

Personality Traits and Chronic Disease: Implications for Adult Personality Development

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Objective. Personality traits have been associated with chronic disease. Less is known about the longitudinal relation between personality and disease and whether chronic disease is associated with changes in personality.

Method. Participants from the Baltimore Longitudinal Study of Aging ($N = 2,008$) completed the Revised NEO Personality Inventory and a standard medical interview at regularly scheduled visits; the Charlson Comorbidity Index, a weighted sum of 19 serious diseases, was derived from this interview. Using data from 6,685 visits, we tested whether personality increased risk of disease and whether disease was associated with personality change.

Results. Measured concurrently, neuroticism and conscientiousness were associated with greater disease burden. The impulsiveness facet of neuroticism was the strongest predictor of developing disease across the follow-up period: For every standard deviation increase in impulsiveness, there was a 26% increased risk of developing disease and a 36% increased risk of getting more ill. Personality traits changed only modestly with disease: As participants developed chronic illnesses, they became more conservative (decreased openness).

Discussion. This research indicates that personality traits confer risk for disease, in part, through health-risk behaviors. These traits, however, were relatively resistant to the effect of serious disease.

Key Words: Disease burden—Illness—Openness—Personality change—Personality traits.

THE development of chronic disease is often a life-changing event, with significant physiological, social, and psychological consequences. As such, developing a severe illness and living with the burden of disease could both lead to long-term changes in self-perception. Although psychological factors, such as personality traits, have been implicated in the development of disease (Deary, Weiss, & Batty, 2010), whether getting sick and enduring chronic illness contributes to personality change is notably understudied in the personality development literature (but see Lüdtke, Roberts, Trautwein, & Nagy, 2011; Steunenberg, Twisk, Beekman, Deeg, & Kerkhof, 2005). To that end, this study addresses two major questions about the reciprocal nature of personality traits and illness burden: (a) Are personality traits associated with an increased risk of significant disease? and (b) As people develop significant illness, does their personality change? A secondary goal of this research is to examine whether the associations between personality and illness vary by demographic factors (age, sex, ethnicity, and education) and to test factors that may contribute to these associations. Using the five-factor model (FFM) of personality as an organizing framework, we address these questions in a large community sample of urban-dwelling adults.

Personality traits have long been known to contribute to disease states and disease progression. The early Type

A literature linked a hostile and aggressive personality to cardiovascular outcomes, such as heart attack and stroke (Williams et al., 1980). Later research identified interpersonal hostility as the driving characteristic of the association between Type A behavior and cardiovascular disease (MacDougall, Dembroski, Dimsdale, & Hackett, 1985). Recent work has demonstrated that other FFM traits, in addition to low agreeableness, are associated with illness. For example, higher neuroticism and lower conscientiousness have been found to predict aggregate morbidity (Chapman, Roberts, Lyness, & Duberstein, in press) and self-rated health (Turiano et al., 2012). The same traits are also associated with increased risk for specific diseases (Goodwin & Friedman, 2006), such as major depression (Kendler & Myers, 2010) and Alzheimer's disease (Terracciano et al., in press; Wilson, Schneider, Arnold, Bienias, & Bennett, 2007). Personality traits are also associated with disease progression. Among those living with HIV, for example, disease progression is slower for more open, extraverted, and conscientious individuals (Ironson, O'Cleirigh, Weiss, Schneiderman, & Costa, 2008).

The longitudinal relation between personality and disease is typically conceived of as personality as a predictor of the onset of disease. There is reason to suspect, however, that disease could likewise have an effect on personality development. A sudden change in personality or other psychological

factors may be one of the first indicators of acute disease. For example, a severe spike in high blood pressure is often detected because of sudden changes in personality and increased irritability (Mayo Clinic, 2012). Patients who are ultimately diagnosed with Lyme disease often present with irritability, depression, and attention deficits (Czupryna et al., 2011). Once the acute phase of the disease is treated, the psychological changes associated with the disease usually return to normal. In contrast, individuals suffering from chronic diseases may face permanent changes to their personality. For example, compared with before the onset of disease, patients with Alzheimer's are described as more neurotic and less extraverted and less conscientious by their caregivers (Pocnet, Rossier, Antonietti, & von Gunten, 2011).

