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Avoidant/Restrictive Food Intake Disorder: A Three-Dimensional Model of Neurobiology with Implications for Etiology and Treatment

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Abstract

Purpose of review: DSM-5 defined Avoidant/Restrictive Food Intake Disorder (ARFID) as a failure to meet nutritional needs leading to low weight, nutritional deficiency, dependence on supplemental feedings, and/or psychosocial impairment. We summarize what is known about ARFID and introduce a three-dimensional model to inform research.

Recent findings: Because ARFID prevalence, risk factors, and maintaining mechanisms are not known, prevailing treatment approaches are based on clinical experience rather than data. Furthermore, most ARFID research has focused on children, rather than adolescents or adults. We hypothesize a three-dimensional model wherein neurobiological abnormalities in sensory perception, homeostatic appetite, and negative valence systems underlie the three primary ARFID presentations of sensory sensitivity, lack of interest in eating, and fear of aversive consequences, respectively.

Summary: Now that ARFID has been defined, studies investigating risk factors, prevalence, and pathophysiology are needed. Our model suggests testable hypotheses about etiology and highlights cognitive-behavioral therapy as one possible treatment.

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Keywords

Avoidant/restrictive food intake disorder; eating disorder; feeding disorder

Introduction

Avoidant/Restrictive Food Intake Disorder (ARFID) was introduced to the psychiatric nomenclature four years ago[1] as a reformulation of Feeding Disorder of Infancy and Early Childhood.[2] ARFID expanded upon Feeding Disorder of Infancy and Early Childhood to acknowledge that avoidant and restrictive eating symptoms can occur across the lifespan. DSM-5 provides three example presentations of ARFID, which can occur independently or in combination. Specifically, individuals with sensory sensitivity may avoid eating specific foods—often meats, vegetables, and/or fruits—due to aversions to specific tastes, textures, or smells. Others with ARFID may restrict the amount they eat due to lack of interest in eating or low appetite. Still others may avoid specific foods or stop eating entirely following a traumatic experience with eating, such as choking, vomiting, or other forms of gastroenterological distress. In all cases, to warrant an ARFID diagnosis, the avoidant and restrictive eating must lead to significant medical or psychosocial problems that require independent clinical attention. Examples of ARFID sequelae that would meet diagnostic criteria include poor growth and/or low weight, vitamin deficiencies, dependence on tube feeding or high-energy supplements to meet calorie needs, and psychosocial impairment (e.g., avoidance of eating opportunities at work or school; difficulty eating with others). Below we summarize what is currently known about ARFID and highlight future research directions including our new three-dimensional model of neurobiology with implications for etiology and treatment.

What is currently known?

Epidemiology

Given its status as a recently defined disorder, ARFID has not been included in any large-scale epidemiological studies. Therefore, its incidence and prevalence in the general population are unknown. A questionnaire-based study recently investigated the prevalence of ARFID in a primary school setting among 8–13 year olds in Switzerland, and found that 3.2% met criteria for ARFID via self-reported symptoms.[3] The prevalence of ARFID in healthcare settings is generally higher. For example, a series of case reviews and clinical studies across eating disorder treatment programs in North America found that between 7.2% and 17.4% of patients across sites had ARFID[4,5,6] In a similar retrospective chart review of individuals seeking treatment for eating disorders in Japan, 11% met criteria for ARFID.[7] Further, ARFID was even more common (22.5%) among youth in a day treatment program for eating disorders.[8] By contrast, a retrospective review of 2,231 consecutive referrals (aged 8–18 years) to pediatric gastrointestinal clinics in the Boston area showed an ARFID prevalence of only 1.5%.[9] A recent latent class analysis of three pediatric surveillance studies (in which pediatricians and child psychiatrists were asked to report on any children < 12 years with a newly diagnosed restrictive type eating disorder) performed across Canada, the United Kingdom, and Australia suggested that one of two identified

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clusters representing between 25–34% of children with incident restrictive type eating disorders mapped onto symptoms consistent with ARFID.[10] These studies suggest that despite variation in estimates, ARFID is commonly seen in clinical settings and might be common among children in the general population. Further studies are needed to investigate the epidemiology of ARFID in children, adults, and the elderly.[11]

