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Impairment of Social Function in Young Females With Recent-Onset Anorexia Nervosa and Recovered Individuals



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ABSTRACT

Purpose: A subgroup of individuals with anorexia nervosa (AN) displays social difficulties; however, it is not clear if individuals with comorbid autism spectrum disorders account for these difficulties.

Methods: We compared social function using the Autism Diagnostic Observation Schedule in 43 young females with first-episode AN who did not have comorbid autism spectrum disorder, 28 individuals recovered from adolescent-onset AN, and 41 healthy comparison individuals (age range 14–22 years). We measured adaptive behavior with the Vineland-II parent questionnaire, and aspects of social cognition with psychological tests, such as the Reading-the-Mind-in-the-Eyes test, Profile of Nonverbal Sensitivity short version, The Awareness of Social Inference Test, Animated Triangles, and the CANTAB Affective Go/No-go task.

Results: Participants with first-episode AN and those recovered from AN displayed difficulties in social function, which were not associated with body mass index or other state factors of the disorder in those with first-episode AN. Mood problems and anxiety were not associated with these difficulties. Parents rated participants with first-episode AN lower than recovered and control participants on the Socialization Domain of Vineland-II. Finally, only participants recovered from AN demonstrated deficits in specific domains of social cognition: perceiving nonverbal bodily gesture and vocal prosody.

Conclusions: Young females with first-episode AN and those recovered from AN displayed impairments in social function, which may represent more stable traits of the disorder. Only participants recovered from AN demonstrated deficits in social cognition.

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IMPLICATIONS AND CONTRIBUTION

On the group level, young females with anorexia nervosa without autism spectrum disorders displayed impaired social function but no inferior social cognition capacity. Recovered individuals were impaired in their social function showed few social cognitive deficits. State factors of the illness did not explain impairment of social function.

Conflicts of Interest: The authors report no conflicts of interest.

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Social difficulties are inherent in current models of vulnerability and maintenance of anorexia nervosa (AN) [1], and a better understanding of these may refine interventions and improve outcome for this serious disorder. However, evidence of social difficulties in young individuals with a recent onset AN and in those recovered from AN is limited. We studied these difficulties at two descriptive levels: social function and social cognition.

Social function encompasses interactive behavior and capacity for socioemotional reciprocity [2]. Social cognition is broadly defined as the mental operations underlying social interaction [3]. Deficits in social cognition are documented in individuals with AN [4,5]. However, the relationship between behavioral and cognitive aspects of social difficulties is not clear, and instruments to reliably quantify difficulties in social function are scarce. The Autism Diagnostic Observation Schedule (ADOS) measures observable interactive behavior in a standardized form [6]. It is intended to qualify a diagnosis of autism spectrum disorder (ASD), a spectrum of developmental disorders characterized by sociocommunicative deficits, and mental and behavioral inflexibility. However, the ADOS quantifies behaviors without assumptions of possible underlying mechanisms, and we therefore used the ADOS to assess social function in AN.

We investigated social function in young females with firstepisode AN and those recovered from adolescent-onset AN, both groups without comorbid ASD. In exploratory analyses, we assessed social function via parent reports and social cognition via experimental tests. Finally, we explored associations between social function and social cognition across diagnostic groups.

We hypothesized that young females with first-episode AN would show impairments in social function and that those recovered from AN would perform intermediate between those with first-episode AN and controls.

Methods

Subjects

We invited young females with a recent onset of their first episode of AN (International Classification of Diseases [ICD-10]: F50.0 or F50.1) from the Centre for Child and Adolescent Mental Health Services (CAMHS; 14- to 17-year-old patients), the Capital Region of Denmark. Recent onset was defined as onset within a maximum of 12 months. Prior psychological treatment was allowed but infrequent, as CAMHS is usually the first line of treatment in Denmark. Inclusion criteria for this group included a body mass index (BMI) percentile corrected for age <25th for 14–15 year olds and a BMI <18.5 kg/m² for participants 16 years or older. All eligible CAMHS patients between July 2012 and March 2015 were invited if possible. In addition, we recruited a few young adults with first-episode AN from the Stolpegaard Psychotherapy Centre (18- to 21-year-old patients), the Capital Region of Denmark. Our first-episode AN participants thus were representative of a young population with a recent onset, without substantial epiphenomena of chronicity.

Second, we invited young females recovered from AN (ICD-10: F50.0 or F50.1) with onset in late childhood or adolescence. They were identified through a quality assurance survey. Recovery was defined as Eating Disorders Examination (EDE) global score within one standard deviation (SD) of non-AN norms [7], \geq 9 points on the Morgan Russell Outcome Assessment Schedule [8], and no current eating disorder pathology or current treatment for an eating disorder. We established the

absence of eating disorder pathology for the last 3 months with EDE diagnostic questions and normal body weight for the last 12 months via self-report.

Finally, we invited control participants via advertisements in state schools, halls of residence, and colleges in the catchment area of the hospital. Inclusion criteria were a normal body weight and absence of psychiatric disorders throughout their lifetime (minor exceptions included transient childhood tics and adjustment disorders).

Comorbidity is well documented during and after the course of AN [9], and accordingly, we did not exclude recovered individuals with past or present psychopharmacological treatment. To address our primary question of whether social deficits were confined to individuals with comorbid ASD, exclusion criteria further included ASD for all three groups. As a screening for undetected ASD, parents completed the Social Communication Questionnaire [10] and the Asperger Syndrome Screening Questionnaire-Revised Extended Version [11]. If ratings were above the established cutoffs, we interviewed parents with the Autism Diagnostic Interview-Revised [12]. Combined, these procedures ensured that no participants fulfilled diagnostic criteria for childhood autism (F84.0) or Asperger syndrome (F84.5).

We assessed psychiatric symptoms with the Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present and Lifetime version [13] and the Beck Youth Inventory [14], and intelligence with the Reynolds Intellectual Assessment Scales [15]. Clinical data at the time of treatment for AN were available for the recovered participants, enabling comparison of AN severity at onset between the clinical groups. Finally, the regional Scientific Ethical Committees (project number H-2-2012-027) and the Danish Data Protection Agency approved the study, and participants and legal caretakers gave informed consent according to the guidelines of the Danish Health and Medicines Authority.

Outcome measures

We measured social function with the ADOS-2, Danish version, module 4 [6]. During the ADOS observation, the observer orchestrates a series of situations with a social press for communication and interaction. The ADOS algorithm yields two subscales: Communication and Reciprocal Social Interaction, and a "Communication and Social Interaction Total" (ADOS-Total). Our main outcomes were the ADOS-Total and the two subscales. In module 4, the clinical cutoff for ADOS-Total is ≥ 7 , for the Communication Subscale is ≥ 2 , and for the Reciprocal Social Interaction Subscale is ≥ 4 . These cutoffs represents a 90% sensitivity and a 93% specificity for distinguishing ASD from nonspectrum cases [16].

Interrater reliability of the main rater and an experienced ADOS-certified psychologist blind to group was monitored in 15 randomly chosen cases, and weighted Kappa for all items was satisfactory (.77).

We used the Socialization Domain of Vineland-II [17] as an additional probe of parent-reported social function measuring adaptive behaviors in everyday life.

We documented social cognitive performance with tests tapping into the following four subdomains identified by the Measurement and Treatment Research to Improve Cognition in Schizophrenia [18].

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