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ORIGINAL ARTICLE

Evidence for the continuous latent structure of mania in the Epidemiologic Catchment Area from multiple latent structure and construct validation methodologies

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Background. Although psychiatric diagnostic systems have conceptualized mania as a discrete phenomenon, appropriate latent structure investigations testing this conceptualization are lacking. In contrast to these diagnostic systems, several influential theories of mania have suggested a continuous conceptualization. The present study examined whether mania has a continuous or discrete latent structure using a comprehensive approach including taxometric, information-theoretic latent distribution modeling (ITLDM) and predictive validity methodologies in the Epidemiologic Catchment Area (ECA) study.

Method. Eight dichotomous manic symptom items were submitted to a variety of latent structural analyses, including factor analyses, taxometric procedures and ITLDM, in 10105 ECA community participants. In addition, a variety of continuous and discrete models of mania were compared in terms of their relative abilities to predict outcomes (i.e. health service utilization, internalizing and externalizing disorders, and suicidal behavior).

Results. Taxometric and ITLDM analyses consistently supported a continuous conceptualization of mania. In ITLDM analyses, a continuous model of mania demonstrated 6.52:1 odds over the best-fitting latent class model (LCM) of mania. Factor analyses suggested that the continuous structure of mania was best represented by a single latent factor. Predictive validity analyses demonstrated a consistent superior ability of continuous models of mania relative to discrete models.

Conclusions. The present study provided three independent lines of support for a continuous conceptualization of mania. The implications of a continuous model of mania are discussed.

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Introduction

Although psychiatric diagnostic systems (e.g. DSM-IV; APA, 1994) conceptualize mania and associated bipolar disorder phenomenology as categorical, there have been no appropriate latent structure investigations conducted to support or refute this conceptualization. In contrast to the DSM, most influential theories of mania and bipolar disorders have suggested a dimensional conceptualization of these constructs. For example, Kraepelin (1921) proposed that affective temperaments form the constitutional foundation for manic-depressive illness, and observed that non-disordered relatives of manic-depressive patients frequently exhibited these temperaments. Intellectual descendants of Kraepelin have subsequently provided empirical evidence to support the validity of a continuous conceptualization of manic-depressive illnesses (e.g. Judd & Akiskal, 2003; Merikangas *et al*. 2007). Aside from the theoretical importance of identifying the latent structure of mania, there are concerns associated with potentially incorrectly conceptualizing mania as a categorical construct, including statistical problems (e.g. attenuated statistical power; Cohen, 1983), spurious psychiatric co-morbidity (Haslam, 2003) and inappropriate denial of treatment services.

Appropriate statistical methodologies for determining whether mania is a categorical or dimensional construct include taxometrics (Waller & Meehl, 1998) and information-theoretic latent distribution modeling (ITLDM; Markon & Krueger, 2006). Using multiple latent structure methodologies to investigate the continuous *versus* discrete nature of a construct has long

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been recommended, but is rarely done in practice (Lenzenweger, 2004). No taxometric or ITLDM studies have examined mania or bipolar disorders, although Meyer & Keller (2003) have conducted a taxometric investigation of hyperthymic temperament. Their study had several methodological issues (e.g. use of a single taxometric procedure) but nonetheless found evidence for a continuous latent structure.

Although ITLDM and taxometric analyses are crucial to determining whether mania is a categorical or dimensional construct, the results from such analyses can be misleading due to flaws in study design (Grove, 1991) or limitations of the analytic procedures (Ruscio *et al*. 2004). Consequently, researchers have argued that latent structural findings are provisional until their meaning has been clarified through construct validation (Waldman & Lilienfeld, 2001; Watson, 2003), and have provided examples of structural findings that do not survive such scrutiny (e.g. schizoid taxon; Nichols & Jones, 1985; dissociative taxon; Watson, 2003).

One approach to evaluating the construct validity of taxometric and ITLDM findings is to compare the relative abilities of multiple discrete and continuous models of mania to predict theoretically relevant constructs. If mania is continuous, it should demonstrate superior predictive ability relative to a discrete model because of the loss of statistical power associated with dichotomizing a continuous variable (Cohen, 1983). Alternatively, if mania is discrete, then discrete models of the construct should out-predict continuous models because the extra variability afforded by continuous models would represent measurement error that would attenuate statistical relationships (Ruscio *et al*. 2006). Two previous studies have used this methodology to examine the construct validity of discrete and continuous models of depression (Aggen *et al*. 2005; Prisciandaro & Roberts, 2009). Both studies found that discrete models of depression did not significantly predict relevant outcomes once continuous models of depression were statistically controlled.

The present study is the first to properly evaluate whether mania is a discrete or continuous construct. Given the various criticisms that have been raised regarding using taxometrics as a standalone methodology (e.g. lack of agreement over implementation decisions), the present investigation used an expanded, comprehensive approach to examine the latent structure of mania. In Part I of the present study, both taxometric and ITLDM analyses were conducted on the manic symptom data from the Epidemiologic Catchment Area (ECA) program (Eaton & Kessler, 1985). In Part II of this study, an examination of the construct validity of taxometric and ITLDM findings was performed by testing the relative predictive abilities of various discrete and continuous models of mania.

Part I Method

Sample and measure

Data from the ECA program (Eaton & Kessler, 1985) were obtained from a representative group of over 20 000 individuals by probability sampling of five geographic catchment areas. Participants were administered the Diagnostic Interview Schedule (DIS; Robins *et al*. 1981) in the first wave of the ECA program, which included an assessment of manic symptoms (in accordance with DSM-III criteria; APA, 1980) over various recall periods (e.g. past 2 weeks, lifetime). Symptoms assessed from 2 weeks prior to participants' interviews were used because these data are minimally affected by long-term recall biases, and because it is clear that reported symptoms occurred during the same time period. The DIS contained nine dichotomous questions designed to assess symptoms of mania. These questions were collapsed into the eight diagnostic criteria of a Manic Episode [elevated or expansive mood ('elevated mood')[1]†, increased activity or physical restlessness ('increased activity'), increased talkativeness or pressured speech ('talkativeness'), flight of ideas or racing thoughts ('racing thoughts'), inflated self-esteem or grandiosity ('grandiosity'), decreased need for sleep ('decreased sleep'), distractibility, and excessive involvement in activities with high potential for painful consequences ('risky behavior'); APA, 1980] using syntax provided with the ECA data. Because of the probing structure of the DIS, an affirmative response to each symptom question indicated that the symptom did not only occur following drug or alcohol use. Test–retest reliability of diagnoses of Manic Episodes made using the DIS have been found to be acceptable ($\kappa = 0.56$; Semler *et al*. 1987). Concordance between diagnoses of Manic Episodes made by lay interviewers and psychiatrists using the DIS have also been found to be acceptable ($\kappa = 0.65$; Robins *et al*. 1981, 1982). Finally, concordance between DIS diagnoses of Manic Episodes and diagnoses based on semi-structured clinical interviews have been found to be good in clinical populations ($\kappa = 0.63$; Helzer *et al*. 1985; $\kappa = 0.86$; Wittchen *et al*. 1985).

