A Taxometric Investigation of the Latent Structure of Worry

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Researchers have described 2 types of worriers, normal and pathological, who differ in the frequency, intensity, and controllability of their worry experiences. Although normal and pathological worry are generally treated as separate though related phenomena, no study has tested for separateness against the alternative hypothesis that all worry exists along a single dimension. In the present study, worry ratings of 1,588 college students were submitted to taxometric procedures designed to evaluate latent structure. Results provided evidence for the dimensionality of worry. These findings suggest that generalized anxiety disorder (GAD), whose central feature is worry, may also be quantitatively rather than qualitatively different from normal functioning. The authors argue that a focus on normal and pathological extremes has constrained the study of worry phenomena and that dimensional conceptualization of worry may significantly enhance understanding of both worry and GAD.

The past two decades have witnessed increased clinical and empirical interest in the subject of worry (see Davey & Tallis, 1994). Among clinical scientists, this interest has been encouraged in part by the adoption of chronic, excessive, uncontrollable worry as the central feature of generalized anxiety disorder (GAD; American Psychiatric Association, 1994) and by the recognition that worry is also highly prevalent in other anxiety disorders (e.g., see Barlow, Blanchard, Vermilyea, Vermilyea, & DiNardo, 1986; Borkovec, 1994; Brown, Antony, & Barlow, 1992) and in depression (Starcevic, 1995). As the worry literature has grown, it has become increasingly clear that this "pathological" form of worry is associated with considerable psychological dysfunction (Boehnke, Schwartz, Stromberg, & Sagiv, 1998; Borkovec, 1994). However, what is not yet clear is how pathological worry is related to—and distinguishable from—the "normal" worry that is so commonly experienced by psychologically healthy adults and children (Borkovec, 1994; Muris, Meesters, Merckelbach, Sermon, & Zwakhalen, 1998; Perrin & Last, 1997; Turner, Beidel, & Stanley, 1992). Unlike its pathological counterpart, normal worry is generally viewed as a constructive strategy for dealing with potentially stressful life events (Davey, 1994), helping to facilitate problem-

solving, information-seeking, and active coping behaviors that reduce anxiety (cf. Billings & Moos, 1981; Endler & Parker, 1990). As knowledge of both normal and pathological worry has grown, questions about differences between the two processes have become among the most widely investigated in the field (see Davey & Tallis, 1994).

In recent years, studies examining the relationship between normal and pathological worry have revealed several characteristics that differentiate the two processes. Whereas normal and pathological worriers tend to report very similar worry content (Borkovec, Shadick, & Hopkins, 1991), pathological worriers (typically defined as individuals diagnosed with GAD) worry about a greater number of topics (Roemer, Molina, & Borkovec, 1997), spend more time engaged in worry (Craske, Rapee, Jackel, & Barlow, 1989), are more likely to worry about minor things (Roemer et al., 1997), and are less likely to associate their worry with a recognizable external precipitant (Craske et al., 1989) than nonanxious individuals. Pathological worriers are also more likely than normal worriers to describe their worrying as uncontrollable (e.g., England & Dickerson, 1988; Parkinson & Rachman, 1981). Research on the cognitive processes of normal and pathological worriers has indicated that normal worriers demonstrate an attentional bias away from threatening material, whereas pathological worriers diagnosed with GAD exhibit an attentional bias toward such material (MacLeod, Mathews, & Tata, 1986) and interpret ambiguous external information as more threatening than do normal worriers (Eysenck, Mogg, May, Richards, & Mathews, 1991). Finally, psychophysiological research has revealed that pathological worriers exhibit greater restriction in heart rate variability (Thayer, Friedman, & Borkovec, 1996) and greater rigidity in higher cortical activity (Borkovec, Ray, & Stoeber, 1998) than nonanxious individuals.

Taken together, these studies suggest that pathological worry is indeed different from the normal worry of nonanxious individuals. However, this literature is limited in two important ways. First, although virtually all research on pathological worry has been conducted with individuals diagnosed with GAD, recent evidence

indicates that only about one fifth of individuals reporting clini-
cally significant levels of worry fully meet the *Diagnostic and
Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; Amer-
ican Psychiatric Association, 1994) criteria for GAD (A. M. Rus-
cio, in press). Thus, it may be that pathological worriers are more
similar to normal worriers than past research has suggested. Sec-
ond, it is unclear from these results whether differences uncovered
between worry groups reflect differences of degree or differences
in kind. Although normal and pathological worry have tradition-
ally been studied as separate but related phenomena (e.g., Craske
et al., 1989; Roemer et al., 1997; Tallis, Davey, & Capuzzo, 1994),
no research has yet assessed whether these processes are qualita-
tively distinct or lie along a single continuum. That is, differences
that have been found between normal and pathological worriers
may reflect points of demarcation between two discrete types of
worry experiences or may instead reflect differing levels of worry
severity associated with the lower and upper extremes of a single
worry dimension. Although the latent structure of worry remains
an open question, no study to date has directly tested whether
worry is a typological or dimensional phenomenon.

The answer to the question of latent structure has critical im-
plications for the ways in which worry is conceptualized and
investigated. As Davey (1994) noted,

> How one defines worrying is of critical importance to our understand-
> ing of the process, especially since we are still at a relatively young
> age in the scientific study of worry. The definition that one adopts is
> important because it delineates the bounds of the phenomenon, and
> this will (either rightly or wrongly) tend to focus the search for the
> causes of the phenomenon. (p. 35)

Thus, knowledge of worry's structural nature should facilitate
theoretical advances, stimulate research in promising new direc-
tions, and help to integrate empirical knowledge within the worry
and anxiety literatures. Likewise, knowledge of worry's latent
structure may have important implications for the ways in which
this phenomenon is assessed in clinical and research settings,
leading to decreased measurement error and greater predictive
power (Fraley & Waller, 1998; Meehl, 1992; J. Ruscio & Ruscio,
in press). Finally, information about the structure of worry may
help to identify individuals who would benefit from specific clin-
ical interventions aimed at reducing worry (Flett, Vrendenburg, &
Krames, 1997). Thus, knowledge of worry's latent structure has
theoretical, empirical, and practical implications for our under-
standing of this complex and important phenomenon.

The present study examined the nature of the boundary between
normal and pathological worry using a family of statistical proce-
dures in the taxometric method. These procedures—performed
with variables from two commonly used measures of pathological
worry—assessed whether the latent structure of worry is typolog-
ical or dimensional.

Method

Participants

Participants for the present investigation included 1,588 undergraduate
students enrolled in Introductory Psychology at Pennsylvania State Uni-
versity during the 1998–1999 academic year. Participants completed mea-
sures for the present study as part of a larger departmental questionnaire
battery. Participation was voluntary and was rewarded with extra credit.

