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Neurobiological and Metabolic Biomarkers as Diagnostic Tools for Anorexia Nervosa in Adolescents: A Literature Review

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AUTHOR BIOGRAPHY

Tara Nyati is a junior at the American School of Dubai, where she serves as Co-Editor-in-Chief of the school's scientific and medical journal and Secretary General of ASDMUN. Her passions lie at the intersection of medicine and ethics, and her research is informed by a personal investment in improving eating disorder diagnosis and treatment. In college, Tara plans to study biochemistry and medical humanities, with aspirations to become a physician.

ABSTRACT

Anorexia nervosa carries the highest mortality rate of any mental health disorder, yet its early diagnosis in adolescents remains critically hindered by overlapping symptomatology, high comorbidity rates, and environmental factors. Moreover, as precision medicine gains traction, the lack of reliable biological indicators for this disorder remains a significant gap. This review synthesizes the current literature on neurobiological and metabolic biomarkers as prospective diagnostic tools for anorexia nervosa in adolescents, interpreting the findings into two categories: trait biomarkers, which reflect stable physiological predispositions detectable prior to symptom onset and often persist post-recovery, and state biomarkers, which emerge during active stages of the disorder. Key biomarkers examined include electrophysiological abnormalities, structural brain changes, neural network dysfunction, and metabolic dysregulation across amino acid, lipid, and hormonal profiles. The implications of applying biomarkers to clinical diagnostic procedures are further identified, alongside critical limitations and ethical concerns regarding this practice. This review highlights the need for longitudinal, demographically generalizable research in this field and the implementation of changes in healthcare policy to ultimately move toward a precision-medicine standard for the early detection of anorexia nervosa in adolescents.

Keywords: *Anorexia Nervosa, Adolescents, Mental Health, Biomarkers, Diagnostic, Electrophysiology, Neuroimaging, Metabolite*

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INTRODUCTION AND BACKGROUND

Mental illnesses are increasingly recognized as multisystem disorders, rather than isolated conditions of the brain, resulting from dysregulated interactions between enteric, immune, neural, and endocrine systems (Glannon, 2022). The metabolic and neurological connection specifically is exemplified by dysfunctions in the gut-brain axis and its stress response pathway: the Hypothalamic-Pituitary-Adrenal (HPA). These pathways respond to environmental and psychological stressors, and are crucial to homeostatic brain functioning (Glannon, 2022). In the context of eating disorders (EDs)—a type of mental illness defined by extreme weight loss or a failure to gain weight, persistent food restriction, and a distorted body image—the HPA axis exhibits chronic hyperactivity, manifesting in high concentrations of cortisol in affected patients, in response to the "physiological" stress of starvation (Dell'Osso et al., 2016; Seidel et al., 2021; Calcaterra et al., 2024).

Despite their severity, EDs are notoriously difficult to diagnose due to the overlapping symptomatology across subtypes, high rates of comorbidities with other mental illnesses, inadequate healthcare provider training, and academic debate surrounding certain diagnostic criteria (Horovitz, 2025). These challenges are further exacerbated in adolescents, who often struggle to articulate feelings of distress and whose symptoms are frequently masked by ongoing emotional development and impaired access to treatment, which is usually dictated by familial dynamics and socio-economic status (Horovitz, 2025). Puberty further places adolescents at a heightened risk of social isolation and adverse peer influence, both of which significantly amplify disordered behaviour and thinking patterns and hinder detection (Horovitz, 2025).

Therefore, the identification of neurological and metabolic biomarkers, measurable indicators of biological functioning, poses an opportunity for diagnostic clarification and improvement (Glannon, 2022). Detecting these biological signatures before symptoms manifest and rigid thought processes take root holds promise for leading to both a more accurate diagnosis and more effective patient outcomes (Glannon, 2022). The use of biomarkers can enable medical professionals to adopt more personalized treatment approaches to EDs targeting specific dysfunctional pathways, thereby improving long-term recovery for the most vulnerable demographic, affected adolescents (aged 10-19 years). This review synthesizes existing empirical literature to identify viable biomarkers for the early detection and diagnosis of anorexia nervosa (abbreviated as anorexia), an ED with the highest mortality rate amongst ED subtypes (Auger et al., 2021), with a focus on adolescents.