Although all ECA participants were administered all manic symptom questions, a subsample ($n = 10\,105$) was selected from the ECA data for methodological reasons. Included participants belonged to the community sample (institutionalized individuals were

† The notes appear after the main text.

excluded because their inclusion could have produced a spurious taxon; Grove, 1991), were assessed for past 2-week symptoms, and completed the interview. The remaining sample contained more females than males [unweighted (weighted): 58.6% (53.2%) *v.* 41.4% (46.8%)], was predominantly White [unweighted (weighted): 56.8% (54.1%), 25.8% (22.7%) Black, 13.3% (17.3%) Hispanic, 1.7% (2.7%) Asian, 0.8% (0.9%) American Indian, and 1.6% (0.5%) other], and ranged in age from 18 to 96 years, with a mean age of 46 (unweighted; weighted = 41.9) years. The base rate of Manic Episodes was: lifetime (observed $n = 50$) 0.49% (unweighted; weighted = 0.46%); past 2 weeks (observed $n = 15$) 0.15% (unweighted; weighted = 0.16%). Missing data were negligible (2.2%) and were list-wise deleted (Kline, 2005).

Analytic strategy

Taxometric analyses were conducted using Ruscio's taxometric programs (Ruscio *et al*. 2006) for the R platform (R Development Core Team, 2009). All other analyses were conducted using MPlus version 5.1 (Muthén & Muthén, 2007).

Preliminary dimensionality analyses

Incorrectly specifying mania as unidimensional *versus* multidimensional can create difficulties in interpreting ITLDM results (Markon & Krueger, 2006). To address this potential difficulty, factor analyses were conducted. The sample was randomly divided into two equal subsamples. Exploratory factor analysis (EFA) was conducted on the first subsample, using the weighted least squares mean and variance adjusted (WLSMV) estimator (Muthén, 1989). A sample weight was applied to adjust for systematic non-response and differential selection probabilities. The number of factors to extract was determined by parallel analysis (Horn, 1965) with 1000 sets of random data. The results from the EFA model were used to construct a confirmatory factor analysis (CFA) model in the second subsample using WLSMV estimation. In addition to the above-mentioned sample weight, information regarding the stratification and clustering of the data was also modeled (Muthén, 2004). CFA model fit was evaluated according to Hu & Bentler's (1999) guidelines [Comparative Fit Index (CFI) > 0.95, Tucker–Lewis Index (TLI) > 0.95, root mean square error of approximation (RMSEA) < 0.05].

Taxometrics

Taxometric statistical methods have consistently demonstrated their ability to determine whether a construct has a discrete or a continuous latent structure (Meehl, 1973; Meehl & Yonce, 1994, 1996; Ruscio, 2000). Rigorous taxometric investigations include multiple non-redundant procedures, additional consistency checks, and simulation techniques (Ruscio *et al*. 2006). Although the ECA data have several strengths for taxometrics (e.g. large sample size, unselected sample), they also present several challenges. Specifically, the items available for the present analysis were dichotomous with sparse endorsement, and the hypothesized manic taxon had a very low base rate. Regarding the former concern, research has demonstrated that, given a large sample size (Ruscio, 2000) and proper implementation of analytic techniques (e.g. use of simulations and the inchworm consistency test; Waller & Meehl, 1998; Ruscio *et al*. 2004; Ruscio & Marcus, 2007), taxometric procedures are able to validly distinguish between discrete and continuous structures. Regarding the latter concern, simulation research has demonstrated that, with sufficient inclusion of taxon members along with a large sample size, taxometric procedures can detect base rates as low as 0.1–0.3% (Ruscio & Ruscio, 2004*b*). Ultimately, suitability for taxometric analysis was determined by simulating many sets of taxonic and dimensional data (Ruscio & Kaczetow, 2008); if the taxometric results from simulated taxonic and dimensional data could be distinguished from one another, then the data were suitable for analysis.

MAXCOV (MAXimum COVariance; Meehl & Yonce, 1996) was conducted on all possible input/output indicator configurations. Summed input indicators were not used because recent evidence suggests that using them for MAXCOV results in significantly less accurate results (Walters & Ruscio, 2009). Subsamples were created by dividing the sample into a large number of overlapping windows (i.e. 986 to 3944; derived using formula 6.5 in Ruscio *et al.* 2006, p. 138), with 90% overlap to allow for the inchworm consistency test and to provide more interpretable results (Ruscio *et al*. 2006). Ten internal replications were implemented in each run, and all MAXCOV curves were combined by averaging the covariance estimates for each subsample. The MAMBAC (Means Above Minus Below A Cut; Meehl & Yonce, 1994) was also conducted. Summed input indicators were used, and the first and last cuts were made 25 cases from each input indicator's distributional tails (Ruscio & Ruscio, 2004*a*). Internal replications were implemented as described above. To improve interpretability (Ruscio *et al*. 2006), the number of cuts for MAMBAC was held equal to the maximum number of overlapping windows used for MAXCOV (i.e. 3944). MAMBAC was repeated until all indicators had served as output, and curves were

combined by averaging mean-difference estimates at each cut along the input indicators.

Supplementary consistency tests included base rate divergence (Ruscio & Ruscio, 2004*b*) and the case removal consistency test (Ruscio, 2000). The inchworm consistency test was also conducted by repeating MAXCOV analyses several times with each successive run containing an increased number of overlapping windows (Waller & Meehl, 1998). Finally, the comparison of simulated taxonic and dimensional data and observed data was used as an interpretational tool in addition to a consistency test (Ruscio & Kaczetow, 2008). Taxonic data were simulated using a modification to the base rate classification method (Ruscio, 2009); individuals with the highest total item scores were assigned to the taxon based on the observed base rate of past 2-week Manic Episodes in the present sample. One hundred sets of taxonic and dimensional data were generated and submitted to the same analyses as the research data. Results from the research data were compared to results from simulated taxonic and dimensional data both visually and using fit indices (i.e. the comparison curve fit index; CCFI). CCFI values >0.60 support taxonic structure, values <0.40 support dimensional structure, and values between 0.40 and 0.60 are interpreted as ambiguous (Ruscio *et al*. 2007*a*).

ITLDM

ITLDM (Markon & Krueger, 2006) consists of estimating a variety of latent class (LCM) and latent trait (LTM) models using logistic modeling, and subsequently comparing their parsimony-adjusted fit using Bayesian Information Criteria (BIC). Specifically, (nominal) LCMs with between 2 and k classes (where $k=8=$number of indicators); LTMs (i.e. 'discrete metrical') with between 2 and k latent values [with each model distributed according to a binomial distribution, B($k-1$, 0.5), rescaled to a mean of 0 and a standard deviation (S.D.) of 1]; and a continuous normally distributed LTM were estimated. In addition to information regarding the stratification and clustering of the data, a sample weight was included to properly model the complex sample design of the data. Comparisons between LTMs with few *versus* many latent values evaluated the relative fit of discrete metrical and continuous models, respectively. Furthermore, comparisons between LTMs and LCMs with the same number of values/classes evaluated whether the target construct consisted of ordered or unordered categories. For each comparison, the exponential of 0.5 times the negative BIC difference between the two models was interpreted as the posterior odds of one model over the other (Raftery, 1995). Descriptively, a BIC difference of 0–2 equals 'weak' evidence, 2–6 equals 'positive' evidence, 6–10 equals 'strong evidence', and >10 equals 'very strong' evidence in favor of the model with the lower BIC value (Raftery, 1995).

