# Purging disorder: considerations for clinical presentation, epidemiology, risk factors and interventions

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Abstract

Purging disorder (PD), a DSM-5 other specified feeding or eating disorder, involves purging in the absence of binge eating among individuals who are not underweight. Epidemiological studies indicate that PD is more common in women than men and affects 1.3% to 4.8% of girls. Clinical presentation includes elevated body image concerns, mood disturbance and perfectionism, similar to other eating disorders. However, PD is characterised by greater somatic complaints compared to other eating disorders, including greater postprandial gastrointestinal distress compared to bulimia nervosa (BN). Studies of biological factors support greater release of postprandial gut peptides that trigger satiation and satiety in PD compared to BN, and these biological factors are linked to the clinical presentation of PD. Prospective risk factors for PD onset differ from those for anorexia nervosa onset and include higher premorbid body mass index, body dissatisfaction and dieting. Relative to its prevalence in the general population, PD is rare in eating disorder treatment settings. Treatment dropout is high in PD, with more than a third of patients discontinuing treatment early. 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. Recommendations for tailoring cognitive behavioural therapy include applying techniques for mood intolerance to address emotional and gastrointestinal distress that maintains purging in the absence of binge eating. Such efforts may benefit patients with PD as well as those who purge across eating disorder diagnoses.

Keywords: purging disorder, epidemiology, biology, risk factors, intervention, treatment, outcome

## Introduction

More than 10 years ago, the *Diagnostic and Statistical Manual for Mental Disorders, Fifth Edition (DSM-5)* included purging disorder (PD) as a named other specified feeding or eating disorder (OSFED), described by "Recurrent purging behavior to influence weight or shape (e.g., self-induced vomiting; misuse of laxatives, diuretics or other medications) in the absence of binge eating" (1). This review covers the topics of diagnosis, epidemiology, clinical presentation, biological factors, risk factors, interventions and outcomes for PD.

## Diagnostic considerations

Like all OSFED, PD can only be diagnosed among those who have a clinically significant disorder that does not meet the full criteria for anorexia nervosa (AN), bulimia nervosa (BN) or binge eating disorder (BED). Consistent with the requirement of clinically significant distress or impairment, a systematic review found that PD was associated with impaired health-related quality of life in children and adolescents (2). There is partial symptom overlap between PD and both AN and BN because purging occurs in all three. However, the overlap is partial because only PD requires the presence of recurrent purging; only AN requires the presence of low weight; and only BN requires the presence of binge eating. Thus, successful differential diagnosis requires understanding how the DSM-5 defines purging, underweight and binge eating.

Purging includes a range of behaviours that evacuate matter from the body, such as self-induced vomiting and misuse of laxatives, diuretics or other medications. An example of other medication use includes insulin omission in type 1 diabetes because insufficient insulin contributes to hyperglycaemia and excess urination. The guidelines for underweight are an adult body mass index (BMI) <18.5 kg/m2 or, for children and adolescents, a BMI <5th percentile for age and sex. Finally, a binge episode involves experiencing a loss of control while consuming a large amount of food (e.g., a package of cookies) within a limited period of time, and binge episodes are frequently associated with later feelings of guilt. Given significant variability across people and contexts in food consumption, determining whether an amount of food is definitely larger than what most people would eat can be challenging. As a rule of thumb, consumption of more than 1,000 kcal (4,184 kJ) within two hours at home and outside of special occasions is more than most people consume. Women with PD often endorse a loss of control over their eating but do not report consuming more than most people would eat under similar circumstances. In clinical interviews, women with PD reported eating approximately 500 kcal (2100 kJ), on average, when they lost control of their eating. In contrast, women with DSM-5 BN reported consuming an average of approximately 2,700 kcal (11,300 kJ) (3).

The International Classification of Diseases, 11th Revision (ICD-11) (4) does not require that binge episodes involve a large quantity of food, and the ICD-11 diagnosis of BN subsumes many cases of DSM-5 PD (5). However, cases of

DSM-5 PD in which purging does not compensate for food consumption, referred to as non-compensatory purging, would not be diagnosed as ICD-11 BN.

Beyond the boundaries set by formal diagnostic criteria for AN and BN, the DSM-5 provided no provisional diagnostic criteria for PD, and no approach was offered for differential diagnosis and diagnostic hierarchies among the OSFED examples. Consequently, characterisation of PD in research and clinical settings has varied.

