Atypical early-onset eating disorders

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SUMMARY
This article reviews the recent changes to the DSM diagnostic classification of feeding and eating disorders with particular reference to children and adolescents. The common clinical presentations of the ‘atypical’ feeding and eating problems of middle childhood and early adolescence are reviewed using clinical case vignettes, and the limited evidence base regarding management is summarised. There are many gaps in the evidence base and this is likely to be an area of rapid development for the field subsequent on the new terminology outlined in DSM-5.

LEARNING OBJECTIVES
• Be able to describe the recent changes in terminology of DSM-5 for eating disorders in children and adolescents.
• Be able to provide information to young people and parents on the short- and long-term medical consequences of low weight in children.
• Be able to assess risk in children presenting with atypical eating disorders.

DECLARATION OF INTEREST
None.

Understanding of the epidemiology and management of feeding and eating disorders in children and young people has developed significantly since we first wrote on this subject for Advances in psychiatric treatment (2014). With the publication of DSM-5 (American Psychiatric Association 2013), we have seen new diagnostic terminology and groupings for the range of clinical feeding and eating disorders seen in this age group (Box 1). DSM-5 has finally removed the non-specific distinction between feeding disorders (which imply a feeding relationship and lack of autonomy, and were historically classified as starting before age 6) and eating disorders (which imply independent eating behaviour), and reframed feeding problems as food intake disorders, thus removing age-related criteria. Many individuals will now fall under the diagnosis of avoidant/restrictive food intake disorder (ARFID), a new term in DSM-5, and likely therefore to be adopted for ICD-11. This term describes restricted food intake in children or adults that is not accompanied by body-weight and shape-related psychopathology. The other major changes in DSM-5 relate to the diagnosis of eating disorders not otherwise specified (EDNOS), which has been a major research focus for the past 5 years.

Eating difficulties can, of course, be present in the context of other disorders, such as depression, obsessive–compulsive disorder (OCD) and pervasive developmental disorders. Physical illness is often associated with loss of appetite or food aversion, to which psychological factors can contribute. In these cases, when food avoidance is marked and merits treatment in its own right, a secondary diagnosis of feeding or eating disorder can be made.

Early-onset obesity and hyperphagic short stature have not traditionally been considered eating disorders, but can also present with marked abnormalities of eating. The DSM-5 work group decided, having reviewed the evidence, that obesity did not merit classification as an eating disorder, although mental dysfunction may be involved in the aetiology of specific obesity phenotypes. Furthermore, obesity is both a risk factor for and strongly associated with some eating disorders, particularly binge eating, as well as a common consequence of many of the medications used to treat mental illness (Marcus 2009). However, it is recognised that within obese and overweight populations, a proportion of individuals will have significant eating pathology and other psychiatric comorbidity, and that psychological factors are important in the maintenance of overweight (White 2012). The management of obesity is beyond the scope of this article, but it is noteworthy that many eating disorders services for young people do not currently offer comprehensive services for the treatment of eating pathology in the context of overweight.

This article principally addresses the less commonly recognised or atypical feeding and eating disorders in children and young adolescents, namely rumination disorder, pica, ARFID and binge eating disorder. For a detailed review of the management of anorexia nervosa and bulimia nervosa in children and adolescents see Nicholls & Barrett (2014).
Atypical early-onset eating disorders