Part I Results

Preliminary dimensionality analyses

Parallel analysis suggested that only one interpretable factor could be extracted from the data. Thus, a one-factor model was estimated in the first subsample, which provided a good fit to the data ($\chi^2=9.79$, df $=11$, $p=0.55$; RMSEA $=0.00$). The unidimensional CFA model constructed from these findings in the second subsample provided a good fit to the data ($\chi^2=20.25$, df $=11$, $p=0.04$; CFI $=0.96$, TLI $=0.96$, RMSEA $=0.01$). All factor loadings were statistically significant (mean loading $=0.58$, mean $r^2=0.37$).

Taxometrics

Indicator validity was excellent (mean indicator validity $=5.26$; Meehl, 1995) and within-class correlations were low (mean absolute value of $r=0.04$; Meehl, 1995). MAXCOV and inchworm consistency test plots are presented in Fig. 1. MAXCOV results were all consistent with a dimensional solution: in all cases, the line representing the results from the research data fit within ± 1 S.D. from the average results for simulated dimensional data, and fell on or outside of the boundaries of ± 1 S.D. from the average results for simulated taxonic data. CCFI values confirmed this superior fit, ranging from 0.26 to 0.30 (mean 0.28). Averaged MAMBAC results are presented in Fig. 2, along with averaged results from simulated taxonic and dimensional data. The averaged research curve resembles both the simulated dimensional and taxonic curves. The associated CCFI value was also ambiguous (CCFI $=0.45$).

Consistent with a dimensional interpretation, mean base rate estimates produced by MAXCOV (mean $=$ 0.24, S.D. $=0.30$) and MAMBAC (mean $=0.77$, S.D. $=$ 0.43) analyses were divergent. Further suggesting a dimensional interpretation, following targeted case removal, deviations from predicted base rate increases were substantial (MAXCOV deviation $=0.10$; MAMBAC deviation $=0.77$) and in the opposite direction than would be predicted by the presence of a taxon[2].

ITLDM

ITLDM results are presented in Table 1. The two estimated models with the best relative fit to the data

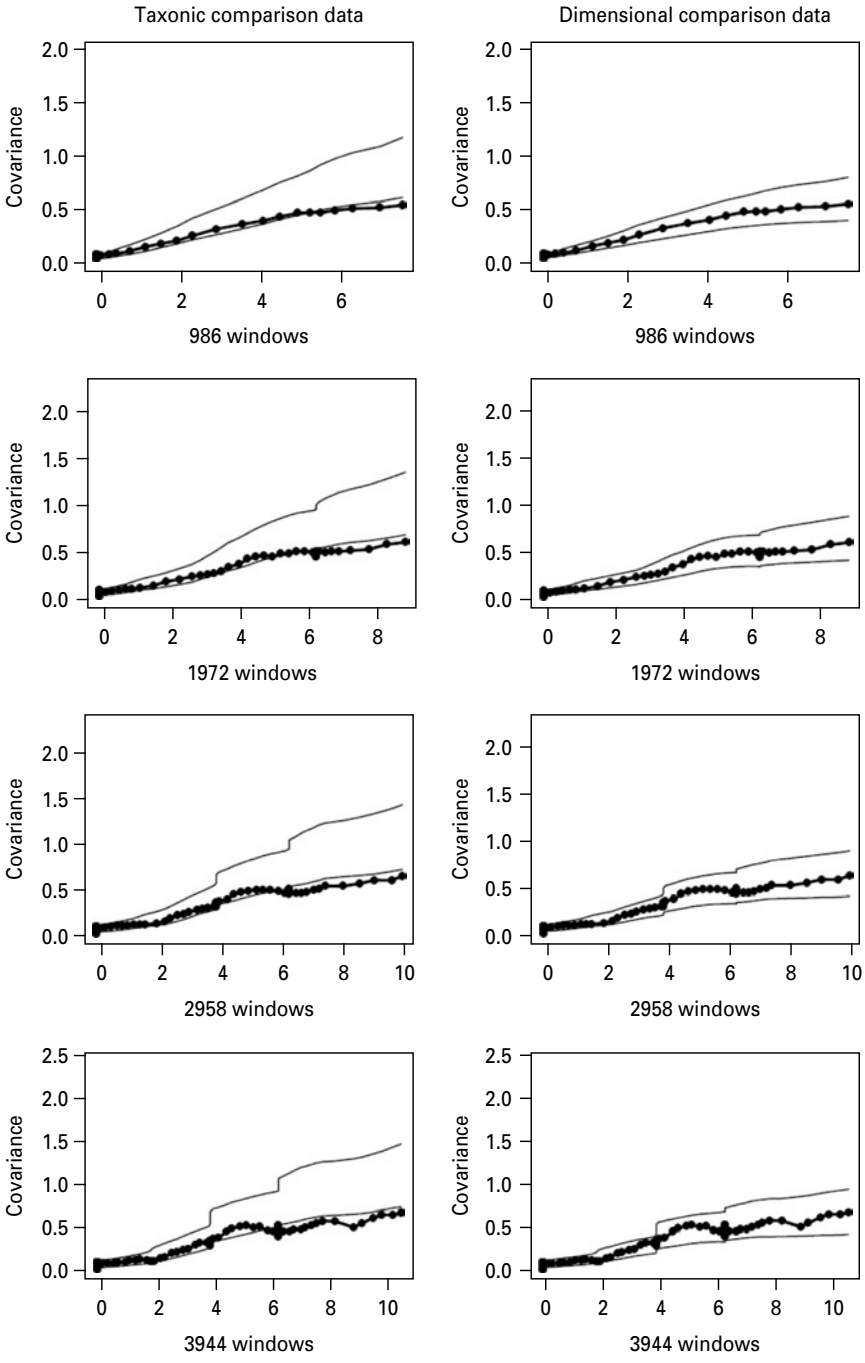

Fig. 1. Averaged maximum covariance (MAXCOV) results overlaying lines that represent ±1 S.D. from the average results for simulated taxonic (left) and dimensional (right) data. Plots in each successive row contain an increased number of overlapping windows: 986, 1972, 2958, 3944.

were the standard normal LTM (BIC = 9531.07) and the two-class LCM (BIC = 9534.82). The BIC difference between the two-class LCM and the standard normal LTM (3.75) corresponded to 'positive' evidence in favor of the LTM. In other words, the odds of the LTM over the two-class LCM were 6.52:1.

To guard against the possibility of spurious continuous findings, all analyses were repeated for lifetime symptom data that contained a larger number, and higher base rate, of hypothesized taxon members. These analyses also supported a continuous conceptualization of mania[3]. In sum, all taxometric and

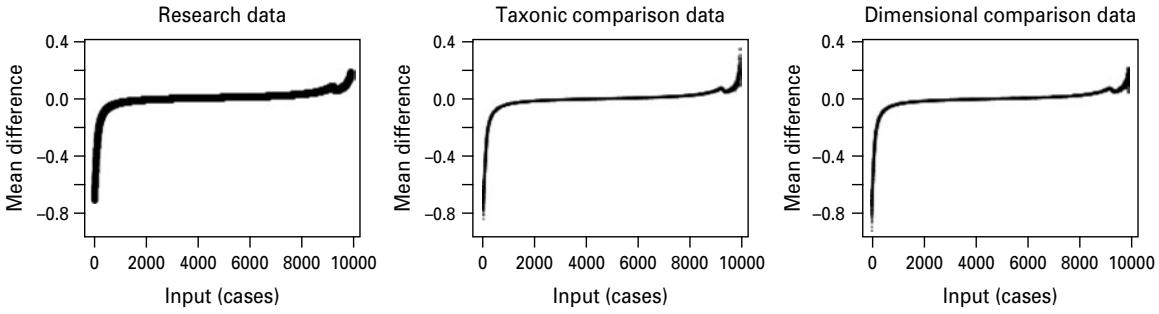

Fig. 2. Averaged means above minus below a cut (MAMBAC) results from research data (far left), simulated taxonic data (center), and simulated dimensional (far right) data.

