Chapter 1

What Is ARFID?

ARFID made its diagnostic debut in the *DSM-5* (American Psychological Association, 2013) as a reformulation of *DSM-IV* feeding disorder of infancy and early childhood. Although *DSM-5* provides a fairly clear definition of ARFID, research on its prevalence, distinction from classical eating disorders, and etiology is still emerging.

Definition

ARFID is defined by a pattern of eating that is limited in variety (e.g., avoidance of specific foods) and/or volume (e.g., restriction of amount) and associated with important medical and psychosocial consequences (*DSM-5* criterion A). Individuals with ARFID typically attribute their avoidant or restrictive eating pattern to the sensory characteristics of food (i.e., sensory sensitivity), a fear of aversive consequences of eating (e.g., choking, vomiting), and/or a lack of interest in eating or food (e.g., low hunger, lack of enjoyment of eating). But ARFID is more than just picky eating or skipping a meal once in a while. To qualify for the ARFID diagnosis, a person's pattern of eating must be associated with significant negative consequences. These include one or more of the following: significant weight loss/growth falloff, nutritional deficiencies, dependence on tube feeding or reliance on energy-dense supplements, or psychosocial impairment (*DSM-5* criterion A). Avoidant/restrictive eating in ARFID cannot be due to lack of available food or cultural norms (*DSM-5* criterion B), nor can it be motivated by weight and shape concerns (*DSM-5* criterion C). If avoidant/restrictive eating co-occurs with other psychiatric or medical illnesses, the eating disturbance must be severe enough to require independent clinical attention (*DSM-5* criterion D) to warrant a comorbid ARFID diagnosis.

As a diagnostic group, ARFID is very heterogeneous. It can include the low-weight, short-stature grade schooler with a low appetite whose babyhood reflux challenged his early feeding, and who now limits his diet to bland white foods he has learned are easy on his stomach. Mealtimes in his family are tense because he is unable to eat the meal that the rest of the family members are eating and his parents vacillate between vehemently pressuring him to eat more and exasperatedly leaving him alone. ARFID also includes the slender high schooler with celiac disease who, following a gluten-contamination episode, has significantly restricted her already narrow diet and precipitously lost weight and incurred vitamin deficiencies. It may also include the overweight young adult with an iron deficiency who has been reluctant to enter the dating world due to embarrassment about his intake of so-called kid foods and his avoidance of all fruits, vegetables, and meats. While these cases are unified by their avoidant/restrictive eating patterns, the rationales for restriction and the medical and psychosocial sequelae differ.

In 2013, ARFID supplanted *DSM-IV's* feeding disorder of infancy or early childhood, which was sometimes used in clinical practice but rarely studied in the scientific literature. Feeding disorder of infancy or early childhood had been defined by weight loss or failure to gain weight as expected; lack of medical or psychiatric comorbid diagnosis that could account for the feeding disturbance; and an onset before six years old. However, the diagnosis was too narrow to be clinically useful. For example, one diagnostic study of individuals presenting to a pediatric feeding disorders clinic found that just 12% met criteria for feeding disorder of infancy or early childhood (Williams, Riegel, & Kerwin, 2009). The others presented with clinically significant feeding difficulties that fell outside the confines of the diagnosis. In response to this diagnostic dilemma, the *DSM-5* Work Group used the new ARFID diagnosis to both revise and expand upon this earlier diagnostic category in several important ways. First, while low weight or failure to

gain weight is a common sequela of avoidant/restrictive eating, other complications can occur. The ARFID diagnosis allows for the eating pattern to be associated with nutrition deficiencies, reliance on enteral feeding or nutrition supplements, or psychosocial impairment. Importantly, this diagnostic reformulation acknowledged that food avoidance/restriction does not always or only lead to weight loss, but can be associated with normal weight or even overweight or obesity. Indeed, some individuals with ARFID may remain in the healthy weight range via the use of tube feeding or energy-dense supplements, while others, reliant on carbohydrates or energy-dense processed foods, carry excess weight. Supportive evidence that feeding difficulties can occur across the weight spectrum includes results from one study of youth presenting to a pediatric feeding disorders clinic, which found that 71% were not underweight (Williams et al., 2009).