NEUROBIOLOGICAL BIOMARKERS

Neurobiological biomarkers are quantifiable biological characteristics that indicate abnormalities in the brain and nervous system, which could serve as useful indicators, either for the identification of different physiological states (state biomarkers) or as symbols of persisting traits throughout the disorder and recovery

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process (trait biomarkers) (Lema et al., 2018). They can be separated into four key domains based on the detection method: blood, immunohistochemistry (application of antibodies to detect specific proteins in tissues or fluids), neuroimaging, and electrophysiology (Reddy & Abeygunaratne, 2022). In the following sections, neurobiological biomarkers identified through electrophysiological and neuroimaging-based approaches are organized based on their functional impact on the adolescent brain.

Electrophysiological Biomarkers

Electrophysiological biomarkers involve the electrical properties of biological cells and tissues; more specifically, in the brain, these signatures are identified using electroencephalography (EEG). EEGs are a non-invasive method that utilizes electrodes to detect electrical impulses passing between neurons and documents the brainwave data collected in an electrogram for interpretation (Berchio et al., 2022). While individual neurons produce electrical fields below the limit of detection, when large populations synchronise firing, their resultant oscillations are perceptible by EEGs. Functionally, these brainwaves act as a regulatory mechanism for states of consciousness and cognitive processing, with wave frequency (in Hertz) corresponding to different, specific physiological and mental states (Miller, 2015). There are five brainwave subtypes, increasing in frequency from delta to gamma, and collectively, deviations from healthy developmental patterns in timing, amplitude, or frequency across these brainwaves can act as biomarkers.

Studies highlight that anorexic male and female adolescents (ranging from nine to 17) exhibit increased neural reactivity in response to visual food stimuli, manifesting as augmented P300 and late positive potential (LPP) event-related potentials (ERP)—stimulus-instigated electrical responses in the brain (Novosel et al., 2014; Wu et al., 2018; Biehl et al., 2019; Berchio et al., 2022). Following visual food stimuli, higher-amplitude P300 waves (indicators of attention and working memory) suggest that an anorexic brain assigns disproportionate importance to food cues compared to a healthy brain (Berchio et al., 2022). Furthermore, studies have identified concurrent increases in LPP responses, an ERP reflecting the continued allocation of attention to emotionally salient stimuli (Novosel et al., 2014; Wu et al., 2018). A heightened LPP in response to visual food stimuli suggests a pronounced attentional bias, greater emotional salience, and difficulty disengaging from food-related thoughts. Amplified P300 and LPP brainwaves in anorexic adolescents can serve as biomarkers, signaling anorexia-like behaviours and thus expediting the diagnostic process. Nevertheless, the impermanent nature of these signatures characterizes them as state-dependent, appearing post-entrenchment in anorexia, rather than indicative of a disorder-wide trait.

Similarly, in anorexic adolescents, studies identified a state-dependent functional reduction in neural circuits responsible for action monitoring, a continuous cognitive process that ensures goal-directed behaviour through controlling metacognition (the awareness of one's own thought processes), information assimilation,

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and inhibition (Torigoe et al., 1999; Yue et al., 2020). This decrease manifests as lower P300 amplitudes and prolonged N200 latencies during tasks requiring inhibition, such as overriding the impulse to partake in disordered thinking or eating habits (Yue et al., 2020). Functionally, an increased N200 latency and a delayed evaluation stage suggest responses to conflict are sluggish and overriding habitual behaviour is more challenging (Berchio et al., 2022), while lower P300 amplitude indicates a lack of energy to prioritize non-disorder-related tasks. Taken together, the disordered pattern of these brainwaves means affected adolescents experience greater difficulty "metacognitively" reflecting on their actions and are more likely to maintain disordered behaviours.

One singular, persisting abnormality in the EEG recordings of adolescents suffering from anorexia, even post-weight restoration, are changes in posterior theta brainwave oscillations in the parietal and occipital regions of the brain (Grunwald et al., 2001; Hatch et al., 2011; Lackner et al., 2016). Elevated posterior theta power in the occipital and parietal region serves as a key electrophysiological signature of anorexia. Since occipital and parietal regions govern spatial awareness and visual processing, augmented theta potentially drives body image distortion. Furthermore, excessive theta power has been linked to strengthened top-down control which manifests as cognitive rigidity in anorexic adolescents. Because these oscillations persist post-weight restoration, they can be utilized as a biomarker, specifically by serving as a symbol of pre-existing vulnerability and thus expediting diagnostic processes.