### Epidemiology

Point prevalence estimates for adolescent girls have ranged from 1.3% to 4.8%, depending on age group and how PD is defined (6-9), with lower prevalence in pre- and peri-pubertal girls. Like other eating disorders, PD is more common in girls than boys. However, across population-based samples, boys make up approximately 20% of children diagnosed with PD (7, 8). Mirroring gender differences in adolescents, PD affects approximately 2% of women and 0.5% of men (10-12). PD may be less common in non-Western contexts, with one study reporting a 0.2% point prevalence in women screened in Malaysia (13).

Compared to its prevalence in non-clinical samples, PD appears to be underrepresented relative to AN and BN in clinical settings (14-19). Among outpatients, AN outnumbered PD 7:1 (18, 20), BN outnumbered PD from almost 4:1 to 6:1 (18, 20) and BED outnumbered PD almost 6:1 (20). Among inpatients, the AN:PD ratio was almost 17:1 and BN:PD ratio was almost 10:1 (19). In a large sample of 1,711 PD patients (14), males accounted for only 2.2% of PD patients – far less than what has been observed in non-clinical samples, consistent with evidence that males are less likely to receive eating disorder treatment (21).

Several factors may contribute to underrepresentation of PD in treatment. Poor recognition of illness severity, stigma and shame, lack of encouragement to seek treatment and low motivation represent barriers to treatment seeking (22). Studies support that each barrier may play a role in underrepresentation of PD in clinical settings. Fatt et al. (23) found that self-identification of a body image problem was a significant predictor of treatment seeking and examined differences across DSM-5 eating disorders on this factor. Despite using purging to influence weight or shape, just over half of girls with PD and less than a third of boys with PD described themselves as having a body image problem, suggesting poor recognition of severity, particularly for boys. Poor recognition appeared to be more common in PD than BN, where >80% of girls and >67% of boys described themselves as having a body image problem. Like AN, PD may represent a more ego-syntonic condition in which the person views use of vomiting or purgatives as a harmless weight-management strategy. Those who do not match the stereotype for an eating disorder – which primarily depicts individuals as being underweight – may be less likely to recognise themselves, or be recognised by others, as having problems that cross the threshold into being a disorder (24). Higher purging frequency is associated with greater concerns about stigma (25) and is often hidden from others out of shame, reducing the likelihood of encouragement from friends of family to seek treatment. Higher purging frequency is also associated with greater treatment ambivalence (25).

Poor detection of eating disorders in health settings represents another major barrier to care; only 10% of individuals with PD were detected and referred to treatment versus approximately 50% of those with AN or BN (26). Universal screening for eating disorders is needed to improve detection, including asking everyone about use of purging to control weight or shape (27). Finally, PD may be misdiagnosed as another eating disorder given its partial symptom overlap with AN and BN, or PD may be identified as atypical AN due to an absence of guidelines for differential diagnosis when criteria are met for both. Folding cases of PD under the umbrella of another eating disorder diagnosis raises the risk of inadequate assessment and treatment. Once a diagnosis has been assigned, clinicians may be biased by heuristics in their clinical judgements which could negatively impact prognosis (28).

### Clinical presentation and correlates

Patients with PD have an elevated drive for thinness, body dissatisfaction, problems with interoception, fears of adulthood, problems regulating impulses, social insecurity, interpersonal sensitivity, obsessive/compulsive features, depressive features and somatisation (16). Although characteristic of many eating disorders, these features were higher in patients with PD compared to those with AN, BED or atypical AN, suggesting that PD falls on the higher end of the severity spectrum (16). A recent meta-analysis supports the interpretation of PD as presenting with comparable severity to BN based on evidence of similar eating pathology, general psychopathology or physical health impairments (29). Yet, Krug and colleagues (16) found distinctions between PD and BN, suggesting that PD was different from BN without being less severe than BN. Compared to BN, Krug et al. (16) and others have found that those with PD have a later age of onset (6, 30), lower impulsivity (31, 32), higher emotional reactivity (20) and a lower frequency of vomiting (31) but use more methods of purging (33) and endorse greater somatic concerns (14). Use of multiple purging methods may reflect greater presence of non-compensatory purging in PD – that is, purging that occurs in the absence of any eating episode (34), and may be linked to elevated risk for alcohol and other substance use disorders in PD (35).