Notably, deletion of the age-of-onset criterion validated that individuals of all ages can have clinically significant avoidant/restrictive eating that does not always begin in early childhood. Many individuals with frank food avoidance/restriction who would now be diagnosed with ARFID were effectively diagnostic orphans of *DSM-IV*. For example, the adult who had been a relatively normal eater before the experience of a choking episode, which resulted in food avoidance and profound weight loss, may have straddled the *DSM-IV* eating disorder not otherwise specified (EDNOS) and specific phobia diagnoses. The EDNOS diagnosis would have been too vague to meaningfully capture his presentation, while the specific phobia diagnosis would have underemphasized his weight loss. By contrast, the ARFID diagnosis parsimoniously characterizes the full problem.

Finally, *DSM-5* noted that ARFID *can* occur in the context of medical or psychiatric comorbidity if the eating problem requires independent clinical attention. The ARFID diagnosis does not presume the etiology of the feeding disturbance, and thus promotes detection of clinically significant disturbances in groups that likely went unrecognized under *DSM-IV* feeding disorder criteria. Indeed while some psychiatric or medical populations may be at greater risk for eating or feeding difficulties than others, most of these issues do not warrant treatment that is outside the scope of what would be expected based on the primary diagnosis. In a recent record review of more than 2,000 youth ages 8–18 years presenting to 19 Boston-area pediatric gastrointestinal clinics for an initial evaluation, our research team found just 1.5% met criteria for ARFID (Eddy et al., 2015). Although eating and feeding issues were rife in this medical population, those with a frank avoidant/restrictive eating disorder stood out.

Feeding or eating difficulties are common in childhood, occurring in roughly 25% of youth (Chatoor, 2002; Micali et al., 2011). However, ARFID is distinguished by its persistence and clinical severity. Childhood selective eating is prevalent but often improves during middle childhood, even without treatment (Jacobi, Schmitz, & Agras, 2008; Mascola, Bryson, & Agras, 2010). By contrast, the more severe food avoidance or restriction in ARFID does not remit on its own. Instead, the food avoidance and restriction that define ARFID can lead to medical or mental health consequences that further exacerbate food avoidance and restriction and serve to maintain the illness (Thomas et al., 2017a). Risks of such eating patterns include poor growth, diabetes, cardiovascular disease, fatigue, poor self-esteem, family mealtime conflict, peer social isolation, and difficulties with relationships and work. Indeed, quality of life is significantly impacted in ARFID. In an epidemiological study, individuals with ARFID (n = 46, ages 24–60 years, living in Australia) had lower mental health–related quality of life and more days of being unable to function due to emotional or physical health problems than people without eating disorders (Hay et al., 2017). Similarly, in an online study, adults with symptoms of ARFID (n = 82) self-reported greater internalizing distress than those without ARFID, and comparable levels of distress to individuals with symptoms of other eating disorders (Zickgraf, Franklin, & Rozin, 2016).

Much of what is known about avoidant or restrictive eating is based on the feeding disorders literature prior to 2013. While the ARFID diagnosis is new, the research in this area is nascent and has proliferated since the publication of *DSM-5*.

Prevalence

Although little epidemiological research has been conducted to date, available data suggest that ARFID is as common as other better-known eating disorders. In an Australian population-based survey of male and female adolescents and adults ages 15 years and older, Hay et al. (2017) found that the three-month point prevalence of ARFID was 0.3% (95% confidence

interval 0.1–0.5) in 2013 and 0.3% (95% confidence interval 0.2–0.6) in 2014. Notably, these prevalence estimates were similar to those for other specific eating disorders (0.4% and 0.5% for anorexia nervosa, and 1.1% and 1.2% for bulimia nervosa), and lower than for the heterogeneous other specified feeding or eating disorder (OSFED) category (3.2%). A second published study reported a point prevalence of 3.2% (i.e., 46 of 1,444) in children ages 8–13 years surveyed through schools in Switzerland in which students self-reported symptoms consistent with an ARFID diagnosis via questionnaire (Kurz et al., 2015). Youth with self-reported ARFID were more likely to be either underweight or overweight compared to youth who did not report ARFID symptoms (Kurz et al., 2015). In contrast to other eating disorders, which predominate in females, in both of these epidemiological studies, males and females were equally likely to be diagnosed with ARFID (Hay et al., 2017; Kurz et al., 2015). In fact, in our own team's study at Boston-area pediatric gastrointestinal clinics, the majority of youth with ARFID were male (22 of 33; 67%) (Eddy et al., 2015).

