ORIGINAL ARTICLE

Individual Growth Curve Analysis Illuminates Stability and Change in Personality Disorder Features

The Longitudinal Study of Personality Disorders

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Background: The long-term stability of personality pathology remains an open question. Its resolution will come from prospective, multiwave longitudinal studies using blinded assessments of personality disorders (PD). Informative analysis of multiwave data requires the application of statistical procedures, such as individual growth curve modeling, that can detect and describe individual change appropriately over time. The Longitudinal Study of Personality Disorders, which meets contemporary methodological design criteria, provides the data for this investigation of PD stability and change from an individual growth curve perspective.

Methods: Two hundred fifty subjects were examined for PD features at 3 different time points using the International Personality Disorders Examination during a 4-year study. Stability and change in PD features over time were examined using individual growth modeling.

Results: Fitting of unconditional growth models indicated that statistically significant variation in PD features existed across time in the elevation and rate of change of the individual PD growth trajectories. Fitting of additional conditional growth models, in which the individual elevation and rate-of-change growth parameters were predicted by subjects' study group membership (no PD vs possible PD), sex, and age at entry into the study, showed that study group membership predicted the elevation and rate of change of the individual growth curves. Comorbid Axis I psychopathology and treatment during the study period were related to elevations of the individual growth trajectories, but not to rates of change.

Conclusions: From the perspective of individual growth curve analysis, PD features show considerable variability across individuals over time. This fine-grained analysis of individual growth trajectories provides compelling evidence of change in PD features over time and does not support the assumption that PD features are traitlike, enduring, and stable over time.

Arch Gen Psychiatry. 2004;61:1015-1024

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PERSONALITY DISORDERS (PDs) are assumed to be stable over time. The prevailing diagnostic nomenclature (*DSM-IV*,[1] *DSM-III*,[2] and *DSM-R-III*[3]) for these prevalent disorders[4,5] embraces this assumption in asserting that PDs are "enduring patterns" of behavior that are "inflexible," and "stable over time."[1] However, data supporting this core assumption, drawn from properly designed longitudinal studies, are essentially nonexistent, and the long-term stability of PDs remains essentially undocumented terrain.

Initial studies of the stability of PDs generally used 2-wave test-retest research designs.[6] Such studies are typically conducted across short time spans and examine the stability of individual differences by examining correlation coefficients and comparing group averages of PD features at the 2 time points. However, the inadequacy of the test-retest design for illuminating stability or change has long been noted in the longitudinal research methodology literature.[7-10] Two waves of longitudinal data represent an extremely limited design for investigating change because (1) the amount of change between the first and second occasions of measurement cannot tell us anything about the shape of each person's individual growth trajectory between those times, and (2) estimates of true change are difficult to obtain from the observed 2-wave data.[8,10]

Alternatively, the methodological superiority of the prospective multiwave longitudinal design for the study of stability and change has long been well documented.[7-12] In addition to using a multiwave design, a proper longitudinal study involves the collection of data across sensibly spaced intervals, wherein the variable of interest is continuous in nature, can be equated across occasions of measurement, and remains construct valid for the entire study pe-

Table 1. Demographic Characteristics of the Longitudinal Study of Personality Disorders Sample (N = 250) for Subjects Available at 3 Waves of the Study

	No. (%) of Subjects	
	Father	Mother
Parents' education, y		
1-12	31 (12.4)	47 (18.8)
13-15	40 (16.0)	51 (20.4)
≥16	172 (68.8)	147 (58.8)
Not available	7 (2.8)	5 (2.0)
Parents' occupation		
Laborer/service	5 (2.0)	6 (2.4)
Operatives (machine)	7 (2.8)	3 (1.2)
Craftsman/foreman	8 (3.2)	4 (1.6)
Clerical/sales	10 (4.0)	42 (16.8)
Management/official	67 (26.8)	32 (12.8)
Professional/technical	131 (52.4)	105 (42.0)
Not available or homemaker	22 (8.8)	58 (23.2)

riod.[10] Moreover, for interview-based assessments, blinded assessment is a critical requirement such that the same subject is never seen by the same interviewer more than once to ensure against "halo effects," which diminish validity and elevate stability estimates artifactually.[13,14] Finally, the most informative extraction of meaning from longitudinal data for the investigation of stability and change takes the individual growth curve (or path) as the critical unit of analysis.[7,10,11]

The Longitudinal Study of Personality Disorders (LSPD),[4,15] begun in 1990, is a prospective multiwave longitudinal study of personality pathology, normal personality, and temperament sponsored by the National Institute of Mental Health, Washington, DC. A major goal of the LSPD is the life-span study of the stability of PD symptomatology. The LSPD subjects were drawn initially from a nonclinical population, thus avoiding the usual confounds attending the study of hospital/clinic PD cases (eg, treatment/time confounds, Berkson's bias, and selection of extreme cases). Herein, we describe an analysis of individual growth trajectories of PD features from the LSPD.[4,15] Our analyses transcend the previous LSPD[15] report by examining stability and change in a more informative manner and by illustrating the power, richness, and utility of the individual growth curve (IGC) approach (also known as hierarchical linear modeling,[16,17] multilevel modeling,[18] covariance components models,[19] or random-coefficient regression models[20]) for longitudinal data analysis in psychopathology.

In the previous LSPD report,[15] the stability of individual differences in the PDs over time was examined using the between-wave correlation approach, and individual differences were found to be relatively stable during the 4-year span. However, this approach provided only thin 2-wave snapshots of the entire spectrum of interindividual differences in growth. Average levels of PD symptomatology, assessed using repeated-measures multivariate analysis of variance (MANOVA),[15] provided some evidence of PD feature reduction over time (albeit with small effect sizes). However, the previous MANOVA approach (1) could not accommodate the existing unequal spacing of PD assessments across study subjects, (2) did not disentangle important aspects of individual change such as initial levels of symptomatology and rate of change as typically implemented, and (3) implausibly assumed comparable growth across all subjects, wherein heterogeneity of growth is more likely.[21] As such, although the previously reported correlational and MANOVA approaches represented a reasonable first pass through the LSPD[15] data, they did not tap the true richness therein. An analysis of individual growth trajectories[10,11] is required at this point to represent this richness and to improve our understanding of the stability or change in personality disorders over time.

