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Why Eating Disorders Return: A Clinical Review of Habit Learning, Relapse, and Recovery

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Abstract

Eating disorders are often described in terms of their symptoms; however, what defines them clinically is that their symptoms persist and return. Despite the awareness, motivation, and symptom-focused treatment individuals receive, eating disorder behaviors frequently recur, showing that existing models are not capable of comprehensively explaining why these behaviors are so challenging to change. This narrative review approaches eating disorders from an action-control perspective, separating goal-directed and habit-like forms of behavior. Using this framework, restriction, binge eating, and compensatory behaviors are viewed as learned action patterns. This review integrates research theories, transdiagnostic models of compulsivity, and clinical outcome studies to explain why eating disorder behaviors are challenging to modify and prone to relapse. Clinical findings related to ambivalence, limits of volitional control, relapse patterns, comorbidity, and generalizations in treatment demonstrate why short-term, symptom-based treatment methods are insufficient for durable change. Viewing eating disorders as habitual behaviors is central to relapse prevention, long-term monitoring, and identifying behaviors, context, and clinical factors that need to be addressed in treatment.

Keywords: *Eating Disorders; Behavioral Persistence; Habit Learning; Relapse; Action Control; Treatment Resistance*

1. Introduction

Long-term outcome research shows that recovering from eating disorders does not have a simple path from illness to recovery, because patients recover, remain ill, or cycle between improvement and relapse over years. Periods of improvement do not mean the underlying issues have been resolved. In clinical practice, many individuals leave treatment prematurely because they believe they have recovered, then return to similar eating disorder patterns. Most models focus on symptom, belief, and motivation aspects. Although these aspects are practical in describing the experience and creating a path for treatment, they do not adequately explain why eating disorders persist once they are established. Many individuals know the risks of the eating disorder behaviors but struggle to stop pursuing them. The gap between insight and action arises because knowing that a behavior is harmful does not guarantee that the individual can act on that knowledge. Walsh highlights this idea by stating that weight restoration alone does not prevent relapse (Walsh, 2013). This review addresses this gap by portraying eating disorders as disorders of behavioral persistence: learned behavioral patterns rather than only matters of belief or intention. This paper explains persistence in terms of action control characteristics and uses this framework to examine relapse, maintaining mechanisms, comorbidity, and limits to treatment generalization. Implications for long-term care and relapse prevention are discussed.

2. Methodology

This review uses a narrative style to combine evidence on behavioral persistence in eating disorders from an action control framework. Literature was searched through PubMed, PsycINFO, ResearchGate, and Google Scholar, with backward citation searches between January and February 2025. The search terms were primarily related to eating disorders, relapse and recurrence, habit learning, goal-directed control, compulsivity, and treatment generalization. Articles were chosen if they were peer-reviewed empirical studies or major theoretical papers that successfully explained mechanisms of persistence or long-term results. Articles were prioritized if they

provided relevant evidence on action control processes, relapse patterns, or how well treatment findings transfer to real-life clinical care. Rather than an exhaustive review, selection prioritized conceptual coverage and theoretical relevance.

3. Literature Review

Persistence and the Limits of Symptom-Based Models

Results from long-term research show that some patients recover, some remain diagnosed, and many experience recurrence, even if they have periods of improvement (Keel & Brown, 2010). They report that anorexia nervosa remission ranges from 29% at short follow-up to 84% at long-term follow-up, with continued anorexia nervosa (AN) present in up to 18% of inpatient samples and crude mortality up to 8%, bulimia nervosa shows remission rates above 70% by 10 years, but roughly 30% of patients remain chronically ill when related eating disorder not otherwise specified (EDNOS) cases are included, and approximately 25% of bulimic-spectrum EDNOS cases follow a chronic course (Keel & Brown, 2010). Current models describe eating-disorder behaviors and the underlying beliefs, but they do not explain why these behaviors persist once they begin. The continued presence of eating disorders is a serious challenge for theories of maintenance. For example, weight regain during anorexia nervosa treatment does not make the eating disorder behaviors disappear, which can lead the person to fall back into the disorder (Walsh, 2013).

