

Anorexia nervosa – on the autistic spectrum?

Clare Allely wonders whether parallels between the two conditions might lead to novel therapeutic approaches

Numerous parental and anecdotal clinical reports, including some research studies, have highlighted that unusual eating behaviours and habits are often present in children with autistic spectrum disorders (ASD). Some researchers have argued that feeding difficulties in infancy may be an early marker for later diagnosis of ASD.

Studies have found neuropsychological, behavioural and cognitive similarities in individuals with ASD and individuals with anorexia nervosa, which highlights the question of whether anorexia should be considered within the spectrum of autistic disorders.

Is there no end to the variety of odd objects autistic children will eat? There are days when I believe my son would be quite happy with a daily menu of baby lotion, rabbit food – and perhaps a little toilet paper for roughage! (Feinstein, 2000)

Autism spectrum disorders (ASDs) are a group of neurodevelopmental disorders characterised by difficulties in three key domains: socialisation; communication; and restrictive and repetitive behaviours. Recent parental and anecdotal clinical reports, plus some research studies, have noted the presence of unusual eating behaviours in children with ASDs. They may refuse foods, be overly selective, or have an aversion to specific textures, colours, smells and temperatures. ‘Rigidity’ is also seen when it comes to the brands of particular foods.

In one study examining the mealtime behaviours of 24 young children (three to six years old) with ASD compared with those of 24 children with typical development, it was found that mothers reported negative mealtime behaviours in about half of the sample of children with ASD (Provost et al., 2010). A variety of negative mealtime behaviours were highly prevalent, including: throwing or dumping food; picky eating; gagging problems when eating; stuffing the mouths and cheeks with food; eating the same food in a repetitive way; and having routines or rituals with food or eating. Half of the children with ASD required their food to be prepared in a particular way. Others

have found similar behaviour: Ahearn (2001) came across a boy with autism who only ever ate a certain brand of macaroni and cheese, made to a particular recipe.

Other unusual eating behaviours such as pica have also been found in some individuals with ASDs. Pica behaviour is characterised by compulsive and recurrent consumption of non-nutritive items. Despite being one of the most frequently exhibited eating disorders in individuals with autism and other developmental disabilities, it remains underidentified, underreported to physicians and undertreated by interventions (Ali, 2001). Targets for pica behaviours are incredibly varied. In one study, 29.2 per cent of parents of children aged between 30 months and 18 years, who had been diagnosed with an ASD for at least six months, reported pica behaviours with sand and paper being most common (Kerwin et al., 2005). Other pica targets in order of frequency were string, metal, toys, coins, leaves, clothing, rocks/stones, Play-Doh, wood, soap, plastic and dirt.

Comorbidity

Eating disorders are categorised as disturbances of eating habits or weight-control behaviour that have a detrimental impact on physical health and/or psychosocial function (Mühlau et al., 2007). The DSM-IV diagnostic criteria for anorexia nervosa (AN) include the ‘refusal to maintain body weight at or above a minimal normal weight’ – specifically, maintaining a body weight at a level less than 85 per cent of normal weight for age and height. This is in addition to an intense fear of putting on weight, disturbed self-evaluation of own body weight and shape and amenorrhoea. Many AN patients also engage in compulsive exercising.

Interestingly, a significant number of studies have indicated a propensity for very low body weight (Sobanski et al., 1999) or comorbid eating disorders (Gillberg et al., 1995) among individuals

questions

Are there similarities in the behaviours of individuals with autistic spectrum disorder and individuals with anorexia nervosa?

Are there neuropsychological similarities in individuals with autistic spectrum disorders and anorexia nervosa?

resources

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with ASDs. Gillberg et al. (1995) found that the comorbidity of anorexia nervosa and Asperger's syndrome (AS) was as high as 12 per cent in their patients, giving rise to the possibility that some individuals with anorexia may have some autistic features. High rates of ASD and autistic symptomatology in adult eating disorders have been observed (Gillberg & Råstam, 1992).

Increased risks for underweight and disturbed eating behaviours among individuals with ASDs (in particular Asperger's syndrome) have also been found (Sobanski et al., 1999). Bölte and colleagues (2002) examined the association between ASDs and low body weight in subjects with autism or

Asperger's syndrome between the ages of 10 and 40 years of age. None of the subjects met diagnostic criteria for anorexia nervosa. Only hyperactive behaviour showed a significant association with BMI. Although low body weight is often present in male subjects with autism or Asperger's syndrome, the researchers argued that this association is inconsistent and partly mediated by hyperactivity.

