

**ARFID: A BRIEF  
EVIDENCE REVIEW FOR  
EATING DISORDERS  
AWARENESS WEEK 2024**

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# ARFID: EVIDENCE REVIEW

## Introduction

Avoidant/restrictive food intake disorder (ARFID) is a relatively new feeding and eating disorder (FED), categorised in May 2013 within the Diagnostic Statistical Manual of Mental Disorders, Fifth Edition (DSM-5). ARFID is characterised by extreme restriction in dietary intake, to the point where an individual's nutritional intake, weight/growth, and psychological/social functioning are negatively impacted. Due to the novelty of this FED, much is unknown about ARFID with limited investigation provided on disorders-related causes, symptom presentation, treatment and outcome. Widespread research and validation of standardised tools for disorder identification and recovery are highly warranted to further clinician, researcher and public understanding of ARFID.

## What is ARFID and how does it differ from other eating disorders?

Avoidant/restrictive Food Intake Disorder (ARFID) is a severe feeding and eating disorder marked by food avoidance and/or restricted food intake. Individuals with ARFID can restrict the amount of food eaten, and therefore do not get enough calories, or they can restrict the range of foods eaten and therefore do not get all the nutrients needed for maintaining health[1].

ARFID differs from the generalised term "picky eating". Many people may experience picky eating at some point in their lives. Individuals with ARFID experience severe health and psychological consequences resulting from their disordered eating, which is not the case for picky eating. Also, some individuals with ARFID are not picky about the types of foods they eat, but they limit the amount of food they eat due to low appetite or lack of interest in food.

Referrals for ARFID are increasing, but health services lack an evidence base to support individuals with ARFID effectively [2].

ARFID differs from anorexia nervosa (AN) in that dietary restriction is not motivated by body image or weight concerns. Rather, ARFID is characterised by eating problems based on symptoms such as diminished appetite or lack of interest in food, sensory-based food aversion, and fear of adverse consequences associated with eating [3]. Additionally, individuals do not need to have a low weight to meet criteria for ARFID. Although for some individuals ARFID can lead to low weight, the weight of individuals with ARFID varies across the full spectrum [3]. For example, an individual could eat a typical quantity of food, but their diet could be limited to a few foods resulting in nutritional deficiencies and/or psychological distress and would meet criteria for ARFID.

### **How prevalent is ARFID? How does this compare with other eating disorders? Are there any particular groups of the population for whom ARFID appears to be more common?**

Reported ARFID prevalence rates across literature vary highly [4], ranging between 0.3%–17.9% in global populations [5; 6; 7; 8; 9; 10; 11; 12; 13; 14; 15; 16], and 0.9%–32% in clinical eating disorder (ED) populations [17; 18; 19; 20; 21; 22; 23; 24; 25; 14; 26]. Within the UK and Republic of Ireland, the minimum incidence of diagnosed ARFID in those aged 5–17 years old is reported to be 3.09 per 100,000 individuals [27], however it should be noted that this is a preliminary finding that has not been peer-reviewed yet. Also, it should be highlighted that this represents individuals who have received a clinical diagnosis within the UK and Ireland, and there are likely to be many individuals who have not been able to access diagnostic services who may meet criteria for ARFID. There are also high proportions of individuals with ARFID also suffering from gastrointestinal (GI) symptoms, with ARFID prevalence rating from 1.5%–32% across reports in GI clinics [28; 29; 30; 31]. While encompassing a diverse range of rates, multiple prevalence reports demonstrate that rates of ARFID are higher than rates seen in other EDs, such as in AN (in which global prevalence rates range between 0.044%–1.95%) or BN (in which global prevalence rates range between 0.1%–2.95%) [32; 33].

Individuals with a DSM-5 diagnosis of ARFID are on average younger [18; 20; 4] and in contrast with other EDs, exhibit higher prevalence rates in male relative to female individuals [18; 28; 20; 34; 35; 10; 25; 36; 4]. However, recent community-based reports also observe relatively equal male-to-female distributions in those with ARFID [9; 37; 13; 15; 38], and further research is warranted to validate associations between gender and disorder prevalence. There is also a high proportion of Autistic individuals exhibiting comorbidity with ARFID, with reported ARFID prevalence rates ranging between 3% to as high as 55% [39; 40; 27].