Assessment

There are currently no validated assessment tools for the specific psychopathology of ARFID. This gap impacts identification of the condition in clinical settings, evaluation of treatment efficacy, and ascertainment of epidemiology and natural course. Prior research suggests that ARFID can be differentiated from anorexia nervosa (AN) based on the rationale for food restriction. A small study of treatment-seekers completing the Eating Pathology Symptoms Inventory (EPSI[12]) found that while individuals with AN endorsed higher scores than those with ARFID on EPSI Restraint (a self-report measure of purposeful dieting), the two groups scored similarly on EPSI Restriction (a self-report measure of actual deficits in calorie intake).[13]

A recently developed questionnaire for school-aged children, the Eating Disturbances in Youth-Questionnaire (EDY-Q), has been used as a self-report measure to identify ARFID,[3] and was shown to have a four-factor structure, possibly distinguishing emotional food avoidance, selective eating, food restriction due to fear of aversive consequences, and weight problems.[14] The Eating Disorder Assessment for DSM-5 (EDA-5)[15] can be used to confer ARFID diagnoses; however its psychometric properties in relation to ARFID have not yet been tested. A new semi-structured multi-informant interview—the Pica, ARFID, and Rumination Disorder Interview (PARDI)—has recently been developed to diagnose ARFID in children and adults. The PARDI also provides dimensional ratings of relevant presentations (selective sensory-based eating; low interest or low appetite food avoidance; and restrictive eating due to fear of aversive consequences) and overall ARFID severity.[16]

Course and Outcome

At this point, little is known about the longitudinal course and outcome of ARFID. Selective eating (though not necessarily ARFID) in childhood has been identified as a risk factor for future psychiatric symptoms.[17] While picky eating (accepting a food one day but eschewing it the next, or systematically avoiding some non-preferred foods, such as broccoli) is common in youth—particularly in preschoolers—children typically expand their diets as they mature. However, there are no data evaluating spontaneous remission rates among individuals with frank ARFID, which differs from developmentally normative picky eating in that individuals with ARFID may not reincorporate previously dropped foods, or may avoid entire categories of food (e.g., all vegetables). Indeed, in our clinical experience, many individuals who present for treatment in adulthood report longstanding selective eating patterns dating back to infancy or childhood (e.g., refusal of all but one type of infant formula, difficulty with transition to solid foods).

ARFID can lead to severe medical sequelae due to malnutrition.[6] Retrospective studies have shown that these patients are at risk for amenorrhea,[18] bradycardia, prolonged QT interval

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on electrocardiogram, and electrolyte abnormalities such as hypokalemia.[19] One recent case report of ARFID in an adolescent male of normal weight demonstrated vitamin A, E, B12, D, K, and folate deficiencies as well as spinal cord degeneration secondary to his significantly restricted diet.[20] Indeed, while weight loss and faltering growth are common signs of avoidant or restrictive eating, nutritional deficiencies can be observed independent of low weight. In our clinical practice we have also seen nutritional excesses, such as elevated mercury levels due to repeated consumption of high-mercury foods (e.g., tuna) in the context of an otherwise very limited diet. Prior research suggests that ARFID often develops in the context of childhood medical problems that lead to gastrointestinal dysfunction or pain.[20,21] Clinically we often find that individuals with the fear of aversive consequences presentation initially begin restricting intake due to their belief that certain foods will cause them pain or discomfort but then find that, rather than resolving the pain, restriction and associated weight loss only exacerbate gastric motility problems and make regular eating even more challenging.

Psychiatric comorbidities, including anxiety disorders,[5,6] autism spectrum disorder,[8] and attention deficit hyperactivity disorder (ADHD)[8] are common among individuals with ARFID. A retrospective chart review of 34 pediatric patients with ARFID indicated that 50% also had generalized anxiety disorder.[5] Co-occurring psychiatric disorders have implications for treatment. Consistent with a recent case report,[23] it has been our clinical experience that comorbid ADHD treated with stimulant medication is sometimes a barrier to increasing caloric intake in individuals with ARFID who are underweight, because a common side effect of stimulant medication is decreased appetite.

Only a handful of studies documenting clinical outcomes of ARFID exist. One recent medical record review of acute medical hospitalizations in an academic medical center demonstrated that youth with ARFID required longer hospital stays and were more likely to require enteral nutrition for stabilization compared to those with AN.[19] Outcomes at one-year follow-up (stability and rates of readmission) were similar between groups. In another record review, Forman and colleagues found that youth with ARFID presenting to adolescent medicine services were followed for less time and were less likely to reach healthy weights compared to those with AN or atypical AN.[24] By contrast, Nakai and colleagues[18] examined outcomes of 15–40 year-olds with ARFID compared to individuals with AN, all of whom were treated in Japan, finding that individuals with ARFID were more likely to recover (51.9% vs. 35.5%) and less likely to die (0% vs. 15%). Again, there is a paucity of outcomes data in ARFID and more work in this area is needed. Current studies compare ARFID to AN, but many individuals with ARFID are not low-weight, so AN may not be the most appropriate comparison group.

