# Personality and the Problems of Everyday Life: The Role of Neuroticism in Exposure and Reactivity to Daily Stressors

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**ABSTRACT** This article investigates mechanisms through which neuroticism leads to distress in daily life. Neuroticism may lead to distress through exposing people to a greater number of stressful events, through increasing their reactivity to those events, or through a mechanism unrelated to environmental events. This article evaluates the relative importance of these three explanations. Subjects were 339 persons who provided daily reports of minor stressful events and mood for 6 weeks. Exposure and reactivity to these minor stressors explained over 40% of the distress difference between high- and low-neuroticism subjects. Reactivity to stressors accounted for twice as much of the distress difference as exposure to stressors. These results suggest that reactions within stressful situations are more important than situation selection in explaining how neuroticism leads to distress in daily life.

Everyone has a unique quota of distress and ill-health. Some people are habitually anxious or sad, whereas others are calm and happy. Some people suffer many physical ailments, whereas others are rarely ill. It is

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now thought that these individual differences in health and well-being are rooted, in part, in enduring personality characteristics (Friedman, 1990). Yet how personality is expressed in health and well-being remains largely a mystery. In this article we test the hypothesis that it is through stressful daily events that the effects of personality are revealed.

Specifically, we propose that personality can influence health and psychological well-being in three ways. First, personality may explain why some people get into stressful situations and others do not; anticipating and preventing the occurrence of stressful events can be an effective way of maintaining well-being. Second, personality may affect how people react once they are in stressful situations; certain people may habitually cope ineffectively under stress. Finally, personality may lead to health and psychological outcomes through a mechanism unrelated to environmental events; personality may have an endogenous or direct effect on well-being.

Stress researchers have traditionally conceptualized personality in the second way—as a variable that modifies the impact of stressors on health outcomes (see Cohen & Edwards, 1989, for a review of this literature). Studies of dispositions such as locus of control (Johnson & Sarason, 1978; Parkes, 1984), hardiness (Kobassa, Maddi, & Kahn, 1982), self-consciousness (Mullen & Suls, 1982; Suls & Fletcher, 1985), and the Type A behavior pattern (Rhodewalt, Hays, Chemers, & Wysocki, 1984; Suls, Gastorf, & Wittenberg, 1979) are examples of research in this tradition. More recently, researchers have paid increasing attention to how personality determines the types of situations people encounter (Buss, 1984, 1987; Diener, Larsen, & Emmons, 1984; Emmons, Diener, & Larsen, 1986; Snyder, 1983; Snyder & Ickes, 1985). For example, Smith and Rhodewalt (1986) have argued that Type A personalities are more prone to coronary heart disease because they select stressful situations as well as overreact in them.

Despite the current attention to both exposure and reactivity to stressors, the relative importance of these processes in linking personality to well-being has not been established. Although people may select (or be selected into) stressful situations, this process may be a relatively minor contributor to individual differences in distress or illness. Similarly, although personality modifies the relationship between stressors and disorder, this process may play a relatively small role in explaining individual differences in well-being. Finally, it is important to note that the relationship between personality and health outcomes may be

largely unmediated by either exposure or reactivity to stressful events.

This issue has both practical and theoretical significance. Consider intervention programs designed to reduce the health-damaging effects of Type A behavior. Should these programs teach people to avoid stressful situations, to cope more effectively with stressful situations, or should they emphasize strategies that are not at all tied to stressful situations (e.g., relaxation techniques)? Without knowing the relative importance of exposure, reactivity, and unmediated processes in leading to the outcomes of personality, one cannot begin to address this question.

This article investigates a specific disposition, neuroticism, and a specific outcome, psychological distress. Of the primary dimensions of personality, neuroticism is the chief determinant of psychological distress (Watson & Clark, 1984). We will assess the relative importance of exposure to daily stressors, reactivity to those stressors, and a mechanism unrelated to daily stressors in explaining the relation between neuroticism and distress in daily life.

## Neuroticism, Stressors, and Distress

What is the evidence that these three mechanisms—exposure, reactivity, and unmediated processes—explain the link between neuroticism and psychological distress? Evidence for the exposure mechanism was reported in two recent studies. In a 6-year study of women with school-aged children, neuroticism was a strong predictor of exposure to major life events (Fergusson & Horwood, 1987). In the second study, neuroticism predicted increased involvement in interpersonal conflicts (Buss, Gomes, Higgins, & Lauterbach, 1987). Because these studies rely on self-reports of stressors, however, it is unclear whether neuroticism increases *actual exposure* to stressful events or merely *reports* of stressful events. It has been demonstrated in laboratory studies that neuroticism increases the recall of negatively toned information (Lishman, 1972; Lloyd & Lishman, 1975; Martin, Ward, & Clark, 1983; Young & Martin, 1981). If this recall bias operates in real-life situations, it may account for some or all of the association between neuroticism and exposure to stressors.

The evidence linking neuroticism to stressor reactivity is more clear-cut. The state-trait theory of anxiety predicts that the effects of neuroticism on anxiety will be most evident under stress (Endler & Edwards,

1982; H. J. Eysenck & M. W. Eysenck, 1985; Spielberger, Gorsuch, & Lushene, 1970), and the theory has been verified in many studies (e.g., Bolger, 1990; Ormel, Stewart, & Sanderman, 1989).

The third putative mechanism linking neuroticism to distress involves neither exposure nor reactivity to stressful events. To the extent that neuroticism is similar to negative affectivity—the disposition to experience negative affect even when provoking events are absent (Watson & Clark, 1984)—the relationship between neuroticism and daily distress should be unmediated by stressful events.

## A Daily Diary Approach

We used a daily diary method to evaluate the relative importance of exposure, reactivity, and unmediated explanations for the expression of neuroticism in daily distress, tracking people as they moved through many minor stressful situations. In that way we could gauge people's exposure to stressors and their emotional reactions to the stressors. We could then see whether neuroticism predicted individual differences in exposure and reactivity and whether exposure and reactivity, in turn, explained the relationship between neuroticism and daily distress.

The diary methodology also allowed us to study same-day and later-day reactions to a stressor, enabling us to distinguish people's initial emotional reactions to a stressor from their speed of recovery from that stressor. This distinction may be critical to understanding the link between neuroticism and distress. High- and low-neuroticism persons may be equally upset by a stressful event on the day it occurs, but high-neuroticism persons may remain distressed longer than low-neuroticism persons, and consequently show higher average distress levels.

The data are from a 6-week study of daily stress in married couples. Husbands and wives provided independent daily reports of their psychological distress and their experience of daily stressful events. Because certain daily events were experienced by both husbands and wives (e.g., marital arguments), we could test whether neuroticism affected spouses' agreement that a stressful event occurred. As noted earlier, the possibility that neuroticism biases stressor reports poses interpretive problems. In this study, evidence of reporting bias could be gauged by comparing husbands' and wives' reports of stressors.

We addressed the following questions. First, are persons high in neuroticism more distressed on average than persons low in neuroticism?

Second, do persons high in neuroticism report greater exposure to daily stressors than persons low in neuroticism, and, for events that spouses share (e.g., marital arguments), do spouses agree that these events actually occurred? Third, are persons high in neuroticism more emotionally reactive—concurrently and over time—to the daily stressors they experience? Fourth, what is the relative importance of exposure, reactivity, and unmediated mechanisms in explaining why persons high in neuroticism are more distressed than persons low in neuroticism?