Structural Biomarkers

Structural biomarkers are anatomical changes in the brain which can be identified using magnetic resonance imaging (MRI). This method was employed—alongside neuropsychological tests assessing cognitive flexibility, inhibitory control, and working memory—in a study investigating differences in executive function and brain structure between adolescent girls (between the ages of eight and 15) with familial high risk (FHR) for developing an ED and healthy, no-risk girls (Pappaianni et al., 2025). The findings revealed three anatomical biomarkers present in MRI scans of girls with FHR.

The study identified structural differences in terms of grey matter regions in girls with FHR compared to healthy controls. Of the disparities identified in grey matter amounts between FHR girls and healthy controls, one statistically significant region was the right supramarginal gyrus (Pappaianni et al., 2025), an area in the inferior parietal lobe responsible for empathy and perspective-taking. A decreased volume of grey matter in this region directly correlates to a reduction in the density of neurons and, subsequently, impaired execution of associated functions, an idea supported by studies demonstrating patients have significantly lower cognitive empathy scores (Kerr-Gaffney et al., 2019). In contrast to the volume reductions observed in social-cognitive regions, girls with FHR exhibit higher grey matter density and hyperconnectivity in the right fusiform gyrus, an area implicated in the recognition of complex visual stimuli (Pappaianni et al., 2025). Increased grey matter

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density in the right fusiform gyrus suggests a disproportionate allocation of significance to visual processing. In anorexic adolescents, this structural expansion correlates with hyperactivity in the region, as evidenced by an exaggerated hemodynamic response when images of oneself are viewed, which indicates that the brain over-prioritizes self-image processing (Inoue et al., 2015).

Collectively, the structural biomarkers identified demonstrate there is a simultaneous under-development of regions facilitating social interaction and over-development of visual-processing areas within the brain of individuals with or predisposed to anorexia. This suggests a mismatch in neural prioritization wherein abnormalities in the anorexic brain specifically wire it to prioritize the "physical self" (how one looks) over the "social self" (how one relates to others). Furthermore, because the anatomical irregularities identified were found in adolescent girls with FHR (Pappaianni et al., 2025), these signatures can be categorized as trait biomarkers, part of the brain's physical architecture rather than a result of certain anorexic states, like starvation. Therefore, the presence of these indicators in adolescent MRI scans may serve as warning signs and enable the early detection of anorexia.

Network-Related Biomarkers

Network-related biomarkers measure how well spatially distinct regions of the brain communicate with each other. In anorexic adolescents, these signatures indicate the brain's internal communication is often misaligned, causing affected individuals to prioritize certain, disordered thoughts over biological needs, a phenomenon known as excessive top-down control (Frank et al., 2019). Network-related biomarkers can be identified via functional brain imaging which encompasses technologies like functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) which aim to map dynamic neural activity by detecting metabolic, blood-flow-related, or electrical changes. Ongoing research has identified abnormalities in several key neural networks that drive anorexia-related symptoms, the most crucial of which are highlighted below.

The executive control network is a large-scale network, primarily involving the prefrontal cortex, which controls high-level cognitive functions. fMRIs of anorexic patients indicate abnormal connectivity within this network; more specifically between the insula and pre-frontal cortex (Kullmann et al., 2014; Gaudio et al., 2015). The insula processes bodily sensations like hunger and, when its signalling with the pre-frontal cortex becomes dysregulated, it creates a broken feedback loop. This manifests as a profound disconnect between intellectual awareness and behavioural capacity, which includes difficulty perceiving and acting on hunger cues. This network disruption becomes particularly significant when considered through the lens of top-down control, defined as the process by which higher-order brain regions regulate and override lower-level automatic responses (Frank et al., 2019). In healthy individuals, top-down control allows the prefrontal cortex to modulate

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emotional and instinctive reactions whereas, in anorexic patients, this control reinforces disordered behaviours and, coupled with simultaneous disruptions to parallel networks which can increase top-down control, makes ED-based habits harder to override.

