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Purging Disorder: Recent Advances and Future Challenges

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Abstract

Purpose of review—This review aims to help specialists remain up-to-date on research from the past two years on epidemiology, risk factors, biological correlates, treatment and outcomes for purging disorder (PD), a DSM-5 Other Specified Feeding and Eating Disorder (OSFED).

Recent findings—PD affects 2.5 to 4.8% of adolescent females in population-based samples, but PD remains relatively rare in treatment settings. Higher premorbid body mass index, body dissatisfaction, and dieting prospectively predict PD onset. In studies of biological correlates, women with PD demonstrated significantly greater postprandial increases in the satiety peptide, PYY, compared to women with bulimia nervosa and controls, and these differences predicted greater gastrointestinal distress in PD. Less than half of those with PD are free from an eating disorder at the end of treatment and at one or more years of follow-up, supporting the need for improved interventions.

Summary—Purging disorder may occupy a space that falls between anorexia and bulimia nervosa, making it “not quite” anorexia and “not quite” bulimia and difficult to reliably distinguish from each. Improved recognition and understanding of PD requires more research specifically designed to test models of risk and maintenance factors to advance interventions for those who purge without binge eating.

Keywords

purging disorder; epidemiology; risk factors; biology; treatment

Introduction

According to the DSM-5, purging disorder (PD) is characterized by recurrent purging in the absence of binge eating in individuals who are not underweight (1). The DSM-5 definitions for binge eating, purging, and underweight are critical to understanding this characterization. Binge eating involves consumption of a large amount of food (e.g., a package of cookies) in a limited amount of time accompanied by a sense of loss of control while eating. Purging involves the forceful evacuation of matter from the body, most often through self-induced vomiting but also through misuse of laxative, diuretics, or other medications. Finally, the guideline for underweight is an adult body mass index (BMI) <18.5 kg/m^2 or, for minors, a

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BMI < 5th percentile for age and sex. PD is not anorexia nervosa (AN) because individuals with AN are underweight and individuals with PD are not. PD is not bulimia nervosa (BN) because individuals with BN have binge episodes and those with PD do not. In this sense, PD falls between AN and BN in its clinical presentation, raising questions about whether it represents a distinct disorder of eating or a partial or subthreshold variant of already recognized eating disorders. In July 2017, a comprehensive meta-analysis (2) aimed to answer that question. This current review seeks to update readers on the research published since that review.

Epidemiology

Point prevalence estimates for PD in adolescent girls range from 2.5% to 4.8%, depending on age group (3) and illness definition (4). In a study of 9,031 US girls aged 9–15 years and followed prospectively from 1996 to 2013, the lifetime prevalence of PD was estimated at 6.2% over the course of the study (3). Broken down by age group, point prevalence was 0.4% in girls 9–12 years, 1.9% at 13–15 years, 2.5% at 16–18 years, 2.5% at 19–22 years, 2.5% at 23–27 years, and 1.3% at 28 years and older. By comparison, AN peak prevalence was 0.7% from ages 16–18 years, BN peak prevalence was 1.1% from ages 19–22 years, making PD more than twice as common as these two syndromes across these ages (3). These estimates also indicate elevated risk extends to older ages in PD, in line with prior findings for a later onset age for PD compared to AN and BN (5). A study of 5,072 Australian adolescent girls and boys between the ages of 11 and 19 years, estimated the point prevalence of PD to be 3.2%, and found significantly greater prevalence in girls (4.8%) compared to boys (1.6%) (4). PD also was more likely to occur among adolescents who were overweight. PD estimates were reduced to 2.6% or 1.5%, when adding a criterion requiring moderate or severe distress, respectively. Applying these criteria to AN and BN, prevalence estimates were 0.6%/0.5% for AN and 4.6%/3.3% for BN, suggesting PD is more common than AN but less common than BN. Across eating disorders, requiring moderate or severe distress particularly impacted PD prevalence estimates (4), suggesting recurrent purging in the absence of binge eating and underweight may be ego-syntonic for many adolescent girls.