Participants were predominantly female (71%) and Caucasian (89%) and
had a mean age of 18.74 years ($SD = 1.97$).

This sample was chosen for the present investigation on the basis of
several considerations. First, a recent study revealed a high base rate of
clinically significant worry in a college student sample, with 28% of cases
receiving worry scores falling within 1 standard deviation of the mean of
GAD-diagnosed clients (A. M. Ruscio, in press). This research also indi-
cated that approximately 6% of college students meet diagnostic criteria for
GAD on self-report questionnaires (A. M. Ruscio, in press). Thus, the true
base rate of an existing pathological worry taxon was likely to fall between
6% and 28% in a comparable sample, providing a sufficient base rate for
taxon detection (Meehl, 1995). Second, studies indicate that the severity of
pathological worry and other GAD symptoms reported by college students
with GAD is equivalent to the severity reported by GAD-diagnosed indi-
viduals in clinical settings (e.g., Roemer, Borkovec, Posa, & Borkovec,
1995). This suggested that the latent structure of pathological worry could
be tested using college student data. Finally, taxometric procedures require
large numbers of cases to generate reliable and interpretable results, and the
ready availability of student data permitted access to an appropriately large
sample.

Measures

Participants completed two self-report questionnaires assessing the fre-
quency, intensity, and uncontrollability of their worry experiences. The
first was the Penn State Worry Questionnaire (PSWQ; Meyer, Miller,
Metzger, & Borkovec, 1990), a 16-item trait measure of clinically signif-
icant worry. Items of the PSWQ are rated on a 5-point Likert-type scale
indicating how typical or characteristic each item is of the respondent. Five
of the questionnaire items are reverse-scored. Sample items include "I am
always worrying about something"; "I find it easy to dismiss worrisome
thoughts"; and "When there is nothing more I can do about a concern, I
don't worry about it anymore." The PSWQ has been shown to have
excellent psychometric properties in college, community, and clinical
samples (cf. Molina & Borkovec, 1994; van Rijsoort, Emmelkamp, &
Vervaeke, 1999). The items of the PSWQ assess central features of
pathological worry, including the generality of worry over time and situ-
ations, the intensity of worry experiences, and the perception of worry as
uncontrollable. These items were drawn from clinical and research expe-
rience with GAD clients and other high worriers and were therefore viewed
as meaningful and potentially valid indicators of the conjectured worry
taxon.

The Generalized Anxiety Disorder Questionnaire (GAD-Q-IV; New-
man, Zuellig, Kachin, & Constantino, 1997) is a 15-item measure that
assesses *DSM-IV* GAD symptomatology. Five items of the GAD-Q-IV
assess the excessiveness, frequency, and controllability of worry experi-
enced in the past 6 months and were considered for inclusion in taxometric
analysis; items assessing nonworry aspects of GAD (e.g., associated symp-
toms; global distress and impairment) were not considered. All five of the
worry-related items have dichotomous response options. Items of the
GAD-Q-IV reflect current *DSM* conceptualizations of pathological worry,
derived from years of empirical and clinical observations of GAD. More-
over, the GAD-Q-IV worry-related items assess certain features of patho-
logical worry—such as its excessiveness and persistence—that the PSWQ
does not directly address but that research has found to differentiate
pathological and normal worriers. Thus, use of items from both instruments
was expected to yield a highly valid pool of indicators that could differ-
entiate members of the two putative latent classes.

Procedure

Indicators drawn from the PSWQ and GAD-Q-IV were submitted to a
series of taxometric procedures developed by Meehl and his colleagues
(e.g., Meehl, 1973, 1995; Meehl & Golden, 1982; Meehl & Yonce, 1994,

1996; Waller & Meehl, 1998) expressly for the purpose of assessing latent structures. Taxometric procedures search for patterns of relationships between variables that are uniquely characteristic of two underlying groups, or taxa. Here, taxa refer to nonarbitrary classes—that is, categories that truly exist in nature—rather than to continuously distributed variables that are dichotomized for purposes of analytic or administrative convenience. Rather than relying on traditional null-hypothesis significance tests, the taxometric method seeks convergence of results across a variety of mathematically diverse procedures using multiple sets of indicator variables (Meehl, 1995, 1999). In recent years, taxometric procedures have been used to investigate the latent structure of a number of important psychological constructs, including depression (J. Ruscio & Ruscio, 2000), psychopathy (Harris, Rice, & Quinsey, 1994), schizotypy (Golden & Meehl, 1979; Lenzenweger, 1999; Lenzenweger & Korfine, 1992), dissociation (Waller, Putnam, & Carlson, 1996; Waller & Ross, 1997), hypnotic susceptibility (Oakman & Woody, 1996), self-monitoring personality (Gangestad & Snyder, 1985), Type A behavior (Strube, 1989), and adult attachment (Fraley & Waller, 1998).

The latent structure of worry was evaluated by two mathematically distinct taxometric procedures: MAXCOV (maximum covariance; Meehl, 1973; Meehl & Yonce, 1996) and MAMBAC (mean above minus below a cut; Meehl & Yonce, 1994). These procedures were performed with three nonredundant sets of indicators drawn from the PSWQ and GAD-Q-IV. As a further test of latent structure, each analysis was used to estimate the base rate of pathological worry in the sample, and subsequent consistency tests examined the degree of correspondence between these estimates. The convergence of results across different procedures, indicator sets, and analyses served as a gauge of the validity of the structural solution.

Results

Selection and Construction of Indicators

The taxometric procedures employed in the present study use input indicators—that is, variables that form the abscissa above which curves depicting a latent structural solution are plotted. Input indicators must therefore have adequate variability to permit the graphing of a stable curve. If the input is measured on a 5-point scale (as are the items of the PSWQ), the resulting curve will consist of only 5 points and may yield an equivocal or invalid interpretation, especially for a low base-rate taxon. Thus, rather than using individual PSWQ or GAD-Q-IV items as input indicators in the present study, three approaches were followed to construct input indicators suitable for taxometric analysis. Because the three approaches differed in their item selection procedures, each generated a set of indicators that only partially overlapped with the others, resulting in quasi-independent taxometric solutions and a more rigorous test of consistency across analyses.

Paired indicators. Using the first approach to indicator construction, indicators were formed by summing PSWQ items in pairs. Of the 16 PSWQ items, 8 were selected on the basis of several criteria. Because the total score of the PSWQ was expected to differentiate normal and pathological worriers, items sharing particularly high correlations with the total score were identified as those most likely to be valid indicators of a pathological worry taxon. To avoid redundancy between indicators and thus minimize within-group ("nuisance") covariance (Gangestad & Snyder, 1985; Meehl, 1973), we paired these items in a manner that yielded higher correlations between paired items than between unpaired items. The final 8 items yielded 4 paired indicators,[1] each ranging in value from 2 to 10. Complete data on the paired indicators were available for 1,576 participants.