METHODS

SUBJECTS

The 258 subjects in the LSPD[15] were drawn from a population consisting of 2000 first-year undergraduate students.[4] Subjects were assigned to a possible PD (PPD) group or a no PD (NPD) group according to the International Personality Disorder Examination *DSM-III-R*–Screen (IPDE-S)[4] (response rate, 84.2%). The PPD subjects met the diagnostic threshold for at least 1 specific *DSM-III-R* PD, whereas NPD subjects (1) did not meet the *DSM-III-R*–defined threshold for diagnosis and (2) had fewer than 10 PD features across all disorders. Extensive detail concerning the initial subject selection procedure and sampling is given elsewhere.[4] The 258 subjects consisted of 121 men (46.9%) and 137 women (53.1%). The PPD group included 134 (68 men and 66 women); the NPD group, 124 (53 men and 71 women). All subjects gave voluntary written informed consent and received an honorarium of $50 at each wave. Of the initial 258 subjects, 250 completed all 3 assessment waves and are included in this analysis. Five PPD and 3 NPD subjects did not complete all 3 waves, for a final sample of 64 men (49.6%) and 65 women (50.4%) in the PPD group and 53 men (43.8%) and 68 women (56.2%) in the NPD group. Race and ethnicity in the final sample were as follows: 9 (3.6%) African American; 12 (4.8%) Latin or Hispanic; 180 (72.0%) white; 43 (17.2%) Asian-Pacific Islander; 2 (0.8%) Native American; and 4 (1.6%) other. Mean age at study entry was 18.89 years (SD, 0.51 years). Additional sample characteristics are summarized in **Table 1**.

PD ASSESSMENTS

The LSPD has a prospective multiwave longitudinal design, with subjects initially undergoing evaluation at 3 time points (ie, first, second, and fourth years in college). Although not required for application of individual growth modeling,[10] the LSPD data are balanced, in that all subjects have 3 waves of data, and time structured, in that everyone undergoes repeated assessment on the same 3-wave schedule,[10] although the time between assessments varies from case to case. Interview assessments were conducted by experienced PhD or advanced MSW clinicians. Finally, as the LSPD is a naturalistic prospective study, subjects were free to seek psychological treatment of their own accord.

The IPDE-S is a 250-item self-administered true-false PD screening inventory developed by Armand W. Loranger, PhD. The diagnostic efficiency and psychometric properties of the IPDE-S in a 2-stage screen application were described previously.[4]

The IPDE is the well-known semistructured interview that assesses *DSM* and *ICD-10* PD features[22-24] and was used in the

World Health Organization/Alcohol, Drug Abuse, and Mental Health Administation–sponsored International Pilot Study of Personality Disorders.[24] The *DSM-III-R* criteria were assessed in this study. Clinically experienced interviewers received training in IPDE administration and scoring by Dr Loranger and were supervised throughout the project by one of us (M.F.L.) who was blind to the subjects' identity, putative PD status, and all previous assessment information. The interrater reliability for IPDE assessments was excellent at all 3 waves, ranging from 0.84 to 0.92 for all PD dimensions. The interviewers were blind to the putative PD group status of the subjects and to all prior LSPD PD assessment data, and subjects never underwent assessment by the same interviewer more than once.

The Structured Clinical Interview for *DSM-III-R*–Non-Patient Version[25] is a semistructured *DSM-III-R* Axis I clinical interview for use with nonpatients. The interview was administered first, followed by the IPDE.

STATISTICAL ANALYSIS

We used IGC analysis to investigate change in PD features over time. This method of analyzing within-subject change was popularized by Rogosa and colleagues[7,11] and represents the most powerful way to assess change in a continuous dimension over time within subjects.[10,11,16,17] The dependent variable used in these analyses was the number of PD features rated as present on the IPDE, which yielded continuous dimensional scores for the 11 *DSM-III-R* Axis II PDs, the 3 clusters (A, B, and C), and total PD features. Dimensional measures of PD ensured the greatest sensitivity to the investigation of stability and change (qualitative diagnoses would not be appropriate for a study of change in this framework).

The IGC approach hypothesizes that, for each individual, the continuous outcome variable (eg, the number of PD features) is a specified function of time, called the individual growth trajectory, plus error. This trajectory is often specified as a simple linear function of time, in which case it contains 2 important unknown individual growth parameters—an intercept and a slope—that determine the shape of individual true growth over time. The individual intercept parameter represents the net elevation of the trajectory over time, ie, the true mean level of the PD features for the individual at the onset of the study (or, alternatively, whenever the origin of the time scale has been defined). The individual slope parameter represents the rate of change over time and is the within-person rate of change in PD features over time in the present study. Despite the obvious theoretical importance of individual slope in studies of change, it has not been widely examined in research on psychopathology. Once an individual growth trajectory has been specified (at level 1) to represent the individual change over time, a level 2 model can be specified to describe the investigators' hypotheses about the way that the individual growth parameters contained in the level 1 model are related to between-subjects factors (eg, subject sex and diagnostic group).

The IGC approach has several advantages. First, interindividual variability in assessment intervals can be tolerated in specifying the individual trajectories, unlike repeated-measures MANOVA. Second, although estimation of an individual growth trajectory and its precision generally requires more than 2 observations on the individual over time, the IGC modeling approach is highly tolerant of missing data, permitting subjects who have incomplete data to participate in the analyses. Third, using modern software, the IGC modeling approach simultaneously uses data on all individuals at every time point to concurrently investigate within- and between-individual change, with concomitant improvements in precision and power. Fourth, when lengthy multiwave data are available, IGC modeling permits the flexible specification and rich investigation of nonlinear individual change over time.