Lastly, operationalizing a definition of recovery from ARFID is complex. Beyond no longer meeting full criteria for ARFID, it is unclear what degree of weight restoration, dietary diversity, and nutritional repletion is expected to categorize an individual as recovered. For example, for youth who have always followed a low-percentile trajectory on standard growth curves, restoration to the median height/weight or body mass index centile may not be realistic. Similarly, it is unclear how many foods within each of the basic food categories (e.g., fruits, vegetables, proteins, grains, dairy) must be regularly consumed to resolve

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nutritional deficiencies or reduce psychosocial impairment. Finally, for the many individuals with ARFID who take vitamin supplements (e.g., multivitamins) prophylactically, supplementation may mask the severity of malnourishment, thus thwarting assessment of baseline medical sequelae and their potential resolution with treatment. This problem is akin to the difficulty in assessing menstrual status in patients with AN who take oral contraceptives, which ultimately led, in part, to amenorrhea being omitted as a diagnostic criterion. Clearly further research is needed to inform the optimal definition of ARFID recovery to assist in evaluating both treatment response and longitudinal outcomes.

Treatment

There is a vast literature on the treatment of pediatric feeding disorders in young children, particularly behavioral interventions designed to increase dietary volume and variety. However, no randomized controlled trials have evaluated the efficacy of any type of ARFID treatment in adolescents or adults. Although individual case reports have highlighted the potential efficacy of cognitive-behavioral therapy[25,26]and family-based therapy[27] for adolescents with ARFID, larger-scale studies are lacking.

In young children with ARFID who are consuming insufficient calories, strategies to increase dietary volume include oral nutritional formula supplementation, tube feeding, and intensive behavioral interventions delivered in day treatment or inpatient settings. Medical supervision of refeeding—including monitoring of cardiopulmonary status and electrolyte balance—is important. Because tube feeding is typically intended as a temporary measure, tube weaning is often an important subsequent treatment component. Typical tube weaning approaches involve a reduction in caloric supplementation by tube feed in order to stimulate the drive to eat and transition to oral feeding. Medical monitoring (e.g., hydration status, weight) is necessary during tube weaning, which can result in weight loss due to inadequate nutrient intake.[28] In a recent randomized controlled trial (RCT) of 1- to 6-year-olds with ARFID (n = 20, 9 of whom were reliant on tube feeds), a 5-day manualized behavioral intervention significantly increased grams consumed at each meal, increased bite acceptance, and reduced mealtime disruptive behaviors, compared to a no-treatment wait-list control condition.[28] Moreover, a meta-analysis of 11 of studies (only two of which were RCTs) evaluating day or inpatient programs for increasing dietary volume in children with ARFID—most of whom had significant co-occurring medical problems—demonstrated an overall 71% success rate, with a range of 43–100%, in weaning those on tube feeds.[22] In studies using tube weaning as the primary intervention, success rates ranged from 81.4% to 100%.[29,30,31]However, while the studies involving behavioral intervention without tube weaning reported weight stabilization or gain,[30,32,33,28] those evaluating tube weaning resulted in weight loss.[29,30,31,34,35] In other words, although tube weaning is often successful in eliminating tube dependence, it should only be undertaken when the patient is able to tolerate at least a small amount of short-term weight loss and there is a clear plan for replacing tube calories with oral intake.

While underscoring the potential efficacy of intensive behavioral interventions for increasing dietary volume in young people with ARFID, these findings highlight the need for stronger designs such as RCTs featuring credible control conditions and standardized outcome