## METHOD

### Sample

The sample comprised 339 persons (166 married couples and 7 individuals) who independently completed diaries every day for 42 consecutive days. All respondents were married, were from the Detroit metropolitan area, and had participated 1 year previously in a community survey of stress and coping. Neuroticism was measured in this earlier survey. The response rate in the survey was 76%. Of the original eligible sample of couples, 34% participated in the diary study. Bolger, DeLongis, Kessler, and Wethington (1989) compared couples who participated in the diary study with those who did not on a variety of background variables that were measured in the original survey (including neuroticism). There was a small difference between the samples on frequency of marital arguments: The diary sample tended to show less marital conflict. (For more detailed information on data collection and response rates, see Bolger, DeLongis, Kessler, and Schilling [1989].)

### Measures

*Neuroticism.* In the original survey, each subject completed an 11-item short form of the Neuroticism scale from the Eysenck Personality Inventory (H. J. Eysenck & S. B. G. Eysenck, 1964). Examples of neuroticism items include: Would you call yourself a nervous person? In general, are your feelings easily hurt? Do you often feel fed-up? Are you a worrier? Cronbach's $\alpha$ for the scale was .78. Although the Eysenck scale is a continuous variable, to simplify the analysis, particularly the decomposition procedure (see statistical model), we treated it as a dichotomous variable. We divided neuroticism at the median such that 172 persons were classified high in neuroticism and 167 persons were classified low in neuroticism.

*Daily stressors.* The list of stressors in the daily diary was based on pilot work that identified the most common daily events in a community sample of married couples. The diary contained a checklist of the 22 most common events. For analytic purposes we reduced this list to nine categories by combining events that were both conceptually similar and had similar relationships to daily distress. For example, we combined "spouse sick," "child sick," and "a lot of demands made by your family" into a single "family demand" variable (see Bolger, DeLongis, Kessler, & Schilling, 1989).

The nine stressor categories were (*a*) overload at home, (*b*) overload at work, (*c*) family demand, (*d*) other demand, (*e*) transportation problem, (*f*) financial problem, (*g*) argument with spouse, (*h*) argument with child, and (*i*) argument with other (not spouse or child). We used dummy (0, 1) variables to code the occurrence of each stressor. For example, if a respondent had a financial problem on a particular day, the financial problem variable was coded 1 for that person-day, and 0 otherwise. Because previous analyses revealed major differences in the emotional effects of different types of stressors (e.g., arguments were more upsetting than overloads) (Bolger, DeLongis, Kessler, & Schilling, 1989), we kept each stressor category distinct in the present analyses.

*Daily distress.* Each day, respondents indicated how strongly they had felt each of 18 emotions over the previous 24 hours. The emotion items were drawn from the anxiety, depression, and hostility subscales of the Affects Balance Scale (Derogatis, 1975). Anxiety was measured using items such as "nervous," "tense," and "afraid"; hostility using "irritable," "angry," and "resentful"; and depression using "helpless," "worthless," and "depressed." Respondents rated each item on a 4-point scale from "not at all" to "a lot." Cronbach's $\alpha$ was .92. We scaled the measure so that it ranged from 0 to 100, where 0 was the lowest possible score and 100 was the highest possible score.

## Statistical Model

*Overview.* The data analysis consisted of two major steps. First, we estimated a model linking neuroticism, daily stressors, and daily distress. Then we used the results of this model to decompose the neuroticism-distress relationship into exposure to stressors, reactivity to stressors, and unmediated components. Because the statistical model and the decomposition procedure are uncommon, we discuss them in more statistical detail than is usual in a substantive article. We also provide graphical descriptions of the model and the decomposition procedure. Readers who wish to focus on the graphical descriptions alone should skip forward to the section entitled "Graphical representation of the hierarchical linear model."

*Hierarchical linear model linking neuroticism, daily stressors, and daily distress.* We analyzed our data using a multilevel or hierarchical linear model (Bryk & Raudenbush, 1987; Mason, Wong, & Entwisle, 1984; Strenio, Weisberg, & Bryk, 1983). The model allows us to obtain separate estimates of stressor reactivity for each person and to express individual differences in reactivity as a function of neuroticism.

Thus, our model specifies that each person in the population has his or her own characteristic relationship between daily events and mood. For the sake of simplicity, we begin our discussion with a model that includes only one stressful event. It generalizes in a straightforward way to include more than one stressful event. The model can be written as follows:

$$D_{it} = a_{0i} + a_{1i}S_{it} + e_{it} \quad (1)$$

where $D_{it}$ is the distress of Person $i$ on Day $t$, $S_{it}$ indicates whether a stressor occurred to Person $i$ on Day $t$ (coded 0 if no stressful event occurred, and 1 if a stressful event occurred), $a_{0i}$ is the intercept for Person $i$ (i.e., Person $i$'s distress on days when no stressful event occurred), $a_{1i}$ is Person $i$'s slope or reactivity to a stressful event (i.e., the number of units that Person $i$ is higher on distress on days when a stressful event occurred compared to days when an event did not occur), and $e_{it}$ is a random component of the distress of Person $i$ on Day $t$. The intercepts for each individual ($a_{0i}$) and slopes for each individual ($a_{1i}$) are assumed to be drawn randomly from a population with a normal distribution. Similarly, the random components of each individual's daily distress score ($e_{it}$) are assumed to be drawn from a normal distribution with a mean of 0 and to show no autocorrelation over time.[1]

We also specify that the coefficients of this model vary as a function of each person's level of neuroticism, as follows:

1. Equation 1 specifies a time-series relationship between stressors and distress for each person. It is not uncommon in time series models that temporally adjacent error terms are positively correlated. Autocorrelation biases standard errors of parameter estimates and thereby leads to biased statistical tests (see Caspi, Bolger, & Eckenrode, 1987, for a discussion of these issues as they relate to daily mood; see also Larsen & Kasimatis, 1991, for a discussion of autocorrelation in daily physical symptoms). In preliminary analyses we used the PROC AUTOREG program in SAS (SAS Institute, 1984) to estimate Equation 1 and to test for the presence of first-order autocorrelated errors (first-order autocorrelation exists when immediately adjacent error terms ($e_{it}$ and $e_{it+1}$) are correlated). We ran this analysis separately for all 339 subjects in our sample. We found only slight evidence of autocorrelation: Across all 339 subjects, the average correlation between adjacent error terms was $-.035$.

$$a_{0i} = b_0 + b_1 N_i + g_i \tag{2}$$

and

$$a_{1i} = b_2 + b_3 N_i + d_i \tag{3}$$

Thus, Equations 2 and 3 show that Person $i$'s intercept and slope are functions of an intercept, a neuroticism component ($N_i$ is coded 0 for low and 1 for high neuroticism), and a random component. The random components, $g_i$ and $d_i$, are assumed to be drawn independently from normal distributions with mean 0.

Substituting for $a_{0i}$ and $a_{1i}$ in Equation 1 we obtain a single-equation model showing daily distress as a function of neuroticism and the daily stressful event:

$$D_{it} = (b_0 + b_1 N_i + g_i) + (b_2 + b_3 N_i + d_i) S_{it} + e_{it}$$

and therefore,

$$D_{it} = b_0 + b_1 N_i + b_2 S_{it} + b_3 N S_{it} + g_i + d_i S_{it} + e_{it} \tag{4}$$[2]

*Graphical representation of the hierarchical linear model.* The hierarchical model linking neuroticism, daily stressors, and daily distress is illustrated in Figure 1. Note that Figure 1 is not based on empirical data; rather, it is a device to convey the main elements of a hierarchical model. Figure 1 shows that each individual has his or her own characteristic relationship (regression line) between stressors and distress. It also shows that individuals fall into two groups: the high-neuroticism group and the low-neuroticism group. There are intercept differences between the groups: High-neuroticism persons tend, on average, to be more distressed than low-neuroticism persons even when no stressful event occurs. There are also slope or reactivity differences between the groups: High-neuroticism persons tend to become more upset than low-neuroticism persons when stressful events occur. (Reactivity is defined as the difference in a person's distress between days when a stressor occurs and days when one does not.)