Table 1. *Fit criteria for discrete and continuous models of mania*

	Criterion		
	k	ln(L)	BIC
Latent class models			
2 classes	17	−4689.23	9534.82
3 classes	26	−4681.21	9601.58
4 classes	35	−4672.09	9666.12
5 classes	44	−4665.76	9736.24
6 classes	53	−4663.99	9815.48
7 classes	62	−4659.92	9890.13
8 classes	71	−4653.03	9959.14
Latent trait models			
2 values	17	−4689.23	9534.82
3 values	18	−4688.93	9543.42
4 values	19	−4687.28	9549.32
5 values	20	−4687.54	9559.04
6 values	21	−4687.53	9568.22
7 values	22	−4687.33	9577.02
8 values	23	−4687.54	9586.64
Normal	16	−4691.95	9531.07

k, Number of parameters; ln(L), log likelihood; BIC, Bayesian Information Criterion.

ITLDM results supported a dimensional interpretation of mania in the ECA data.

Part II Introduction

Part II of the present study evaluated the relative predictive abilities of discrete and continuous models of mania using the methodology detailed in Prisciandaro & Roberts (2009). Two sets of model comparisons were made across outcomes: (1) between empirically derived discrete and continuous models from Part I of the present study and (2) between rationally selected discrete and continuous models. For the rationally selected continuous model, we created a single additive scale of mania with each manic symptom contributing 1 point. For the discrete model, we chose the predominant diagnostic model of mania: DSM Manic Episodes.

Three types of outcomes (i.e. dependent variables) were selected for predictive analyses: (1) psychiatric health service utilization, which is elevated among individuals with bipolar disorders (Weissman *et al*. 1991; Merikangas *et al*. 2007); (2) psychiatric disorders, which are highly co-morbid with bipolar disorders (Kessler *et al*. 1997; McElroy *et al*. 2001); and (3) suicidal behavior, which is elevated among individuals with bipolar disorders (Sharma & Markar, 1994; Kessler *et al*. 1999; Kallner *et al*. 2000).

The main criterion used to evaluate models' relative predictive abilities was whether the discrete model of mania predicted unique variance in outcomes once the continuous model was statistically controlled (and vice versa).

Part II Method

Sample

The sample for Part II of the present study was the same as for Part I. Missing data were negligible (4.2%) and were list-wise deleted (Kline, 2005).

Discrete and continuous models of mania

Empirically derived models

The best-fitting continuous and discrete models of mania from Part I ITLDM analyses were selected for subsequent predictive analyses (see Table 2 for model parameters). As reported in Part I, the best-fitting continuous model was the unidimensional, standard normal LTM (BIC = 9531.07). All factor loadings were statistically significant (mean loading = 0.69, mean $R^2 = 0.48$). This model was represented as a manifest continuous variable for regression analyses by computing factor scores from the standard normal LTM. The best-fitting discrete model was the two-class LCM (BIC = 9534.82). Class 1 ($n = 9474$, 'symptom free')

Table 2. *Standard normal LTM factor loadings and R^2 values, and two-class LCM item response probabilities for the eight DSM criteria of a manic episode*

	Standard normal LTM		Two-class LCM	
	Factor 1	R^2	Class 1 'Symptom free'	Class 2 'Potentially symptomatic'
Racing thoughts	0.81	0.66	0.00	0.22
Talkativeness	0.78	0.60	0.00	0.17
Distractibility	0.76	0.57	0.01	0.28
Decreased sleep	0.74	0.51	0.01	0.22
Risky behavior	0.64	0.41	0.00	0.05
Elevated mood	0.62	0.39	0.00	0.01
Grandiosity	0.60	0.37	0.01	0.17
Increased activity	0.55	0.31	0.01	0.13

LTM, Latent trait model; LCM, latent class model.

consisted of individuals with near-zero response probabilities across all symptoms (mean probability = 0.01) and class 2 ($n = 406$, 'potentially symptomatic') consisted of individuals with at least minimal response probabilities across all symptoms (mean probability = 0.16). This model was represented as a manifest discrete variable for regression analyses by calculating participants' posterior probabilities for each class, assigning participants to the class to which they were most likely to belong, and creating a dichotomous variable to reflect these class assignments.

Rationally selected models

The rationally selected continuous model of mania was a single additive scale of manic symptoms, with each symptom contributing 1 point. The empirically derived and rationally selected dimensional models of mania were strongly associated ($r = 0.97$, $p < 0.001$). The rationally selected discrete model was past 2-week DSM-III diagnoses of Manic Episodes ($n = 15$). The association between the empirically derived and rationally selected categorical models of mania was minimal but statistically significant ($r = 0.08$, $p < 0.001$).

Outcome variables

Outcome variables reflecting psychiatric diagnoses and suicidal behavior were assessed as part of the DIS. Health service utilization outcomes were assessed using a supplementary structured interview (Shapiro *et al*. 1985). Psychiatric diagnoses were assessed on a lifetime basis and were coded '0' = absent, '1' = present. Consistent with recommendations (e.g. Boyd *et al*. 1984; Meyers *et al*. 1984), DSM-III (APA, 1980) hierarchy rules were not observed. To reduce diagnostic outcomes, Krueger's (1999) dimensional model of common mental disorders was estimated using CFA. Suicidal behavior variables were assessed on a lifetime basis and were coded '0' = no, '1' = yes. Items covered: (1) thoughts of death; (2) desire to die; (3) thoughts of committing suicide; and (4) suicide attempts. Health service utilization variables (17 total) were assessed on a lifetime basis and were coded '0' = no, '1' = yes. Participants were asked if they had ever gone to a wide variety of people and places where 'someone might get help for problems with emotions, nerves, drugs, alcohol, or their mental health', including spiritual and natural healers, mental health and medical professionals, friends and relatives, and support groups.

Analytic strategy

Factor analyses

As described above, Krueger's (1999) model of common mental illnesses was estimated using CFA. Factor scores were computed for the two superordinate factors (internalizing and externalizing), and the resulting variables were used as outcomes in subsequent predictive analyses. Additionally, two separate unidimensional CFA models were estimated from the suicidal behavior and health service utilization variables, respectively; outcome variables were created by computing factor scores from each of these models. CFAs were conducted using the WLSMV (Muthén, 1989), and each incorporated the ECA sample weight in addition to information regarding the stratification and clustering of the data (Muthén, 2004).

