The Genetic Origins of Avoidant/Restrictive Food Intake Disorder (ARFID)

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ARFID is a serious eating disorder characterized by persistent and extreme restrictive or selective eating for reasons that cannot be explained by another physical or mental condition, and is not the result of distorted body image. While genetic research on other eating disorders has contributed significantly to advances in biological understanding and treatment development, the genetic basis of ARFID remains poorly understood. A narrative literature review was conducted using PubMed, PsycINFO, and Google Scholar to identify peer-reviewed studies on ARFID genetics, epigenetics, and microbiome findings published within the past decade. Although findings across articles were inconsistent and no single gene was shown to have a definitive association with ARFID, several candidate genes were found as plausible areas of investigation. For example, ZSWIM6 has been associated with ARFID risk in an autism-enriched cohort; ADCY3, which is involved in appetite regulation and energy balance, has been associated with ARFI-broad phenotypes in a large population cohort; and DAT1 shows preliminary epigenetic associations across ARFID subtypes. Additionally, evidence suggests potential biological distinctions among ARFID subtypes, providing support for the view that ARFID is a heterogeneous disorder. Overall, this review highlights critical gaps in the literature and underscores the need for larger, subtype-specific, and methodologically rigorous studies to clarify the disorder's genetic basis.

Keywords: ARFID, avoidant/restrictive food intake disorder, genetics, eating disorders, ZSWIM6, DAT1, ADCY3, dopamine, appetite regulation, autism spectrum disorder, subtypes, epigenetics

Introduction

Avoidant/Restrictive Food Intake Disorder (ARFID) is a relatively new eating disorder formally recognized in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) in 2013. Since then, awareness of ARFID has increased, with estimates suggesting that up to 15% of the general population may be affected [1], though prevalence rates vary depending on the population studied and the diagnostic criteria applied. Before this, DSM-4 recognized a "feeding disorder of infancy or early childhood" but limited it to children under the age of six [1]. Because its classification in the DSM-5 is relatively recent, research on ARFID remains limited [2].

The primary feature of individuals with ARFID is avoidance and/or restriction of intake of foods because of sensory characteristics, aversive consequences (e.g. of trying to eat), or a lack of interest in eating. The resulting persistent failure to meet expected requirements for nutrients and/or energy is the key disturbance in this category. In order to meet the criteria for diagnosis, a disturbance must lead to significant weight loss, failure to gain weight, nutritional deficiency, use of supplementation, and/or marked interference with psychosocial functioning. There are three primary presentations of the disturbance: sensory aversive (aversion from foods due to their sensory attributes), aversive avoidance (food avoidance following trauma/negative consequences such as choking or vomiting), and low interest/restricted (characterized by a lack of interest in eating and limited motivation to consume food) [1]. These are not mutually exclusive and individuals may present symptoms that fall within more than one category at any given time. In this sense, the current DSM-5 categories should be seen as descriptive of a variety of overlapping clinical presentations rather than as consistent labels for distinct groups of individuals. Importantly, ARFID is distinct from other categories of eating disorders in that there is no fear of weight gain or distortion of body image [3].

Although genetic factors have long been investigated in relation to eating disorders, little is known about the genetic basis of ARFID. For example, numerous studies on Anorexia Nervosa (AN), Bulimia Nervosa (BN), and Binge Eating Disorder (BED) have examined serotonergic genes, including the 5-HT2A gene, in relation to appetite regulation and mood [4]. However, current evidence reports inconsistent and location-based associations rather than a uniformly confirmed 5-HT2A signal, highlighting the complex nature of psychiatric genetic research [5]. Research on these genes has improved understanding of the biological mechanisms associated with eating disor-

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ders and has guided further studies, despite the mixed clinical effectiveness of serotonin-related medications[5]. In comparison, the genetic and biological origins of ARFID remain understudied, and no genes have been established as definitive risk factors for the disorder.

Given the limited research on ARFID, a comprehensive synthesis of existing literature is needed. Identifying specific genetic factors that contribute to the development of ARFID would enable the development of more targeted approaches for assessment and intervention. In addition, understanding genetic risk factors would create opportunities to improve early screening and prevention efforts, potentially reducing long-term physical and mental health consequences.

