Stability and Change in Psychological Distress and Their Relationship With Self-Esteem and Locus of Control: A Dynamic Equilibrium Model

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The proposed model assumes (a) that each person has a stable and characteristic symptom level, (b) that external events act to deflect symptom levels from this stable level, and (c) that adaptive mechanisms tend to normalize these deviations. The model is used to examine (a) the dynamics of psychological distress (PD), (b) the role of personality traits (i.e., self-esteem and locus of control), and (c) the contamination of these traits by the current level of PD. The analyses show that the structural model adequately fits data of 2 longitudinal community studies. Two thirds of the variance in distress could be attributed to differences in stable symptom levels, leaving 1/3 for environmental change agents. Both personality traits were substantially contaminated by PD levels. Finally, high symptom levels were strongly related to low self-esteem and external control. The applications of the model and the origins of stability and change in PD are discussed.

In the last decade, a number of longitudinal epidemiological community studies have been conducted that suggest some degree of stability in psychological symptom levels over time (Aneshensel, 1985; Brown, Andrews, Harris, Adler, & Bridge, 1986; Costa, McCrae, & Zonderman, 1987; Henderson, Byrne, & Duncan-Jones, 1981, Ingham, Kreitmier, Miller, Sashidharan, & Surtess, 1986, Kessler, Price, & Wortman, 1985 [review]; Kobasa, Maddi, & Courington, 1981, Lin & Ensel, 1984; McFarlane, Norman, Streiner, & Roy 1983; Ormel, 1980; Pearlman, Lieberman, Menaghan, & Mullan, 1981; Tennant, Bebbington, & Hurry, 1981; Warheit, 1979; Williams, Ware, & Donald, 1981) whereas many subjects remain at relatively stable (generally low) symptom levels, others show marked symptom change. Similar observations have been made in the broader domain of negative affect (Costa & McCrae, 1980; Watson, 1988a; Watson & Clark, 1984).

Such findings pose the problem of explaining the processes by which symptom levels wax and wane over time. Heady and Wearing (1989) distinguished three types of models that try to shed some light on this issue. Personality models assume that symptom levels depend primarily on personality factors. Exogenous models emphasize the disruptive effects of exogenous life events. Finally, adaptation models are based on the assumptions that major life events cause changes in symptom levels and that these activate adaptive mechanisms that seek to reduce the negative impact of these events. Adaptation models imply that people adapt so rapidly and completely to life events that practically no impact on psychological symptoms can be observed. Because all three models have theoretical drawbacks and because empirical support in favor of the models is equivocal, Heady and Wearing (1989) propose an alternative dynamic equilibrium model. They argue that it is reasonable to consider that each person has his or her normal level of life events and normal symptom level, as life event scores and symptom levels tend to be fairly stable over time. Both so-called "equilibrium levels" can be predicted on the basis of stable personality characteristics, such as neuroticism, as has been demonstrated in many empirical studies. Because of exogenous forces, the pattern of life event exposure may change, which in turn may lead to changes in symptom level. In other words, exogenous forces cause life events and symptom levels to deviate from their characteristic normal equilibrium levels. These changes are likely to be temporary, however, because stable personality characteristics will usually ensure that the levels of life event exposure and symptoms return to their normal levels. Contrary to what is assumed in adaptation models, this process of returning to normal equilibrium levels takes time and is accompanied by an observable impact on the psychological symptoms.

The stability and change model that we developed, which is presented in this article, is essentially a dynamic equilibrium model. In its present form, however, the stability and change model does not include measures of event exposure. It can, however, be extended to do so, as is made evident in the discussion.

Our attempt is threefold. First, we describe the model of stability and change. This model assumes that each person has
a stable and characteristic symptom level. In addition, it assumes that environmental (and other) change agents act to deflect symptom levels from this stable, characteristic level and that adaptive mechanisms tend to neutralize the effects of change agents and to restore the characteristic symptom level. The model represents the causes of symptom change and stability in terms of nonobserved latent variables, reflecting the effects of change agents and stable attributes of the person and his or her environment. The fundamentals of this model have been proposed by Duncan-Jones (1985), Duncan-Jones, Fergusson, Ormel, and Horwood (in press), and Ormel (1983). Subsequently, the model is extended by introducing two personality variables, self-esteem and locus of control, which are modeled as potential causes of between-subjects differences in stable, characteristic symptom level. It has often been suggested that these two personality factors are related to high symptom levels (low self-esteem: Brown et al., 1986; Emmons & Diener, 1985; Ingham et al., 1986; Pearlin & Schooter, 1978; Tyler, 1978; external locus of control: Abramowitz, 1969; Emmons & Diener, 1985; Johnson & Sarason, 1979; Kobasa et al., 1981; McFarlane et al., 1983; Moyal, 1977; Turner & Nosh, 1983; Wheaton, 1980). However, there is evidence indicating that the measurement of self-esteem and locus of control is contaminated by current mental health state, at least in as far as patient samples are concerned (Bianchi & Fergusson, 1977; Hirschfeld & Klerman, 1979; Ingham et al., 1986; Koeter, Ormel, Van den Brink, & Dijkstra, 1989). Consequently, the correlation between symptom levels and personality measures has an equivocal interpretation, because it cannot be established which part of the association reflects contamination and which part reflects real effects. We show that unbiased estimates of both contamination and real effects can be obtained using our stability and change model. Finally, the model is fitted to data from two longitudinal studies: a random sample of 226 subjects from the general Dutch population and a cohort of 389 non-university students. Because both studies used different samples, different numbers of follow-up, and different measures of the same constructs, we can assess the robustness of the model.