Regression analyses

Regression models were estimated using the %SREGSUB macro (SAS Institute Inc., 2002) in SAS version 9.1.3

(SAS Institute Inc., USA), which allows for the analysis of subpopulations of complex survey data and includes information regarding the weighting, stratification and clustering of the data into parameter and variance estimations. Separate sets of predictive comparisons were conducted for the empirically derived and the rationally selected models of mania. Within each set of comparisons, separate stepwise regression models were estimated for each of the four outcome variables. Step 1 included a set of continuous or discrete predictors, whereas step 2 added the set of predictors not included in step 1. Each stepwise model was estimated twice for each outcome: once with the continuous predictor entered at step 1, and once with the discrete predictor entered at step 1. The improvement in model fit from step 1 to step 2 represented the degree to which one model of mania provided unique predictive validity beyond that of the alternative model. Of particular interest were the unique relationships between predictor variables and outcomes in step 2 models. β values involving predictors with meaningful scales of measurement (i.e. both of the rationally selected models and the discrete empirically derived model) were Y standardized. Alternatively, β's involving the continuous empirically derived model were fully (XY) standardized. Separate Bonferroni corrections were applied to significance tests of ΔR^2 and β to control experiment-wise α inflation. With a desired α of 0.05 for each type of test, Bonferroni corrections suggested an α of 0.0016 for individual ΔR^2 (0.05/32) and β (0.05/32) significance tests.

Part II Results

Factor analyses

Krueger's (1999) model of common mental illnesses provided a good fit to the data ($\chi^2 = 134.02$, df = 19, $p < 0.001$; CFI = 0.95, TLI = 0.95, RMSEA = 0.02), and all factor loadings were statistically significant (mean loading = 0.74, mean $R^2 = 0.56$). Internalizing and externalizing factors were significantly correlated ($r = 0.43$, $p < 0.001$). The suicidal behavior CFA model provided an acceptable fit to the data ($\chi^2 = 142.21$, df = 2, $p < 0.001$; CFI = 0.98, TLI = 0.95, RMSEA = 0.07), and all factor loadings were statistically significant (mean loading = 0.83, mean $R^2 = 0.70$). Finally, the health service utilization CFA model provided a good fit to the data ($\chi^2 = 126.83$, df = 26, $p < 0.001$; CFI = 0.95, TLI = 0.97, RMSEA = 0.02), and all factor loadings were statistically significant (mean loading = 0.71, mean $R^2 = 0.51$). The four outcome variables that were created from these models (by computing factor scores for all participants) had acceptable levels of skew and kurtosis (<3 and <10 respectively; Kline, 2005).

Regression analyses

Empirically derived models

The results regarding the empirically derived models' abilities to predict outcomes are presented in Table 3. This table shows that the inclusion of the discrete model at step 2 did not result in a statistically significant improvement in R^2 for any of the four outcomes. In addition, the discrete model did not uniquely predict any of the four outcomes when the continuous model was considered simultaneously. Table 3 also shows that the inclusion of the continuous model at step 2 resulted in a statistically significant improvement in R^2 for each of the four outcomes (mean $\Delta R^2 = 0.02$, mean increase in $R^2 = 89\%$). Furthermore, the continuous model significantly uniquely predicted each of the four outcomes when the discrete model was considered simultaneously (mean $\beta = 0.20$). In sum, the continuous model demonstrated unambiguously superior predictive validity relative to the discrete model; the latter model had no incremental validity over the former model.

Rationally selected models

The results regarding the rationally selected models' abilities to predict outcomes are presented in Table 4. This table shows that the inclusion of the discrete model at step 2 led to a statistically significant improvement in R^2 for internalizing, externalizing and suicidal behavior (mean $\Delta R^2 = 0.003$, mean percentage increase in $R^2 = 7\%$), but not for health service utilization. However, the discrete model only uniquely predicted one of the four outcomes (suicidal behavior: $\beta = 0.16$) when the continuous model was considered simultaneously. Table 4 also shows that the inclusion of the continuous model at step 2 resulted in a statistically significant improvement in R^2 for each of the four outcomes (mean $\Delta R^2 = 0.04$, mean percentage increase in $R^2 = 2296\%$). Furthermore, the continuous model significantly uniquely predicted each of the four outcomes when the discrete model was considered simultaneously (mean $\beta = 0.07$). In sum, although the discrete model demonstrated some incremental predictive validity relative to the continuous model, the increase in R^2 associated with the inclusion of the discrete model was very small, and the discrete model only uniquely predicted one outcome when the continuous model was considered simultaneously.

Taken together, findings from Part II provide strong and consistent support for continuous models of mania, and weak and inconsistent support for discrete

Table 3. *Multiple regression analyses with empirically derived continuous or discrete models of mania alone predicting four outcomes, followed by analyses with both models of mania simultaneously predicting outcome*

	Internalizing disorders		Externalizing disorders		Suicidal behavior		Health service utilization	
	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β
I. Addition of discrete predictors to models with continuous predictors only								
Step 1								
Standard normal LTM	0.06*	0.25*	0.04*	0.19*	0.04*	0.20*	0.03*	0.18*
Step 2								
Standard normal LTM	0.00	0.24*	0.00	0.20*	0.00	0.20*	0.00	0.18*
Two-class LCM		0.01		−0.01		0.01		0.00
II. Addition of continuous predictors to models with discrete predictors only								
Step 1								
Two-class LCM	0.03*	0.17*	0.02*	0.12*	0.02*	0.14*	0.02*	0.12*
Step 2								
Two-class LCM	0.03*	0.01	0.02*	−0.01	0.02*	0.01	0.02*	0.00
Standard normal LTM		0.24*		0.20*		0.20*		0.18*

Regression coefficients involving the standard normal LTM were XY standardized. Coefficients involving the two-class LCM were Y standardized.

LTM, Latent trait model; LCM, latent class model; ΔR^2, change in proportion of variance accounted for; β, regression coefficient.

* $p < 0.0016$.

Table 4. *Multiple regression analyses with rationally selected continuous or discrete models of mania alone predicting four outcomes, followed by analyses with both models of mania simultaneously predicting outcomes*

	Internalizing disorders		Externalizing disorders		Suicidal behavior		Health service utilization	
	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β
I. Addition of discrete predictors to models with continuous predictors only								
Step 1								
Manic Symptoms Scale	0.06*	0.08*	0.03*	0.06*	0.04*	0.07*	0.03*	0.06*
Step 2								
Manic Symptoms Scale	0.00*	0.08*	0.00*	0.06*	0.00*	0.07*	0.00	0.06*
Manic Episodes		0.18		0.22		0.16*		0.04
II. Addition of continuous predictors to models with discrete predictors only								
Step 1								
Manic Episodes	0.00*	0.23*	0.01*	0.26*	0.00*	0.21*	0.00	0.08
Step 2								
Manic Episodes	0.05*	0.18	0.03*	0.22	0.04*	0.16*	0.03*	0.04
Manic Symptoms Scale		0.08*		0.06*		0.07*		0.06*

LTM, Latent trait model; LCM, latent class model; ΔR^2, change in proportion of variance accounted for; β, Y standardized regression coefficient.

* $p < 0.0016$.

models of mania. To guard against the possibility of spurious continuous findings, all analyses were repeated for lifetime symptom data that contained a larger number, and higher base rate, of diagnosed Manic Episodes. These analyses also supported a continuous conceptualization of mania[4].