For many complex disorders, modern psychiatric genetics has been unable to pinpoint single genes as straightforward causes. Rather, genetic contributions to psychiatric conditions typically involve polygenic risk as well as gene–environment interactions, in which genetic susceptibility interacts with developmental, environmental, or epigenetic factors. Replication of findings across independent cohorts is crucial for establishing credible genetic associations. This review is accordingly framed around candidate genetic and microbiome-associated findings in ARFID, rather than established causal mechanisms. Specifically, this review aims to identify primary studies reporting genetic, epigenetic, or microbiome-level associations with ARFID or ARFID-related phenotypes; characterize the plausible biological relevance of identified candidate genes to feeding behavior; compare candidate findings with genetic evidence from other eating disorders, noting areas of apparent overlap and divergence with appropriate caution; and critically evaluate the strength, consistency, and limitations of existing evidence. This review does not make causal relationships between any specific gene and ARFID, as current evidence does not support such conclusions.

Methods

This project has been conducted as a **narrative literature review** of primary research mainly published in the last 5-10 years. This type of review does not rely on a pre-registered protocol, nor does it follow a systematic meta-analytic approach for the synthesis of findings. The qualitative synthesis of findings considers evidence quality, methodological design and consistency of findings between studies.

Search Strategy

Relevant studies were identified through searches of Google Scholar, PubMed, and PsycINFO using the following search terms: "ARFID genetics," "avoidant restrictive food intake disorder," "ARFID subtypes," "eating disorder genetics," "DAT1 eating disorder," "ADCY3 appetite," and "ZSWIM6 autism feeding." In addition, a number of forward and backward citation searches were conducted on key papers. All identified sources were checked to establish whether they were peer reviewed and therefore suitable for inclusion. Google Scholar was used in order to establish whether any further, as yet unpublished, sources existed that would have been relevant to the current review.

Inclusion and Exclusion Criteria

Studies were included if they met the following criteria. First, they focused on human subjects. Animal models were noted only where they provided relevant biological framework. Second, they contained information on the genetics (e.g. genetic and epigenetic studies), the microbiome (e.g. metagenomic studies), or both for ARFID or for feeding behaviors that are related to individuals with ARFID. Reviews and analyses of other eating disorders were included for comparison but clearly distinguished from primary genetic research for ARFID. Third, they were published in peer-reviewed journals. Non-peer reviewed clinical websites and consumer summaries were excluded where peer-reviewed alternatives existed.

A total of 23 studies were included in the review and these are listed in table 1. Of these, four studies reported primary data on the genetics or microbiome of individuals with ARFID or similar phenotypes.

Study Selection Rationale

The candidate genes discussed in this review—ZSWIM6, DAT1, and ADCY3—were selected because they were repeatedly referenced across multiple independent searches and were specifically identified in studies directly examining ARFID or ARFID-related phenotypes. The ermF microbiome marker was included because it was reported in the only published study applying metagenomic sequencing directly to an ARFID population, and is discussed separately from human genetic evidence. Gene-comparison data from AN, BN, and BED literature were included where they allowed meaningful comparison with ARFID findings.

Synthesis Method

Studies were organized by evidence type (GWAS, candidate gene study, epigenetic study, microbiome study, or comparative eating disorder genetics review) and by candidate gene or marker. Methodological weight was assigned qualitatively, with GWAS and large population-based studies considered stronger evidence than small candidate-gene or pilot studies. Speculative interpretations and mechanistic hypotheses are confined to the Discussion section and are distinguished from empirical findings in the Results.

Table 1 Summary of primary studies reporting candidate genetic or microbiome findings in ARFID or ARFID-related phenotypes

Author (Year)	Phenotype	N	Population	Study Type	Key Finding / Replication Status
Koomar *et al.*[6] (2021)	ARFID risk in autism cohort	∼6,000	Autism cohort; US (Simons Simplex)	GWAS / SNP array	Narrow-sense heritability ≈45%; GWAS hit near ZSWIM6; genetic overlap with neuroticism, metabolic traits, ASD. Autism-enriched sample limits generalizability. Not independently replicated.
Cimino *et al.*[7] (2021)	ARFID subtypes; DAT1 epigenetics	94	Clinical; Italian pediatric	Epigenetic (methylation)	Higher DAT1 methylation in impulsive/irritable subtype. Epigenetic, not sequence-level variation. Pilot study (n=94); not replicated.
Ye *et al.*[8] (2023)	ARFID gut microbiome	∼30 ARFID + controls	Pediatric clinical; China	16S rDNA + metagenomics	Elevated ermF (macrolide resistance gene) in ARFID group. Microbiome evidence, not host genetics. Small sample; not replicated.
Bjørndal *et al.*[9] (2025)	ARFI-broad trajectories; ADCY3	35,751	Norwegian population cohort (MoBa)	GWAS / population cohort	ARFI-broad classified as persistent/transient/emergent at ages 3 & 8; follow-up through age 14. ADCY3 association identified; SNV heritability 8–16%. Not subtype-mapped. Causality not established.