Incidence and prevalence

A recently published UK study (Micali 2013) found an increase in the number of patients newly diagnosed with an eating disorder over a 10-year period, with the incidence highest for girls aged 15–19 and for boys aged 10–14. Particular increases in atypical eating disorders were noted, whereas for anorexia nervosa and bulimia nervosa the incidence was relatively stable. This finding supports other studies that suggest a stable incidence of eating disorders, but clinical presentations increasing in younger age groups (van Son 2006). Furthermore, the number of hospital admissions of young people up to 14 years of age has risen year on year for over a decade in England (Hospital Episode Statistics: www.hesonline.nhs.uk). It is not clear whether these increases reflect rising incidence, greater recognition or changing practice. Definitive data of rising incidence are needed before searching for possible explanations. Childhood-onset eating disorders are still relatively rare and data are therefore sparse in epidemiological samples. National Surveillance methodology has shown that new cases of childhood eating disorders (<13-year-olds) in the UK and Ireland have an overall incidence of 3.01/100,000 (Nicholls 2011a), comparable to figures for Canada (Pinhas 2011) and Australia (Madden 2009). The majority of individuals had anorexia nervosa or anorexia nervosa-like presentations (in the British sample, 37% had anorexia nervosa, 43% EDNOS and only 1.4% bulimia nervosa). Almost 20% of the children showed determined food avoidance and were underweight without weight or shape concerns. These children would now be diagnosed with a form of ARFID in DSM-5. The proportion of boys relative to girls in the younger age group has also increased (Micali 2013).

Atypical feeding/eating problems are common in young children and the challenge is delineating normal developmental variants from clinically significant disorder (Box 2). Around 50% of parents report that their child avoids certain foods, and around 20% report multiple feeding problems (Crist 2001; Equit 2013). How many of these would be clinically significant problems has not been established. Usually, the distinction is made on the

<table>
<thead>
<tr>
<th>BOX 1 The range of feeding and eating disorder presentations seen in children and adolescents</th>
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<tbody>
<tr>
<td><strong>Previous terminology (DSM-IV, ICD-10 and the literature)</strong></td>
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<tr>
<td>Feeding disorder of infancy and early childhood (includes infantile anorexia; post-traumatic feeding disorder)</td>
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<tr>
<td>Pica</td>
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<td>Ruminator disorder</td>
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<tr>
<td>Food avoidance emotional disorder (FAED)</td>
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<tr>
<td>Selective eating or sensory food avoidance</td>
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<tr>
<td>Pervasive refusal syndrome</td>
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<td>Anorexia nervosa</td>
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<td>Atypical anorexia nervosa</td>
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<td>Bulimia nervosa</td>
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<td><strong>DSM-5 terminology</strong></td>
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<td>ARFID</td>
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<tr>
<td>Not formally recognised as an eating disorder</td>
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<tr>
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<tr>
<td>Many will now meet criteria for anorexia nervosa</td>
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<tr>
<td>Bulimia nervosa</td>
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<tr>
<td>Some will now meet criteria for bulimia nervosa or purging disorder</td>
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<tr>
<td>Many now meet criteria for anorexia nervosa, bulimia nervosa, or other specified feeding or eating disorder (OSFED) e.g. binge eating disorder, purging disorder</td>
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**BOX 2 Vignette 1: Eating disorders in younger children**

Mel, aged 10, attends her general practitioner (GP) with her mother for an assessment of her eating. Two months ago, her mother was worried about Mel losing weight; a year ago Mel had been described as plump. The GP wonders if the child might have an eating disorder and notes that her body mass index (BMI) is 13.5. Although he recognises that this is low for an adult, he is not sure whether it is low for a child. Mel has an evident preoccupation with thinness and weight and shape concerns. However, she thinks that her current weight is fine and tells her GP that a calorie-restricted diet is associated with living longer. There have been significant rows at home, which have escalated to the point that Mel is throwing food and has on one occasion hit her mother with a plate. The GP is not sure whether children as young as Mel could present with an eating disorder. Neither does he know what medical parameters to measure with respect to her low BMI. He is also uncertain about her medical risk, whether to refer her to child and adolescent mental health services (CAMHS) or to paediatrics, and how urgently he needs to act. He telephones you for advice on the diagnosis and how to assess the risk.

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Atypical feeding/eating problems are common in young children and the challenge is delineating normal developmental variants from clinically significant disorder (Box 2). Around 50% of parents report that their child avoids certain foods, and around 20% report multiple feeding problems (Crist 2001; Equit 2013). How many of these would be clinically significant problems has not been established. Usually, the distinction is made on the
basis of accompanying weight loss, behavioural or emotional problems, or nutritional deficit.

