Psychological Bulletin
2007, Vol. 133, No. 6, 976–1006

0033-2909/07/$12.00 DOI: 10.1037/0033-2909.133.6.976

Anorexia Nervosa and Autism Spectrum Disorders: Guided Investigation of Social Cognitive Endophenotypes

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Death by suicide occurs in a disproportionate percentage of individuals with anorexia nervosa (AN), with a standardized mortality ratio indicating a 57-fold greater risk of death from suicide relative to an age-matched cohort. Longitudinal studies indicate impaired social functioning increases risk for fatal outcomes, while social impairment persists following recovery. Study of social cognition in AN may elucidate impaired processes that may influence therapeutic efficacy. Symptoms of autism spectrum disorders (ASD) are overrepresented in those who evidence a chronic course. Relative to that in AN, social information processing in ASD is well characterized and may inform systematic study in AN. This article (a) reviews impaired interpersonal processes in AN, (b) compares the phenotype of AN with that of ASD, (c) highlights deficits of social cognitive disturbance in ASD relative to AN, and (d) proposes a new framework to understand the interaction of individuals with AN with their social context.

Keywords: anorexia nervosa, social cognition, autism, interpersonal functioning, eating disorders

Individuals with anorexia nervosa (AN) have a nearly 12-fold greater risk of death from all causes and a 57-fold greater risk of death from suicide relative to their age-matched peers (Keel et al., 2003). Such troubling statistics speak to the incapacitating, perplexing, and isolating nature of AN, a severe psychiatric illness that negatively impacts the biological, emotional, and psychosocial functioning of both the affected individual and her or his family (Harris & Barraclough, 1998; Treasure, Whitaker, Whitney, & Schmidt, 2005). In fact, AN exacts a toll on the family reported to exceed that experienced by families with a child with a severe psychiatric disorder such as schizophrenia (Treasure et al., 2001). Although guiding epidemiologic data are currently lacking, there has been increasing concern regarding several notable trends: (a) an upsurge of AN in both younger and older ages than has been previously reported (Decaluwe & Braet, 2003; Johnson, Cohen, Kasen, & Brook, 2006; Marcus & Kalarchian, 2003), (b) increased incidence in minority groups hitherto thought protected (Decaluwe, Braet, & Fairburn, 2003; Tanofsky-Kraff et al., 2003), and (c) recent published reports of extensive illness burden among family members (Treasure et al., 2001; Treasure, Whitaker, Whitney, & Schmidt, 2005; Whitney & Eisler, 2005). Such patterns highlight the broad impact of these illnesses and underscore the urgency of alternative approaches to illness management.

While advances have been made in the treatment of adolescents with a relatively short duration of illness (Lock & le Grange, 2005), progress in the treatment of adults with AN remains limited, at best (W. S. Agras et al., 2004; Kaplan, 2002). Of note, a report issued by the National Institute of Mental Health (W. S. Agras et al., 2004) emphasized the absence of innovative treatment approaches over the past several decades. This conclusion was recently echoed in an evidence-based review calling for novel intervention strategies that target core biological and cognitive features yet remain acceptable to patients (Berkman et al., 2006). Thus, there is a need for treatment development sensitive to the phenomenology of individuals with AN.

One relatively unexplored domain for intervention in AN is interpersonal functioning (McIntosh, Bulik, McKenzie, Luty, & Jordan, 2000). This neglect is surprising given the profound inter-

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This work is supported by National Institute of Mental Health Grant 1-R01-MH-078211-01, Neurodevelopmental Process of Social Cognition in Anorexia Nervosa and Autism, and National Institute of Neurological Disorders and Stroke Grant 1-K23-MH-070-418-01 awarded to Nancy L. Zucker. Kevin A. Pelphrey is supported by a career development award from the National Institute of Mental Health and by a John Merck Scholars Award. Molly Losh is supported by National Institute of Health Career Development Award K12 RR023248.

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personal deficits reported in individuals with AN, deficits often reflexive of trait levels of disturbance (I. C. Gillberg, Rastam, & Gillberg, 1995). Indeed, a growing body of evidence highlights premorbid impairment in interpersonal functioning (C. Gillberg & Rastam, 1992), the interference of interpersonal skills on treatment outcome (Goodwin & Fitzgibbon, 2002), and the negative impact of sustained social impairment on illness prognosis (Keel, Dorer, Franko, Jackson, & Herzog, 2005; Rastam, Gillberg, & Wentz, 2003). Of interest, McIntosh et al. (2005, 2006) compared the effects of three manualized psychotherapies in adult AN, including interpersonal psychotherapy. Interpersonal psychotherapy did not fare well, demonstrating inferior outcomes to specialist supportive clinical management (the active control condition). Indeed, if the hypotheses put forth in this review have credence, this finding is consistent with our proposed model of interpersonal functioning in AN. It may be that for an intervention in the social domain to be effective, more basic social cognitive processes need to be addressed prior to a more macro-level, goal-oriented intervention like interpersonal psychotherapy. In contrast, interventions that enhance social cognitive processes may address existing barriers to treatment progress and provide innovative domains to enhance and create intervention strategies.