## **What are some of the common presenting symptoms of ARFID, and how do they impact a person's physical and mental health?**

Inherent to ARFID is a persistent lack of obtaining required dietary needs for reasons separate to body image disturbances or fear of gaining weight [1; 5]. Difficulties in meeting dietary needs in ARFID is driven by either a lack of interest in food, avoidance of food based on sensory characteristics or concerns about aversive consequences of eating, such as choking [1; 41; 42; 25]. ARFID and its commonly presenting symptoms have been proposed to have three underlying profiles associated with brain function; altered regulation of appetite, differences in sensory processing (such as touch/texture/shape), and an increased activation of brain regions participating in an individual's response to fear [43]. However, research is needed to confirm whether this proposed model is correct.

Collectively, such behaviours exert a significant impact on an individual's physical health. Indeed, a common and often diagnostic consequence from restrictive eating habits displayed in ARFID is weight loss, compromised growth, significant malnutrition and/or a dependence on supplements [1; 41]. People with ARFID can experience vitamin and mineral deficiencies [44; 45]. ARFID has also been found to exert a significant impact on the individual's mental health [46; 47; 16]. A wide array of studies have reported an increased prevalence ranges in those with ARFID across mental health conditions, such as 9.1%-72.0% in anxiety disorders [48; 18; 28; 24; 12; 49; 26; 16], and 4.0%-33.0% in depression/mood disorders [48; 18; 24; 12; 49; 26]. Cooney et al. (2018) [24] reports that amongst those with ARFID, 57.1% present with one and 14.3% present with more than one co-occurring psychiatric diagnosis.

## What are the potential causes and risk factors for the development of ARFID?

While the exact causes of ARFID are not fully understood and more research is needed, several factors may contribute to its development:

**Traumatic Events:** Experiences related to food, such as food poisoning or choking incidents, can lead to the development of ARFID by creating fear or aversion to specific foods [50].

**Anxiety, Obsessive Compulsive Disorder (OCD) or Other Mental Health Issues:** Disturbances in emotional and mental well-being can contribute to ARFID. Anxiety or obsessive thoughts about food can lead to avoidance of certain foods or food groups [49].

**Autism:** ARFID is more common in autistic individuals, as sensory sensitivities and rigid eating habits which are features of ARFID are often associated with autism [51]. Nonetheless it is important to recognise ARFID as a separate diagnosis from autism, not all autistic individuals experience eating difficulties. Conversely not all individuals with ARFID are autistic.

**Genetics:** There may be a genetic component to ARFID, as some individuals may have a genetic predisposition to sensory sensitivities or food aversions. An initial twin study from Sweden has found that 79 per cent of the risk of developing ARFID could be explained by genetic factors [52]. Dinkler et al., 2023 [52] identified that the heritability (or genetic component) of ARFID is on par, and in many instances higher than heritability estimates for other EDs or other psychiatric and neurodevelopmental conditions, suggesting that genetics may play a major role in the development of ARFID.

**Neurobiology:** An initial brain imaging study has found individuals with ARFID differ from individuals without ARFID in brain activation patterns within brain regions involved in food cue and reward processing [53].

Despite the aforementioned evidence-based risks associated with ARFID, researchers continue to face challenges regarding how to characterise ARFID and clearly distinguish between risk factors in picky eating and risk factors in ARFID. Research is highly warranted to better specify disorder-specific risks and follow-up on additional speculated risk factors of ARFID from previous literature.

### **To what extent is there a connection between neurodivergence and ARFID specifically?**

There is a notable overlap between ARFID and neurodevelopmental conditions. Picky eating, or food selectivity, has been consistently reported to be more frequent and more persistent in autism and ADHD compared to neurotypical peers [54; 55; 56; 57]. With the inclusion of ARFID in the DSM-5, such restrictive eating habits became potentially clinically relevant, accounting for individuals who significantly restrict their food intake without fear of weight gain associated with other EDs [34].

ADHD has been reported to be over-represented in ARFID populations, with recent studies suggesting that ADHD was found to be significantly more prevalent in ARFID populations than other EDs, such as AN populations [26; 58]. Beyond this, there is a notable lack of research into the overlap between ADHD and ARFID. More research has been done exploring the link between autism and ARFID, perhaps due to the similar sensory sensitivities implicated in both autism [1; 59; 60] and ARFID [34] that highlight the possible overlap between Autism and ARFID [49]. Subsequent prevalence estimates of ARFID in Autistic populations have been reported from 3.0-54.8% [18; 49; 61; 4], with a recent genetic study also finding that up to 17% of parents of Autistic children are at heightened risk, suggesting a lifelong risk of disordered eating [39]. Beyond this, however, research into the co-occurrence of Autism and ARFID remains in its infancy. The prevalence of ARFID in adolescence and adulthood is largely unknown, and underlying mechanisms remain poorly understood.