**Examples of atypical eating disorders**

**Rumination disorder**

Rumination disorder is characterised by effortless regurgitation of recently ingested food, the stomach contents appearing in the mouth without retching or nausea. Food may then be re-chewed before being swallowed again, or in some cases spat out. Typically, this process is repeated and can appear to be pleasurable and a form of self-stimulation.

Complications of rumination disorder include malnutrition secondary to inadequate nutrient or calorie retention, halitosis, dental damage, electrolyte abnormalities and abdominal pain. It is seen most commonly in infants and individuals with psychiatric or neurological disorders (Olden 2001). Sensory and/or emotional deprivation are also associated with rumination in children, and increased incidence is therefore seen in institutionalised children, infants in intensive care units and in normal infants with attachment disorders. In older children, rumination can be associated with weight loss and vomiting (Khan 2000). However, we have experience of treating a number of previously healthy children who developed rumination disorder in the context of anxiety and somatisation disorders (Box 3).

The most significant practice point for clinicians is to exclude alternative causes of regurgitation or vomiting, the most obvious being gastrointestinal conditions such as gastro-oesophageal reflux, pyloric stenosis and gastrointestinal infections. Regurgitation also needs to be distinguished from vomiting or possetting.

**Avoidant/restrictive food intake disorder (ARFID)**

The DSM-5 category ‘avoidant/restrictive food intake disorder’ replaces DSM-IV’s ‘feeding disorder of infancy and early childhood’. Key features are the removal of age criteria and clarification of the ways in which the threshold for disorder can be reached, i.e. as a result of weight loss, nutritional impairment or by virtue of the psychological impact of a highly restricted diet on both personal development and family function. Unlike patients with anorexia nervosa, bulimia nervosa and EDNOS, young people with presentations that would be encompassed by the criteria for ARFID typically recognise their degree of underweight (i.e. there is no body image distortion), many would like to be heavier and may not know why they find this difficult to achieve. They are more likely to have other medically unexplained symptoms, or comorbid medical or neurodevelopmental disorders.

Bryant-Waugh et al (2010) propose three as yet unvalidated subgroups of ARFID, in the hope of stimulating research and clarification of terminology: children who avoid food; children with fears about eating; and children with sensory problems about eating.

**Children who avoid food**

Terms such as food avoidance emotional disorder (FAED), non-fat-phobic anorexia nervosa, restrictive eating and infantile anorexia nervosa have been used to describe children who avoid food for no clear reason. In middle childhood (5–13 years of age) about 20% of patients presenting with a clinically significant eating difficulty involving weight loss fall into this category (Madden 2009; Nicholls 2011a), with a proportionately higher number of boys. They may give any number of reasons for not eating enough, but often report not feeling hungry or just ‘can’t eat’ or ‘it hurts my tummy’. Comorbid OCD or depression may be present, but often the food avoidance exists as an isolated symptom.

Children with food avoidance may be as severely physically compromised as those with anorexia nervosa. Their parents may attribute weight loss or food avoidance to undiagnosed physical disorder, and in many cases this may indeed be a factor. Some children have unidentified organic pathology, such as inflammatory bowel disease, food allergy or intracranial pathology.

**Children with fears about eating**

Some children and young people describe specific fears about eating and these presentations are best conceptualised as phobic disorders. Phobias...
involving food can occur in isolation or as part of a more generalised anxiety disorder or OCD. The nature of the specific fear varies with, among other things, the child’s developmental stage. Common fears are fear of vomiting (emetophobia), fear of contamination or poisoning and fear of choking or swallowing (sometimes known as functional dysphagia). Food phobias usually follow a period of eating that is normal for developmental stage. Clear trigger events (e.g. choking) can be identified in some but not all cases. Phobias of this kind can be chronic and lead to significant functional impairment.