In a separate survey of individuals 15 years and older in metropolitan and rural districts in South Australia, the 3-month prevalence of PD was 0.3% (6). In addition to being less prevalent, PD cases had a mean age of 67.5 years, with just over one third (36%) being male. These features differ from prior descriptions of PD (2) and may reflect the diagnostic hierarchies used to create mutually exclusive DSM-5 categories (6). Despite the absence of trumping rules for OSFED in the DSM-5 (1), some studies, including this one, use a diagnosis of atypical AN as a rule out for a diagnosis of PD. This means that among individuals who are not underweight, those who purge in the absence of binge eating are diagnosed with atypical AN if they have lost weight, restricting PD diagnoses to those who purge in the absence of binge eating but have maintained a stable weight. This ad hoc diagnostic hierarchy mirrors the official hierarchy between diagnoses of AN and BN in the DSM-5. However, it lacks empirical support because there are no studies that have established the predictive validity of distinctions among atypical AN with and without purging and PD without and without weight loss.

Two articles addressed the prevalence of PD in clinical settings (7, 8). In both, AN is overrepresented in patient samples relative to its population-based prevalence with nearly 2 AN patients for every 1 BN patient in both outpatient (7) and inpatient settings (8). However, the gap between population-based and patient-based representation for PD is even greater. Among 651 consecutive adolescent intakes PD accounted for 4.5% of outpatients (7). AN outpatients outnumber PD outpatients 7:1, and BN outpatients outnumber PD outpatients almost 6:1. Among inpatients, the AN:PD ratio is almost 17:1 and BN:PD ratio is almost 10:1 (8). Nakai and colleagues (8) conducted a comprehensive retrospective review of patients seeking treatment at the Kyoto University Hospital between 1963 and 2004 to establish the prevalence of DSM-5 eating disorders across cohorts from: 1963–1974, 1975–1984, 1985–1994, and 1995–2004. PD was found in 0% of the first cohort, 2.1% of the second cohort, 3.5% of the third cohort, and 2.3% of the fourth cohort, providing no clear evidence for an increase in the relative proportion of eating disorder patients seeking treatment for PD. This trend differed from observations for AN, which accounted for decreasing proportions of patients over successive cohorts, and differed from observations for BN and BED, which both accounted for increasing proportions of patients over cohorts (8). Representation in patient cohorts reflects at least three factors: 1) population-based prevalence, 2) illness severity, and 3) illness recognition (9). Given population-based prevalence estimates and medical risks of purging (10), lack of recognition may be a particularly relevant barrier to treatment for this “other” eating disorder.

Prospective Risk Factors

A handful of recent studies have examined prospective risk factors for the development of PD in community-based samples. The largest of these comes from the population-based Avon Longitudinal Study of Parents and Children (ALSPAC) (11). The ALSPAC recruited all pregnant women (N=14,541) living in the Avon area of the UK to provide data on themselves and from their children (N=13,617) born between April 1, 1991 and December 31, 1992. In one of several papers from this sample, researchers examined BMI percentile trajectory from birth to 12.5 years of age to examine prospective risk for an eating disorder diagnosis at 14, 16, or 18 years in 1,502 children. PD contributed 133 of the 536 detected eating disorder cases (25%), with a 9:1 female to male ratio. Parametric growth models demonstrated that, prior to their PD onset, boys demonstrated a significant increase in BMI percentile by the age of 6 years compared to boys who did not develop eating disorders. For girls who later developed PD, BMI percentile was significantly greater beginning at 5 years of age compared to girls without eating disorders. For girls and boys who went on to develop BN, BMI percentiles diverged significantly from non-eating disorder controls as early as 2 years of age. Similar patterns emerged for elevated premorbid BMI trajectories for BED. In contrast, premorbid BMI trajectories were significantly lower for girls (by age 4) and boys (by age 2) who went on to develop AN compared to those who did not develop any eating disorder. Premorbid BMI trajectories differed significantly between AN and both PD and BN, which did not differ significantly (11). Higher premorbid weight may explain why those with PD fear gaining weight and do not become underweight when they resort to purging. However, it also raises questions about the boundary between PD and atypical AN. If those with PD (and BN) develop purging and other extreme weight control behaviors in response

to higher premorbid BMI and these behaviors contribute to weight loss, how do we distinguish between atypical AN and PD (and BN) when behavioral symptoms overlap?

