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### **Abstract**

Anorexia nervosa (AN) is a severe psychiatric disorder characterized by self-starvation, impaired body perception, and intense fear of weight gain. It stands as the mental illness with the highest mortality rate. This literature review combines findings from three primary sources and additional peer-reviewed research to examine the etiology, neurobiological correlations, treatment limitations, and overall stigma surrounding anorexia. There is increasing evidence suggesting AN as a complex biopsychosocial disorder that stems from genetic susceptibility, neurochemical disturbances (notably reward and habit system dysfunction). Those with AN also tend to desire perfectionism, with environmental factors like trauma and culture heavily influencing the outcome of the disorder. Neuroimaging studies show structural and functional brain abnormalities that are partly reversible with clinical improvement. Still, despite decades of clinical research, treatment outcomes remain modest, with high relapse rates and limited efficacy from gold-standard therapies such as family-based treatment and cognitive-behavioral therapy. The review also points to the role of stigma in avoiding treatment and prolonging suffering. It calls for urgent innovation in neuroscience-informed therapies, public health interventions, and personalized treatment strategies. Through critical synthesis of existing literature, this review aims to expose the current problems of anorexia and explore the need for more effective, interdisciplinary solutions.

### **Introduction & Rationale**

Anorexia nervosa (AN) is a severe psychiatric disorder characterized by self-induced starvation, a relentless fear of weight gain, and a distorted body image, often leading to dangerously low body weight—typically a body mass index (BMI) below 18.5 kg/m<sup>2</sup> in adults—but sometimes occurring even when one appears “normal” in weight. It most commonly emerges during adolescence and disproportionately affects females, though any gender can be afflicted.

AN remains one of the deadliest mental illnesses globally, exhibiting the highest mortality rate among psychiatric disorders, estimated to be up to six times higher than age- and sex-matched populations, with standardized mortality ratios around 5–6. Deaths are often due to starvation-induced organ failure, cardiovascular complications, and an elevated suicide risk.

Beyond its lethality, AN poses profound societal challenges. Its prevalence among young women is estimated at 0.3–4.3%, with rising rates observed worldwide—from 3.4% between 2000–2006 to 7.8% by 2018—and has worsened in the wake of the COVID-19 pandemic. The disorder is also costly: the economic burden spans medical treatment, lost productivity, and long-term care, intensified by significant barriers to treatment access. Only about one-third of affected adults seek care, and many relapse after initial intervention. Given its deadly nature, stubborn chronicity, and widespread impact on individuals and societies, AN demands urgent scholarly attention. This review delves into its multifaceted etiology, neurobiological underpinnings, treatment challenges, and societal consequences to underscore why combating anorexia is both a public health imperative and a scientific frontier.

## **Etiology & Risk Factors**

### **1. Genetics (Heritability 28–58%; Key Loci)**

Anorexia nervosa (AN) exhibits strong familial aggregation. Twin studies estimate heritability in the range of 28–58%, indicating that over half of the risk may be genetically inherited (Ramsay et al., 2024). There have also been findings of numerous polymorphisms related to the genes that regulate not only eating behaviors in humans, but also emotions, personality, and reward mechanisms. Additionally, first-degree relatives have roughly a 12-fold increased risk compared to the general population (Gibson, 2025). Molecular genetic studies have implicated several genes and polymorphisms. The BDNF Val66Met (rs6265) variant is particularly notable: this SNP affects activity-dependent secretion of BDNF, a neurotrophin crucial to appetite regulation and synaptic plasticity. Dopaminergic genes—including DRD2, DRD3, COMT, and DAT1—have been associated with reward and motivation dysfunctions in AN. Additional loci of interest linked to social cognition and emotional processing include OXTR, TFAP2B, and KCTD15 (Almaghrbi et al., 2024). Despite these findings, many genetic association studies are limited by small sample sizes, inconsistent phenotyping, and methodological challenges. Epigenetic research is emerging, showing that malnourishment may alter gene expression via DNA methylation, potentially further maintaining the disorder (Cowdrey et al., 2011). Large-scale efforts like ANGI (Anorexia Nervosa Genetics Initiative) are underway to identify novel risk loci in well-defined phenotype subgroups, including chronic or resistant cases (Muhlheim et al., 2024). Genetic summary: AN's moderate to high heritability underscores a significant biological component, but it arises from the combined effect of multiple variants, likely impacting brain function, reward pathways, and stress-emotion regulation, rather than any single gene.