In our analyses, the hypothesized levels 1 and 2 statistical models were fitted simultaneously to the LSPD data using full-maximum likelihood estimation and the HLM-5 computer program.[26] We conducted our analyses sequentially. First, we conducted a set of unconditional growth analyses[10] in which we posited a linear individual change trajectory at level 1, but did not attempt to predict interindividual variation in the growth parameters by between-subject factors. Such unconditional analyses are useful for partitioning the outcome variation into variance components that describe the net variation in slope and intercept across individuals. Second, we conducted a set of conditional analyses in which we examined systematic interindividual differences in intercept and slope as a function of 3 between-subject predictors, namely study group (PPD vs NPD), subject sex, and age in years at entry into the study. These predictors yield fixed effects in the prediction of the slope and intercept values retained from the level 1 analysis. The fitting of the level 2 model also yields estimates of residual variance that describe remaining interindividual variability in the individual slopes and intercepts (as well as their covariance) after accounting for the hypothesized fixed effects, giving rise to the variance components (ie, σ^2_0, σ^2_1, σ_{01}) (**Table 2**).

In addition, supplementary analyses were conducted to determine whether statistical interactions among the group, sex, and age at entry variables were required as predictors in the level 2 model or whether inclusion of additional level 2 predictors (ie, Axis I disorder and treatment) would substantially improve the model fit for a particular PD dimension. Improvement in model fit was assessed by comparison of deviance statistics. Fixed-effects and variance components were tested for statistical significance using the provided z statistics (2-tailed). Sample comparisons of simple proportions were performed using the χ^2 test.

RESULTS

CLINICAL CHARACTERISTICS OF THE SAMPLE

As reported previously,[15] the lifetime *DSM-III-R* Axis I diagnoses (**Table 3**) of the study subjects are for definite and probable disorders. Eighty-one (62.8%) of the PPD subjects received an Axis I diagnosis compared with 32 (26.4%) of the NPD subjects (χ^2_1=33.30; P<.001). Forty-one PPD subjects (31.8%) vs 21 NPD subjects (17.4%) reported a history of treatment by wave 3 (χ^2_1=6.97; P<.01). Finally, as of wave 3, 16% of the sample received a probable or definite diagnosis for at least 1 Axis II PD (or PD not otherwise specified).

ASSESSMENT SCHEDULE CHARACTERISTICS

The PD features of each of the 250 study subjects were assessed 3 times during the 4-year study period. The average age of study subjects at the assessment waves were 18.89 years (SD, 0.51 years) at wave 1, 19.83 years (SD, 0.54 years) at wave 2, and 21.70 years (SD, 0.56 years) at wave 3. The time between assessments for each subject was calculated in years, using each subject's date of birth and exact assessment date, and then centered on age at entry into the study for each study subject (with age at entry being included as a predictor at level 2). The mean time from entry into the study (wave 1) to wave 2

Table 2. Definition and Interpretation of Parameters in the Multilevel Model for Growth (Change)

Level 1 Model: The level 1 individual growth trajectory is assumed to be linear in time over the period of the study, as follows:

$Y_{ij} = \pi_{0i} + \pi_{1i}T_{ij} + \varepsilon_{ij}$ (equation 1: level 1),

where:

- Y_{ij} is the outcome score (eg, number of PD features) of individual *i* at time *j* ;
- T_{ij} is the time at which assessment *j* of subject *i* took place, measured in years and *centered* for each subject on each individual subject's age at entry into the study;
- π_{0i} is the *intercept* parameter or "elevation" of the hypothesized growth trajectory for individual *i* (that is, the *true initial status* of a subject on the PD feature variable Y at the beginning of the study);
- π_{1i} is the *slope* parameter for individual *i* (that is, the true rate of change [yearly] in the number of PD features over time);
- ε_{ij} is a level 1 residual, or the unexplained portion of the outcome, across all occasions of measurement, for individual *i* in the population (the net [vertical] scatter of the observed data around individual *L*' s hypothesized change trajectory). It is assumed to be normally distributed with mean zero and variance defined by σ^2_ε.

Level 2 Model: At level 2, each individual growth parameter from the level 1 model is predicted by important time-invariant characteristics of the individual (eg, study group, sex), such that:

$\pi_{0i} = \gamma_{00} + \gamma_{01}(X_i) + \zeta_{0i}$ (equation 2: level 2)

$\pi_{1i} = \gamma_{10} + \gamma_{11}(X_i) + \zeta_{1i}$,

where:

- γ_{00} is the population average of the level 1 intercepts, π_{0i}, for individuals with a level 2 predictor value of 0;
- γ_{01} is the population average difference in level 1 intercepts, π_{0i}, for a 1-unit difference in the level 2 predictor (*X*) (alternatively, the impact of predictor $[X_i]$ on initial status);
- γ_{10} is the population average of the level 1 slopes, π_{1i}, for individuals with a level 2 predictor value of 0;
- γ_{11} is the population average difference in the level 1 slope, π_{1i}, for a 1-unit difference in the level 2 predictor (alternatively, the impact of predictor $[X_i]$ on the individual rates of change);
- X_i is a generic level 2 time-invariant predictor, of which there may be several, such as subject study group (NPD vs PPD) or biological sex;
- ζ_{0i} and ζ_{1i} are the level 2 residuals that represent those portions of the level 2 outcomes that remain unexplained by the level 2 predictors and are assumed to be drawn from a bivariate normal distribution with mean vector 0 and unstructured error covariance matrix. Their variances are represented by σ^2_0 and σ^2_1, respectively, and their covariance as σ_{01}.