A lack of understanding or effort cannot explain the behaviors and thoughts, because many individuals recognize that their behavior is risky and they want to change; however, they still find themselves stuck in the same patterns. Over time, eating-disorder behaviors get stronger and more complex, which makes it hard to change, especially when they involve strict rules and repeated actions (Klein & Walsh, 2004; Walsh, 2013). By examining these patterns, it becomes clear that the issue is not confusion. As a result, both clinical observations and long-term results indicate that learning is a valuable means of understanding why the disorder persists. Eating-disorder behaviors that occur repeatedly can develop into learned patterns that become stronger over time, especially when they provide a sense of pleasure or short-term relief (Klein & Walsh, 2004; Walsh, 2013).

Goal-Directed and Habitual Control

Control systems that change are different in how actions are learned, selected, and represented. Goal-directed and habitual systems are parallel to each other and compete for control over behavior, determining their relative influence according to changes in learning history, environmental stability, and task demand (Yin & Knowlton, 2006; Graybiel, 2008).

Similar behaviors can occur even though they have fundamentally different underlying reasons at various points in a person's life. Goal-directed behaviors are linked to both the action outcome and the value expected from the outcome. Actions are chosen according to what is believed to be the desired consequence, making the behavioral tendency adjust to the outcome value (Balleine & O'Doherty, 2010).

This indicates that the action is based on what is expected to happen, rather than being automatically elicited by the situation. Thus, flexibility is an essential feature of goal-directed control because actions are chosen. When control shifts toward the habit system, however, the link between action and outcome weakens, and goal-directed responding declines even when the physical demands of the task remain constant (Yin & Knowlton, 2006).

Goal-directed behavior necessitates a precise understanding of the relationship between actions and outcomes, instead of relying on stimulus-response learning or simple repetition, which allows goal-directed control to make the behavior adjust according to the demands of a changing environment. At the same time, goal-directed actions require mental effort because selecting an action based on anticipated outcomes requires combining information about the action and the value of the outcome. The strength of the relationship between action and outcome depends on current goals and requires the use of working memory, planning, and executive control systems (Rangel et al., 2008).

From a neural perspective, this type of control requires associative corticostriatal circuits. This involves the prefrontal cortex and dorsomedial striatum, which are essential areas for the sensitivity to outcome devaluation and contingency degradation (Yin & Knowlton, 2006; Balleine & O'Doherty, 2010).

As a result, goal-directed control is vulnerable. Because it requires ongoing evaluation, especially in situations where excessive training or highly stable environments are present, these situations can cause behavior to shift toward a more automatic form of control (Graybiel, 2008). This distinction is clinically relevant because behaviors that shift from goal-directed to habitual control become less sensitive to outcomes, making them harder to modify even when goals, beliefs, or motivation change.

Table 1 summarizes the main behavioral features, experimental models, and control systems that separate goal-directed action from habit-like responding, providing a helpful guide for understanding how eating disorders could lose sensitivity to outcomes and become less sensitive over time.

Table 1. Goal-Directed vs Habitual Action Control: Core Features and Circuit

Feature	Goal-directed control (A–O / R–O)	Habit-like control (S–R)	Connection to ED persistence
Associative Structure	Actions are chosen because of the expected outcome	Behavior triggered automatically by cues	Knowing it is harmful but still doing it.
Outcome Devaluation	Outcome losing value stops the behavior	Behavior continues despite devaluation	Changing value ≠ stopping behavior.
Contingency Degradation	Behavior stops when it is no longer working	Behavior persists even when ineffective	Consequences lost meaning.
Effect of Repetition	Behavior remains flexible	Repetition makes behavior automatic	Repetition strengthens routine.
Key Circuits	Prefrontal cortex and dorsomedial striatum	Dorsolateral striatum	Persistent behaviors are often driven by learned routines rather than current goals.

Note. A–O = action–outcome; R–O = response–outcome; S–R = stimulus–response.

