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Prevalence of and Familial Influences on Purging Disorder in a Community Sample of Female Twins

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Abstract

Objective—Purging Disorder (PD) was recently included as an Otherwise Specified Feeding or Eating Disorder (OSFED) in the DSM-5; however, limited information is available on its prevalence, and its etiology is unknown.

Method—Data from 1790 monozygotic and 1440 dizygotic European American female twins (age range = 18 – 29 years) from the Missouri Adolescent Female Twin Study were used to investigate prevalence and familial influences for PD. A structured clinical interview assessed lifetime DSM-IV criteria for eating disorders and PD. After adjustment for age, twin correlations and biometrical twin models were used to estimate familial (i.e., genetic plus shared environmental) influences on PD.

Results—One hundred and twenty one (3.77%; 95% CI: 3.14, 4.49) women met criteria for lifetime PD. Twin correlations suggested that genetic, shared environmental, and nonshared environmental factors influenced liability to PD. Nonshared environmental factors accounted for 56% [35%, 79%] of the variance in PD. Although familial effects accounted for a significant proportion of variance (44% [21%, 65%]), it was not possible to disentangle the independent contributions of additive genetic effects (20% [0%, 65%]) and shared environmental effects (24% [0%, 57%]).

Discussion—PD is a prevalent form of eating pathology. Familial factors are relevant to the development of PD but do not demonstrate the magnitude of heritable factors found for other eating disorders.

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Keywords

purging disorder; twins; genetics; women; eating disorders

Introduction

Purging disorder (PD) is a new diagnosis included as an Otherwise Specified Feeding or Eating Disorder (OSFED) in the DSM-5¹. Inclusion of PD in the DSM-5 was supported by evidence of the clinical significance of PD as well as its potential distinctiveness from other eating disorders on both psychological and biological factors (see² for review). Prior population-based studies of women have shown that the prevalence of PD ranges from 1.1% to 5.3%^{3–6} and the mean age of onset is around age 20⁷. Recent data support that PD may follow a distinct longitudinal course from Anorexia Nervosa (AN) and Bulimia Nervosa (BN), demonstrating greater chronicity than AN and greater mortality than BN⁸. Despite this information, limited data have established the lifetime prevalence of PD in population-based studies in the US, and etiological studies of PD have yet to be undertaken. In particular, unlike information on AN and BN, the extent to which familial (i.e., genetic plus shared environmental) factors influence PD is unknown.

Studies have estimated that the heritabilities of AN, BN, and Binge Eating Disorder (BED) are 28%–74%, 28%–83%, and 39%–45%, respectively⁹. Although no twin studies of PD have been published, extant research has examined genetic and environmental factors for specific purging behaviors (i.e., self-induced vomiting, laxative use, and diuretic use). An early report¹⁰ found that the heritability of vomiting was 72%, whereas a more recent study¹¹ suggested that this heritability was much lower at 8%. Additional research has indicated that the heritability of inappropriate compensatory behaviors, a broader category which includes self-induced vomiting and laxative misuse as well as fasting and excessive exercise, is between 28% and 52%^{12–14}. Even though these studies have provided important information regarding familial influences on purging and related behaviors, they have not examined the familial effects of PD. Because purging can occur transdiagnostically as a feature of the binge-purge subtype of AN and within BN, prior heritability studies of purging behaviors may simply reflect heritability of these disorders. Thus, additional work is necessary to elucidate the familial risk associated with PD. To address gaps in the literature, we investigated the lifetime prevalence of PD and extent to which familial factors influenced liability to PD.

Methods

Participants

Participants were part of the Missouri Adolescent Female Twin Study (MOAFTS)^{15; 16}, a population-based longitudinal study of female twins who were identified from state birth records as a twin pair born between July 1st 1975 and June 30th 1985 in Missouri to a mother who was a state resident. Twins were recruited using a cohort sequential sampling design, with ascertainment of successive 6-month cohorts of 13, 15, 17, and 19 year old twin pairs over a two-year period and continued recruitment of 13 year olds for an additional two