Behavioural and brain similarities

Studies have also found behavioural or cognitive similarities in individuals with ASD and individuals with AN, such as poor cognitive flexibility (Tchanturia et al., 2011), difficulties with set shifting and excessive attention to details (Odent, 2010), and impairment with interpersonal functioning and social interactions (Coombs et al., 2011). Greater autistic traits in young people (8–16 years) with early-onset eating disorders were also suggested in another study by Pooni et al. (2012). They found that repetitive and stereotyped behaviour was more often seen in the early-onset group compared with typically developing peers, and there was also a trend towards higher levels of autistic social impairment. However, there were no differences between the two groups in terms of prevalence of ASDs.

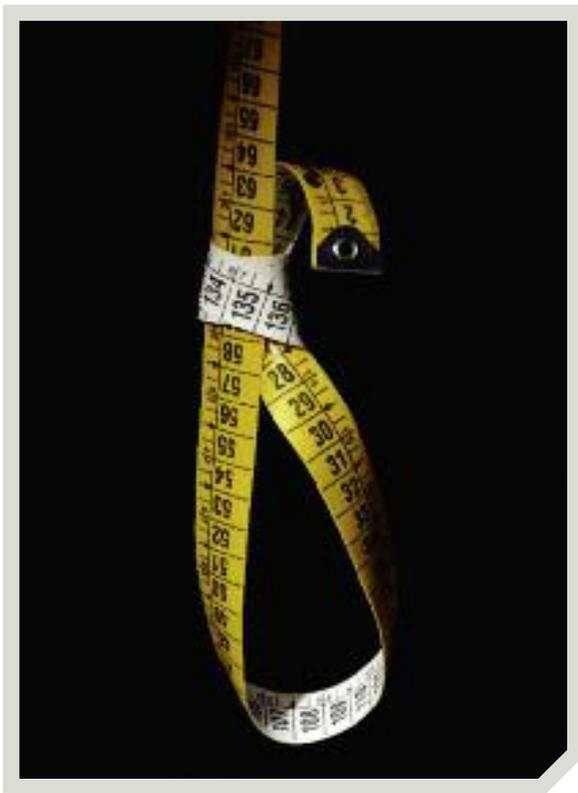
These behavioural similarities are consistent with studies looking at brain functions (using brain scanning and electroencephalography) of both these groups. In both ASDs (Waiter et al., 2005) and AN

(Uher & Treasure, 2005) studies have shown a left hemisphere preponderance (or right hemisphere deficits). Oxytocin processing impairments have also been demonstrated in both disorders. A study of mid-day blood samples from 29 autistic and 30 age-matched normal children (Modahl et al., 1998) showed that the autistic group had lower blood oxytocin levels compared with controls, and oxytocin increased with age in the control group only. Interestingly, oxytocin deficits have also been found in anorexia, with one study finding levels of oxytocin in the cerebrospinal fluid of women with 'restricting anorexia' to be significantly less than that in individuals with bulimia and control subjects (Demitrack et al., 1990).

Starvation itself has been shown to have a significant effect on brain function and to produce what appears to be an autistic presentation in the sufferer (Treasure, 2013). The Minnesota Starvation Study by Keys et al. (1950) involved 36 males who lived on a severely restricted diet for six months. Participants in this study had their calorific intake halved for six months, followed by three months of rehabilitation and re-feeding. During the starvation phase of the study, participants exhibited incessant preoccupation with food coupled with a variety of emotional, social and personality changes. Social isolation and avoidance of old and new relationships were observed. Changes to both personality and social behaviour did, however, revert to normal after re-feeding.

Should anorexia be considered within the spectrum?

