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Do health behaviors explain the effect of neuroticism on mortality? Longitudinal findings from the VA Normative Aging Study

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ABSTRACT

Studies have shown that higher levels of neuroticism are associated with greater risk of mortality. Yet what accounts for this association? One major theoretical position holds that persons higher in neuroticism engage in poorer health behaviors, such as smoking and excessive drinking, thus leading to earlier death. We tested this hypothesis using 30-year mortality in 1788 men from the VA Normative Aging Study. Using proportional hazards (Cox) models we found that one health behavior, smoking, attenuated the effect of neuroticism on mortality by 40%. However, 60% remained unexplained, suggesting that the effects of other pathways (e.g., biological) also influence the relationship between neuroticism and mortality.

1. Introduction

Over the past two decades, personality traits have emerged as important predictors of all-cause mortality (Friedman et al., 1993; Maier & Smith, 1999). One of the best documented of these effects, along with conscientiousness, is the association between neuroticism and mortality. At least half a dozen studies have shown that people higher in neuroticism die sooner than those who are not as high (Abas, Hotopf, & Prince, 2002; Christensen et al., 2002; Denollet et al., 1996; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007; Schulz, Bookwala, Knapp, Scheier, & Williamson, 1996; Shipley, Weiss, Der, Taylor, & Deary, 2007; Wilson, Mendes de Leon, Bienas, Evans, & Bennett, 2004). Moreover, other studies have found that variables similar to neuroticism also predict mortality (e.g., MMPI Cynicism; Almada et al., 1991).

Over 20 years ago, Friedman and Booth-Kewley (1987) proposed that negative emotionality, or neuroticism, was predictive of general proneness to disease. The weight of the empirical evidence on neuroticism and mortality is consistent with this perspective (Hampson & Friedman, in press; Smith & Gallo, 2001; Suls & Bunde, 2005). Although a small number of studies have found no relationship between neuroticism and mortality (e.g., Huppert & Whittington, 1995; Iwasa et al., 2007; Taga, Friedman, & Martin, in press – although the latter did find a protective effect specifically among widowed men), and two have observed a protective effect (Korten et al., 1999; Weiss & Costa, 2005), the majority of findings have supported the conclusion that higher neuroticism confers a higher mortality risk. Moreover, high initial or average neuroticism paired with an increasing rate of neuroticism change further raises the mortality risk (Mroczek & Spiro, 2007). One of the key unanswered questions in this area is what explains the connection (Friedman, 2000; Roberts & Bogg, 2004; Roberts et al., 2007). This study took up the question, testing the possibility that health behaviors fully or partially account for the link between high neuroticism and shorter life.

1.1. Theories of neuroticism, health, and mortality

Recent theoretical and empirical work has identified several candidate hypotheses that might explain the connection between personality and health (Contrada, Cather, & O'Leary, 1999; Hampson & Friedman, in press; Hampson, Goldberg, Vogt, & Dubanoski, 2007; Rozanski, Blumenthal, & Kaplan, 1999; Segerstrom, 2000; Siegler et al., 2003; Smith, 2006; Smith & Spiro, 2002). One such hypothesis states that high levels of particular traits lead to worse (or better) health behaviors (Hampson, 2008; Roberts et al., 2007). Neuroticism, or negative emotionality, is predictive of the amount of negative affect (anxiety, depression) and perceived stress (Almada et al., 1991; Bolger & Schilling, 1991; Larsen & Ketelaar, 1991; Mroczek & Almeida, 2004; Suls, Green, & Hillis, 1998; Watson & Clark, 1994). Excessive negative emotions and stress may lead to poor health behaviors such as smoking or excessive drinking that in turn contribute to worse health (Friedman, 2000; Smith, 2006). In a sense, persons high in neuroticism may self-medicate with tobacco, alcohol, or drugs to alleviate their chronically high levels of negative affect and perceived stress (Eysenck, 1973,

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1991; Eysenck & Eaves, 1980). This self-medication, while perhaps giving psychological respite, has a physical health cost.

Theoretical models of neuroticism posit various mechanisms for why people high in this trait experience greater anxiety and potential self-medication through smoking or drinking. For example, Gray's Behavioral Inhibition System (BIS) is hypothesized to underlie the activation of negative emotion and behavioral withdrawal due to threat sensitivity (Gray, 1981, 1994).

