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# ANOREXIA NERVOSA: AETIOLOGY, ASSESSMENT, AND TREATMENT

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ABSTRACT :

*Anorexia nervosa is a vital reason for physical and psychosocial grimness. Late years have gotten propels comprehension of the fundamental psychobiology that adds to disease onset and support. Hereditary elements infl uence chance, psychosocial and relational components can trigger onset, and changes in neural systems can manage the sickness. Significant advances in treatment, especially for juvenile patients with anorexia nervosa, point to the benefi ts of specific family-based mediations. Grown-ups with anorexia nervosa too have a sensible possibility of accomplishing recuperation or if nothing else generous change, however no specifi c approach has demonstrated clear prevalence, proposing a blend of re-sustenance and anorexia nervosa-specifi c psychotherapy is most eff ective. To effectively fi ght this puzzling sickness, we need to upgrade comprehension of the hidden organic and psychosocial components, enhance procedures for counteractive action and early mediation, and better focus on our medications through enhanced comprehension of specifi c infection systems.*

## INTRODUCTION

Anorexia nervosa is a highly distinctive serious mental disorder. It can aff ect individuals of all ages, sexes, sexual orientations, races, and ethnic origins; however, adolescent girls and young adult women are particularly at risk. This disorder is characterised by an intense fear of weight gain and a disturbed body image, which motivate severe dietary restriction or other weight loss behaviours such as purging or excessive physical activity.1–3 Additionally, cognitive and emotional functioning are markedly disturbed in people with this disorder. Serious medical morbidity and psychiatric comorbidity are the norm.4,5 Anorexia nervosa in adults and older adolescents commonly has a relapsing or protracted course,6 and levels of disability and mortality are high,7,8 especially without treatment. Even partial syndromes (ie, subsyndromal anorexia nervosa) are associated with adverse health outcomes.9 Quality of life is poor and the burden placed on individuals, families, and society is high.10 This Review, like the Lancet Seminar published in 2010,4 which included all eating disorders, focuses on factors associated with anorexia nervosa that are of particular relevance to clinicians, such as recent developments in diagnosis, epidemiology, pathogenesis, treatment, and prognosis.

## CLASSIFI CATION AND DIAGNOSIS

Low bodyweight or low body-mass index (BMI) is the central feature of anorexia nervosa. Tables 1 and 2 give an overview of diagnostic criteria for anorexia nervosa according to DSM2 and ICD11. Restricting and binge-

purge subtypes and remission and severity specifi ers exist. Amenorrhoea is no longer required in the new DSM-5 diagnostic criteria and is also expected to be dropped in ICD-11.12 Main reasons for eliminating this criterion are based on confl icts with inclusion of male individuals, adolescents who have not yet reached menarche, and women who use exogenous hormones into the diagnostic criteria. This change is also based on a large body of accumulated evidence13 showing no meaningful clinical diff erences between women with anorexia who menstruate and those who do not.

## EPIDEMIOLOGY

In high-income countries, the lifetime prevalence of anorexia nervosa in the general population is reported to be around 1% in women and less than 0•5% in men.9 Accurate point prevalence has been more diffi cult to calculate, with studies often failing to identify any cases of DSM-IV-defi ned anorexia nervosa. If the broader DSM-5 criteria A and C (low weight in the presence of overvaluation of weight or shape) are applied, the point prevalence is about 0•3–0•5%.16 Some studies,17 but not all clinical incidence studies, support an increase in anorexia nervosa in adolescents in the past two to three decades.9 Anorexia nervosa typically begins in early-tomid-adolescence, although it can emerge at any age.18 The sex ratio in adults is 1:8, with more female individuals affl icted.19 In children, the sex distribution is less skewed.20 Outcomes diff er across age groups, with higher rates of full recovery and lower mortality in adolescents than in adults (mean mortality 2% vs 5%).21

## PSYCHIATRIC AND PHYSICAL COMORBIDITY

Nearly three-quarters of patients with anorexia nervosa report a lifetime mood disorder, most commonly major depressive disorder.22 Between 25% and 75% of patients with anorexia nervosa report a lifetime history of at least one anxiety disorder,23 which typically precedes anorexia nervosa and starts in childhood.24 Obsessivecompulsive disorder occurs in 15–29% of individuals with anorexia nervosa,25 with up to 79% experiencing obsessions or compulsions at some point in their lives.26 In population-based studies, the prevalence of alcohol misuse or dependence in individuals with anorexia nervosa is between 9% and 25% for anorexia nervosa and typically lower in those with the restricting subtype.27,28 Register-based studies29 confi rmed aggregation of autism spectrum disorder in probands with anorexia nervosa and in their relatives; however, the relation between anorexia nervosa and autism spectrum disorder seems to be non-specifi c.29

