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ORIGINAL CONTRIBUTION

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Childhood autism, feeding problems and failure to thrive in early infancy

Seven case studies

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Abbreviations: ASD: Autistic spectrum disorder; ED: Eating disorder; AN: Anorexia nervosa; SE: Selective eating; RE: Restrictive eating; FTT: Failure to thrive; FR: Food refusal; FP: Food phobia; PE: Picky eating; PEB: Perseverant eating behaviour; BMI: Body mass index; OCD: Obsessive compulsive disorder; ITFA: Inappropriate texture for age.

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Abstract Despite longstanding clinical experience of unusual feeding difficulties in children with autism, there is no published literature describing their association with early onset FTT. This paper examines literature that may link feeding problems and abnormal growth with developmental and psychiatric conditions and describes seven cases of children with autism, who showed growth failure caused by severe feeding problems starting in the first year of life. Inadequacies in existing classifications systems are highlighted. The presence of severe or atypical feeding problems and FTT in infancy should alert professionals to possible underlying ASD. The aetiology of feeding disorders in autism appears to involve an unusually complex interactional model with biological vulnerabilities due to dysfunction in sensory, cognitive and emotional response interacting with dysfunctional attachment and learnt behaviours to produce a severe and intractable problem. Effective treatment therefore requires a novel multifaceted approach that can address each of these areas.

Key words autism – feeding problems – failure to thrive

Introduction

Feeding problems are common in childhood, reported to occur in 25–35% of normally developing children and up to 80% of those with developmental delay [5]. Failure to thrive (FTT) as a result of abnormal feeding behaviour, however, is relatively infrequent, occurring in approximately 3% of infant populations. Fifty percent of infants exhibiting FTT in the second year of life, show early onset and cross the threshold for FTT at around 6 months of age [3, 43].

Classification of feeding and eating problems in young children is problematic. The term feeding disorder, applied to the younger end of the spectrum, implicitly acknowledges the interactive nature of infant-caregiver feeding dyad. Described in similar terms in both ICD10 and DSM-IV (Table 1), the broad scope of the diagnostic criteria does not differentiate between the different types of infant/toddler feeding disorders with biological and behavioural aetiologies. The physical process of feeding can be disrupted through structural abnormalities or neurodevelopmental disabilities. Disruption of the process of learning to eat and accept new tastes during the critical "window" of opportunity can result in both oral-sensory and oral-motor dysfunction [2, 16]. Infants experiencing early adverse feeding experience such as recurrent vomiting (especially gastro-oesophageal reflux) are particularly at risk.

An attempt to refine the DSM IV criteria for the behavioural feeding disorders [6] (namely disorders of; (a) homeostatic state regulation, (b) of reciprocal

Table 1 DSM-IV diagnostic criteria—feeding disorder in infancy or early childhood

1. Feeding disturbance as manifested by persistent failure to eat adequately with significant failure to gain weight or significant loss of weight over at least 1 month
2. The disturbance is not due to an associated gastrointestinal or other general medical condition (e.g., oesophageal reflux)
3. The disturbance is not better accounted for by another developmental disorder (e.g., Rumination Disorder) or by lack of available food
4. The onset is before the age of 6 years

interaction, (c) of separation (Infantile Anorexia), (d) sensory food aversions, (e) those associated with a medical condition and (f) of post-traumatic origin) are often quoted but may have limited validity being overly dependent on case reports [2]. Unfortunately, much of the data around aetiology relate to clinic samples attending tertiary specialist clinical services [5, 12].

In contrast to the well-defined eating disorders (anorexia and bulimia nervosa), associated with preoccupation with weight and shape, the eating problems in middle childhood are characterised by idiosyncratic terminology, sparse literature and lack of robust research [12, 26]. In particular, their relationship to growth disorder is unclear.

Systematic review suggests there are four types of eating problems relevant to the preschool population which are not associated with abnormal cognitions or preoccupations about body or weight [12]. There appears to be a growing consensus that exclusive categories of feeding problems do not adequately capture their mixed aetiologies and even when there are major organic factors there is a complex interplay between biological, social and behavioural factors [8]. This has led to others suggesting alternative categories (Table 2). However, apart from persistent RE where growth may be affected, none of the other categories, and in particular SE, suggest the presence of an underlying regulatory abnormality leading to compromised growth. The precise nature of possible mediating mechanisms is as yet unknown.

The majority of what we know of prognosis comes from clinical experience and expertise [12]. On this basis, SE is thought to have a very good outcome. In most there is resolution by the teenage years with less than 1% becoming adult selective eaters. Appetites of those with RE increase and they tend to grow into thin but healthy adults. Although the majority of children with FTT in infancy achieve height and weight in the normal range [33] they tend to low BMI and height and weight for age [3].