In addition to the physiological changes that occur with chronic illness, the tremendous lifestyle changes that accompany disease may feed back on to how individuals perceive themselves. Chronic disease can have a widespread effect on nearly every aspect of the individual's life. Disease impairs mobility (Guralnik et al., 1993), places burden on loved ones (Emanuel, Fairclough, Slutsman, & Emanuel, 2000), and can be financially devastating (Wolff, Starfield, & Anderson, 2002). For someone suffering from a chronic disease, all of these factors, over time, may have lasting effects on personality. For example, dependence on a caregiver may lead the individual to perceive himself as less competent and less capable than when he/she was independent. Yet, whether disease burden and increases in burden are associated with changes in personality have yet to be explored.

This study uses a large, community-based sample to examine the cross-sectional and longitudinal relations between personality traits and illness burden. Given that high neuroticism and low conscientiousness are associated with specific diseases (Goodwin & Friedman, 2006), increases in comorbidity over time (Chapman et al., in press), declines in physical functioning (Jaconelli, Stephan, Canada, & Chapman, 2012), and premature mortality (Terracciano, Löckenhoff, Zonderman, Ferrucci, & Costa, 2008; Wilson, Mendes De Leon, Bienias, Evans, & Bennett, 2004), we expected these two traits to be associated with greater illness burden when measured concurrently and to confer greater risk for developing disease over time.

To date, the research on personality and illness burden has focused almost exclusively on domain-level associations. Yet the five broad domains include a number of relatively heterogeneous specific traits, or facets, that do not always follow their factor-level associations. These narrower traits tend to have greater predictive power than the broad domains (Paunonen, Haddock, Forsterling, & Keinonen, 2003), and facet-level associations can sometimes go in opposite directions, obscuring the effect at the broad domain level. Thus, to fully understand the relation between personality traits and illness burden, it is informative to examine these associations at the more specific facet level, in addition to the broad domains. A number of these more specific traits

have been linked to factors that increase risk for poor health. Impulsivity-related traits, for example, have been associated with obesity (Sutin, Ferrucci, Zonderman, & Terracciano, 2011; Van Hout, van Oudheusden, & van Heck, 2004), inflammation (Sutin, Terracciano, Deiana, Naitza, et al., 2010), and health-risk behaviors, such as cigarette smoking and other substance use (Mitchell, 1999; Turiano, Whiteman, Hampson, Roberts, & Mroczek, 2012). Depression-related scales have likewise been associated with indices of poor health (Eaton et al., 2008), whereas those who are less moody and more cheerful (Andersen et al., 2012) and those who have a disposition to be active tend to exercise more and tend to fare better as they age and live longer (Terracciano, Löckenhoff, Zonderman, et al., 2008). Thus, we expected the traits that reflect the different aspects of trait impulsivity (N5: impulsiveness, E5: excitement-seeking, low C5: self-discipline, and low C6: deliberation) and depression (N3: depression, N6: vulnerability) to be associated with greater illness burden measured concurrently and with greater risk for developing disease over time, whereas there should be a reduced risk for those high on E4: activity.

Given the dearth of research on illness burden and changes in personality, there is less evidence to draw from to make hypotheses for how disease will shape personality development. We can, however, draw from related literatures to make preliminary predictions about these relations. As described previously, inflammatory processes contribute to depression and illness can place limits on the individual's ability to socialize and be active. Further, experimental findings suggest that some people become more conservative when primed with thoughts of death (Nail, McGregor, Drinkwater, Steele, & Thompson, 2009). Therefore, we expected that the development of disease would be associated with increases in neuroticism and declines in extraversion and openness.

METHOD

Participants

Participants ($N = 2,008$) were drawn from the Baltimore Longitudinal Study of Aging (BLSA), an ongoing multidisciplinary study of normal aging implemented by the National Institute on Aging. Participants in the BLSA are generally healthy and educated ($M = 16.48$ years of education, $SD = 2.41$); the present sample is 71% white, 22% black, and 7% other ethnicity and 49% women. Age in this sample ranged from 19 to 96 ($M = 57.28$, $SD = 17.00$). Concurrent assessments of personality and disease burden were available for 6,685 visits (mean assessment per participant = 3.33, $SD = 2.41$, range 1–15); assessments took place between 1989 and 2010.