## **What is the typical age of onset of ARFID, and how long does the illness last?**

Extant literature reports the typical age of onset to be younger in those with ARFID relative to EDs such as AN or bulimia nervosa (BN) [48; 18; 20; 3; 62]. Since the introduction of the ARFID diagnosis within the DSM-5, studies comparing age of diagnosis between those with ARFID and those with AN denote a minimum difference of 2 years in disorder onset between those with ARFID vs those with AN, ranging from 12.9 vs. 15.6 years regarding age at diagnosis [48], 8.3 vs. 16.4 years when individuals presented for treatment, [3] and 14.3 vs. 18.8 years of age from multiple clinical populations [62] respectively. In line with reported trends that the age of onset of ARFID is younger than in other EDs, it is important to note that this DSM-5 disorder diagnosis replaced the DSM-IV diagnosis of “feeding disorder of infancy or early childhood”, encompassing those under 6 years of age. Contrary to the term ARFID replaces, ARFID can be diagnosed across the lifespan, following recognition that ARFID symptoms affect individuals of all ages and not just those earlier in development [1; 63].

Duration of illness for those with ARFID is reported to be similar [64], or longer [20; 65] than duration observed in other EDs [38], with Lieberman et al. (2019) [65] identifying a mean difference of 22.89 months in illness duration between those with ARFID vs. those with AN (29.28 months in ARFID vs. 6.39 months in AN). However, lacking evidence-based research available across the lifespan warrants further investigation to further understand the duration of illness and additional long-term outcomes associated with ARFID. Adolescents with low-weight ARFID have been reported to experience similar long-term outcomes as those with AN [64], while children with ARFID have been reported to experience longer duration of illness than children with AN (26.3 vs. 6.4 months) [65]. Importantly, children with ARFID also display long-standing histories of gastrointestinal symptomatology [28; 65; 30; 31] relative to other EDs, which may further exacerbate disorder duration.

## **How does ARFID impact a person’s quality of life, including their social relationships, academic or occupational performance, and overall well-being?**

Interference to psychosocial functioning is part of the diagnostic criteria for ARFID outlined in the DSM-5 [1], highlighting the impact that ARFID has beyond physical and mental health. Studies demonstrate interference to social and occupational functioning (e.g., 66; 64], with one mixed-method study reporting 100% of participants experienced difficulties in family functioning, 46% in occupational functioning and 41% in social functioning, with all participants fulfilling the diagnostic criterion of psychosocial interference for an ARFID diagnosis [25]. Lower school functioning has also been reported in children aged 8-10 years old with ARFID when compared to healthy controls [66], while general health-related quality of life has been found to be significantly impacted in childhood [66] with recent evidence to suggest this becomes more pronounced during adulthood [16]. Compared to other EDs such as AN or BN, one study found that individuals with ARFID reported with lower mental-health-related, but similar physical-health-related quality of life [7].

## **What are the current evidence-based treatments available for ARFID and how effective are they in improving outcomes?**

Current research for evidence-based treatments is lacking. This is one of the reasons why neither the National Institute of Health and Care Excellent (NICE) or the Scottish Collegiate Guidelines Network (SIGN) guidelines on the treatment of EDs make any recommendations on the treatment of ARFID. The omission of ARFID from such guidelines perpetuates the limited and variable NHS service provision for ARFID.

Evidence-based interventions for ARFID are limited by low sample numbers and wide ranges of measurements used to evaluate disorder-related outcome [67]. Despite impediments, current reports highlight advances made in treatment approaches over the last decade [67], and highlight the efficacy of cognitive-behavioural therapy (CBT) [43] and family-based treatment [68; 69; 70], food exposure [43], as well as psychological intervention (consisting of positive reinforcement [68; 69]) and anxiety management [71; 72].

Behavioural interventions such as positive/contingent reinforcement, group- and parent-based intervention, psychoeducation and goal setting, report positive outcomes such as increases in BMI [73; 74], a 38% increase in caloric needs consumed orally [74], and significant improvements in eating behaviour reported by the Children's Eating Behaviour Questionnaire (CEBQ) (Enjoyment of Food CEBQ score increased from 2.38 to 2.85; Food Fussiness CEBQ score decreased from 4.74 to 4.05) [75].