Habit Bias and Compulsivity

Different disorders, such as substance use disorder, obsessive-compulsive disorder, and related compulsive syndromes, have similar behavior patterns of repetition, continuity, and lack of sensitivity to the outcomes (Voon et al., 2015; Godier & Park, 2014). In all of these disorders, the behaviors continue even in cases where the relationship between the action and its outcomes changes, or negative interactions with consequences occur. This shows that compulsivity is connected to disruptions in the process, in actions being selected and done, rather than being a consequence of the specific beliefs or the context of the disorder itself. This pattern reflects a systematic reliance on stimulus-response habits rather than goal-directed action selection, even when flexible options are available. In the contemporary action control framework context, this pattern is perceived as habit bias (Everitt & Robbins, 2005; Voon et al., 2015). From this perspective, habit bias explains the control mechanisms of behaviors, focusing on the underlying mechanisms rather than simply addressing symptom severity or motivational failures. Therefore, compulsive behavior intensifies in situations where dominant action overrides goal-directed control. This is clearest in substance use disorder, where repeated drug use shifts action-outcome learning toward stimulus-response control, making behavior insensitive to manipulations such as outcome devaluation and punishment (Everitt & Robbins, 2005). On a similar note, obsessive-compulsive disorder also has impaired goal-directed control and excessive reliance on habits, making persistent responses even though the action is no longer needed (Gillan et al., 2016). Together, these findings show that obsessive-compulsive disorder has a shared behavioral phenotype with other compulsive disorders, rooted in action-control imbalance across diagnoses (Robbins et al., 2012; Voon et al., 2015). Two factors frequently implicated in habit dominance across compulsive conditions are repetition and stress. Excessive repetition makes stimulus-response associations stronger, which increases how automatic the behavior is and reduces how sensitive it is to outcomes. Similarly, stress extends this bias by shifting control toward habitual responding and weakening goal-directed processes (Everitt & Robbins, 2005; Voon et al., 2015). These factors in these different disorders make the case for habit-based models a strong explanation for behavioral persistence. This supports shared learning mechanisms without implying the disorders are identical. These models are related but not interchangeable. Addiction involves escalating reward through dopaminergic systems, obsessive-compulsive disorder involves avoidance learning, and habit involves stimulus-response stamping. The claim here is the narrow one that eating disorders share the imbalance between goal-directed and habitual control, not the broader features of any single model.

Eating Disorders as Persistent Learned Behaviors

What keeps eating disorders going is the repeated eating behavior itself. In different diagnostic systems, eating disorders form their own diagnostic category, defined by behavioral patterns such as binge eating, restriction, and

compensatory behaviors (Walsh, 2011; Keel et al., 2011). In discussions of the Diagnostic and Statistical Manual of Mental Disorders (DSM), the proposed DSM-5 criteria aimed to reduce reliance on EDNOS (Keel et al., 2011). However, EDNOS remained the most common diagnosis in the samples studied (Keel et al., 2011). This shows that eating pathology commonly presents as a behavioral pattern and does not mean it fits into one category (Fairburn et al., 2003; Keel et al., 2011). This is also supported by transdiagnostic models, since they share the same maintaining processes for different diagnoses (Fairburn et al., 2003). Sharing a control system is not the same as sharing a learning process. Bingeing is driven by appetitive reinforcement while restriction is driven by distress reduction. The claim is that both converge on the same outcome, which is cue-driven behavior insulated from goals, rather than that they arise from the same reinforcer. Eating disorders start as goal-directed behaviors but can transfer into more automatic forms of control (Steinglass & Walsh, 2006; Walsh, 2013).

In anorexia nervosa, restriction is the dominant behavior. Walsh describes it as starting with minor restrictions that might look like innocent restrictions, but they could become something bigger and dangerous that is resistant to change, like a maladaptive habit (Walsh, 2013). This happens when the behavior turns into something that is stimulus-response-based and less sensitive to outcomes, becoming something that could even get triggered by cues, even in situations where pursuing that outcome is neither rewarding nor helpful (Steinglass & Walsh, 2006; Walsh, 2013). This could also be supported by neurobiological models, which suggest that altered reward processes and strong top-down control could exacerbate ongoing restrictions, even if they carry the risk of leading to starvation, and the dangers are apparent to the individual (Kaye et al., 2013; Walsh, 2013).