As described in Diagnostic considerations, above, women with PD often subjectively experience a loss of control while eating despite consuming an average of only 500 kcal (2100 kJ) prior to purging (36). Using an *ad lib* meal as a

| Eating disorder | CCK | GLP-1 | PYY | Insulin |
|---|---|---|---|---|
| Purging disorder | No difference from controls<br>Higher than bulimia nervosa | No difference from controls<br>Higher than bulimia nervosa | Higher than controls<br>Higher than bulimia nervosa | No difference from controls<br>Higher than bulimia nervosa |
| Anorexia nervosa | Inconclusive | Inconclusive | Mixed, but may be higher than controls | Lower than controls, but with higher sensitivity |
| Bulimia nervosa | Higher than controls<br>Lower than purging disorder | Higher than controls<br>Lower than purging disorder | No difference from controls<br>Lower than purging disorder | No difference from controls, but with reduced sensitivity<br>Lower than purging disorder |
| Binge eating disorder | Inconclusive | No difference from controls | Inconclusive | Higher than controls, but with reduced sensitivity |

**Table 1.** Summary of altered biological factors in purging disorder and other eating disorders (adapted from Williams and Keel (58))

Abbreviations: CCK = cholecystokinin; GLP-1 = glucagon-like peptide 1; PYY = peptide tyrosine tyrosine

Note: "Inconclusive" reflects a pattern of mixed results in which studies have reported levels that are significantly higher, significantly lower and levels that do not differ from controls.

behavioural measure of satiation, observed food intake was significantly correlated with self-reported food intake in a sample that included women with PD, BN and no eating disorder; women with BN consumed significantly more food compared to women with PD and controls to achieve the same subjective state of feeling full (36). After eating, both women with BN and women with PD experienced significant increases in nausea, stomach ache and desire to vomit, which was not observed in healthy women (36). Greater somatic concerns and gastrointestinal distress following intake of a normal amount of food in PD may reflect distinct disruptions in physiological responses to food intake that support distinguishing between PD and BN in the DSM-5 (37).

## Biological correlates

PD has been linked to distinct physiological responses to food intake that correspond to both the absence of binge eating and presence of vomiting after normal amounts of food (see Table 1). Women with BN have demonstrated lower cholecystokinin (CCK) responses (38) and glucagon-like peptide 1 (GLP-1) levels (39) following a standardised liquid meal compared to women with PD and controls. Given the roles of CCK and GLP-1 in producing satiation (a signal to the brain to stop eating), these differences may explain the presence of objectively large binge episodes in BN and their absence in PD. Despite consuming the same amount of food in the standardised meal, women with PD reported significantly greater increases in nausea and stomach ache after eating compared to both women with BN and healthy women (3). These increases were predicted by postprandial increases in the gut satiety peptide, PYY. 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 (3). Finally, compared to women with BN, those with PD demonstrated a significantly greater insulin response to a fixed test meal, with controls demonstrating a response that did not differ significantly from either BN or PD (40). Combined with prior findings, these results suggest unique biological correlates for PD, in which women demonstrated intact CCK and GLP-1 responses and an elevated PYY response to food intake. In contrast, BN is characterised by blunted CCK and GLP-1 responses and an intact PYY response to food intake. The unique combination of intact satiation signals and excessive satiety signalling in PD may contribute to maintenance of purging after normal amounts of food.

Keel et al. (41) recently reported that delayed gastric emptying was associated with self-induced vomiting in both PD and BN and predicted increased gastrointestinal distress after food intake (41). A single dose of 10 mg of metoclopramide effectively increased the gastric emptying rate and eliminated group differences in gastric emptying. However, metoclopramide caused increased PYY release. As a consequence, all participants experienced an exacerbation of PYY response to the test meal and associated nausea and stomach ache. Although these results provided compelling evidence that PYY causes gastrointestinal distress, findings suggest the need for caution when attempting to isolate and correct a specific physiological process within a complex system. Importantly, biological correlates of PD do not necessarily reflect maintenance or aetiological factors as they may be consequences of eating disorder behaviours.