However, some people have more sensitive Behavioral Inhibition Systems than others, rendering them more responsive to real or perceived threats – these persons are typically high-neuroticism (Gray, 1994). They may be prone to alleviating the unpleasantness that arises from BIS activation through smoking or excessive drinking. These detrimental health behaviors are potential "agents of respite." Similarly, Carver and Scheier's theory of self-regulation hypothesizes the existence of monitoring systems that yield feedback to the individual (Carver & Scheier, 1990, 1998). For example, if the pursuit of a goal is not going well, the monitoring or regulating systems will activate negative emotions, which in turn may lead to poor health behaviors that nevertheless alleviate the negative feelings.

However, neuroticism is not always hypothesized to be bad. As Tamir, Robinson, and Solberg (2006) have argued, neurotic individuals are often portrayed as victims of their frequent negative affect and maladaptive appraisals of threats. However, some neurotic individuals possess threat-identifying skills that are adaptive (Tamir et al., 2006). This study does not look at such mechanisms, yet it is important to acknowledge that neuroticism may not always be bad for health and health behaviors. Indeed, Friedman (2000) speculated that some persons high in neuroticism may enjoy better health, and presumably greater longevity, because of "neurotic vigilance" that leads to good health behaviors (see also Hampson & Friedman, in press; Taga et al., in press). However, at present there is no clear, agreed-upon way to distinguish between health and unhealthy neurotics. Thus, the present study confined itself to the more general finding in the extant literature that neuroticism is related to certain detrimental health behaviors, keeping open the possibility that future work may show our findings hold primarily for "unhealthy neurotics".

The goal of the current study was to test the extent to which detrimental health behaviors explain the neuroticism–mortality link. Health behaviors such as smoking are considered mediators in this type of model (Friedman, 2000; Hampson, 2008; Roberts et al., 2007). In this kind of conceptual framework, neuroticism leads to poorer health behaviors, which in turn damage health and increase mortality risk. As Hampson (2008) argues, health behaviors act as a bridge that joins personality traits to health and ultimately, to mortality or longevity. Because mediator mechanisms such as poor health behaviors do their damage over long periods of time, lifespan studies that follow individuals over many years or decades are ideal for testing these hypotheses (Hampson, 2008; Hampson & Friedman, in press). In addition, recent conceptual work has called for research on personality and health behaviors to go further and examine their downstream consequences on health and mortality (Hampson & Friedman, in press). It is important to note, however, that not all the personality-related poor health behaviors actually lead to worse health or mortality, as there are likely individual differences in how such behaviors influence health, in addition to dosage effects and interactions among health behaviors (Hampson & Friedman, in press).

Nonetheless, empirical work has supported the general notion that high neuroticism leads to problems in physical health (keeping in mind that there are likely exceptions). For example, people high in neuroticism are at higher risk of developing hypertension (Spiro, Aldwin, Ward, & Mroczek, 1995), as well as obesity and metabolic syndrome (Hampson & Friedman, in press). Thus, it is not surprising that many studies have found that high neuroticism is a risk factor for mortality (Abas et al., 2002; Christensen et al., 2002; Denollet et al., 1996; Schulz et al., 1996; Wilson et al., 2004). A recent meta-analysis has confirmed high neuroticism as detrimental for longevity (Roberts et al., 2007). It is important to bear in mind that some studies have found no relationship (Huppert & Whittington, 1995; Iwasa et al., 2007) and that two studies have identified high neuroticism as a protective factor (Korten et al., 1999; Weiss & Costa, 2005). However, these latter two studies have two factors in common. Both used samples where the age at entry was greater than 70, and both used relatively short follow-up periods for mortality (3–4 years for Korten et al., and 5 years for Weiss and Costa). In addition, the neuroticism effect was in the correct direction for women (it was a risk factor; not protective) in the Korten et al. (1999) study, but the hazard ratio was not significant. Additionally, among the Korten et al. men, only very high neuroticism (being in the upper quintile) conferred a protective effect. The great majority of the evidence rests behind the conclusion that high neuroticism elevates mortality risk.