Theoretical Background

Symptom Stability and Change

Consider a sample of subjects studied at a series of at least three different points in time, \( t_1, t_2, \ldots, t_n \). Assume that symptom levels are measured without error by a variable, \( S_t \), which represents the level of psychological symptoms reported at time \( t \). (Later we deal with measurement error.) The stability and change model assumes that the subject's symptom level remains perfectly stable in the absence of change agents at his or her characteristic level. It can be expressed by the following general equation:

\[
S_t = SS_t + C_t
\]

(1)

in which \( S_t \) denotes the symptom level of the \( t \)th subject at the \( t \)th time, \( SS_t \) indicates the nonobserved stable and characteristic symptom level for the \( t \)th subject, and \( C_t \) denotes symptom change resulting from the effects of nonobserved change agents that cause the symptom scores, \( S_t \), to deviate from the stable and characteristic value, \( SS_t \). In the absence of change agents, \( C_t \) equals zero.

The observed symptom scores are thus functions of two nonobserved latent variables: the stable, characteristic symptom level (or symptom stability, \( SS \)) and the symptom change variable (\( C_t \)). The symptom stability variable reflects the effects of stable attributes of the person and his or her social environment that cause the subject to have a characteristic and stable symptom level. Such attributes could include personality traits, such as neuroticism (Eysenck & Eysenck, 1975) or negative affectivity (Diener & Larsen, 1984; Watson & Clark, 1984) or pervasive social and economic conditions (Brown et al., 1986; Costa et al., 1987). In addition, we assume that adaptive mechanisms are activated when symptom change occurs, which will act to restore and sustain symptoms at their normal level (cf. the dynamic equilibrium assumption). The effectiveness of the adaptive mechanisms depends on the psychobiological make-up of the person and, perhaps, on stable environmental factors. Psychotherapy and social support can be considered to foster these adaptive processes (Duncan-Jones et al., in press). The latent symptom-change variable, on the other hand, reflects the effects of various transitory social, psychological, and biological events, here denoted as change agents.

The problem posed by Equation 1 is that of estimating the subject's stable and characteristic symptom level and hence the deviation from this characteristic level at time \( j \). Such an estimation requires that certain assumptions are made. Following Duncan-Jones (1985), we impose two assumptions that will serve to identify the model described in Equation 1. (a) The subject's characteristic symptom level is statistically independent of symptom change. This implies that the nonobserved variables \( SS \) and \( C_t \) are uncorrelated. (b) The subject's deviation from the stable, characteristic level at any point in time \( C_t \) depends on the deviation at the previous point in time and on the extent of nonnormal exposure to change agents during the interval. As there are two assumptions, the across-time structure of the symptom-change variables, \( C_t \), is modeled as a first-order autoregressive process: (a) The effects of change agents decay gradually, and (b) the adaptive mechanisms, activated by symptom change, need time for their normalization processes. This autoregressive component of the model may be described as the following:

\[
C_t = B \times C_{t-1} + E_t
\]

(2)

in which \( C_t \) denotes the symptom-change level at time \( j \), \( C_{t-1} \) is the corresponding variable at time \( j - 1 \), \( B \) is a regression coefficient that describes the relationship between change at time \( j - 1 \) and time \( j \), and \( E_t \) represents the component of \( C_t \) that is not explained by the auto-regressive process and that results from exposure to actual change agents in the interval. The \( E_t \)'s are assumed to be uncorrelated.

The structure of relationships implied by these assumptions
can be seen most readily from the left part of the path diagram in Figure 1. The model shows that at any time, the observed symptom scores, $S_j$, are functions of two nonobserved latent variables, $SS$ and $C_j$.

The coefficients in Figure 1 describe the model in terms of standardized parameters and latent variables with means of zero and unity variance. $^2$ (The raw score variables are the freestanding variables.) In this standardized model, a certain proportion of variation in symptoms is explained by a common latent variable $SS$ (stable, characteristic symptom level), which influences symptom levels at all time periods. The squares of the standardized maximum likelihood estimates—parameters linking the stable, characteristic symptom variable to the symptom levels at time $j$—provide estimates of this proportion. This common factor model is supplemented by a first-order autoregressive model that describes the dynamics of symptom change over time and accounts for the effects of adaptive mechanisms and change agents.

**True Scores and Measurement Error**

For the sake of simplicity, we assume that two independent indicators of the level of symptoms are available at each time period. The measurement model we used assumes that the variation in the observed indicators of symptom level reflects variation attributable to two nonobserved variables: the "true" symptom level and random errors of measurement (Alwin & Jackson, 1982). More formally, this true score model may be described as the following:

$$s_{ij} = \lambda_{i} \times S_{ij} + U_{ij}$$  \hspace{1cm} (3)$$

in which $s_{ij}$ denotes the observed symptom level for the $i$th indicator observed at the $j$th time period, $S_{ij}$ is the nonobserved true distress level at time $j$, and $U_{ij}$ denotes random errors of measurement. The parameter $\lambda_{i}$ indicates the regression on the true score $S_{ij}$ of the indicator $s_{ij}$. It is assumed that the errors of measurement $U_{ij}$ are uncorrelated with the true score $S_{ij}$ and with each other. With the help of the true score model (not

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$^2$ In the unstandardized parametrization of the stability and change model, the $B_1$, $B_2$, structural coefficients are fixed at unity, and the variances of $SS$ and $C_j$ ($\sigma^2$ in LISREL) are estimated. The $B_1$ and $B_2$ and $E_j$ are also estimated. If the intervals between $T_j$ are of equal length and if the sample is obtained from a population in which mean symptom levels and variances did not change substantially during the period under study, the $B_1$ and $B_2$ may be set equal as well as the $E_j$. The disturbance terms of the true symptom score variables ($S_{ij}$) are fixed at zero.
displayed for reasons of economy), the covariances among the unobserved true symptom scores can be estimated (see Tables 2 and 5).