### **2. Neurobiology: Reward & Habit Circuitry Dysfunction**

Neuroimaging and fMRI studies consistently point to dysregulated reward processing and heightened habit formation in AN patients. Abnormalities in the brain's reward system, particularly when it becomes overly sensitive, less responsive, or malfunctions in its ability to process and respond to rewards. A normal reward system would result in natural rewards like food, social interaction, and exercise, but a dysregulated system would have heightened sensitivity to these rewards or even depend on artificial rewards. Acute and recovered AN patients show abnormal physiological and neural responses to food cues and other rewards, especially within the dorsal striatum and associated habit systems (Wagner et al. 2007). In essence, this indicates that the ability to detect and identify the significance of stimuli is lost, and is a very plausible explanation for why patients diagnosed with anorexia nervosa are less motivated to engage in recovery. Additionally, a landmark meta-analysis argues that anorexic cognition is characterized by a shift from goal-directed to habit-based reinforcement, not just limited to food but generalized across behaviors. Accordingly, recent ecological momentary assessment confirms that the daily life of AN patients is governed by heightened habit frequency, even in non-dietary contexts. Together, these findings support a sequential two-stage model: disrupted reward sensitivity reduces motivation to eat, and compulsive habitual behaviors entrench extreme dietary restriction. Moreover, resting-state connectivity studies reveal altered synchronization between reward and habit-learning circuits, suggesting a neurobiological scaffold for chronicity.

### 3. Environment & Psychology

#### ***Perfectionism***

Perfectionism is a robust, trait-like risk factor identified since the 1940s. Individuals with AN often present with rigid goal-setting, high achievement orientation, and resistance to change. They set almost impossible goals, and experience severe frustration when they fall short. This raises questions on the correlation between perfection and anorexia. Multitudes of reports have noted that there is a strong correlation between the two, mostly due to the idea that anorexia is a limiting disease where one mistake could ruin weeks, even months of progress. Many of those affected believe that to experience the disorder successfully, one must be perfect in their regimen. Finally, and most importantly, elevated perfectionism is associated with a longer course of illness and slower recovery times (Ramsay et al., 2024).

#### ***Trauma & Psychological Comorbidity***

Traumatic experiences, including abuse and familial dysfunction, raise the risk of developing AN by triggering maladaptive coping mechanisms. Comorbid conditions—especially anxiety, depression, and OCD—share underlying phenotypes and may mediate the onset or perpetuation of restrictive eating.

#### ***Socio-Cultural Pressures & Social Media***

The internalization of societal thin ideals, propagated through both traditional media and rapidly evolving digital platforms, remains a potent and well-documented risk factor for anorexia nervosa (AN) (Levine & Murnen, 2009). Adolescents and young adults—particularly girls—are uniquely vulnerable to these influences during critical developmental windows when self-concept and identity formation are most malleable. A scoping review spanning 50 studies across 17 countries concluded that social media usage consistently heightens risk for disordered eating via mechanisms such as upward social comparison, thin- and fit-ideal internalization, and self-objectification (Holland & Tiggemann, 2016). Platforms like TikTok, Instagram, and Pinterest now algorithmically prioritize body-focused content, making it easy for young users to fall into echo chambers that glorify restrictive eating, calorie counting, and excessive exercise. Notably, the rise of so-called “pro-ana” or “thinspiration” communities online has created a uniquely dangerous feedback loop. Here, individuals not only consume but actively engage with content that reinforces ED cognitions, such as body-checking videos, starvation diaries, and weight loss transformation posts. These communities often reward dangerous behaviors with likes and validation while discouraging recovery efforts. Influencers with millions of followers frequently edit and manipulate their bodies in posts, perpetuating impossible beauty standards that followers mistakenly perceive as natural. This manufactured perfection feeds into body dissatisfaction, low self-esteem, and a compulsive drive for thinness, especially in teenagers whose media literacy is still underdeveloped. Worse yet, engagement with these platforms is often paired with negative peer feedback and cyberbullying, which amplifies shame, perfectionism, and social isolation—each of which is a well-established psychological predictor of anorexia nervosa (Fardouly & Vartanian, 2015). Despite growing awareness, regulatory efforts remain minimal, and algorithmic amplification of this content persists. As social media continues to shape adolescent identity and peer dynamics, its role in the etiology of eating disorders demands more urgent intervention, not only in clinical settings but also at the policy level, where content moderation, age protections, and digital literacy campaigns remain grossly inadequate.