Personality

Personality traits were assessed with the Revised NEO Personality Inventory (NEO-PI-R), a comprehensive

measure of the FFM (Costa & McCrae, 1992). The NEO-PI-R consists of 240 items answered on a 5-point Likert format ranging from strongly disagree to strongly agree. The NEO-PI-R assesses 30 facets, six for each dimension of the FFM. Raw scores were standardized as T-scores ($M = 5$, $SD = 1$) using combined-sex norms reported in the manual. In the current sample (on the first assessment for each participant), the internal consistencies were 0.91, 0.87, 0.87, 0.88, and 0.92 for neuroticism, extraversion, openness, agreeableness, and conscientiousness, respectively. Available longitudinal data over intervals of 10 years indicate that stability coefficients for the five factors are approximately 0.80 (Terracciano, Costa Jr., & McCrae, 2006).

Illness Burden

Participants reported their medical history to a certified nurse practitioner at regularly scheduled visits. From this history, illness burden was assessed with the Charlson Comorbidity Index (CCI; Charlson, Pompei, Ales, & MacKenzie, 1987). The CCI is the weighted sum of 19 clinical conditions found to increase risk of mortality, including myocardial infarction, congestive heart failure, peripheral vascular disease, dementia, cerebrovascular disease, chronic pulmonary disease, connective tissue disease, ulcer disease, mild liver disease, diabetes, hemiplegia, moderate or severe renal disease, diabetes with end-organ damage, any tumor, leukemia, lymphoma, moderate or severe liver disease, metastatic solid tumor, and AIDS. We used an adapted version of the CCI, which defines each condition by the International Classification of Diseases, Ninth Revision diagnosis codes and combines leukemia and lymphoma with any tumor (Deyo, Cherkin, & Ciol, 1992). This version has been found to consistently predict mortality (Schneeweiss et al., 2004). In the present sample, at the first assessment of personality, the CCI had a mean of 0.53 ($SD = 0.95$, range 0–8 diseases), and at the most recent assessment, the CCI had a mean of 1.03 ($SD = 1.34$, range 0–10 diseases).

Covariates

In addition to demographic covariates (age, sex, ethnicity, and education), additional analyses control for behavioral and physiological markers known to increase risk of morbidity. Specifically, we controlled for smoking (i.e., smoked more than 100 cigarettes; 44%) and BMI ($M = 26.14$, $SD = 4.47$).

Statistical Overview

To test the concurrent associations between personality traits and illness burden, we ran a series of linear regressions predicting illness burden from each of the factors and facets, controlling for basic demographic characteristics (age, sex, ethnicity, and education). To evaluate the consistency of these associations, we ran the regressions twice, once on the baseline assessment and once on the most recent assessment; we

focus primarily on the findings that were significant across both time points. We then repeated the analyses controlling for factors that increased risk of disease (smoking and BMI) and used bootstrapping techniques (Preacher & Hayes, 2008) to test smoking and BMI as mediators of the personality–disease relations. To determine whether any of these associations varied by demographic factors, we used Aiken and West's (1991) method for interactions to test age, sex, ethnicity, and education as moderators of the relation between personality and illness burden. Again, we focus on the mediators and moderators that were consistent across both time points.

We examined the longitudinal relations between personality and illness burden in two ways. First, we used logistic regression to examine whether personality conferred risk for developing a disease ($n = 1,457$ with at least two assessments of both personality and disease). Among those who did not have any illness at baseline ($n = 972$), we contrasted those who developed at least one disease by follow-up ($n = 254$) with those who remained disease free ($n = 718$), and among those who had at least one illness at baseline ($n = 485$), we contrasted those who developed at least one additional disease by follow-up ($n = 165$) with those who had a stable illness burden across baseline and follow-up ($n = 320$). Again, we controlled for the basic demographic factors.