Abbreviations: NPD, no personality disorder (PD); PPD, possible PD.

was 0.95 years (SD, 0.14 years); from wave 1 to wave 3, 2.82 years (SD, 0.23 years). Centering the assessment intervals on age at entry and including age at entry as a predictor at level 2 accounts for each subject's unique chronological age when he or she began the study and causes the individual level 1 intercepts to represent the true value of the wave 1 assessments as the subjects' initial status.

IGC MODELING OF PD FEATURES

The IGC modeling analyses were performed separately for the outcome variables of total PD features, cluster PD features, and each individual *DSM* PD. The heterogeneity in the individual growth trajectories is impressive, and they are plotted, using an exploratory ordinary least squares approach, for total PD features in **Figure 1**.

Unconditional Analyses: Is Change Present in the Data?

First, an unconditional growth model (ie, containing no level 2 predictors) was fitted for all PD dimensions, providing estimates of the average elevation and rate-of-change parameters and their natural variation across all subjects on entry into the study. The results of these fits are given in **Table 4** and **Table 5**, which contain estimates of the fixed-effects and variance components for the unconditional growth trajectories for each of the various PD dimensions. The estimated average elevation of the individual growth trajectories on entry into the study (intercepts) differed significantly from 0 for all PD dimensions ($P<.001$; all large effects). In addition, each intercept contained significant variability (σ^2_0) ($P<.001$) available for prediction at level 2 in subsequent conditional models.

The estimated average rates of change also differed significantly from 0 for all of the PD dimensions, except for the paranoid PD dimension, indicating that much change over time was evident in the PD data (typically medium or large effects). In addition, all of the variance components associated with rate of change (σ^2_1), with the exception of cluster C disorders (compulsive, avoidant, passive-aggressive, and dependent PDs) and paranoid PD, were statistically significant, suggesting that there were significant amounts of variation in change that could potentially be predicted in subsequent level 2 models. Finally, the estimated slopes from the unconditional growth analyses support interesting conclusions with respect to the rate at which PDs actually change with time. Specifically, we estimate that total PD features decrease by 1.4 PD features with each passing year. At the level of clusters, annual rates of change over the study period were as follows: 0.35 cluster A features per year, 0.65 cluster B features per year, and 0.40 cluster C features per year.

Conditional Analyses

Next, we distinguished the individual growth trajectories by the subjects' group, sex, and age at study entry. We introduced level 2 predictors to explain any between-person variation in the individual elevation and rate-of-change parameters. The 2 primary between-subjects factors of interest were the group membership (PPD vs NPD) and subject sex. In addition, we included each subject's age at entry into the study as a predictor at level 2 to account for interindividual variation in change associated with actual age (ie, developmental level). For all 15 models fitted, intermodel comparisons of goodness-of-fit (deviance) statistics disclosed that the inclusion of group, sex, and age at study entry as level 2 predictors significantly improved the fit beyond that achieved in the unconditional growth models (schizoid PD, $P<.05$; all others, $P<.01$). The results of the conditional analyses are presented in **Table 6** and **Table 7**, which includes estimates of the fixed-effects and variance components associated with each level 2 predictor (study group, sex, and age at entry), the approximate *P* value for testing that these effects are 0 in the population, and an estimate of the effect size correlation coefficient (*r*).[27] Table 5 also

Table 3. Definite and Probable Lifetime *DSM-III-R* Axis I SCID-NP Diagnoses for Sample of 250 Subjects

Disorder	Subjects, No. (%)* NPD (n = 121)	PPD (n = 129)	χ^2	*P* Value†
Major depression	16 (13.2)	47 (36.4)	17.84	<.001
Bipolar/bipolar NOS	1 (0.8)	6 (4.7)	3.36	.07
Dysthymia (current only)	3 (2.5)	13 (10.1)	6.02	.01
Other affective disorder	10 (8.3)	32 (24.8)	12.22	<.001
Alcohol abuse	2 (1.7)	7 (5.4)	2.56	.11
Alcohol dependence	2 (1.7)	13 (10.1)	7.86	.005
Other drug abuse	3 (2.5)	3 (2.3)	0.006	.94
Other drug dependence	0	4 (3.1)	3.81	.05
Social phobia	6 (5.0)	22 (17.1)	9.18	.002
Simple phobia	8 (6.6)	15 (11.6)	1.88	.17
Panic	3 (2.5)	1 (0.8)	1.15	.28
Obsessive-compulsive	4 (3.3)	8 (6.2)	1.14	.28
Anorexia	4 (3.3)	4 (3.1)	0.01	.93
Bulimia	2 (1.7)	11 (8.5)	5.99	.01
Eating disorder NOS	0	1 (0.8)	0.94	.33
Any Axis I diagnosis	32 (26.4)	81 (62.8)	33.30	<.001

Abbreviations: NOS, not otherwise specified; NPD, no personality disorder (PD); PPD, possible PD; SCID-NP, Structured Clinical Interview for *DSM-III-R*–Nonpatient Version.

*Values are expressed as the number of cases and percentages per diagnostic category observed in the 2 subject groups. Cases consist of definite and probable lifetime Axis I diagnoses combined for the entire study period. Some subjects have more than 1 Axis I diagnosis.

†Significance based on Pearson χ^2_1 test (2-tailed).