Developmental and psychiatric disorder and growth

There have been observations since the 1940s, of a relationship between psychiatric disturbance in children and depressed height, weight and skeletal maturation. In a longitudinal study of 40 psychiatric patients aged 7–18, abnormal growth patterns of weight, height and head circumference are described in 63% [21]. Low BMI in males has been reported in a study of 51 adolescents with OCD [18]. In a study of 33 male children and adolescents with schizoid personality disorder or Asperger's disorder, low BMI was associated with both conditions but further reduced if associated with abnormal eating behaviour [17]. Similar findings were observed in a study of 36, 7–18 year olds, with Asperger syndrome [35]. The role of hyperactivity in mediating these effects is unclear, with no association found in one study [35] and in another, the presence of 28% of males with autism or Asperger syndrome with a BMI below the 5th centile said to be partly explained by hyperactivity [4].

Although AN is characterised by the presence of abnormal cognitions, a number of authors have, nevertheless, observed a link between it and previous feeding disorder in early childhood [20, 29]. The co-

Table 2 Types of feeding and eating problems in preschool children

Type of problem	Associated behaviour	Growth features
RE*	Poor appetite, normal nutrition, small amounts	Normal initially, Low height, low weight if persistent
SE*	Narrow range of food, extreme faddiness	Weight usually normal
FR*	Episodic, intermittent, situational	Not stated
FP*	Food avoidance, functional dysphagia, vomiting	Not stated
ITFA**	Only pureed or semi-solid food, full range food types	Weight normal or occasionally low
PE***	Limited number of foods and food types, strong preferences in presentation and preparation	Unaffected
PEB****	Narrow range of food, food refusal, extreme fear and contamination responses	Normal

*Lask and Bryant-Waugh (2000) cited in Fox and Joughin [12]
**Fox and Joughin [12]
***Jacobi et al. [21]
****Harris [15]

occurrence of AN and autism was initially suggested in case reports [11, 13]. Although no excess of eating disorders was identified within a clinic population with autism [4], a controlled follow up study of AN, showed a significant excess, with 18% of cases having evidence of an ASD at the time of onset of AN [14]. AN has also been shown to co-occur with other childhood onset developmental disorders, as well as ASD [39].

There are few data on the physical characteristics of children with autism. Abnormal linear growth and skeletal maturation have been reported but weight has not been found to be depressed. In a study of 117 children with infantile autism aged 2–15 years, the BMI of the males, but not the females, was significantly lower than controls, with 75% of males below the 50th percentile and 21% below the 5th centile, with no relationship with either intelligence or socioeconomic status. It is suggested that low BMI may be one of several physical features found in autism [25].

The co-occurrence of disorders such as ASD, AN, SE and OCD is suggestive of overlapping aetiologies. It has been proposed that a common central regulatory mechanism could influence both personality features and appetite regulation [26].

Feeding and eating problems in childhood autism

There is a notable discrepancy between the wealth of clinical experience of eating problems in autism and paucity of its systematic study. Pica, overeating, anorectic behaviour, complete food refusal as well as unusual food restriction and preferences are recognised [14]. Studies have tended to use retrospective parental survey with the methodological limitations of recall bias with contradictory results, suggesting both an absence [9] and an excess [40] in the incidence of abnormal feeding behaviours.

Other specific behaviours such as refusal to feed self and excessive drinking (suggesting poor self-regulatory processes), although not subject to systematic study, are nevertheless recognised in diagnostic interviews schedules such as the Diagnostic Interview for Social and Communication Disorders [41].

A small number of case series characterised by the presence of SE have been consistent in noting the association with autistic or autistic-like rigid or obsessional behaviour together with increased sensory sensitivity and food anxiety and phobia, suggesting an interrelationship between these features as a possible underlying mechanism [10, 26] (G. Harris, personal communication). A continuum is proposed from normal erratic food faddiness through to the autistic severely selective child [10]. However, feeding disorder in children with autism and Asperger syndrome is said to be of relatively late onset in the second year when their feeding style changes [15].

Studies of autistic children suggest around 50% with high levels of selectivity by food category or texture [1] or food cravings [27]. Although the significance of these studies is uncertain as comparison was not made with typically developing children or children with other developmental problems, neophobia in the general population is uncommon [26].

Despite the apparent association between autism and eating problems, no study has found a significant association between deficits in overall nutritional status and autism [27, 33], apart from one report of a high prevalence of iron deficiency [23].