Most of the individuals understand the risks it causes, but they still pursue the action because of the short-term reinforcement described as a mechanism that reduces anxiety. At the same time, hunger and dietary restriction have more than a physical dimension, because they fulfill psychological needs (Kaye et al., 2013). People can feel deprived because they are not eating what they want, even when they are eating as much as they need, because the feeling is tied to the psychological reward system rather than to actual calorie intake. When these restrictions are constantly used with short-term relief or as a control mechanism, these behaviors become stable. Moreover, the behavior turns into a routine that keeps coming back (Walsh, 2013; Kaye et al., 2013). One objection to the habit account is that restriction never loses its value for the patient, which should keep the behavior goal-directed rather than habitual. The answer is that the habit is not the pursuit of thinness but the anxiety-reduction cycle around eating: restriction lowers food-related distress, that relief reinforces the behavior, and it becomes bound to mealtime cues (Steinglass & Walsh, 2006; Kaye et al., 2013). The patient keeps valuing thinness while the restrictive behavior itself runs on automatic distress-relief loops.

Fairburn's cognitive behavioral model describes binge eating as a process where an individual has strict dieting rules, and when they even have a tiny slip-up, it makes them feel like they failed their diet, which makes them follow the cycle again and again, and these behaviors cause fear of weight gain which makes the unhealthy cycle keep going (Fairburn et al., 2003). Similarly, Berner and Marsh link the compensatory behaviors in bulimia nervosa to dysfunction in dorsal frontostriatal circuits, impaired self-regulation, and habit learning involving reward-related circuits, which may explain why binge-purge behaviors become difficult to break over time (Berner & Marsh, 2014). Similar binge-eating behavior can occur without deprivation. For instance, when access to preferred food is restricted, the resulting deprivation and cue-triggered motivation can drive binge patterns (Lowe & Levine, 2005). Therefore, understanding behaviors correctly is vital, because patterns such as restricted food choices and excessive food consumption are linked to relapse and worse long-term results (Steinglass & Walsh, 2006; Walsh, 2013).

Looking at this with the habit learning aspect of anorexia nervosa, it could be said that behaviors themselves can potentially turn into disorders. Over time, these learned routines are explained by frontostriatal systems and learning processes, rather than by a lack of motivation or knowledge, grounding the account empirically (Berner & Marsh, 2014; Walsh, 2011).

Table 2 uses action-control frameworks to explain eating disorders, where actions that were goal-directed could become habit-like patterns, even if they do not align with the person's conscious intentions or desired outcomes.

Table 2. Eating-disorder behaviors as persistent learned action patterns

Repeating behavior	Early function (goal-directed)	Later pattern (habit-like)	Clinical Conclusions
Restriction	Thinness, weight loss, and control are priorities	Persistent, cue- and stress-sensitive restriction	Consistent restriction, knowing the harm
Binge Eating	Managing distress or breaking diet rules	Cue-driven eating episodes	Relief reinforces the cycle
Binge–Purge Cycles	Compensating for perceived overeating	Ritualized cycle triggered independently of the binge	Purging persists after its original purpose fades
Compulsive Exercise	Managing anxiety, weight, and mood	Cue-driven routine that continues despite injury or exhaustion	Exercise is maintained by relief, not goals

Awareness, Ambivalence, and Limits of Volitional Control

Clinically, eating disorders are characterized by ambivalence rather than simple resistance: wanting recovery does not necessarily mean wanting to give up the meaningful experiences they perceive as valuable in the disorder (Williams & Reid, 2010). In anorexia nervosa, individuals described these restrictive behaviors as providing a sense of control, security, and emotional regulation, even though they understood the physical damage these behaviors cause (Serpell et al., 1999), which suggests that these experiences can become coping mechanisms and a source of identity, instead of being viewed as unwanted behavior. This shows individuals could fear weight gain, loss of control, or emotional exposure, even though they can understand the long-term health risks this illness causes (Williams & Reid, 2010). As a result, this suggests that motivation is a dynamic and content-dependent state that changes over time, rather than remaining constant (Manwaring et al., 2021).

These internal conflicts indicate that knowledge does not always translate into action, even in cases where the individual wants to recover by planning treatment goals and expressing their desire to do so. They still may not act on those intentions when they have to make weight-related decisions or deal with emotional distress (Vitousek et al., 1998). Clinical work with treatment-resistant eating disorders reflects this by stating that awareness and agreement exist in situations where behavior change stays limited (Vitousek et al., 1998). This gap is caused by the difficulties of taking control over behavior in scenarios where there are high emotions, rather than problems with understanding.