## **Neuroimaging & Brain Structure in Anorexia Nervosa (AN)**

Neuroimaging has been instrumental in uncovering the profound structural changes that accompany acute anorexia nervosa. Across multiple MRI-based studies, acute phases of AN are consistently associated with global reductions in both gray matter (GM) and white matter (WM) volumes, alongside increases in cerebrospinal fluid (CSF) space—likely a compensatory change due to brain tissue loss (Seitz et al., 2014; Frank et al., 2013). A meta-analysis by Seitz and colleagues (2014) reported average global GM volume reductions of approximately 5–11% and WM volume reductions of 3.7–3.8% in acutely underweight patients. These findings highlight the extensive neurobiological impact of severe malnutrition, with volume losses visible across nearly all brain regions, including the frontal cortex, parietal lobes, and cerebellum. Such widespread structural compromise aligns with the clinical presentation of AN, which often involves cognitive rigidity, poor interoceptive awareness, and impaired decision-making. Importantly, follow-up studies demonstrate that many of these volumetric deficits are reversible. Longitudinal MRI studies in adolescents and young adults with AN show that cortical thinning and total brain volume reductions tend to normalize with sustained weight restoration and nutritional rehabilitation (King et al., 2015; Bernardoni et al., 2016). This suggests that many of the observed alterations in GM and WM reflect a state-dependent effect of malnutrition rather than irreversible structural damage. Notably, full recovery of brain volume often lags behind physical weight restoration, implying a delayed but ongoing neurobiological recovery trajectory. This lag can complicate clinical judgments about patient readiness for discharge or tapering of intensive treatment.

However, there is growing evidence that certain structural deficits may persist beyond acute recovery, pointing toward potential trait or scar markers of the disorder. For example, diminished volumes in cerebellar white matter and certain regions of cortical WM at the time of hospital admission have been linked to slower treatment response and poorer prognosis (Friederich et al., 2012). These abnormalities may either reflect pre-existing vulnerabilities that predispose individuals to developing AN or long-term consequences of prolonged malnutrition, particularly in cases of early-onset or chronic illness. The distinction between state and trait markers is critical, as it informs both prognosis and the timing of therapeutic interventions. Understanding whether these abnormalities precede illness onset or emerge as a consequence could eventually enable predictive biomarker development or early neurodevelopmental screening in high-risk individuals.

### ***2. Regional Gray Matter Changes: Cingulate, SMA, Cerebellum***

In addition to global brain volume reductions, voxel-based morphometry (VBM) studies have revealed consistent patterns of regional gray matter (GM) loss in individuals with anorexia nervosa (AN), particularly in areas involved in cognitive control, motor planning, and self-referential processing. Meta-analyses of VBM studies have identified significant GM volume reductions in the medial and posterior cingulate cortex (MCC and PCC), supplementary motor area (SMA), precuneus, and bilateral cerebellum (Seitz et al., 2014; Gaudio et al., 2011; Zhang et al., 2018). These localized deficits offer critical insight into the neurobiological underpinnings of AN and its characteristic symptoms.