years. Importantly, the MOAFTS sample was demographically representative of the Missouri population at the time the twins were born, with nearly 15% of twins being African American and the remainder being of European descent (as reported by the mother at the time of birth). A baseline interview was conducted with the twins beginning in 1995 (median age = 15 years). When possible, interviews were also conducted with at least one parent (usually the mother) at the time the twins entered the study. Zygosity was determined by standard questions¹⁷ that have shown approximately 95% agreement with genotyping methods¹⁸. The Wave 4 young adult follow-up interview was conducted an average of six years after the baseline assessment (median age = 22 years). Since all members of the target cohort were 18 years old or older and study participation was no longer contingent upon parental consent, all individuals from the original sampling frame were invited to participate, even if they had not participated at baseline. Only those twins who themselves refused future contact or whose parents had refused all future contact for the family were excluded from being recontacted at Wave 4. Analyses were conducted on 3230 European American twins who completed the Wave 4 assessment. As the etiological underpinnings of PD may vary across ethnic groups and the proportion of African American twins was modest, we restricted these analyses to European American women. There were 1516 (853 monozygotic [MZ] and 663 dizygotic [DZ]; 94% of overall sample) complete twin pairs for which both co-twins participated. Data from an additional 198 (84 MZ and 114 DZ; 6% of overall sample) individual twins who had missing co-twin information were also included in the analyses, out of a total of 1999 European American pairs (3998 twins) originally identified from birth records. This represents 81% of the original European American sampling frame. Additional details about response rates across assessments may be found elsewhere¹⁶. The protocol was approved by the Washington University School of Medicine Institutional Review Board, and all twins gave informed consent before study participation.

Measures

At Wave 4, an adaptation of the Semi-Structured Assessment on the Genetics of Alcoholism (SSAGA¹⁹) was administered over the phone to all participants to assess DSM-IV²⁰ lifetime criteria for multiple psychiatric (e.g., eating disorders, Major Depressive Disorder) and substance use (e.g., Alcohol Use Disorder, Nicotine Dependence) disorders. The SSAGA was based on and is similar to other commonly used lay-administered diagnostic interviews, such as the Diagnostic Interview Schedule²¹ and the Composite International Diagnostic Interview²², in that it has multiple sections, each asking specific questions pertaining to DSM-IV criteria for the respective disorder. A unique strength of the modified version of the SSAGA is that it also permitted assessment of eating disorders not included in DSM-IV, such as PD. Lifetime history of PD was operationalized as a binary (yes/no) variable if the participant endorsed the following: 1) recurrent self-induced vomiting, laxative use, or diuretic use in the absence of binge eating; 2) an intense fear of weight gain or becoming fat; 3) binge eating (without loss of control) less than 1 day per month; and 4) no lifetime diagnosis of DSM-5 AN or BN¹. Of relevance to the current report, all questions regarding inappropriate compensatory behaviors were not conditional on endorsement of binge eating, permitting evaluation of these behaviors in the absence of binge eating. Work by Swanson et al.²³ suggest that a significant proportion of clinically significant eating pathology is missed by following skip rules in structured interviews. Although other diagnostic sections in the

SSAGA have reported good reliability and validity¹⁹, unfortunately, there are no psychometric data available for the eating disorders section. Additional information regarding the eating disorder assessment can be found elsewhere²⁴.

Multiple psychiatric and substance use disorder diagnoses assessed at Wave 4 were also examined in this study. We included DSM-IV Major Depressive Disorder, DSM-IV Alcohol and Cannabis Abuse and Dependence, and DSM-IV Nicotine Dependence. Of note, clustering was not required for Cannabis Dependence only. Finally, demographic characteristics included maternal and paternal education (based on twin and parental reports), body mass index (calculated as weight (in kilograms)/height (in meters) squared), and age at menarche.

Statistical Analyses

SAS²⁵ was used for data management and descriptive analyses were conducted in Stata²⁶. Survey commands were used for analyzing bivariate associations. Under the survey command, the Pearson chi-square (χ^2) statistic is corrected for the twin sampling design using the Rao and Scott²⁷ second order correction, converting the Pearson χ^2 into an F -statistic. We conducted linear regression analyses to test for significant mean differences between groups, and Huber-White robust variance estimators were used to adjust standard errors for the non-independence of observations in twin data. Twin modeling was conducted in Mx^{28; 29}. Using raw data, individual differences in liability to PD were assessed from three sources: additive genetic (A), shared environmental (C), and nonshared environmental (E) effects. Additive genetic effects refer to the sum of the individual effects of multiple genes on a given trait. Shared environmental effects are those factors that make family members similar, and nonshared environmental effects are factors that make members within a family uncorrelated for a trait. Importantly, measurement error is included in the nonshared environmental effect estimate.