Top-down upregulation is further reflected in reversed communication between the hypothalamus and ventral striatum, a phenomenon observed through fMRI scans of young adults with anorexia (Frank et al., 2019). In healthy individuals, the hypothalamus (involved in maintaining homeostasis) initiates hunger signals to the ventral striatum (the brain's reward centre), prompting the pursuit of food as a rewarding stimulus. However, brain imaging demonstrates that in anorexic patients the ventral striatum overrides hypothalamic signals rather than responding to them, suppressing the biological drive to eat before it can meaningfully influence behaviour (Frank et al., 2019).

Moreover, excessive top-down control causes greater connectivity from the orbitofrontal cortex and insula to the inferior frontal gyrus, linking food perception to restraint in young women with anorexia (Kullmann et al., 2014). Both the orbitofrontal cortex and insula assign a positive value to food, thus motivating eating in healthy individuals. However, augmented connectivity between these regions and the inferior frontal gyrus (involved in motor inhibition), a phenomenon specific to anorexic patients, means that when food is perceived or even thought of, the signal does not travel to reward and motivation hubs (Kullmann et al., 2014). Instead, it is rerouted to the inferior frontal gyrus, triggering an automatic "stop" response. This suggests the anorexia-driven aversion to eating is not purely psychological, but rather the result of a network rewiring which now pairs food perception with inhibition. Therefore, heightened top-down control can serve as a biomarker for increased susceptibility to anorexia.

Additionally, neural networks govern how individuals perceive their own bodies. For instance, the default mode network (DMN) consists of a series of brain regions that communicate together, each of which controls processes like interoception, "self-relevant mentalizing", and recollection (Frank et al., 2019). fMRIs of anorexic individuals depict elevated default mode network connectivity, thought to be a result of low blood sugar levels due to starvation (Ishibashi et al., 2018). Overactivity within this network means the brain is stuck in constant self-referential processing which directly translates into symptoms like body dysmorphia. This inward hyperconnectivity may come at the expense of outward social cognition, a theory which aligns with the lower empathy scores observed in anorexic patients (Kerr-Gaffney et al., 2019).

Finally, the salience network, encompassing widespread brain regions (including the aforementioned insula), filters relevant environmental stimuli (Frank et al., 2019). In anorexic patients, the network's "filtration abilities" are damaged, leading to overactivity in regions processing body image and concurrent underactivity in areas associated with hunger cues (McFadden et al., 2014), a finding supported by structural biomarkers

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showing differences in grey matter concentrations across brain regions (Pappaianni et al., 2025). The hypoglycaemia-induced nature of irregular connectivity in the DMN and salience network makes these indicators state-dependent, as hypoglycaemia fails to persist post-weight restoration. Despite this, both biomarkers underscore one of anorexia's most formidable tenets: body dysmorphia. For this reason, they hold promise for earlier disorder detection.

METABOLIC BIOMARKERS

Metabolic biomarkers emerge from metabolomics, the study of the metabolome, the complete set of small molecules called metabolites (including amino acids, glucose, etc.) found in a biological sample (Mayo-Martínez et al., 2021). Known as "downstream effectors", metabolites represent the final outcome of a series of changes in the body's DNA or proteins (Mayo-Martínez et al., 2021). Since these structures are highly sensitive to lifestyle factors, metabolites serve as ideal indicators of diagnostic and prognostic efforts.

Two primary analytical techniques are used to identify these biomarkers: nuclear magnetic resonance (NMR) and mass spectrometry (Mayo-Martínez et al., 2021). NMR utilises a strong magnetic field to identify the quantity and identity of compounds in a sample, but it lacks sensitivity, often failing to recognize rare or trace metabolites. For this reason, NMR is often complemented with mass spectrometry which ionizes molecules within a sample and then separates them based on their mass-to-charge ratio with extreme precision, thus detecting metabolites present even in tiny concentrations.

For anorexia research, the majority of biomarkers remain underexplored or undiscovered. Despite this, researchers agree on the importance of metabolic biomarkers in detecting anorexia; both because the disorder produces severe metabolic dysfunction, and because emerging evidence suggests metabolic disturbances may drive the psychiatric symptoms of the condition (Mayo-Martínez et al., 2021). Given this inconclusive but consequential bidirectional relationship, metabolic biomarkers occupy a unique diagnostic position: they may reflect disease symptoms while simultaneously highlighting its origins.

