Patterns of Covariation of DSM-IV Personality Disorders in a Mixed Psychiatric Sample

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The covariation patterns of DSM-IV personality disorders (PDs) were studied in 431 consecutively admitted psychiatric patients. The co-occurrence rate was greater than 50% for all DSM-IV PDs. Both bivariate association tests and loglinear models showed distinct significant covariation patterns among PDs which were stable across confounder strata. DSM-IV PD clusters were not replicated, with the exception of cluster A. Principal-component analysis (PCA) showed the presence of 3 latent dimensions, thus explaining the DSM-IV PD covariation patterns. These results seem to stress the inadequacy of the DSM-IV categorical model of PD assessment. The need for a reduction of axis II categories and the inclusion of a dimensional model in the diagnostic assessment of DSM-IV PDs are discussed.

CONSISTENT DATA from the literature show that at least 50% of the patients diagnosed as having any type of personality disorder (PD) receive 2 or more PD diagnoses.[1-4] Moreover, the use of structured interviews for PD diagnosis increased the rate of PD co-occurrence when compared with clinical interview or chart review.[1]

This finding is frequently referred to as "comorbidity," that is, the coexistence of 2 or more independent disorders.[4,5] However, interpreting the co-occurrence of PDs as the co-presence of independent disorders seems problematic. In fact, previous studies on the co-occurrence of PD diagnoses[1,3,5-13] showed significant associations between several PDs. A substantial variation in the size and direction of PD covariation was observed across studies, mainly because of their methodological heterogeneity.[4] Moreover, co-occurrence, as well as covariation, depends on the prevalence of the respective PDs. This could be influenced by several confounders such as differences in the diagnostic threshold, method of assessment, patient severity,[4] subject gender,[13] and diagnostic system. Furthermore, it could occur in several ways, such as by including overlapping criteria, emphasizing multiple diagnoses rather than differential diagnosis, or demarcating different categories along a shared spectrum of pathology.[14] The stability of PD covariation patterns across these confounders should be tested before making any conclusion about their generalizability. Unfortunately, only a few studies[3-5] have tried to assess their effect.

Despite these methodological problems, the evidence of significant covariation among PDs raised doubts about the validity and clinical usefulness of the DSM-III-R categorical model of PDs. Some authors[5] suggested that the categorical diagnostic system of PDs could be maintained with several deep modifications, ranging from the elimination of overlapping criteria to the collapsing of present categories into superordinate clusters on the basis of statistically based hierarchies. On the contrary, other authors[15-17] claimed that the significant covariation observed among PDs could be explained by the presence of common underlying personality dimensions, and suggested that a dimensional model would be more appropriate. A number of studies[7,8,11,12,18] were performed to identify the dimensions underlying the covariation of PDs. Unfortunately, these studies did not provide consistent results, perhaps due to substantial methodological variability.[4] No definitive evidence was found for DSM-III-R clusters; rather, some studies showed strong similarities between some of the dimensions underlying DSM-III PDs and those identified by the 5-factor personality model.[4,11]

With regard to DSM-III-R, DSM-IV[19] made a noticeable effort to reduce the overlap between PD criteria and sharpen the boundaries between individual PDs. Diagnostic thresholds were modified for several PDs, sadistic PD was removed, and depressive and passive-aggressive (negativistic) PDs were included as diagnostic categories necessitating further study.

All of these modifications, which could poten-

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tially deeply influence the PD base rate and phenomenology, stress the need for a reassessment of PD covariation.

Starting from these considerations, the aims of this study were to (1) analyze the patterns of covariation of DSM-IV PDs in a mixed psychiatric sample, (2) evaluate the potential confounding effect of subject gender and severity (i.e., male *v* female, presence *v* absence of any axis I diagnosis, and inpatient *v* outpatient), and (3) identify the dimensions underlying the covariation patterns of DSM-IV PDs and test their replicability across the confounder levels.