1.2. Neuroticism and health behaviors

There is empirical evidence that neuroticism is associated with two key health behaviors that, in turn, are related to poor physical health and mortality. These are smoking and excessive alcohol use. Researchers have known for a long time that higher neuroticism is associated with cigarette smoking (Eysenck, 1973; Gilbert, 1995; Goodwin & Hamilton, 2002; Kirk, Whitfield, Pang, Heath, & Martin, 2001; Lerman et al., 2000). Persons high in neuroticism are more likely to smoke, tend to smoke more, and have greater difficulty quitting smoking (Almada et al., 1991; Rausch, Nichinson, Lamke, & Matloff, 1990). They also tend to use cigarettes and other tobacco products to self-medicate feelings of anxiety and worry, and to alleviate the negative affect that they experience much of the time (Audrain, Lerman, Gomez-Caminero, Boyd, & Orleans, 1998; Eysenck, 1991; Eysenck & Eaves, 1980; Lerman et al., 2000). Further, anxiety and smoking are correlated and likely have common biological underpinnings (Johnson et al., 2000). These relationships may exist in part because neuroticism and smoking may share some of the same genetic predispositions, especially polymorphisms that regulate serotonin and monoamine oxidase (Kirk et al., 2001; Lerman et al., 1999; Lesch et al., 1996). All in all, the fact that neuroticism and smoking are positively associated is well established.

The association between neuroticism and alcohol abuse is not as well established. Even so, several studies have documented that higher neuroticism is related to alcohol abuse and dependence (Almada et al., 1991; Grekin, Sher, & Wood, 2006; Larkins & Sher, 2006; Read & O'Connor, 2006) as well as greater negative consequences from drinking (Fischer, Smith, Annus, & Hendricks, 2007). Presumably, the same theoretical processes of self-medication and alleviation of negative emotion underlie the neuroticism–drinking association.

We tested whether smoking or drinking (or both) partially or fully explained the association between neuroticism and mortality. As Hampson and Friedman (in press) point out, the main competitor hypothesis to health behavior models of personality and health are biological explanations that argue for physiological mechanisms (e.g., Wiebe & Smith, 1997). Further, Hampson, Goldberg, Vogt, and Dubanoski (2006) have argued that the effect of neuroticism on health and mortality might be better suited to biological or physiological explanations. It may be the case that neuroticism works through both health behaviors as well as physiological mechanisms (e.g., higher production of cortisol or inflammatory cytokines, e.g., Segerstrom, 2000; Segerstrom & Miller, 2004). Thus, it is more realistic to expect that smoking and drinking will partially, but not fully, explain the neuroticism–mortality relationship.

1.3. Present study

We tested the above questions in the present study, conducting a survival analysis (proportional hazards model), and drawing upon measurements of neuroticism made in 1975 to predict time-to-death over 30 years in a sample of older men. Subsequently, we added health behaviors (smoking and drinking) to the model to determine if they explained, either fully or partially, the association between neuroticism and mortality. We hypothesized that health behaviors would at least partially explain the mortality risk of high neuroticism. In essence, we tested a mediator model. Mediator variables can explain why a predictor is related to an outcome, and in the present study we reasoned that health behaviors were a key mediator, or conduit, by which neuroticism influences mortality. In a few prior studies of neuroticism and mortality, health behaviors such as smoking have been controlled. However, none has explicitly compared the effects (hazard ratios) of neuroticism with and without such health behaviors in the model, to ascertain the size of any potential mediation. Moreover, no study of neuroticism and mortality has employed a 30-year follow-up period.

2. Method

2.1. Sample

Data were from the VA Normative Aging Study (NAS), a longitudinal investigation of aging in men founded at the Boston VA Outpatient Clinic in 1963 (Bosse', Ekerdt, & Silbert, 1984). Over 6000 men were screened for the absence of serious physical or mental illness between 1961 and 1970 to assemble a panel of 2280 initially healthy participants. The present study included the 1788 men for whom we had neuroticism measurements from 1975 as well as assessments of health behaviors from the same approximate period (mid-1970s). In 1975, the beginning of the period in which we tracked survival, the age range for the 1788 men was 23–89 (M = 51.15, SD = 9.34).

