Ten-year course of borderline personality disorder: Psychopathology and Function from the Collaborative Longitudinal Personality Study

John G. Gunderson
Harvard Medical School, McLean Hospital

Robert L. Stout
Decision Science Institute/PIRE

Thomas H. McGlashan
Yale University School of Medicine

M. Tracie Shea
Department of Veteran Affairs and Brown University Medical School

Leslie C. Morey
Texas A & M University - College Station

Follow this and additional works at: https://wesscholar.wesleyan.edu/div3facpubs

Recommended Citation
Ten-Year Course of Borderline Personality Disorder

Psychopathology and Function From the Collaborative Longitudinal Personality Disorders Study

John G. Gunderson, MD; Robert L. Stout, PhD; Thomas H. McGlashan, MD; M. Tracie Shea, PhD; Leslie C. Morey, PhD; Carlos M. Grilo, PhD; Mary C. Zanarini, EdD; Shirley Yen, PhD; John C. Markowitz, MD; Charles Sanislow, PhD; Emily Ansell, PhD; Anthony Pinto, PhD; Andrew E. Skodol, MD

Context: Borderline personality disorder (BPD) is traditionally considered chronic and intractable.

Objective: To compare the course of BPD's psychopathology and social function with that of other personality disorders and with major depressive disorder (MDD) over 10 years.

Design: A collaborative study of treatment-seeking, 18- to 45-year-old patients followed up with standardized, reliable, and repeated measures of diagnostic remission and relapse and of both global social functioning and subtypes of social functioning.

Setting: Nineteen clinical settings (hospital and outpatient) in 4 northeastern US cities.

Participants: Three study groups, including 175 patients with BPD, 312 with cluster C personality disorders, and 95 with MDD but no personality disorder.

Main Outcome Measures: The Diagnostic Interview for DSM-IV Personality Disorders and its follow-along version (the Diagnostic Interview for DSM-IV Personality Disorders–Follow-Along Version) were used to diagnose personality disorders and assess changes in them. The Structured Clinical Interview for DSM-IV Axis I Disorders and the Longitudinal Interval Follow-up Evaluation were used to diagnose MDD and assess changes in MDD and in social function.

Results: Eighty-five percent of patients with BPD remitted. Remission of BPD was slower than for MDD ($P < .001$) and minimally slower than for other personality disorders ($P = .03$). Twelve percent of patients with BPD relapsed, a rate less frequent and slower than for patients with MDD ($P < .001$) and other personality disorders ($P = .008$). All BPD criteria declined at similar rates. Social function scores showed severe impairment with only modest albeit statistically significant improvement; patients with BPD remained persistently more dysfunctional than the other 2 groups ($P < .001$). Reductions in criteria predicted subsequent improvements in DSM-IV Axis V Global Assessment of Functioning scores ($P < .001$).

Conclusions: The 10-year course of BPD is characterized by high rates of remission, low rates of relapse, and severe and persistent impairment in social functioning. These results inform expectations of patients, families, and clinicians and document the severe public health burden of this disorder.

the perspective of the study’s full 10-year follow-up and by concurrently examining both changes in psychopathology (remission and relapse) and social functioning. Psychopathology is the primary focus of clinical interventions, whereas the associated social dysfunction, via direct costs and effects on others, is the primary public health concern. By examining both, this report allows us to examine how these 2 domains interact.

METHODS

DESIGN

The CLPS is a multisite, naturalistic, repeated-measures, longitudinal study of individuals with 4 personality disorders, BPD, schizotypal personality disorder, avoidant personality disorder (AVPD), and obsessive-compulsive personality disorder (OCPD), and a comparison group of patients with major depressive disorder (MDD) without personality disorder.

SAMPLE

The CLPS was approved by the institutional review boards at all participating sites. All patients gave written informed consent after procedures were fully explained. Each of the 4 sites (Brown University, Columbia University, Harvard University, and Yale University) recruited consecutive eligible patients from multiple clinical subsites (N=19 subsites). The resulting samples were most frequently ascertained from psychiatric outpatient clinics (43%) and from psychiatric hospitals (12%). All patients were aged 18 to 45 years, an age range that would best generalize to clinical samples and would allow follow-up through the most relevant stage of life. Our personality disorder samples were identified by semistructured interview (Diagnostic Interview for DSM-IV Personality Disorders, see later) with confirmation of the diagnosis (cell assignment) from self-report measures or by independent clinical judgment. Because the cell-assigned diagnoses are informed by clinical judgment and ensure that our samples are mutually exclusive, this narrower definition of our personality disorder samples was used. While the CLPS also includes subjects with schizotypal personality disorder, that diagnostic group was excluded from this report because their follow-up data involved ratings from in vivo observations that were progressively more difficult to obtain as more assessments were conducted via telephone. Their omission increased the homogeneity of the comparison group with other personality disorders (OPD) by combining the 2 cluster C personality disorders, ie, AVPD and OCPD. Thus, the study’s 3 study groups were patients with BPD (n=175), patients with cluster C OPD (n=312, including 158 with AVPD and 154 with OCPD), and patients with MDD (n=95). Our MDD study group was notable for having been selected to exclude comorbid personality disorder. The 3 diagnostic study groups shared similar age and socioeconomic status; there were, however, more women (79%) in the BPD group than in the OPD (64%) or MDD (60%) cells (P=.01).

Sixty-six percent of the patients who entered the study completed the full 10 years of follow-up. This report includes 111 patients with BPD (63% of those with BPD who entered), 211 with OPD (including 97 with AVPD [61%] and 114 with OCPD [74%]), and 62 with MDD (65%). Differences in attrition by study cell, age, and sex were not statistically significant.