One study collapsed data across three separate school-based prevention trials of high-risk adolescents and young adults, producing a sample of 1,272 females (12). In multivariable analyses for PD onset, greater body dissatisfaction and more frequent dieting were significant prospective predictors. Examining predictors for onset of the other eating disorders suggested somewhat different patterns, with negative affect and lower BMI significantly predicting AN onset; body dissatisfaction, overeating, and fasting predicting BN onset; and body dissatisfaction, overeating, and functional impairment predicting BED onset. Thus, body dissatisfaction increased risk across PD, BN and BED, but overeating uniquely predicted disorders characterized by binge episodes (12). Utilizing data from the same sample, Stice and Desjardins (13) employed classification tree analysis to examine the unique pathways girls followed in the eventual development of eating disorders. Highest risk for PD onset occurred in girls who dieted more, held more positive thinness expectancies, and experienced elevated but not extremely high negative affect. Similar to prior results in the same sample(12), lower BMI was a unique branch for development of AN, and overeating was a unique branch for the development of BN and BED(13). Taken together, results suggest that individuals with PD may lack precursors to core symptoms for AN (low weight), share elevated premorbid BMI and body dissatisfaction as risk factors with BN and BED, but then lack a precursor for binge eating (overeating) that increases risk for BN and BED. These findings raise questions about potential biologically-based differences in regulation of eating and weight between PD and other eating disorders.

Biological Correlates

My own program of research has focused on the key feature that distinguishes PD from both BN and healthy eaters. In our clinical interviews, we have established that women with PD are more likely than non-eating disorder controls to subjectively experience a loss of control over their eating, but they do not objectively consume more than most people would eat under similar circumstances. In our most recent study, mean caloric intake prior to purging was 535 kcal in PD, compared to 2,722 kcal in BN (14). Reflecting these self-reported differences, women with BN ate significantly more in an *ad lib* meal (316 grams) compared to women with PD (230 grams) or controls (226 grams) to reach the same level of fullness, and we found no differences between healthy women and those with PD. To achieve the same level of subjective fullness (a score of 77 on a 100 mm visual analogue scale), women with BN consumed 40% more kcal compared to women with PD and controls. We found a significant correlation between the amount of food consumed during the *ad lib* meal and food intake prior to purging in PD and BN, suggesting that differences between PD and BN may be linked to factors that regulate satiation. Although there were no differences in feelings of fullness after the meal, groups differed significantly in feelings of nausea, stomach ache, and desire to vomit. Healthy women experienced very low levels of these before and after the meal. In contrast, both women with BN and women with PD experienced significant increases in gastrointestinal distress (14).