2. This model is more complex than those used in previous analyses of diary data. In previous work it has been usual to assume that all persons in the analysis have a common slope, but that each person has his or her own regression intercept. The effects of these individual-specific intercepts have been taken into account either by residualizing each person's data from his or her own mean (e.g., Bolger, DeLongis, Kessler, & Schilling, 1989) or by including dummy variables for each person as predictor variables (e.g., Cutrona, 1986). The present model allows each person to have his or her own intercept and slope, and as such, is more sensitive to individual differences in daily stress processes.

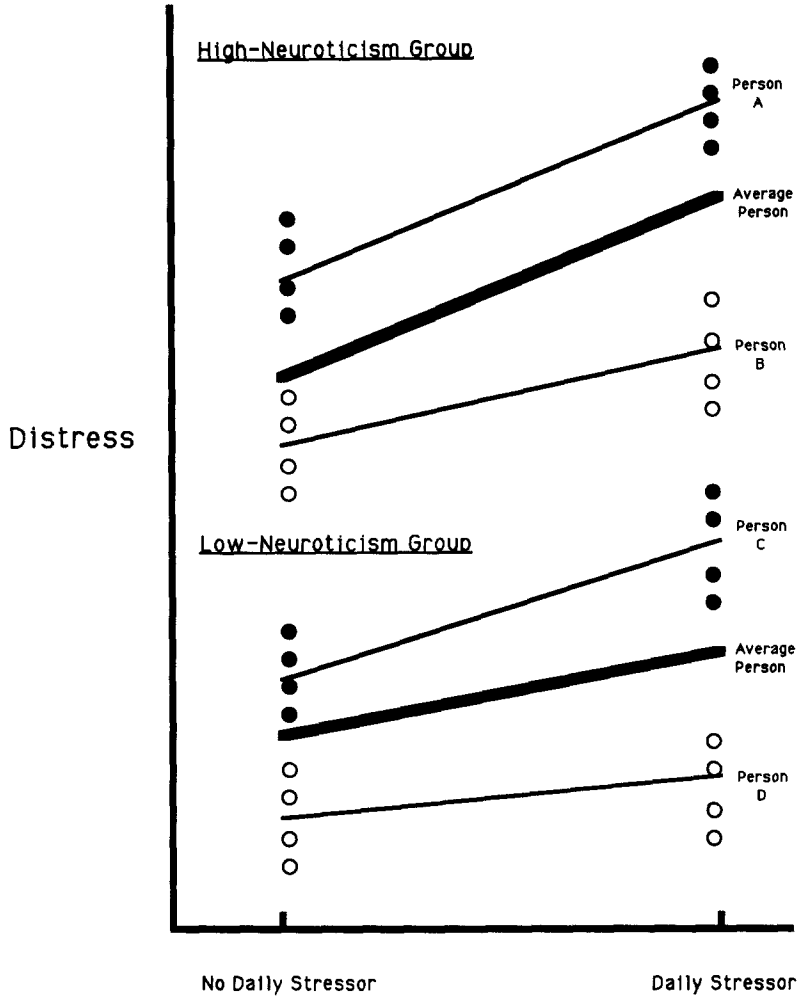



**Figure 1**
**Hierarchical Linear Model of the Relationship between Neuroticism, Daily Stressors, and Daily Distress**

The bold line for the high-neuroticism group shows the intercept and slope for the average person in that group. The lighter lines represent deviations from the group average. The line labeled "Person A" displays the model for a high-neuroticism individual who is, on average, above the group mean on distress on days when no stressful event occurs (i.e., has a high intercept) and whose reactivity to stress is the same as the group average. The line labeled

"Person D" displays the model for a low-neuroticism individual who is, on average, less distressed than the group mean for "no stressor" days, and who is slightly less reactive to stressors than the group is on average. Note, for Persons A, B, C, and D, the distribution of points around their means for "no stressor" and "stressor" days. These points are meant to represent the raw data obtained in this study: reports of stressful events and mood for particular individuals on particular days. The model uses each person's time-series data on stressors and distress to obtain each person's intercept and reactivity to stressors.

We can now begin to identify the specific terms in Equation 4 as they are represented in Figure 1. (Readers interested in graphical accounts only should now skip forward to the section entitled "Graphical illustration of the decomposition procedure.") For the low-neuroticism group, $N_i$ equals zero, and therefore the terms $b_1N_i$ and $b_3NS_{it}$ in Equation 4 equal zero. The model therefore reduces to

$$D_{it} = b_0 + b_2S_{it} + g_i + d_iS_{it} + e_{it} \tag{5}$$

In Figure 1, the bold line for the low-neuroticism group represents the average or expected value of $D_{it}$ for days when no stressor occurs and for days when a stressor occurs. The expected value of $D_{it}$, conditional on $S_{it}$, is

$$E(D_{it}/S_{it}) = E(b_0 + b_2S_{it} + g_i + d_iS_{it} + e_{it}) = b_0 + b_2S_{it} \tag{6}$$

As shown in Figure 1, $b_0$ is the group mean for days on which no stressful event occurs and $b_2$ is the stressor reactivity, the number of units higher on distress that group members are on days when a stressful event occurs compared to days when one does not occur.

For the high-neuroticism group, $N_i$ equals 1, and therefore the terms $b_1N_i$ and $b_3NS_{it}$ in Equation 4 equal $b_1$ and $b_3S_{it}$, respectively. Therefore, the model reduces to

$$D_{it} = (b_0 + b_1) + (b_2 + b_3)S_{it} + g_i + d_iS_{it} + e_{it} \tag{7}$$

In the same manner as for the low-neuroticism group, the expected value of this function, conditional on $S_{it}$, is

$$(b_0 + b_1) + (b_2 + b_3)S_{it} \tag{8}$$

which is represented by the heavy line for the high-neuroticism group in Figure 1. In this case, $(b_0 + b_1)$ is the group mean on days when no stressful event occurs and $(b_2 + b_3)$ is the group stress reactivity.

For both the low- and high-neuroticism groups, the components $g_i$, $d_i$, and

$e_{it}$ are the random parts of the model. Thus $g_i$ represents random variation in intercepts due to specific persons in the low- and high-neuroticism groups. Figure 1 shows that the value of $g_i$ for Person A is positive, giving that person a higher-than-average distress score for days when no stressful event occurs, whereas the value of $g_i$ for Person B is negative, giving that person a lower-than-average distress score for days when no stressful event occurs. The $d_i$ component represents random variation in stressor reactivity within the low- and high-neuroticism groups; it captures how each person's reactivity differs from the group average. For Person A the $d_i$ component is zero, as this person is at the high-neuroticism group mean for stressor reactivity. For Person C the component is positive, as this person is more reactive to stress than the typical person in the low-neuroticism group. The $e_{it}$ component represents the random fluctuation in a person's daily distress scores around his or her average: Each $e_{it}$ represents the deviation of a single data point around a person's mean distress for either "no stressor" or "stressor" days.