ANALYSES

Cumulative Kaplan-Meier survival analyses assessed rates of remission and relapse with a Wilcoxon χ² test for group equality. Remission was defined as meeting 2 or fewer criteria for BPD. In comparing BPD rates of remission with those of OPD, we used 12-month durations at 2 or fewer criteria for greater clinical significance, whereas in comparing rates of remission of the BPD study group with those of MDD, we used what has become the MDD standard (a Psychiatric Status Rating ≤2, reflecting minimal or no symptoms) of a 2-month duration. Remission from OPD was defined as remaining at 2 or fewer AVPD criteria for patients in the AVPD cell and remaining at 2 or fewer OCPD criteria for those in the OCPD cell. Relapse for BPD was defined as returning to 5 or more criteria (the DSM-IV threshold) for 2 or more months after having remitted. For OPD, relapse was defined as returning to 4 or more criteria (the DSM-IV thresholds) for 2 or more months for AVPD and OCPD cells separately.

Point prevalence analyses were used to assess changes in mean scores for number of BPD criteria and for each individual BPD criterion, for GAF and GSA scores, and for scores on 6 LIFE subscales (and their total). This examination offers an alternative way to document change that is perhaps more clinically recognizable than survival analyses. To characterize individual patterns of improvement, using only those participants who provided at least 5 years of data, we analyzed individual change in GAF scores across follow-up. First, we tabulated how many participants improved their baseline GAF scores by at least 10 points at some time during follow-up as well as...
the amount of improvement. We then calculated how many consecutive years these persons stayed at a GAF score no more than 5 points worse than their best GAF score. Finally, we noted the lowest postpeak GAF score. These analyses together depict the maximum amount of improvement and how long that improvement was sustained. We contrasted the BPD and OPD groups on these measures using t tests for continuous measures and χ² tests for dichotomous measures.

Hierarchical linear modeling (HLM) analyses were used to test for between-group differences in functioning for the GAF score, the GSA score, and the continuous measures listed in Table 1. The HLM analyses included main effects for BPD vs OPD vs MDD, a term for linear change over time, and interaction terms for time × BPD vs OPD and time × BPD vs MDD. For more detailed examination of dichotomized variables over time, ie, employment (full time vs not) and marital status (married or cohabiting vs not), generalized estimating equation analyses with a logistic link function were used. Both the HLM and generalized estimating equation analyses covaried for age, education, and sex. In all of these analyses, we used multiple imputation to accommodate missing data. For each separate dependent variable, 23 imputed samples were generated using PROC MI in SAS version 9.2 statistical software (SAS Institute, Inc, Cary, North Carolina); results were aggregated across imputations using PROC MIANALYZE. It should be noted that the effective df for tests aggregated by multiple imputation are computed as a function of the actual sample size and missingness; thus, estimated df will vary from test to test. Kaplan-Meier survival analyses examined changes over time on the subgroups whose level of function was considered good based on GAF scores higher than 70.

Lagged HLM analyses with number of BPD criteria and GAF score as time-varying predictors were used to test our hypotheses regarding which predictors would predict subsequent (the next year’s) scores in the other domain. Thus, in 1 analysis, year 2 BPD criteria were used to predict year 3 GAF scores, year 3 BPD criteria were used to predict year 4 GAF scores, and so on. In a separate analysis, the roles of GAF score and BPD criteria were reversed. These analyses also included tests for age, sex, and education as covariates, a main effect for study year, and a year × time-varying predictor interaction. These analyses used multiple imputations for missing data as described earlier.

**RESULTS**

Figure 1A shows that the cumulative rates of remission for BPD over 10 years were 91% (95% confidence interval [CI], 86-96) using the 2-month definition of remission and 85% (95% CI, 78-91) using the 12-month definition, with the greatest rate of change occurring in the earlier years. While the overall rates of remission at 10 years were high for all 3 diagnostic study groups, the time to remission for BPD was significantly longer than for MDD (χ² = 73.91; P < .001) (using the 2-month standard for MDD) but only minimally longer than for cluster C OPD (χ² = 4.90; P = .03) (using the 12-month definition).

Figure 1B shows the cumulative relapse rates for patients with BPD who had remitted using both the 2- and 12-month definitions and how these compare with the MDD and OPD cells. The 10-year relapse rate for BPD was 11% (95% CI, 4-17) for the more clinically significant 12-month definition of remission—a rate that rose to 21% (95% CI, 13-29) using the 2-month definition. Relapses largely occurred in the first 4 years before leveling off. Using the 12-month definition of remission, the relapse rate for the OPD study group at 10 years was 25% (95% CI, 18-31), significantly higher than for BPD (χ² = 7.00; P = .008). The relapse rate for the MDD study group, using a 2-month definition, was significantly higher: 67% (95% CI, 57-78) in the MDD group relapsed by 10 years compared with 21% (95% CI, 13-29) for BPD (χ² = 44.74; P < .001).

The mean number of criteria met for BPD decreased from 6.7 to 4.3 in the first year and thereafter steadily decreased at a rate of 0.29 criteria per year to a low of 1.7 at 10 years. Only 9% of the patients with BPD remained stably disordered (≥5 criteria) at 10 years. As Figure 2 illustrates, the rates of decline for each of the 9 DSM-IV BPD criteria were similar, with those that were most prevalent at baseline remaining most prevalent after 10 years.