**Personality and Symptoms**

Extending the model with personality measures may provide insight into the causes of between-subjects differences in stable, characteristic symptom levels. However, introducing personality factors into the model is not as simple as it may look at first glance. The measurement of personality traits may be contaminated by the subject's mental health state at that particular time, as has been stated in the introduction. Therefore, the correlation between personality and symptom measures has an equivocal interpretation, because it contains correlation that is due to contamination. This problem can be dealt with by controlling for contamination of the measurement of personality by symptom levels. To include this control into the model, it is necessary for the personality trait (P) to be assessed at at least two points in time. The model, as presented in Figure 1, assumes that the two measures of P (i.e., P1 and P2) are influenced by the changing components of psychological distress at the corresponding time periods, C1 and C2. This additional assumption implies that the change agents that cause the symptom levels S_j to deviate from their stable, characteristic values also cause the personality measures P_j to deviate from the subject's true (and stable) level of P. Thus the model compensates for sources of variance in the measures of P that are related to changing symptom levels. The coefficients \( \lambda_1 \) and \( \lambda_2 \) represent estimates of the degree of contamination. The coefficient \( \phi \) represents the uncorrelated relationship between the trait and the stable, characteristic symptom level.

Later both models are fitted to two sets of longitudinal data. First we describe the two data sets.

**Method**

**Study 1**

**Subjects and Design**

Three interviews were conducted—in 1976 (T1), in 1977 (T2), and in 1984 (T3)—with 226 Dutch adults (52% men, 48% women; age in 1976 \( M = 39, SD = 12, \text{range} = 22–65 \)). Fifty-seven percent were employed, 35% were housewives, and 8% were unemployed or retired. With regard to education, 28% had attended primary school only, 25% completed technical or vocational training, 34% attended high school, and 13% had a college or similar degree. Of the 384 originally selected subjects, 258 (67%) completed all three interviews. These 384 subjects were randomly selected from a list of 1,622 names, representing a 1 in 5,000 sample of the Dutch adult population (Ormel, 1980; Ormel, 1983; Ormel, Sanderman, & Stewart, 1988). Most of the nonresponse occurred at T1. Of those interviewed at T1, 86% were reinterviewed at T2 and T3. Death and emigration accounted for nearly 25% of the nonresponse. Because information on one or more symptoms or personality items was lacking at one or more points in time for 32 people, the analyses presented here are based on 226 subjects. Comparison of responders and nonresponders revealed no major differences on a broad range of sociodemographic, distress, and personality-related variables—except for age and external locus of control, which were associated with nonresponse.

**Measures**

**Psychological distress (S)** Two instruments were used to assess the nonspecific psychological symptoms: (a) the self-report Negative Affect (NA) subscale from Bech-Neufeldt's (1968) Affect Balance Scale and (b) the Psychological Symptom (PS) section of the Groningen Symptom Schedule, a semistructured interview covering 14 symptoms (Ormel, 1980, 1983; Ormel et al., 1988). The time frame for both instruments was the 4 weeks prior to the interview. PS items included nervous tension, depressive mood, and worrying. Interviewers rated each symptom on a 5-point scale, ranging from 0 if the symptom was absent during the previous 4 weeks to 4 if the symptom had been present for more than a fortnight. Only if the symptom had bothered the subject for more than half of the day did it rate present for that day. At T1 and T2, the same interviewers administered the symptom schedule. At T3, 7 years after T2, for the most part different interviewers carried out the interview. NA items include restlessness, unhappiness, loneliness, and feeling upset because of criticism. The original NA item on boredom was excluded, as it did not fit into the scale. Each symptom was rated according to the frequency it was experienced during the previous 4 weeks on a scale ranging from 0 (never) to 4 (daily) or higher.

Reliability of both scales (NA, PS) as measured by Cronbach's alpha, fluctuated around .75 (see Table 1). Details regarding the measurement procedures and the validity of the measures have been presented elsewhere (Ormel, 1980, 1983; Ormel et al., 1988). The NA, and PS measures are used as indicators of psychological distress.

**Locus of control (LC)** Andressen and Van Cadsand's (1983) 14-item version of Rotter's Locus of Control Scale was used. A high score on the scale represents an internal locus of control (i.e., the generalized belief that things happening in one's life can be influenced). A low score represents an external locus of control. Validity studies with several samples (\( N = 2,038 \)) indicated that internals have higher achievement motivation, are more extraverted, sociable, and active and are less neurotic and dogmatic than externals. This picture is consistent with the literature (cf. Lefcourt, 1976). The inventory was administered at T2 and T3; Cronbach's alphas were .81 and .83, respectively (see Table 1).

