathways by which optimism might influence pulmonary health, including direct physiological effects, the use of more active and problem-focused coping strategies, greater psychological well-being, and better health habits (11).

Direct pathways linking explanatory style to lung function may involve influences on underlying chronic inflammatory processes. Such processes may be regulated through complicated immune phenomena in which many cells (i.e., neutrophils, eosinophils, and T lymphocytes) and associated cytokines

play a role (39) Decline in lung function in COPD has been shown to be related to inflammatory changes in the airways and lung parenchyma (40). Thus, optimism and pessimism may more directly influence pulmonary outcomes through immune-function modulation. It is noteworthy, for example, that Kamen-Siegel, Rodin, Seligman, and Dwyer (20) demonstrated a reduced CD4/CD8 ratio associated with pessimistic explanatory style, and studies in patients with COPD have found a reduced CD4/CD8 ratio to be associated with lower levels of lung function and degree of airflow limitation (16,21)

Alternatively, explanatory style may influence the degree of stress to which individuals are exposed, which in turn influences neuroendocrine and immune function (19). Because optimistic individuals actively engage in planning and problem solving, they may experience fewer stressors, or they may have more resources with which to deal with stress. In fact, a number of studies have suggested that optimism is associated with decreased levels of stress and mood disturbance (41,42) and may retard the activation of stress-related immune processes, such as chronic hyperarousal or hyporesponsiveness or both, which may impact inflammatory airways disease (19) In a study on explanatory style, stress, and illness, a pessimistic explanatory style was associated with higher reporting of daily hassles over time (41). Several other studies have found evidence of a relation of optimism and pessimism with stress, immune function, and other health outcomes (7,8,43).

Explanatory style may also influence health through psychosocial pathways, by promoting a sense of control, positive social interactions, and other health-enhancing behaviors (11,44) For example, optimists tend to be less socially isolated and more likely to experience positive emotions (7,45) Fredrickson (46) proposed a "broaden and build" model that posits that positive emotions build individuals' resources in multiple domains, including physical, cognitive, and social. These resources may in turn promote health and buffer the effects of negative emotions. Moreover, optimism has been found in other studies to be associated with increased likelihood of engaging in health-promoting behaviors, such as avoiding smoking, exercising, and not drinking to excess, which in turn may influence pulmonary function (see 13, 47 for review). For more than 20 years, cigarette smoking has been recognized as the single most important risk factor for an abnormal rate of decline in pulmonary function in adult life (48,49). When compared with lifelong nonsmokers, cigarette smokers experience a two- to threefold increase in rate of decline in FEV₁ per year (50). Deleterious effects of cigarette smoking on lung function decline were evident in the present analyses. Notably, however, optimistic explanatory style remained an independent predictor of pulmonary function when controlling for cigarette smoking in these analyses. This suggests that additional and perhaps more direct mechanisms linking explanatory style and pulmonary function need to be considered

It is interesting to speculate whether examination of the association between emotion and pulmonary function might, at least in part, shed light on as yet unexplained socioeconomic disparities in lung health. That is, it has long been recognized

that both lung function measures and rates of respiratory symptoms show a gradient relation to socioeconomic status (SES), with lower lung function and higher rates of symptoms as SES decreases (51) These findings persist independent of smoking habit, age, and occupational status, leaving a significant proportion of this relation unexplained Explanatory style may arise partly as a consequence of one's environment; the social milieu in which one is embedded may influence the manner in which people explain causes of life events Partly through their influence on optimism or pessimism, factors such as poverty, unemployment, or work stress may increase the likelihood of impoverished lung health In other research we have found that individuals with less education were significantly more likely to be pessimistic, which lends some support to this hypothesis (52) However, our study findings are limited in that they pertain to White men and thus cannot be generalized to women or to non-White populations

A common question that arises after presenting evidence of a link between psychological factors and health outcomes revolves around what are appropriate next steps Should further research corroborate our findings of a link between optimism and pulmonary function, a program of intervention may be warranted (7,53) The heritability factor in optimism has been estimated at .25 (54,55), which suggests that optimism may be partially learned (29) Training programs that reliably increase optimism have been developed in which individuals are encouraged to engage in planning behaviors or to develop a more positive outlook on life (10,53,56) To date, smoking cessation and occupational changes have been thought to be the only interventions that may alter the course of lung function The results of this study suggest that modifying cognitive or emotional styles, such as optimistic or pessimistic explanatory style, may help to delay or attenuate potential pulmonary decline in individuals at risk of poor pulmonary health