The cingulate cortex, especially the posterior and medial subregions, is central to emotion regulation, self-reflection, and interoceptive awareness—abilities often impaired in individuals with AN. Reduced volume in the MCC and PCC may contribute to the distorted body image and impaired self-evaluation

observed in these patients. The precuneus, which plays a role in visuospatial imagery and aspects of self-consciousness, also shows significant thinning, potentially underlying the body dysmorphia and overvaluation of thinness central to the disorder (Gaudio & Quattrocchi, 2012). These impairments are not merely secondary to starvation but may reinforce the obsessive, self-critical thinking that sustains disordered eating behaviors. Similarly, GM reductions in the supplementary motor area (SMA)—which coordinates motor planning and habitual action—may be linked to the rigid routines and compulsive exercise seen in AN. The SMA's hyperactivation, often observed even in the absence of external cues, has been hypothesized to reflect an overlearned behavioral response pattern, contributing to the habitual restriction and over-control of food intake (Frank et al., 2020).

The bilateral cerebellum, traditionally associated with motor control, has increasingly been implicated in emotional regulation and cognitive flexibility. GM deficits here may help explain the difficulty with cognitive set-shifting and emotion processing, both of which are commonly impaired in individuals with AN and often predict poor treatment response. Emerging research suggests that cerebellar volume loss may persist even after weight restoration, raising the possibility that cerebellar alterations are a trait marker or consequence of prolonged illness duration (Ehrlich et al., 2019). Taken together, these regional abnormalities suggest that AN is not merely a behavioral or psychological disorder but one that is deeply entrenched in the brain's structure and function. Targeting these specific circuits, particularly those involving the cingulate cortex and SMA, may provide a more precise neurobiological basis for future therapeutic interventions, including neuromodulation and cognitive remediation therapy.

### ***3. Functional Correlates & Behavioral Side***

The structural brain abnormalities observed in individuals with anorexia nervosa (AN) are not just incidental findings—they map closely onto core behavioral symptoms and functional impairments. Reduced gray matter (GM) volume in the supplementary motor area (SMA) and anterior/mid-cingulate cortex has been functionally linked to deficits in motor planning, habit formation, and interoceptive awareness (Kaye et al., 2013; Ehrlich et al., 2015). These regions are integral to coordinating voluntary action and monitoring bodily signals, suggesting that their atrophy may directly underlie hallmark AN behaviors such as rigid food routines, compulsive exercise, and the inability to accurately sense hunger or fullness. In this way, brain-based dysfunctions likely reinforce the behavioral loop of restriction and control, rather than merely reflecting its consequence.

Further, involvement of the precuneus and cerebellum suggests impairments in self-referential processing, visuospatial representation of the body, and emotion regulation—all of which are consistently disrupted in AN patients. The precuneus, for example, plays a crucial role in imagining oneself from a third-person perspective and maintaining a coherent self-image. Its dysfunction is therefore tightly associated with body dysmorphia and distorted body image, a core feature of the disorder (Gaudio & Quattrocchi, 2012). The cerebellum, though traditionally associated with motor coordination, also supports cognitive flexibility and affective integration. Volume loss in this region has been tied to difficulties in shifting attention away from food- and weight-related thoughts, contributing to the cognitive rigidity and obsessive rumination that complicate treatment.

Functional MRI (fMRI) studies bolster these structural observations. Patients with AN exhibit hypoactivation in the reward circuitry, including the ventral striatum and orbitofrontal cortex, during tasks

involving food stimuli or positive feedback (Wierenga et al., 2015). This suggests a blunted reward response, which may explain both the lack of appetite and diminished motivation to recover, even in life-threatening conditions. Additionally, aberrations in resting-state functional connectivity—particularly within the default mode network (DMN) and salience network—have been reported in both acutely ill and weight-restored individuals. These networks are central to internal thought processes, self-monitoring, and assigning importance to bodily cues. Persistent dysregulation, even post-recovery, raises concerns about trait-like neural signatures that may drive relapse or underlie a “neurobiological scar” of the illness (Boehm et al., 2014).