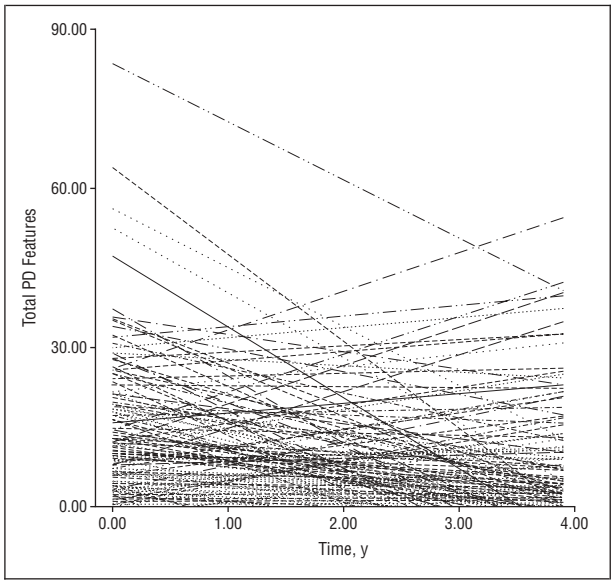

Figure 1. Ordinary least squares individual growth trajectories for total personality disorder (PD) features in 250 subjects during the study period. Time is reported in years since the beginning of the study and centered for each subject using the subject's age at entry into the study.

contains estimates of the variance components from the level 2 models, which were also tested for statistical significance.

With respect to elevation of the individual growth trajectories, group was a statistically significant predictor of individual elevation parameters in PD features for all PD dimensions across time (all $P<.001$, except schizotypal PD [$P<.002$] and schizoid PD [$P<.07$]), with effect sizes ranging from 0.12 to 0.44 (median, 0.30), indicating medium effects. Sex was less substantially predictive of elevation parameters; although statistically significant ($P\leq.05$) for 8 of 15 curves, most effect sizes were 0.18 or less (median, 0.14), indicating small effects. The PPD status corresponded to higher elevation for all PD dimensions, and male subjects displayed higher elevation, except for histrionic and dependent PD. Age at entry into the LSPD predicted little variation in PD growth curve elevations, except for narcissistic PD ($P<.09$). All of the variance components estimates for elevation (σ^2_0) indicated that there remained significant variation in elevation that could be modeled beyond the 3 predictors that we had selected.

The critical growth parameter for investigating stability and change in PD features over time is the individual slope parameter, as it directly indexes the rate and direction of individual change over time. In the level 2 prediction of slope, group membership was significantly predictive of the rate of change in PD features for total PD features; cluster A, B, and C dimensions; and the paranoid, borderline, narcissistic, histrionic, avoidant, obsessive-compulsive, and dependent PDs (all $P\leq.05$). For these PD dimensions, the median effect size was 0.26, indicating a medium-sized effect, and the direction of the fixed effects shows that PPD subjects were showing higher rates of change (ie, PD feature declines). Group status was less predictive of rate of change for schizoid ($P<.43$), schizotypal ($P<.06$), passive-aggressive ($P<.24$), and antisocial ($P<.10$) PDs. Inspection of the fixed effects for sex indicate that it was less predictive of slope and, therefore, essentially unrelated to change in PD features over time, attaining statistical significance only for narcissistic PD ($P<.03$). Overall, the effect sizes for sex in relation to the rate of change were quite small (median effect size, $r=0.07$). Age at entry into the LSPD was minimally associated with the rate-of-change parameters (Table 4). Overall, study group status was the factor most strongly associated at level 2 with rate of change in the PDs. For many PD dimensions, namely clusters A and B and schizoid, schizotypal, antisocial, borderline, narcissistic, and histrionic PD, the variance component estimates for rate of change (σ^2_1) indicated there was also additional significant variation in elevation that could still be modeled beyond the 3 predictors that we had selected.

Finally, in sensitivity analyses suggested by the arithmetic and distributional properties of the dependent variable (as a count of features), we refitted all of our unconditional and conditional models by replacing the existing outcome by its square root. This yielded a pattern of results completely consistent with those reported herein for the untransformed PD variables.

The change observed in PD features over the study period is clearly shown in fitted growth trajectories recovered from the IGC analysis for the total PD features index presented in **Figure 2**. In this figure, we plot fitted growth trajectories for prototypical members of each group and sex. Prototypical PPD subjects display marked change over time.

Table 4. Parameters of the Unconditional Growth (Baseline) Model of PD Feature Change From a Prospective Multiwave Longitudinal Perspective: Intercept and Slope (N = 250)*

	Elevation (Intercept) of Individual Trajectory (π_{0i})			Rate of Change (Slope) of Individual Trajectory (π_{1i})		
Disorder	Fixed Effect (γ_{00})	*P* Value	ES *r*†	Fixed Effect (γ_{10})	*P* Value	ES *r*†
Total PD	9.61	.001	0.64	−1.40	.001	0.38
Cluster A	2.08	.001	0.52	−0.35	.001	0.29
Cluster B	4.37	.001	0.56	−0.65	.001	0.33
Cluster C	3.16	.001	0.62	−0.40	.001	0.29
Paranoid	0.52	.001	0.40	−0.04	NS	0.08
Schizoid	0.51	.001	0.37	−0.07	.004	0.18
Schizotypal	1.05	.001	0.50	−0.24	.001	0.37
Antisocial	1.00	.001	0.41	0.11	.01	0.16
Borderline	0.97	.001	0.48	−0.14	.001	0.24
Narcissistic	1.21	.001	0.47	−0.30	.001	0.39
Histrionic	1.18	.001	0.52	−0.31	.001	0.44
Dependent	0.71	.001	0.47	−0.09	.001	0.22
Avoidant	0.70	.001	0.47	−0.09	.002	0.20
Passive-aggressive	0.76	.001	0.48	−0.09	.01	0.17
Compulsive	0.99	.001	0.55	−0.12	.003	0.19

Abbreviations: NS, not significant; PD, personality disorder.

*The unconditional level 1 model examines average "within-individual" change over time.[10] The fixed-effects and variance components parameters were tested to determine whether they differ from zero. Any *P* values greater than .10 are designated as NS.