REFERENCES

- (1) Alexander F: *Psychosomatic Medicine: Its Principles and Applications*. New York: Norton, 1950
- (2) Wright RJ, Rodriguez M, Cohen S: Review of psychosocial stress and asthma: An integrated biopsychosocial approach *Thorax* 1998; 53: 1066-1074
- (3) Camilli AE, Burrows B, Knudson RJ, Lyle SK, Lebowitz MD: Longitudinal changes in forced expiratory volume in one second in adults: Effects of smoking and smoking cessation *American Review of Respiratory Diseases* 1987; 135: 794-799
- (4) Burrows B, Lebowitz MD, Camilli A, Knudson RJ: Longitudinal changes in forced expiratory volume in one second in adults: Methodologic considerations and findings in healthy nonsmokers *American Review of Respiratory Diseases* 1986; 133: 974-980
- (5) Bone RC, Higgins MW, Hurd SS, Reynolds HY: NHLBI workshop summary: Research needs and opportunities related to respiratory health of women *American Review of Respiratory Diseases* 1992; 146: 528-535
- (6) Murphy SL: Deaths: Final data for 1998 *National Vital Statistics Reports* 2000; 48: 26
- (7) Peterson C, Bossio LM: *Health and Optimism*. New York: Free Press, 1991

- (8) Peterson C, Seligman MEP, Vaillant GE: Pessimistic explanatory style is a risk factor for physical illness: A thirty-five year longitudinal study. *Journal of Personality and Social Psychology* 1988, 55 23-27
- (9) Peterson C, Seligman MEP, Yurko KH, Martin LR, Friedman HS: Catastrophizing and untimely death. *Psychological Science* 1998, 9 127-130
- (10) Seligman MEP: *Learned Optimism*. New York: Knopf, 1991
- (11) Kamen LP, Seligman MEP: Explanatory style and health. *Current Psychology: Research and Reviews* 1987, 6 207-218
- (12) Maruta T, Colligan RC, Malinchoe M, Offord KP: Optimists vs pessimists: Survival rate among medical patients over a 30-year period. *Mayo Clinic Proceedings* 2000, 75 140-143
- (13) Peterson C: Explanatory style as a risk factor for illness. *Cognitive Therapy and Research* 1988, 12 117-130
- (14) Rich EA: The role of immune abnormalities and inflammation in causing and perpetuating COPD. In Cherniack NS (ed), *Chronic Obstructive Pulmonary Disease*. New York: Harcourt Brace Jovanovich, 1991. 235-248
- (15) Snider GL: Chronic obstructive pulmonary disease—A continuing challenge. *American Review of Respiratory Diseases* 1986, 133 942-944
- (16) O'Shaughnessy TC, Ansari TW, Barnes NC, Jeffrey PK: Inflammation in bronchial biopsies of subjects' chronic bronchitis: Inverse relationship of CD8+ T lymphocytes with FEV1. *American Journal of Respiratory and Critical Care Medicine* 1997, 155 852-857
- (17) Keatings VM, Jatakanon A, Wordsell YM, Barnes PJ: Effects of inhaled and oral glucocorticoids on inflammatory indices in asthma and COPD. *American Journal of Respiratory and Critical Care Medicine* 1997, 155 542-548
- (18) Keatings VM, Collins PD, Scott DM, Barnes PJ: Differences in interleukin-8 and tumor necrosis factor-alpha in induced sputum from patients with chronic obstructive pulmonary disease or asthma. *American Journal of Respiratory and Critical Care Medicine* 1996, 153 530-534
- (19) McEwen BS, Stellar E: Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine* 1993, 153 2093-2101
- (20) Kamen-Siegel L, Rodin J, Seligman MEP, Dwyer J: Explanatory style and cell-mediated immunity in elderly men and women. *Health Psychology* 1991, 10 229-235
- (21) de Jong JW, van der Belt-Gritter B, Koeter GH, Postma DS: Peripheral blood lymphocyte cell subsets in subjects with chronic obstructive pulmonary disease: Association with smoking, IgE, and lung function. *Respiratory Medicine* 1997, 91 67-76
- (22) Kaptein AA, Brand PL, Dekker FW, et al: Quality-of-life in a long-term multicentre trial in chronic nonspecific lung disease: Assessment at baseline. *European Respiratory Journal* 1993, 6 1479-1484
- (23) Bell B, Rose CI, Damon A: The Normative Aging Study: An interdisciplinary and longitudinal study of health and aging. *International Journal of Aging and Human Development* 1972, 3 5-17
- (24) Malinchoe M, Offord KP, Colligan RC: PSM-R: Revised optimism-pessimism scale for the MMPI-2 and MMPI. *Journal of Clinical Psychology* 1995, 51 205-214
- (25) Seligman MEP: *Helplessness: On Depression, Development, and Death*. San Francisco: Freeman, 1975
- (26) Schulman P, Castellon C, Seligman MEP: Assessing explanatory style: The content analysis of verbatim explanations and the Attributional Style Questionnaire. *Behavior Research and Therapy* 1989, 27 505-512
- (27) Peterson C, Luborsky L, Seligman MEP: Attributions and depressive mood shifts: A case study using the symptom-context method. *Journal of Abnormal Psychology* 1983, 92 96-103
- (28) Peterson C, Bettes BA, Seligman MEP: Depressive symptoms and unprompted causal attributions: Content analysis. *Behavior Research and Therapy* 1985, 23 379-382
- (29) Colligan RC, Offord KP, Malinchoe M, Schulman P, Seligman MEP: CAVEing the MMPI for an optimism-pessimism scale: Seligman's attributional model and the assessment of explanatory style. *Journal of Clinical Psychology* 1994, 50 71-95
- (30) Butcher JN, Dahlstrom WG, Graham JR, Tellegen A, Kaemmer B: *MMPI-2 Minnesota Multiphasic Personality Inventory-2 Manual for administration and scoring*. Minneapolis: University of Minnesota Press, 1989
- (31) Ferris BGJ: Epidemiology standardization project. *American Review of Respiratory Diseases* 1978, 118 1-88
- (32) Weinfurt KP: Repeated measures analyses: ANOVA, MANOVA, and HLM. In Grimm LG, Yarnold PR (eds), *Reading and Understanding More Multivariate Statistics*. Washington, DC: American Psychological Association, 2000. 317-363
- (33) Bijleveld CCJH, van der Kamp LJT, Mooijaart A, et al: *Longitudinal Data Analysis: Designs, Models, and Methods*. London: Sage, 1998
- (34) Bryk AS, Raudenbush SW: *Hierarchical Linear Models: Applications and Data Analysis Methods*. Newbury Park, CA: Sage, 1992
- (35) Ware JH, Weiss S: Statistical issues in longitudinal research on respiratory health. *American Journal of Respiratory and Critical Care Medicine* 1996, 154 S212-S216
- (36) Griffith KA, Sherrill DL, Siegel EM, et al: Predictors of loss of lung function in the elderly: The Cardiovascular Health Study. *American Journal of Respiratory and Critical Care Medicine* 2001, 163 61-68
- (37) Burns MO, Seligman MEP: Explanatory style across the life span: Evidence for stability over 52 years. *Journal of Personality and Social Psychology* 1989, 56 471-477
- (38) Peterson C, de Avila ME: Optimistic explanatory style and the perception of health problems. *Journal of Clinical Psychology* 1995, 51 128-132
- (39) Romagnani S: TH1 and TH2 subsets: Regulation of differentiation and the role in protection and immunopathology. *Internal Archives of Allergy and Immunology* 1992, 98 279-285
- (40) Saetta M, Finkelstein R, Cosio MG: Morphological and cellular basis for airflow limitations in smokers. *Thorax* 1998, 53 129-136
- (41) Dykema J, Bergbower K, Peterson C: Pessimistic explanatory style, stress, and illness. *Journal of Social and Clinical Psychology* 1995, 14 357-371
- (42) Peterson C, Seligman MEP: Causal explanations as a risk factor for depression: Theory and evidence. *Psychological Review* 1984, 91 347-374
- (43) Segerstrom SC, Taylor SE, Kemeny ME, Reed GM, Visscher BR: Causal attributions predict rate of immune decline in HIV-seropositive gay men. *Health Psychology* 1996, 15 485-493
- (44) Peterson C, Bossio LM: Optimism and physical well-being. In Chang EC (ed), *Optimism and Pessimism: Implications for Theory, Research, and Practice*. Washington DC: American Psychological Association, 2000. 127-146