Study-Level Summary

The primary studies reporting candidate genetic or microbiome findings related to ARFID are summarized in Table 1. Studies are sorted by evidence type to prevent preliminary candidate-gene or pilot findings from being weighted equivalently to GWAS or large epidemiological investigations.

Results

Candidate Genes Associated with ARFID

ZSWIM6: Koomar et al.[6] conducted the first genome-wide investigation of ARFID risk using a large, phenotypically diverse autism cohort. Given the scarcity of direct genetic research on ARFID, the authors examined genetic overlap between ARFID and related neurodevelopmental conditions, such as Autism Spectrum Disorder (ASD). Although ARFID and ASD share certain features, including sensory sensitivities, the disorders remain clinically distinct. Koomar et al. estimated narrow-sense heritability of ARFID at approximately 0.45 within this cohort, suggesting that common genetic variation may contribute substantially to ARFID risk. The authors also identified a genome-wide significant association near the ZSWIM6 gene and observed genetic overlap between ARFID risk and traits such as neuroticism, metabolic features, and ASD. While the autism-enriched cohort with noted power and generalizability limitations means these results should not be interpreted as evidence that ZSWIM6 is directly associated with ARFID in the general population, these findings provide preliminary evidence that ARFID may have a measurable heritable component and may share genetic pathways with neurodevelopmental and metabolic conditions.

Building upon the work of Koomar et al.[6], Kennedy et al.[2] conducted a comprehensive review discussing studies that identified a possible association between ARFID-like feeding difficulties and a *de novo* variant of the ZSWIM6 gene, which has previously been linked to schizophrenia and intellectual disability. However, these projects were conducted on patients with ASD who are also at high risk for ARFID, limiting the generalizability of results to individuals with ARFID alone. Kennedy et al. further proposed that genetic patterns may differ across ARFID subtypes, although no subtype-specific genetic analyses were conducted in their review.

DAT1: Cimino et al.[7] investigated the epigenetic regulation of the DAT1 gene, which encodes a dopamine transporter involved in synaptic dopamine reuptake. It is important to note that DAT1 methylation findings represent epigenetic variation—modification of gene expression through chemical changes to DNA—rather than direct genetic sequence variation. This distinction is fundamental: epigenetic changes are potentially reversible and environmentally influenced, and should not be combined with inherited sequence-level genetic risk. Studying 94 children aged 24–36 months without comorbid disorders, the researchers categorized participants into three ARFID subtypes: impulsive/irritable, sensory food aversion, and post-traumatic feeding disorder. While no subtype showed a direct genetic sequence association with DAT1, children in the impulsive/irritable group exhibited significantly higher DAT1 methylation levels. These methylation differences are consistent with, but do not establish, differential dopaminergic regulation across ARFID presentations. The study also found significant differences between subtypes, providing empirical support for the hypothesis that distinct ARFID subtypes may involve different neurobiological mechanisms[2].

ADCY3: Bjørndal et al.[9] examined 35,751 children using data from the Norwegian Mother, Father, and Child Cohort (MoBa). This study classified ARFI-broad—a broader phenotype describing restrictive eating that does not necessarily meet full ARFID diagnostic criteria—as persistent, transient, or emergent, with assessments at ages 3 and 8 and develop-

mental follow-up reported through age 14. Children with persistent ARFI-broad showed more difficulties across multiple developmental domains from infancy through adolescence, including elevated rates of attention difficulties, emotional dysregulation, and language delays, as well as comorbidities such as celiac disease. The study identified an association between ARFI-broad phenotypes and the ADCY3 gene, which is involved in appetite regulation and energy balance, with single-nucleotide variant (SNV, where a single nucleotide in a genetic sequence is altered) heritability estimated at 8–16%. However, this association did not establish causality or provide subtype-level mapping, and no direct evidence links ARFID patients to ADCY3 dysfunction or abnormal cAMP signaling.