Second, we tested whether increases in disease burden were associated with change in personality using Hierarchical Linear Modeling (HLM; Raudenbush, Bryk, & Congdon, 2004). We selected BLSA visits that had both the personality and the illness assessment ($n = 6,685$). Normative changes in personality modeled with HLM in this sample have been described in detail elsewhere (Terracciano, McCrae, Brant, & Costa Jr., 2005). Based on this previous research, we fit a quadratic model at Level 1 to account for nonlinear changes in personality. To test whether developing significant disease was associated with changes in personality, we included illness burden and an interaction between illness burden and age as time-varying covariates. We controlled for the demographic factors at Level 2:

$$\text{Level 1: Personality} = \beta_0 + \beta_1 (\text{age}) + \beta_2 (\text{age}^2) + \beta_3 (\text{illness burden}) + \beta_4 (\text{illness burden} \times \text{age}) + r$$

$$\text{Level 2: } \beta_0 = \gamma_{00} + \gamma_{01} (\text{sex}) + \gamma_{02} (\text{ethnicity [black]}) + \gamma_{03} (\text{ethnicity [other]}) + \gamma_{04} (\text{education}) + u_0$$

$$\beta_1 = \gamma_{10} + \gamma_{11} (\text{sex}) + \gamma_{12} (\text{ethnicity [black]}) + \gamma_{13} (\text{ethnicity [other]}) + \gamma_{14} (\text{education}) + u_1$$

$$\beta_2 = \gamma_{20} + u_2$$

$$\beta_3 = \gamma_{30} + u_3$$

$$\beta_4 = \gamma_{40} + u_4$$

RESULTS

We first compared participants with only one assessment of personality and disease with participants who had more

than one assessment. Of the 551 participants with only one assessment of both personality and disease, 26% were deceased, 32% were still active in the study but had not returned yet for a second assessment, and 42% had withdrawn from the study. There was no difference between those who had one assessment versus at least two assessments in terms of sex ($\chi^2 = 3.04$, *ns*) or ethnicity ($\chi^2 = 1.62$, *ns*), but those who had only one assessment were younger (mean age = 53.20 vs. 58.82; $F(1, 2007) = 44.61$, $p < .01$) and slightly less educated (mean years of education = 16.22 vs. 16.58; $F(1, 2007) = 8.55$, $p < .01$). There was no difference in illness burden between those with one versus at least two assessments, $F(1, 2007) = 0.09$, *ns*. After accounting for differences in age and education, there was no difference on any of the five personality domains between those who had one versus at least two assessments (median $F(1, 2004) = 1.41$, range 0.01–3.42, all *ns*).

Concurrent Associations Between Personality and Illness Burden

The associations between personality traits and disease, controlling for age, sex, ethnicity, and education are shown in Table 1. At the domain level, participants higher in neuroticism and lower in conscientiousness had more serious, chronic diseases at both visits. At the facet level, those prone to depression (N3: depression) and those vulnerable to stress (N6: vulnerability) had more diseases, whereas those who lived a fast-paced life (E4: activity), those who preferred novelty (O4: action), those who were capable (C1: competence), and those who were highly disciplined (C5: self-discipline) had fewer diseases. All of these associations were also significant at the most recent visit except for C1: competence and C5: self-discipline. These associations were of similar magnitude, but because of the smaller sample size, both correlations were reduced to trends ($p < .10$). In addition, although not associated at baseline, N5: impulsiveness and C4: achievement striving were associated with comorbidity at follow-up. Controlling for BMI and smoking reduced some of these associations to trends ($p < .10$; see Table 1).

The slight reduction of the effect when BMI and smoking were included as covariates suggested that these factors could be mediators between personality and illness burden. And indeed, smoking was a significant mediator for several of these relations. At both baseline and follow-up, individuals higher in neuroticism and lower in conscientiousness had greater disease burden, in part, because they had a history of smoking (neuroticism: point estimates = 0.0006 [95% confidence interval, CI = 0.0002–0.0012] and 0.0013 [95% CI = 0.0005–0.0025], respectively, for first and most recent; conscientiousness: point estimates = –0.0006 [95% CI = –0.0013 to –0.0001] and –0.0014 [95% CI = –0.0026 to –0.0006], respectively, for first and most recent). Smoking similarly mediated, in part, the association between N3:

Table 1. Concurrent Associations Between Illness Burden and Personality Traits

Personality	Illness burden	
	Baseline	Follow-up
Neuroticism	0.05*	0.05*** ^a
Extraversion	–0.02	–0.03
Openness	–0.02	–0.03
Agreeableness	–0.01	–0.01
Conscientiousness	–0.04*** ^a	–0.06*** ^a
Facets		
N1: Anxiety	0.04	0.02
N2: Angry hostility	0.02	0.04
N3: Depression	0.06**	0.05*
N4: Self-consciousness	0.01	0.01
N5: Impulsivity	0.02	0.05*** ^b
N6: Vulnerability	0.07**	0.06*
E1: Warmth	0.00	0.01
E2: Gregariousness	0.00	–0.01
E3: Assertiveness	0.00	0.01
E4: Activity	–0.06**	–0.07*
E5: Excitement-seeking	–0.01	0.00
E6: Positive emotions	–0.04	–0.05
O1: Fantasy	0.00	0.00
O2: Aesthetics	0.01	0.01
O3: Feelings	0.00	–0.04
O4: Actions	–0.05*	–0.06*
O5: Ideas	–0.02	–0.03
O6: Values	–0.02	–0.02
A1: Trust	–0.02	–0.01
A2: Straightforwardness	0.00	–0.03
A3: Altruism	0.00	0.00
A4: Compliance	–0.01	–0.01
A5: Modesty	0.01	0.01
A6: Tender-mindedness	0.01	0.00
C1: Competence	–0.06**	–0.04
C2: Order	–0.02	–0.03
C3: Dutifulness	–0.04	–0.04
C4: Achievement striving	0.00	–0.06*
C5: Self-discipline	–0.05*** ^a	–0.05
C6: Deliberation	–0.01	–0.03

Notes. $N = 2,008$ at Time 1 and $N = 1,457$ at Time 2. Results are standardized β 's predicting illness burden from personality traits, controlling for age, sex, ethnicity, and education.

^aCorrelation reduced to a trend ($p < .10$) when controlling for smoking and BMI.

^bCorrelation reduced to nonsignificance when controlling for smoking and BMI.

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

depression (point estimates = 0.0003 [95% CI = 0.0001–0.0008] and 0.0006 [95% CI = 0.0001–0.0016], respectively, for first and most recent) and N6: vulnerability (point estimates = 0.0004 [95% CI = 0.0001–0.0010] and 0.0012 [95% CI = 0.0004–0.0024], respectively, for first and most recent) and illness burden. BMI did not mediate any of these relations. In addition, neither smoking nor BMI mediated the relation between E4: activity or O4: actions and illness burden.

We next tested for demographic moderators of these associations. Very few interactions were consistent across both the baseline and most recent assessments. Age moderated

the association between two facets of openness and illness burden at both time points. Among older participants, those who were receptive to their own feelings (O3: feelings) and those willing to try new things (O4: actions) had lower burden, whereas those who scored lower on these traits had higher illness burden; these associations were not apparent among younger participants ($\beta_{O3 \times \text{age}} = -0.09$ and -0.10 , both $ps < .05$, respectively, for first and most recent, and $\beta_{O4 \times \text{age}} = -0.08$ and -0.05 , both $ps < .05$, respectively, for first and most recent). In addition, sex moderated the association between O5: ideas and disease: Men who scored higher on O5: ideas had less illness burden than men who scored lower on this facet; there was no association between O5: ideas and burden among women ($\beta_{O5 \times \text{sex}} = -0.09$ and -0.08 , both $p < .05$, respectively, for first and most recent). Neither ethnicity nor education moderated the associations between personality and illness burden across both baseline and follow-up.

Longitudinal Relations Between Personality and Illness Burden

We first considered baseline personality as a predictor of disease at the most recent visit. Among those who were disease free at baseline, we tested whether baseline personality was associated with developing one or more chronic diseases by follow-up, controlling for the covariates. At the factor level, none of the traits was associated with the development of disease between baseline and follow-up. At the facet level, N5: impulsiveness was associated with an approximately 25% increased risk, whereas E6: positive emotions were associated with an approximately 20% decreased risk of developing a disease between baseline and the most recent assessment (see Table 2). In addition, among those already living with disease at baseline, we tested whether personality was associated with getting more ill by follow-up. At the factor level, extraversion was associated with an increased risk of more disease, whereas conscientiousness was associated with a decreased risk of developing more disease. At the facet level, those who were impulsive (N5: impulsiveness), warm (E1: warmth), and happy (E6: positive emotions) were at increased risk, whereas those who were orderly (C2: order), disciplined (C5: self-discipline), and deliberate (C6: deliberation) had a decreased risk of becoming more sick.