Treasure (2013) revisited Gillberg and Råstam's (1992) hypothesis that AN should be considered within the spectrum of autistic disorders, conducting a literature search on studies exploring the behavioural traits and cognitive, emotional and neuro-anatomical intermediate phenotypes that are shared between individuals with ASD and anorexia nervosa. Wherever possible



High rates of ASD and autistic symptomatology in adult eating disorder have been observed

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studies were selected for inclusion if they involved samples of individuals who had recovered from the acute phase of their illness (i.e. anorexia nervosa) as well as studies that included first degree relatives and twins in order to investigate the impact of familial and/or genetic links. It was discovered that individuals with eating disorders do appear to share some familial (genetic) behavioural traits (social impairment and restricted and repetitive behaviours) and intermediate phenotypes (weak central coherence, and impaired set shifting and theory of mind) in individuals diagnosed with ASD. These areas also appear to be both causal and maintaining factors.

Treasure also found that studies indicated shared behavioural and intermediate neuropsychological traits in individuals with eating disorders and ASD. These traits are partly familial but are also heightened during the acute illness state, producing a pseudo-autistic state that is reversed following recovery. They may also be traits experienced as 'secondary to starvation'.

These findings have important clinical implications, as they emphasise the requirement for tailored treatment for eating disorders in individuals who are exhibiting these pseudo-autistic states. Neuropsychological feedback (Lopez et al., 2008) or cognitive remediation may be effective for those high in rigidity and weak in coherence (Tchanturia et al., 2008). Individuals who exhibit a deficit or difficulty with theory of mind and social impairments may benefit from work with the family that aims to improve communication (Treasure et al., 2012; Whitney et al., 2012) or derive clinical benefits from being administered oxytocin (Odent, 2010).

Others have also proposed that AN is a neuropsychiatric developmental disorder (NPDD). Autistic disorder is a classic presentation of an NPDD. Support for this

position came from an interesting case presented by Kerbeshian and Burd (2009) of a 12-year-old girl (Barbara) with high-functioning autistic disorder who developed Tourette's syndrome and obsessive-compulsive disorder. She went on to exhibit a distinct onset of partial anorexia nervosa, which included fear of gaining weight, body image distortions, food preference idiosyncrasies including avoidance of fat, dietary restriction, a pursuit of thinness, episodic self-induced vomiting, the missing of her menstrual cycles, and a 10 per cent decrement in expected weight for height. However, she did not meet the required 15 per cent

decrement in expected weight for height to qualify for the full syndrome.

Barbara's persistent preoccupation with and intense fear of weight gain or of being fat in AN may be considered to be a

preoccupation with stereotyped and restricted patterns of interest in a high-functioning person with autistic disorder. If AN and the comorbidities in Barbara's case are related, it may be expected that you would observe common features of non-pharmacological treatment approaches across these disorders. This may particularly be the case in individuals with ASD or AN, based on the literature which has outlined the various similarities between these two disorders. Similarly, treatment approaches typically used with individuals with neuropsychiatric developmental disorders might be effective in higher-functioning individuals with eating disorders (Kerbeshian & Burd, 2009).

Another group of researchers found a subgroup of patients with AN who also presented with symptoms and signs consistent with underlying neurodevelopmental disorders such as autistic spectrum disorders (Frampton et al., 2012). They explored whether neurobiological status (indexed by regional cerebral blood flow) at initial presentation predicts neuropsychological

status at four-year follow-up in a sample of female children with early-onset anorexia nervosa. Significant differences between subgroups (based on neurobiological status at initial presentation) and matched controls in long-term visual memory and cognitive inhibition were found at follow-up. These early findings provide some evidence that neurobiological abnormalities at initial presentation predict neuropsychological status at follow-up, suggesting a distinct neurodevelopmental subtype of early-onset anorexia nervosa. In essence, there appears to be a common pathway between early onset anorexia nervosa and a neurodevelopmental subtype such as autism.

Conclusion

So what exactly do all these findings mean for the question 'Should AN be considered within the spectrum of autistic disorders?'. Most of the relevant literature suggests there are some striking parallels in the cognitive, behavioural and pathological features of ASDs and AN. Therefore, it does seem reasonable to consider AN to be within the spectrum of autistic disorders. The findings also indicate that they may both fall on the same neurodevelopmental trajectory which leads us on to suggest that if this is indeed the case, there may be some value in comparing therapeutic approaches across the two conditions. As Kerbeshian and Burd (2009) suggested: 'treatment approaches for AN could all benefit if a NPDD perspective were to be applied to AN in addition to the already existing rich and fruitful research and clinical agenda'.

"there may be some value in comparing therapeutic approaches"



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