To evaluate the likely extent of nuisance covariance, we computed correlations between the paired indicators within groups of individuals expected to represent relatively pure taxon and complement groups: the lower and upper quartiles of the distribution of PSWQ total scores. Nuisance correlations averaged .15, well within the tolerance limits of both the MAXCOV and MAMBAC procedures (Meehl & Yonce, 1994, 1996). In the full sample—where large, positive correlations are desirable—the four items were correlated at an average level of .61. These values were substituted into a formula provided by Meehl and Yonce (1996, p. 1146) to estimate the average separation that would be achieved by the paired indicators if taxa were present in the sample. The average degree of separation was estimated to be 2.17 within-group standard deviations. Given a sample size of 1,576 cases and a separation of this magnitude, both MAXCOV and MAMBAC typically yield clear and consistent results, even when the taxon base rate is relatively low (see Meehl, 1995). Therefore, the four paired indicators were used in both taxometric procedures.

Unpaired indicators. The second approach selected a set of PSWQ items whose sum served as the input indicator. Consistent with the first approach, items were chosen on the basis of high correlations with the PSWQ total score. However, as item pairing was no longer a goal, low content redundancy was sought between all items. These considerations led to the selection of eight items,[2] only five of which overlapped with items chosen by the first approach. Complete data on the eight unpaired indicators were available for 1,577 cases.

Using the same procedure described earlier, nuisance correlations were found to average .16, still well within the tolerance limits of taxometric procedures. The average manifest correlation was .57 in the full sample, yielding an average estimated separation of 1.95 standard deviations. Given the strength of these parameter estimates and their appropriateness for taxometric analysis, the unpaired indicators were used in a second series of analyses.

Dichotomous indicators. The final indicator set was composed entirely of dichotomous indicators. This permitted the inclusion of dichotomous GAD-Q-IV items in analyses while serving as a consistency test for the traditional (continuous indicator) MAXCOV procedure. Using this third approach to indicator selection, all items of the PSWQ were dichotomized prior to analysis. Then, in each analysis, two dichotomous indicators were removed for covariance computation and all remaining indicators were summed to form the input variable.

The cutpoint used to dichotomize the items was determined by examining PSWQ item endorsement patterns in the sample and by considering the theoretical meaningfulness of different cut values. PSWQ mean item responses ranged from 1.98 to 4.28 (*Mdn* = 3.00) among non-GAD participants. A cutpoint of 4 was thus considered to be the most appropriate place to dichotomize, given its expected efficacy for separating normal and pathological worriers at the item level and its capacity to yield enough cases above and below the cut to produce adequate indicator variability.

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[1] The four paired indicators consisted of the following PSWQ item pairs: Items 3 and 10, 4 and 5, 9 and 16, 14 and 15.

[2] The eight unpaired indicators included PSWQ Items 2, 4, 5, 9, 12, 13, 14, and 16.

Therefore, for each PSWQ item, responses of 1, 2, or 3 were reclassified as "absent" and responses of 4 and 5 were reclassified as "present" with respect to the content of the item.

The 16 dichotomized PSWQ items and the five worry-specific GAD-Q-IV items formed a pool from which indicators were selected for the modified (dichotomous indicator) MAXCOV procedure. Past research suggests that MAXCOV curves based on eight dichotomous indicators yield an acceptable number of input intervals and retain enough cases within each interval to achieve stable covariance values and uncover true latent structures (e.g., Gangestad & Snyder, 1985; Trull, Widiger, & Guthrie, 1990; J. Ruscio, 2000). In keeping with this literature, eight items[3] were chosen from the PSWQ and the GAD-Q-IV according to the same criteria used by the previous selection approaches: relatively low content redundancy and high correlations with a scale composed of all dichotomous PSWQ and GAD-Q-IV items. These indicators were available for 1,559 participants and included five items selected by the other approaches.

Using the same procedure described earlier, nuisance correlations were estimated to average .03. The average interindicator correlation in the full sample was .45, leading to an estimated average separation of 1.75 standard deviations. As these parameter estimates were highly suitable for taxometric analysis, the eight dichotomous indicators were used in the modified MAXCOV procedure.

MAXCOV Analyses

Paired indicators. The MAXCOV procedure was first performed using all 12 possible combinations of the four paired-item PSWQ indicators. In each analysis, the covariance between two output indicators was computed for every value of an input indicator, then plotted above this corresponding input value to form the MAXCOV curve. Each curve was smoothed using the "hanning" method (Hartwig & Dearing, 1979) to reduce the effects of sampling error, thereby easing interpretation (Meehl, 1973; Meehl & Golden, 1982) and permitting comparison with smoothed curves yielded by Monte Carlo trials (Meehl & Yonce, 1996). None of the 12 resulting curves exhibited the clear peak that is characteristic of taxonic data, nor the tapering of covariance values toward zero at the endpoints that would be expected of curves with low estimated nuisance covariance. Rather, these curves were relatively flat, favoring a dimensional solution. An average of the raw and smoothed MAXCOV curves is displayed in the top panel of Figure 1.[4]

Unpaired indicators. The MAXCOV procedure was next performed 28 times using every possible configuration of the eight unpaired PSWQ indicators. In each analysis, two output indicators were removed for covariance computation, and the remaining six items were summed to form the input indicator. Following the recommendations of Meehl and Yonce (1996), the input variable was standardized and divided into intervals .25 standard deviations in width. To ensure the stability of covariance values on which the MAXCOV curve was based, we combined extreme values of the input indicator such that a minimum of 20 cases fell in each interval of the input indicator. Although some of the 28 resultant curves contained slight elevations, the full panel of curves lacked the sharp, consistent peaks indicative of taxonic data. These results thus provided additional support for the dimensionality of worry.
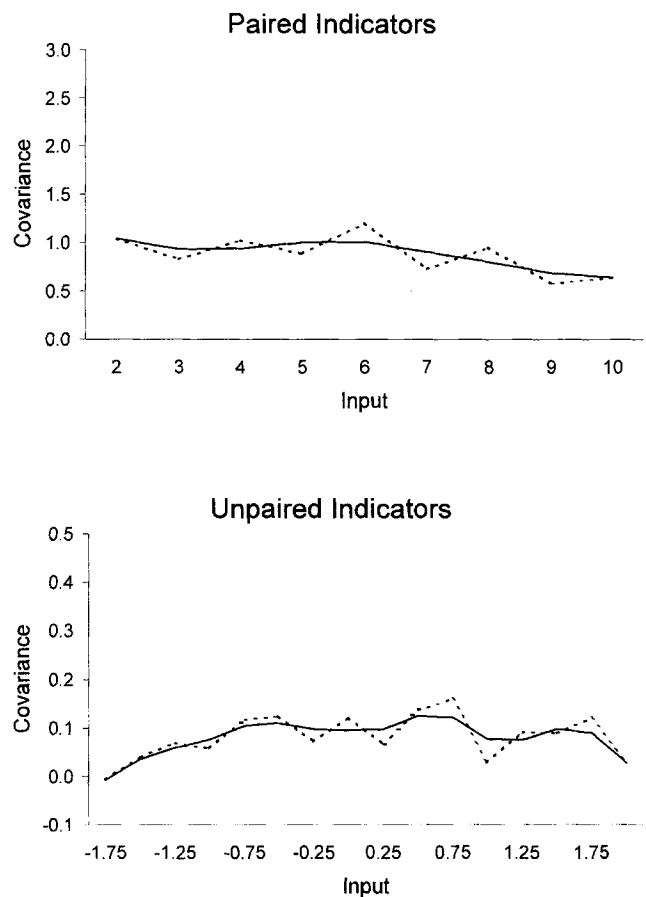