A handful of studies have examined ARFID prevalence in specialty services via medical record review, estimating that roughly 5% of children (Norris et al., 2014) and up to 15% of adolescents (Fisher et al., 2014; Forman et al., 2014; Ornstein et al, 2013) evaluated in pediatric or adolescent medicine eating disorder programs could be diagnosed with ARFID based on a retrospective application of criteria. ARFID was found even more frequently (22.5%) in a record review of 7–17-year-olds participating in a partial hospital (day) program for eating disorders (Nicely et al., 2014).

While less is known yet about the relative occurrence of ARFID within different racial or ethnic minority groups, ARFID has already been reported outside North America, Europe, and Australia. For example, Nakai et al. (2016) reported that between 9% and 11% of individuals aged 15–40 years seeking treatment for an eating disorder through a hospital-based eating disorder program in Japan could be diagnosed with ARFID. Similarly, among adults receiving inpatient eating disorders treatment in Japan, Tanaka et al. (2015) reported that 8.9% were diagnosed with ARFID. Further, a recent case report documented ARFID in a school-aged boy of Colombian descent presenting for treatment in Canada (Schermbrucker et al., 2017).

Thus, these emerging data demonstrate that ARFID occurs in males and females in the general pediatric, adolescent, and adult population, and furthermore that individuals with ARFID present to eating disorder services. Notably, the majority of these published studies capture patient encounters occurring before the publication of *DSM-5* and, therefore, prior to both clinical and popular recognition of ARFID. Taken together, these findings suggest that the actual prevalence of ARFID may be underestimated in published reports.

Distinction from Classical Eating Disorders

ARFID is different from anorexia nervosa, bulimia nervosa, binge eating disorder, and related forms of OSFED. The primary distinction is that, in ARFID, avoidant or restrictive eating behaviors are not motivated by shape or weight concerns. Whereas desire for a thin ideal drives typical dieting, and overvaluation of body shape and weight is considered to be a core feature of classical eating disorders, in ARFID, shape and weight concerns are typically absent or within the normal range. The prototypical low-weight patient with ARFID, characterized by lack of interest in eating, may endorse unhappiness about being so thin and feel proud and visibly happy with weight gain during treatment. By contrast, the low-weight patient with a classical eating disorder will express frank fat phobia or engage in behaviors that thwart weight gain, and will typically experience intense anxiety during weight restoration. Furthermore, the preferred foods for individuals with ARFID, which are often energy-dense, high-fat, and high-carbohydrate, differ vastly from those preferred by individuals with more classical eating disorders, which are often low-calorie and include foods that individuals with ARFID actively avoid (e.g., fruits and vegetables). Indeed, as part of an ongoing study of low-weight eating disorders, our team examined food records over a four-day period and found that individuals with ARFID consumed a significantly smaller percentage of their calories from protein compared to those with anorexia nervosa (Izquierdo et al., 2018). Notably, ARFID is also distinguished from other feeding disorders including pica – characterized by intake of non-nutritive, non-food substances – and rumination disorder, which is defined by repeated regurgitation and re-chewing, re-swallowing, or spitting out previously ingested foods.

Clinical impressions about the distinctions between ARFID and classical eating disorders are borne out in emerging data as well. Data from adolescent medicine clinic record reviews suggest that those with ARFID are generally younger (Forman et al., 2014; Norris et al., 2014) and more likely to be male (Forman et al., 2014; Norris et al., 2014) than those with other eating disorders. Compared to individuals with anorexia nervosa or bulimia nervosa, individuals with ARFID predictably score lower on measures of eating disorder psychopathology (Nakai et al., 2016; Nicely et al., 2014; Ornstein et al., 2017). One contrast of clinical interest with diagnostic implications exists between individuals with putative ARFID and those with anorexia nervosa who deny or minimize their experience of weight and shape concerns (non-fat phobic anorexia nervosa; Becker, Thomas, & Pike, 2009). Because both disorders present with low weight in the absence of frank body image disturbance, differential diagnosis can be challenging (Thomas, Hartmann, & Killgore, 2013). Recognizing that explicit endorsements (e.g., of fat phobia) do not always match internal beliefs, our team used measures of implicit associations with dieting to explore between-group differences. In a sample of low-weight adolescent females, we tested the hypothesis that individuals with anorexia nervosa, whether or not they explicitly express fear of weight gain, have implicit beliefs biased in favor of thinness and dieting, while those with ARFID do not. Consistent with our hypothesis, we found that implicit bias toward dieting was high among those with anorexia nervosa and did not differ between those who did versus did not endorse fat phobia. By contrast, in ARFID, bias toward dieting was lower than in anorexia nervosa and did not differ from healthy controls (Izquierdo et al., 2017).