Expressing motivation does not guarantee behavioral change. Clinical outcome data show that relapses commonly occur even after symptom remission, indicating that knowledge and motivation alone are insufficient for durable change (Keel et al., 2005; Waller, 2012). For example, in anorexia nervosa, lower motivation after treatment and declining motivation during follow-up are associated with higher relapse risk (Carter et al., 2012). These findings show that volitional control weakens outside acute treatment phases, contributing to vulnerability even when motivation is present (Waller, 2012).

Relapse as a Predictable Feature of Eating Disorders

Relapse is better understood as part of the illness course than as treatment failure. In a prospective follow-up of adults with anorexia nervosa, 41% of the patients relapsed during the 1-year follow-up, with the highest risk between 4 and 9 months post-treatment (Carter et al., 2012). Seven percent of participants met criteria for both weight relapse and binge-purge relapse, and a significant proportion of those who did not relapse maintained only marginal weight at 1-year follow-up (Carter et al., 2012). This pattern suggests that relapse risk is concentrated in a limited post-treatment window rather than distributed evenly across the course of recovery.

A similar perspective also works for understanding bulimia nervosa (BN) and binge eating disorder (BED). For example, in a five-year community prospective study, the results showed high amounts of instability since each year about one-third remitted and one-third relapsed. Moreover, many individuals met criteria for a clinically severe eating disorder at each assessment point. By contrast, binge eating disorder in the same study showed better

outcomes, with greater improvement and fewer relapses at follow-up (Fairburn et al., 2000). Relapse risks also occur within a broader psychiatric context. For example, Axis I comorbidity was common in treatment-seeking samples for anorexia nervosa and bulimia nervosa, with rates of 73% in anorexia nervosa, 60% in bulimia nervosa, and 82% in mixed presentations (Herzog et al., 1992). From a long-term result perspective, psychiatric comorbidity predicted a poorer outcome for both binge-eating disorders and purging-type bulimia nervosa (Keel et al., 2005). The literature on anorexia nervosa reported a recovery rate of less than half on average, and close to 20% remaining chronically ill with symptoms such as vomiting, bulimic symptoms, and purgative abuse (Steinhausen, 2002).

Context, Treatment Generalization, and Real-World Care

A persistent question in this literature is whether treatments developed under tightly controlled research conditions translate into routine clinical settings. Most treatment trials are conducted with tightly structured protocols, including careful sample screening and extensive monitoring. While it is true that these tools improve treatment outcomes in the study, they may also overestimate the effectiveness of these treatments when applied to real-life situations that we encounter in everyday practice. Participation and retention problems are essential to consider here, since adults with anorexia nervosa show high rates of treatment refusal and dropout, with serious consequences for generalizability. Some authors have argued that randomized trials may be premature if these problems are not addressed directly (Halmi et al., 2005). Even when trials are completed, patient behavior diverges from what the trial conditions captured. Comorbid conditions often complicate treatment, especially when chronic disorders require additional care, making it harder to observe how well the treatment effects go (Gowers et al., 2007). However, it would be wrong to ignore the context because most of the interventions are done according to stepped or transitional care aspects, instead of isolated patient treatments. In the post-hospitalization study, patients entered the trial only after regaining at least 90% of their ideal body weight during structured inpatient care, so both treatment arms included medical monitoring alongside either cognitive behavioral therapy (CBT) or nutritional counseling, and the results may generalize only to similar settings (Pike et al., 2003). The findings may therefore not reflect outcomes for patients entering routine outpatient care. Similarly, findings from the ANTOP trial show no substantial evidence that any of the three treatment arms outperformed the others (Herzog et al., 2022). In that trial, body mass index (BMI) was the primary outcome. The authors noted, though, that BMI captures only one aspect of recovery. This matters because BMI does not capture psychological functioning or daily-life outcomes (Herzog et al., 2022). These findings matter for the habit model because they show that standard treatment often fails to disrupt the cue-driven nature of the behavior, allowing the habit to persist even when weight is restored. Clinician training, experience, and treatment structure also play a role. It is also essential to consider how far these findings generalize beyond the settings in which they were produced. In binge eating disorder, group CBT and group interpersonal psychotherapy (IPT) are typically given in a centralized and structured format (Wilfley et al., 1993). Some trials combining medication with CBT have focused on binge abstinence at post-treatment (Grilo et al., 2005). Long-term follow-up findings also depend on the treatment setting, since the type of care received shapes outcomes (Wilson et al., 2002). For example, in adolescents treated in intensive specialty programs, outcomes at follow-up were poorer than those seen in typical treatment settings, which may reflect the intensity of the treatment rather than its effects (Strober et al., 1997). Finally, qualitative research indicates that how patients experience treatment depends heavily on the quality of care and on contextual factors, which makes standardization difficult. Patients' descriptions of psychodynamic psychotherapy for bulimia nervosa and of the therapeutic relationship shape how treatment is understood and experienced, which limits generalization (Poulsen et al., 2010).