Developmental and psychosocial characteristics of infants with FTT and how this may relate to autism

Critical review of the evidence in childhood FTT has moved the focus from maternal and social factors, through attachment theory to the role of infant factors. As in other behavioural phenomena, FTT should now be understood in a multidimensional context [34]. It is not within the remit of this paper to examine the complex family and social factors that are known to affect feeding and growth.

Learning to regulate self, suck, swallow and to time onset and termination of feeding by giving signals of hunger and fullness is the first stage eating behaviour [6]. Inability to master this stage results in ineffective feeding. Infants with FTT may lack normal sensitivity to internal hunger or satiation cues for example, at the age of 1 year, those with FTT did not compensate for additional calorie intake compared to controls [22]. Biological factors affecting self-regulation may be of particular importance in autism because of the known associations with sensory processing abnormalities. A range of abnormal responses (especially in response to sound, visual stimuli, touch, pain, temperature and smell) to is said to distinguish autism from mental retardation [14].

Disturbances in interactional patterns of the infant and caregiver with lack of engagement may lead to lack of pleasure, appetite and development of dysfunctional patterns such as vomiting [6]. Studies of the temperament and social responsiveness of infants with FTT suggest the coexistence of unusual qualities of social and emotional responsiveness. For example, subgroups of children with FTT are described as "irritable and non-cuddly" or as apathetic, apprehensive, withdrawn with poor development of vocalisation [34]. Another study of infants with FTT identified "difficultness" of temperament as one of

their defining features [15]. Unfortunately, most of these studies were not designed to explore these neurodevelopmental characteristics further.

A study describing seven infants, aged 13–30 months, with normal development, with serious feeding problems from shortly after birth of whom three needed nasogastric tube feeding and two, gastrostomy, all showed lack of interest in feeding and food refusal. All experienced weight loss when attempts made to wean off supplements [37]. Another small study of five hospitalised infants under 1 year with FTT [28] found that vomiting, feeding aversion and inadequate calorie intake were associated with poor social responsiveness.

In a study of the "biobehavioural" aspects of in infants under 12 months with FTT, low reactivity of the autonomic nervous system, felt to be indicative of low environmental adaptability, was associated with low behavioural responsiveness. These infants were described as difficult to interact with, because of limited and unusual facial expression, poor eye contact, minimal smiling and diminished vocal expressiveness [36]. Ambiguity of non-verbal response was observed in 1-year-old infants with FTT who also showed abnormalities of oral motor function such as aversion to touch and abnormal tongue and lip movement [24].

The decline in growth velocity after the first year of life is accompanied by decline in the rate of increase of appetite. It is a time at which many children start to be seen as "fussy" or "picky", and refusal to try new foods increases as a normal phase of development. However, normally developing children tend to imitate others and will attempt new foods if they observe others eating. Conversely, those with low levels of sociability and therefore social imitation, are more likely to exhibit neophobia [15].

Outcome studies for infants with refusal to eat are inconsistent with respect to "behavioural problems" and psychomotor development. Psychosocial problems appear to be correlated with severe persistent feeding problems [12]. Early studies of children with FTT suggested a close association with both developmental delay and with behavioural difficulties [30]. Subsequent studies of a population sample have not supported these cognitive findings [3]. Although some psychomotor faltering was evident in the second year of life, FTT did not relate to eventual cognitive abilities at 6 years apart from mild impact on quantitative and memory skills. A systematic review of the long-term outcome of failure to thrive in infancy, suggested that subsequent behavioural or emotional problems are seen in just a minority and academic and IQ tests showed no significant differences from controls, but the nature of the problems evident in that minority is unclear [31].

Summary of issues from the literature

There is a suggested relationship between low body size and some psychiatric disorders including autism but the cause and significance of this is unclear. Autism appears to be associated with feeding problems and abnormal food behaviour. The association has not been examined systematically. Infants with FTT show abnormalities of biological regulation and social responsiveness. Outcome studies have not clarified the relationship between these factors and eventual social function. It appears, therefore, that the nature of feeding problems in autism which are associated with growth failure in infants has not been adequately explored and characterised.

The case studies

These seven children derive from the author's clinic population of approximately 350 children with autism, diagnosed following assessment in the child development services of two UK health districts. Parental permission was obtained for publication. They are a sample of those with unusually severe feeding problems, and in whom retrospective clinical data indicated the presence of significant failure to thrive in early infancy as indicated by a fall across two major weight centile lines and associated with abnormal Body Mass Index which fell below the 0.4th centile in all cases. Three required medical intervention for maintenance of growth by nasogastric tube and/or gastrostomy feeding, six fulfilled criteria for ICD10 [42] diagnosis of childhood autism and one for the broader spectrum.