## PATHOGENESIS

### Genetic factors

Anorexia nervosa is strongly familial43 and heritability estimates range from 28% to 74%.44 Two genome-wide association studies (GWAS),45,46 currently understood to be underpowered in view of the presumed genetic architecture of anorexia nervosa, predictably did not detect genome-wide signifi cant loci. Boraska and colleagues45 conducted sign tests to compare results from the discovery sample with those from the replication sample, and 76% of the results from the replication sample were in the same direction as the discovery sample, a result highly unlikely to be due to chance ($p=4 \times 10-6$). This observation strongly suggests that true fi ndings exist and that an increase of the sample size will yield signifi cant results; the fi eld is currently accruing larger samples. A study30 reported a signifi cant negative genetic correlation between anorexia nervosa and BMI and a signifi cant positive genetic correlation between anorexia nervosa and schizophrenia, encouraging a deeper exploration of metabolic factors in anorexia nervosa and a new genetic association between anorexia nervosa and schizophrenia.

### Neurobiological factors

Most neurobiological studies have been done in currently ill or recovered patients, thus abnormalities might result from state-related consequences of malnourishment or so-called scarring

### Neurocognition and social cognition

Neurocognitive markers of anorexia nervosa include set shifting diffi culties (ie, diffi culties switching

between diff erent tasks or task demands)47 and poor central coherence (ie, a preference for local [detail-focused] over global [bigger picture] processing).48 These impairments have also been noted in unaff ected sisters of people with anorexia nervosa and to some extent persist after recovery.49

## Structural neuroimaging

Patients with acute anorexia nervosa have been reported to have global reductions in grey and white matter, increased cerebrospinal fl uid and regional grey matter decreases in the left hypothalamus, and in rewardrelated regions of the basal ganglia and the somatosensory cortex.53 Studies in patients who recovered from anorexia nervosa and longitudinal studies (before and after treatment) suggest that brain tissue abnormalities might recover with weight regain.54,55 Many earlier structural studies have methodological limitations .

| Organ systems or organ | Pathological findings | Leading systems |
|---|---|---|
| CNS | Morphological and functional cerebral changes; volume reduction in cerebral grey and white matter | Cognitive deficits |
| Dental system and parotis glands | Impaired dental status, dental caries, increased serum amylase | Dental caries, enlargement of the parotid glands |
| Endocrine system and reproductive function | Hypothalamus-pituritary-gonadal-axis, low $T_3$ syndrome, hypercortisol | Amenorrhoea in women, symptoms of hypothyroidism , depression, elevated stress levels |
| Cardiovascular system | Hypotension, bradycardia, arrhythmia | Syncope |
| Gastrointestinal tract | Impaired gastric emptying, gastric dilation, gastro-duodenal ulcers | Constipation, ileus, upper gastrointestinal bleeding |
| Haematological and immune system | Bone marrow hypoplasia, anaemia with reduced leucocytes and immunoglobulin | Anaemia, (bacterial) infections, compromised immune competence |
| Renal tract | Hypokalaemia, hypophosphataemia, hypernatraemia | Nephrolithiasis, oedema, syncope |
| Bone | Reduced bone density (osteopenia) or osteoporosis | Bone fractures and concomitant pain, spinal compression |

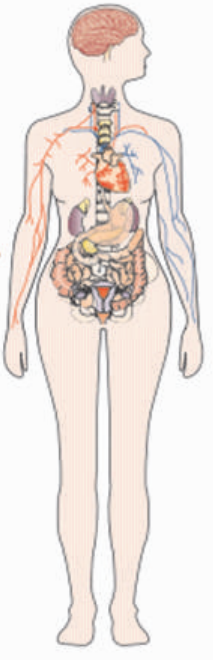

*Figure:* **Impaired organ function in anorexia nervosa**

## Developmental factors

Several adverse experiences occurring around universal stages and transitions of development are associated with an increased prevalence of anorexia nervosa. These developmental risk factors include adverse prenatal, perinatal, and neonatal events, such as dysmaturity or prematurity67,68 as well as feeding and sleeping diffi culties in infancy.68 Throughout childhood, emerging personality traits associated with anxiety, depression, perfectionism, and autism spectrum68 have been identifi ed as risk factors for anorexia nervosa. Puberty and adolescence are characterised by profound changes, vulnerabilities, and the transition to adulthood, and they represent the period of fi rst onset of anorexia nervosa. A possible explanation for the crucial role of puberty for the onset of anorexia nervosa might lie in hormonal changes and dysregulations that interact with neurotransmitter functioning, brain maturity, and genetic factors.69