†ES *r* indicates effect size *r*, which is interpreted as follows: 0.10, small effect; 0.24, medium effect; and 0.37, large effect.[27(p446)] Model estimation was performed with full-maximum likelihood using the HLM-5 program.[26]

PRESENCE OF POTENTIAL STATISTICAL INTERACTION EFFECTS IN THE LEVEL 2 MODEL

We also investigated whether statistical interactions among the level 2 predictors had any impact on the prediction of the PD outcome variables. In 1 set of supplementary IGC analyses, we included a group × sex interaction term as an additional predictor at level 2, along with the group, sex, and entry age main effects, and we refitted all of the hypothesized statistical models. For 14 of the 15 models fitted (ie, total PD, 3 clusters, and 11 PDs), inclusion of the group × sex interaction did not result in improved fit over and above corresponding models containing only the main effects for group, sex, and age at entry. For the antisocial PD outcome, however, inclusion of the group × sex interaction resulted in an improvement in model fit (χ^2_2=9.31; $P<.01$), and the interaction was predictive of elevation ($P<.003$) but not slope ($P=.29$). In addition, we refitted all hypothesized models and included age at entry × sex and age at entry × group interactions, none of which improved model fit over corresponding main effects models.

SUPPLEMENTARY ANALYSES INCLUDING OTHER LEVEL 2 PREDICTORS

We also considered whether other important level 2 variables might predict elevation and/or rate of change in the PD dimensions. One such hypothesized variable was the presence of any Axis I disorder for study subjects before or during the study period, as it is reasonable to suspect that stability of PD features could be influenced by an Axis I disorder in evidence before or during the study period. Indeed, 45.2% of the total sample had some form of lifetime (or current) Axis I disorder as of wave 3. In 12 of the 15 fitted PD models, including the presence of an Axis I disorder as a predictor at level 2 provided an improvement in model fit beyond the fit achieved with only the main effects of group, sex, and entry age ($P<.05$). However, the presence of an Axis I disorder was related significantly only to the elevation parameter values (for 9 PD dimensions) and not to any of the slope parameters (ie, change).

Another potential level 2 predictor of interest was the presence of some form of psychological/psychiatric treatment in subjects before or during the study period. In this sample, 24.8% of the subjects reported receiving treatment at some point during their lives, before or during the study. When included as a level 2 predictor, treatment exposure provided an improvement in model fit in 10 of 15 cases (all $P<.05$). The impact of treatment, however, was limited entirely to elevation (intercept) values in 7 of those 10 models ($P\leq.05$) (total PD, cluster A and B totals, and paranoid, schizotypal, borderline, and narcissistic PDs) and had no statistically significant relation to rate of change (ie, slope) for any of the 15 PD dimensions.

COMMENT

The current diagnostic nomenclature (*DSM-IV*) clearly asserts that PDs are "enduring patterns" of behavior that are "inflexible," "stable" over time, and "of long duration."[1] However, empirical data supporting such assertions are virtually nonexistent. The LSPD[4,17] was begun 14 years ago, in part to investigate this core *DSM* assumption. In a previous LSPD report,[15] it was concluded that "PD features display relatively high levels of individual difference stability and appreciable mean level

Table 5. Parameters of the Unconditional Growth (Baseline) Model of PD Feature Change From a Prospective Multiwave Longitudinal Perspective: Variance Components and Deviance Statistics (N = 250)*

Disorder	Variance Components								Deviance (–2 Log-Likelihood)
	σ^2_ε	*P* Value	σ^2_0	*P* Value	σ^2_1	*P* Value	σ_{01}	*P* Value	
Total PD	39.43	.001	107.16	.001	2.54	.054	–16.21	.001	5359.76
Cluster A	4.10	.001	8.84	.001	0.36	.012	–1.41	.001	3658.75
Cluster B	8.37	.001	36.20	.001	1.51	.001	–7.35	.001	4273.91
Cluster C	8.23	.001	10.81	.001	0.04	NS	–0.60	NS	4081.56
Paranoid	0.77	.001	0.86	.001	0.04	NS	–0.16	.001	2237.30
Schizoid	0.41	.001	1.40	.001	0.05	.002	–0.16	.001	2097.82
Schizotypal	1.35	.001	2.43	.001	0.10	.03	–0.50	.001	2717.62
Antisocial	1.29	.001	3.98	.001	0.16	.002	–0.28	.01	2975.97
Borderline	1.02	.001	2.50	.001	0.13	.001	–0.57	.001	2561.69
Narcissistic	1.26	.001	4.22	.001	0.29	.001	–1.11	.001	2769.15
Histrionic	0.96	.001	3.15	.001	0.24	.001	–0.87	.001	2552.02
Dependent	0.79	.001	1.29	.001	0.03	NS	–0.02	.001	2315.69
Avoidant	0.91	.001	1.07	.001	0.01	NS	–0.07	NS	2405.48
Passive-aggressive	1.25	.001	1.11	.001	0.01	NS	–0.04	NS	2609.54
Compulsive	1.33	.001	1.36	.001	0.06	NS	–0.22	.005	2649.39

Abbreviations: NS, not significant; PD, personality disorder.
*The variance components parameters and deviance statistics in this table correspond to the unconditional level 1 models reported in Table 4. The variance components parameters were tested to determine whether they differ from zero. Any *P* values greater than .10 are designated as NS. Deviance statistics are based on 6 estimated parameters. Model estimation was performed with full maximum likelihood using the HLM-5 program.[26]

stability, with some change occurring over time," although "the changes were relatively small." However, that analysis had limited resolving power for issues of stability and change, as it was inadequately sensitive to the heterogeneity of individual growth trajectories and the unique spacing of assessments for each subject in the LSPD. The present study, which used the more powerful IGC analysis approach, was able to characterize the nature and amount of change observed in PD dimensions in the LSPD subjects more precisely. Specifically, although a previous MANOVA-based analysis[15] hinted at some change in PD feature levels over time (with small effect sizes), it did not speak to the rate of change as reflected in the true growth (change) of each subject apart from their initial level of PD features. Our IGC analysis clarified that indeed there was considerable change noted in PD features over time. Group membership (PPD vs NPD) emerged as an important factor that predicted not only the elevation of the individual growth trajectories (at study entry) as a medium-sized effect, but also showed a comparable medium-sized effect in predicting the rates of individual change in PD features over time. Clear evidence of statistically significant individual change was observed for nearly all PD dimensions studied, and this change was typically and uniformly in the direction of decreasing PD features over time. In sum, PD features do not appear to be as inflexible and enduring as that suggested by the *DSM* criteria.[1-3]