Their developmental and medical profiles are shown in Table 3, nature of the feeding disorder in Table 4 and growth details in Table 5.

Discussion

The frequency of growth failure in autism is unknown. The seven cases are a sample from a clinic population where adequate retrospective confirmatory growth data (particularly of height and BMI) was available. This study suggests an over-representation of children with autism with a BMI below the 0.4th centile. They therefore represent an extreme group. The increased prevalence of low BMI may indicate an early manifestation of the phenotype seen in older children [25], or a predisposition triggered particularly early by feeding problems. However, as five cases subsequently achieved weights and BMI within the normal range, this explanation is less likely.

Table 3 Developmental and medical profiles

Case	Sex	Age at diagnosis (year)	Cognitive levels		Medical problems and intervention
			Non-verbal	Verbal	
1	Male	3.10	Average	Moderate delay + disorder	–
2	Male	4.10	Average	Average	+ Gastroesophageal reflux to 3 months Functional rectal outlet obstruction with severe constipation Eosinophilic enteropathy, dysmotility syndrome Gastrostomy feeds at 39 months Colostomy at 8 years
3	Male	11.4	Average	Mild delay	+ NJT feeds at 9 months
4	Male	3.8	>Average	Moderate delay	–
5	Male	3.11	>Average	Average	–
6	Male	4.6	Average	Mild-moderate delay	+ Severe constipation at 4 weeks Allergic colitis NJT feeds at 42 months Gastrostomy feeds at 54 months
7	Female	5.4	Average	Mild delay	– 32 weeks gestation

Table 4 Feeding history

Case	Problems breast or bottle feeding	Problems weaning to solids	Abnormal food behaviour	Outcome
1	–	+	Refused solids until 2 years. SE—3 foods at 4 years. Excess drinking of milk to point of vomiting	Small improvement in SE at 5 years Height normal, wt centile low
2	–	+	Refused spoon Spat out or vomited solids SE-6 foods at 6 years RE Refusal to feed self Contamination fears at 6 years	Normal growth attained after gastrostomy, colostomy and relief of constipation resulted in improved intake Selectivity largely unchanged at 8 years
3	+	+	Poor suck Refused solids Unable to take food off spoon Refusal to feed self until 9 years ITFA	At 13 year food rituals and obsessive behaviour Anxious about cleanliness ITFA Normal growth
4	+	+	Distress and refusal if infant milk feed delayed No enjoyment of any food RE SE—1 food type at 4 years Refusal to feed self	Not known Family moved out of UK
5	–	+	Refused solids Disinterested and passive at feed time RE Refusal to feed self till >3 years	Problem gradually resolved just >3 years Normal growth attained
6	–	+	Gag on lumps No chewing ITFA RE SE—5 foods at 4 years	Normal growth on gastrostomy feeding Obsessive and ritualistic food behaviour
7	–	+	RE	Stable and unchanged RE at 7 years

BMI use in childhood is relatively recent and centiles have been developed primarily for use in management of obesity rather than FTT. It is particularly difficult to interpret BMI in infancy because of its dramatic increase in the first 8 months. BMI charts in FTT have uncertain clinical value and further experience is needed in using them for this purpose [7]. However, in the assessment of nutritional status

European Child & Adolescent Psychiatry (2008) Vol. 17, No. 4

Table 5 Growth details

Case no	Age at start of FTT (months)	Weight centile			BMI centile	
		Pre-FTT	Lowest	Post-FTT	Lowest	Post-FTT
1	14	50–75	<0.4	2–9	<0.4	9–25
2	4	9	<0.4	9–25	<0.4	75
3	9	9	<0.4	0.4–2	<0.4	2–9
4	40*	9–25	<0.4	0.4–2	<0.4	9–25
5	8	50	2	25–50	<0.4	25–50
6	18	25–50	0.4–2	9	<0.4	9
7	17	50	<0.4	2	<0.4	2–9

*Likely to be earlier from history but incomplete medical records

in older children and adolescents a BMI of below the 9th centile signals pathology because of the association with future significant health implications e.g., osteopenia and risk of bone fracture.