## Environmental factors

Female gender has consistently been shown to be a risk factor for anorexia nervosa.68 The increase of anorexia nervosa in low-income and middle-income countries suggests that cultural transitions associated with industrialisation, urbanisation, and globalisation might be associated with environmental risk constellations for the development of anorexia nervosa.9 These con stellations might include the adoption of the so-called

western lifestyle, including nutritional habits and thin ideal internalisation. However, although body dissatis faction has been identifi ed as a risk factor for the development of any eating disorder,70 risk factors associated with thin ideal internalisation and associated sociocultural pressures have not been confi rmed for anorexia nervosa. Appraising the potential infl uence of environmental factors, it has to be taken into account that the incidence of anorexia nervosa is relatively low worldwide despite these pervasive sociocultural pressures to be thin.

## Treatment

### Initial assessment and investigations

Initial assessment of the patient with anorexia nervosa includes an in-depth interview, a physical examination, and investigations to establish severity and nature of eating disorder symptoms and diagnosis, comorbid psychological and physical symptoms, diagnoses and risk, past treatments, current motivation for treatment, and available supports. An early task is to build good rapport with the patient, as they are often highly ambivalent about and fearful of treatment.72 Whenever possible, it is important to involve signifi cant others (family, partners) in assessment and subsequent treatment. Table 3 summarises recommended physical investigations to be done at assessment. We have also formulated indicators of high risk, requiring rapid intensive specialist consultation and intervention (panel 1). Pathways (levels) of care A Finnish epidemiological study74 reported that about 50% of people with anorexia nervosa in the community do not access treatment. Those who do engage in treatment typically show varying degrees of ambivalence about change.75 However, with treatment, at least 40% of people with anorexia nervosa (and more in younger samples) will make a full recovery.21 Additionally, access to care from a specialist service with expertise in anorexia nervosa might be associated with better outcomes.76

### Evidence-based prevention programmes

Prevention eff orts can be divided into universal, selective, and indicated, depending on whether they address the general population or populations with increased risk (eg, children of eating disordered mothers; elite athletes) or those exhibiting early signs of a disorder. Eating disorder prevention has focused on either risk factors (eg, body dissatisfaction) or eating disorder pathology or caseness. A systematic review116 of eating disorders prevention programmes for young people between the ages of 12 and 25 years identifi ed six reviews and 46 universal prevention trials, with psychoeducation the most commonly tested intervention (26 trials).116 The review also identifi ed six reviews and 40 trials in at-risk populations, where psychoeducation and cognitive dissonance programmes were equally common (12 each). Meta-analyses of controlled trials from other reviews were summarised as indicating that “prevention programmes generally produce large eff ects on outcomes related to eating disorder knowledge, and only small net eff ects for other important prevention targets such as reducing exhibited risk factors, changing attitudes, and reducing eating pathology”.

## CONCLUSIONS

The past 5 years have seen substantial advances in the knowledge of anorexia nervosa. Recent treatment studies suggest that patients with anorexia nervosa have a realistic chance of recovery, especially if treated early, or at least, to achieve substantial improvement. However, there is widespread agreement that several challenges remain in the management of anorexia nervosa (panel 3) and new interventions are needed to improve outcomes, especially in adults with the disorder. Such interventions should target specifi c disease mechanisms. The neuropsycho logical constructs introduced within the research domain criteria (RDoC) matrix off er a new systematic basis for determining the neural substrates underlying the biological predisposition to anorexia nervosa.120 A clearer understanding of how anorexia nervosa behaviour is encoded in neural circuits would provide a key for developing more eff ective treatments. To conclude, we still need to discover how to provide better, faster, and lasting improvements in the management of this enigmatic disorder. Contributors All authors contributed to the search and selection of the medical literature and to the writing of the Review.

## Declaration of interests

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