Treatment utilises cognitive–behavioural principles in combination with psychoeducation, graded desensitisation and exposure, behavioural rewards, family therapy and, in some cases, anxiolytic medication.

Children with sensory problems about eating

Some children avoid foods because of its sensory features, such as shape, colour, texture or smell. Other terms for this presentation include selective eating and sensory food aversion. ‘Faddy’ or ‘picky’ eating occurs in over 20% of toddlers and can be considered normal at particular developmental stages. In a small number, particularly boys and children with neurodevelopmental disorders, the behaviour persists into middle childhood and adolescence. An association with sensory sensitivity and features of autism spectrum disorder or attention-deficit hyperactivity disorder (ADHD) is a factor in around 40% of these children (Rastam 2013) (Box 4).

Food refusal

Not clearly identified within the ARFID subgroups are young people who refuse food. Food refusal as an isolated behaviour is an experience most parents encounter at some point during their child’s development. Behaviours associated with food refusal in toddlers include whining or crying, tantrums and spitting out food. In older children it is often associated with other defiant behaviours, such as delaying eating by talking, trying to negotiate what food will be eaten, getting up from the table during meals and refusing to eat much at a meal but requesting food immediately afterwards (Crist 2001). Much of the nutritional intake of these children is gained through snacking between meals.

Food refusal can also occur in children of otherwise biddable and compliant temperament, or in conditions such as cerebral palsy, intellectual disability and other developmental disorders. In these children, food refusal may be one of only a few ways that they are able to communicate that something is either physically or emotionally wrong. In its extreme form, food refusal may be associated with refusal to talk and walk, a presentation known as pervasive refusal syndrome. This is not a formally recognised diagnosis.

Binge eating disorder

Binge eating is thought to be relatively common, particularly in children whose parents seek treatment for their overweight. It can be detected through screening for eating disorder psychopathology, but this is not often carried out in primary care. Consequently, binge eating is probably underdiagnosed except in specialist obesity services.

There has been more focus recently on factors that may contribute to ‘loss-of-control eating’ in young people (Tanofsky-Kraff 2007), in the hope that this might prevent future eating disorders and obesity. For example, there is evidence that when parents control their child’s intake too much, this can potentiate preferences for high-fat, energy-dense foods, limit children’s acceptance of a variety of foods and disrupt their regulation of energy intake by altering responsiveness to internal cues of hunger and satiety (Birch 2001). Comorbid psychiatric and social problems are common, including depression, anxiety, low self-esteem, body dissatisfaction, weight concerns and poorer quality of life.

General principles of assessment

A suggested assessment schedule includes:

- full history from both the child and family, including the history of the eating/feeding difficulty, recent stressors, family history, comprehensive review of past treatment,
time line, comorbidity, related risk factors, psychological risk assessment (including self-harm and suicide, other mental health concerns, best assessed individually if child is able); if there is weight loss, differential diagnoses need to be excluded (Box 5);

• review of a food diary (typically 3 days) to assess nutritional adequacy of range and quantity;
• physical examination and medical risk assessment; there are several potentially useful frameworks for this, including the Junior MARSIPAN risk assessment framework if underweight is severe (Box 6);
• validated standardised assessment measures such as the Child Eating Disorder Examination (Ch-EDE; Bryant-Waugh 1996), the Child Eating

BOX 5 Differential diagnosis of acute weight loss

Differential diagnosis includes:
• endocrine: diabetes mellitus, hyperthyroidism, glucocorticoid insufficiency
• gastrointestinal: coeliac disease, inflammatory bowel disease, peptic ulcer
• oncological: lymphoma, leukaemia, intracerebral tumour
• chronic infection: tuberculosis, HIV, viral, other
• psychiatric: depression, autism spectrum disorder, obsessive–compulsive disorder (OCD)

(Royal College of Psychiatrists 2012a)

Multidisciplinary discussion and feedback to the family, followed by planning with the young person and family, form the basis of a collaborative approach to treatment. An understanding of potential predisposing, precipitating and perpetuating factors based on the information obtained may help in building an understanding with the family of the nature of the eating difficulty and factors influencing it, including those that are and are not open to change. However, the need to regain medical stability and a nutritionally adequate diet may be the priority over understanding aetiology, and psychological factors may become more evident and relevant during the process of nutritional rehabilitation.