Amino Acid Dysregulation

Amino acids are the building materials for proteins and are key indicators of the body's current energy state, since they are central to biochemical processes like cellular metabolism and energy production. The amino acid profile in the body is in constant flux, primarily driven by food intake and proteolysis (the breakdown of muscle tissue to get amino acids). By severely restricting their food consumption, especially of carbohydrates and fats (Mayo-Martínez et al., 2021), the metabolic system of anorexic patients begins to undergo proteolysis. Amino acids produced from this process are subsequently converted into glucose in the liver for energy.

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Collectively, several studies using NMR and mass spectrometry on blood and faecal samples converge on a consistent finding: anorexic patients exhibit a "disordered" amino acid profile (Föcker et al., 2020; Miyata et al., 2021; Monteleone et al., 2021).

For certain amino acids—specifically, glutamine, glycine, and tryptophan—patients experience hyperaminoacidemia, an abnormally high concentration of amino acids and a direct signature of proteolysis (Föcker et al., 2020). Conversely, for branched-chain amino acids (BCAAs) like leucine and valine, patients show decreased levels (Miyata et al., 2021; Monteleone et al., 2021). Unlike other forms of amino acids, BCAAs cannot be naturally produced by the body and must be obtained exclusively from dietary sources, making a decrease in their levels a direct marker of malnutrition. Furthermore, BCAAs are heavily involved in muscle protein synthesis; therefore, their absence may reduce long-term muscle rebuilding, leading to a self-reinforcing cycle of muscle deterioration. This may provide a rationale for the reduced muscle mass and strength anorexic patients exhibit compared to healthy controls, even after weight restoration (Polito et al., 1998). Apart from BCAAs, the levels of tyrosine, a precursor for neurotransmitters like dopamine and norepinephrine, are also lower in anorexic individuals (Miyata et al., 2021; Monteleone et al., 2021). Dopamine governs reward, motivation, and pleasure, while norepinephrine regulates alertness and stress response. When tyrosine is depleted, the corresponding neurotransmitter levels may decrease, leading to the anhedonia, motivational deficits, and heightened stress levels associated with anorexia. Collectively, the dysregulation of amino acid concentrations serves as a biomarker for anorexia; however, because these abnormalities are a result of proteolysis, this marker is state-dependent, only serving as an indicator during periods of severe restriction and malnutrition.

Lipid Mobilization

A second consequence of carbohydrate and fat restriction is the breakdown of internal fat stores. This process is observable through alterations in lipid profiles from anorexic patients which indicate increased levels of freed fatty acids like acylcarnitine and glycerophospholipids (Föcker et al., 2012). An increase in these lipid molecules, both of which are involved in energy metabolism, indicates the body is rapidly mobilising stored fat for energy via a process called beta oxidation. This biological indicator manifests as extremely low body weight, the primary symptom of anorexia.

Moreover, abnormalities involving lipid molecules can also influence an individual's mental state (Shih et al., 2016). As a result of beta oxidation, fatty acid by-products are released such as arachidonic acid (ARA). This is a precursor to oxylipins, lipid-derived pro-inflammatory signalling molecules involved in the immune and stress responses. As a result of increased ARA, oxylipin production spikes and there is a subsequent rise in oxylipin concentration in the brain. Consequently, the brain experiences a rise in neuroinflammation (Shih et

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al., 2016): an inflammatory response driven by the central nervous system's immune cells. This phenomenon is associated with conditions such as anxiety and depression (Shih et al., 2016) and may account for the high levels of comorbidities that anorexic patients often face. Therefore, the elevation of lipid molecules in blood and stool samples serves as a measurable metabolic signature of anorexia. However, its presence depends on the state of patients, appearing phenotypically during stages of extreme malnutrition (Föcker et al., 2020), thereby limiting its application to early detection efforts.

Elevated Ghrelin Levels

A meta-analysis of studies examining blood samples across acute and recovered anorexia patients found that levels of the ghrelin were elevated (Seidel et al., 2021). Produced in the superior section of the stomach, ghrelin is the only known hormone created outside the brain that directly stimulates appetite in the brain, acting as a messenger in the larger gut-brain axis. As a part of appetite regulation, ghrelin also stimulates gastric mobility, accelerating stomach movement to prepare the body for more food intake. There are two types of ghrelin: acyl ghrelin, which increases hunger signals, and desacyl ghrelin, which is ghrelin's inactive form and is thought to reduce hunger. Increased levels of both these ghrelin-types in anorexic patients suggest the body is undergoing an adaptive response to cachectic stress. However, when patients are compared to lean, BMI-matched individuals, ghrelin levels still differ (Seidel et al., 2021), indicating that augmented amounts of the hormone are not solely due to low body mass. Another explanation is that chronically elevated ghrelin levels foster ghrelin resistance in patients by desensitizing the brain's ghrelin receptors, therefore requiring progressively higher concentrations of the hormone to instigate eating behaviours, as patients fail to respond appropriately to ghrelin's appetite-stimulating cues at normal thresholds.