Clinical Consequences for Long-Term Management and Relapse Prevention

Long-term follow-up data show that relapse and chronicity remain risks across 10-15 years, and that recovery trajectories vary by patient and by how recovery is defined (Strober et al., 1997). Diagnostic crossovers support this. Crossovers between anorexia nervosa and bulimia nervosa are common and typically occur within the first 5 years of the illness (Tozzi et al., 2005). These patterns suggest that traditional symptom-based treatment approaches may fail to capture the underlying vulnerability, even when short-term results appear favorable. Comorbidity is not incidental; it is integral to the disorder and elevates relapse risk (Herzog et al., 1992). Chronic course and poor outcome are also documented in anorexia nervosa follow-up cohorts (Bulik et al., 2000). In bulimia nervosa, roughly 1 in 10 patients continues to binge and purge for longer than a decade (Keel et al., 1999), and follow-up studies after inpatient treatment show that symptom status can remain unstable over time, indicating vulnerability to relapse comparable to that in anorexia nervosa (Zeeck et al., 2011). In binge eating disorder, comorbidity is associated with greater binge frequency and broader psychopathology (Grilo et al., 2009). Taken

together, these findings indicate that insight, symptom reduction, and short-term treatment gains should not be equated with durable recovery. Even when weight and core symptoms improve, destructive eating patterns and general impairment remain clinically relevant (Sysko et al., 2005). Moreover, comorbid psychiatric and medical conditions may compound poor outcomes (Patel et al., 2018). Relapse prevention may therefore need to focus on long-term monitoring and realistic planning to make treatment sustainable. If habits are the mechanism, this points to specific interventions: cue exposure and inhibitory training, practicing restored eating across varied contexts to prevent context-specific learning, and habit-reversal techniques (Steinglass & Walsh, 2006). Clinical predictions made at discharge also warrant attention (Fichter et al., 2008). Reimagining eating disorders as a persistent, high-relapse syndrome could affect the prognosis, relapse prevention, and clinical expectations directly by creating an environment for treatment that focuses on long-term management instead of limited-time solutions.

Table 3 combines long-term clinical results and findings to explain the need for sustained clinical monitoring by highlighting relapse risk, comorbidity, and instability over time.

Table 3. Clinical consequences for long-term management and relapse prevention

Long-term patterns	What do the data show?	Why does this matter clinically?
Long periods of illness	Some individuals remain ill for 10 to 15 years, though others recover	Early improvement does not mean full recovery
Specific time relapse risk	Relapse risk is highest shortly after discharge	Follow-up care should focus on the post-discharge period
Outcome instability in BN	Symptom status remains unstable at follow-up	Ongoing assessment is needed, not one-time outcome checks
Ongoing binge–purge behavior	About 1 in 10 still binge and purge for over a decade	Long-term symptom monitoring should continue past treatment
High rates of comorbidity	Mood, anxiety, and substance-use disorders are common	Comorbidity worsens prognosis and raises relapse risk
BED severity and comorbidity	Comorbidity causes impairment and worse symptoms	Relapse prevention should focus on more than eating behaviors
Limits of symptom-based outcomes	Weight or symptom change misses daily functioning	Recovery should require functional and behavioral change
Poor predictions at discharge	Clinicians struggle to predict long-term results	Structured monitoring after discharge should be used

4. Future Directions

Future studies should use persistence models to develop clinically measurable indices. First, transdiagnostic longitudinal designs should track symptom progression, impairment, diagnostic crossover, and relapse over time. These studies should examine persistence beyond symptom counts. Cue sensitivity and behavioral rigidity, in which the same cues, routines, or decisions resurface under stress, should be measured directly. Second, outcome definitions should not rely on disorder features alone, because weight restoration achieved in treatment does not mean the individual has recovered, nor does it capture functional impairment or daily-life difficulties. Future work should therefore develop outcome criteria that integrate symptoms, functioning, and behavioral mechanisms to

determine whether the behavior becomes less cue-driven over time. Research should assess treatment effectiveness in routine clinical settings. Third, relapse prediction should target the early post-discharge period. Methods to identify high-risk situations in real settings, such as combining repeated assessments with ecological momentary assessment, should be employed. Finally, comorbidity-informed models should be used to determine whether specific patterns of co-occurrence alter the learning and relapse process.