Critically, these functional deficits not only correlate with structural findings but also predict clinical severity and treatment response. For example, lower SMA activation during motor inhibition tasks has been linked to poorer outcome trajectories, while altered cerebellar connectivity may signal a higher risk of chronicity (Ehrlich et al., 2019). As neuroimaging methods continue to evolve, there is growing interest in using these brain-based markers as predictive tools for personalized treatment plans, offering a much-needed shift from symptom-based diagnostics to biologically-informed care.

#### ***4. White Matter Integrity and Network Disruption***

While gray matter abnormalities in anorexia nervosa (AN) have received considerable attention, white matter (WM) integrity—which supports communication between brain regions—is equally critical to understanding the disorder’s neural basis. Studies using diffusion tensor imaging (DTI), a form of MRI that measures water diffusion along axons, consistently reveal impaired structural connectivity in individuals with acute AN. A 2021 meta-analysis by Barona et al. identified significant reductions in fractional anisotropy (FA)—a key marker of WM integrity—in the corpus callosum (CC) and cingulum bundle, among other tracts, suggesting widespread disruptions in interregional signaling (ScienceDirect; Frontiers in Psychiatry).

The corpus callosum, the brain’s largest WM tract, facilitates interhemispheric communication, allowing the two cerebral hemispheres to share and integrate cognitive and emotional information. Decreased FA in this region may partially explain emotional dysregulation, impaired cognitive flexibility, and the inability to reconcile conflicting internal signals, such as feeling hungry but fearing food. The cingulum bundle, a critical WM tract connecting the cingulate cortex to the hippocampus and frontal lobes, supports emotion regulation, attention, and memory retrieval—functions that are consistently impaired in AN. Damage to this tract could underlie the rigid, perseverative thinking and ruminative self-focus that characterize the illness. Interestingly, although total WM volume tends to normalize following nutritional rehabilitation, evidence suggests that microstructural alterations may persist well into weight recovery. Studies have found continued reductions in FA in long-term recovered patients, particularly within the superior longitudinal fasciculus and anterior thalamic radiations, indicating lasting deficits in large-scale brain network integration (ResearchGate; Frontiers). These persistent disruptions could reflect a “neural scar”—a remnant of prolonged malnutrition—or even premorbid vulnerability, highlighting the importance of early detection and intervention.

From a functional standpoint, these WM changes likely affect cortico-limbic circuitry, especially the fronto-striatal and fronto-limbic loops that govern habit formation, impulse control, and emotional processing. Disruption of these networks may contribute to both the compulsive nature of food avoidance

and the emotional numbing seen in chronic cases. Emerging research suggests that these deficits may not only predict treatment resistance but also guide future neuromodulatory interventions (e.g., transcranial magnetic stimulation) that aim to restore disrupted connectivity.

In summary, WM abnormalities in AN are not limited to passive consequences of malnutrition; they may represent core features of the disorder, driving its persistence and complicating recovery. Further integration of DTI with functional MRI (fMRI) and machine learning could help identify connectome-level biomarkers of illness severity, chronicity, and treatment prognosis.

## **Clinical Characteristics & Outcomes**

Anorexia nervosa (AN) carries the highest mortality rate of any psychiatric disorder. A comprehensive meta-analysis reported standardized mortality ratios (SMRs) between 5 and 6—meaning AN patients are approximately six times more likely to die than the general population—driven by medical complications and suicide (Treasure, et al). Suicide accounts for a significant portion of these deaths; eating disorders contribute to around 20% of mortality, with AN showing yearly death rates of about 5.4 per 1,000 individuals.

### ***Relapse & Prognosis***

AN often follows a chronic or relapsing course. Nearly half of patients relapse within one year post-discharge, with reported relapse rates between 30–72% in the first two years post-treatment. Full recovery is achieved in only about 50% of cases; 20–30% have a partial recovery, and approximately 20% develop a chronic form of the disorder 🧠 Costs & Stigma (≈1 page)

### ***Treatment Costs & Access***

Anorexia nervosa (AN) is not only one of the most fatal psychiatric disorders, but it is also one of the most financially devastating to treat. A typical residential or inpatient treatment episode can cost approximately \$80,000, with total costs for full recovery (including follow-up outpatient care, therapy, medical monitoring, and nutritional rehabilitation) often exceeding \$250,000 per individual (Glamour, 2022; Wikipedia). These staggering figures rival or surpass the costs of treating cancer, yet the reimbursement landscape for eating disorders remains fragmented and insufficient.