Figure 1. Averages of curves yielded by traditional (continuous indicator) maximum covariance (MAXCOV) analyses. Stippled lines represent raw covariances; solid lines represent covariances smoothed by hanning. Top panel: The average of 12 curves generated by the four paired indicators. Bottom panel: The average of 28 curves generated by the eight unpaired indicators. The scale of each input indicator is standardized and contains 17 intervals with a width of .25 standard deviations.

An average of the raw and smoothed MAXCOV curves yielded by the unpaired indicators appears in the bottom panel of Figure 1.

Dichotomous indicators. The modified MAXCOV procedure was performed 28 times. In each analysis, two output indicators were removed for covariance computation, and the remaining six items were summed to form an input indicator ranging in value from 0 (normal worry responses on all items) to 6 (pathological worry responses on all items). Because of the decreased stability of MAXCOV curves derived from dichotomous indicators, the 28 individual graphs were averaged into a single, more reliable com-

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[3] The eight dichotomous indicators included dichotomized PSWQ Items 2, 4, 5, 7, 12, and 13 and GAD-Q-IV Items 2 and 3.

[4] Although interpretations were based on the full panels of curves, only averaged curves are presented herein to conserve space. All MAXCOV curves were averaged using medians to reduce the influence of extreme values on the final curve, whereas all MAMBAC curves were averaged using means because of the small number of curves contributing to the average. Full panels of curves for all taxometric analyses described in the present study are available from Ayelet Meron Ruscio.

posite curve for interpretation (cf. Gangestad & Snyder, 1985). This curve (appearing in the top panel of Figure 2) was low and humped, with the peak covariance value reaching only .03. Monte Carlo research with the modified MAXCOV procedure has found

Dichotomous Indicators

Simulated Dimensional Data

Simulated Taxonic Data

Figure 2. Averages of raw (unsmoothed) curves yielded by the modified (dichotomous indicator) maximum covariance (MAXCOV) procedure. Top panel: The average of 28 curves generated by the eight dichotomous indicators. Middle panel: The average of 10 samples (each an average of 28 curves) generated by dimensional data simulated to match the parameters of the present sample. Bottom panel: The average of 10 samples (each an average of 28 curves) generated by taxonic data simulated to match the parameters of the present sample.

that, among samples with roughly equivalent parameters to those estimated in the present study, 100% of taxonic samples yield a covariance peak greater than .05, whereas only 3% of dimensional samples exceed this value (J. Ruscio, 2000). This suggested that the data underlying the present MAXCOV curve were dimensional.

To further aid interpretation, a small-scale Monte Carlo simulation was conducted using dichotomous indicators. Ten taxonic samples and 10 dimensional samples were created whose sample size, number of indicators, base rate, and separation precisely matched those of the present sample. The modified MAXCOV procedure was performed within each of the 20 samples. All taxonic samples produced covariance curves with clearly recognizable sharp peaks, whereas the dimensional samples yielded smooth, low humps very similar to the curve obtained in the present study. Averages of the 10 taxonic and 10 dimensional curves appear in the lower panels of Figure 2. Taken together, the dichotomous indicator MAXCOV results further corroborated the dimensionality of worry.
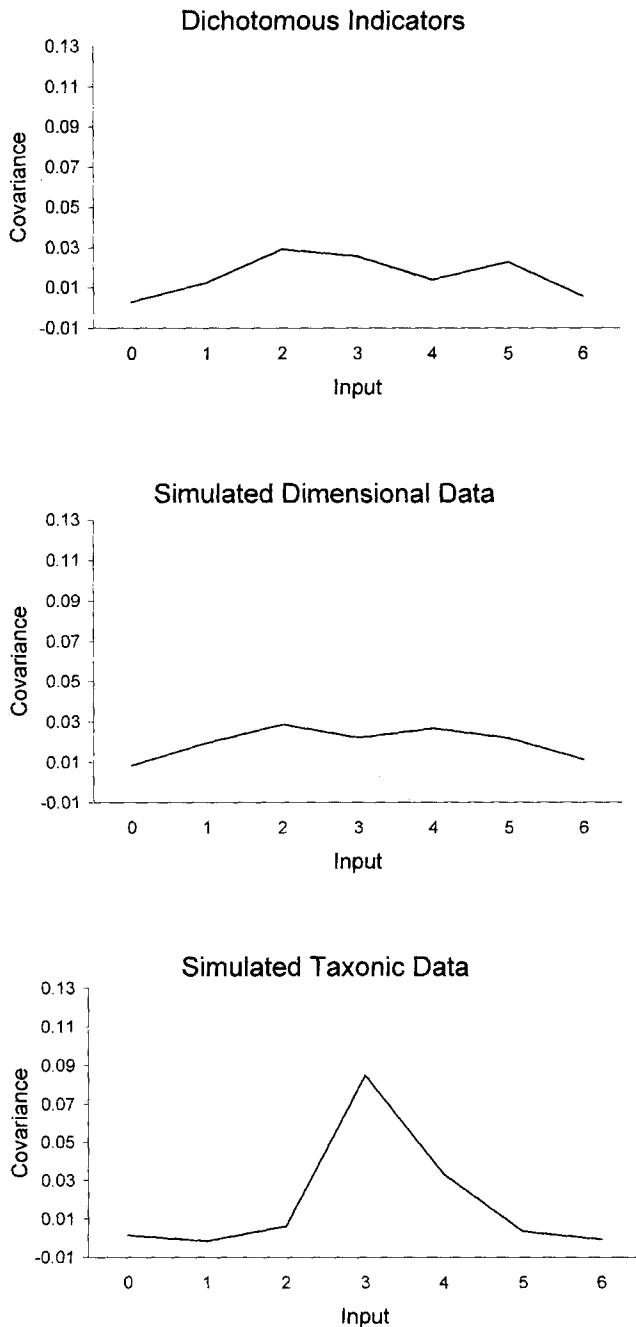
MAMBAC Analyses

Paired indicators. The MAMBAC procedure was first conducted using the four paired PSWQ indicators. Because the small response scales of these indicators would have provided only a crude sorting of cases in MAMBAC analyses, each analysis was performed by removing one of the paired indicators to serve as the output variable, then summing the remaining three indicators into a single input variable. Following this procedure, MAMBAC was conducted four times. The resulting curves were dish-shaped rather than peaked, providing support for a dimensional solution. An average of these curves is displayed in the top panel of Figure 3.