Additionally, ghrelin has been shown to play a role in the dopaminergic system, the primary neural circuit controlling motivation. More specifically, elevated levels of ghrelin are associated with increased dopamine levels in the nucleus accumbens, and subsequent heightened activation in key regions of the brain associated with reward (Seidel et al., 2021). Through this mechanism, ghrelin increases the incentive value of food, a phenomenon that manifests as the obsessive fixation on food anorexic patients exhibit. Interestingly, augmented ghrelin levels were found to benefit a patient's performance on the Iowa Gambling Task, a neuropsychological assessment designed to simulate real-life decision making in uncertain situations (Paslakis et al., 2019). Anorexic patients with elevated ghrelin demonstrated sharpened cognitive functions in processes like decision making and delayed gratification compared to healthy controls, who displayed increased impulsivity, irrationality, and reward sensitivity (Seidel et al., 2021). This finding demonstrates ghrelin's cognitive impacts are context-dependent and that in high-stress situations, patients may default to hyperrationality and excessive top-down control, overriding lower-level desires for rewards like food. This rationale aligns with the

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aforementioned neural network-based biomarkers and demonstrates how increased ghrelin levels act as a metabolic indicator for anorexia.

Following treatment, though, anorexic patients demonstrated a significant decrease in ghrelin levels compared to acute stages of the disorder (Seidel et al., 2021). However, ghrelin levels were still significantly elevated in recovered patients compared to healthy individuals (Seidel et al., 2021), suggesting this hormone is potentially a trait biomarker.

APPLICATIONS, LIMITATIONS AND THE ETHICS OF BIOMARKER-INFORMED DIAGNOSIS

Presently, psychiatry is the only medical specialty without reliable biomarkers. This discrepancy is particularly consequential for anorexia, where current manualized treatments yield recovery in only 31% of patients over nine years (Eddy et al., 2017), suggesting that the field's understanding of the disorder remains fundamentally incomplete. Establishing connections between a patient's physiology and clinical phenotypes would enable precision medicine: the personalized selection of therapeutic interventions that account for the multisystem complexities of disease and minimize the side effects of ineffective treatment (Bryant et al., 2025). Biomarkers represent a critical step toward this standard, offering tools for early detection and improved therapy outcomes. However, it must be acknowledged that anorexia is shaped by a host of factors that biological signatures alone cannot capture, including exposure to Western media, childhood trauma, bullying, and negative school experiences (Bryant et al., 2025). Furthermore, the biomarkers identified thus far carry significant limitations of their own: the instability of state-dependent markers, the inaccessibility of the most diagnostically powerful signatures, the unresolved question of metabolic bidirectionality and context-based interpretation, and the demographic constraints of patient samples—all of which restrict the promise of biomarker-informed diagnosis.

Ambiguity in State and Trait Biomarker Classification

Out of the eleven neurobiological and metabolic biomarkers described in this paper, only three can be interpreted as trait or trait-leaning indicators, all of which are neurobiological: elevated posterior theta oscillations, disparities in grey matter volume and subsequent hyperconnectivity in certain brain regions, and excessive top-down control. This asymmetry in trait-stability between neurobiological and metabolic biomarkers is not incidental; neurobiological biomarkers are more stable, since they capture structural differences in the brain's architecture. On the other hand, metabolic biomarkers reflect the body's immediate biochemical state, which fluctuates in direct response to food intake, restriction, and energy availability. As a result, most of the metabolic biomarkers identified are state-dependent, rendering them ineffective at enabling early diagnosis as

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they are visible only during active starvation, the stage at which anorexia is already clinically apparent. Several biomarkers identified, however, resist clean classification into "state" and "trait" categories and occupy an ambiguous middle ground. The persistence of elevated ghrelin levels post-weight restoration suggests trait-like stability (Seidel et al., 2021); yet, given the scarcity of longitudinal data, it remains unclear whether this elevation is simply biological predisposition. Similarly, irregular connectivity in the DMN and salience network is driven by starvation-induced hypoglycaemia and therefore state-dependent by origin (Ishibashi et al., 2018), but both networks govern body image processing, a symptom that clinically exists long before and after weight restoration in the majority of anorexic patients.