Discussion

The present study is the first to examine whether mania is a discrete or a continuous construct using appropriate methodologies. In an unselected epidemiologic sample of approximately 10 000 individuals, the manic symptom questions from the structured DIS were submitted to a wide variety of taxometric procedures and consistency tests, in addition to ITLDM. The relative predictive validities of various continuous and discrete models of mania were also examined. The results all converged on a continuous solution.

These results provide support for prominent theories of bipolar disorders that conceptualize mania as an extreme variant of human functioning (e.g. Kraepelin, 1921; Depue *et al*. 1987). They are also consistent with studies demonstrating the validity of subthreshold mania (e.g. Merikangas *et al*. 2007). Taxometric studies that have investigated other bipolar mood constructs, such as depression (e.g. Ruscio & Ruscio, 2000; Prisciandaro & Roberts, 2005; Slade & Andrews, 2005) and hyperthymic temperament (Meyer & Keller, 2003), have also found these constructs to be continuous[5]. However, the latent structures of key bipolar mood constructs (e.g. mixed mania, mood cycling) have not yet been examined using appropriate statistical methodologies. Future studies should examine the reliability of the present study's findings, and also conduct similar investigations on related bipolar constructs.

The present study had several methodological strengths (e.g. large sample size, high indicator validity) that allowed it to overcome challenging aspects of the data (e.g. sparse, dichotomous data). Although unselected community samples are generally preferred in structural investigations because they minimize the likelihood of spurious dimensional or categorical findings (Ruscio *et al*. 2006), the present study's extremely low number of currently manic individuals potentially increased the likelihood of spurious dimensional findings. However, all analyses were repeated on lifetime manic symptom data, with an increased number of manic individuals, and these results converged on a continuous interpretation. Nevertheless, future research should evaluate the structure of mania in samples that are more likely to contain currently manic individuals.

The present study's support for a unidimensional representation of mania is not entirely consistent with previous factor analytic studies, which have found between two and seven latent factors (Murphy & Beigel, 1974; Cassidy *et al*. 1998; Swann *et al*. 2001; Sato *et al*. 2002; Akiskal *et al*. 2003). However, these studies have extracted factors reflecting depression and psychosis, suggesting that symptoms unrelated to mania were included. Nonetheless, future factor analytic studies of DSM mania should include more than one item for each DSM criterion to ensure sufficient coverage. Although the present study used indicators created from DSM-III (APA, 1980) criteria, present-day Manic Episode criteria (APA, 1994) are nearly identical to those in DSM-III. Finally, because irritable mood was only assessed in a highly selected group of individuals in the ECA, the absence of irritable mood as a symptom indicator in the present study was an additional limitation; the results of our study are thus more applicable to euphoric mania than to a conceptualization of mania that accepts irritable mood in the absence of euphoric/elevated mood.

If the results from the present study are supported by future investigations, this may argue for the use of a continuous representation of mania. If continuous conceptualizations of mania are more valid than discrete representations, categorical selection methods for research (e.g. selecting participants diagnosed with DSM Manic Episodes) would reduce statistical power (Cohen, 1983), prevent proper investigation of dimensional theoretical models of mania (e.g. Depue *et al*. 1987), disguise potential non-linear relationships between mania and other variables (Ruscio *et al*. 2004), and create spurious statistically significant effects (Maxwell & Delaney, 1993). Instead, correlational research designs that sample individuals at all levels of manic symptom severity should be used. To sample individuals at all levels of symptom severity, continuous measures of manic symptoms must be further developed as existing measures may not reflect the true latent structure of mania.

As Akiskal and colleagues have demonstrated (e.g. Judd & Akiskal, 2003), the issue of clinical significance needs to be revisited for bipolar disorders; it is clear that subthreshold expressions of mania can result in pervasive impairment. The findings from the present study further support Akiskal and colleagues' reasoned arguments for expansion of the bipolar spectrum. It is important to recognize, however, that support for a continuous conceptualization of mania suggests that there are *no* points at which a certain number of symptoms or a certain level of symptom severity inherently denotes the presence of a manic disorder. Instead, a continuous conceptualization of mania necessitates that empirical associations between mania and important outcomes (e.g. functional impairment) form the primary basis for determining treatment resource allocation. For example, if the relationship between mania and impairment is non-linear, treatment efforts could be directed at individuals whose levels of manic symptoms place them at the cusp of a substantial increase in functional impairment. If, instead, the relationship between mania

and impairment is linear, a graded continuum of care could be provided to affected individuals, such that different degrees of manic severity are met with concordant levels of treatment intensity (e.g. ranging from psycho-education to hospitalization). Cost–benefit analyses could be used to determine which specific treatments are most effective at particular levels of severity. Before such a plan can be instituted, however, research is needed to determine the form of the relationship between mania and functional impairment, and to further investigate the cost–benefit ratios of specific treatments at varying levels of manic symptom severity.

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Declaration of Interest

None.

Notes

[1] In the ECA, only participants who endorsed $\geqslant 3$ co-occurring symptoms of mania and denied elevated or expansive mood were asked a question regarding irritable mood. Because the question was not asked of all participants, irritable mood was not included in the present analyses.

[2] Taxonic data can be simulated using a variety of methods, and the method of simulation chosen can significantly impact the taxometric results. Thus, it is important to evaluate the robustness of the taxometric results across different simulation methods. All taxometric analyses were reconducted using an alternate method of taxonic data simulation. The base rate classification method (Ruscio, 2009) assigns individuals with the highest total indicator scores to the taxon group based on the mean base rate estimate from taxometric analyses of the research data. All MAXCOV and MAMBAC curves derived from the research data were more similar visually to curves derived from simulated dimensional data than to curves from simulated taxonic data. CCFI values obtained from MAXCOV (mean CCFI = 0.15) and MAMBAC (CCFI = 0.04) analyses further supported a dimensional interpretation of the data. Overall, these supplementary results suggest that the present study's dimensional taxometric findings were not solely determined by the taxonic data simulation method used. For further details, see www.buffalo.edu/~robertsj/psychmed.2010.supplement.pdf or www.drtprogram.cjb.net/psychmed.2010.supplement.pdf.

[3] Although past 2-week symptom data are preferable to lifetime data (and simulations suggested that the former could distinguish taxonic and dimensional structures), they are also potentially problematic because the base rate of Manic Episodes over a 2-week period is small (0.15%) and item endorsement is sparse. All analyses were repeated for lifetime symptom data to evaluate the robustness of obtained findings in data with a larger base rate of Manic Episodes (0.49%). Preliminary dimensionality analyses suggested a single latent factor. Twelve sets of MAXCOV analyses across three methods of taxonic data simulation unambiguously supported a continuous interpretation of the data (mean CCFI = 0.20). Two of three sets of MAMBAC analyses supported a continuous interpretation (mean CCFI = 0.22); the third set was ambiguous (CCFI = 0.46). ITLDM analyses suggested that a standard normal LTM of mania provided the best relative fit to the data. The BIC difference (1.06) between this model and the second-best fitting model (LTM with four latent values) corresponded to 'weak' evidence in favor of the standard normal model. In other words, the odds of the normal LTM over the four-valued LTM were 1.7:1. Overall, these results support a continuous interpretation of the data, and suggest that the results presented from the past 2-week data were not unduly influenced by methodological concerns regarding an insufficient number of hypothesized taxon members in the sample. For further details, see www.buffalo.edu/~robertsj/psychmed.2010.supplement.pdf or www.drtprogram.cjb.net/psychmed.2010.supplement.pdf.