Unpaired indicators. The MAMBAC procedure was next conducted using each of the eight unpaired PSWQ items in turn as the output indicator. Following the rationale described above, cases were sorted on an input indicator representing the sum of the seven remaining items. The eight resulting MAMBAC curves were clearly dish-shaped with no noticeable taxonic humps, providing additional evidence for a latent dimension of worry. An average of these curves appears in the bottom panel of Figure 3.

Consistency of Base-Rate Estimates

In addition to yielding a curve reflecting latent structure, each MAXCOV and MAMBAC analysis also estimates the base rate of the taxon (here, pathological worry) in the sample. If worry is taxonic, estimates yielded by different taxometric procedures should cohere around the true base rate of the taxon. Significant variability between base-rate estimates suggests that these values do not denote an actual latent class, providing evidence for dimensionality.

All MAXCOV curves were smoothed by hanning to reduce the effects of sampling error on base-rate computation. Then, following procedures described by Meehl and Yonce (1994, 1996), the base rate of pathological worry was estimated from every MAXCOV and MAMBAC curve. As is shown in Table 1, these values varied tremendously across procedures, indicator sets, and analyses. Base-rate estimates derived from the individual curves spanned almost the entire range of possible values, as reflected by the remarkably high standard deviations corresponding to the
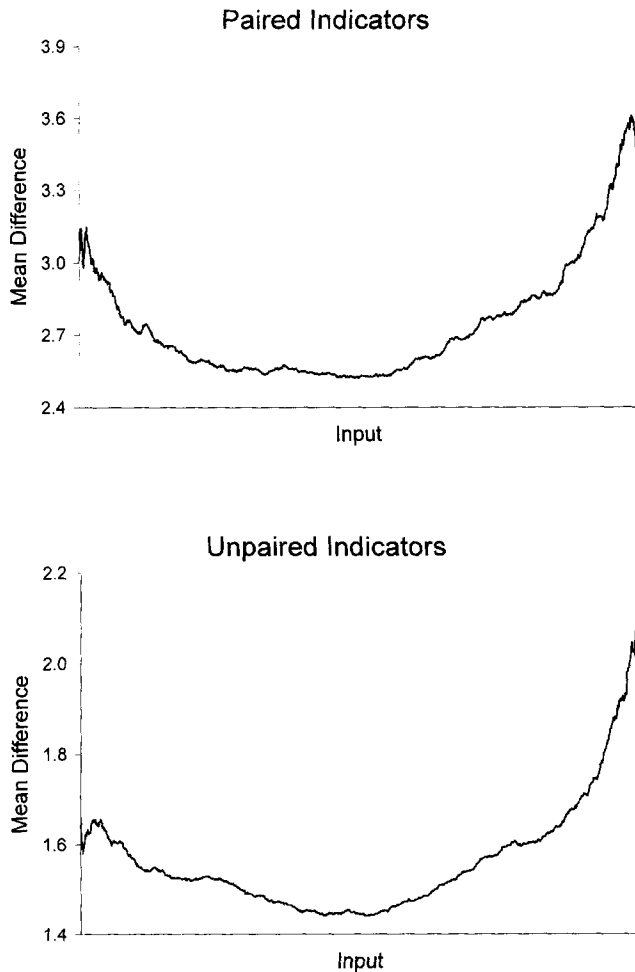
Figure 3. Averages of raw (unsmoothed) curves yielded by the mean above minus below a cut (MAMBAC) procedure. Top panel: The average of four curves generated by the four paired indicators. Bottom panel: The average of eight curves generated by the eight unpaired indicators.

distribution of estimates. Moreover, none of the mean or averaged values fell between the theoretically derived base-rate boundaries of 6% and 28% proposed at the outset of the study.

Discussion

Theorists and investigators have long distinguished between normal and pathological worriers, seeking to identify characteristics differentiating the two groups. However, no study to date has directly assessed the structural nature and fundamental interrelationship of these worry phenomena. The present investigation sought to determine whether normal and pathological worry constitute discrete phenomena or whether worry is better represented by a single severity continuum with normal and pathological extremes. Two taxometric procedures, MAXCOV and MAMBAC, were performed with several indicator sets estimated to have high validity and low nuisance covariance. Curves generated by both procedures were characteristically dimensional in shape, and base-rate estimates derived from these curves failed to cohere around a

consistent value. Taken together, these results provided strong support for the dimensionality of worry.

The present findings raise potentially important implications for the theoretical conceptualization of worry. These results first suggest that theories attempting to explain how and why worry becomes problematic must move beyond factors associated with the presence or absence of pathological worry. Instead, it will be useful for theorists to consider causal and maintenance factors associated with varying levels of worry severity within the full range of worry presentations. As theories shift from their present focus on worry extremes to a consideration of all worriers, two major changes will likely occur: expansion of current theory to account for a broader range of disturbances among a far larger population of worriers, and development of hypotheses proposing new kinds of relationships (e.g., linear, curvilinear, threshold, twisted pear) between worry and other variables of interest. Theories of worry will be advanced by the reformulation of prior empirical findings within the bounds of a dimensional framework, framing differences between normal and pathological worriers as differences in degree rather than in kind. Theoretical advances will also be made by integrating disparate and formerly segregated literatures concerned only with normal worriers (e.g., test anxiety literature) or pathological worriers (e.g., clinical literatures), contributing to a more comprehensive understanding of worry phenomena.