Figure 3A shows change in GAF scores over time. The clinically modest levels of functional improvement for BPD (mean GAF scores increased from 53 to 57), OPD (mean GAF scores increased from 62 to 64), and MDD (mean GAF scores increased from 61 to 69) were each, nonetheless, statistically significant over time (F₁,₄₀ = 26.36; P < .001). Across follow-up, 66% of subjects with BPD and 53% of subjects with OPD had at least 1 year when their GAF score was at least 10 points better than at intake. This difference is statistically significant (χ² = 6.32; P = .01). Of those who improved 10 points or more, the mean (SE) improvement was 12.21 (0.54) points and the mean (SE) number of years of sustained improvement was 2.00 (0.05) years; these measures did not differ between BPD and OPD. Improvements typically were not sustained. The worst GAF score following the best year was a mean (SE) of 16.45 (0.57) points lower; the size of decrement in GAF score did not differ for BPD vs OPD.

An HLM examination of the averaged mean GAF scores over time covaried for age, education, and sex showed that the averaged GAF score for BPD (GAF score 56) was significantly worse than for OPD (GAF score 62) (b = −1.14; t₁₄₇ = −2.29; P = .02), although the difference narrowed over time (b = 0.51; t₁₄₇ = 3.21; P = .002). A similar pattern holds when BPD was compared with MDD (GAF score 65) (b = −3.80; t₁₄₇ = −5.77; P < .001), although with a smaller change in the difference over time (b = −0.43; t₁₄₇ = −2.01; P = .04). Significant covariates were age (b = −0.27; t₁₄₇ = −6.53; P < .001) and education (b = 1.67; t₁₄₇ = 8.76; P < .001). We next looked at subgroups with good (GAF score > 70), fair (GAF score 61-70), and poor (GAF score < 61) functioning. The fractions of both subjects with OPD and subjects with MDD who scored either good or poor uniformly ranged between 20% and 40%. A much higher fraction of the subjects with BPD rated poor (range, 61%-81%; mean, 69%) and a much lower percentage rated good (range, 3%-14%; mean, 9%). A more focused examination of the attainment of good functioning (GAF score > 70) by survival analysis showed that at baseline no subjects of the BPD sample had good functioning and that by 10 years only 21% achieved this (Figure 4). This fraction for the BPD sample was much lower than the frequency of good functioning attained in either the OPD sample (48%) or MDD sample (61%) (χ² = 19.54; P < .001).
The LIFE ratings of GSA scores (Figure 3B) fell uniformly in the range from poor to mild impairment. The GSA scores mirrored the relatively low levels of change found with the GAF, although again all 3 diagnostic cells showed statistically significant improvement ($b=0.474; t_{401}=8.43; P<.001$). An HLM examination covarying for age, sex, and