2.2. Measures

2.2.1. Neuroticism

Neuroticism was assessed in 1975 via the EPI-Q (Floderus, 1974), a short version of the Eysenck Personality Inventory (Eysenck & Eysenck, 1968). EPI-Q neuroticism is assessed by nine dichotomous items that yield scores ranging from 0 to 9. The EPI-Q has been used primarily in Swedish twin studies (Floderus-Myrhed, Pedersen, & Rasmuson, 1980), and has demonstrated good construct validity (Levenson, Aldwin, Bosse, & Spiro, 1988; Mroczek & Spiro, 2003; Mroczek, Spiro, Aldwin, Ozer and Bosse', 1993). McCrae, Costa, and Bosse (1978) successfully retrieved a clear neuroticism component from the EPI-Q using principal components analysis with Varimax rotation. Mean neuroticism in 1975 was 3.17 (SD = 2.39). The internal consistency (Cronbach alpha) was 0.77.

2.2.2. Health behaviors

Both health behaviors (smoking and drinking) were measured separately from the survey that assessed neuroticism, thus reducing potential response sets that could arise among scales on the same questionnaire. Yet, smoking and drinking were indexed within two years of the neuroticism assessments in most cases, so there still may be some small inflation of the correlations due to close measurement. Smoking was measured via the Cornell Medical Index (CMI), using the assessment closest in time to the 1975 neuroticism measurement. NAS men are given the CMI once per 3-year cycle, so the time frame was from 1972 to 1978, with smoking assessed for the majority of participants within two years of the neuroticism measurement (1973–1977). The CMI asks whether one smokes a pack of cigarettes per day (20 cigarettes) or not. Thus, smoking was represented by a dichotomous variable that indicated whether a participant was a pack-a-day smoker or not in 1975. The mean of this dichotomous variable was 0.17 (SD = 0.38) meaning that 17% of the sample smoked a pack (20 cigarettes) per day or more in 1975. Drinking was assessed in a 1973 survey via a question asking if they had more than two drinks per day, each day. The mean was 0.25 (SD = 0.43), meaning 25% had more than two drinks per day.

2.2.3. Mortality in the NAS

Vital status of NAS participants is monitored by periodic mailings, and when notified, death certificates are obtained and coded for cause of death. Of the 1788 participants who were alive in 1975 and had valid measurements for neuroticism, smoking, and drinking, 665 died during the 30 years of follow-up from 1975 to 2005. The vast majority of deaths were due to heart disease or cancer. Very few were due to accidents and almost none to suicide. We used exact date of death to calculate survival time as the interval from the 1975 survey until death. For censored observations (survivors), we used an end date of December 31, 2005. Among the 665 decedents, survival time ranged from 1 to 29 years. Mean survival time for the decedents was 17.40 years (SD = 7.90).

2.2.4. Data analysis

As noted above, we used exact date of death to calculate survival time. Due to the large number of survivors, we had many right-censored observations, but proportional hazards models (Cox, 1972) are ideal for this kind of data structure in which the baseline is general and the shape of the hazard function is unconstrained. We have used these models in the past, utilizing personality traits to predict onset of hypertension (Spiro et al., 1995) as well as change in traits to predict mortality (Mroczek & Spiro, 2007). Our age-adjusted hazard rate models were expressed as follows:

$$h(t_{ij}) = h_0(t_j)e^{[\beta_1 \mathrm{age}_{1i} + \beta_2 \mathrm{neuroticism}_{2i}]} \quad (1)$$

$$h(t_{ij}) = h_0(t_j)e^{[\beta_1 \mathrm{age}_{1i} + \beta_2 \mathrm{neuroticism}_{2i} + \beta_3 \mathrm{health\ behaviors}_{3i}]} \quad (2)$$

In the above equations, $h(t_{ij})$ is an individual i's risk of dying (or hazard: h) at time t (see Singer & Willett, 2003, p. 512). The term $h_0(t_j)$ represents the general baseline hazard function. It is the risk of dying when all predictors are set to 0. The terms in the exponent, $\beta_1(\mathrm{age}_{1i})$, $\beta_2(\mathrm{neuroticism}_{2i})$, and $\beta_3(\mathrm{health\ behaviors}_{3i})$, are the effects of these variables on risk of dying. Note that the first model tests the effect of neuroticism on mortality, whereas the second introduces the health behaviors (smoking and drinking) to determine whether the hazard ratio for neuroticism declines or is rendered non-significant. In the tables below, we report exponentiated β coefficients (as shown in the formulae), also known as hazard ratios.