[4] Because the base rates of manic symptoms and Manic Episodes in the present study's 2-week recall period were low, models of mania, especially the rationally selected discrete model, may have been limited in their ability to predict outcomes. Therefore, all predictive analyses were repeated for lifetime symptom data to evaluate the robustness of regression findings in data with a larger base rate of Manic Episodes (0.49% = 50 potential taxon members). The inclusion of the empirically derived or rationally selected continuous model at step 2 resulted in a statistically significant improvement in R^2 for each of the four outcomes (empirically derived: mean $\Delta R^2 = 0.03$, mean percentage increase in $R^2 = 41\%$; rationally selected: mean $\Delta R^2 = 0.09$, mean percentage increase in $R^2 = 580\%$). Furthermore, in all cases, the continuous model significantly uniquely predicted each of the four outcomes when the discrete model was considered simultaneously (mean $\beta = 0.30$). The inclusion of the empirically derived discrete model at step 2 led to a statistically significant improvement in R^2 for health service utilization ($\Delta R^2 = 0.002$, percentage increase in $R^2 = 2\%$), but not for internalizing, externalizing and suicidal behavior. The inclusion of the rationally selected discrete model at step 2 led to a statistically significant improvement in R^2 for externalizing (mean $\Delta R^2 = 0.001$, mean percentage increase in $R^2 = 1\%$), but not for internalizing, health service utilization and suicidal behavior. However, in no cases did the discrete model uniquely predict any of the four outcomes when the continuous model was considered

simultaneously. Taken together, findings from these supplementary analyses provide strong and consistent support for continuous models of mania, and weak and inconsistent support for discrete models of mania. For further details, see www.buffalo.edu/~robertsj/psychmed.2010.supplement.pdf or www.drtprogram.cjb.net/psychmed.2010.supplement.pdf.

[5] Although a few taxometric studies (e.g. Solomon *et al*. 2006; Ruscio *et al*. 2007*b*) have supported a discrete structure of depression, all available construct validity investigations have supported a continuous interpretation (Aggen *et al*. 2005; Prisciandaro & Roberts, 2009).

References

Aggen SH, Neale MC, Kendler KS (2005). DSM criteria for major depression: evaluating symptom patterns using latent-trait item response models. *Psychological Medicine* **35**, 475–487.

Akiskal HS, Azorin JM, Hantouche EG (2003). Proposed multidimensional structure of mania: beyond the euphoric-dysphoric dichotomy. *Journal of Affective Disorders* **73**, 7–18.

APA (1980). *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edn. American Psychiatric Association: Washington, DC.

APA (1994). *Diagnostic and Statistical Manual of Mental Disorders*, 4th edn. American Psychiatric Association: Washington, DC.

Boyd JH, Burke Jr. JD, Gruenberg E, Holzer III CE, Rae DS, George LK, Karno M, Stoltzman R, McEvoy L, Nestadt G (1984). Exclusion criteria of DSM-III: a study of co-occurrence of hierarchy-free syndromes. *Archives of General Psychiatry* **41**, 983–989.

Cassidy F, Forest K, Murry E, Carroll BJ (1998). A factor analysis of the signs and symptoms of mania. *Archives of General Psychiatry* **55**, 27–32.

Cohen J (1983). The cost of dichotomization. *Applied Psychological Measurement* **7**, 249–253.

Depue RA, Krauss S, Spoont MR (1987). A two-dimensional threshold model of seasonal bipolar affective disorder. In *Psychopathology: An Interactional Perspective* (ed. D. Magnusson and A. Ohman), pp. 95–123. Academic Press: Orlando, FL.

Eaton WW, Kessler LG (1985). *Epidemiologic Field Method in Psychiatry: The NIMH Epidemiologic Catchment Area Program*. Academic Press: Orlando, FL.

Grove WM (1991). Validity of taxometric inferences based on cluster analysis stopping rules. In *Thinking Clearly About Psychology: Essays in Honor of Paul E. Meehl, Vol. 2: Personality and Psychopathology* (ed. D. Cicchetti and W. M. Grove), pp. 313–329. University of Minnesota Press: Minneapolis, MN.

Haslam N (2003). The dimensional view of personality disorders: a review of the taxometric evidence. *Clinical Psychology Review* **23**, 75–93.

Helzer JE, Robins LN, McEvoy LT, Spitznagel EL, Stoltzman RK, Farmer A, Brockington IF (1985). A comparison of clinical and diagnostic interview schedule diagnoses. Physician reexamination of lay-interviewed cases in the general population. *Archives of General Psychiatry* **42**, 657–666.

Horn JL (1965). A rationale and test for the number of factors in factor analysis. *Psychometrika* **30**, 179–185.

Hu L, Bentler PM (1999). Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. *Structural Equation Modeling* **6**, 1–55.

Judd LL, Akiskal HS (2003). The prevalence and disability of bipolar spectrum disorders in the US population: re-analysis of the ECA database taking into account subthreshold cases. *Journal of Affective Disorders* **73**, 123–131.

Kallner G, Lindelius R, Petterson U, Stockman O, Tham A (2000). Mortality in 497 patients with affective disorders attending a lithium clinic or after having left it. *Pharmacopsychiatry* **33**, 8–13.

Kessler RC, Borges G, Walters EE (1999). Prevalence of and risk factors for lifetime suicide attempts in the National Comorbidity Survey. *Archives of General Psychiatry* **56**, 617–626.

Kessler RC, Rubinow DR, Holmes C, Abelson JM, Zhao S (1997). The epidemiology of DSM-III-R bipolar I disorder in a general population survey. *Psychological Medicine* **27**, 1079–1089.

Kline RB (2005). *Principles and Practice of Structural Equation Modeling*, 2nd edn. Guilford Press: New York.

Kraepelin E (1921). *Manic-Depressive Insanity and Paranoia*. Livingstone: Edinburgh.

Krueger RF (1999). The structure of common mental disorders. *Archives of General Psychiatry* **56**, 921–926.

Lenzenweger MF (2004). Consideration of the challenges, complications, and pitfalls of taxometric analysis. *Journal of Abnormal Psychology* **113**, 10–23.

Markon KE, Krueger RF (2006). Information-theoretic latent distribution modeling: distinguishing discrete and continuous latent variable models. *Psychological Methods* **11**, 228–243.

Maxwell SE, Delaney HD (1993). Bivariate median splits and spurious statistical significance. *Psychological Bulletin* **113**, 181–190.

McElroy SL, Altshuler LL, Suppes T, Keck Jr. PE, Frye MA, Denicoff KD, Nolen WA, Kupka RW, Leverich GS, Rochussen JR, Rush AJ, Post RM (2001). Axis I psychiatric comorbidity and its relationship to historical illness variables in 288 patients with bipolar disorder. *American Journal of Psychiatry* **158**, 420–426.

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

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

Meehl PE, Yonce LJ (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 PE, Yonce LJ (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.

Merikangas KR, Akiskal HS, Angst J, Greenberg PE, Hirschfeld RMA, Petukhova M, Kessler RC (2007). Lifetime and 12-month prevalence of bipolar spectrum disorder in the National Comorbidity Survey replication. *Archives of General Psychiatry* **64**, 543–552.