**Self-esteem (SE)** This attribute was measured with the 17-item Zelf-Waardering (ZW—self-esteem) scale from the Dutch Personality Inventory (Luteyn, Starren, & Van Dyk, 1985), administered at T1 and T3. Cronbach's alphas were .73 and .75, respectively (see Table 1). According to the test manual (pp. 14–15), the ZW scale correlates substantially with other measures of self-esteem and with related concepts including ego strength and vigor.

**Study 2**

**Subjects and Design**

In 1984, questionnaires were mailed to 1,524 students who would graduate in the next 6 months from 16 professional schools in the northern Netherlands. The response rate was 42% (63% men, 37% women; age in 1984 \( M = 23, SD = 2, \text{range} = 21–33 \) years), and the sample of respondents represented representative for the population of Dutch professional graduates with respect to gender and type of education (Schaufeli, 1988). Four follow-ups were conducted after graduation with 6-month intervals (T2 to T5). Drop-out rates ranged from 7% to 20%. In total, 411 respondents participated in every follow-up. These participants did not differ significantly from the dropouts with respect to age, gender, type of training, level of psychological distress, self-esteem, and locus of control (Schaufeli, 1988). Twenty-two respondents were excluded from the analyses because of missing data, leaving a final sample of 389 respondents.
Measures

Psychological distress. Psychological distress over the previous week was measured using the 17-item PSYCH subscale of the Dutch version of the Hopkins Symptom Checklist (HSC; Luteyn, Hamel, Bouman, & Kok, 1984). Instead of the five original HSC-symptom dimensions found in American samples, the Dutch version contains only two dimensions (i.e., psychological distress and psychosomatic distress). According to the test manual (pp. 13-15), the PSYCH scale correlates substantially positively with self-reported psychological symptoms, with ratings of psychologists, and with other questionnaires, including the Depression subscale of the Minnesota Multiphasic Personality Inventory.

Moreover, Watson, Clark, and Tellegen (1988) stated that the HSC was "roughly interchangeable" (p. 1068) with their NA scale, suggesting that psychological (psychosomatic) distress and negative affectivity are conceptually closely related. Values of Cronbach's alpha of the PSYCH scale ranged in our study from 89 to 92 (see Table 5). For each of the five time periods, the items were randomly divided into two sets to obtain two indicator measures of the true level of psychological distress at each time period ($\delta_{ij}, \delta_{i+1,j+1} = 1, 2, 3, 4, 5$). By applying such a randomization procedure across time, correlated measurement errors are very unlikely (Schaufler, 1988). Table 4 shows the alpha values, ranging from 75 to .81.

External locus of control. Only those eight items from the Dutch version of Rotter's Locus of Control scale (Andressens & Van Cadsand, 1983) that describe an external locus of control were administered at T3 and T5. Cronbach's alpha was 74 and .81, respectively (see Table 4).

Self-esteem. Self-esteem was measured at times T3 and T5 by a six-item selection from the Dutch version of Rosenberg's (1965) Self-Esteem Scale (Helbing, 1982). The construct validity of the SE scale is satisfactory as indicated by the positive relationships between self-esteem and the pattern of correlations with personality dimensions (e.g., from the Edwards Personality Preference Schedule; Helbing, 1982). In our study, alpha coefficients were .93 and .92 at T3 and T5, respectively (see Table 4).

Results

Study 1

Because the data fitting was in part repetitive, we describe the procedure followed in Study 1 in detail and use the results as a baseline for the second data set. Two models were fitted to the variance/covariance matrix of the observed variables, using LISREL VI (Joreskog & Sorbom, 1985): the true score model and the stability and change model. The means, standard deviations, and correlations of the observed raw data of Study 1 are shown in Table 1.

Table 1 indicates that as expected, the personality traits were fairly stable over time. LC had a stability coefficient of .64 over a 7-year period, SE had a coefficient of .65 over a period of 8 years. Moreover, this stability was higher than that of the distress measures across the corresponding intervals ($r = .44$ and .57 over the 7-year period; $r = .37$ and .52 over the 8-year period). Both symptom measures (NA and PS) were substantially correlated ($r = .69, .62, and .56$ at T1, T2, and T3, respectively), thereby providing support for their construct validity.

True Score Model

The true score model (not shown for reasons of economy) yields optimal estimates of (a) the six regression coefficients linking the observed indicators, $PS_j$, $NA_j$ ($j = 1, 2, 3$), to the corresponding true symptom variables, $x_j$, and (b) the across-time covariances between these symptom variables. The standardized regression coefficients are moderately high, ranging between .74 and .83, suggesting a reasonable reliability for the indicators. The estimated across-time correlations between the $\delta$s are displayed above the diagonal in Table 2. Compared with the correlation between the corresponding overall raw score indexes (PD) below the diagonal in Table 2, the true score correlations are higher because of the correction for measurement error implicit in the true score model.

The pattern of both estimated and raw score correlations suggests that common factors influence distress levels and that at the same time, there is a tendency for distress levels to shift with increasing time intervals. This is consistent with the structure of the stability and change model.

Stability and Change Model

The estimates presented in Figure 2 are standardized and concern the relationships between the latent variables. The autoregression parameter covering the 7-year interval between $C_2$ and $C_3$ has been constrained to equal the 7th power of the autoregression between $C_3$ and $C_4$. The reason for doing this is that the two intervals in this study are of unequal length; the second interval is approximately 7 times as long as the first interval.