- (45) Anderson CA, Arnoult LH: Attributional style and everyday problems in living: Depression, loneliness, and shyness. *Social Cognition* 1985, 3: 16-35
- (46) Fredrickson BL: What good are positive emotions? *Review of General Psychology* 1998, 2: 300-319
- (47) Scheier MF, Carver CS: Effects of optimism on psychological and physical well-being: Theoretical overview and empirical update. *Cognitive Therapy and Research* 1992, 16: 201-228
- (48) Fletcher C, Peto R, Tinker C, Speizer FE: *The Natural History of Chronic Bronchitis and Emphysema*. Oxford, England: Oxford University Press, 1976
- (49) U.S. Department of Health and Human Services: *The Health Consequences of Smoking - Chronic Obstructive Lung Disease. A Report of the Surgeon General*. Washington, DC: U.S. Government Printing Office, 1984
- (50) Hammond EC, Horn D: Smoking and death rates: Report on forty-four months of follow-up of 187,783 men. I. Total mortality. *Journal of the American Medical Association* 1958, 166: 1159
- (51) Steinberg M, Becklake MR: Socio-environmental factors and lung function. *South African Medical Journal* 1986, 70: 270-274
- (52) Kubzansky LD, Kawachi I, Sparrow D: Hope for the heart: Optimism, socioeconomic status, and coronary heart disease. New York Academy of Sciences Conference, Bethesda, MD, 1999
- (53) Beck AT, Rush AJ, Shaw BF, Emery G: *Cognitive Therapy of Depression*. New York: Guilford, 1979
- (54) Plomin R, Scheier MF, Bergeman CS, et al.: Optimism, pessimism, and mental health: A twin/adoption analysis. *Personality and Individual Differences* 1992, 13: 921-930
- (55) Gillham JE, Shatte AJ, Reivich KJ, Seligman ME: Optimism, pessimism, and explanatory style. In Chang EC (ed), *Optimism and Pessimism: Implications for Theory, Research, and Practice*. Washington, DC: American Psychological Association, 2000, 301-320
- (56) Hollon SD, Shelton RC, Loosen PT: Cognitive therapy and pharmacotherapy for depression. *Journal of Consulting and Clinical Psychology* 1991, 59: 88-99