Yet in spite of endorsing different rationales for food avoidance and restriction, a subset of individuals with ARFID are just as low weight as those with anorexia nervosa (Nakai et al., 2016; Nicely et al., 2014). Interestingly, available data suggest that individuals with ARFID and ARFID-like symptoms have typically lost less weight immediately prior to seeking treatment compared to individuals with anorexia nervosa or related presentations (Pinhas et al., 2017; Strandjord et al., 2015). Instead, those with low-weight ARFID appear to have been chronically low weight prior to seeking treatment, rather than experiencing the acute weight loss that so often characterizes anorexia nervosa (Strandjord et al., 2015). Speaking to medical severity, one preliminary study found that at presentation for treatment, 77% of individuals with ARFID (20/26) had bone density Z-scores of < -1. A further 25% (7/26) had Z-scores of < -2, indicating they were already in the osteoporosis range (Norris et al., 2014). Bone loss was greater in the ARFID group than in those with anorexia nervosa in that particular study (Norris et al., 2014) but comparable to rates of osteopenia and osteoporosis in anorexia nervosa that have been reported in the literature (Misra & Klibanski, 2014).

The psychiatric comorbidity profiles of individuals with ARFID also differ somewhat from those of individuals with other eating disorders. Clinically speaking, anxiety and depression seem to co-occur in ARFID just as often as they do in anorexia nervosa or bulimia nervosa. However, other conditions including autism spectrum disorder, oppositional defiant disorder, and attention deficit/hyperactivity disorder may be more common in ARFID than in the other eating disorders. The high prevalence of eating and feeding difficulties in autism spectrum disorder is well documented (e.g., Berry et al., 2015; Buie et al., 2010; Emond et al., 2010; Lucarelli et al., 2017; McElhanon et al., 2014). However, little data about psychiatric comorbidity and personality styles in ARFID compared to the other eating disorders are available. In a sample of 36 children and adolescents presenting for one of our team's ongoing research studies of ARFID, we found that 39% met criteria for a comorbid psychiatric disorder via structured clinical interview, with anxiety disorders (9/36) and attention deficit/hyperactivity disorder (4/36) being most common. Our data may underestimate the comorbidity with lower functioning autism spectrum disorders, as IQ < 70 was an exclusion criterion for the study. Very little data speak to different cognitive styles in individuals with ARFID versus anorexia nervosa. In a second study, we compared low-weight females with ARFID to those with anorexia nervosa on a task of monetary delay discounting, that is, the degree to which the subjective value of a reward decreases based on delay of receipt. In anorexia nervosa, delay discounting is often low; in other words, those with anorexia nervosa have a tendency to forgo smaller immediate rewards (e.g., high-calorie foods) in favor of larger long-term rewards (e.g., a thin body). By contrast, our preliminary data suggest that those with ARFID are more similar to healthy controls in that they were more likely to choose immediate than

delayed rewards, in comparison to those with anorexia nervosa. These findings suggest individuals with ARFID may have less self-control or greater impulsivity than those with anorexia nervosa (who are often abstemious) (Coniglio et al., 2017). These findings seem consistent with our clinical impression that, in ARFID, food restriction is less calculated and purposeful than in classical eating disorders.

Finally, diagnostic crossover among the classical eating disorders has been well documented (Eddy et al., 2008; Milos et al., 2005), but whether crossover from ARFID to any of the other eating disorders commonly occurs has not yet been studied. There are data to suggest that there may be continuity between childhood feeding problems and adolescent or adult eating disorders. Some longitudinal studies suggest that childhood digestive problems and selective eating (eating little, pickiness, eating slowly, low interest) increase the risk for anorexia nervosa, while digestive problems, pica, and dieting increase the risk for bulimia nervosa (Marchi & Cohen, 1990). Further, another study found that childhood conflicts around eating and difficulty with family meals, which were both associated with picky eating, also increased the risk of developing eating disorders in adolescence (Kotler et al., 2001). Conversely, it is possible that some of those with ARFID, particularly those who are low weight, may develop secondary body image disturbance during the course of illness or even treatment. In an ongoing National Institutes of Health (NIH)-funded study entitled 'Neurobiological and Behavioral Risk Mechanisms of Youth Avoidant/Restrictive Eating Trajectories' (1R01MH108595), our team is actively exploring the course of illness for those with ARFID and thus more information on the potential for crossover is emergent.