The standardized estimates can be interpreted as follows:

- The coefficients linking the true symptom levels to the symptom stability factor are estimates of the correlation between true symptom scores and the symptom stability factor. The coefficients range from .79 to .86, suggesting that between 63% and 75% of the variance in true distress scores represents between-subjects variation in stable, characteristic distress level. Accordingly, between 25% and 37% of the variance in the true distress scores is attributable to the effects of (nonobserved)

\[3\] Specification of the model in LISREL is complicated. Details can be obtained from Johan Ormel. The specification was as follows: Measurement part of the model (a) To scale the latent variables, we fixed at unity one of the regression coefficients linking the latent variable to its observed indicator measures (lamas in LISREL). (b) Both regression coefficients linking the personality trait to its indicators were fixed at unity. All other regression coefficients linking latent and observed variables including the contamination coefficients ($E_j$), as well as all measurement error variances, were not restrained and thus were estimated. Structural part of the model See Footnote 2.

\[4\] The measurement error terms corresponding with the Negative Affect (NA) indicators appeared to have a low correlation over time (circa 0.10). These correlations are theoretically justifiable because they show that the NA indicators are correlated over time independently of the true symptom level of the subject. This suggests the presence of systematic method variance. To account for this method variance, a so-called instrument factor (not shown in Figures 2 and 3) on the NA indicators was introduced into the model (Joreskog & Sorbom, 1982) By this, one degree of freedom was lost.

\[5\] This constraint could be handled in COSAN (McDonald, 1983) but not in LISREL. The COSAN analysis showed that the 7-year autoregression was close to zero Therefore, we fixed $b_9$ at zero in the LISREL analysis.
Table 1
Observed Means, Standard Deviations, Correlations, and Cronbach's Alpha Coefficients (on the Diagonal) of Study 1 Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>M</th>
<th>SD</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. PS:T1</td>
<td>3.32</td>
<td>3.24</td>
<td>.69</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>2. NA:T1</td>
<td>2.31</td>
<td>2.36</td>
<td>.69</td>
<td>.70</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. PS:T2</td>
<td>3.33</td>
<td>3.46</td>
<td>.52</td>
<td>.47</td>
<td>.75</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>4. NA:T2</td>
<td>2.05</td>
<td>2.02</td>
<td>.54</td>
<td>.62</td>
<td>.62</td>
<td>.72</td>
<td></td>
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<tr>
<td>5. PS:T3</td>
<td>2.33</td>
<td>3.73</td>
<td>.37</td>
<td>.42</td>
<td>.44</td>
<td>.45</td>
<td>.79</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>6. NA:T3</td>
<td>1.77</td>
<td>1.89</td>
<td>.38</td>
<td>.52</td>
<td>.42</td>
<td>.57</td>
<td>.56</td>
<td>.69</td>
<td></td>
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<td>7. SE:T1</td>
<td>26.32</td>
<td>4.98</td>
<td>-.45</td>
<td>-.37</td>
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<td>-.35</td>
<td>-.27</td>
<td>-.24</td>
<td>.73</td>
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<td>8. SE:T2</td>
<td>26.81</td>
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<td>-.37</td>
<td>-.33</td>
<td>-.30</td>
<td>-.34</td>
<td>-.39</td>
<td>-.43</td>
<td>.65</td>
<td>.75</td>
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<tr>
<td>9. LC:T2</td>
<td>33.61</td>
<td>7.44</td>
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<td>-.23</td>
<td>-.29</td>
<td>-.29</td>
<td>-.18</td>
<td>-.24</td>
<td>.15</td>
<td>.13</td>
<td>.81</td>
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<td>27.90</td>
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<td>-.12</td>
<td>-.14</td>
<td>-.10</td>
<td>-.25</td>
<td>-.22</td>
<td>.07</td>
<td>.21</td>
<td>.64</td>
<td>.83</td>
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</table>

Note n = 226. PS = Psychological Symptom section of the Groningen Symptom Schedule, NA = Negative Affect subscale from the Affect Balance Scale; SE = Self-Esteem scale; LC = External/Internal Locus of Control Scale; T = time.

change agents that cause distress levels to deviate from their stable, characteristic level. Roughly speaking, the model shows that about two thirds of the variance in the true distress scores is explained by the latent variable reflecting the constant effects of stable person and environmental factors (i.e., the stable, characteristic symptom level), whereas the remaining third of the variance is explained by the latent variable reflecting the effects of environmental-change agents (i.e., the symptom-change variable).

2. The 1-year autoregression amounts to .30, suggesting that the distress-change variables are interrelated to each other by relatively weak autoregressive processes. Apparently, adaptive mechanisms largely succeed in eliminating the deviations from a person's stable and characteristic distress level within a period of 1 year.

3. The standardized coefficients linking the symptom-change variables to the self-esteem measures suggest that 3% to 10% of the variance in observed self-esteem scores can be attributed to the effects of symptom change. For locus of control, these percentages range from 5% to 7%. Thus, the change agents that lead to changes in levels of distress also lead to changes in reported self-esteem and locus of control.

4. The symptom-stability factor has a high negative correlation with self-esteem (−.59) and has a low negative correlation (−.32) with locus of control. Thus, higher stable, characteristic symptom levels are somewhat more frequently found among low-self-esteem and externally oriented subjects. Finally, self-esteem and internal locus of control have a low correlation (.18).