Despite these immense treatment costs, access to care remains abysmally low. An estimated 30 million Americans experience an eating disorder in their lifetime, yet only around 20% receive any form of treatment (National Eating Disorders Association, 2023; Daily Telegraph). The remaining majority are effectively locked out of the recovery pipeline, hindered by a range of structural and economic barriers. These include inadequate insurance coverage, limited geographic access to specialized treatment centers, and prohibitive out-of-pocket costs, especially for those who require extended or residential care.

Among those who do access treatment, insurance hurdles present an additional obstacle to sustained recovery. Most U.S. insurers use narrow medical necessity criteria—commonly BMI thresholds, electrolyte levels, or heart rate—to determine admission, coverage length, and discharge. This overly medicalized gatekeeping approach ignores the complex psychological and behavioral components of AN,

often leading to premature discharges when patients show physical stabilization, even if they remain acutely ill from a psychiatric standpoint (Glamour, 2022). Such discharges not only undermine treatment gains but also increase the likelihood of relapse and readmission, driving up long-term costs and compounding emotional distress for patients and families alike. This systemic failure to ensure affordable, sustained, and evidence-based treatment access is not just a logistical issue—it is a moral one. When individuals are denied or prematurely pulled from treatment due to insurance technicalities or lack of funds, a potentially treatable illness becomes chronic or fatal. This disproportionately impacts low-income individuals and those without strong parental or financial support, further exacerbating health inequities. Numerous case studies have highlighted how patients with high medical acuity have died shortly after being discharged from underfunded or insurance-limited care, despite clear warning signs of deterioration.

The financial landscape of eating disorder treatment thus presents a paradox: the most severe and deadly psychiatric illness is both grossly underfunded and underprioritized, despite being eminently treatable with early and sustained care. Addressing these systemic barriers through policy reform, insurance regulation, and public health funding is critical if we are to improve recovery outcomes, reduce relapse rates, and save lives.

## **Stigma**

Beyond biology and economics, social stigma remains one of the most pervasive and insidious barriers to anorexia nervosa (AN) treatment and recovery. Stigma not only deters individuals from seeking care, but it also worsens outcomes by reinforcing internalized shame, social isolation, and systemic neglect. A 2022 study from the University of South Australia found that individuals with eating disorders were perceived far more negatively than those with depression, with participants frequently attributing traits such as “weak character,” “self-inflicted behavior,” and “lack of willpower” to those with AN (Frontiers in Psychology; Wikipedia). This framing reflects a long-standing societal misunderstanding: where depression elicits empathy, AN is often met with blame. The disorder’s highly visible symptoms—extreme weight loss, ritualized behaviors, food refusal—invite scrutiny and moral judgment rather than support. This compounds the isolating nature of the illness and feeds into its core mechanisms of control, secrecy, and self-punishment.

These attitudes have tangible clinical effects. Research has shown that individuals who internalize eating disorder stigma report increased symptom severity, longer illness duration, and more frequent relapses (PMC; NCBI). Stigma leads to lower self-esteem, reduced trust in medical providers, and a greater likelihood of avoiding or prematurely discontinuing treatment. Many patients delay disclosure of symptoms for years, particularly if they feel their illness will not be taken seriously—a delay that can deepen both psychological and physical deterioration.

Stigma also operates on a systemic level, particularly harming those with atypical anorexia—individuals who meet all diagnostic criteria for AN except being underweight. Despite enduring severe restriction, obsessional thinking, and profound emotional distress, these patients are often dismissed by both healthcare providers and peers as “not sick enough” (Glamour, 2023). This denial of care reinforces dangerous messages: that eating disorders only matter when they are visible, or that thinness validates suffering. For individuals in larger bodies, the stigma is even more potent, as weight loss may be praised

rather than flagged as a symptom, delaying diagnosis and reinforcing disordered behaviors. The consequences of this double-edged stigma—personal and institutional—are severe. When stigma intersects with financial barriers and limited insurance coverage, a common scenario emerges: people wait too long to seek help, only accessing treatment when symptoms have become deeply entrenched or life-threatening. This not only complicates recovery but also increases the total cost and duration of treatment.