Collectively, this demonstrates that the boundary between state and trait indicators is often heavily blurred, an incongruence which constrains the application of neurobiological and metabolic biomarkers to the early anorexia diagnosis.

Tensions Regarding Clinical Accessibility

A further limitation is the seemingly inverse relationship between a biomarker's diagnostic power and its clinical accessibility. More specifically, the neurobiological biomarkers identified as trait-stable are detectable only through EEG, MRI, and fMRI technologies, all of which are expensive and require specialist administration. For adolescents, whose access to treatment is constrained by familial dynamics and socioeconomic status (Horovitz, 2025), the likelihood of these modalities being incorporated into routine checkups is low. As a result, the biomarkers with the highest predictive power, those capable of identifying vulnerability before disorder onset, sit behind the highest access barriers. Metabolic biomarkers, conversely, are far more compatible with routine adolescent healthcare; blood and stool sampling are non-invasive, low-cost, and already embedded in standard checkups. Yet, as previously established, the overwhelming majority of these biomarkers are state-dependent, emerging only once severe restriction is underway. This tension indicates that the accessibility and practice of brain imaging in standard adolescent health checks must be increased to apply biomarker-informed expedited diagnosis.

Metabolic Bidirectionality and Interpretative Issues

An additional limitation constraining the diagnostic utility of metabolic biomarkers is the question of causal directionality. The relationship between metabolic dysfunction and psychiatric symptoms characteristic of anorexia is bidirectional; the disorder produces metabolic dysregulation, yet emerging evidence suggests such disturbances may themselves precipitate anorexia and instead function as a causal factor (Mayo-Martínez et al., 2021). This ambiguity is acutely problematic in the context of biomarkers: elevated ARA-driven oxylipin production and subsequent neuroinflammation may represent the brain's inflammatory response to starvation

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(Shih et al., 2016), or it may reflect a pre-existing neuroinflammatory vulnerability that contributed to the onset of disordered behaviour in the first place. Similarly, depleted levels of neurotransmitter precursors may be a downstream consequence of prolonged restriction (Miyata et al., 2021; Monteleone et al., 2021), or an upstream metabolic condition that increases the susceptibility of reward and stress regulation systems prior to restriction. While the biomarker is clinically real in either situation, the diagnostic meaning and the therapeutic interventions it should inform differs; for instance, a pre-existing metabolic condition will persist independent of weight restoration and thus demands a different type of therapy. This interpretive ambiguity does not affect neurobiological biomarkers to the same extent, as evidence of structural brain changes was found in girls with FHR before the onset of the disorder (Pappaianni et al., 2025). The absence of an equivalent, directionally interpretable anchor for metabolic biomarkers means that, in isolation, they cannot reliably be used for early diagnosis.

This interpretative difficulty within metabolic biomarkers particularly impacts ghrelin. As previously noted, elevated ghrelin levels in anorexic patients were found to benefit performance on the Iowa Gambling Task, a finding that stands in direct contradiction to ghrelin's effect on healthy individuals (Paslakis et al., 2019; Seidel et al., 2021). This presents a concrete diagnostic problem: elevated ghrelin levels cannot be interpreted in isolation, because the same hormonal reading produces neurologically opposite cognitive outcomes depending on the population in which it is measured. This context-dependency undermines ghrelin's standalone validity as a diagnostic indicator and raises a limitation: biomarkers do not exist independently of the behavioural and environmental components of the individual possessing them, and their interpretation therefore cannot be reliant on presence or magnitude.