*The multiple-stressor case.* The data set we analyzed contained not one but nine stressful events. Thus for our purposes Equation 4 needs to be expanded as follows:

$$D_{it} = b_0 + b_1 N_i + \sum_j b_{2j} S_{jit} + \sum_j b_{3j} N S_{jit} + g_i + \sum_j d_{ji} S_{jit} + e_{it} \quad (9)$$

$S_{jit}$ $(j = 1, 2, \ldots, 9)$ represents nine dummy (0, 1) variables that indicate whether Respondent $i$ experienced Stressor $j$ on Day $t$. $NS_{jit}$ $(j = 1, 2, \ldots, 9)$ represents nine interaction terms between Stressor $j$ and the neuroticism dummy (0, 1) variable. These interaction terms are also dummy variables and their coefficients $b_{3j}$ indicate the mean difference in reactivity to Stressor $j$ between respondents in the high-neuroticism group and those in the low-neuroticism group.

We showed earlier that our single-stressor model, Equation 4, could be written as two submodels: Equation 5, showing the determinants of distress for the low-neuroticism group, and Equation 7, showing these determinants for the high-neuroticism group. Using Equations 5 and 7, we then showed that the average or expected value of distress for the low- and high-neuroticism groups for no-stress and stress days was given by Equations 6 and 8, respectively. In the same manner, using the multiple-stressor Equation 9 as our starting point, we can show the expected value of distress for the low-neuroticism group, conditional on $S_{jit}$, is

$$b_0 + \sum_j b_{2j} S_{jit} \quad (10)$$

The reactivity of a low-neuroticism respondent to Stressor $j$ is thus $b_{2j}$.

Similarly, the expected value of the high-neuroticism submodel is

$$(b_0 + b_1) + \sum_j (b_{2j} + b_{3j}) S_{jit} \tag{11}$$

The reactivity of a high-neuroticism respondent to Stressor $j$ is, therefore, $b_{2j} + b_{3j}$, and the difference in reactivities between low- and high-neuroticism groups is $b_{3j}$.

Because our data were longitudinal, we could investigate the tendency for stressors to affect distress beyond the day of their occurrence. We could also examine whether this process depended on level of neuroticism. To do this we used the following equation:

$$\begin{aligned} D_{it} = {} & b_0 + b_1 N_i + \sum_j b_{2j} S_{jit} + \sum_j b_{3j} N S_{jit} \\ & + \sum_j b_{4j} P_{jit} + \sum_j b_{5j} N P_{jit} + g_i + \sum_j d_{ji} S_{jit} \\ & + \sum_j f_{ji} P_{jit} + e_{it} \end{aligned} \tag{12}$$

The nine dummy variables ($P_{jit}$) indicate whether Respondent $i$ experienced Stressor $j$ on the day *prior* to Day $t$. Interactions of these variables with the neuroticism group dummy variable yield nine additional dummy variables ($NP_{jit}$). The coefficients of these interaction variables, $b_{5j}$, estimate neuroticism-related differences in the tendency for yesterday's stressors to affect today's distress. The term $f_{ji} P_{jit}$ represents the individual random components of the prior-day stressor effects.

*Decomposing the effect of neuroticism on distress.* Using information from the models above, we can compare the relative importance of exposure to stressors, reactivity to stressors, and unmediated processes in explaining the overall mean difference in distress between the two neuroticism groups. The following is an algebraic description of the technique. More complete expositions of the decomposition technique can be found in Winsborough and Dickinson (1971), Kessler (1979), or Iams and Thornton (1975). Readers who would rather skip the algebra and rely on a graphical description should turn to the next section of the article.

To simplify the algebra, we will assume that stressors only have a concurrent (same-day) effect on distress, i.e., that the quantities $b_{4j}$, $b_{5j}$, and $f_{ji}$ in Equation 12 are zero. The method we will describe, however, can be generalized to include both concurrent and cross-day reactivity effects.

It is a characteristic of linear regression that if the means of all predictor variables are substituted into the regression equation, the predicted value of the dependent variable will be its mean (see Neter, Wasserman, & Kutner,

1985, pp. 43–46). Therefore, by computing the relevant means—over persons and days—for each stressor, and by substituting these means (probabilities of exposure to each stressor) into Equations 10 and 11, we can predict the mean level of distress for the low- and high-neuroticism groups, respectively. The mean level of distress for the low-neuroticism group ($M_L$) can be written as

$$M_L = b_0 + \sum_j b_{2j} S_{Lj} = b_0 + \sum_j b_{Lj} S_{Lj} \tag{13}$$

Similarly, the mean level of distress for the high-neuroticism group ($M_H$) can be written as

$$M_H = b_0 + b_1 + \sum_j (b_{2j} + b_{3j}) S_{Hj} = b_0 + b_1 + \sum_j b_{Hj} S_{Hj} \tag{14}$$

Here $S_{Lj}$ and $S_{Hj}$ give the probabilities of exposure to Stressor $j$ for the low- ($L$) and high- ($H$) neuroticism groups. Similarly, $b_{Lj}$ and $b_{Hj}$ index how reactive the low- and high-neuroticism groups are to Stressor $j$. Using Equations 13 and 14, we can show that the difference in distress between the two groups, assuming, for simplicity, only one stressor (i.e., dropping the $j$ subscript), is

$$M_L - M_H = b_0 - (b_0 + b_1) + b_L S_L - b_H S_H$$

By adding $(-b_L S_H + b_L S_H)$, which is equal to zero, to the right-hand side of the equation and collecting terms this becomes

$$M_L - M_H = -b_1 + b_L (S_L - S_H) + (b_L - b_H) S_H$$

Expanding $b_L$ in the second term to $(b_L - b_H + b_H)$, this becomes

$$M_L - M_H = -b_1 + (b_L - b_H + b_H)(S_L - S_H) + (b_L - b_H) S_H$$

This can then be rewritten as

$$\begin{aligned} M_L - M_H = {} & -b_1 + b_H (S_L - S_H) + (b_L - b_H) S_H \\ & + (b_L - b_H)(S_L - S_H) \end{aligned}$$

Including all nine stressors as predictors and rearranging terms, the equation is:

$$\begin{aligned} M_L - M_H = {} & \sum_j b_{Hj} (S_{Lj} - S_{Hj}) + \sum_j (b_{Lj} - b_{Hj}) S_{Hj} \\ & + \sum_j (b_{Lj} - b_{Hj})(S_{Lj} - S_{Hj}) - b_1 \end{aligned} \tag{15}$$

The quantity $M_L - M_H$ can be viewed as the decrease in distress that is required to make the high-neuroticism group's distress equal that of the low-neuroticism group. We can see from Equation 15 that this mean difference can be decomposed into four components. The first term is what we referred to earlier as the exposure component of the mean difference in distress. This term estimates the decrease in the mean distress level of the high-neuroticism group if this group's reactivity level were held constant but its *exposure to all stressors* were made to equal that of the low-neuroticism group. The second term is the reactivity component. This estimates the decrease in the mean distress level of the high-neuroticism group if this group's exposure to stressors were held constant but its *reactivity to all stressors* were made equal to that of the low-neuroticism group.

The third term is the interaction component. This is the effect of simultaneously changing the neuroticism group's exposure and reactivity to stressors, over and above the effect of changing each alone. If this term is relatively large, then the effect of changing both exposure and reactivity may be much smaller or much greater than the sum of the effects of changing exposure alone and changing reactivity alone (see Iams & Thornton, 1975).

The final term is the unmediated or residual component, the component of the distress difference not attributable to exposure, reactivity, or interaction. It can also be interpreted as the difference in distress that would exist between the high- and low-neuroticism groups in the absence of any stressful events, i.e., the difference in the regression intercepts of each group.