One particularly important theoretical consequence of the present results is their potential to focus the search for the origins of worry. Several researchers have suggested that knowledge of latent structure may provide clues about the etiology of a trait or disorder by ruling out etiological models that are incompatible with a particular structural solution (Gangestad & Snyder, 1985; Haslam, 1997; Oakman & Woody, 1996). Haslam (1997) has argued that dimensional latent structure is incongruent with a variety of specific causal models, such as an all-or-none genetic or environmental effect or an interaction of all-or-none genetic factors, environmental factors, or both. Instead, he suggested that dimensional structure is consistent with other etiological models, such as a single, continuously graded environmental factor; multiple, additive genetic factors; multiple, additive environmental factors; or an additive combination of genetic and environmental factors. Thus, the present findings argue against the ability of

Table 1

Base-Rate Estimates Derived From Smoothed MAXCOV and MAMBAC Curves

Procedure and indicators	No. of estimates	Taxon base-rate estimates		
		Range	M	SD
MAXCOV analyses				
4 paired indicators	12	.19–.83	.53	.25
8 unpaired indicators	28	.10–.74	.43	.21
8 dichotomous indicators	1	—	.34	—
MAMBAC analyses				
4 paired indicators	4	.37–.60	.48	.10
8 unpaired indicators	8	.37–.52	.44	.06

Note. MAXCOV = maximum covariance; MAMBAC = mean above minus below a cut.

all-or-none factors (e.g., experiencing a particular traumatic stressor, inheriting a particular worry- or anxiety-linked gene) to predict the level of worry that will typically be experienced by a given individual. Instead, these findings direct the search for the origins of worry toward graded and additive etiological models that emphasize the individual's degree of exposure to one or more experiential factors (e.g., significant stressors, quality of the attachment relationship in childhood, extent of current interpersonal problems), hereditary factors (e.g., predisposition to neuroticism, GAD, or other anxiety or mood disorders), or both. In this way, the dimensional solution uncovered herein may help to rule out several unlikely etiological models of worry while delineating areas for further study that are most likely to enhance etiological understanding of worry phenomena.

Along with their implications for theories of worry, the present findings also have potentially important consequences for our understanding of GAD. If pathological worry—the defining feature of GAD—is indeed continuous with normal worry, this raises potential questions about the status of GAD as a discrete clinical entity that is qualitatively distinguishable from normal functioning. At the same time, it must be noted that GAD and severe worry are not one and the same (A. M. Ruscio, in press) and that the combination of pathological worry with other core cognitive and physiological symptoms of GAD (e.g., concentration problems, restlessness, sleep disturbance) and with clinically significant distress and impairment may serve to delineate a GAD taxon. The present findings thus do not directly address the latent structure of GAD, but rather underscore the need for more focused research into the underlying nature of GAD and the relationship between GAD and worry. Given the present results, the first step in such a research program should include a taxometric evaluation of the latent structure of GAD. If GAD is found to be taxonic, subsequent investigations would most profitably search for the variables or processes with which worry interacts to produce GAD. Alternatively, if GAD—like worry—is found to be dimensional, ensuing research would usefully evaluate the degree of similarity between the two dimensions and identify the elements that distinguish them. Results of these studies may significantly enhance our understanding of the etiology and manifestation of generalized anxiety, leading to advances in theory, research, and treatment of the disorder.

The present findings have yet another set of implications: those pertaining to the assessment of worry. First, these findings suggest not only that the normal and pathological labels are relatively uninformative indicators of worry severity, but that they may be misleading as well. This conclusion is reinforced by recent research indicating that high levels of worry are quite "normal" among individuals without GAD and that some GAD-diagnosed "pathological" worriers report less severe levels of worry than many "normal" worriers (Ruscio, in press). Both studies argue for the removal of these "loaded" labels from the professional vernacular, replacing them with references to the degree of worry severity—from low to high—that is experienced by individuals, irrespective of their GAD diagnostic status. Second, the dimensional results uncovered herein indicate that worry will be most validly assessed by measures that yield continuous worry scores, such as the PSWQ. Attempts to form groups of normal and pathological worriers—whether by the number or severity of worry experiences, by cut scores on continuous worry measures, or by DSM-IV

criteria for GAD—would not only be theoretically inaccurate, but would also likely result in a significant loss of statistical power. By contrast, a shift to continuous measures of worry should result in more powerful empirical investigations and an improved understanding of worry phenomena. A third, related point is that comparative designs, which seek to identify differences between normal and pathological worriers, no longer seem appropriate in light of the purely quantitative differences between these individuals. Instead, a more profitable approach may be to study worry as a unitary construct, identifying correlates or predictors of the full range of worry severity scores. Fourth, the present findings indicate that, in order to optimally differentiate individuals across the entire worry continuum, measures of worry should include moderately worded items designed to yield a wide range of possible scores (e.g., "When there is nothing more I can do about a concern, I don't worry about it any more") rather than items that force responses into the extremes of the worry continuum in an effort to separate normal and pathological worriers (e.g., "I never worry about anything" or "I worry all the time").

Finally, the present findings may inform the search for mechanisms underlying change in worry, thereby stimulating the development of increasingly effective worry interventions. Strube (1989) has argued that taxa are "not consistent with programs aimed at, or expectant of, gradual or partial change. The presence of a taxon instead suggests that change will occur in an all-or-none fashion" (p. 984). By implication, clinical problems that are dimensional in nature may be more receptive to incremental intervention because they allow change to occur gradually, even partially, without requiring a global shift to a dramatically different plateau of functioning. Moreover, if worry reduction follows a gradual course rather than a step function of change, it may not be appropriate to view treatment as successful only when worry drops to a predetermined minimum level. Instead, a smaller but clinically significant reduction in worry severity may also be regarded as a meaningful and perhaps even successful result of treatment.

If the process of therapeutic change is thus conceptualized as a gradual decline in symptom severity rather than as a shift from pathological to normal states, several implications ensue for the application and evaluation of worry interventions. First, there is a need to use measures of change that are sensitive to movement along the full worry continuum and to evaluate treatment effectiveness according to the degree of reported reduction in worry severity. Second, research is needed to determine which worriers are most likely to need and benefit from worry-focused interventions. Research on the relationship between worry and GAD (A. M. Ruscio, in press) has indicated that worry severity may not be a necessary or sufficient criterion for identifying individuals who are significantly distressed or impaired by their worrying, suggesting that other factors (e.g., uncontrollability of worry, severity of somatic hyperarousal, perception of worry as problematic) may play an important role in assessing the need for intervention. Finally, there is a need to determine whether individuals experiencing different degrees of worry severity may best be helped by different kinds of interventions. It may be that available treatments are not equally effective for all worriers, or that the intensity and duration of sufficient treatment vary as a function of

worry severity. Thus, practical applications of the present findings may be considerable.

The present sample was, in many ways, highly appropriate for a taxometric investigation of worry. Appropriate features included the large size of the sample, its mixed-gender composition, its significant proportion of cases reporting high levels of worry, and its inclusion of the full range of possible worry scores on the PSWQ ($M = 48.64$, $SD = 13.83$, range $= 16-80$). Moreover, it is noteworthy that a nonnegligible proportion of the present sample (7%) met the diagnostic criteria for GAD on the GAD-Q-IV and that these individuals reported levels of PSWQ worry severity ($M = 67.56$, $SD = 8.88$) that were virtually identical to the levels reported by treatment-seeking patients diagnosed with GAD ($M = 67.66$, $SD = 8.86$; Molina & Borkovec, 1994), suggesting that the severity of pathological worry experienced by the most anxious students in this sample was comparable to that of clinical patients. These features of the present sample enhanced the power of taxometric procedures to uncover an existing taxon, and failure to find a taxon in the sample bolsters confidence in the validity of these results. However, the present sample was limited by its restriction to college students, a relatively young and homogeneous population that is traditionally considered to be high-functioning relative both to clinical samples and the general population. It has generally been the case in the worry literature that results emerging from research with college samples have subsequently been replicated with clinical samples (Borkovec & Newman, 1999). Furthermore, to our knowledge, there is no empirical or theoretical reason to suspect structural differences in worry among different age groups or assessment settings that would limit the generalizability of the present findings. However, the applicability of the present findings to clinical or community samples remains an open question, and additional research is warranted to replicate these findings and to extend them to other populations of interest.