Models controlled for age, which was categorized by quartiles (i.e., 18–19, 20–22, 23–24, and 25–29 year olds) and included as a set of definition variables that were freely estimated in Mx. This allowed the prevalence rates for PD to differ for each age group. With respect to twin correlations, genetic effects are present if the r_{MZ} is greater than the r_{DZ} , and nonshared environmental effects are present if the r_{MZ} is less than one. If the r_{DZ} is greater than half the r_{MZ} , then there is evidence for shared environmental effects. Finally, if the twin correlations are equal, there is no evidence for genetic effects on PD.

For biometrical twin modeling, multiple submodels of the full ACE model were fit to the data that constrained A, C, and E to zero. For all analyses, minus 2 log-likelihood ($-2LL$) and standard chi-square difference tests (χ^2)²⁸ were used to compare the fit of nested models. P -values less than 0.05 for the χ^2 indicated a poorer fit to the data compared with the full model. In other words, p -values less than 0.05 suggested that the parameter(s) could not be dropped from the model without providing a significant decrement in model fit. Akaike's Information Criteria (AIC; $-2LL - 2df$)³⁰ was also included for comparison of non-nested models. Although lower AIC values generally indicate better-fitting and more parsimonious models, using the AIC to compare univariate models may be problematic³¹; thus, the AIC should be used for illustrative purposes only.

Results

One hundred and twenty one (3.77%; 95% CI: 3.14, 4.49) women met criteria for lifetime PD, as compared to 44 (1.37%; 1.00, 1.84), 24 (0.75%; 0.48, 1.11), and 3 (0.09%; 0.02, 0.27) women who met criteria for DSM-5 AN, DSM-IV BN, and DSM-IV BED, respectively. Compared to women without PD, women with PD were more likely to have mothers with less than a high school education (9.26% vs. 14.88%, $p = 0.04$), as well as significantly higher rates of Major Depressive Disorder (18.09% vs. 37.19%; $p < 0.0001$), Alcohol Abuse (19.71% vs. 33.88%; $p = 0.0002$), Alcohol Dependence (7.81% vs. 17.36%; $p = 0.0004$), Alcohol Use Disorder (21.89% vs. 38.02%; $p < 0.0001$), and Nicotine Dependence (18.27% vs. 32.23%; $p = 0.0002$). There were no significant differences between the two groups on age, zygosity, body mass index, age at menarche, or Cannabis Abuse/Dependence (Table 1). In women with PD, the most frequently endorsed inappropriate compensatory behavior was self-induced vomiting (71.07%), whereas the least endorsed item was laxative use (25.62%) (Table 2). Additionally in these individuals, two had cotwins who reported lifetime AN, three had cotwins who reported lifetime BN, and one had a cotwin who reported lifetime BED.

Among MZ twins with PD, there were six concordant and 52 discordant pairs; among DZ twins, there were three concordant and 40 discordant twin pairs. An additional three MZ and eight DZ twins with PD had missing cotwin information. The MZ twin correlation (r_{MZ}) was 0.45 [0.20, 0.66], whereas the DZ twin correlation (r_{DZ}) was 0.35 [0.01, 0.63]. Given that the r_{MZ} was less than one and was greater than the r_{DZ} , but less than twice the r_{DZ} , there was evidence for familial (i.e., genetic and shared environmental) influences, as well as nonshared environmental effects on PD.

Results from biometrical twin modeling procedures are shown in Table 3. Comparisons between the full ACE model and the three nested submodels (i.e., CE, AE, and E models) indicated that the only model which resulted in a significant decrement in fit was one where only nonshared environmental factors influenced liability to PD ($\chi^2 = 14.61$; $df = 2$; $p < 0.001$). Although the AIC was lowest for a model with shared and nonshared environmental factors (AIC = -5061.04; $\chi^2 = 0.26$; $df = 1$; $p = 0.61$), a model with additive genetic and nonshared effects also provided an adequate fit to the data (AIC = -5060.84; $\chi^2 = 0.46$;

$df = 1$; $p = 0.50$). In light of the non-significant decrement in fit (per χ^2) in the CE and AE models, but strong evidence of familial influences (significant χ^2 for the E only model), we retained the full ACE model as the best-fitting model. Nonshared environmental factors accounted for 56% [35%, 79%] of the variance in PD. Although familial effects were highly significant, accounting for the remaining 44% [21%, 65%] of the variance, it was not possible to disentangle the separate contributions of additive genetic effects (20% [0%, 65%]) and shared environmental effects (24% [0%, 57%]) because the 95% confidence intervals both included zero.