Finally, we examined whether getting sick was associated with personality change. Counter to our expectations, increases in illness burden were primarily unrelated to changes in personality. At the domain level, only one significant effect emerged for the effect of illness burden on the slope of personality (the illness burden by age interaction): Increases in disease were associated with declines in openness ($\gamma = -0.18$, $p < .05$). At the facet level, this association was due primarily to declines in O2: aesthetics ($\gamma = -0.18$, $p < .05$) and O3: feelings ($\gamma = -0.20$,

Table 2. Logistic Regressions Predicting Change in Illness Burden From Baseline Personality

Personality	Illness burden	
	Became sick	Got sicker
Neuroticism	1.17 (0.98–1.38)	1.06 (0.85–1.32)
Extraversion	0.92 (0.78–1.08)	1.26 (1.03–1.55)*
Openness	1.08 (0.92–1.27)	1.04 (0.86–1.26)
Agreeableness	0.99 (0.84–1.18)	1.08 (0.86–1.35)
Conscientiousness	0.95 (0.81–1.12)	0.77 (0.62–0.96)*
Facets		
N1: Anxiety	1.06 (0.90–1.25)	0.98 (0.79–1.22)
N2: Angry hostility	1.13 (0.96–1.32)	1.15 (0.92–1.45)
N3: Depression	1.12 (0.94–1.33)	0.97 (0.78–1.21)
N4: Self-consciousness	1.09 (0.92–1.28)	0.94 (0.77–1.15)
N5: Impulsivity	1.26 (1.06–1.49)**	1.36 (1.08–1.71)**
N6: Vulnerability	1.08 (0.92–1.28)	0.96 (0.77–1.21)
E1: Warmth	0.97 (0.82–1.14)	1.29 (1.05–1.60)*
E2: Gregariousness	0.96 (0.82–1.12)	1.11 (0.91–1.36)
E3: Assertiveness	1.02 (0.87–1.19)	1.17 (0.94–1.45)
E4: Activity	0.92 (0.79–1.08)	1.10 (0.90–1.36)
E5: Excitement-seeking	0.98 (0.83–1.17)	1.20 (0.96–1.51)
E6: Positive emotions	0.82 (0.70–0.97)*	1.24 (1.01–1.52)*
O1: Fantasy	1.10 (0.94–1.28)	1.10 (0.90–1.34)
O2: Aesthetics	1.18 (1.00–1.39)	0.94 (0.77–1.14)
O3: Feelings	1.05 (0.89–1.24)	0.96 (0.77–1.18)
O4: Actions	0.94 (0.81–1.10)	1.10 (0.89–1.34)
O5: Ideas	1.04 (0.88–1.23)	1.01 (0.83–1.24)
O6: Values	0.97 (0.83–1.13)	1.11 (0.91–1.36)
A1: Trust	1.01 (0.85–1.20)	1.18 (0.92–1.51)
A2: Straightforwardness	0.93 (0.79–1.10)	1.00 (0.80–1.25)
A3: Altruism	0.99 (0.83–1.17)	1.20 (0.96–1.49)
A4: Compliance	0.88 (0.75–1.03)	1.03 (0.84–1.26)
A5: Modesty	1.13 (0.97–1.32)	0.89 (0.72–1.10)
A6: Tender-mindedness	1.07 (0.91–1.25)	1.15 (0.93–1.44)
C1: Competence	1.06 (0.90–1.25)	0.91 (0.73–1.12)
C2: Order	0.98 (0.85–1.14)	0.77 (0.62–0.96)*
C3: Dutifulness	0.98 (0.83–1.17)	0.92 (0.73–1.16)
C4: Achievement striving	0.95 (0.81–1.11)	0.88 (0.71–1.08)
C5: Self-discipline	0.94 (0.81–1.10)	0.81 (0.67–0.98)*
C6: Deliberation	0.89 (0.76–1.05)	0.78 (0.63–0.97)*

Notes. $N = 1,457$. Analyses controlled for age, sex, ethnicity, education, and time interval.