### Table 1. Social Functioning as Measured by Longitudinal Interval Follow-up Evaluation Subscales

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Baseline</th>
<th>2 y</th>
<th>4 y</th>
<th>6 y</th>
<th>8 y</th>
<th>10 y</th>
<th>Significant Covariates</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Employment</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BPD</td>
<td>2.82 (2.18)</td>
<td>2.34 (1.40)</td>
<td>2.28 (1.27)</td>
<td>2.25 (1.30)</td>
<td>2.04 (1.24)</td>
<td>2.13 (1.13)</td>
<td>Age (+), education (-)</td>
</tr>
<tr>
<td>No.</td>
<td>145</td>
<td>99</td>
<td>100</td>
<td>84</td>
<td>75</td>
<td>64</td>
<td></td>
</tr>
<tr>
<td>OPD</td>
<td>2.05 (1.13)</td>
<td>1.93 (1.07)</td>
<td>1.99 (1.07)</td>
<td>1.92 (1.06)</td>
<td>1.91 (1.01)</td>
<td>1.84 (0.93)</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>216</td>
<td>216</td>
<td>206</td>
<td>184</td>
<td>172</td>
<td>151</td>
<td></td>
</tr>
<tr>
<td>MDD</td>
<td>2.14 (1.18)</td>
<td>1.70 (0.96)</td>
<td>1.86 (1.13)</td>
<td>1.69 (0.79)</td>
<td>1.76 (0.80)</td>
<td>2.05 (0.94)</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>78</td>
<td>63</td>
<td>63</td>
<td>61</td>
<td>54</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td><strong>Spouse role</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BPD</td>
<td>3.20 (1.29)</td>
<td>2.51 (1.29)</td>
<td>2.29 (1.15)</td>
<td>2.13 (1.21)</td>
<td>2.26 (1.10)</td>
<td>2.24 (1.16)</td>
<td>Age (+), education (-)</td>
</tr>
<tr>
<td>No.</td>
<td>87</td>
<td>45</td>
<td>48</td>
<td>52</td>
<td>46</td>
<td>49</td>
<td></td>
</tr>
<tr>
<td>OPD</td>
<td>2.58 (1.21)</td>
<td>2.12 (1.00)</td>
<td>2.16 (1.07)</td>
<td>2.00 (1.12)</td>
<td>2.11 (1.03)</td>
<td>2.17 (1.01)</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>158</td>
<td>92</td>
<td>92</td>
<td>101</td>
<td>102</td>
<td>101</td>
<td></td>
</tr>
<tr>
<td>MDD</td>
<td>3.03 (1.38)</td>
<td>1.92 (1.15)</td>
<td>1.93 (0.81)</td>
<td>1.83 (0.87)</td>
<td>1.88 (1.07)</td>
<td>1.86 (0.97)</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>34</td>
<td>25</td>
<td>28</td>
<td>24</td>
<td>26</td>
<td>28</td>
<td></td>
</tr>
<tr>
<td><strong>Parent role</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BPD</td>
<td>3.13 (1.31)</td>
<td>2.59 (1.11)</td>
<td>2.40 (1.00)</td>
<td>2.33 (1.07)</td>
<td>2.35 (1.08)</td>
<td>2.48 (1.15)</td>
<td>Age (+), education (-)</td>
</tr>
<tr>
<td>No.</td>
<td>166</td>
<td>144</td>
<td>134</td>
<td>123</td>
<td>107</td>
<td>93</td>
<td></td>
</tr>
<tr>
<td>OPD</td>
<td>2.55 (1.18)</td>
<td>2.33 (0.99)</td>
<td>2.30 (1.01)</td>
<td>2.09 (0.98)</td>
<td>2.17 (1.01)</td>
<td>2.13 (0.96)</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>291</td>
<td>266</td>
<td>252</td>
<td>230</td>
<td>203</td>
<td>184</td>
<td></td>
</tr>
<tr>
<td>MDD</td>
<td>2.26 (1.06)</td>
<td>2.20 (1.05)</td>
<td>2.13 (0.97)</td>
<td>2.10 (1.01)</td>
<td>1.93 (1.01)</td>
<td>1.90 (0.93)</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>91</td>
<td>82</td>
<td>78</td>
<td>70</td>
<td>58</td>
<td>52</td>
<td></td>
</tr>
<tr>
<td><strong>Friend role</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BPD</td>
<td>3.17 (1.22)</td>
<td>2.62 (1.12)</td>
<td>2.56 (1.20)</td>
<td>2.53 (1.26)</td>
<td>2.53 (1.18)</td>
<td>2.69 (1.16)</td>
<td>Age (+), education (-)</td>
</tr>
<tr>
<td>No.</td>
<td>175</td>
<td>157</td>
<td>146</td>
<td>139</td>
<td>124</td>
<td>111</td>
<td></td>
</tr>
<tr>
<td>OPD</td>
<td>2.79 (1.24)</td>
<td>2.46 (1.13)</td>
<td>2.44 (1.12)</td>
<td>2.34 (1.15)</td>
<td>2.42 (1.23)</td>
<td>2.61 (1.25)</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>312</td>
<td>287</td>
<td>277</td>
<td>259</td>
<td>233</td>
<td>211</td>
<td></td>
</tr>
<tr>
<td>MDD</td>
<td>2.44 (1.03)</td>
<td>1.80 (0.87)</td>
<td>1.95 (1.02)</td>
<td>2.00 (0.94)</td>
<td>2.03 (0.98)</td>
<td>2.29 (1.11)</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>95</td>
<td>87</td>
<td>84</td>
<td>78</td>
<td>68</td>
<td>62</td>
<td></td>
</tr>
<tr>
<td><strong>Recreation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BPD</td>
<td>3.46 (1.18)</td>
<td>2.76 (1.29)</td>
<td>2.73 (1.21)</td>
<td>2.87 (1.17)</td>
<td>2.60 (1.04)</td>
<td>2.60 (1.08)</td>
<td>Age (+), education (-)</td>
</tr>
<tr>
<td>No.</td>
<td>175</td>
<td>156</td>
<td>146</td>
<td>139</td>
<td>124</td>
<td>111</td>
<td></td>
</tr>
<tr>
<td>OPD</td>
<td>2.72 (1.18)</td>
<td>2.38 (1.11)</td>
<td>2.52 (1.12)</td>
<td>2.46 (1.08)</td>
<td>2.42 (1.10)</td>
<td>2.48 (1.10)</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>312</td>
<td>287</td>
<td>277</td>
<td>259</td>
<td>233</td>
<td>211</td>
<td></td>
</tr>
<tr>
<td>MDD</td>
<td>2.94 (1.07)</td>
<td>2.07 (1.09)</td>
<td>2.18 (1.10)</td>
<td>2.08 (0.92)</td>
<td>2.15 (0.98)</td>
<td>2.34 (1.13)</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>95</td>
<td>67</td>
<td>84</td>
<td>78</td>
<td>68</td>
<td>62</td>
<td></td>
</tr>
<tr>
<td><strong>Satisfaction</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BPD</td>
<td>3.57 (0.95)</td>
<td>3.01 (1.10)</td>
<td>2.91 (1.12)</td>
<td>2.90 (1.16)</td>
<td>...</td>
<td>...</td>
<td>Age (+), education (-)</td>
</tr>
<tr>
<td>No.</td>
<td>175</td>
<td>157</td>
<td>146</td>
<td>139</td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>OPD</td>
<td>3.06 (0.89)</td>
<td>2.65 (0.99)</td>
<td>2.64 (1.01)</td>
<td>2.58 (1.08)</td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>312</td>
<td>286</td>
<td>277</td>
<td>257</td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>MDD</td>
<td>3.23 (0.84)</td>
<td>2.35 (0.90)</td>
<td>2.39 (1.05)</td>
<td>2.27 (1.03)</td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>95</td>
<td>86</td>
<td>84</td>
<td>78</td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviations:** BPD, borderline personality disorder; MDD, major depressive disorder; OPD, other personality disorders; ellipses, not applicable.

*Reported means use nonmissing data; hierarchical linear modeling analyses were based on multiple imputation. Scores were measured on a scale of 1 to 5, with 1 indicating no impairment; 2, satisfactory or good; 3, mild or fair; 4, moderate or poor; and 5, severe or very poor. All 3 diagnostic cells improved significantly on every measure of social impairment, with $P<.001$.