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measures.[36] Collecting both short- and long-term outcomes (e.g., oral intake, need for oral supplements or tube feeds, nutritional value of caloric intake, weight, nutritional deficiencies, amenorrhea, and psychosocial status) will also be important to establish best practices for increasing dietary volume in underweight patients with ARFID. In addition, there is a need to include older patients and those who have less severe clinical manifestations of ARFID in research. It is also notable to us that while tube feeding—particularly long-term ambulatory feeding by gastrostomy tube—is usually considered a last resort for patients with AN, it is sometimes offered to youth with ARFID as a first-line treatment prior to less invasive interventions. Supporting our clinical observations, in a retrospective study of hospitalized patients undergoing refeeding, those with ARFID were substantially more likely than those with AN to require enteral feeding to meet caloric requirements, highlighting the severity of food refusal in the ARFID population.[19] Given that long-term reliance on tube feeding is (1) part of the diagnostic criteria for ARFID; (2) can pose unintended iatrogenic effects (e.g., reinforcement of the sick role, reduced expectation for solid food consumption); and (3) may require intensive behavioral intervention to discontinue; more research is needed to determine when and whether the risks of tube feeding outweigh the benefits in the treatment of low-weight patients with ARFID.

For young children with ARFID who exhibit selective eating (i.e., limited dietary variety), strategies to increase dietary variety include systematic desensitization or operant conditioning paradigms, which can be offered across levels of care and regardless of body mass index. In systematic desensitization, patients are typically exposed repeatedly to new foods through play with no stated expectation for consumption. In contrast, operant conditioning paradigms feature verbal or object rewards from a therapist or caregiver for consumption of a target food. In an RCT evaluating 10 outpatient sessions of either systematic desensitization or operant conditioning to increase acceptance of novel foods in 2- to 6-year-olds with feeding difficulties similar to ARFID, there was a trend for operant conditioning to result in the acceptance of 3.3 more foods, on average, compared to systematic desensitization.[37] Due to the popularity of systematic desensitization in clinical settings, more research is needed on the differential efficacy of these two approaches, applicability to older patients (for whom operant rewards such as “take a bite and you can go play” would not be developmentally appropriate), and potential moderators of treatment outcome.

Despite the promising outcomes of at least some behavioral interventions, the treatment of ARFID even in children is not without controversy. Perhaps most notably, Satter’s influential Feeding Dynamics Model[38] suggests that parents should never apply pressure to encourage children with avoidant and restrictive eating behavior to increase or expand their dietary intake. Indeed, in a recent cross-sectional study of college students, perceived parental pressure to eat during childhood was predictive of higher levels of picky eating in young adulthood.[39] Of course, it is unclear whether parental pressure is a cause or consequence of childhood picky eating, and the generalizability to clinical populations is unknown. Berlin and colleagues[40] have argued that the Feeding Dynamics model does not apply to youth on the tails of the body mass index distribution (which may include those with low-weight feeding disorders now classified as ARFID). However, given the resonance

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of the Feeding Dynamics Model among both caregivers and health professionals, as well as the uncertain generalizability of inpatient and day hospital behavioral modification techniques to caregivers in the home setting,[41] more research is needed to evaluate alternative treatment approaches such as those offered by speech and occupational therapists (e.g., oral-motor skill development, sensory play) for youth with ARFID. Another important research question is whether caregiver support is differentially effective for increasing dietary volume (i.e., to promote weight gain) versus dietary variety (i.e., to introduce novel foods). Clinically we have sometimes found that non-underweight patients with ARFID who have previously felt pressured to try novel food by family members prefer individual to family-based therapy, when given a choice.

Lastly, there are currently no RCTs to support the use of medication in ARFID treatment. In clinical practice, cyproheptadine is sometimes used to stimulate appetite for young people with ARFID who are underweight or lack interest in eating or food. A recent non-randomized case record review from a pediatric feeding clinic found that patients who were given cyproheptadine gained significant weight over a 6.5-month period, but remained in the underweight range at the end of the study and did not significantly surpass weight gain in a control group who did not take appetite-stimulating medication.[42] Similarly, while a recent recent case report describes the use of mirtazapine—an antidepressant medication with appetite-stimulating properties–to reduce barriers to eating in ARFID with fear of aversive consequences,[43] further research is needed on the efficacy of antidepressant and anti-anxiety medications for the treatment of ARFID across the age span.

Directions for future research

Little is known about what causes ARFID. Although some young people present to treatment with exasperated caregivers who describe their children's eating habits as purposefully picky or oppositional, in contrast, we suspect that biology is a major contributor. To shape the next generation of research, we propose a three-dimensional model wherein biological abnormalities in sensory perception, homeostatic appetite, and fear responsiveness underlie the three primary ARFID presentations of sensory sensitivity, lack of interest in eating, and fear of aversive consequences, respectively. This model informs a novel cognitive-behavioral treatment approach that we have recently designed at Massachusetts General Hospital (MGH) to address each of these maintaining mechanisms in children, adolescents, and adults.