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

$p < .05$). A trend did emerge for a decline in extraversion ($\gamma = -0.13$, $p = .09$) with increases in disease. A facet-level analysis revealed that this association was driven by E6: positive emotions: Participants who increased in disease declined in their tendency to experience positive emotions ($\gamma = -0.19$, $p < .05$).

DISCUSSION

This research tested the dynamic relation between serious, chronic disease and personality trait development in adulthood. Individuals high in neuroticism and low in conscientiousness were more likely to already be living with a serious chronic disease at baseline and facets of these traits were the strongest predictors of developing disease/additional diseases over time. Disease, however, was primarily

unrelated to changes in personality; illness burden was only associated with declines in openness and one facet of extraversion. Thus, personality traits were largely resistant to the effect of disease.

Vulnerability to Disease

Across different populations and different ways of measuring personality, higher neuroticism and lower conscientiousness are consistently associated with risk of premature mortality (Terracciano, Löckenhoff, Zonderman, et al., 2008; Wilson et al., 2004). Researchers now face the task of mapping the path from these traits to longevity. This pathway is likely mediated through both behavioral and physiological mechanisms. These traits, for example, have been linked to a number of behavioral risk factors that increase risk of morbidity, including smoking and illicit drug use (Terracciano, Löckenhoff, Crum, Bienvenu, & Costa, 2008), physical inactivity and function (Rhodes & Smith, 2006; Tolea et al., 2012), and diet (Möttus et al., 2012). And, indeed, in the current sample, smoking partially mediated several of the relations between personality and disease.

In addition to behavioral mechanisms, physiological factors likely contribute to the relation between personality and morbidity and mortality. Traits have been associated with a number of pre-disease states. Individuals high in neuroticism and low in conscientiousness, for example, tend to have higher levels of chronic inflammation (Chapman et al., 2011; Sutin, Terracciano, Deiana, Naitza, et al., 2010) and unhealthy lipoprotein profiles (Sutin, Terracciano, Deiana, Uda, et al., 2010), and tend to be overweight/obese (Roehling, Roehling, & Odland, 2008). Even when measured in childhood, these traits go on to predict inflammatory markers in midlife (Appleton et al., 2011) and self-reported health status nearly 30 years later (Kubzansky, Martin, & Buka, 2009). At the other end of the age spectrum, neuroticism and conscientiousness remain potent vulnerabilities to illness burden in older adulthood (Chapman et al., *in press*).

This research adds a more nuanced picture of the association between personality and vulnerability to disease. In addition to the five broad domains, we found that many facets were associated with illness burden and disease vulnerability. In particular, impulsivity-related traits, which have been previously implicated in obesity (Sutin et al., 2011), addiction (Bechara, 2005), and inflammation (Sutin et al., 2012), increased risk of disease over time. Although unrelated to illness burden at baseline, N5: impulsiveness was the strongest predictor of sickness over the follow-up period: Individuals who scored high on this facet had an almost 30% increased risk of becoming sick for the first time by follow-up and a nearly 40% increased risk of getting more ill between baseline and follow-up. The effect of N5: impulsiveness on illness burden may have a cumulative effect with age. With age, individuals tend to be less resilient to poor lifestyle choices; the damage to the body

accumulated by people high in impulsiveness may catch up with them as they get older.

A number of other facets were also related to illness burden. Both depression-related traits (N3: depression and N6: vulnerability) and activity-related traits (E4: activity and O4: actions) were associated with disease concurrently. Acute depression (Mezuk, Eaton, Albrecht, & Golden, 2008; Penninx et al., 2001) and stress (Everson-Rose & Lewis, 2005) are known psychological risk factors for chronic illnesses, such as diabetes and cardiovascular disease. The current findings suggest that a disposition toward depression and stress, in addition to acute states, also increases risk. In contrast to the depression-related traits, those who have a tendency to be active and those who like to try new things were less likely to be living with disease. Surprisingly, neither the activity-related nor the depression-related traits posed an additional vulnerability to disease over time. As such, these traits may pose the greatest risk for the development of disease rather than disease progression.