## Prospective risk factors

A handful of studies have examined prospective risk factors for PD in community-based samples. The largest of these comes from a longitudinal study in which 14,541 pregnant women living in the Avon area of the UK were enrolled to provide data on themselves and allow data to be collected on their children, beginning at birth (42). In one paper using this sample, researchers examined BMI trajectory from birth to 12.5 years of age to examine the prospective risk of an eating disorder diagnosis at 14, 16 or 18 years of age in the children (43). PD contributed 133 of the 536 detected eating disorder cases (25%), with a 9:1 female: male ratio. Prior to PD onset, girls (by age 5) and boys (by age 6) demon-

strated a significant increase in BMI percentile compared to those who did not develop eating disorders. For children who went on to develop BN, BMI percentiles increased and diverged significantly from non-eating disorder controls as early as 2 years of age. 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 eating disorders (43). Findings support that factors driving differences in eating behaviours and weight at a young age set the stage for differences in the central features of the various eating disorders. It also suggests how a higher premorbid weight may buffer those with PD against becoming underweight when they purge without bingeing.

Stice et al. (30) collapsed data across three separate school-based prevention trials of high-risk adolescents and young adults, resulting in a sample of 1,272 female participants who endorsed body image concerns. Body dissatisfaction appeared to be a common risk factor across PD, BN and BED, but overeating emerged as a predictor of disorders characterised by large binge episodes and did not uniquely contribute to risk for onset of PD or AN (30). Instead, more frequent dieting predicted PD onset and emerged as the first risk factor in analyses determining unique pathways for developing PD (44). Findings indicated highest PD risk in girls who dieted more, held more positive thinness expectancies and experienced elevated but not extremely high negative affect (44). Similar to findings from children living in Avon, lower BMI was a unique branch for developing AN.

Children who go on to develop PD may not experience the same drive to consume large amounts of food that give rise to the very early elevations in BMI seen in BN and BED but may, nonetheless, gain more weight during childhood than their peers. In cultures that idealise thinness and stigmatise higher body weights, these children may turn to dieting with the belief that this will make both their bodies and lives better. When dieting does not produce the desired effects, feelings of ineffectiveness and negative mood may provide the nudge to try purging. Purging produces dehydration which, in the short run, creates the illusion of weight loss. In addition, findings from an ecological momentary study of PD indicated that purging alleviates negative affect (45). Thus, purging may be maintained as a strategy to control weight and mood.

Risk factors shared between PD and other eating disorders suggest that existing eating disorder interventions may be effective for PD. Risk factors that are specific to PD offer insights into how these interventions may be tailored to meet the specific needs of those with PD.

### Interventions and outcomes

Limited information exists to guide the prevention and treatment of PD. However, most research suggests that targeting risk and maintenance factors shared across eating disorders provides a good starting point. A brief intervention to reduce internalisation of the thin ideal produced a statistically and clinically significant reduction (62%) in risk of PD onset in samples combined from three randomized controlled trials (RCTs) (46). The intervention also significantly reduced risk for subthreshold BN/BN onset, but not subthreshold/full threshold AN or BED over three-year follow-up. Such findings support a more central role for thin ideal internalisation in risk for PD and BN, compared to AN and BED.

Several other RCTs have included PD along with patients with BN or subthreshold BN (47-50) but did not include enough participants with PD to report their outcomes separately. Findings from larger case series of patients receiving cognitive behavioural therapy (CBT) suggest caution in generalising findings from BN to PD. Tasca et al. (17) reported outcomes for patients evaluated in a tertiary care centre, including 122 with PD and 415 with BN, among whom 25 with PD (20%) and 126 with BN (30%) were referred for CBT in their day program. In addition to a lower likelihood of referral for treatment, PD was associated with higher dropout compared to BN (37% vs. 20%) and lower likelihood of good outcome (48% vs. 57%). Similar patterns emerged for patients treated at a specialised eating disorders unit. Riesco et al. (51) described treatment outcomes in 57 patients with PD, and Fernández-Aranda et al. (52) described outcomes for 484 patients with BN over the same period from the same centre in an online supplement. Dropout was higher in PD compared to BN patients (37% vs. 28%) and full remission was lower in PD compared to BN patients (18% vs. 25%). Although direct comparisons between PD and BN were not presented, the pattern of differences points to less favourable outcomes for PD.