Microbiome Findings

ermF: Ye et al.[8] compared fecal microbiome profiles of children aged 3–12 with ARFID against healthy controls using 16S rDNA and metagenomic sequencing. The study reported significant differences in antibiotic resistance gene abundance, with macrolide resistance genes—including ermF—elevated in the ARFID group. Current research has established that ermF belongs to a family of **antibiotic resistance genes**, which are pieces of DNA most commonly found in bacterial organisms that contain genetic information for the synthesis of proteins that result in resistance to particular antibiotics[10]. This gene is not a human gene, but a bacterial resistance gene, serving as a microbial biomarker rather than evidence of possible inherited genetic risk. The elevated ermF levels in the ARFID group therefore reflect differences in gut bacterial ecology, not inherited host genetic variation, and do not support a "genetic origins" framing of ARFID. A causal or mechanistic relationship between ermF abundance and ARFID-related feeding behaviors has not been established; further research is needed before any conclusions can be drawn regarding the significance of this finding[11].

Identified Genes and Their Functions

This section examines each of the candidate genes identified above, describing their established functions and their plausible, though unconfirmed, relevance to ARFID. As noted, the different subtypes of ARFID may be associated with different neurobiological mechanisms, though subtype-specific genetic evidence remains limited.

ZSWIM6: The ZSWIM6 gene has been associated with intellectual and neurodevelopmental disorders, including ASD and schizophrenia[12,13]. Its molecular function is predicted to involve coding for proteins that interact directly with DNA[13]. ZSWIM6 has been associated with conditions such as Acromelic Frontonasal Dysostosis (AFND) and Neurodevelopmental Disorder with Movement Abnormalities, Abnormal Gait, and Autistic Features (NEDMAGA), both of which involve developmental delays commonly observed in children with ARFID. Given these neurodevelopmental associations, researchers have proposed that ZSWIM6 may be relevant to ARFID, particularly the sensory-based presentation.

DAT1: DAT1 encodes the central dopaminergic neurotransmitter transporter protein (DAT). Franke et al.[14] demonstrated associations between DAT1 variants and ADHD, which is frequently observed as a comorbid condition in children with ARFID. As noted in the Results section, the evidence linking DAT1 to ARFID is epigenetic in nature, involving methylation patterns rather than sequence-level genetic variation, and should be interpreted with appropriate caution. DAT1 methylation status is not equivalent to transporter protein abundance, dopamine tone, or synaptic flux, and inferences about dopaminergic dysregulation based solely on methylation data are not established. DAT1's role in neurotransmitter regulation makes it a biologically plausible candidate mechanism warranting further investigation[7], though no clinical evidence currently supports its use as a treatment target in ARFID populations.

ADCY3: Adenylyl Cyclase 3 (ADCY3) is an enzyme that regulates the synthesis of cyclic adenosine monophosphate (cAMP); which is a signaling molecule involved in numerous cellular processes including olfactory signal processing and appetite regulation[15,16]. Research has associated loss-of-function ADCY3 variants with severe obesity and altered energy balance[16]. Given its role in appetite regulation, ADCY3 is a biologically plausible candidate for association with ARFID, particularly in presentations characterized by reduced appetite. There is however no direct evidence to support the claim that individuals with ARFID have abnormal levels of cAMP or that the dysfunction of this enzyme results in modified feeding patterns. Furthermore, there is as yet no evidence to suggest that the dysfunction of this enzyme will result in a specific ARFID subtype. The association between ADCY3 and ARFI-broad phenotypes described by Bjørndal et al.[9] will require replication in independent ARFID patient cohorts that have been clinically defined.

ARFID-Associated Genes and Genes Associated with Other Eating Disorders

The causes of eating disorders are largely unknown. Research on anorexia nervosa (AN) and similar conditions continues to seek both causes and effective treatments. This section examines connections between different eating disorders that could inform future studies and influence research priorities. Additionally, this section explores whether certain genes may contribute to the development of eating disorders more broadly. While AN, BN, BED, and ARFID are defined by distinct motivations, timing of onset, and patterns of comor-

bidity, claims of genetic overlap must be supported by appropriate genetic correlation analyses, polygenic risk scores, or replicated candidate-gene studies, rather than by phenotypic studies alone.

Anorexia Nervosa

Anorexia Nervosa is typically defined by persistent food restriction for reasons related to the individual's attempt to achieve an unrealistic body image. Although AN and ARFID are very different from a psychological perspective, both disorders involve the restriction of types of food leading to serious weight loss or related nutritional problems. Thus, information regarding the genetic mechanisms involved in individuals with AN could offer clues as to possible biological mechanisms involved in individuals with ARFID.