To probe whether biological factors contributed to these differences, we tested physiological and subjective responses to a standardized test meal. Our prior studies had revealed that women with BN demonstrated lower cholecystokinin (CCK) responses (15) and glucagon-like peptide 1 (GLP-1) levels (16) following the same test meal. Both trigger satiation, and lower function in BN may explain their vulnerability to consume large amounts of food while bingeing. In our most recent study (17), we sought to understand the presence of purging after normal amounts of food in PD. Consistent with prior results in a different sample (15), women with PD reported significantly greater increases in nausea and stomach ache after eating compared to both women with BN and healthy women. These increases were predicted by postprandial increases in the gut satiety peptide, PYY. Unlike satiation peptides, which signal the brain to terminate a meal, the satiety peptide signals the brain to delay onset of the next meal. Women with PD demonstrated a significantly elevated postprandial PYY response compared to both controls and women with BN, who did not differ from each other (17). Compared to healthy women, both women with BN and those with PD demonstrated higher fasting ghrelin levels, and although ghrelin decreased after eating in all women, the levels remained higher in both eating disorder groups. Yet changes in subjectively reported hunger did not remain higher both eating disorder groups. For women with BN, hunger stayed higher after eating compared to controls and women with PD (17). Combined with our prior findings, results suggest unique biological correlates for PD, in which women demonstrated elevated ghrelin, intact CCK and GLP-1 responses, and elevated PYY response to food intake. In contrast, BN was characterized by elevated ghrelin, blunted CCK and GLP-1 response, and intact PYY response to food intake. These alterations may contribute to maintenance of large binge episodes in BN by increasing hunger and diminishing satiation whereas the unique combination of intact satiation signals and excessive satiety signal in PD may contribute consumption of normal amounts of food followed by an urge to vomit. Importantly, biological correlates of PD do not necessarily reflect etiological or maintenance factors. Molecular genetic studies of PD have provided a clearer picture of potential causes versus consequences of illness.

Molecular Genetic Studies

In a second paper from ALSPAC (18), researchers examined the mothers' reports of how well they felt cared for during their childhood and then provided blood samples of their own DNA to examine associations between lifetime eating disorder diagnoses and two genes for the oxytocin receptor (rs53576 and rs2254298). Those with PD histories experienced low care from their moms when they were growing up compared to those without eating disorders but showed no genotypic differences from controls. Conversely, BN was associated with the rs53576 G/G genotype and the combination of low maternal care with carrying the A allele for the rs2254298 oxytocin receptor gene. Restrictive eating disorders also were linked to carrying the A allele of the rs2254298 genotype, regardless of maternal care history. Candidate gene studies have come under criticism for lack of replication (19), and findings do not prove that AN and BN are genetically influenced and PD is not, but they echo behavioral genetic findings for PD (20). Unlike robust support for significant heritability and an absence of shared familial influences for AN and BN in adult twin studies (19), the one large twin study for PD could not distinguish genetic from shared familial influences on PD (20).

In a third paper using the ALSPAC sample (21), researchers identified which women had a history of an eating disorder, had an eating disorder during pregnancy, or never had eating disorders. When their babies were born, the researchers sampled blood from the babies' umbilical cord to determine DNA methylation. Prior research had shown that pregnant women with lower body weight had babies with lower DNA methylation and that lower DNA methylation predicted lower body fat during infant development. Compared to women with no history of an eating disorder, the babies of women with a past eating disorder history had lower DNA methylation, and DNA methylation was lowest in babies of women who had an eating disorder during their pregnancy. Effects of eating disorder symptoms on DNA methylation were strongest for dietary restriction and purging without binge eating (16). Thus, epigenetic consequences of PD may resemble those for AN but differ from those for BN or BED.

Treatment and Outcomes

There have been no published randomized controlled trials (RCTs) focused on the treatment of PD and no RCTs in which outcomes have been specifically reported for patients with PD. One recent case series (22) described treatment outcome in 57 patients with PD. Mean duration of treatment was 4 months, and the mean number of sessions attended was 10. Among PD patients, 37% dropped out of treatment, 21% continued to have PD, 25% had partial remission, and 18% no longer had an eating disorder after treatment. Drop out was predicted by lower harm avoidance, reward dependence, and self directedness. Similarly, predictors of partial or full remission included higher harm avoidance, higher persistence, higher self-directedness, and lower self worry. This report also provided treatment outcomes for 82 patients with atypical AN and 37 patients with subthreshold BN. No significant differences in treatment outcome were observed across groups. However, outcome predictors differed between PD and both atypical AN and subthreshold BN (22).