General principles of treatment

The basis of treatment lies in engaging both the young person and the family, since motivation and therapeutic alliance are key determinants of treatment outcome. Treatment decisions are based primarily on diagnosis, clinical risk and severity of the disorder. These will determine the treatment setting. Also relevant are systemic/family factors, such as how much support the family has and is able to offer.

There is no evidence base regarding the effectiveness of residential treatment for bulimia nervosa or atypical eating disorders/ARFID and decisions therefore need to be made on the basis of risk, treatment needs and service availability. A therapeutic hospital admission may be needed if the burden of care required exceeds the capacity of the family and/or where risks are high. Weight loss, physical sequelae and nutritional status will require careful monitoring, and the clinical consequences of weight loss need to be managed. When refeeding in the context of acute weight loss, the risk of refeeding syndrome needs to be acknowledged (see below). For chronic low weight, the impact of malnutrition on growth and a clear plan for improving nutrition need to be established.

Medical treatment and physical well-being

Assessment of medical stability is an important component of risk assessment, as mentioned earlier, and nutritional rehabilitation will be the first-line treatment for some children. Early intervention is essential to prevent or reverse significant physical

BOX 6 Components of the Junior MARSIPAN risk assessment framework

• Body mass index (BMI) and weight
• Cardiovascular health
• Electrocardiograph (ECG) abnormalities
• Hydration status
• Temperature
• Biochemical abnormalities
• Disordered eating behaviours
• Engagement with management plan (young person and family)
• Activity and exercise
• Muscular weakness
• Self-harm and suicide
• Other mental health diagnoses
• Other medical conditions

(Royal College of Psychiatrists 2012a)
complications. Nutritional assessment must encompass both low weight and rapidity of weight loss, pubertal/menarcheal status, body mass index (BMI) centile or % median BMI (BMI/median BMI for age and gender), haemodynamic stability and future predicted intake (more commonly over-than underestimated). Rapid weight loss (more than 1 kg/week) can cause medical instability even if the child is not underweight. Muscle weakness and peripheral neuropathy are signs of serious nutritional deficit. Local protocols agreeing thresholds for paediatric admission can be helpful. Clinical guidelines such as the Junior MARSIPAN report (Royal College of Psychiatrists 2012a) emphasise the importance of a collaborative approach between paediatric and mental health services. This is especially important given the deficits in knowledge and training with respect to children with eating disorders who are under-weight (Hudson 2013).

Distinction should be made between children who are appropriately prepubertal and those in whom puberty is delayed. Chronic physical illness or genetic factors resulting in pubertal delay need to be taken into account. Information about the child’s growth will give a more complete picture. Significant delay is usually defined as more than 2 standard deviations from the mean. Menses are deemed to be delayed if there is failure of onset within 4.5 years of the start of puberty, or by chronological or bone age of 14 years. Onset of weight loss during puberty may result in pubertal arrest. Tanner staging, pelvic ultrasound appearances and discrepancy between the bone age and chronological age of the patient can help in evaluating the degree of pubertal delay.

**Acute medical management**

Hudson et al (2012) used data from population-based surveillance systems in the UK and Ireland to examine the physical burden of eating disorders in children under 13. Over a third of the children had medical instability at presentation (60% bradycardia, 54% hypotension, 34% dehydration, 26% hypothermia); 52% required admission at diagnosis (73% to a paediatric ward); 41% of those with medical instability were not underweight, that is, they had BMI z-scores above −2.0 (in the 2nd centile). These findings emphasise the importance of physical examination in all young people presenting with eating difficulties.