METHOD

The sample consisted of 431 subjects consecutively admitted from January to October 1997 to the Clinical Psychology and Psychotherapy Unit of the Scientific Institute H. San Raffaele, Milan, Italy, a specialized unit in the diagnosis and treatment of PDs. The exclusion criteria were as follows: IQ of 75 or less; axis I diagnosis of schizophrenia, schizoaffective disorder, delusional disorder, or organic mental disorder; and educational level less than elementary school. All subjects provided informed consent to participate in the study after a detailed description. One hundred sixty-two subjects (37.6%) were male and 269 (62.4%) were female. The mean age was 29.81 ± 8.34 years (mean ± SD). Three hundred twenty-seven subjects (75.9%) were unmarried, 72 (16.6%) were married, 30 (7.0%) were divorced, and 2 (0.5%) were widowed. Two hundred thirteen subjects (49.4%) were inpatients and 218 (50.6%) outpatients. Two hundred sixty-two subjects (60.8%) received at least 1 axis I diagnosis; the most frequently diagnosed axis I disorders were anxiety disorders (n = 120, 27.8%), eating disorders (n = 70, 16.2%), mood disorders (n = 33, 7.7%), substance abuse/dependence disorders (n = 19, 4.4%), and brief/NOS psychotic disorder (n = 10, 2.3%). Twenty-one subjects (4.9%) received other axis I diagnoses (e.g., sleep disorders, tic disorder, etc.). The cumulative frequency and percentage of subjects with specific axis I diagnoses exceeded the frequency and percentage of subjects with at least 1 axis I diagnosis because of multiple diagnoses. DSM-IV axis I disorders were clinically diagnosed by the clinicians who evaluated the subjects in treatment, blind to the axis II diagnosis. The low base rate of several axis I diagnoses prevented the analysis of the confounding effect of specific axis I disorders on DSM-IV PD covariation patterns. The relatively high base rate of anxiety disorder and eating disorder diagnoses, as well as the low base rate of mood disorders, observed in this sample could be explained by the presence in our hospital of 2 large divisions specializing in the treatment of anxiety and eating disorders, respectively.

All subjects were administered the Structured Clinical Interview for DSM-IV Axis II Personality Disorders, Version 2.0 (SCID-II).[20] The SCID-II is a 140-item (organized by diagnosis) semistructured interview designed to diagnose the 12 DSM-IV PDs. SCID-II was preceded by the administration of its self-report screening questionnaire. To increase diagnostic validity, all additional available sources of information (e.g., chart data, informant, treatment response, etc.) were used in this study. Subjects with axis I diagnoses were administered the SCID-II at acute symptom remission, according to the judgment of the treating clinician, by expert trained raters to avoid confounding effects of axis I disorders on axis II diagnoses.[21] In this study, with the exception of depressive PD categorical diagnosis (Cohen $\kappa = .68$), all other joint-interview interrater reliability coefficients (Cohen κ for categorical diagnosis and intraclass correlation coefficient for dimensional assessment) were more than .80 for categorical and dimensional PD diagnoses. This is consistent with previously published data.[22]

The bivariate association between categorically diagnosed DSM-IV PDs was assessed using the odds ratio (OR). An OR greater than 1.0 shows a positive association between 2 variables, whereas an OR less than 1.0 indicates negative association. In the case of independence, the OR equals 1.0. Whenever a cell in the contingency table had zero frequency, a 0.5 constant was added to avoid OR undefinition.[23] The Yates-corrected chi-square (χ^2) test was used to test the hypothesis that the ORs were significantly different from 1.0.[23]

Hierarchical log-linear models were used to identify the specific PD association patterns needed to adequately reproduce the matrix of PD observed frequency. The independence model was chosen as a baseline model. The following interactions were then entered in the model in successive steps: (1) 2-way interactions, including bivariate PD associations with P less than .0008 (i.e., Bonferroni-corrected nominal significance level, .05/66 = .0008); (2) 2-way interactions with P less than .01; (3) 2-way interactions with P less than .025; (4) 2-way interactions with P less than .05; and (5) 3- and 4-way interaction model, based on DSM-IV clusters (cluster A, B, and C). Considering the exploratory nature of these log-linear analyses, the model selection was based on the significance of the likelihood-ratio χ^2 statistic (G^2) difference between competing models and minimization of the Akaike information criterion (AIC) and Bayesian information criterion (BIC).[24] The goodness-of-fit of the best model was tested using the G^2 statistic. A nonsignificant G^2 value shows that the model adequately reproduced the observed frequencies.[24]

OR homogeneity across sample strata defined by subject gender and severity was tested. To find a balance between the low power of the homogeneity χ^2 test[23] and the excessive fluctuation of the significance level due to the large number of comparisons for each confounder, Bonferroni correction was applied to an extremely liberal nominal significance level ($P = .20/66 = .003$). The use of log-linear models to test OR homogeneity[24] was prevented by the excessive cell sparseness observed in several confounder strata.

Principal-component analysis (PCA) was used to identify the latent dimensions explaining the covariation of DSM-IV PDs. PCA was applied to the correlation (Pearson r) matrix of dimensionally assessed (i.e., number of traits) DSM-IV PDs. This procedure was justified by the almost perfect agreement observed between Pearson r and coefficient phi correlation matrices (i.e., the correlation matrices of dimensionally assessed and categorically scored DSM-IV PDs, median phi = −.04, median $r = -.05$; correlation between matrices, $r = .97$, $P < .001$, Wilcoxon matched-pairs test = −1.11, P (2-tailed) >.20). On the contrary, tetrachoric correlation coefficients (median $r = -.67$) clearly overestimated the size of DSM-IV PD correlations with regard to phi (Wilcoxon test = −4.88, P