Etiology

The causes of ARFID are unknown. Some data suggest that there may be biological contributors, but biomarkers have been largely understudied and their putative relationship to frank ARFID, versus picky eating, is not known. While there are no twin or adoption studies of ARFID specifically, twin and adoption studies of related traits suggest that taste preferences are at least partially genetic (Breen, Plomin, & Wardle, 2006). Anecdotally, many of our patients have shared that their first-degree relatives (e.g., parents, siblings) also have avoidant/restrictive eating, further highlighting the possibility of a genetic component to risk. We hypothesize that certain biological factors reflected in sensory sensitivity, anxiety, and low appetite play an etiologic role, and these hypotheses are described more fully in Chapter 4.

A body of research has focused on the role of the family meal environment and family dynamics around eating as they relate to early feeding and development of healthy or unhealthy eating behaviors in typically developing youth (see Savage, Fisher, & Birch, 2007 for reviews). Indeed, parents provide the first models of eating, creating an atmosphere (e.g., warm and relaxed versus harsh and tense) and setting expectations for mealtimes in terms of food volume, variety, and pace. Ellyn Satter has described a *division in responsibility in feeding* in which parents set the expectation of *what, when*, and *where* meals will be served, and then children decide *whether* and *how much* to eat (Satter, 1986). To promote healthy eating in normally developing youth, parents can make a variety of healthy foods available and model eating them in variety and in healthy amounts (Savage et al., 2007). There is evidence to suggest that parents with greater food neophobia have children who have greater food neophobia and higher levels of picky eating (Dovey et al., 2008). Further, there are data to suggest that in population-level studies, parental pressure to eat is associated with child low weight, whereas parental restrictions on child eating are associated with increased weight gain (Birch & Fisher, 2000). However, we highly doubt that parents cause ARFID. Indeed, the application of these data and recommendations to youth with the more severe form of picky eating and frank ARFID is not clear. In our experience, by the time families present for treatment, they routinely describe having tried *everything* – pressure, no pressure; rewards, punishment – suggesting that the recommendations that may be useful for most healthy children cannot be readily applied to those with ARFID.

Indeed, other environmental factors including agricultural subsidies, fast-food advertising, busy two-career families, and the high cost of fruits and vegetables compared to processed foods make it difficult for parents to present their children with a wide variety of healthy foods at all eating opportunities (Brownell & Horgen, 2004). Furthermore, the rise of 'kids' menus' and child-targeted food products (e.g., pre-packed lunch packs, squeezable yogurts and purees) that encourage homogenized food

choices in youth may also contribute to the development of avoidant or restrictive eating patterns.

At the intersection of biological and environmental contributors are medical and psychiatric comorbidities that can challenge eating and feeding, setting the stage for the development of ARFID. For example, food allergies that require certain restrictions may increase vulnerability in some to more food avoidance or restriction. Similarly, autism spectrum disorder, which is associated with increased sensory sensitivity and cognitive inflexibility, sets the stage for selective eating, which will become entrenched for some. In addition, medical advances have meant an increase in preterm births in the past few decades. Individuals who are born early are more likely to have low birth weight and medical complications that can challenge early nutrition (Kumar et al., 2017; Villar et al., 2018), which may also increase risk for feeding disorders.

In sum, the clinical significance of ARFID is evident in the pattern of nutritional and psychological compromise it leaves in its wake. All available evidence demonstrates that ARFID is a real, identifiable, and heterogeneous problem. ARFID appears to occur at rates similar to other eating disorders and to affect males and females of all ages. While it shares some commonalities with classical eating disorders – including patterns of aberrant food avoidance and restriction, medical risks, and comorbid anxiety – ARFID is also clearly distinct. Unlike the classical eating disorders, the core psychopathology of ARFID is not overvaluation of weight and shape, and clinical impressions coupled with preliminary data demonstrate comorbidity and personality characteristics that also separate ARFID from anorexia nervosa and bulimia nervosa. Of course, similarities between ARFID and feeding, eating, and anxiety disorders may also suggest the possible efficacy of similar treatment strategies.