Many studies have examined associations between AN and serotonergic genes, particularly the 5-HT2A gene and the -1438A allele, found in approximately 46% of individuals with anorexia[4]. Additional studies have found correlations between AN and other serotonin genes[17], which are involved in food intake, mood, and body regulation. However, current meta-analytic literature reports inconsistent and geographically dependent associations rather than a uniformly confirmed 5-HT2A signal[5], and the reproducibility of serotonin receptor gene associations should not be overstated. The 5-HT2A gene has also been associated with anxiety and depression, which are often comorbid with eating disorders[18]. Donato et al.[17] noted a variety of appetite regulation genes associated with AN while also observing that research on eating disorders remains limited and focused largely on AN. So far, no direct evidence has been found linking ARFID to any of the appetite regulation genes noted by Donato et al.[17]. ARFID has been connected to dopaminergic genes like DAT1, which indicates a general connection between eating disorders and neurotransmitter gene pathways, though the nature of this overlap remains unclear.

Bulimia Nervosa

Bulimia nervosa (BN) involves the desire to achieve a specific body image and consists of episodes of consuming large amounts of food immediately followed by compensatory behaviors such as purging. Because BN involves irregular eating patterns and psychological distress surrounding food, some underlying neurobiological mechanisms may have relevance to ARFID. On the other hand, aversive ARFID is often associated with a fear of choking or negative consequences of eating rather than with body image concerns, suggesting that ARFID and BN may differ substantially in etiology.

From a genetic perspective, Donato et al.[17] identified a correlation between BN and the 5-HT2A gene, similar to findings reported for AN. However, although Cimino et al.[7] reported a potential association between the DAT1 gene and BN, this finding has not been widely replicated, and additional research is needed to clarify this. There is currently little direct evidence examining genetic overlap between ARFID and BN.

Binge Eating Disorder

Binge Eating Disorder (BED) is characterized by recurrent episodes of consuming large amounts of food accompanied by a sense of loss of control. BED is not primarily driven by concerns with body image and thus, like ARFID, BED does not involve weight or shape concerns. Those with BED often eat excessively without intentional control, representing, in some respects, the opposite extreme on the feeding-behavior spectrum from ARFID, where restricted intake is not motivated by weight concerns but by avoidance, fear, or lack of interest. BED has also been associated with trauma and genetic factors[19].

From a genetic perspective, Micali et al.[20] conducted a longitudinal study examining eating disorder outcomes in women and their children, with 4,948 questionnaires completed by the time children reached age 16. Although the ADCY3 gene has been associated with weight and diet regulation, this study found no association between ADCY3 and BED. In contrast, Davis et al.[21] found a possible association between BED and the DAT1 gene, suggesting that DAT1 may influence appetite regulation and reward processing in the context of BED.

Discussion

Despite the limited literature, this review identified candidate associations between ARFID and multiple genes, including ADCY3, ZSWIM6, and DAT1. Each represents a biologically plausible candidate given known roles in appetite regulation, neurodevelopment, and neurotransmitter signaling. For example, ADCY3 has been linked to obesity and appetite control[15,16]. While obesity represents one end of the appetite-regulation spectrum, individuals with restrictive ARFID report diminished hunger and lack of interest in food, suggesting a possible relevance of ADCY3 to this presentation. The microbiome marker ermF was also identified; however, this reflects gut bacterial ecology rather than host genetic variation and should be considered separately from the candidate human genetic findings.

Overall, ARFID does not appear to have a strong or consistent genetic overlap with other eating disorders, despite the identification of possible common candidate genes. Instead, emerging evidence suggests that different subtypes of ARFID may have different neurobiological underpinnings. In addition to Cimino et al.[7], who reported epigenetic and genotype differences across ARFID subtypes, the functional roles of candidate genes offer biologically plausible subtype-specific hypotheses. For instance, ZSWIM6, associated with neurodevelopmental conditions such as ASD, may be relevant to sensory-

Table 2 Summary of candidate gene associations with eating disorders. Entries indicate whether peer-reviewed studies have reported a positive association ("Reported"), reported a null finding ("Not reported"), or where literature is insufficient ("Not enough literature to conclude"). Associations labeled "Reported" reflect candidate-level evidence only and do not imply causality or consistent replication.