Table 2
Observed (Below Diagonal) and Estimated (Above Diagonal) Correlations Between Symptom Totals

<table>
<thead>
<tr>
<th>Symptom level</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 T1</td>
<td>.81</td>
<td>77</td>
<td>.65</td>
</tr>
<tr>
<td>2 T2</td>
<td>.62</td>
<td>81</td>
<td>.73</td>
</tr>
<tr>
<td>3 T3</td>
<td>.50</td>
<td>57</td>
<td>.82</td>
</tr>
</tbody>
</table>

Note n = 226. Cronbach's alpha (on the diagonal) and observed correlations concern the overall measure of psychological distress (negative affect and psychological symptoms for all three time periods). The estimated correlations were obtained from the true score model. T = time.

![Figure 2](image-url)

Figure 2: Fitted symptom stability and change model—standardized estimates for Study 1 (n = 226). SS = stable and characteristic symptom level, or symptom stability factor; SE = self-esteem at time j; LC = locus of control at time j; Tj = true symptom level at time j; PSj = psychological symptoms at time j; NAj = negative affect at time j; Cj = symptom-change variable, or changing component of symptoms at time j.)
Table 3
Measures of Fit for Study 1

<table>
<thead>
<tr>
<th>Model</th>
<th>$\chi^2$</th>
<th>$df$</th>
<th>$p$</th>
<th>GFI</th>
<th>Absolute NR</th>
</tr>
</thead>
<tbody>
<tr>
<td>True score</td>
<td>4.89</td>
<td>5</td>
<td>.43</td>
<td>.994</td>
<td>0.15</td>
</tr>
<tr>
<td>Symptom stability and change</td>
<td>21.95</td>
<td>27</td>
<td>.74</td>
<td>.981</td>
<td>0.31</td>
</tr>
</tbody>
</table>

Note $n = 226$. NR = normalized residual; GFI = goodness-of-fit index

Goodness of Fit

Table 3 shows the log-likelihood chi-squared statistic and Jöreskog and Sorbom's (1982) goodness-of-fit index (GFI). The GFI may range between 0 and 1; 1 indicates a perfectly fitting model. The GFI provides better insight into the fit of the model than the chi-squared statistic because the latter is rather sensitive to deviations from multivariate normality (Boomsma, 1983), and the observed variables were not normally distributed. The values of the two indexes, chi-squared and GFI, suggest an acceptable fit for the true score model and for the stability and change model.

The GFI's discussed in the previous paragraph assess the overall fit of the model. Further insight into the fit on the level of individual relationships can be obtained from the normalized residuals (NRs), which represent the divergence between the observed covariances and the covariances as predicted by the model. The NR is defined as the observed covariance minus the predicted covariance divided by the square root of its asymptotic variance. Absolute values of NRs larger than 2.0 in magnitude are indicative of specification error in the model. Mean and range of the absolute values of the NRs are presented in the last two columns of Table 3. Both mean values are quite low; their range indicates that the critical level of 2.0 is never exceeded.

The stability and change model predicts that the observed correlation between the personality indicators and the distress indicators are exclusively explained by (a) the correlation between the trait self-esteem (locus of control) and the stable, characteristic symptom level and (b) the contaminating effects of symptom change on reports of self-esteem (locus of control). This prediction can be verified by comparing the 24 observed correlations between the measures of personality and distress (see Table 1) with the 24 predicted correlations as obtained with the model displayed in Figure 2. The differences are very small indeed, which is reflected in the small NRs (absolute mean = 0.42; maximum = 1.03). Thus, the model appears to explain adequately the observed correlations between the measures of distress and personality.

Study 2

Although in Study 2, two additional waves were included, the specification of the model is similar to the procedure followed in Study 1.

Table 4 contains the means, standard deviations, and correlations of the observed raw data of Study 2. The split-half reliability of the PSYCH scale is satisfactory, ranging from .78 to .82. As expected, the 1-year stability of self-esteem (73) is somewhat higher than that of the psychological distress measures (ranging from .54 to .69). The stability of the locus of control (61), however, is within the latter range.

True Score Model

The standardized coefficients linking the symptom indicators to the true score symptom variables ranged between .87 and .92, suggesting good reliability for the indicators. The estimated correlations between the true score variables across time are displayed above the diagonal; the correlations between the overall distress indexes are displayed below the diagonal in Table 5.

The true score correlations are higher than the correlations between the raw score indexes, because of the correction for measurement error implicit in the true score model. Both the estimated and raw score correlations suggest that strong common factors influence distress levels, and at the same time the distress levels tend to shift with increasing time intervals. This is consistent with the structure of the stability and change model and the findings of Study 1.

Symptom Stability and Change Model

The autoregressive parameters were constrained within LISREL to be equal over the four 6-month intervals. All standardized coefficients displayed in Figure 3 differ significantly from zero and may be interpreted as follows:

1. The standardized estimates of the relation between true distress scores and the symptom-stability factor (ranging from .75 to .86) and between the symptom-change variables (.49 to .67) imply that approximately two thirds of the variance in true distress can be ascribed to differences in stable, characteristic distress levels, and approximately one third can be ascribed to the effects of nonobserved change agents. These estimates are remarkably similar to those found in Study 1.

2. The 6-month autoregressions range between .23 and .42. In Study 1, the 1-year autoregression was .30, which equals a 6-month auto-regression of .55. This suggests that the symptom-change variables in Study 2 are related by a slightly weaker autoregressive process than in Study 1.

3. Both self-esteem and locus of control are slightly contaminated by symptom change. The standardized coefficients link-
ing the self-esteem (external control) measures to the symptom-change variables indicate that approximately 7% (2%) of the variance in the measures of self-esteem (external control) can be attributed to changes in distress level that are due to the effects of change agents.