5. Discussion

The framework helps explain why eating disorder persistence cannot be attributed solely to beliefs, insight, or symptom severity. Repeated performance of the same behavior in stable environments can shift control toward habit-like responses. This habit bias has been described in different compulsive syndromes. It explains why restriction, bingeing, and purging turn into routines and inflexible behaviors rather than flexible decisions, since eating disorders are not purely a matter of beliefs and attitudes but of learned automatic action patterns (Everitt & Robbins, 2005; Voon et al., 2015; Godier & Park, 2014). Wanting to recover can coexist with thoughts that sustain the eating-disorder behavior, such as concerns about emotional regulation or a sense of control (Serpell et al., 1999; Williams & Reid, 2010).

Patients may express a desire to recover; however, they might not be able to act on their intentions in emotionally intense situations, indicating a limitation in volitional control rather than a lack of understanding or motivation (Vitousek et al., 1998). Motivation can also change over time and is not a reliable predictor of lasting behavioral change. Given these outcomes, relapse is not unexpected (Manwaring et al., 2021).

Results from long-term studies show instability in symptom patterns, high rates of recurrence, diagnostic crossover, and persistence in binge-type behaviors (Keel & Brown, 2010; Zeeck et al., 2011; Tozzi et al., 2005). Outcomes from intensive specialty settings may not generalize to routine outpatient treatment (Strober et al., 1997). Qualitative research further supports this by showing that the therapeutic relationship and treatment context influence how interventions are experienced, limiting the direct transfer of standardized protocols (Poulsen et al., 2010). The habit account is not without limits. Evidence for habit-based control in eating disorders comes largely from laboratory paradigms and theoretical proposals rather than direct measurement of habitual behavior in patients, and Steinglass and Walsh (2006) originally framed the account as a hypothesis rather than a demonstrated mechanism. It is also worth noting that family-based treatment produces good outcomes in adolescent anorexia nervosa, which sits uneasily with a framework emphasizing entrenched automatic control. It also remains unclear when the shift from goal-directed to habitual control occurs, since the elevated relapse risk in the months after discharge is difficult to reconcile with a process that requires extended repetition to consolidate.

Finally, there are significant limitations in the evidence base, since most of the research has focused on anorexia nervosa, leaving other diagnoses underrepresented. Definitions of recovery vary across studies. Moreover, the usual outcome markers, such as weight gain or short-term symptom reduction, do not always translate into durable functional recovery. However, the overall conclusion is consistent: symptom reduction by itself is not enough for permanent recovery. Treatment has a higher chance of achieving durable recovery when it includes long-term monitoring and relapse prevention focused on the cues and situations that trigger learned eating disorder patterns.

Conclusion

In conclusion, defining eating disorders by their symptoms is not enough, and there should be more focus on behavioral persistence and symptom duration because an individual having awareness, motivation, and symptom-focused treatment does not translate into full recovery in today's existing treatment models. Eating disorders do not follow a straightforward path to recovery because the behaviors become entrenched automatic patterns that resist change even when insight is present. In the long term, restriction, bingeing, and purging become context-specific repetitive patterns that are difficult to change, even when the patient knows the behavior is harmful. Treatment can create short-term change, but this does not necessarily amount to durable recovery. To ensure that generalization does not lead to treatment failure, symptom-focused treatments and relapse prevention should be used in conjunction, taking into account the context of the patient's illness and disorder, as the results vary significantly for each patient. There should also be long-term monitoring to identify learned patterns and prevent them from developing, ensuring that eating disorder behaviors do not become ingrained. Future research should use persistence models that are clinically measurable and usable. Therefore, using transdiagnostic longitudinal designs would be beneficial to track symptom changes, diagnostic crossover, and relapse that occur in different years.

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