**Refeeding**

Refeeding acutely malnourished children and young people carries more risk than chronic malnutrition, so caution should be exercised. Ideally, the clinician should have experience in refeeding, but in its absence, guidelines should be followed closely. The Junior MARSIPAN report outlines key principles to approaching refeeding, including the importance of continuous review and the risks of being overly cautious (Royal College of Psychiatrists 2012a). Starting calorie intake should not be lower than intake before admission. For most young people, starting at 1000–1200 kcal per day is safe. However, electrolytes and clinical state need careful monitoring and transfer to a paediatric unit may be required if, for example, serum phosphate levels fall to <0.4 mmol/l. For the highest-risk individuals (those with very rapid weight loss, very low, abnormal biochemistry before refeeding or a low baseline white blood cell count), a more cautious approach is advised, with phosphate and vitamin supplementation, but here the key is to increase energy as soon as it is safe to do so, i.e. every 2 days. Refeeding syndrome comprises a potentially serious constellation of biochemical and cardiovascular anomalies, the most common of which is hypophosphataemia. It is most likely to occur in the first few days of refeeding, but may occur up to 2 weeks afterwards. For most young people, refeeding should be manageable orally, particularly if a structured meal plan is used.

For a comprehensive review of the medical aspects of eating disorders, see Nicholls et al (2011b).

**Longer-term medical management**

Surprisingly little is known about the longer-term outcomes of early-onset underweight and eating disorders. Most published data on physical instability and growth in malnutrition come from specialist centres or from low-income countries where aetiology differs. In terms of sequelae of low weight, effects that in adults are known to be reversible with weight gain may be irreversible in children. For example, growth slows down and even stops during a period of starvation. After starvation is over, catch-up growth can occur but it is still unclear to what extent. Bone density is also affected, and in younger patients the problem of bone loss is compounded by failure of bone accretion, since adolescence is a critical time for bone accrual. Between 25% and 40% of young people with anorexia nervosa will have osteopenia on bone density scan. The long-term fracture risk is around three times that in the general population. Interpretation of reduced bone density in anorexia nervosa in young people should consider the impact of pubertal delay and growth failure on bone size.
Nutritional rehabilitation remains the treatment of choice for low bone density. Calcium supplementation can be considered, although it is likely to have limited value in an underweight child. There is no evidence at present that oestrogen given as an oral contraceptive improves bone mineral density and it risks stunting from premature epiphyseal fusion. However, physiological oestrogen doses that mimic puberty (i.e. oestrogen patches) have potential therapeutic use (Misra 2011).

**Psychological treatment of specific disorders**

There are no randomised clinical trials for the treatment of atypical eating disorders in children and adolescents, and treatment approaches therefore borrow from the evidence in other disorders. Family-based treatment (Lock 2013) or systemic therapy (Eisler 2007) are the best evidenced interventions for anorexia nervosa in adolescents, and so will usually be the first-line intervention for younger patients. This treatment comprises around 20 sessions over 6–12 months. Some young people respond early (in the first four sessions), whereas those with obsessional features may take longer (Lock 2005). Notably, about 15% of those in receipt of family-based treatment will require hospital admission for medical stabilisation (Lock 2010). For a review of family therapy and parental counselling for anorexia nervosa and bulimia nervosa see Nicholls & Barrett (2014). The principles of family-based treatment have been increasingly applied to other problem eating behaviours, especially where the initial focus is weight gain or nutritional rehabilitation.

In general, the evidence for individual therapies is limited with respect to younger children. Nutrition, cognitive and emotional development will all have an impact on ability to engage with treatment, and support from caregivers is essential. Interventions incorporating elements of dialectical behaviour therapy such as mindfulness, distress tolerance skills training and cognitive–behavioural therapy (CBT) are being piloted for binge eating (Mazzeo 2013).

**Psychopharmacology**

The evidence base for psychopharmacology for younger patients remains limited. In practice, given high rates of comorbidity and lack of evidence, prescribing is often symptom- rather than diagnosis-based. For ARFID, psychopharmacological interventions will be guided by comorbidity, such as obsessionality/OCD, low mood, aggression, rigidity and inflexibility, sleep disturbance and anxiety.