In a step of analysis prior to the Cox models, we ran bivariate correlations between all variables in the model, to see if neuroticism was related to health behaviors in a simple way. We also tested the quadratic effect of neuroticism, and tested interactions between neuroticism and the health behaviors.

3. Results

Table 1 shows the simple, zero-order correlations among age, neuroticism, smoking and drinking along with means and standard deviations. Note that neuroticism is positively correlated with both drinking and smoking. Drinking and smoking are significantly

Table 1
Correlations among predictor variables ($N = 1.788$).

Variables	1	2	3	4
1. Age	–			
2. Neuroticism	−0.06***	–		
3. Drinking	−0.08***	0.10***	–	
4. Smoking	−0.21***	0.14**	0.19***	–
Mean	51.15	3.17	0.25	0.17
SD	9.34	2.39	0.43	0.38

** $p < 0.01$.
*** $p < 0.001$.

correlated as well. A first step in testing mediators is establishing that the predictor is associated with the mediators. In this case, neuroticism is related to both.

3.1. Neuroticism, health behaviors and mortality

Table 2 displays three models of neuroticism and mortality. As noted earlier, Tables 2 and 3 report exponentiated β coefficients, as shown in formulae 1 and 2. These exponentiated coefficients are known as hazard ratios (or risk ratios). Model 1 shows that neuroticism in 1975 significantly predicts mortality. The hazard ratio is 1.05 (95% CI: 1.01–1.08) meaning that a 1-point increase on the EPI-Q is associated with a 5% increase in risk of dying. Given that the standard deviation for neuroticism was 2.39, this means that a one standard deviation increase on EPI-Q neuroticism is associated with an approximate 12% increase in mortality risk. As a check on the latter, we standardized the neuroticism variable so that it was expressed in standard deviation units, and indeed, the hazard ratio was 1.12. Thus, for every standard deviation increase on EPI-Q neuroticism, there is 12% higher risk of dying.

In Model 2, we added smoking. The hazard ratio for smoking was large and significant at 2.13 (95% CI: 1.73–2.61). This means smoking a pack per day or more was associated with a 113% increase in mortality risk. Importantly, the addition of smoking lowered the hazard ratio for neuroticism to 1.04 (95% CI: 1.01–1.07), representing a 20% decrease. Using the regression coefficients associated with the hazard ratio, we tested whether the coefficient for neuroticism in Model 2 was significantly different from that in Model 1, and it was, $t(1787) = 21.05$, $p < 0.001$.[1]

In Model 3 we added drinking. Drinking was itself not predictive of mortality, but lowered the hazard ratio for neuroticism to 1.03 (95% CI: 1.01–1.07). Again, using the regression coefficients associated with the hazard ratio, we tested whether the hazard ratio for neuroticism in Model 3 was significantly different from that in Model 2, and it was, $t(1787) = 5.26$, $p < 0.01$. As we knew from the zero-order correlations (Table 1), drinking was positively correlated with both neuroticism and smoking. Although it was not itself predictive of mortality, the drinking variable may have controlled irrelevant variance, and helped to further clarify the neuroticism–mortality association. Nevertheless, it is important to point out that this does not constitute evidence that drinking mediates the neuroticism–mortality association, as drinking is not directly predictive of mortality in any model. Drinking simply seems to serve as a clarifying influence through its correlations with neuroticism and smoking.

[1] The confidence intervals for neuroticism overlap heavily in Models 1, 2 and 3. We believe this is because confidence intervals in the Cox model are asymptotic and therefore asymmetric around its point estimate (the hazard ratio). See Singer and Willett (2003, p. 530) for more details. In addition, the size of the units that a predictor is scaled in has an effect in the confidence interval. The EPI-Q is expressed in raw units (0–9). However, when we standardized the EPI-Q and thus created a scale in standard deviation units, we observed the confidence interval become less overlapping.

Table 2
Hazard ratios for neuroticism and health behaviors on mortality risk ($N = 1788$).

Risk factor	Hazard ratio (95% C.I.)		
	Model 1	Model 2	Model 3
Age	1.10 (1.09–1.11)***	1.11 (1.10–1.12)***	1.11 (1.10–1.12)***
Neuroticism	1.05 (1.01–1.08)**	1.04 (1.03–1.07)**	1.03 (1.01–1.07)**
Smoking		2.13 (1.73–2.61)***	2.10 (1.71–2.60)***
Drinking			1.08 (0.90–1.31)
-2LL	9246	8736	8686
AIC	9250	8742	8694

** $p < 0.01$.
*** $p < 0.001$.

Table 3
Hazard ratios testing neuroticism as a mediator of smoking and mortality ($N = 1788$).