*Graphical illustration of the decomposition procedure.* Figure 2 provides a graphical illustration of the procedure used to decompose the neuroticism-distress relationship. To simplify the exposition, we again assume that distress is predicted by only one stressful event. Note also that Figure 2 is not based on empirical data; it is intended as an aid to understanding the decomposition procedure. Figure 2, Panel A, shows the relationship between experiencing a stressful event and becoming distressed for the high- and low-neuroticism groups. Figure 2 is, in fact, a simplified version of Figure 1, in which we display only the regression lines for the average low- and high-neuroticism individual. Unlike Figure 1, however, we have also indicated the mean exposure to stressors and the mean distress levels for both groups.

There are three noteworthy features of Figure 2, Panel A. Notice that high-neuroticism individuals are more distressed on average than low-neuroticism individuals. Notice also that they have higher exposure *and* higher reactivity to stressors than the low-neuroticism individuals. Finally, notice that for both the low- and high-neuroticism groups, the mean exposure to stressors predicts the mean distress for that group. Thus, for the low-neuroticism regression line, notice that the low-neuroticism mean exposure to stressors predicts the low-

neuroticism mean on distress. Similarly, for the high-neuroticism regression line, notice that the high-neuroticism mean exposure to stressors predicts the high-neuroticism mean on distress.

Panels B, C, and D, respectively, show the unmediated, exposure, and reactivity components of the mean difference in distress. (The interaction component, described in the previous section, is not displayed because in the empirical data we will present later this was small enough to be ignored.) We turn first to the unmediated component (Panel B). We referred to this earlier as the difference in distress that would have existed in the absence of any stressful event. This effect is captured by the difference in intercepts between the low- and high-neuroticism groups (the intercept is the level of distress for days when no stressful event occurs). Panel B shows that by lowering the intercept of the high group to equal that of the low group, the overall mean difference in distress between the groups would decrease.

Panel C shows the exposure effect, i.e., the effect of lowering the high-neuroticism group's exposure to stressors to the level of the low-neuroticism group, while keeping their reactivity constant. To see this, attend only to the high group's regression line. Notice that if (on the x-axis) we lower the exposure of the high group to that of the low group, we observe (on the y-axis) a corresponding decrease in the high group's distress.

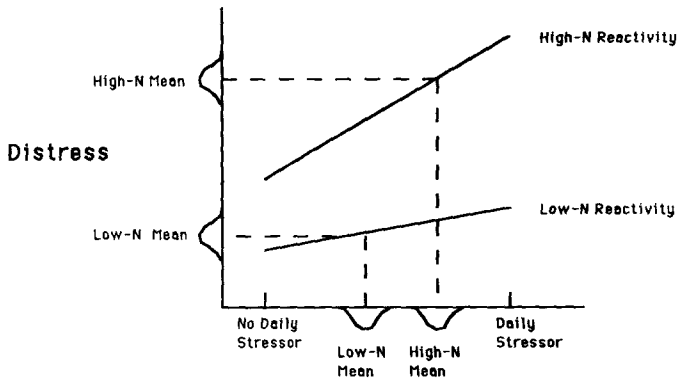
Panel D shows the reactivity effect, i.e., the effect of holding the high-neuroticism group's exposure to stress constant but lowering its reactivity to stressors to that of the low-neuroticism group. To see this, look first at the point on the high-neuroticism group's regression line corresponding to its mean exposure to stressors. Now, observe the effect of dropping down perpendicularly to the low group's regression line (i.e., lowering reactivity while holding exposure constant). Once again there is a decrease in the mean difference in distress.

## RESULTS

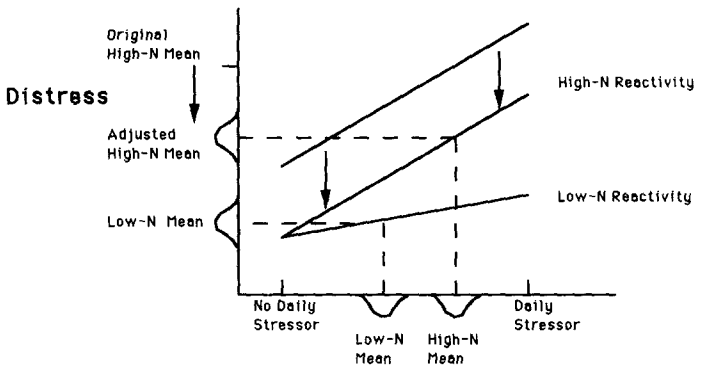
### Neuroticism and Daily Distress

On average, high-neuroticism subjects were more distressed than low-neuroticism subjects over the 6-week daily diary period. We determined this by first calculating the mean distress across all days separately for each subject and then expressing these means as a function of neuroticism and gender in a 2 × 2 analysis of variance (ANOVA). Using the ANOVA model we calculated the mean difference in distress for low- and high-neuroticism persons, adjusting for gender and the Gender × Neuroticism interaction to take account of the fact that husbands and

A: Original Neuroticism-Distress Relationship




B: Equate High- and Low-N Intercepts



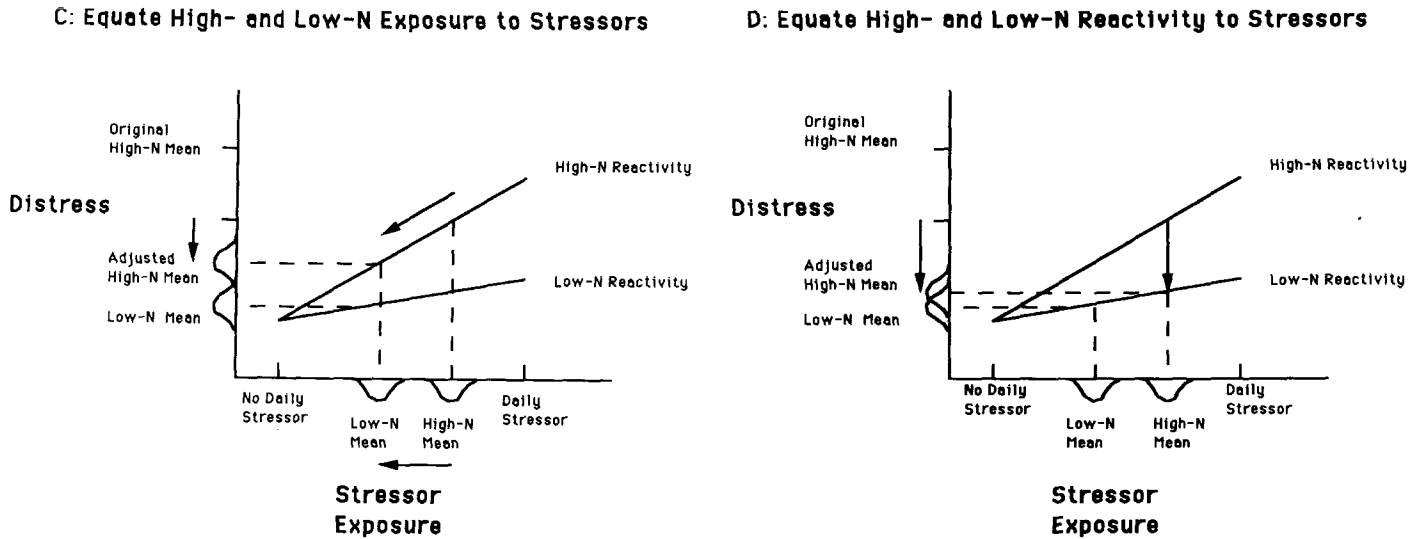



**Figure 2**
Decomposing the Neuroticism-Distress Relationship into Unmediated, Exposure, and Reactivity Components

wives were matched in the sample.[3] There was a 5.8 unit difference in distress between the high- and low-neuroticism groups, $t(335) = 4.70$, $p < .0001$. This corresponds to 8% of the between-person variance in daily distress, which is equivalent to a partial correlation of .28 or a standardized effect size of .59 (see Cohen, 1988, p. 20). We were interested in explaining this 5.8 unit difference in distress in terms of differences in exposure and reactivity to daily stressors.