Glazer et al. (3) provided follow-up data from the Growing Up Today Study (GUTS) for 563 adolescents diagnosed with PD. At one or more years following diagnosis of PD in this multi-wave longitudinal study, 13% continued to have PD, 17.9% had sub-PD, 4.8% migrated to a diagnosis of BN, 4.4% to sub-BN, 0.9% migrated to diagnosis of BED, 1.6% to sub-BED, 36.2% no longer met criteria for an eating disorder, and 20.1% provided no data at follow-up. Collapsing across these outcomes, persistence of PD syndrome (31%) was more likely than crossing over to a disorder characterized by binge eating (12%). Combining findings from both studies (3, 22) suggests that likelihood of full remission (18% to 36%) is similar to likelihood of persistence of PD (21% to 31%).

Conclusion

From 2017 to the present, 13 empirical articles have contributed to greater understanding of the prevalence, causes and treatment of PD. However, we could be doing better. Most of the reviewed studies come from large samples in which PD criteria could be retroactively applied rather than from studies that set out to examine PD. Without an intention to study PD, sample sizes often are not large enough to permit analyses of PD specifically. For example, another 8 empirical articles published since 2017 included "purging disorder" as a key word, but analyses collapsed across all forms of OSFED due to insufficient sample

sizes. Similarly, published RCTs including PD participants analyzed outcomes in an expanded definition of BN (23–26). This approach is consistent with revisions to the most recent edition of the International Classification of Diseases (ICD-11) published in June of 2018, in which binge eating is defined as: “a distinct period of time during which the individual experiences a subjective loss of control over eating, eating notably more *or differently* than usual, and feels unable to stop eating *or limit the type or* amount of food eaten.” (emphasis added;(27)) The new definition of binge eating absorbs much, but not all, of PD into a broader definition of BN because many individuals with PD experience some loss of control over eating prior to purging despite not having eaten an objectively large amount of food (28). This may not critically impact prevalence estimates for PD because many studies have defined the disorder as the presence of purging in the absence of any self-reported binge eating. However, it is likely to impact the composition of samples with ICD-11 BN and make them different from samples with DSM-5 BN. Based on findings in the current review, increased heterogeneity of “BN” samples may slow research advances for BN and could largely eliminate research advances for PD. Hopefully, the field will recognize that such a move is premature and continue to favor the DSM-5 definition of eating disorders, which permits examination of PD as an other specified eating disorder. To truly advance research on PD, more effort should be placed in designing adequately powered studies to specifically test hypotheses regarding risk and maintenance factors that could lead to improved interventions. Moving forward, treatments may need to acknowledge that those with PD have histories of elevated weight in childhood and adolescence and that purging may be an effective but very harmful means of weight control for these individuals because this likely influences distress and motivation to seek treatment. Treatments should acknowledge that individuals with PD experience a physiologically distinct response to eating that increases stomach ache, nausea and desire to vomit after eating amounts of foods that others find tolerable. Finally, treatments should acknowledge that purging in PD does not follow large binge episodes and tackle purging directly rather than relying on therapies that count on purging ceasing after the ego-dystonic symptom of binge eating has been effectively treated. This is a lot to ask of a field, but the eating disorders field has shown an ability to accomplish a lot over a very short period of time. The key to our success seems to be ensuring that we understand and accept the challenge before us.

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Key Points

1. Purging disorder affects approximately 2.5% of adolescents each year, with a higher prevalence in girls than boys.
2. Premorbid higher BMI percentile and dieting to lose weight increase risk for developing purging disorder.
3. Women with purging disorder experience a significantly greater postprandial increase in a gut peptide that triggers satiety compared to individuals with bulimia nervosa or healthy controls, and these changes predict the elevated gastrointestinal distress reported by women with purging disorder.
4. At end of treatment and in naturalistic follow-up of a community-based sample, purging disorder or subthreshold purging disorder persists in 31% and 36% of individuals, and full remission is observed in 21% and 36%, respectively.