Closely related to the question of sample appropriateness is the issue of worry severity. That is, is there reason to suspect that a pathological worry taxon does exist, but that the level of worry severity present in the sample was too low to permit its detection? The distribution of PSWQ total scores in the current sample suggests that this was not likely to be the case. Of the full sample, 31% of participants had a PSWQ total score greater than or equal to 56, a value falling one standard deviation below the mean of individuals diagnosed with GAD and thus viewed as a threshold for "clinically significant" worry (Molina & Borkovec, 1994). Moreover, 18% of cases had PSWQ total scores that equaled or surpassed the mean score of GAD-diagnosed college students in past research, whereas 10% of cases had PSWQ total scores that equaled or surpassed the mean of GAD-diagnosed clinical samples in past research (Molina & Borkovec, 1994). Monte Carlo investigations of taxometric procedures have indicated that taxa with base rates as low as 10% can be reliably detected (Meehl & Yonce, 1994, 1996). These findings suggest that worry was indeed sufficiently severe in the present sample to detect a pathological worry taxon, had one existed.

Another potential limitation of the study concerns the exclusive use of self-report data in analyses. By its very nature, worrying is an internal, cognitive process that is typically measured only by self-report. Although a growing body of research has revealed physiological correlates of worry activity (e.g., Borkovec, Ray, &

Stoeber, 1998; Thayer, Friedman, & Borkovec, 1996), these findings are still preliminary, and physiological variables have not yet been sufficiently validated to serve as independent indicators of worry phenomena. In order to increase diversity of measurement in the present study, three nonredundant indicator sets were selected from two common measures of pathological worry. All three indicator sets evidenced excellent validity and low nuisance covariance, and each set yielded highly consistent results across taxometric procedures and consistency tests. Thus, although the inclusion of indicators from qualitatively different measures would have provided even greater confidence in the structural solution, the impressive coherence of findings across multiple measures, indicator sets, and procedures conveys considerable support for this solution.

The dimensional structure of worry has critical implications for the ways in which worrying is conceptualized, assessed, investigated, and treated. These implications may be particularly significant because contemporary theories and investigations of worry emphasize differences rather than similarities between normal and pathological processes (cf. Davey & Tallis, 1994). It is hoped that empirically derived knowledge of latent structure will help to construct a more complete and coherent understanding of worry phenomena and that future research efforts—guided in part by this knowledge—will yield more rapid insight into the etiology and maintenance of worry and related psychopathology.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Barlow, D. H., Blanchard, E. B., Vermilyea, J. A., Vermilyea, D. B., & DiNardo, P. A. (1986). Generalized anxiety and generalized anxiety disorder: Description and reconceptualization. *American Journal of Psychiatry, 143,* 40–44.

Billings, A. G., & Moos, R. H. (1981). The role of coping responses and social resources in attenuating the impact of stressful life events. *Journal of Behavioral Medicine, 4,* 139–157.

Boehnke, K., Schwartz, S., Stromberg, C., & Sagiv, L. (1998). The structure and dynamics of worry: Theory, measurement, and cross-national replications. *Journal of Personality, 66,* 745–782.

Borkovec, T. D. (1994). The nature, functions, and origins of worry. In G. Davey & F. Tallis (Eds.), *Worrying: Perspectives on theory, assessment and treatment* (pp. 5–33). Chichester, England: Wiley.

Borkovec, T. D., & Newman, M. G. (1999). Worry and generalized anxiety disorder. In A. S. Bellack & M. Hersen (Series Eds.) & P. Salkovskis (Vol. Ed.), *Comprehensive clinical psychology: Vol. 6. Adults: Clinical formulation and treatment* (pp. 439–459). Oxford, England: Elsevier Science.

Borkovec, T. D., Ray, W. J., & Stoeber, J. (1998). Worry: A cognitive phenomenon intimately linked to affective, physiological, and interpersonal behavioral processes. *Cognitive Therapy and Research, 22,* 561–576.

Borkovec, T. D., Shadick, R., & Hopkins, M. (1991). The nature of normal and pathological worry. In R. Rapee & D. H. Barlow (Eds.), *Chronic anxiety and generalized anxiety disorder* (pp. 29–51). New York: Guilford Press.

Brown, T. A., Antony, M. M., & Barlow, D. H. (1992). Psychometric properties of the Penn State Worry Questionnaire in a clinical anxiety disorders sample. *Behaviour Research and Therapy, 30,* 33–37.

Craske, M. G., Rapee, R. M., Jackel, L., & Barlow, D. H. (1989). Qualitative dimensions of worry in *DSM-III-R* generalized anxiety disorder

subjects and nonanxious controls. *Behaviour Research and Therapy, 27,* 397–402.

Davey, G. C. L. (1994). Pathological worrying as exacerbated problem-solving. In G. Davey & F. Tallis (Eds.), *Worrying: Perspectives on theory, assessment and treatment* (pp. 35–59). Chichester, England: Wiley.

Davey, G., & Tallis, F. (Eds.) (1994). *Worrying: Perspectives on theory, assessment and treatment.* Chichester, England: Wiley.

Endler, N. S., & Parker, J. D. A. (1990). Multidimensional assessment of coping: A critical evaluation. *Journal of Personality and Social Psychology, 58,* 844–854.

England, S. L., & Dickerson, M. (1988). Intrusive thoughts: Unpleasantness not the major cause of uncontrollability. *Behaviour Research and Therapy, 26,* 279–282.

Eysenck, M. W., Mogg, K., May, J., Richards, A., & Mathews, A. (1991). Bias in interpretation of ambiguous sentences related to threat in anxiety. *Journal of Abnormal Psychology, 100,* 144–150.

Flett, G. L. Vrendenburg, K., & Krames, L. (1997). The continuity of depression in clinical and nonclinical samples. *Psychological Bulletin, 121,* 395–416.

Fraley, R. C., & Waller, N. G. (1998). Adult attachment patterns: A test of the typological model. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 77–114). New York: Guilford.

Gangestad, S., & Snyder, M. (1985). "To carve nature at its joints:" On the existence of discrete classes in personality. *Psychological Review, 92,* 317–349.