## Neuroticism and Exposure to Stressors

Figure 3 shows the mean exposure to each stressful event by level of neuroticism. These means were obtained by first calculating the mean exposure to stress for each person (expressed as the percentage of days over the 6-week period on which each event occurred) and then, as before, using an ANOVA model to average these means by level of neuroticism, adjusting for gender of respondent and the Gender × Neuroticism interaction. Because each stressor was coded as a dummy (0, 1) variable, each person's stressor exposure (e.g., to arguments with child) is simply the arithmetic mean of his or her scores for that stressor across the 6-week period (this was then multiplied by 100 to obtain a percentage score).

Figure 3 is noteworthy in several respects. First, high-neuroticism respondents are, in general, more exposed to stressors than low-neuroticism respondents. In seven of the nine stressor categories, high-neuroticism respondents are more likely than low-neuroticism respondents to report a stressful event, although only two of these seven differences (for argument with spouse and argument with other) were statistically significant at $F(1, 335)$, $p < .05$, two-tailed. Second, interpersonal conflicts are consistently more prevalent for high-neuroticism respondents. The difference in mean stressor exposure is 1.8% of days for argument with spouse, 1.9% for argument with child, and 3.3% for argument with other.

We noted in the introduction that high-neuroticism subjects might have a lower threshold than low-neuroticism subjects for reporting stressful events. For example, a person high in neuroticism might report a minor disagreement as an argument, whereas a person low in

3. We used the SAS PROC GLM program to do these calculations (SAS Institute, 1985).

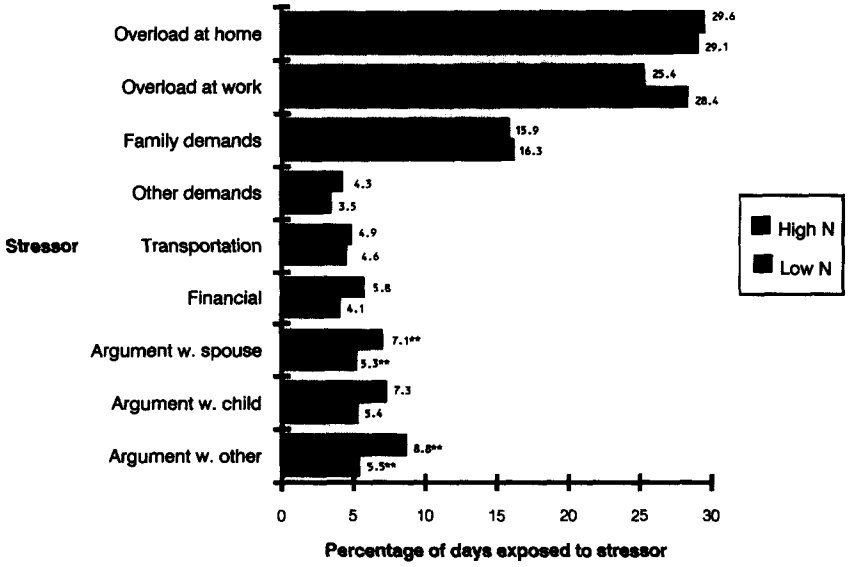



**Figure 3**
**Neuroticism and Exposure to Daily Stressors**

Note. Low N = low neuroticism; High N = high neuroticism.
$^{**}p < .05$, two-tailed.

neuroticism would consider the same event too insignificant to report. We checked for this possibility by analyzing respondents' and spouses' reports of marital arguments and found no evidence for it. Specifically, we performed a $4 \times 2 \times 42$ repeated measures analysis of variance (MANOVA) with Neuroticism (husband-low, wife-low; husband-low, wife-high; husband-high, wife-low; and husband-high, wife-high) as a between-couple factor, and with Gender (husband, wife) and Time (Day 1 through Day 42) as repeated-measurement factors. Although we found that wives were more likely in general to report a marital argument on a particular day than husbands (6.5% of days vs. 5.4% of days), the discrepancy between husbands and wives did not differ as a function of neuroticism. Therefore, compared to low-neuroticism respondents, high-neuroticism respondents do not appear to have a lower threshold for reporting marital arguments.

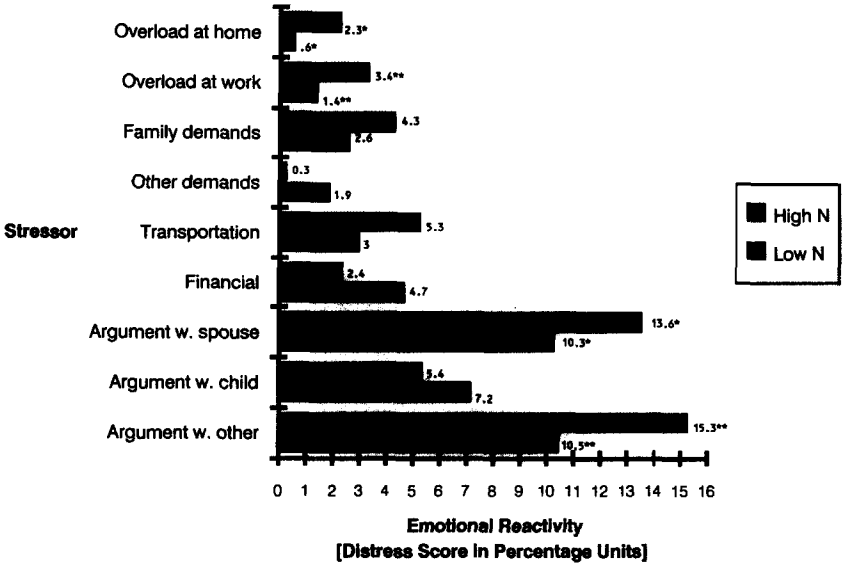



**Figure 4**
**Neuroticism and Reactivity to Daily Stressors**

Note. Low N = low neuroticism; High N = high neuroticism.
*$p < .1$, two-tailed.
**$p < .05$, two-tailed.

## Neuroticism and Reactivity to Stressors

Figure 4 shows differences in stressor reactivity between the low- and high-neuroticism groups for each of the nine stressors. Recall that we defined reactivity as the difference in a person's distress on days when a stressor occurs compared to days when it does not. As in the case of stressor exposure, we first obtained estimates of stressor reactivity for each person (see Equation 1) and then expressed these estimates as a function of neuroticism (see Equation 3), adjusting for gender of respondent and the Gender × Neuroticism interaction.[4]

Figure 4 shows that *both* low- and high-neuroticism respondents experience greater distress on days when stressors occur than on days when they do not. Across the set of nine stressors, however, high-neuroticism respondents experience more event-related distress than

4. We used the SAS PROC REG program (SAS Institute, 1985) to obtain individual estimates of reactivity and used the SAS PROC GLM program to express these as a function of neuroticism and to adjust for gender.

do low-neuroticism respondents, Wilks's Lambda = .94, $F(9, 327) = 2.21, p < .03$. In univariate tests, the significance level for the neuroticism effects was at least $p < .1$, two-tailed, $F(1, 335)$, for four events: overloads at home, overloads at work, arguments with spouse, and arguments with other persons. As shown in Figure 4, when these events occurred, high-neuroticism respondents became more distressed than low-neuroticism respondents by 1.7, 2.0, 3.3, and 4.8 units, respectively.