Three-dimensional model of the neurobiology of ARFID

Prior categorical models of feeding disorders have described individual presentations (e.g., sensory sensitivity, lack of interest in eating, and fear of aversive consequences) as separate diagnostic entities (depicted in Figure 1a). Indeed, DSM-5 describes three primary ARFID presentations including sensory sensitivity, lack of interest in food or eating, and fear of aversive consequences,[1] which generally coincide with the Great Ormond Street criteria for feeding disorders (i.e., selective eating, food avoidance emotional disorder, and functional dysphagia, respectively).[44] While more research is needed to evaluate the accuracy of this putative ARFID nosology, these three primary presentations are commonly seen in clinical

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settings[9,43,44] and a recent epidemiological study confirmed that all three were present among the 8–13-year-olds who met criteria for ARFID.[3,14]

We hypothesize that a given individual's ARFID presentation can be plotted as a single point along a three-dimensional space, meaning that the three prototypic presentations vary in severity and are not mutually exclusive (depicted in Figure 1b). Indeed, available data supporting our dimensional model suggest that ARFID varies in severity, and that nearly half of individuals with ARFID who present for psychological treatment exhibit eating difficulties in multiple ARFID domains.[45] A typical example would be a young person with longstanding selective eating (i.e., sensory sensitivity) and chronic low appetite (i.e., lack of interest in eating) who loses weight precipitously following an acute choking episode (thus developing fear of aversive consequences).[43] Consistent with the National Institute of Mental Health's Research Domain Criteria (RDoC) approach, we hypothesize that abnormalities in taste perception, homeostatic appetite, and fear responsiveness underlie the three primary ARFID presentations of sensory sensitivity, lack of interest in eating or food, and fear of aversive consequences, respectively. We are currently testing our hypotheses in a study funded by the National Institute of Mental Health (1R01MH108595: "Neurobiological and Behavioral Risk Mechanisms of Youth Avoidant/Restrictive Eating Trajectories," https://projectreporter.nih.gov/project_info_description.cfm?aid=9244073&icde=34034868&ddparam=&ddvalue=&ddsub=&cr=4&csb=default&cs=ASC&pball=). In this multimodal study, we are collecting data across several domains—ranging from self-report to hormones to neuroimaging—on 100 boys and girls ages 10–22 years with ARFID and 50 healthy controls matched for age and pubertal development to identify the potential neurobiological underpinnings of the three primary ARFID presentations.

First, individuals with the sensory sensitivity presentation of ARFID often describe non-preferred foods as tasting intensely negative. The traditional clinical understanding of this presentation is that individuals with sensory sensitivity simply lack experience with non-preferred foods and will come to prefer them with repeated exposure. While we do see a role for exposure in the treatment of ARFID with sensory sensitivity, we hypothesize that oversensitivity in taste perception itself—rather than mere extreme cognitive or affective reactions to certain tastes—contributes to the sensory sensitivity presentation. Indeed, there is evidence that adults who self-identify as picky eaters rate both bitter and sweet solutions as significantly more intense than do those who do not identify as picky eaters.[46] Similarly, children whose parents describe them as "fussy" eaters are more likely to be classified as supertasters—by perceiving to 6-N-Propylthiouracil (PROP) to be extremely bitter—compared to non-fussy children.[47]

Second, individuals with the lack of interest in food or eating presentation of ARFID often describe that they do not feel hungry at mealtimes, forget to eat, and/or feel full more quickly than others. Prior research has suggested that fasting women with restrictive eating disorders have decreased activation of the hypothalamus (control center for integration of appetitive signals) and anterior insula (houses primary taste cortex, integrates visceral signals and interoceptive experiences) versus controls.[48] Thus we hypothesize that the lack

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of interest presentation of ARFID might be associated with differences in activation of the brain's appetite-regulating centers.

Lastly, individuals with the fear of aversive consequences presentation of ARFID react with intense fear and avoidance following a traumatic experience with food. While many people experience choking, vomiting, or abdominal pain after eating at some point in their lives, only a small minority go on to develop ARFID, suggesting that this smaller subset may have entered the traumatic eating experience with a pre-existing vulnerability that gave rise to a phobic response. In line with evidence suggesting that psychophysiological reactivity to fear stimuli distinguishes phobic-type anxiety disorders (i.e., social and specific phobias) from other anxiety disorders.[49] we hypothesize that, hyperactivation of this defense motive system (i.e., amygdala, anterior cingulate, and VPFC hyperactivation) might be manifest among individuals with ARFID.