There are no studies of psychopharmacological interventions for bulimia nervosa, binge eating disorder or ARFID in children and adolescents, although adult studies have used selective serotonin reuptake inhibitors (SSRIs), other antidepressants, mood stabilisers and anti-obesity medications as adjunctive treatments (Flament 2012).

**Treatment of anorexia nervosa**

There have been a few open and randomised controlled trials of psychopharmacology for anorexia nervosa in children and adolescents. Two (Kafantaris 2011; Norris 2011) showed no greater benefit of olanzapine over placebo. Similarly, a randomised trial of risperidone v. placebo found no effect on eating disorder rating scales or weight (Hagman 2011). However, an open naturalistic randomised controlled trial of quetiapine v. placebo reported greater improvement in concerns with eating, weight and shape, and decreased comorbid anxiety and depression in the quetiapine group (Court 2010).

**Treatment of avoidant/restrictive food intake disorder**

Since the diagnosis of ARFID includes a range of different clinical presentations, treatment will vary from child to child. Assessment will have identified the key areas of impact and risk, including the extent of nutritional compromise, the effect on weight and growth, how much the eating problem interferes with social and emotional development or function, and associated distress or impairment. It will also reveal the extent to which the child is motivated to change aspects of their eating behaviour.

The Royal College of Psychiatrists (2012b) suggests that treatment should closely mirror that for similarly presenting eating disorders. It would therefore typically involve a combination of nutritional advice or intervention, psychological interventions and medical monitoring. Eating a highly limited range of foods may have no effect on growth and development, but may compromise, for example, bone health and other micronutrient-related parameters. In some cases, reassurance that the behaviour is not doing the child any damage is all that is required, but in others children will be nutritionally compromised, or find themselves socially disadvantaged by their eating, unable to go away on school trips or stay at friends’ houses.

If the child is ready to address their eating problem, a CBT model based on age-appropriate food records, relaxation and reward, led by the...
child, can be rapidly effective. Over the years, the child may have developed an avoidance-reinforced anxiety associated with new foods. This may be anticipatory nausea (with sight or smell triggers), fear of vomiting (textures) or fear of choking. Early in treatment, as new foods are faced, symptoms will occur. If the child is not committed to change at this stage, the anxiety will result in avoidance again. An exposure-based CBT approach is described in Nicholls et al (2001).

If low weight is a feature this will need to be assessed and addressed as for anorexia nervosa.

Commonly, nutritional compromise will be long-standing, which has implications for both the nature and speed of the intervention. Expectations may also differ, i.e. a full range of foods may never be eaten, and the desired outcome will focus on maximising function and limiting risk. For example, adding pizza to the diet may enable a child to stay with friends; adding a multivitamin may improve fatigue levels and bone health.

If food avoidance results from symptoms of emotional problems (such as worry or a form of somatisation), treatment will focus on emotional well-being, including helping the child to find alternative ways of naming and identifying their feelings (note that appetite loss secondary to depression would not normally be included, since this would be expected to respond to treatment for depression). Parents work to support the child in their rehabilitation, much the same as for a somatisation disorder. Behavioural techniques have a role in changing concrete, measurable aspects of behaviour, but have little effect on thoughts, beliefs and feelings. A case example of ARFID and its management plan are outlined in Bryant-Waugh (2013).

Individuals experiencing comorbid mental health problems such as depression or anxiety might benefit from CBT and other treatments for the comorbid condition. This is particularly the case for children with an evident phobic component to the presentation.

Young people with ARFID are likely to experience impaired social functioning. In turn, this may affect family functioning, especially if there is great stress surrounding mealtimes. Telephone interviews with families who perceived their children to be picky eaters showed high rates of stress in caregivers (Goh 2012), a finding that highlights the potential for family and group work.