We also tested whether high- and low-neuroticism respondents differ in the speed with which they recover emotionally from stressful events. To do this, we looked at differences in reactivity between high- and low-neuroticism respondents on the day following a stressful event, controlling for whether or not a stressful event also occurred on this day. A global test of the significance of the difference in reactivity, in terms of the nine $b_5$ coefficients in Equation 12, was not significant, Wilks's Lambda = .98, $F(9, 327) = 0.77$, *ns*.

## Neuroticism and Daily Distress: Exposure, Reactivity, and Residual Explanations

*Decomposition of neuroticism-distress relationship.* We have seen that persons high in neuroticism are more likely than those low in neuroticism to experience stressful situations and to become distressed by those situations. Recall that the mean difference in distress between low- and high-neuroticism respondents was 5.8 units and that using Equation 15, this difference can be decomposed into four components: exposure, reactivity, interaction, and unmediated.

Substituting the exposure and reactivity estimates from Figures 3 and 4 into Equation 15, we calculated the exposure, reactivity, interaction, and unmediated components. The exposure component is .8 units, indicating that high-neuroticism respondents would experience a mean level of distress .8 units lower if we held their reactivity to all stressors constant, yet lowered their exposure to those stressors to the level experienced by low-neuroticism respondents. The reactivity component is 1.7 units, indicating that high-neuroticism respondents would experience a mean level of distress 1.7 units lower if we held their exposure to all stressors constant but lowered their reactivity to those stressors to the level of low-neuroticism respondents. Note that this component is twice as large as the exposure component.

The interaction component is only −0.1 units, too small to affect

our conclusions about exposure and reactivity to daily stressors. Exposure and reactivity to environmental stressors account for $[.8 + 1.7 + (-0.1)]/5.8 = 41\%$ of the overall mean difference in distress; thus, 59% of the mean difference in distress is not attributable to environmental events.[5]

*Contribution of particular stressors to the neuroticism-distress relationship.* Not all events contributed equally to explaining the neuroticism-distress relationship. We noted earlier that high-neuroticism respondents were consistently more reactive than low-neuroticism respondents to overloads at home and at work. In addition, high-neuroticism respondents were more exposed and more reactive to adult interpersonal conflicts, namely, arguments with spouse and with others. These interpersonal conflicts are the most important daily stressors in explaining the neuroticism-distress relationship. The two types of conflict account for 1.19 units or 50% of what we were able to explain of the neuroticism-distress relationship. The two overloads account for .95 units or 40% of the explained portion of the neuroticism-distress relationship.

## Results Using Between-Person Estimates of Stress-Reactivity

In this study we used time-series data to calculate within-person estimates of reactivity to stressors. It is instructive to briefly compare the results obtained using the within-person approach with the results that would have been obtained had we adopted the more conventional between-person approach. Reactivity is defined as the difference in distress associated with a unit change in exposure to stressors. Between-person estimates of reactivity are obtained by regressing individual differences in distress on individual differences in exposure to stressors. Between-person estimates of effects need not be similar to within-

5. In response to a reviewer's query, we examined the extent to which the neuroticism-distress relationship found in this study could be attributed to the overlap between neuroticism and self-esteem. We reran all analyses controlling for self-esteem (measured by the Rosenberg scale). Controlling for dichotomized self-esteem, the original neuroticism effect on mean daily distress was reduced from 5.8 units to 5.4 units. In addition, our estimates of exposure and reactivity effects were virtually unchanged. This suggests that the neuroticism-related exposure and reactivity processes identified in this study are independent of self-esteem.

person estimates. In the present study, we found that the effects of daily stressors and their interactions with neuroticism explained 26% of the within-person variance in distress, whereas these variables explained 41% of the between-person variance in distress.

Predictably then, when between-person estimates of reactivity are substituted into the distress-decomposition equation (Equation 15), we explain a greater proportion of the mean difference in distress. Instead of explaining 41% of the neuroticism effect, we now explain 72% of the effect. The decomposition is as follows: 5.8 (total effect) = 2.2 (exposure) + 3.0 (reactivity) − 1.0 (interaction) + 1.6 (residual). In this decomposition, reactivity and exposure effects are relatively similar in size. Furthermore, daily stressors largely account for why neuroticism is associated with distress in daily life: The residual portion is only 28% of the effect (compared with 59% in the previous decomposition). Note that the difference between the two decompositions is due purely to differences in estimates of reactivity: The values for mean distress and mean exposure to stress used in the two decompositions are identical.

## DISCUSSION

This study has five main findings: (*a*) We have shown that reactivity to stressors is twice as important as exposure to stressors in explaining the relationship between neuroticism and distress in daily life. (*b*) Interpersonal conflicts with other adults are the most important types of daily stressors in explaining this relationship. (*c*) High-neuroticism respondents are no more likely than low-neuroticism respondents to show emotional reactivity on days following a stressful event; thus speed-of-recovery differences do not explain the neuroticism-distress relationship. (*d*) Fifty-nine percent of the neuroticism-distress relationship is not mediated by either exposure or reactivity to stressors. (*e*) Using a within-subject approach to assess stress reactivity leads to conclusions very different from those that would be obtained using a more conventional between-subjects approach. We discuss each of these findings in turn.

### Exposure versus Reactivity

In recent years, considerable attention has been paid to the importance of situation selection in determining the outcomes of personality. In this

article we captured this situation selection process in terms of exposure to common daily stressors. We do not wish to imply, however, that all of the stressors the subjects reported in this study were self-selected. People are often exposed to stressors for reasons beyond their control. Yet despite using a liberal definition of selection as exposure to stressors, we found that stressor reactivity was twice as important as stressor exposure in explaining the neuroticism-distress relationship.

Because we did not obtain detailed information on the context in which subjects were exposed to daily stressors, we cannot disentangle the various processes that might underlie the exposure effect. For example, we have not distinguished between chosen and imposed situations in the present study, and it is known that personality is a stronger predictor of exposure to chosen situations than to imposed situations (Emmons et al., 1986). The effect of neuroticism on stress exposure that we find, then, is a result of averaging across chosen and imposed situations. More fine-grained research on this topic is clearly necessary.

How do neuroticism-based differences in reactivity come about? We think they arise primarily through what people do within stressful situations, specifically, through high-neuroticism subjects' use of less effective coping mechanisms when confronted with stress. In a longitudinal study, Bolger (1990) has shown that neuroticism predicts emotional reactivity to a major examination, and that this reactivity can be explained as the result of ineffective coping efforts, i.e., wishful thinking and self-blame.[6,7]

## Interpersonal Conflicts and the Neuroticism-Distress Relationship

We found that just two of the nine stressors—conflicts and tensions with spouse and with other adults—accounted for half of the explained

6. Not all studies have found self-blame to be an ineffective coping mechanism. Several cross-sectional studies (Bulman & Wortman, 1977; Tennen, Affleck, & Gershman, 1986; Timko & Janoff-Bulman, 1985) have shown that self-blame is associated with lower distress.

7. McCrae and Costa (1986) have also linked neuroticism to ineffective coping with stress, although they argue that coping does not mediate the effects of neuroticism and distress (see Bolger, 1990, for a discussion of this issue). More generally, research on personality and adaptation to stress has increasingly emphasized the mediating effects of coping efforts (see, for example, research on optimism by Scheier, Weintraub, & Carver [1986] and locus of control [Parkes, 1984]).

portion of the neuroticism-distress relationship. Although not generally considered to be a defining feature of neuroticism, interpersonal conflict appears to be one of the key mechanisms linking this disposition to distress in daily life. As such, these results are consistent with those of Buss et al. (1987), who found that among married couples neuroticism predicts the use of conflictual tactics of manipulation.