*For covariates, (+) indicates that higher covariate scores go with higher (worse) scale scores; (-) indicates the converse.

*Data were not gathered at years 8 and 10.
education showed that the averaged GSA score over time for BPD (mean GSA score 3.42, poor to fair) was initially worse than for OPD, although the difference decreased over time ($b = -0.045; t_{237} = -3.14; P = .002$) (mean GSA score 3.06, fair), and for MDD (mean GSA score 2.83, fair to mild) ($b = 0.245; t_{449} = 4.89; P < .001$). Significant covariates were age (older age predictive of poorer functioning, $P < .001$) and education (more education predictive of better functioning, $P < .001$).

The LIFE functioning changes over time are shown in Table 1. The HLM analyses using multiple imputation for missing data are reported in Table 2. In these analyses, overall improvement from baseline to follow-up was observed for all subscales ($P < .001$). The differences between BPD and OPD at baseline diminished over time, and even though the mean score for BPD was higher (worse), there were no statistically significant main effects for BPD vs OPD. For the satisfaction and recre-
generalized estimating equation analyses indicate that ... in a repeated-
quent GAF scores was confirmed in a repeated-
ent at each assessment would inversely predict subse-
lihood of being married or cohabiting.

More education, but not age or sex, predicted greater like-
significantly differ from either the OPD or the MDD samples.
rated our finding that the mean level of the BPD sam-
other 2 groups at any period. These analyses corrobo-
P
were the other 2 samples (BPD vs OPD:

significant) for each of the 9 borderline personality disorder criteria on the

Diagnostic Interview for DSM-IV-Personality Disorders, assessed for the 2
years prior to the follow-up point.

Our hypothesis that the number of BPD criteria present
at each assessment would inversely predict subse-
quent GAF scores was confirmed in a repeated-
measure HLM with BPD criteria as a time-varying
predictor (t_{135} = -3.10; P = .002); each additional crite-
ron predicted a decrease of 0.47 point on the following
year’s GAF. There was an interaction between study year
and number of criteria. Notably, the number of BPD cri-
teria in early years predicted subsequent GAF scores less
well than in subsequent years. Age and education also
significantly predicted GAF scores (P < .001 for both):
every 10 years of added age predicted a decrease of 3.22
GAF points, whereas every additional year of education
predicted an increase of 1.71 GAF points.

A parallel HLM analysis with GAF score as the time-
varying predictor, GAF scores did not predict number
of BPD criteria for the next year (t_{109} = -1.01; P = .31).
There was no year × GAF score interaction. Education was a sig-
nificant covariate (t_{170} = -5.39; P < .001), but age and sex
were not. As noted earlier (Figure 1), there was a sig-
nificant decline over time in number of BPD criteria
(t_{295} = -10.06; P < .001).

This report is written at a time when, despite the high
prevalence of BPD in psychiatric facilities, attention to
BPD remains woefully low relative to that paid to other
major psychiatric disorders. Indeed the diagnosis is un-
derused\textsuperscript{22,23} and most mental health care professionals
avoid or actively dislike patients with BPD.\textsuperscript{24} This con-
text helps frame the significance of this study. Its results
Correlate with those of the only other 10-year prospective
study of BPD\textsuperscript{4,5} to demonstrate that BPD psychopa-
therapy improves more than generally expected but that
psychosocial functioning often remains impaired.

The remission rates found for BPD, very similar to those
found in the MSAD\textsuperscript{4,5} exceed what might have been pre-
dicted from usual clinical assumptions as well as from
prior long-term retrospective studies.\textsuperscript{26-28} Notably, this
pattern of remission, occurring in the absence of sus-
tained or BPD-specific treatments,\textsuperscript{29-31} is consistent with
the theory that if patients with BPD can achieve stable
supports and avoid interpersonal stressors they will re-
mit clinically.\textsuperscript{32,33} The relapse data, again mirroring what
was found in the MSAD follow-up,\textsuperscript{4,5} are equally strik-
ing. Only 11% of those who remitted subsequently re-
 lamps. The low relapse rate suggests that during the
remission process, the patients changed either psycholog-
ically, perhaps having acquired more resiliency or new
adaptive skills, or situationally by attaining more sup-
ports or less stress.

The rates of BPD remission found here resemble those
observed in 10-year follow-up studies that used similar
follow-up methods for MDD,\textsuperscript{35} bipolar disorder,\textsuperscript{36} and
panic disorders\textsuperscript{37} but far exceed those for social pho-
bia.\textsuperscript{38} The rates of BPD relapse found here are dramati-
ually lower than for all of these disorders.\textsuperscript{35-38} These com-
parisons underscore the clinically significant and distinct
BPD pattern in which BPD remitted significantly more
slowly than MDD but only minimally more slowly than
OPD and relapsed significantly less often than MDD and
OPD. Insofar as 80% of our BPD sample had lifetime
MDD,\textsuperscript{39} the dramatically faster rate to remission of our

![Figure 2. Prevalence of borderline personality disorder criteria. Positive indicates the cases with a score of 2 (definitely present and clinically significant) for each of the 9 borderline personality disorder criteria on the Diagnostic Interview for DSM-IV-Personality Disorders, assessed for the 2 years prior to the follow-up point.](image-url)
MDD sample (80% by 1 year) compared with BPD (30% by 1 year) underscores how negatively BPD influences the course of MDD. Similarly, the fact that the rate of relapse found in our MDD sample was lower than in other MDD samples presumably reflects our sample’s lack of personality disorder comorbidity. What is evident appears clinically counterintuitive: patients with BPD improve symptomatically more often, more quickly, and more dramatically than expected and, once better, maintain improvements more enduring than for many other major psychiatric disorders.