4. The pattern of correlations between the symptom stability factor and both personality factors is similar to that observed in Study 1, only the relations are stronger. Self-esteem and symptom stability are very highly negatively correlated (−.74). External control is highly positively correlated with the symptom stability factor (52). The most striking difference between both studies is that the two personality traits themselves are substantially more highly correlated in Study 2. Note that the locus of control measure used in Study 2 includes only external control items, whereas the locus of control variable in Study 1 includes both external and internal items. Selective item sampling may play a role, or the external trajectory of the internal–external continuum may be more strongly related to self-esteem than the total continuum. However, differences in strength of correlations of personality traits between both studies may also reflect differences in age between the samples.

Table 5

<table>
<thead>
<tr>
<th>Symptom level</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>.91</td>
<td>.75</td>
<td>.72</td>
<td>.66</td>
<td>.60</td>
</tr>
<tr>
<td>T2</td>
<td>.65</td>
<td>.80</td>
<td>.81</td>
<td>.69</td>
<td>.67</td>
</tr>
<tr>
<td>T3</td>
<td>.62</td>
<td>.70</td>
<td>.92</td>
<td>.70</td>
<td>.71</td>
</tr>
<tr>
<td>T4</td>
<td>.52</td>
<td>.51</td>
<td>.61</td>
<td>.88</td>
<td>.76</td>
</tr>
<tr>
<td>T5</td>
<td>.53</td>
<td>.60</td>
<td>.68</td>
<td>.67</td>
<td>.92</td>
</tr>
</tbody>
</table>

Note n = 389 Cronbach’s alpha and observed correlations concern the overall measure of psychological distress (psychological symptoms for all five time periods). The estimated correlations were obtained from the true score model, T = time.

Goodness of Fit

Although it is clear from Table 6 that both models fit less well than in Study 1, the chi-squared statistic should be interpreted with care, considering the large sample size and the deviations from multivariate normality (which both bias chi-squared upwards). The fit may, however, be considered acceptable for two reasons (Bentler & Bonnet, 1980): First, the chi-squared is lower than twice the number of degrees of freedom; second, the nonsignificant absolute normalized residuals are all well below the critical threshold of 2.0 (mean absolute NR = 0.50; maximum NR = 1.72).

To further examine the validity of the stability and change model, the deviations from the 40 observed (see Table 2) and model-based predicted correlations between the personality and distress indicators were examined. These deviations are small and nonsignificant (mean absolute NR = 0.59; maximum NR = 1.60). As in Study 1, the stability and change model adequately explains the observed correlations between the measures of distress and personality.

Discussion

It is remarkable how the findings of the two studies concur, considering the differences in samples, number of measurement occasions, and instruments. Somewhere in the region of two thirds of the true variance in psychological symptoms apparently has to be attributed to between-subjects variation in stable, characteristic symptom levels. Accordingly, approximately one third of the true variance in psychological distress arises from the effects of change agents that cause subject’s distress level to deviate from its stable, characteristic level. These findings suggest that personality models, which assume that symptom levels depend primarily on personality factors (Costa & McCrae, 1980), are more valid than exogenous models (Block & Zautra, 1981), which rely on the impact of environmental influences when explaining across-time changes in symptom levels. Neither of the models, however, provide any information
about the processes involved in the waxing and waning of symptoms over time. This issue is discussed at the end of this section.

The results of the two studies are also rather conclusive with regard to the found relationship between the personality traits of self-esteem and locus of control and distress. First, both studies found reported personality to be somewhat contaminated by current distress levels; the change agents that led to symptom change also influenced the reporting of locus of control and, in particular, self-esteem. This is in line with studies that report that self-esteem is significantly lowered during episodes of intense distress (Bianchi & Fergusson, 1977; Ingham et al., 1986; Koeter et al., 1989). Second, in both studies, between-subjects differences in self-esteem and locus of control appeared to be highly stable across time. Third, we found that all correlation between the measures of personality and distress that was not due to contamination could be accounted for by the relationship between the stable, characteristic symptom

Table 6
Measures of Fit for Study 2

<table>
<thead>
<tr>
<th>Model</th>
<th>$\chi^2$</th>
<th>df</th>
<th>p</th>
<th>GFI</th>
<th>M</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>True score</td>
<td>44.29</td>
<td>25</td>
<td>.003</td>
<td>978</td>
<td>.03</td>
<td>0.00-0.54</td>
</tr>
<tr>
<td>Symptom stability and change</td>
<td>129.09</td>
<td>70</td>
<td>.000</td>
<td>957</td>
<td>.50</td>
<td>0.00-1.72</td>
</tr>
</tbody>
</table>

Note: $n = 389$, NR = normalized residual, GFI = goodness-of-fit index.
level and the personality traits. This is an important finding, because it is an indication of the validity of the model and supports the trait notion of self-esteem and locus of control. The relationship between the stable, characteristic symptom level and low self-esteem can be interpreted in many ways. It may suggest a strong influence of self-esteem on stable, characteristic symptom level, but it may also be interpreted as the result of reciprocal causal processes, as the outcome of common factors, as operational confounding, or as an indication that symptom stability and self-esteem represent phenomena that are to some extent similar. In the case of locus of control, a causal interpretation faces similar problems. The model and the data are not suitable for solving these causal issues.