Two children presented with gastrointestinal disorders associated with increased risk of FTT and both received a gastrostomy for feeding. Otherwise the group did not present with characteristics typical of those at high risk of FTT. Five had undergone comprehensive investigations for FTT and had negative results for organic aetiology. The infant who was premature established feeding with little difficulty. Although three exhibited abnormal oral behaviour with food none had oromotor or other forms of dyspraxia. Where there were cognitive delays, these were confined to language function. The strong predominance of males reflects that in high functioning autism [39], but is also observed in both feeding disorder [12] (G. Harris, personal communication) and FTT. In the latter case, increased male vulnerability to psychosocial stress has been suggested as a contributing factor [32].

One child was adopted very soon after birth. In one case were there adverse economic factors (relating to unemployment) but no other reported adverse social factors.

In general the combined pictures do not fit the descriptions of feeding disorders in young children and are not consistent with the descriptions of SE, the disorder that has been most associated with autistic features. Consistent with other studies, the cases showed a combination of feeding problems. In all the feeding problems started at, or before, weaning and in six, FTT was evident in the first year of life. Although early onset of feeding problems is not unusual, it is not recognised as a characteristic in existing classifications of disorder, even those with suggested link with autistic traits. Four of the cases refused to feed themselves well into the nursery/preschool stage, a phenomenon not generally discussed in the literature on feeding problems.

Although the overall psychosocial context must be an intrinsic part of the appraisal of aetiology of any feeding disorder, the developmental issues more specific to autism raise additional possibilities which could inform management strategies. Clinical experience points to the frequent presence of abnormal hypo-, or hypersensitivity, in all the sensory modalities. When present in oral, gustatory and olfactory processing, there are obvious implications for the development of feeding patterns. In addition, disturbance of homeostatic and self-regulatory processes are also frequently observed clinically but, as yet, poorly researched. Disturbances relating to hunger and thirst would also have major implications in the aetiology of such feeding disorder. Lastly, some aspects of feeding behaviours in autism could represent a particularly early onset of obsessional or stereotyped behaviour. In addition, an infantile predisposition to anxiety or phobic responses as seen in older children with autism, may play an important role in shaping the final picture.

The literature, especially regarding very young infants, suggests an association between FTT and a range of abnormalities in both biological and social responsiveness. Temperamental features observed in infants with severe feeding problems with FTT may be early manifestations of a social communication disorder and it is possible that the subjects of these studies may have been infants with as yet unrecognised autistic features. The presence of poor oromotor skills, undemanding behaviour and low appetite could be secondary effects of under-nutrition as well as having a role in causing FTT [43]. Under-nutrition may therefore exacerbate subtle early manifestations of abnormal social behaviours, sensory responsiveness and obsessive behaviour, contributing to a more extreme clinical picture as seen in the cases presented.

There was inconsistent access to specialist intervention reflecting more general difficulties in service provision for this type of problem. Whilst most received some dietetic and psychology intervention, and the two most severe cases input from a specialist feeding clinic, none showed substantial improvement in feeding behaviour during engagement in a therapeutic programme. The child who spontaneously improved did so without professional intervention. In

two cases, (Cases 2 and 3) inducing hunger was expected to relax the severity of selectivity and improve self-feeding. In both cases this approach resulted in considerable weight loss. This suggests a similarity to older children with severe SE, with failure to respond to treatment and in some cases, exacerbation of symptoms [26].

These issues underline the severe, complex and likely intractable nature of the problem for which intensive and continued multimodal support is needed. Whilst individual cognitive behavioural therapy is felt to be an important first line treatment for older children [26] there is little literature relevant to the group in question. This study suggests that successful intervention will need to integrate management approaches to dysfunctional sensory processing, attachment, cognitive inflexibility and learnt behaviours, as well as associated anxiety or phobia. SSRI's have a recognised role in anxiety disorder [38] and have been suggested as having a role in adolescent eating disorders but there insufficient evidence for their effectiveness in this age group [19].

Conclusion

This study shows that the nature of feeding problems in childhood autism has not been well characterised but cuts across suggested discreet categories. Its aetiology is likely to have complex biological underpinnings with vulnerabilities due to dysfunction in sensory, cognitive and emotional response interacting with dysfunctional attachment and learnt avoidance and aversion behaviours to produce a severe and intractable problem.

There is an intriguing inter-relationship between growth, feeding problems and developmental and neuropsychiatric disorders which has yet to be explored systematically.

The presence of severe and persistent feeding problems, or atypical patterns of FTT, in very young children should alert clinicians to the possibility of an ASD. In the clinical evaluation of children presenting with ASD it is essential to obtain a detailed history of feeding behaviour and to evaluate their growth.

The management of autism related feeding and growth disorders presents unusual challenges and requires novel approaches. This study suggests that therapeutic intervention will need to integrate work on each of the areas of dysfunction together with management of anxiety or phobia regarding food.

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