Overall, we envision that our three-dimensional neurobiological model will not only uniquely characterize the cross-sectional pathophysiology of specific ARFID presentations, but could also highlight potential risk factors for the disorder. Furthermore, our model could be used to predict likely longitudinal trajectories and identify areas to intervene. For example, individuals who have multiple underlying abnormalities consistent with multiple ARFID presentations may be at greater risk for persistence and relapse compared to individual who have just one abnormality and presentation. In that setting, clinicians could tailor treatment plans to uniquely target underlying biological abnormalities in sensory sensitivity, low appetite, and acute fears when designing treatments. Whereas underlying biological abnormalities could dictate treatment approach (e.g., systematic desensitization for sensory sensitivity, regular eating or appetite-enhancing medication for low appetite), severity within each ARFID domain could dictate treatment intensity (e.g., outpatient treatment for patients who are avoiding certain foods after a traumatic experience, versus inpatient treatment for those who are avoiding food entirely and are at high risk for medical compromise).

Cognitive-behavioral therapy for ARFID

Based on our three-dimensional neurobiological model, our team has recently developed a novel form of cognitive-behavioral therapy for ARFID (CBT-AR).[50] CBT-AR is appropriate for children, adolescents, and adults ages 10 and older who are medically stable, do not have severe developmental disabilities, and are not reliant on tube feeding. Because we believe that individuals with sensory sensitivity perceive flavors—such as bitter or sweet—more intensely that individuals without ARFID, CBT-AR teaches skills for approaching novel foods in a stepwise fashion (i.e., first look, then touch, then smell, then taste, then chew). Given our hypothesis that individuals who present with lack of interest in food or eating exhibit abnormalities in homeostatic appetite, CBT-AR relies on parent support to increase dietary volume for youth who are underweight. Lastly, because we believe that fear of the aversive consequences of eating is maintained by high levels of physiological reactivity, CBT-AR relies on both in vivo and interoceptive exposure for managing phobic responses to traumatic experiences such as vomiting or choking. A recent case report describes the

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successful application of CBT-AR to an 11-year-old girl with low weight, sensory sensitivity, lack of interest in eating, and fear of aversive consequences.[43]

CBT-AR proceeds through four stages, which include (1) psychoeducation and regular eating; (2) re-nourishment and treatment planning; (3) addressing relevant maintaining mechanisms (i.e., sensory sensitivity, lack of interest in eating, fear of aversive consequences); (4) preventing relapse. CBT-AR is meant to last approximately 20 sessions, but up to 30 sessions may be necessary when the patient is underweight. Sessions take place weekly, so that the standard 20-session treatment takes approximately five months, whereas the longer 30-session version takes approximately eight months. For underweight or malnourished patients, the total number of sessions is based on the degree of weight restoration needed, the number of maintaining mechanisms that need to be addressed, and the therapist's clinical judgment. CBT-AR is a time-limited treatment that can be delivered in a family-supported or individual treatment format. Family-supported therapy is generally recommended for patients under age 16, and can also be used for older adolescents or young adults who have significant weight to gain, or patients with mild developmental disabilities. The individual format is generally recommended for adults and for mature adolescents who are highly motivated and do not have significant weight to gain.

In addition to using CBT-AR in routine clinical practice at MGH, our team is currently conducting an open trial of CBT-AR for youth ages 10–22 who are also participating in the neurobiology of ARFID study. Going forward, in the setting of a larger future RCT, we plan to evaluate whether signs and symptoms of illness at baseline (e.g., increased brain activation in response to bitter tastants; reduced neural activation in response to palatable foods; heightened fear response to images of choking or vomiting) predict symptom improvement (e.g., increase in dietary volume, dietary volume, and/or weight) at the conclusion of treatment.