What could account for this pattern of change in PD features over time? Variance components estimated in our level 2 analyses showed that additional variability in the PD rate-of-change parameters remains that could be predicted by factors beyond our hypothesized predictors of group, sex, and entry age. The presence of an Axis I disorder in a study subject or receipt of treatment before or during the study period had little relationship with rates of change for the PDs. Fortunately, the IGC modeling approach will allow for the inclusion of additional predictors (eg, personality and temperament) in future efforts to dissect individual change in PD features over time,[10,11,28] and such future work will be model guided.[29]

Several caveats should be considered with these data. The present sample is more homogenous in age, educational achievement, and social class than the US population at large, and it consists of young adults. All of these features may differentially affect the study results. We note adjustment to university life across the college years (particularly the freshman-year transition) may have played a role in the changes we observed.[15] Also, LSPD subjects were selected from a population of first-year university students that may have been censored for some of the most severely PD-affected individuals. However, 16% of the LSPD sample received a diagnosis of an Axis II condition by the end of the study period using the highly conservative IPDE and a rate that accords well with community studies,[5,30] and 45.2% had a lifetime (or current) Axis I disorder by the end of college. Unfortunately, no other multiwave longitudinal study of PDs includes proper methodological safeguards (eg, blinded assessments, no treatment/time confounds) with which to compare these results (compare Shea et al[31(p2037)]) Additional longitudinal studies of PDs using clinical samples are welcome.

William James[32] claimed: "by the age 30, the character has set like plaster, and will never soften again," which appears true for some aspects of normal personality, but not all.[33-36] However, the *DSM* criteria notwithstanding, clinical experience suggests that some PD features may diminish over time or be generally less stable, and our results support this impression. Continued life-span study of the LSPD subjects will allow us to specify the long-term change or stability for personality pathology. With planned waves 4 and 5 data collections, more complex

Table 6. Predicting Interindividual Differences in Change of PD by Group, Sex, and Age at Entry Into Study: Intercept and Slope (N = 250)*

	Elevation (Intercept) of Individual Trajectory (π_{0i})			Rate of Change (Slope) of Individual Trajectory (π_{1i})		
Disorder	**Fixed-effect Coefficient**	***P* Value**	**ES *r*†**	**Fixed-effect Coefficient**	***P* Value**	**ES *r*†**
Total PD						
Male	3.58	.006	0.18	−0.60	NS	0.09
PPD	10.05	.001	0.44	−2.14	.001	0.33
Age at entry	1.87	NS	0.07	−0.60	.10	0.11
Cluster A						
Male	1.15	.007	0.17	−0.10	NS	0.04
PPD	1.71	.001	0.25	−0.30	.05	0.13
Age at entry	0.11	NS	0.02	−0.15	NS	0.07
Cluster B						
Male	1.79	.02	0.15	−0.28	NS	0.08
PPD	5.08	.001	0.40	−1.20	.001	0.33
Age at entry	1.54	.08	0.11	−0.48	.02	0.15
Cluster C						
Male	0.64	NS	0.09	−0.21	NS	0.08
PPD	3.28	.001	0.41	−0.66	.001	0.26
Age at entry	0.21	NS	0.02	0.05	NS	0.02
Paranoid						
Male	0.05	NS	0.02	0.002	NS	0.00
PPD	0.70	.001	0.30	−0.12	.05	0.13
Age at entry	0.06	NS	0.02	−0.03	NS	0.03
Schizoid						
Male	0.41	.02	0.16	−0.03	NS	0.04
PPD	0.29	.07	0.12	−0.04	NS	0.05
Age at entry	0.04	NS	0.03	−0.07	.10	0.11
Schizotypal						
Male	0.68	.004	0.18	−0.07	NS	0.06
PPD	0.72	.002	0.20	−0.14	.06	0.12
Age at entry	0.004	NS	0.00	−0.06	NS	0.06
Antisocial						
Male	1.39	.001	0.32	−0.07	NS	0.05
PPD	1.07	.001	0.27	−0.14	.10	0.11
Age at entry	0.42	NS	0.10	−0.14	.06	0.12
Borderline						
Male	0.19	NS	0.06	−0.09	NS	0.08
PPD	1.33	.001	0.38	−0.33	.001	0.30
Age at entry	0.14	NS	0.04	−0.08	NS	0.08
Narcissistic						
Male	0.65	.02	0.16	−0.21	.03	0.14
PPD	1.56	.001	0.36	−0.46	.001	0.33
Age at entry	0.54	.09	0.11	−0.12	NS	0.10
Histrionic						
Male	−0.45	.06	0.12	0.08	NS	0.07
PPD	1.11	.001	0.29	−0.27	.001	0.21
Age at entry	0.45	NS	0.10	−0.13	NS	0.10
Dependent						
Male	−0.30	.06	0.12	0.03	NS	0.04
PPD	0.87	.001	0.33	−0.24	.001	0.29
Age at entry	0.06	NS	0.02	0.004	NS	0.01
Avoidant						
Male	0.25	NS	0.09	−0.07	NS	0.07
PPD	0.66	.001	0.26	−0.20	.001	0.21
Age at entry	−0.02	NS	0.01	−0.01	NS	0.00
Passive-aggressive						
Male	0.27	.10	0.10	−0.09	NS	0.08
PPD	0.78	.001	0.28	−0.08	NS	0.08
Age at entry	0.21	NS	0.07	0.04	NS	0.04
Compulsive						
Male	0.42	.03	0.14	−0.09	NS	0.07
PPD	0.97	.001	0.34	−0.14	.05	0.12
Age at entry	−0.04	NS	0.01	0.003	NS	0.00

Abbreviations: NS, not significant; PD, personality disorder; PPD, possible PD.