Demographic Limitations Within Empirical Samples

The final limitation concerns the demographic composition of the samples from which biomarkers have been identified, and the degree to which those samples reflect the adolescent population this review specifically aims to address. Sample demographics from the selected research papers were largely inapplicable in either gender or age categories, or due to inconsistent reporting. Within the neurobiological studies: the electrophysiological biomarker studies included both male and female adolescents; the structural biomarker study was conducted exclusively on adolescent girls, making it age appropriate but lacking generalizability; and the neural network studies described either did not specify sample composition or were conducted on young adult rather than adolescent populations. This age discrepancy constitutes a significant limitation as an adolescent brain is fundamentally distinct from that of a young adult; therefore, biomarkers derived from young adult samples cannot be assumed to present identically in adolescent patients. In particular, prefrontal cortical development, which governs the executive control and top-down networks central to their paper's findings, is not complete until the mid-twenties. Within the metabolic sub-theme, demographic reporting is even more

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sparse; studies utilizing blood-based and faecal samples frequently omitted age and gender stratification, making it impossible to determine whether the metabolic changes identified are generalisable. Collectively, these sample-related constraints mean that the biomarkers identified in this paper require longitudinal, demographically inclusive studies before being reliably utilized as clinical diagnostic tools.

Ethical Considerations of Biomarker-Informed Early Diagnosis

Beyond their practical and interpretive limitations, the application of biomarkers to adolescent populations raises two ethical concerns: the validity of neuroimaging as a predictive instrument and the broader social implications of deploying biomarker-based risk determination in a vulnerable population.

Neuroimaging's capacity to objectively identify structural abnormalities in the adolescent brain is significant. However, a critical distinction exists between the ability to detect an abnormality and to predict clinical outcomes from that abnormality. Brain structure does not directly map onto behaviour with the precision that diagnostic applications demand; for instance, two adolescents presenting identical structural changes may demonstrate entirely different cognitive profiles, shaped by their unique developmental histories, cultural contexts, and lived experiences (Choudhury & Moore, 2016). This interpretive gap is further exacerbated by the adolescent brain's increased neuroplasticity, which means that what registers as an anomalous pattern in one developmental window may represent a transient stage of normal maturation in adolescence. As a result, this blurs the boundary between developmentally "abnormal" and "normal", making neuroimaging data insufficient alone. Further, an overemphasis on biological justifications, termed biodeterminism, risks undermining the role socioeconomic inequalities and varying life experiences play in shaping the same behavioural patterns (Choudhury & Moore, 2016).

In addition, predictive biomarker use in adolescents may result in pre-emptive, erroneous diagnoses. Psychiatric biomarkers employed to identify "at-risk" individuals carry an inherent stigmatizing potential, and adolescents are at a critical point in identity formation—a premature diagnostic label can become a disruptive force in that process (Choudhury & Moore, 2016). Evidence suggests that stigma attached to mental health diagnoses disproportionately affects adolescents, and that it frequently functions as a deterrent to help-seeking behaviour (McDonagh et al., 2021). Compounding this, the neurobiological signatures identified in this paper such as elevated salience network reactivity are characteristics not exclusive to anorexia, but broadly normative features of the adolescent population. The risk of false positives is therefore not negligible, and there is a significant ethical consequence of incorrectly labelling an adolescent as biologically predisposed to anorexia. Unsolicited risk information, delivered to an individual who has not sought diagnosis and may not have developed any clinical presentation, introduces the possibility of a nocebo effect (Colloca & Miller, 2011): the knowledge of predisposition itself may accelerate disorder onset. Furthermore, biomarker screening of this kind

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raises the question of informed consent, particularly when applied to minors with limited capacity to understand the implications of predictive biological testing. Taken together, these concerns do not invalidate the use of biomarker-informed early detection, but they do demand that its implementation be governed by comprehensive ethical frameworks that safeguard adolescents.

CONCLUSION

There is a lack of early diagnostic tools for anorexia, particularly for detection in adolescents, and biomarkers serve as a potential solution for this discrepancy. This review identifies two main types of biomarkers—neurobiological and metabolic—with the former being more trait-stable, while the latter is more state dependent. However, this complementarity is impeded by structural limitations: the most predictively powerful biomarkers remain financially inaccessible, while lower-cost state indicators demand more complex interpretation. Their application to predictive diagnostics also raises ethical concerns such as biodeterminism, nocebo-driven harm, and induced stigma, all of which are exacerbated by the vulnerability of adolescence as a developmental period. Consequently, achieving biomarker-informed diagnosis requires both scientific and policy progress, and key next steps include: conducting longitudinal empirical studies utilizing demographically representative samples for each biomarker, creating comprehensive consent frameworks to prevent unethical conduct, integrating low-cost metabolic screening into routine adolescent health checkups, and subsidizing access to neuroimaging for at-risk and low-income groups.

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