Discussion

This study provides important information on the lifetime prevalence of PD in the US and is the first published study to examine the extent to which familial effects influence liability to

PD in young adult female twins. Approximately 4% of the sample met criteria for PD, with familial (i.e., genetic and shared environmental) effects contributing to 44% of the variance in this disorder. The remaining variance was attributed to nonshared environmental effects. The prevalence of PD in this sample is comparable to estimates reported in other studies of women^{4–6}. In addition, the familial influences of PD were similar in magnitude to what has been reported in prior studies on self-induced vomiting and other compensatory behaviors^{10; 12–14} and with twin studies of AN, BN, and BED⁹. The current study was not able to distinguish whether familial influences reflected genetic or shared environmental effects, a pattern of uncertainty found in studies of contributions to purging behaviors^{10; 11}. This is likely due to low statistical power. Larger samples will be needed to understand the specific contributions of familial influences on PD, particularly if *both* genetic and shared environmental factors are important. As found for other eating disorders, there were significant contributions of nonshared environmental factors (and measurement error) on PD. Sources of nonshared environment (i.e., prior experiences that were not shared between cotwins) that could be important to the development of PD include friends, trauma, weight history, personality differences, and previous psychiatric and substance use history. Additional genetically informative study designs (e.g., discordant MZ twin or prospective twin/family studies)³² will be needed to identify these specific nonshared environmental factors.

Information on exact genetic mechanisms underlying PD, though speculative, could include systems involved in satiation (e.g., serotonin, estrogen) or reward (e.g., dopamine, opioid) and could potentially overlap with those of other eating disorders^{9; 33}. Other research suggests that some environmental risk factors that have been shown to influence other eating disorders may also influence PD³⁴. Indeed, in a comparison of cohorts of Fijian adolescent girls before and after the introduction of Western TV programs into girls' households, the prevalence of self-induced vomiting among the girls increased from 0% to 11%³⁵. At minimum, a significant familial risk indicates that family history of PD, in addition to environmental risk not shared with family members, must be evaluated in order to provide the most effective prevention and treatment.

A unique strength of this study was the absence of skips from the diagnostic section, which allowed us to examine purging regardless of whether participants endorsed binge eating behaviors. In contrast, the recent National Comorbidity Survey-Replication only probed for purging if binge eating was coded as present, which prevented evaluation of the prevalence of this syndrome in the largest epidemiological study in the US with assessments of eating disorders and their symptoms³⁶. As such, the current study adds much needed data on the lifetime prevalence of PD in the US. Still, this study had some limitations. First, as mentioned above, there was low statistical power to distinguish between genetic and shared environmental influences on PD. Larger sample sizes will be necessary in the future to disentangle the separate contributions of familial influences on this disorder. Second, for a PD diagnosis, we excluded individuals who had met lifetime criteria for DSM-5 AN or BN. This exclusion may have underestimated the true prevalence rate of PD since pairs treated as discordant for PD may actually have been concordant for PD if one cotwin had PD with no prior history of AN or BN and her cotwin had PD with a history of another eating disorder.

Had we been able to include additional individuals with PD, our ability to disentangle familial risk into genetic and shared environmental effects would have increased, but unfortunately, the diagnostic interview did not assess past year or past month eating disorders. Thus, exclusion of lifetime AN was the only way to ensure that diagnoses of PD did not reflect the binge-purge subtype of AN. Third, the binge eating questions did not probe for loss of control over eating and, thus, may have conflated objective overeating with binge eating. This could further reduce our estimate of the prevalence of PD as we adopted a more conservative approach in requiring that neither form of excessive food intake (with or without loss of control) was recurrent. Finally, the mean age of our sample was 22 years, and it is possible that some respondents have not yet passed through the risk period for developing PD or other eating disorders. It has been reported that the mean age of onset for PD is 20 years old⁷. However, 20 years of age represented the upper age of assessment in that longitudinal study. Thus, the true age range for peak onset of PD remains unknown. If some twins have yet to develop PD, this would result in an underestimate of lifetime prevalence of PD in late adolescent and young adult women and could also underestimate concordance within twin pairs.

In conclusion, this study found that familial (i.e., genetic plus shared environmental) influences accounted for 44% of the overall liability to PD, and the 95% confidence interval for this estimate did not include zero. These results suggest that PD has a similar or perhaps slightly lower proportion of familial influences compared with that reported for the other eating disorders. Unlike studies of other eating disorders with comparable sample sizes and prevalence estimates, we could not distinguish between genetic and shared environmental influences on PD. Future studies with larger twin samples are needed to determine whether this finding reflects a difference in etiological influences for PD compared to other eating disorders. Specifically, it may be that unlike AN and BN, both genetic and shared environmental risk factors contribute to the etiology for this newly specified eating disorder in the DSM-5¹. Additional exploration of gene-environment interplay, and whether there are overlapping genetic and environmental risks for PD and other eating disorders are needed to comprehensively understand the etiology of PD.