Outcomes

Longer-term outcomes and course of illness for young people meeting criteria for ARFID are largely unknown, and there have been few prospective studies. McDermott et al (2010) examined the persistence of parent-perceived picky eating in a cohort of over 7000 children, from birth to 14 years of age. About 40% of irregular eaters at age 5 were still irregular eaters at age 14, strongly predicted by infant feeding problems and the child’s inability to regulate their sleep and mood. Maternal factors were greater age, not feeling positive about the baby and persistent anxiety during the child’s early years. These risk factors mirror some of those identified for anorexia nervosa (Nicholls 2009). Notably, individuals with sensory aversions to food may have enduring difficulties (Mascola 2010).

Parental sense of self-efficacy may also affect outcomes (Robinson 2013), and carer psychological distress and burden may influence or affect recovery and should therefore be a focus for treatment.

There are no long-term outcome studies of rumination disorder or of binge eating in children, although the association with obesity is well described.

Prevention and early intervention

Prevention and early intervention in eating disorders has been a neglected area in the UK. Prevention and early interventions for ARFID will develop as the field determines the key characteristics of these disorders. However, the link between parenting practices regarding eating and later eating pathology (Loth 2014) suggests that the prevention of intergenerational transmission of eating problems is a potential target for interventions.

Resources

Box 7 gives details of useful online resources for health professionals.

Conclusions

There is some evidence that the changes to the diagnostic criteria introduced in DSM-5 will better reflect the presentations of eating disorders seen in clinical practice (Birgegård 2012), reducing reliance on ‘not otherwise specified’ categories as a diagnosis without loss of information. These changes will in turn have implications for the scope of eating disorders services and pave the way for more research into those forms of feeding and eating disorders for which there is little evidence to guide treatment decisions.

This article highlights the lack of a cohesive evidence base for younger children and in particular the limited evidence with respect to
**BOX 7 Useful online resources for professionals**

**Organisations**
- Academy for Eating Disorders: www.aedweb.org
- Feeding Matters: www.feedingmatters.org
- Institute of Child Health, University College London: www.ucl.ac.uk/ich/education-ich/events
- National Eating Disorder Information Centre, Canada: www.nedic.ca

**Literature/learning modules**
- Growth charts: www.rcpch.ac.uk/child-health/research-projects/uk-who-growth-charts

**ARFID.** Much current treatment is led by comorbid conditions or extrapolated from evidence in other disorders. Hudson et al (2013) have highlighted training and knowledge deficits in middle-grade paediatric doctors of the medical management of underweight children. They advocate enhanced training packages, including how to manage children who are severely underweight, in the curricula for acute resuscitation courses. Although training is important, the absence of literature to guide clinical practice highlights atypical eating disorders as a priority area for research.

**References**
- American Psychiatric Association (2013) Diagnostic and Statistical Manual of Mental Disorders (5th edn) (DSM-5). APA.


**MCQs**

Select the single best option for each question stem

1 National surveillance has shown the incidence of new cases of eating disorders in children under 13 years of age to be:
   a 3 per 100 000
   b 6 per 100 000
   c 0.6 per 100 000
   d 60 per 100 000
   e 3 per 10 000.

2 The differential diagnosis of acute weight loss includes all of the following except:
   a diabetes mellitus
   b depressive disorder
   c hypothyroidism
   d leukaemia
   e tuberculosis.

3 New diagnostic terminology for feeding and eating disorders include:
   a cyclical rumination disorder
   b refeeding rehydration syndrome
   c functional reflux disorder
   d avoidant restrictive food intake disorder
   e anorexia nervosa not otherwise specified.

4 As regards BMI measurement in children:
   a it is more significant if the rate of weight loss is rapid
   b muscle weakness is a primary determinant
   c children in whom puberty is delayed are less likely to have an accurate BMI measurement
   d BMIs are generally less reliable in children
   e it is a linear constant.

5 The following is an essential feature of an eating disorder risk assessment:
   a ECG abnormalities
   b informed consent has been freely given
   c parental consent from both parents
   d locus of control
   e presence of family support.