The relative stability of BPD criteria reported here extends our prior reports after 2 years of follow-up. The earlier reports from CLPS, like the 10-year data from the MSAD, suggested a hybrid model with more stable criteria being traitlike (eg, affective instability, unstable relationships) and with less stable criteria being more symptomlike or statelike (eg, self-injurious behavior, stress/paranoia). In contrast, these 10-year data failed to confirm this division: all 9 criteria had similar rates and levels (about 50%) of decline with a similar rank ordering of prevalence at all times. Our finding is clinically instructive: criteria that we had previously predicted would remain intransigently stable traits proved just as likely to diminish over time as those that we expected would prove more episodic and transient. This finding also is notable for failing to show that any of BPD’s 3 major phenotypes, ie, affective, behavioral, or interpersonal, show a distinctive pattern of stability. This perhaps affirms the overriding single-factor unity of the BPD construct.

Figure 3. Scores on the Global Assessment of Functioning (GAF) (A) and the Global Social Adjustment (GSA) scale (B). A, A score of 100 represents the best level of overall functioning, and a score of 0 represents the lowest level. B, A score of 0 represents the highest level of social functioning, and a score of 5 represents the lowest level. MDD indicates major depressive disorder; OPD, other personality disorders; and BPD, borderline personality disorder.
In any event, the apparent between-study differences are
not well understood. They can be partially explained by
our use of prevalence rates based on our entire sample
in contrast to the MSAD’s use of time-to-remission analy-
ses that apply only to the subjects who had the criteria
at baseline, but they may also be related to differences
in the samples and the assessment instruments. This is-
issue requires more research.

Despite statistically significant overall improvement
in functioning, the magnitude of these improvements
was far less dramatic and far less clinically significant
than the improvements found on measures of psycho-
pathology. The fact that the patients with BPD im-
proved more than those in the comparison groups re-
flected their having lower baseline functioning. The
initially more severe level of the BPD sample’s func-
tional impairment tended to converge toward the levels
of both comparison groups over time. As measured by
mean GSA scores at 10 years, BPD’s social adjustment
(3.1) lagged considerably below that found for MDD
(2.7), bipolar I disorder (2.9), and bipolar II disorder
(2.8) after 14 to 15 years. As measured by GAF score
(ie, mid 50s), our BPD sample was less functional than
observed after long-term retrospective follow-up of
other BPD samples but resembles the MSAD sample.

Why the 2 prospective studies evidenced more dysfunction than the retrospec-
tive studies is unclear. Although it could relate to sever-
it of BPD in the samples or to less effective intervening
therapies, it seems more likely that the use of rigor-
ous—presumably more valid—assessment methods for
diagnosis and functioning established a better estimate.
Our results show that the improvements in the BPD sample’s functioning evident during the first 2 years continued to progress, albeit more slowly. The BPD sample’s improvement in specific areas usually moved them from the poor to the satisfactory range of function. Moreover, the analyses of individual change indicate that while average levels of functioning change slowly, subgroups of patients with BPD (and OPD) episodically experienced substantial fluctuations at the individual level; change in function was more the norm than was stability. Thus, with respect to psychosocial function, the traditional pessimism about this disorder’s prognosis seems partially justified. Younger age, consistent with 2 prior studies—a rate approximating that found in the MSAD for 10 years (mean score, 2.1)—falls inexplicably lower than that found in prospective 14- to 15-year follow-up study 1 identified a very similar course, there now exists a strong empirically based prognostic portrait of BPD that can inform clinicians, families, and patients. By virtue of its distinctiveness, this course offers strong validation for the DSM-IV BPD diagnosis. This validation joins the hard-earned validation of DSM-IV BPD that has come from descriptive and family history or heritability research and from disorder-specific therapies. Because, as reported here, the DSM-IV definition of BPD—like DSM-IV definitions of other major psychiatric disorders—identifies a disorder whose course is disjunctive with social disability, it invites the hope that a revised characterization of BPD might more closely correspond to the disorder’s dysfunction and perhaps with its underlying genotype. While current proposals to redefine BPD for DSM-5 (http://www.dsm5.org and the article by Gunderson) might fulfill this hope, they should proceed with due recognition that the existing definition already has difficult-to-attain validation and conveys clinically essential information about course.

That psychopathology would predict dysfunction is consistent with the MSAD findings that symptomatic improvement was associated with better function and the conclusion that McGlashan reached in his earlier study. The patterns of improved psychopathology and persisting social dysfunction have been noted for other disorders. However, the finding of a course marked by gradually attained, frequent, and persistent remission is distinctive for BPD. Given that the other prospective 10-year follow-up study 1 identified a very similar course, there now exists a strong empirically based prognostic portrait of BPD that can inform clinicians, families, and patients. By virtue of its distinctiveness, this course offers strong validation for the DSM-IV BPD diagnosis. This validation joins the hard-earned validation of DSM-IV BPD that has come from descriptive and family history or heritability research and from disorder-specific therapies. Because, as reported here, the DSM-IV definition of BPD—like DSM-IV definitions of other major psychiatric disorders—identifies a disorder whose course is disjunctive with social disability, it invites the hope that a revised characterization of BPD might more closely correspond to the disorder’s dysfunction and perhaps with its underlying genotype. While current proposals to redefine BPD for DSM-5 (http://www.dsm5.org and the article by Gunderson) might fulfill this hope, they should proceed with due recognition that the existing definition already has difficult-to-attain validation and conveys clinically essential information about course.