Golden, R. R., & Meehl, P. E. (1979). Detection of the schizoid taxon with MMPI indicators. *Journal of Abnormal Psychology, 88,* 217–233.

Harris, G. T., Rice, M. E., & Quinsey, V. L. (1994). Psychopathy as a taxon: Evidence that psychopaths are a discrete class. *Journal of Consulting and Clinical Psychology, 62,* 387–397.

Hartwig, F., & Dearing, B. E. (1979). *Exploratory data analysis.* Beverly Hills, CA: Sage.

Haslam, N. (1997). Evidence that male sexual orientation is a matter of degree. *Journal of Personality and Social Psychology, 73,* 862–870.

Lenzenweger, M. F. (1999). Deeper into the schizotypy taxon: On the robust nature of maximum covariance analysis. *Journal of Abnormal Psychology, 108,* 182–187.

Lenzenweger, M. F., & Korfine, L. (1992). Confirming the latent structure and base rate of schizotypy: A taxometric analysis. *Journal of Abnormal Psychology, 101,* 567–571.

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology, 95,* 15–20.

Meehl, P. E. (1973). MAXCOV-HITMAX: A taxometric search method for loose genetic syndromes. In P. E. Meehl (Ed.), *Psychodiagnosis: Selected papers* (pp. 200–224). Minneapolis, MN: University of Minnesota Press.

Meehl, P. E. (1992). Factors and taxa, traits and types, differences of degree and differences in kind. *Journal of Personality, 60,* 117–174.

Meehl, P. E. (1995). Bootstraps taxometrics: Solving the classification problem in psychopathology. *American Psychologist, 50,* 266–275.

Meehl, P. E. (1999). Clarifications about taxometric method. *Applied & Preventive Psychology, 8,* 165–174.

Meehl, P. E., & Golden, R. R. (1982). Taxometric methods. In P. C. Kendall & J. N. Butcher (Eds.), *Handbook of research methods in clinical psychology* (pp. 127–181). New York: Wiley.

Meehl, P. E., & Yonce, L. J. (1994). Taxometric analysis: I. Detecting taxonicity with two quantitative indicators using means above and below a sliding cut (MAMBAC procedure). *Psychological Reports, 74,* 1059–1274.

Meehl, P. E., & Yonce, L. J. (1996). Taxometric analysis: II. Detecting taxonicity using covariance of two quantitative indicators in successive intervals of a third indicator (MAXCOV procedure). *Psychological Reports, 78,* 1091–1227.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy, 28,* 487–495.

Molina, S., & Borkovec, T. D. (1994). The Penn State Worry Questionnaire: Psychometric properties and associated characteristics. In G. Davey & F. Tallis (Eds.), *Worrying: Perspectives on theory, assessment and treatment* (pp. 265–283). Chichester, England: Wiley.

Muris, P., Meesters, C., Merckelbach, H., Sermon, A., & Zwakhalen, S. (1998). Worry in normal children. *Journal of the American Academy of Child and Adolescent Psychiatry, 37,* 703–710.

Newman, M. G., Zuellig, A. R., Kachin, K. E., & Constantino, M. J. (1997, November). *Examination of the reliability and validity of the GAD-Q-IV: A revised self-report measure of generalized anxiety disorder.* Poster presented at the annual meeting of the Association for Advancement of Behavior Therapy, Miami, FL.

Oakman, J. M., & Woody, E. Z. (1996). A taxometric analysis of hypnotic susceptibility. *Journal of Personality and Social Psychology, 71,* 980–991.

Parkinson, L., & Rachman, S. (1981). The nature of intrusive thoughts. *Advances in Behaviour Research and Therapy, 3,* 101–110.

Perrin, S., & Last, C. G. (1997). Worrisome thoughts in children clinically referred for anxiety disorder. *Journal of Clinical Child Psychology, 26,* 181–189.

Roemer, L., Borkovec, M., Posa, S., & Borkovec, T. D. (1995). A self-report diagnostic measure of generalized anxiety disorder. *Journal of Behavior Therapy & Experimental Psychiatry, 26,* 345–350.

Roemer, L., Molina, S., & Borkovec, T. D. (1997). An investigation of worry content among generally anxious individuals. *Journal of Nervous and Mental Disease, 185,* 314–319.

Ruscio, A. M. (in press). Delimiting the boundaries of generalized anxiety disorder: Differentiating high worriers with and without GAD. *Journal of Anxiety Disorders.*

Ruscio, J. (2000). Taxometric analysis with dichotomous indicators: The modified MAXCOV procedure and a case removal consistency test. *Psychological Reports, 87,* 929–939.

Ruscio, J., & Ruscio, A. M. (2000). Informing the continuity controversy: A taxometric analysis of depression. *Journal of Abnormal Psychology, 109,* 473–487.

Ruscio, J., & Ruscio, A. M. (in press). A structure-based approach to psychological assessment: Matching measurement models to latent structure. *Assessment.*

Starcevic, V. (1995). Pathological worry in major depression: A preliminary report. *Behaviour Research and Therapy, 33,* 55–56.

Strube, M. J. (1989). Evidence for the *type* in Type A behavior: A taxometric analysis. *Journal of Personality and Social Psychology, 56,* 972–987.

Tallis, F., Davey, G. C. L., & Capuzzo, N. (1994). The phenomenology of non-pathological worry: A preliminary investigation. In G. Davey & F. Tallis (Eds.), *Worrying: Perspectives on theory, assessment and treatment* (pp. 61–89). Chichester, England: Wiley.

Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry, 39,* 255–266.

Trull, T. J., Widiger, T. A., & Guthrie, P. (1990). Categorical versus dimensional status of borderline personality disorder. *Journal of Abnormal Psychology, 99,* 40–48.

Turner, S. M., Beidel, D. C., & Stanley, M. A. (1992). Are obsessional thoughts and worry different cognitive phenomena? *Clinical Psychology Review, 12,* 257–270.

van Rijsoort, S., Emmelkamp, P., & Vervaeke, G. (1999). The Penn State Worry Questionnaire and the Worry Domains Questionnaire: Structure, reliability, and validity. *Clinical Psychology and Psychotherapy, 6,* 297–307.

Waller, N. G., & Meehl, P. E. (1998). *Multivariate taxometric procedures: Distinguishing types from continua.* Thousand Oaks, CA: Sage.

Waller, N. G., Putnam, F. W., & Carlson, E. B. (1996). Types of dissociation and dissociative types: A taxometric analysis of dissociative experiences. *Psychological Methods, 1,* 300–321.

Waller, N. G., & Ross, C. A. (1997). The prevalence and biometric structure of pathological dissociation in the general population: Taxo-

metric and behavior genetic findings. *Journal of Abnormal Psychology, 106,* 499–510.

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