Risk factor	Hazard ratio (95% C.I.)	
	Model 1	Model 2
Age	1.10 (1.09–1.11)***	1.11 (1.10–1.12)***
Smoking	2.15 (1.76–2.63)***	2.13 (1.73–2.61)***
Neuroticism		1.04 (1.03–1.07)**
AIC	8801	8736
-2LL	8805	8742

** $p < 0.01$.
*** $p < 0.001$.

We considered the possibility that the reverse mediator model may hold. Did neuroticism mediate the relationship between smoking and mortality? To test this alternative explanation, we ran age and smoking in a first model, then age, smoking and neuroticism in a second model. If the hazard ratio for smoking remained the same of changed very little, this would support the conclusion that neuroticism does not mediate the association between smoking and mortality. Table 3 shows the results. Note that the hazard ratio for smoking was 2.15 in the first model and 2.13 in the second. This difference of between the hazard ratios was not statistically significant. It is not likely that neuroticism mediates the smoking–mortality association. Rather, our evidence is consistent with the conclusion that smoking mediates the neuroticism–mortality association.

Finally, we also tested the quadratic effect of neuroticism as well as interactions between neuroticism and both smoking and drinking, in addition to the three-way interaction. None of these were significant. Summarizing our models, we began with a hazard ratio of 1.05 for neuroticism on mortality, which was reduced 40% to 1.03 in the final model. This reduction appears attributable mainly to the health behavior of smoking. Yet, the final hazard ratio of 1.03 for neuroticism remained significant in the final model, meaning that we did not fully explain the effect of this trait on mortality. Health behaviors, particularly smoking, account for a portion of the neuroticism–mortality relationship, but not all of it.

4. Discussion

Neuroticism predicted mortality over a 30-year period among men in the VA Normative Aging Study. This is one of the longest follow-ups among studies that have examined the neuroticism–mortality association, and demonstrates that the effect of neuroticism holds over periods greater than a quarter-century. Hazard ratios are directly interpretable as measures of effect size. The hazard ratio of 1.05 for neuroticism (5% increased mortality risk per 1 point on the 0–9 EPI-Q) is not small, and the final hazard ratio after controlling for health behaviors is 1.03 and remains significant.

More importantly, one of the health behaviors – smoking – explains a portion, but not all of, the association between neuroticism and mortality.

4.1. Do health behaviors explain the neuroticism–mortality association?

One of the main theoretical models of the personality–health relationship holds that health behaviors are a key pathway by which personality traits influence physical health and ultimately, illness and mortality (Contrada et al., 1999; Friedman, 2000; Hampson, 2008; Hampson & Friedman, in press; Roberts et al., 2007; Smith, 2006). We hypothesized that health behaviors would serve as such a link between neuroticism and 30-year mortality, but found that only one such behavior, smoking, seems to act as a bridge from personality to shorter life.

This is consistent with our hypotheses and with theoretical expectations that health behaviors should account for at least some of the neuroticism–mortality association (e.g., Contrada et al., 1999; Friedman, 2000; Hampson, 2008; Smith, 2006; Smith & Spiro, 2002). Yet there is room for other explanations, such as biological pathways (Segerstrom, 2000; Wiebe & Smith, 1997). As noted earlier, Hampson et al. (2007) provided suggestive evidence that biological or physiological mechanisms may provide a better explanation of the effect of neuroticism on health and mortality. However, our correlation matrix (Table 1) shows clear associations between neuroticism and both smoking and drinking. Plenty of other studies buttress this finding, particularly for smoking (Gilbert, 1995; Goodwin & Hamilton, 2002; Kirk et al., 2001; Lerman et al., 2000) but also for drinking (Almada et al., 1991; Fischer et al., 2007; Grekin et al., 2006; Larkins & Sher, 2006; Read & O'Connor, 2006).