That psychopathology would predict dysfunction is consistent with the MSAD findings that symptomatic improvement was associated with better function and the conclusion that McGlashan reached in his earlier study. The patterns of improved psychopathology and persisting social dysfunction have been noted for other disorders. However, the finding of a course marked by gradually attained, frequent, and persistent remission is distinctive for BPD. Given that the other prospective 10-year follow-up study 1 identified a very similar course, there now exists a strong empirically based prognostic portrait of BPD that can inform clinicians, families, and patients. By virtue of its distinctiveness, this course offers strong validation for the DSM-IV BPD diagnosis. This validation joins the hard-earned validation of DSM-IV BPD that has come from descriptive and family history or heritability research and from disorder-specific therapies. Because, as reported here, the DSM-IV definition of BPD—like DSM-IV definitions of other major psychiatric disorders—identifies a disorder whose course is disjunctive with social disability, it invites the hope that a revised characterization of BPD might more closely correspond to the disorder’s dysfunction and perhaps with its underlying genotype. While current proposals to redefine BPD for DSM-5 (http://www.dsm5.org and the article by Gunderson) might fulfill this hope, they should proceed with due recognition that the existing definition already has difficult-to-attain validation and conveys clinically essential information about course.

That psychopathology would predict dysfunction is consistent with the MSAD findings that symptomatic improvement was associated with better function and the conclusion that McGlashan reached in his earlier study. The patterns of improved psychopathology and persisting social dysfunction have been noted for other disorders. However, the finding of a course marked by gradually attained, frequent, and persistent remission is distinctive for BPD. Given that the other prospective 10-year follow-up study 1 identified a very similar course, there now exists a strong empirically based prognostic portrait of BPD that can inform clinicians, families, and patients. By virtue of its distinctiveness, this course offers strong validation for the DSM-IV BPD diagnosis. This validation joins the hard-earned validation of DSM-IV BPD that has come from descriptive and family history or heritability research and from disorder-specific therapies. Because, as reported here, the DSM-IV definition of BPD—like DSM-IV definitions of other major psychiatric disorders—identifies a disorder whose course is disjunctive with social disability, it invites the hope that a revised characterization of BPD might more closely correspond to the disorder’s dysfunction and perhaps with its underlying genotype. While current proposals to redefine BPD for DSM-5 (http://www.dsm5.org and the article by Gunderson) might fulfill this hope, they should proceed with due recognition that the existing definition already has difficult-to-attain validation and conveys clinically essential information about course.

That psychopathology would predict dysfunction is consistent with the MSAD findings that symptomatic improvement was associated with better function and the conclusion that McGlashan reached in his earlier study. The patterns of improved psychopathology and persisting social dysfunction have been noted for other disorders. However, the finding of a course marked by gradually attained, frequent, and persistent remission is distinctive for BPD. Given that the other prospective 10-year follow-up study 1 identified a very similar course, there now exists a strong empirically based prognostic portrait of BPD that can inform clinicians, families, and patients. By virtue of its distinctiveness, this course offers strong validation for the DSM-IV BPD diagnosis. This validation joins the hard-earned validation of DSM-IV BPD that has come from descriptive and family history or heritability research and from disorder-specific therapies. Because, as reported here, the DSM-IV definition of BPD—like DSM-IV definitions of other major psychiatric disorders—identifies a disorder whose course is disjunctive with social disability, it invites the hope that a revised characterization of BPD might more closely correspond to the disorder’s dysfunction and perhaps with its underlying genotype. While current proposals to redefine BPD for DSM-5 (http://www.dsm5.org and the article by Gunderson) might fulfill this hope, they should proceed with due recognition that the existing definition already has difficult-to-attain validation and conveys clinically essential information about course.

That psychopathology would predict dysfunction is consistent with the MSAD findings that symptomatic improvement was associated with better function and the conclusion that McGlashan reached in his earlier study. The patterns of improved psychopathology and persisting social dysfunction have been noted for other disorders. However, the finding of a course marked by gradually attained, frequent, and persistent remission is distinctive for BPD. Given that the other prospective 10-year follow-up study 1 identified a very similar course, there now exists a strong empirically based prognostic portrait of BPD that can inform clinicians, families, and patients. By virtue of its distinctiveness, this course offers strong validation for the DSM-IV BPD diagnosis. This validation joins the hard-earned validation of DSM-IV BPD that has come from descriptive and family history or heritability research and from disorder-specific therapies. Because, as reported here, the DSM-IV definition of BPD—like DSM-IV definitions of other major psychiatric disorders—identifies a disorder whose course is disjunctive with social disability, it invites the hope that a revised characterization of BPD might more closely correspond to the disorder’s dysfunction and perhaps with its underlying genotype. While current proposals to redefine BPD for DSM-5 (http://www.dsm5.org and the article by Gunderson) might fulfill this hope, they should proceed with due recognition that the existing definition already has difficult-to-attain validation and conveys clinically essential information about course.