In sum, addressing stigma is not a side issue—it is a central pillar of improving anorexia outcomes. Reducing societal blame, validating diverse presentations of AN, and training clinicians to recognize non-stereotypical cases must be prioritized in public health and clinical discourse. Without these changes, millions will continue to fall through the cracks, suffering in silence from an illness they're too ashamed or too dismissed to name.

## **Future Directions & Public Health Implications**

### ***Social Media's Role & the Case for Regulation***

Parallel to biological discoveries is the growing recognition that digital media environments play a powerful etiological role in the development and maintenance of eating disorders. While traditional media have long promoted thin ideals, modern platforms like TikTok, Instagram, and Pinterest algorithmically amplify content promoting extreme dieting, body checking, and “what I eat in a day” videos, often under misleading wellness or fitness labels. A growing body of research confirms that exposure to such content is associated with greater body dissatisfaction, disordered eating, and internalization of thin-ideal messaging, particularly in adolescents and young adults (Fardouly et al., 2015; Tiggemann & Slater, 2014).

### ***Policy Wins & the Need for Structural Support***

Although the treatment landscape for eating disorders remains underdeveloped, recent policy wins offer glimmers of progress. The Anna Westin Act (2021), named after a young woman who died from anorexia, mandated insurance parity for eating disorder treatment in the United States, requiring that mental health care be reimbursed on par with physical health services. While enforcement remains inconsistent, the legislation represents a critical step in acknowledging eating disorders as serious, insurable medical conditions.

Additionally, nonprofit initiatives such as Project HEAL provide vital stopgaps. Their work includes insurance navigation, coverage appeals, and financial assistance for therapy and nutritional rehabilitation services that can mean the difference between recovery and relapse for patients who fall outside traditional coverage criteria. By helping patients meet diagnostic and medical necessity thresholds, these programs reduce access disparities and support continuity of care. However, these are still patchwork solutions to a larger systemic issue. True progress requires an integrated strategy that addresses cost, stigma, digital exposure, and neurobiology simultaneously. As this literature review demonstrates, AN is not just a medical illness—it is a multisystem public health challenge.

## **Conclusion**

Despite decades of clinical effort, anorexia nervosa (AN) remains one of the most intractable challenges in psychiatry. It is characterized by high relapse rates, severe medical complications—including cardiac arrhythmias, osteoporosis, and brain atrophy—and one of the highest mortality rates among psychiatric disorders. While established interventions such as Family-Based Therapy (FBT), Cognitive Behavioral Therapy (CBT), and selective serotonin reuptake inhibitors (SSRIs) are critical components of care, their outcomes are often modest and inconsistent, particularly for chronically ill or adult populations.

However, emerging research offers new hope. Advances in neuroscience-informed treatments—including deep brain stimulation, transcranial magnetic stimulation, and genetic screening for risk loci—promise more targeted, biologically grounded interventions. At the same time, public health strategies—including regulation of harmful social media content, policy changes such as the Anna Westin Act, and restructured insurance coverage models—are gaining momentum, recognizing that anorexia is not only a medical condition, but also a sociocultural crisis. Ultimately, combating anorexia requires an ecosystem-level paradigm shift: one that breaks from the “one-size-fits-all” model and integrates biological science, therapeutic innovation, systemic reform, and community education. The disorder cannot be treated in isolation—it must be approached as a neurobiological illness shaped by social messaging, economic inaccessibility, and public misunderstanding.

The bottom line is clear: if we continue to rely solely on traditional therapies and ignore broader social determinants, we risk perpetuating the status quo of suffering. But if we unite brain science, social action, and policy under a cohesive framework, the potential for transformative change is not only real—it’s long overdue.

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