More generally, it is becoming clear that conflicts and tensions with others are the principal sources of daily stress. In a previous article, Bolger, DeLongis, Kessler, and Schilling (1989) found that interpersonal conflicts were by far the most important stressors in terms of their effects on daily distress. As in the case of daily stress studies in general, future studies on the effects of neuroticism should pay particular attention to interpersonal conflicts as mediators.

## Neuroticism and Speed of Recovery from Daily Stressors

It seemed plausible to us at the outset that some of the aggregate relationship between neuroticism and distress might be because persons high in neuroticism take longer than those low in neuroticism to recover from daily stressors. Previous research has shown that factors such as social support and level of chronic stress affect recovery speed, such that those with the least support and with the greatest levels of chronic stress show subsequent-day effects of daily stressors (Caspi et al., 1987). Analogously, we thought that high-neuroticism individuals might have a slower recovery speed than low-neuroticism individuals. However, we found no tendency for high-neuroticism individuals to remain relatively more distressed on days following an event.

It is important to note that these results do not rule out the possibility that neuroticism affects recovery speed within days. Thus persons low in neuroticism might show no aftereffects of marital arguments within several hours following the event, whereas persons high in neuroticism might remain distressed for the rest of the day. Because we obtained only one measurement each day we were unable to test this possibility.[8]

8. Note, however, that despite using three repeated measurements per day, Larsen and Kasimatis (1991) found no relationship between neuroticism and the duration of physical symptoms.

### Unmediated Portion of the Neuroticism-Distress Relationship

Almost 60% of the neuroticism-distress relationship is not mediated by daily stressful experiences. This finding partially supports Watson and Clark's (1984) view that neuroticism—or negative affectivity—is the disposition to experience distress even in the absence of environmental stressors. To the extent that we have not adequately measured common daily events, of course, the size of this component will be overestimated. Nonetheless, it appears that a substantial portion of the daily distress associated with neuroticism is not explained by daily variation in minor, negative events.

Although the focus of this study was on minor stressors, we can also rule out major life events as possible mediators of the residual portion of the neuroticism-distress relationship. Fewer than half of 1% of the person-days in the study involved any major life events (e.g., becoming unemployed; see Bolger, DeLongis, Kessler, & Schilling, 1989). Thus, major events were sufficiently rare that it is unlikely they contributed to explaining the neuroticism-distress relationship.

### Between-Person versus Within-Person Results

We found that by assessing stressor reactivity in a between-person fashion we were able to explain 72% of the neuroticism-distress relationship. This compares favorably with our ability to explain 41% of the relationship using within-person estimates of reactivity. The between-person results lead to substantive interpretations very different from those of the within-person results. The between-person results imply that daily stressors largely explain why neuroticism and distress are related, whereas the within-person results imply that the majority of the neuroticism-distress relationship is unexplained by daily stressors. Furthermore, the between-person results imply that exposure and reactivity processes are of comparable importance in explaining the neuroticism-distress relationship, whereas the within-person results imply that reactivity is distinctly more important than exposure in explaining the relationship.

We believe that the within-person results are more accurate than the between-person results. Unlike between-person estimates, within-person estimates of stress reactivity cannot be influenced by omitted between-person variables. We suspect that the between-person results

are inflated and distorted because of omitted between-person factors (such as poor living conditions) that are correlated both with individual differences in stressor exposure and with individual differences in distress.[9]

In general, this problem is equivalent to the "ecological fallacy" of assuming that correlations between variables defined at aggregate levels such as states or countries (e.g., between unemployment and crime rates) give us accurate estimates of correlations at the individual level (between being unemployed and committing a crime; see Langbein & Lichtman, 1978). In the same way, it is unwise to assume that correlations between variables defined at the between-person level may give us accurate estimates of correlations between the same variables if they were measured within persons over time. Yet often there is the temptation to draw conclusions about within-person relationships even when data are measured at the between-person level only. Although not necessarily true in general, it is significant that ecological correlations are often higher than their equivalents at the individual level, just as we observed that between-person estimates of stressor reactivity were substantially higher than their equivalents at the within-person level.

## Limitations of the Study

Inferences from this study are limited in several ways. First, we have studied people at a time of relative equilibrium, i.e., during a typical 6-week period. As noted earlier, fewer than half of 1% of the person-days in our study involved any major life events. To adequately explore exposure and reactivity to major life events, it would be necessary to study people over longer periods of time or to design studies around major life transitions. Therefore we cannot assume that the relative importance of exposure versus reactivity for the neuroticism-distress relationship will be the same for major events as it is for minor events.

It is also worth noting that the types of daily stressors people commonly experience may be constrained by such prior life events and

9. Although the within-person estimates of stressor reactivity are smaller than equivalent between-person estimates of reactivity, they are still substantial. As such they further illustrate that daily stressful events have potent effects on well-being even when one controls for individual difference variables such as neuroticism. For an excellent discussion of this and related issues see Lazarus (1990) and associated peer commentaries.

transitions. For example, a person may be in a chronically unpleasant, unrewarding job because prior economic conditions led to the loss of a pleasant, rewarding job. Ideally, then, one would like to understand the selection processes that led people into the equilibrium conditions in which we found them.

A second limitation arises from our use of a crude, checklist approach to measure daily events. It is possible that the stressor reactivity effects we found may be due, in part, to stressor heterogeneity. Although our analyses controlled for the effects of between-event heterogeneity (i.e., arguments with children are less severe than arguments with adults), we could not control for unmeasured within-event heterogeneity (e.g., some arguments with spouse may be more severe than others). Accordingly, it may be that neuroticism appears to influence stressor reactivity, in part, because it influences exposure to objectively more severe versions of the same event. In future research, one way to circumvent this problem would be to obtain detailed accounts of each stressful event and to use independent raters to code these events for severity. An example of such an approach is the system developed by Brown and his colleagues for the study of major life events (see Brown & Harris, 1978).

A final limitation concerns the adequacy of our list of daily stressors. Although we attempted to create a diary instrument that captured the most common daily stressors, we may have missed important categories of events—for example, neglect and inattentiveness in interpersonal relationships. Significantly, this type of "cold treatment" is characteristic of those high on neuroticism (Buss et al., 1987), and it is possible that such behavior evokes similar responses from the spouses of high-neuroticism individuals. If so, we may have missed important mediators of the neuroticism effect, and the unmediated portion of the neuroticism-distress relationship may be smaller than it appears to be in this study.

## CONCLUSION

Recent attempts to understand the relationship of personality to health and psychological outcomes have focused on situation-selection processes. Available evidence suggests that selection processes are important and statistically reliable phenomena. To date, however, no research has attempted to compare the importance of situation selection with

situation reactivity in determining the outcomes of personality. Using a naturalistic design, we examined this issue as it concerned the relationship between neuroticism and distress in daily life. For this personality-outcome relationship, we found that selection processes—as indexed by exposure to daily stressors—were, at best, half as important as reactivity processes.

Whether this pattern of results holds for other personality measures and for other health and psychological outcomes is presently unclear. Future studies need to address this issue. To this end, the design and methodology used here can readily be applied to other personality-outcome relationships. For the neuroticism-distress relationship, however, it appears that our psychological well-being is shaped more by how we react within stressful situations than by our exposure to those situations.

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