Smoking and excessive drinking are well-known risk factors for poor health and mortality, and neuroticism is clearly related to both of these damaging behaviors, not only in this study but in many others. It thus makes theoretical and empirical sense to accept that some of the detrimental effect of neuroticism on health is due to poor health behaviors among high-neuroticism individuals. We must be cautious and not over-interpret the role of drinking, as it never emerged as direct predictor of mortality in any of our models. However, the role of smoking is clear and it does seem to mediate the effect of neuroticism on mortality. However, some 60% of the effect was still unaccounted for in this study, and is presumably explainable via biological or other mechanisms.

4.2. Limitations and future directions

The most prominent limitation of this study is the lack of women or any substantial number of minorities in the sample. The Normative Aging Study was founded by the VA in the 1960s, a time when women were routinely excluded from many scientific studies, especially those conducted by the VA. Also, in the 1960s, Boston has home to relatively few minorities. Therefore it is important to recognize that our results may apply mainly to white men.

Another limitation is the bluntness of our health behavior measures. It would have been ideal to obtain estimates of exact number of cigarettes smoked per day for each respondent. There is undoubtedly more variability in smoking and drinking that is not captured by these dichotomous variables. It is likely that the effect of smoking and drinking are *underestimated* in the present analyses, and so it may be the case that they may account for more of the effect than documented here. Additionally, as indicated above, 17% of the NAS participants were pack-a-day (or more) smokers in 1975. Younger generations who have reached adulthood after the 1970s may not smoke at such rates, meaning that our findings require replication with younger cohorts.

Some readers may also perceive the short proximity in time between the neuroticism and health behavior assessments as a limitation. Most were within a two years of one another. Thus, the closeness in time makes the interpretation of the direction of the mediation less clear, as it is plausible that smoking and drinking influence neuroticism. This is unlikely, given that we tested the reverse hypothesis (see Table 3), and also because theory in this area as well as prior empirical work shows that neuroticism predicts smoking and other poor health behaviors (Gilbert, 1995; Goodwin & Hamilton, 2002; Grekin et al., 2006; Kirk et al., 2001; Larkins & Sher, 2006), not the other way around. Nevertheless, any inflation in the correlation between neuroticism and the health behaviors due to closeness in measurement time may have influenced the effects.

As a first step in investigating the role of health behaviors in the neuroticism–mortality association, we treated both neuroticism and health behaviors as fixed effects at baseline (as in most epidemiological studies). We have shown elsewhere that *change* in neuroticism is related to mortality (Mroczek & Spiro, 2007). To investigate the effects of change in both neuroticism and health behaviors is a complex challenge, and we first wanted to demonstrate that the baseline effects matter. In subsequent work, we will address the impact of changes in both neuroticism and health behaviors.

4.1. Conclusion

In recent years, personality variables have emerged in as important predictors of mortality (Friedman, 2000; Friedman et al., 1993). These findings herald a significant development. They signal that certain psychological factors such as personality traits may prove as useful as traditional biomedical markers in understanding mortality, and eventually, disease processes (Smith, 2006). The discovery of pathways that stretch from personality dispositions toward important health outcomes is the crux of what Krueger, Caspi, and Moffitt (2000) have termed "personological epidemiology." The current study is part of this new area of inquiry, and builds upon previous work by suggesting the mechanisms through which personality traits influence health and mortality. Here, we presented evidence that health behaviors, especially smoking, are at least part of the puzzle. However, a good deal of the relationship remained unexplained even when accounting for some health behaviors. Future investigations may examine other types of health behaviors, or even biological mechanisms (Hampson et al., 2007; Wiebe & Smith, 1997) to account for the remainder.

There is also a practical conclusion that we can draw, and perhaps this is the most important conclusion. It is clear that persons high in neuroticism tend to smoke and drink more. There is no doubt that smoking and excessive drinking can damage health and shave years off one's life expectancy. People high in neuroticism are at higher risk of engaging in these potentially destructive behaviors. Interventions or programs that target people high in neuroticism (and other key traits, such as those low in conscientiousness) may get bigger bang for the buck than more global techniques. Identification of people who are predispositionally at higher risk for certain diseases is a hallmark of the new area of "individualized medicine." It may be possible to use personality traits to similarly identify people who, because of their predispositions, are at risk for engaging in poor health behaviors such as smoking or excessive drinking. This perhaps is how the results of this study and future studies in this area can have lasting impact.

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