Meyer TD, Keller F (2003). Is there evidence for a latent class called 'hypomanic temperament'? *Journal of Affective Disorders* **75**, 259–267.

Meyers JK, Weissman MM, Tischler GL, Holzer III CE, Leaf PJ, Orvaschel H, Anthony JC, Boyd JH, Burke Jr. JD, Kramer M, Stoltzman R (1984). Six-month prevalence of psychiatric disorders in three communities. *Archives of General Psychiatry* **41**, 959–967.

Murphy DL, Beigel A (1974). Depression, elation, and lithium carbonate responses in manic patient subgroups. *Archives of General Psychiatry* **31**, 643–648.

Muthén BO (1989). Dichotomous factor analysis of symptom data. *Sociological Methods and Research* **18**, 19–65.

Muthén BO (2004). *Mplus Technical Appendices*. Muthén & Muthén: Los Angeles, CA.

Muthén LK, Muthén BO (2007). *Mplus User's Guide*, 5th edn. Muthén & Muthén: Los Angeles, CA.

Nichols DS, Jones Jr. RE (1985). Identifying schizoid-taxon membership with the Golden-Meehl MMPI items. *Journal of Abnormal Psychology* **94**, 191–194.

Prisciandaro JJ, Roberts JE (2005). A taxometric investigation of unipolar depression in the National Comorbidity Survey. *Journal of Abnormal Psychology* **114**, 718–728.

Prisciandaro JJ, Roberts JE (2009). A comparison of the predictive abilities of dimensional and categorical models of unipolar depression in the National Comorbidity Survey. *Psychological Medicine* **39**, 1087–1096.

R Development Core Team (2009). *R: A Language and Environment for Statistical Computing*. R Foundation for Statistical Computing: Vienna, Austria.

Raftery AE (1995). Bayesian model selection in social research. *Sociological Methodology* **25**, 111–196.

Robins LN, Helzer JE, Croughan J, Ratcliff KS (1981). National Institute of Mental Health Diagnostic Interview Schedule: its history, characteristics, and validity. *Archives of General Psychiatry* **38**, 381–389.

Robins LN, Helzer JE, Ratcliff KS, Seyfried W (1982). Validity of the Diagnostic Interview Schedule, Version II: DSM-III diagnoses. *Psychological Medicine* **12**, 855–870.

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 (2009). Assigning cases to groups using taxometric results: an empirical comparison of classification techniques. *Assessment* **16**, 55–70.

Ruscio J, Haslam N, Ruscio AM (2006). *Introduction to the Taxometric Method: A Practical Guide*. Lawrence Erlbaum Associates: Mahwah, NJ.

Ruscio J, Kaczetow W (2008). Simulating multivariate nonnormal data using an iterative algorithm. *Multivariate Behavioral Research* **43**, 355–381.

Ruscio J, Marcus DK (2007). Detecting small taxa using simulated comparison data: a reanalysis of Beach, Amir, and Bau's (2005) data. *Psychological Assessment* **19**, 241–246.

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

Ruscio J, Ruscio AM (2004*a*). A nontechnical introduction to the taxometric method. *Understanding Statistics* **3**, 151–193.

Ruscio J, Ruscio AM (2004*b*). Clarifying boundary issues in psychopathology: the role of taxometrics in a comprehensive program of structural research. *Journal of Abnormal Psychology* **113**, 24–38.

Ruscio J, Ruscio AM, Keane TM (2004). Using taxometric analysis to distinguish a small latent taxon from a latent dimension with positively skewed indicators: the case of involuntary defeat syndrome. *Journal of Abnormal Psychology* **113**, 145–154.

Ruscio J, Ruscio AM, Meron M (2007*a*). Applying the bootstrap to taxometric analysis: generating empirical sampling distributions to help interpret results. *Multivariate Behavioral Research* **42**, 349–386.

Ruscio J, Zimmerman M, McGlinchey JB, Chelminski I, Young D (2007*b*). Diagnosing major depressive disorder: XI. A taxometric investigation of the categorical-dimensional debate on the structure underlying DSM-IV symptoms. *Journal of Nervous and Mental Disease* **195**, 10–19.

SAS Institute Inc. (2002). %SREGSUB macro provides additional capabilities for PROC SURVEYREG (http://support.sas.com/kb/24/985.html#ref). Accessed 5 June 2008.

Sato T, Bottlender R, Kleindienst N, Moller H-J (2002). Syndromes and phenomenological subtypes underlying acute mania: a factor analytic study of 576 manic patients. *American Journal of Psychiatry* **159**, 968–974.

Semler G, Wittchen H-U, Joschke K, Zaudig M, Vongeiso T, Kaiser S, von Cranach M, Pfister H (1987). Test-retest reliability of a standardized psychiatric interview (DIS/CIDI). *European Archives of Psychiatry and Neurological Sciences* **236**, 214–222.

Shapiro S, Tischler GL, Cottler L, George LK, Amirkhan JH, Kessler LG, Skinner EA (1985). Health services research questions. In *Epidemiologic Field Method in Psychiatry: The NIMH Epidemiologic Catchment Area Program* (ed. W. W. Eaton and L. G. Kessler), pp. 191–208. Academic Press: Orlando, FL.

Sharma R, Markar HR (1994). Mortality in affective disorder. *Journal of Affective Disorders* **31**, 91–96.

Slade T, Andrews G (2005). Latent structure of depression in a community sample: a taxometric analysis. *Psychological Medicine* **35**, 489–497.

Solomon A, Ruscio J, Seeley JR, Lewinsohn PM (2006). A taxometric investigation of unipolar depression in a large community sample. *Psychological Medicine* **36**, 973–985.

Swann AC, Janicak PL, Calabrese JR, Bowden CL, Dilsaver SC, Morris DD, Petty F, Davis JM (2001). Structure of mania: depressive, irritable,

and psychotic clusters with different retrospectively-assessed course patterns of illness in randomized clinical trial participants. *Journal of Affective Disorders* **67**, 123–132.

Waldman ID, Lilienfeld SO (2001). Applications of taxometric methods to problems of comorbidity: perspectives and challenges. *Clinical Psychology: Science and Practice* **8**, 520–527.

Waller NG, Meehl PE (1998). *Multivariate Taxometric Procedures: Distinguishing Types from Continua*. Sage Publications, Inc.: Thousand Oaks, CA.

Walters GD, Ruscio J (2009). To sum or not to sum: taxometric analysis with ordered categorical assessment items. *Psychological Assessment* **21**, 99–111.

Watson D (2003). Investigating the construct validity of the dissociative taxon: stability analyses of normal and pathological dissociation. *Journal of Abnormal Psychology* **112**, 298–305.

Weissman MA, Bruce ML, Leaf PJ, Florio LP, Holzer III C (1991). Affective disorders. In *Psychiatric Disorders in America: The Epidemiologic Catchment Area Study* (ed. L. N. Robins and D. A. Regier), pp. 53–80. Free Press: New York, NY.

Wittchen H-U, Semler G, von Zerssen D (1985). A comparison of two diagnostic methods: clinical ICD diagnoses vs. DSM-III and Research Diagnostic Criteria using the Diagnostic Interview Schedule (version 2). *Archives of General Psychiatry* **42**, 677–684.