That psychopathology would predict dysfunction is consistent with the MSAD findings that symptomatic improvement was associated with better function and the conclusion that McGlashan reached in his earlier study. The patterns of improved psychopathology and persisting social dysfunction have been noted for other disorders. However, the finding of a course marked by gradually attained, frequent, and persistent remission is distinctive for BPD. Given that the other prospective 10-year follow-up study 1 identified a very similar course, there now exists a strong empirically based prognostic portrait of BPD that can inform clinicians, families, and patients. By virtue of its distinctiveness, this course offers strong validation for the DSM-IV BPD diagnosis. This validation joins the hard-earned validation of DSM-IV BPD that has come from descriptive and family history or heritability research and from disorder-specific therapies. Because, as reported here, the DSM-IV definition of BPD—like DSM-IV definitions of other major psychiatric disorders—identifies a disorder whose course is disjunctive with social disability, it invites the hope that a revised characterization of BPD might more closely correspond to the disorder’s dysfunction and perhaps with its underlying genotype. While current proposals to redefine BPD for DSM-5 (http://www.dsm5.org and the article by Gunderson) might fulfill this hope, they should proceed with due recognition that the existing definition already has difficult-to-attain validation and conveys clinically essential information about course.

That psychopathology would predict dysfunction is consistent with the MSAD findings that symptomatic improvement was associated with better function and the conclusion that McGlashan reached in his earlier study. The patterns of improved psychopathology and persisting social dysfunction have been noted for other disorders. However, the finding of a course marked by gradually attained, frequent, and persistent remission is distinctive for BPD. Given that the other prospective 10-year follow-up study 1 identified a very similar course, there now exists a strong empirically based prognostic portrait of BPD that can inform clinicians, families, and patients. By virtue of its distinctiveness, this course offers strong validation for the DSM-IV BPD diagnosis. This validation joins the hard-earned validation of DSM-IV BPD that has come from descriptive and family history or heritability research and from disorder-specific therapies. Because, as reported here, the DSM-IV definition of BPD—like DSM-IV definitions of other major psychiatric disorders—identifies a disorder whose course is disjunctive with social disability, it invites the hope that a revised characterization of BPD might more closely correspond to the disorder’s dysfunction and perhaps with its underlying genotype. While current proposals to redefine BPD for DSM-5 (http://www.dsm5.org and the article by Gunderson) might fulfill this hope, they should proceed with due recognition that the existing definition already has difficult-to-attain validation and conveys clinically essential information about course.
An implication of this study is that the enthusiasm generated by the successes reported for psychosocial therapies of patients with BPD needs to be qualified by the recognition that these treatments have rarely demonstrated that the patients achieve better functional capacities. Clearly, future studies of therapeutic outcome need to address functional gain, but more importantly, future BPD therapies need to address functional impairment, i.e., to incorporate social learning and rehabilitation strategies. The need for rehabilitative strategies has already been recognized with other major mental illnesses. From a public health viewpoint, it is critical that therapies demonstrate their effectiveness in helping patients with BPD attain and maintain work roles.

The methods and design of this study as well as the confirmatory results from the MSAD permit a much higher level of confidence in our findings than from prior studies. Still, the completion of the study invites reminders of its limitations. The effort to attain a representative clinical urban sample precludes generalization of our findings to nonclinical or rural populations. As with all longitudinal studies, the repeated contacts with research staff may have affected the outcomes. Other limitations in-cluding to nonclinical or rural populations. As with all lon-gitudinal studies, the repeated contacts with research staff may have affected the outcomes. Other limitations in-clude our reliance on the participants as informants (when outside informants may have augmented assessment validity) and our reliance on a measure of employment that did not include homemaking. Finally, we are aware of the many related issues that we did not examine, issues such as predictors of change or the isolation of subgroups based on good or poor outcomes, comor-bidity, or sex.

In summary, the 10-year outcome of patients with BPD in the CLPS demonstrates a distinctive, clinically useful, and diagnostically validating course characterized by remissions more enduring and by functional impairment more severe than many other major psychiatric disorders. This pattern highlights the potential therapeutic rewards of treating patients with BPD, while challenging the next generation of therapies to help them become more effective by improving functional outcomes. It also highlights the imposing public health issue these patients represent and the embarrassingly disproportionate lack of attention the disorder has received.16

Submitted for Publication: June 2, 2010; final revision received December 15, 2010; accepted January 28, 2011.

Published Online: April 4, 2011. doi:10.1001 /archgenpsychiatry.2011.37

Author Affiliations: Department of Psychiatry, Harvard Medical School, McLean Hospital, Belmont, Massachusetts (Drs Gunderson and Zanarini); Decision Sciences Institute/PIRE, Pawtucket (Dr Staut); and Department of Veterans Affairs (Dr Shea) and Department of Psychiatry and Human Behavior, Brown University (Drs Shea and Yen), Providence, Rhode Island; Yale Psychiatric Research Institute and Yale University, New Haven (Drs McGlashan, Grilo, and Ansell), and Department of Psychology, Wesleyan University, Middletown (Dr Sanisol); Connecticut; Department of Psychology, Texas A&M University, College Station (Dr Morey); Department of Psychiatry, Columbia University (Drs Markowitz and Pinto) and Department of Personality Studies, New York State Psychiatric Institute (Drs Markowitz, Pinto, and Skodol), New York; and Sunbelt Collaborative and Department of Psychiatry, University of Arizona College of Medicine, Tucson (Dr Skodol).

Correspondence: John G. Gunderson, MD, McLean Hospital, 115 Mill St, Belmont, MA 02478 (jgunderson@mclean.harvard.edu).

Financial Disclosure: None reported.

Funding/Support: This work was supported by grants R01 MH 080221, R01 MH 01654, R01 MH 50837, R01 MH 50838, R01 MH 50839, R01 MH 50840, R01 MH 50850, R01 MH 69904, and R01 MH 73708 from the National Institute of Mental Health.

Additional Contributions: The manuscript was reviewed and approved for submission by the Publications Committee of the Collaborative Longitudinal Personality Disorders Study.

REFERENCES