Gene	Anorexia Nervosa	Bulimia Nervosa	Binge Eating Disorder	ARFID
5-HT2A (serotonin gene; associated with AN, BN, anxiety, depression; inconsistent across populations [4,5,17])	Reported [4,5]	Reported [17]	Not enough literature to conclude	Not enough literature to conclude
ADCY3 (encodes enzyme producing cAMP; associated with appetite regulation and energy balance [15,16])	Not enough literature to conclude	Not enough literature to conclude	Not reported [20]	Reported [9]
DAT1 (encodes dopamine transporter; epigenetic associations in ARFID subtypes; associated with ADHD [14])	Reported [22]	Hypothesized [7,23]	Reported [21]	Reported (epigenetic) [7]
ZSWIM6 (neurodevelopmental gene; associated with ASD, schizophrenia, intellectual disability [12,13])	Not enough literature to conclude	Not enough literature to conclude	Not enough literature to conclude	Reported [2,6]

based ARFID, whereas ADCY3, given its role in appetite regulation, may be relevant to the restrictive, low-appetite presentation. However, this candidate-level subtype mapping is speculative; no subtype-stratified genetic analyses have been conducted in adequately powered, clinically-defined ARFID cohorts. Drawing direct correspondences between specific genes and specific subtypes would substantially overstate the available evidence. These preliminary patterns warrant hypothesis-driven investigation rather than clinical inference.

A central limitation of the current evidence base is the preliminary and largely unreplicated nature of the findings. The ZSWIM6 association was identified in an autism-enriched cohort with acknowledged power and generalizability limitations, and does not establish that ZSWIM6 is a risk gene for ARFID in the general population. The DAT1 findings are epigenetic rather than sequence-level, derive from a small pilot study (n=94), and have not been independently replicated. The ADCY3 association from Bjørndal et al. [9] , while derived from a large population sample, studied ARFI-broad rather than clinically diagnosed ARFID, and the SNV heritability of 8–16% leaves the majority of phenotypic variance unexplained by the identified variants. None of these findings have been replicated in independent ARFID-specific cohorts. Interpreting any of these associations as established biological mechanisms would be premature, and the review's contribution should be understood as identifying candidate pathways for future investigation.

Although this review focuses on genetic and epigenetic factors, ARFID is unlikely to be explained by genetic variation alone. Gene–environment interactions, in which genetic predispositions interact with developmental experiences, early feeding environments, trauma, or sensory processing differences, are likely to play an important role. The trauma-based ARFID presentation is particularly illustrative: while genetic factors may contribute to temperamental or physiological vulnerability, the onset of avoidant eating following an aversive food-related experience involves an environmental trigger, and these contributions should not be conflated. Trauma associations do not imply a shared genetic etiology with other ARFID presentations. Future research should explicitly model gene–environment interactions rather than treating genetic and environmental risk factors as independent.

The identified candidate findings provide a preliminary foundation for future research. Future studies should prioritize subtype-specific genetic analyses in large, clinically defined ARFID cohorts using genome-wide approaches. Expression quantitative trait loci (eQTL) analyses may help determine whether observed associations reflect sequence-level variation or altered gene expression. Subtype-specific analyses may prove more informative than treating ARFID as a single diagnostic entity, given that its subtypes appear to differ substantially in phenomenology and potentially in neurobiology. Understanding the genetic architecture of ARFID—approached rigorously and empirically—holds the potential to transform how the disorder is understood, but the primary contribution of the current evidence is to identify plausible candidate pathways and underscore how much remains to be discovered.

This review has several important limitations. Firstly, this narrative review was conducted without pre-registration. The review did not follow a systematic approach and evidence was not synthesized in a systematic meta-analytic manner. The synthesis provided largely reflects the qualitative judgment of the author with regards to the quality of the evidence. In addition, the primary genetic studies on ARFID are small and methodologically diverse. The majority of these studies were conducted on autism-enriched or high-risk samples as opposed to clinically diagnosed individuals with ARFID. The comparative eating disorder genetics sections draw on findings from AN, BN, and BED that were not written for aiding ARFID research. Phenotypic overlap between the different eating disorders does not necessarily mean that they share genetic causes. The Harmonizome database entry for ZSWIM6 is a bioinformatics annotation resource, not a primary research study. These limitations emphasize the need for

the development of standardized diagnostic approaches, large-scale biobanks, and collaborative research dedicated specifically to ARFID.

Without further studies, no firm conclusions can be drawn regarding the genetic origins of ARFID or its subtypes. Larger and more subtype-specific studies are necessary to validate these preliminary findings. Future research should also incorporate eQTL analyses and prospective longitudinal designs to better characterize the biological mechanisms underlying ARFID development.

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