Preliminary findings indicate that more effort should be placed on tailoring treatments for the unique configuration of symptoms of PD, based on what we have learned thus far. Treatments should address weight history and that purging in the absence of binge eating may contribute to weight suppression – that is, maintenance of weight that is lower than the highest premorbid weight. In the context of CBT or enhanced CBT (CBT-E), prescription of a normal pattern of eating and abstinence from purging may produce rapid weight gain which could contribute to the increased dropout observed in PD. Greater emphasis on weight and shape concerns earlier in treatment using cognitive dissonance techniques may facilitate greater acceptance of treatment by prioritising physical and emotional health over weight. 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. Krug et al. (16) recommended the mood intolerance module of CBT-E to help PD patients accept and tolerate feelings of excess fullness and anxiety. Combined with exposure and response prevention techniques successfully employed in a case study using CBT-E for an adolescent with PD (53), adaption of mood intolerance to address intolerance of

gastrointestinal distress represents a novel strategy to tailor CBT to PD.

Keel (54) summarised outcomes from 11 follow-up studies that included data from a combined total of 943 individuals with PD with duration of follow-up ranging from just over two months to 10 years. Overall, 40% had no eating disorder at follow-up. Approximately 20% had PD, and another 20% continued to purge at a reduced frequency or to exercise excessively in the absence of binge eating, suggesting that 40% would fall within a broad phenotype for PD. Finally, 20% had BN and virtually no cases of AN or BED were found. Studies consistently supported a greater likelihood of persistence of PD versus crossover to BN or any other eating disorder, suggesting that PD is not a prodromal stage of BN in most cases.

Two studies included in Keel's (54) summary reported outcomes for patients with PD at five or more years following intake. At five-year follow-up of inpatients, Koch, Quadflieg, and Fichter (15, 55) reported that 5% of patients with PD had died compared to 3.7% of purging AN patients and 1.1% of purging BN patients, with significantly higher mortality in PD and AN compared to BN. The 5% crude mortality rate in PD reflected a standardised mortality ratio of 3.9 (95% confidence interval: 2.1–7.2) or a nearly four-fold increase in risk of premature death associated with PD. Where cause of death could be determined, medical complications from the eating disorder or suicide accounted for all but one (55). PD patients were significantly older and had a significantly longer duration of illness at admission compared to purging patients with AN or BN, suggesting that potential delays in recognition, referral or treatment may have contributed to worse outcome. At follow-up, approximately 40% had no eating disorder diagnosis (15).

Forney et al. (56, 57) reported long-term outcomes of PD in a sample of 84 women followed for approximately 10 years. No participants died during the follow-up period. Full recovery was achieved in 30% of the sample (56). Women were significantly more likely to continue to have PD than to cross over to BN (57). Greater concerns with weight or shape predicted illness maintenance, and quality of life was impaired in physical, psychological and social domains (56).

A notable feature of these two longer follow-up studies is that full remission ranged from 30% to 40%, closely matching the estimated 40% remission collapsed across all follow-up studies of PD, including those of much shorter duration. This suggests minimal improvement in outcomes over extended periods of time, highlighting the need for earlier and better interventions.

## Conclusion

PD is a serious eating disorder characterised by the use of vomiting, laxatives or other medications to influence weight or shape in the absence of binge eating in individuals who are not underweight. The disorder affects a substantial number of people, most of whom do not receive treatment. Clinical presentation includes significant disturbances in mood, personality and interpersonal function, with PD falling on the more severe end of the spectrum of eating disorders, along with BN. Women with PD report unique problems with somatic symptoms, including increased gastrointestinal distress, and these features are linked to delayed gastric emptying and elevated release of gut peptides following food intake. This, combined with a premorbid history of elevated weight, may negatively impact acceptability of CBT in PD. Tailored treatment approaches could involve addressing overvaluation of weight and shape earlier in treatment, adapting modules for mood intolerance to address tolerance of gastrointestinal distress, and employing exposure and response prevention to help patients with PD consume normal amounts of food without purging. A minority of those with PD report eating disorder remission in follow-up studies. Eliminating barriers to treatment and adapting interventions for PD could improve outcomes. Finally, addressing purging as a primary symptom in treatment may benefit patients who purge in the context of other eating disorders, helping many patients for whom the gold standard of treatment does not work.

### Funding and declarations of interest

The author receives royalties from Oxford University Press for the following titles: *Eating Disorders, Second Edition (2017); The Void Inside: Bringing Purging Disorder to Light (2020).*

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