*The level 2 analysis detects variability in change across individuals and determines the relationship between predictors and the shape of each person's individual growth trajectory.[10] All components of the level 1 and level 2 models were estimated simultaneously. Sex is coded as 1 for male and 0 for female; group status is coded 1 for PPD and 0 for no PD. Tabled values represent the final estimates of the fixed effects with robust standard errors. The fixed-effects parameters were tested to determine whether they differ from zero. Any *P* values greater than .10 are designated as NS.

†ES *r* indicates effect size *r*, which is interpreted as follows: 0.10, small effect; 0.24, medium effect; and 0.37, large effect.[27(p446)]

Table 7. Predicting Interindividual Differences in Change of PD by Group, Sex, and Age at Entry Into Study: Variance Components and Deviance Statistics (N = 250)*

Disorder	Variance Components								Deviance (–2 Log-Likelihood)
	σ^2_ε	*P* Value	σ^2_0	*P* Value	σ^2_1	*P* Value	σ_{01}	*P* Value	
Total PD									
Male	39.01	.001	77.37	.001	1.40	NS	–10.12	.001	5295.91
PPD									
Age at entry									
Cluster A									
Male	4.11	.001	7.71	.001	0.33	.03	–1.23	.001	3629.74
PPD									
Age at entry									
Cluster B									
Male	8.35	.001	28.17	.001	1.09	.001	–5.51	.001	4219.43
PPD									
Age at entry									
Cluster C									
Male	8.03	.001	8.17	.001	0.01	NS	–0.13	NS	4034.01
PPD									
Age at entry									
Paranoid									
Male	0.77	.001	0.73	.001	0.03	NS	–0.14	.003	2210.52
PPD									
Age at entry									
Schizoid									
Male	0.41	.001	1.32	.001	0.05	.002	–0.15	.001	2084.71
PPD									
Age at entry									
Schizotypal									
Male	1.34	.001	2.17	.001	0.10	.04	–0.46	.001	2691.45
PPD									
Age at entry									
Antisocial									
Male	1.29	.001	3.10	.001	0.15	.003	–0.20	.04	2922.81
PPD									
Age at entry									
Borderline									
Male	1.02	.001	2.05	.001	0.10	.006	–0.46	.001	2520.95
PPD									
Age at entry									
Narcissistic									
Male	1.26	.001	3.41	.001	0.23	.001	–0.88	.001	2725.03
PPD									
Age at entry									
Histrionic									
Male	0.95	.001	2.78	.001	0.22	.001	–0.79	.001	2522.78
PPD									
Age at entry									
Dependent									
Male	0.78	.001	1.10	.001	0.02	NS	–0.15	.002	2285.00
PPD									
Age at entry									
Avoidant									
Male	0.89	.001	0.96	.001	0.003	NS	–0.04	NS	2384.35
PPD									
Age at entry									
Passive-aggressive									
Male	1.25	.001	0.92	.001	0.005	NS	–0.02	NS	2581.17
PPD									
Age at entry									
Compulsive									
Male	1.33	.001	1.06	.001	0.05	NS	–0.17	.002	2610.18
PPD									
Age at entry									

Abbreviations: NS, not significant; PD, personality disorder; PPD, possible PD.

*The variance components parameters and deviance statistics in this table correspond to the conditional models reported in Table 6. The variance components parameters were tested to determine whether they differ from zero. All components of the level 1 and level 2 models were estimated simultaneously. Any *P* values greater than .10 are designated as NS. Deviance statistics are based on 12 estimated parameters. Model estimation was performed with full maximum likelihood using the HLM-5 program.[26]

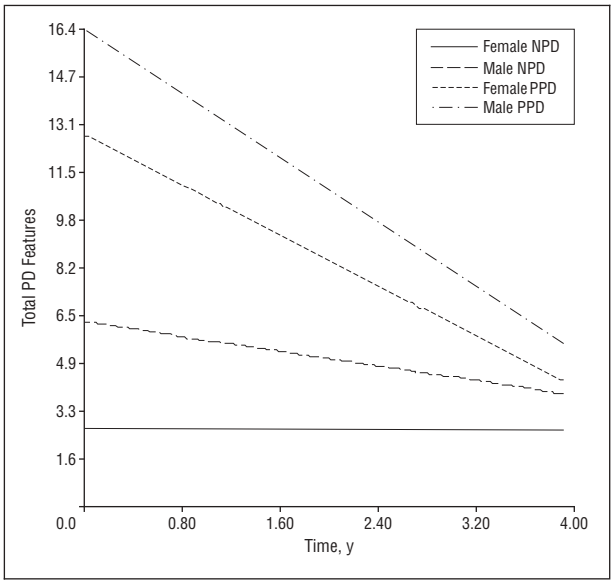

Figure 2. Empirical Bayes trajectories recovered from the hierarchical linear modeling analyses for total personality disorder (PD) features in 250 subjects, by study group and subject sex. Time is reported in years since the beginning of the study and centered for each subject using the subject's age at entry into the study. NPD indicates no PD; PPD, possible PD.

functions (eg, quadratic and cubic) for representing nonlinear individual change will become estimable.[10] We expect that additional waves of data will shed further light on this fascinating issue and should provide a more refined appreciation of the natural history of personality disorders.

Submitted for Publication: September 17, 2003; final revision received February 25, 2004; accepted March 16, 2004.
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Funding/Support: This study was supported in part by grant MH-45448 from the National Institute of Mental Health, Washington, DC (Dr Lenzenweger).
Acknowledgment: We thank Armand W. Loranger, PhD, for providing training and consultation on the use of the International Personality Disorder Examination and Lauren Korfine, PhD, for project coordination in the early phase of the study.

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