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Table 1

Sample characteristics (results shown as n (%) unless otherwise indicated)

	Lifetime Purging Disorder (n = 121)	No Lifetime Purging Disorder (n = 3087)	F (df)	p
<i>Demographics</i>				
Age (mean (sd))	21.53 (0.27)	21.63 (0.07)	0.14 (1, 1714)	0.71
Monozygotic	67 (55.37)	1713 (55.49)	0.00 (1, 1714)	0.98
Maternal Education			3.14 (2, 3419.51)	0.04
Less than high school	18 (14.88)	286 (9.26)		
High school or college	99 (81.82)	2750 (89.08)		
Missing	4 (3.31)	51 (1.65)		
Paternal Education			0.24 (1.99, 3409.62)	0.79
Less than high school	12 (9.92)	303 (9.82)		
High school or college	104 (87.13)	2691 (87.17)		
Missing	5 (3.05)	93 (3.01)		
Current body mass index			1.05 (3, 5134.23)	0.37
Underweight	4 (3.33)	230 (7.46)		
Normal Weight	80 (66.67)	2020 (65.54)		
Overweight	21 (17.50)	500 (16.22)		
Obese	15 (12.50)	332 (10.77)		
Age at menarche (mean (sd))	12.75 (0.13)	13.00 (0.03)	3.42 (1, 1713)	0.06
<i>Psychiatric and Substance Use Disorder History (Lifetime)</i>				
Major Depressive Disorder	45 (37.19)	558 (18.09)	29.65 (1, 1714)	<0.0001
Alcohol Abuse	41 (33.88)	605 (19.71)	14.02 (1, 1711)	0.0002
Alcohol Dependence	21 (17.36)	241 (7.81)	12.52 (1, 1714)	0.0004
Alcohol Use Disorder	46 (38.02)	672 (21.89)	16.88 (1, 1711)	<0.0001
Nicotine Dependence	39 (32.23)	564 (18.27)	14.31 (1, 1714)	0.0002
Cannabis Abuse	3 (2.50)	81 (2.62)	0.01 (1, 1714)	0.93
Cannabis Dependence	3 (2.48)	52 (1.68)	0.45 (1, 1714)	0.50
Cannabis Use Disorder	5 (4.13)	106 (3.43)	0.17 (1, 1714)	0.68

Note: Survey commands in Stata²⁶ were used for analyzing bivariate associations. Under the survey command, the Pearson chi-square (χ^2) statistic is corrected for the twin sampling design using the Rao and Scott²⁷ second order correction, converting the Pearson χ^2 into an F-statistic. Significant differences between women with and without Purging Disorder are indicated by **bold-type**.

Table 2

Prevalence of inappropriate compensatory behaviors in women with Purging Disorder (n = 121)

	n (%)
Any purging behaviors	121 (100.00)
Self-induced vomiting	86 (71.07)
Laxative use	31 (25.62)
Water pill or diuretic use	44 (36.36)
Any non-purging behaviors	100 (82.64)
Strict dieting	66 (54.55)
Fasting	60 (49.59)
Exercise vigorously	67 (55.37)

Table 3

Univariate model-fitting results for Purging Disorder (PD)

Model	a^2	c^2	e^2	-2LL	AIC	χ^2	df	<i>p</i>
ACE	0.20 [0.00, 0.65]	0.24 [0.00, 0.57]	0.56 [0.35, 0.79]	938.70	-5059.30	---	---	---
CE	---	0.40 [0.20, 0.58]	0.60 [0.42, 0.80]	938.96	-5061.04	0.26	1	0.61
AE	0.47 [0.23, 0.66]	---	0.53 [0.34, 0.77]	939.16	-5060.84	0.46	1	0.50
E	---	---	1.00 [1.00, 1.00]	953.31	-5048.69	14.61	2	< 0.001

Note: A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effects; a^2 = proportion of variance in PD due to additive genetic effects; c^2 = proportion of variance in PD due to shared environmental effects; e^2 = proportion of variance in PD due to nonshared environmental effects; -2LL = -2 log likelihood; df = degrees of freedom; AIC = Akaike's Information Criterion; χ^2 = chi-square difference test statistic; df = change in degrees of freedom; *p* = *p*-value associated with χ^2 . The best-fitting model is indicated by **bold-type**.