Conclusions

ARFID was added to the psychiatric nomenclature four years ago as a reformulation of an understudied childhood diagnosis. Now that ARFID has been clearly defined, studies of risk factors, prevalence, pathophysiology, and treatment are needed. Although there is a growing literature on the treatment of pediatric feeding disorders, there is no clear standard of care for ARFID specifically and controversies (e.g., about whether caregiver involvement provides helpful support or iatrogenic pressure; and when and whether tube feeding is the most appropriate intervention) abound. Furthermore, little is known about treatment efficacy for adolescents and adults with ARFID beyond a handful of published case studies, though our team at MGH is currently testing a novel form of cognitive-behavioral therapy. To support the evaluation of treatment efficacy, the field must agree upon clear definitions of recovery by reaching consensus on how much weight must be restored for patients who are consuming insufficient volume, and the degree of dietary diversity that must be achieved to resolve nutritional deficiencies and reduce psychosocial impairment among patients who are consuming insufficient variety. Furthermore, guidelines for determining the appropriate level of care for individuals with ARFID—ranging from outpatient CBT or occupational therapy to tube placement or inpatient care—should be developed to encourage the appropriate

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distribution of limited healthcare resources. Such conversations will support the development of psychometrically valid assessments (such as the PARDI[16]) to screen for the condition, assess relative severity, and evaluate treatment outcomes. Our three-dimensional model of neurobiology highlights testable hypotheses that could inform our understanding of etiology, longitudinal persistence, and treatment response.

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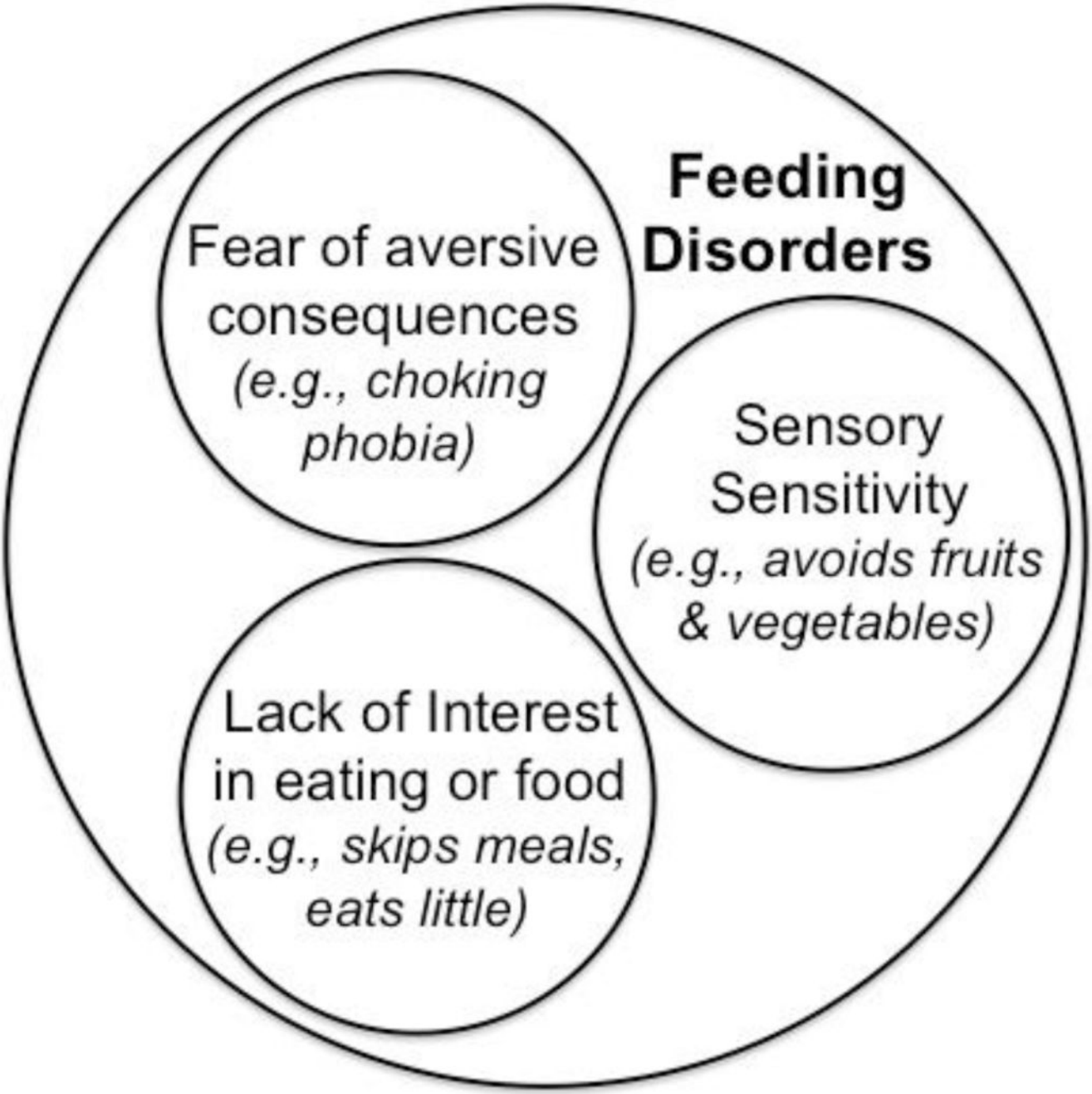