An important issue is the extent to which our results tally with those of others. Studies with three or more measurement points are rare. Two-wave studies are more prevalent (Aneshensel, 1985; Cronkite & Moos, 1984; Ferguson & Horwood, 1984; Henderson et al., 1981; Kanner, Coyne, Schaefer, & Lazarus, 1981; McFarlane et al., 1983; Monroe, 1982; Monroe, Imhoff, Wise, & Harris, 1983; Thoits, 1982; Vet & Ware, 1983; Warheit, 1979; Weissman & Myers, 1978; Williams et al., 1981). Although a variety of general and more specific measures of psychological distress were used in these studies, the observed 1-year correlations ranged between .30 and .70, with an average of around .50. Assuming reliability of around .75, which seems quite reasonable for scales measuring negative affectivity (cf. Watson, 1988b), correction for attenuation would raise this correlation of .50 to .67. This is somewhat lower than the true 1-year correlation found in the current studies (Study 1 \( r = .77 \); Study 2 \( r = .69 \) to .71). The small but systematic difference in correlations between the two studies was probably caused by different time frames; in Study 1, subjects were instructed to take the previous 4 weeks into account when completing the NA and PS scales, whereas in Study 2, only the last week was sampled. Watson (1988a) observed that longer time frames are associated with higher test-retest correlations in various negative affect measures.

Note that the estimated parameters, which constitute the basis for our conclusions concerning symptom stability and change, are not directly affected by the (split-half) reliability of the measures. As a matter of fact, the model estimates the measurement error that is due to unreliability. In the case of very unreliable measures, the model fit will probably be poor, so that the estimated parameters cannot be interpreted. If measurement-error terms are not correlated, the estimates of the relationships among the latent variables are not affected by variations in reliability of the observed variables, because these variations in reliability produce corresponding variations in the observed correlations (assuming the same true score correlation). The estimates only "correct" the correlation with regard to the deflatable effect of unreliability.

The model is based on assumptions that may be erroneous. In particular, the assumption that a subject's stable, characteristic symptom level does not change over time may be justifiable for protracted periods of time during adulthood but not for the life phases of adolescence and old age, during which developmental processes may have an effect. In addition, this assumption may not hold for people who have experienced events that result in permanent personality changes. Elaboration of the model in such an inflated way that it allows particular change agents to cause permanent changes in a subject's stable, characteristic symptom level would not only qualify the inertia assumption but also would render superfluous the assumption of statistical independence between the stable, characteristic symptom level and symptom change. For the time being, these assumptions can be defended on the grounds that they represent the perhaps most simple and parsimonious assumptions needed to model symptom change and stability and lead to strong predictions that can be and were tested on two quite different data sets with positive results.

A strong aspect of the stability and change model is that it can easily be extended with empirical accounts of specific sources of symptom stability and symptom change as have been demonstrated with the measures of self-esteem and locus of control. In general, these measures and their corresponding observed or true score variables may be included in the model as exogenous variables influencing stable, characteristic symptom level or the symptom change variables. The extension of the model with stable attributes of the person and his or her environmental and life event measures also make it possible to test the dynamic equilibrium model proposed by Heady and Wearing (1989). They hypothesized that stable personality traits affect the exposure to life events. Support for this hypothesis has been provided by Ferguson and Horwood (1987), who showed that both neuroticism and the presence of persistent social and economic difficulties were related to life event exposure at various time periods. Heady and Wearing's hypotheses can be modeled in the stability and change model by allowing an effect from the stable person and environmental attributes to both the stable, characteristic symptom level and the life event measures (or change agents).

In the introduction, we stated that stability in a subject's distress level may be the result of dynamic homeostatic processes in which the effects of stable attributes of the person and his or her environment unfold. We assumed that these stable attributes determine a person's characteristic distress level and exposure to change agents. They may also influence the rate at which adaptive mechanisms succeed in reducing deviations from the characteristic symptom level, brought about by variations in the level of exposure to minor and major life events. It is not fully known how these attributes exert their effects. They may, for example, act through inadequate coping patterns (McCrue, 1984). The observed strong relation between the stable, characteristic symptom level and low self-esteem and an external locus of control in the present study provides some support for this point of view.

In a stimulating chapter, Depue and Monroe (1985) distinguished four types of rather stable attributes: (a) The first type of attribute is personality traits that influence the subjective experience of negative affect, in particular the personality dimension of neuroticism (Eysenck & Eysenck, 1975) or negative affectivity (Watson & Clark, 1984). It may even be argued that the stable, characteristic symptom level largely represents the basic personality domain of negative affectivity (Duncan-Jones et al., in press) (b) The second type of attribute is prolonged stress factors that often appear difficult to alter, such as chronic
marital discord, unsafe neighborhoods, and chronic unemployment. Elsewhere we present findings suggesting that such objective stressors are extremely persistent and highly correlated with psychological distress (Ormel, Giel, Sanderman, & Heuvel, 1988). (c) The third type of attribute is persistent primary life-threatening or life-altering medical disorders or physical handicaps that severely limit the daily activities and affect the coping abilities of the sufferers. (d) The fourth type of attribute is chronic psychopathological disorders with presumably somatic origins.

Change agents accounted for approximately one third of the variance in levels of negative effect. Yet no study of life events has been able to account for variance of this magnitude (Kessler et al., 1985). To some extent, this is obviously due to unreliability in the measurement of life events. However, it seems likely that the range of empirically significant change agents is much broader than those studied in life events studies. The range of significant change agents may also include events that neutralize existing long-term difficulties (Tennant et al., 1981), daily hassles and uplifts (Kanner et al., 1981) and therapeutic interventions.

References
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