Teasing apart those who became sick for the first time and those who became more ill between baseline and follow-up revealed an interesting and unexpected pattern for extraversion. At the domain level, extraversion was unassociated with risk of becoming ill for the first time. Among the facets of extraversion, however, higher scores of E6: positive emotions were protective against developing a disease. This finding is consistent with the literature that a positive attitude is good for health and can help to stave off disease (Boehm & Kubzansky, 2012). Once the individual was already sick, however, extraversion surprisingly increased risk of becoming more ill. The protective benefits of extraversion, particularly positive emotions, do not continue once an initial illness has developed; introverted individuals seem to fair better with the onset of illness. The exuberance and high spirit that define extraversion may protect against disease, but the loss of energy that typically accompanies disease may hit extraverts particularly hard and precipitate more illness.

Personality Development

In contrast to our expectations, personality changed very little with disease. The modest effect of disease on personality change was surprising given the social and physiological consequences associated with illness. Our hypothesis that individuals may come to view themselves differently as they develop serious chronic diseases was largely unsupported. Indeed, we only found support for the effect of disease burden on changes in openness to experience: As participants got sick, they preferred more familiar environments and their emotional responses became more muted. These findings are consistent with the predictions of the selective optimization with compensation model (SOC; Baltes & Baltes, 1990) and socioemotional selectivity

theory (SST; Carstensen, 2006). According to SOC and SST, as time horizons shrink, goal selection and pursuit change to maximize remaining time (Lang, Rieckmann, & Baltes, 2002). Carstensen (2006) argues that when time is perceived as finite, people tend to invest less in gathering information, seeking novelty and learning new things. They focus instead on deepening the relationships and interests they deem important. Although typically associated with age, the processes associated with shrinking time horizons are apparent in other situations that limit time, such as developing a serious disease. In this study, as individuals developed disease, they increased in the tendency to be more conventional and less exploratory. With illness, people may be more inclined to stick to the tried and true rather than to explore new things. When they perceive their time as limited, individuals may explore less and invest more in the people and activities that matter most to them (Carstensen, 2006). The result of this process for personality traits may be declines in openness to experience.

In addition to openness, there was a trend for declines in extraversion. This trend was driven primarily by significant declines in E6: positive emotions. Individuals who score high in this facet tend to be cheerful and optimistic; disease reduces their tendency to be happy. As individuals near the end of their lives, there is a terminal decline in well-being with impending mortality (Gerstorf et al., 2008). Extraverts with sensory impairment are also less likely to experience positive affect (Wahl, Heyl, & Schilling, 2012). Similarly, the development of diseases with a high risk of mortality has a particularly devastating effect on the experience of positive emotions.

It is perhaps more remarkable, however, that personality changed relatively little with the development of serious disease. Personality traits tend to be stable in adulthood (Terracciano et al., 2005) and this stability suggests that the normative trajectory is resistant to change. Indeed, the effect of life events on change in personality, when found, is often modest (e.g., Lüdtke et al., 2011). It is of note that serious disease, which has significant physiological, psychological, and social consequences, did not lead to long-term changes in how individuals' described their personalities.

This research had a number of strengths, including a comprehensive measure of personality, a well-validated assessment of disease burden, and a large community-dwelling sample that had multiple assessments of both personality and disease burden. There are a number of limitations to this study that should be considered when evaluating the findings. Perhaps most important, the sample is not representative of the general population. BLSA participants tend to be educated and fairly healthy for their age. Disease may have a greater effect on personality for younger individuals who develop serious illnesses. Future research should expand to more diverse populations. This research also only focused on severe illnesses that are linked to a high risk of mortality. It would be interesting to examine other types

of illness and disease. For example, hearing and/or vision impairments with aging are usually not life threatening but can have significant implications for quality of life and maintenance of social relationships and daily routines. In addition, it would be interesting to examine the reciprocal relations between specific diseases (e.g., type 2 diabetes, dementia) and personality traits. Finally, the number of statistical tests may have increased risk of a Type I error. We chose to report the findings uncorrected because corrections for multiple comparisons can be overly conservative and increase risk of Type II errors. We do, however, emphasize the statistically significant findings that are consistent with theory and previous research on personality and health. In sum, this research indicates that although personality is associated with disease, significant illness burden is largely unrelated to personality development in adulthood.

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