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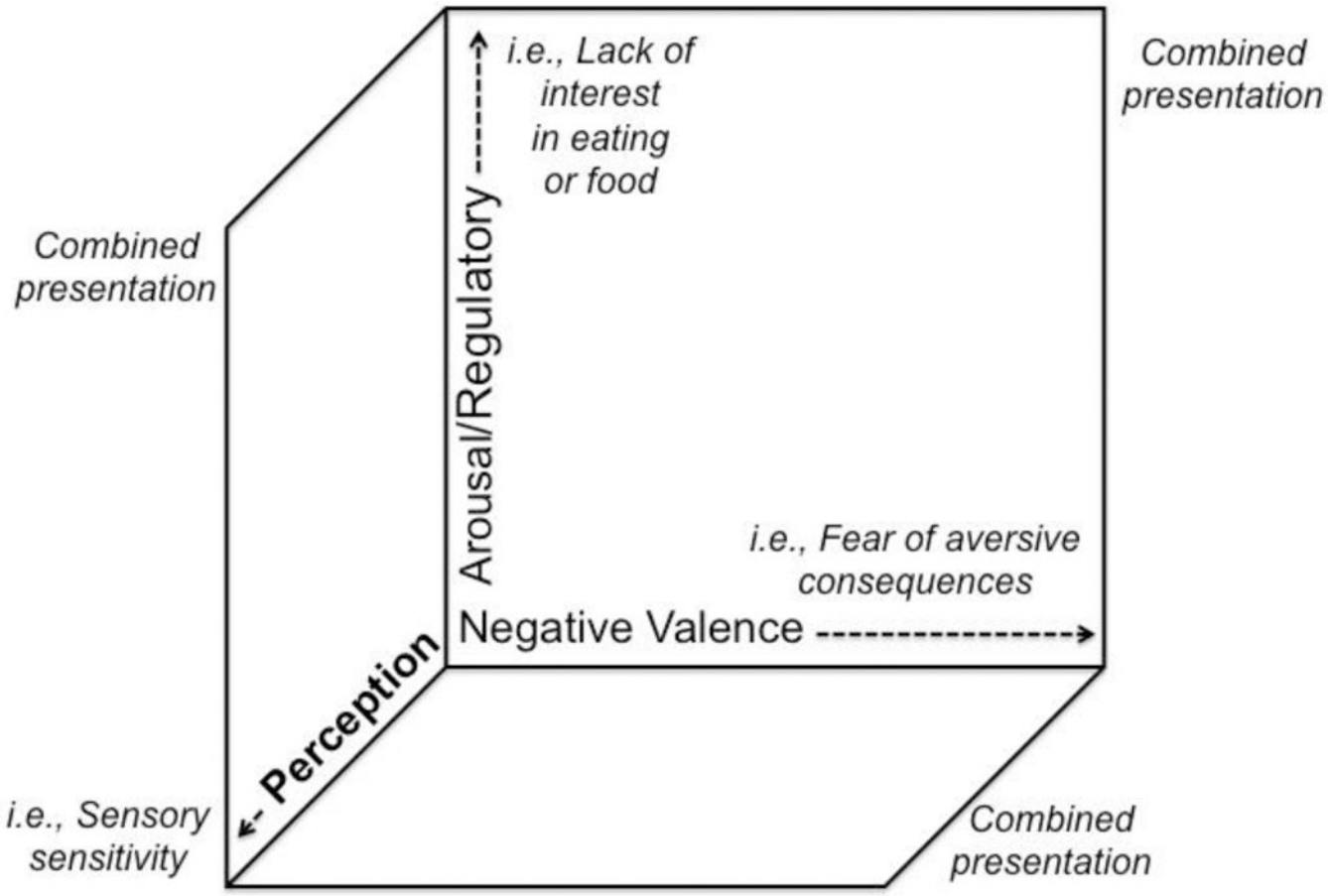

Figure 1. Categorical versus dimensional models of ARFID.

(a) categorical model of avoid/restrictive eating. (b) dimensional model of avoidant/restrictive eating.