Interventions utilising CBT report similar positive outcomes in those with ARFID, such as an increase in BMI (17.65 at baseline vs. 19.49 at discharge) [76] and weight (107.45 lbs at baseline vs. 118.83 lbs. at discharge) in low-weight ARFID, increased dietary diversity with 17–18 new foods introduced pre- vs. post-treatment [77; 76], a reduced fear of trying new foods reported by the Food Neophobia Scale (71 at baseline vs. 43 at two-month follow-up [78]; 61 at baseline vs. 53.5 at discharge [76]), and reductions in clinical ARFID symptoms via scores in the Nine-Item ARFID Screen (15 at baseline vs. 10 at two-month follow-up) [78]. ARFID outcome extends to psychological well-being, with individuals reporting reductions in post-treatment anxiety, depression, and general psychopathology [70; 67].

Importantly, extant literature has focused heavily on treatment-based approaches during childhood and early adolescence, with further research warranted to understand ARFID recovery and outcome during adulthood [67].

### **What are the potential barriers to treatment for individuals with ARFID, such as stigma, lack of awareness, or limited access to specialised care?**

Due to the novelty of the ARFID diagnosis relative to other EDs, there are multiple barriers to ARFID treatment, such as lack of awareness and education of the FED, limited access to standardised care pathways, and existing stigma concerning how people perceive (or rather fail to perceive) the severity of ARFID [79; 80; 81]. Healthcare professionals have not only struggled to characterise or diagnose children with ARFID, but also found difficulty in identifying professionals or organisations capable of further assessment, treatment and support [80]. Professionals may experience further challenges in identification of ARFID, as clinicians need to recognise symptoms of this FED in the context of low-weight or low-BMI states, which are frequently seen in individuals with ARFID but are not necessary conditions for diagnosis [81]. Additionally, the complexity of ARFID symptomatology may contribute towards individuals "falling between the gaps" of specialised healthcare services.

Stigmatisation of ARFID symptomatology may also act as a contributing barrier towards treatment and recovery. Research has clarified upon distinctions between the FED and non-clinical forms of "picky eating" [82], but population-based views of ARFID contrast reported disorder severity. When asked to rate perceptions of EDs, a large-scale survey of young adults reported ARFID being perceived as significantly less pathological than AN [79] despite reports of similar levels of malnutrition [36] and outcome [83; 81]. Due to the multitude of barriers presented to those with ARFID seeking treatment including lack of literature availability and under-recognition of ARFID, it has not currently been determined as to how long it takes for individuals to seek diagnosis, help and further support.

## **What are the long-term outcomes for individuals with ARFID, including rates of recovery, relapse, mortality, and co-occurring mental or physical health conditions?**

While evidence-based determinations of ARFID outcome are still being established, existing literature details that those with ARFID experience similar outcome to those with other EDs, particularly AN, with varying recovery rates. Long-term follow-up retrospective studies report that those with ARFID exhibit similar outcome to those with AN, but in contrast exhibit little diagnostic crossover to other EDs [64; 81], and report with more developmental disorders/family history of mental disorders relative to those with AN (nARFID=46.2% vs. nAN=3.8%/nARFID=46.2% vs. nAN=17.7%) [83], as well as higher presence of additional psychiatric diagnoses ([nARFID=26.3% vs. nAN=24.3%] 64]. Recovery rates in those with ARFID relative to other EDs vary, with evidence-based determinations reporting lower ([Recovery Rate: nARFID=73.7% vs. nAN=78.4%] 64; [Diagnosis Persistence: nARFID=84% vs. nAN=62%] 81) and higher ([Recovery Rate: nARFID=77% vs. nAN=43%] 83; [Rehospitalisation: nARFID=0% vs. nAN=62%] 84) recovery rates in those with ARFID relative to those with AN upon follow-up. Existing evidence firstly suggests that those with ARFID exhibit similar rates of recovery and disorder outcome as with other restrictive EDs such as AN, highlighting the need for further intervention-based developments. Secondly, the heightened presence of additional psychiatric and developmental disorders seen in ARFID relative to other EDs assists in explaining why this FED was designated as a specific diagnosis in the DSM-5.

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