A necessary prerequisite for such treatment innovation is knowledge of the behavioral and cognitive features that characterize interpersonal processes in individuals with AN. Unfortunately, social cognitive processes are vastly understudied in AN—in sharp contrast to other psychiatric disorders such as schizophrenia (Pinkham, Penn, Perkins, & Lieberman, 2003) and autism spectrum disorders (ASDs; Klin, Jones, Schultz, Volkmar, & Cohen, 2002a), in which the social phenotype is being meticulously characterized, both behaviorally and neurocognitively. Importantly, the developmental pathways of schizophrenia and ASD differ. Despite this, the comparison of social cognitive processes across these diagnostic classes has helped to delineate common and distinct areas of dysfunction and provided vital information to understand further the comparative pathophysiology of these syndromes. As researchers in the field of eating disorders widen their lens beyond strict diagnostic syndromes and consider the interaction of an individual with AN with her or his interpersonal context, an entirely novel conceptualization of AN begins to emerge.

Investigation of the social phenotype in AN is in line with the increased emphasis on the definition of endophenotypes in the study of psychiatric illness (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003; Bulik et al., in press; Gottesman & Gould, 2003). Endophenotypes are defined as measurable, heritable traits falling midway between the distal genotype and full diagnostic phenotypic expression of a disease (Gottesman & Gould, 2003). For example, in schizophrenia, patients report difficulty in filtering information from multiple sensory channels (Swerdlow, Light, Cadenhead, Sprock, & Braff, 2005). Neuropsychological tests, such as assessments of prepulse inhibition of the startle response, have been developed to assess this feature (Clementz, Geyer, & Braff, 1997). Evidence of this deficit in patients and family members relative to healthy control participants provides preliminary support of this feature as a putative endophenotype (Cadenhead, Swerdlow, Shafer, & Braff, 2000). Often the evident phenotypic expression of pathology provides clues to underlying cognitive or neurobiological processes that may serve as endophenotypes. The delineation of social cognitive capacities, guided by behavioral observations of this syndrome, can help delineate social cognitive endophenotypes of AN.

To achieve this goal, we explore what is known of the social phenotype of AN and demonstrate areas of overlap with the broad phenotype of ASD. From these similarities, we use the roadmap of systematic inquiry into social cognitive processes of ASD to guide review of the cognitive neuroscience subserving social functioning in AN (Figure 1). This comparison helps to highlight areas of future study in AN. Finally, we present a new framework for the study of AN that integrates previous etiological models that emphasize habit learning (Steinglass & Walsh, 2006) and fear conditioning (Strober, 2004) in AN with the interaction of these learning patterns on the social world.

Why Study Social Cognition in Anorexia?

AN is a disorder in which profound preoccupation with weight and shape contributes to the desired maintenance of a severely low body weight. Accompanying these symptoms is a disturbance in the manner in which the body is experienced. In essence, an individual with AN is disconnected from her internal experiences and thus does not use internal signals of hunger, fatigue, or affective state to guide behavior effectively (Miller, Redlich, & Steiner, 2003). This lack of integration is further compounded by difficulties in interpreting internal emotional experiences, deficits often referred to as poor interoceptive awareness (Fassino, Piero, Gramaglia, & Abbate-Daga, 2004).

Given this profound level of disturbance of the individual with AN in interaction with herself, why study interpersonal interaction in AN? There are several compelling reasons. First, improvement in social acceptance and interpersonal proficiency are arenas in which individuals with AN are motivated to improve (Serpell, Teasdale, Troop, & Treasure, 2004; Serpell, Treasure, Teasdale, & Sullivan, 1999). While limited motivation for treatment is commonly reported (Strober, 2004), a desire for interpersonal acceptance and concern for the opinion of others are factors that may actually serve to sustain symptom severity (Bizeul, Sadowsky, & Rigaud, 2001). Treatments targeting this area of functioning may enhance treatment engagement. Further, if fears of negative evaluation and interpersonal insecurity prolong the illness, then interventions that address these fears may enhance motivation to address the eating disorder symptoms themselves. Second, the only effective treatments for youths to date rely on the involvement of the family (Lock & le Grange, 2005). Despite their relative success, some adolescents and their families fare poorly in family therapy, and this lack of success has been associated with poor interpersonal communication (Eisler et al., 1997). Treatments that enhance communication may potentiate interventions that have already demonstrated preliminary success. Finally, in other severe psychiatric illnesses such as schizophrenia, social cognitive indices such as social perception have been reported to be crucial mediators between neurocognitive impairment and functional status (Sergi, Rassovsky, Nuechterlein, & Green, 2006). Thus, interventions that improve social cognition may offer a potent strategy to impact quality of life in severe chronic illness (Treasure et al., 2005). The study of social cognition, the characterization of social phenotypes, and, ultimately, the delineation of social cognitive endophenotypes may provide an entirely new arena for treatment development in a psychiatric illness that continues to be one of the

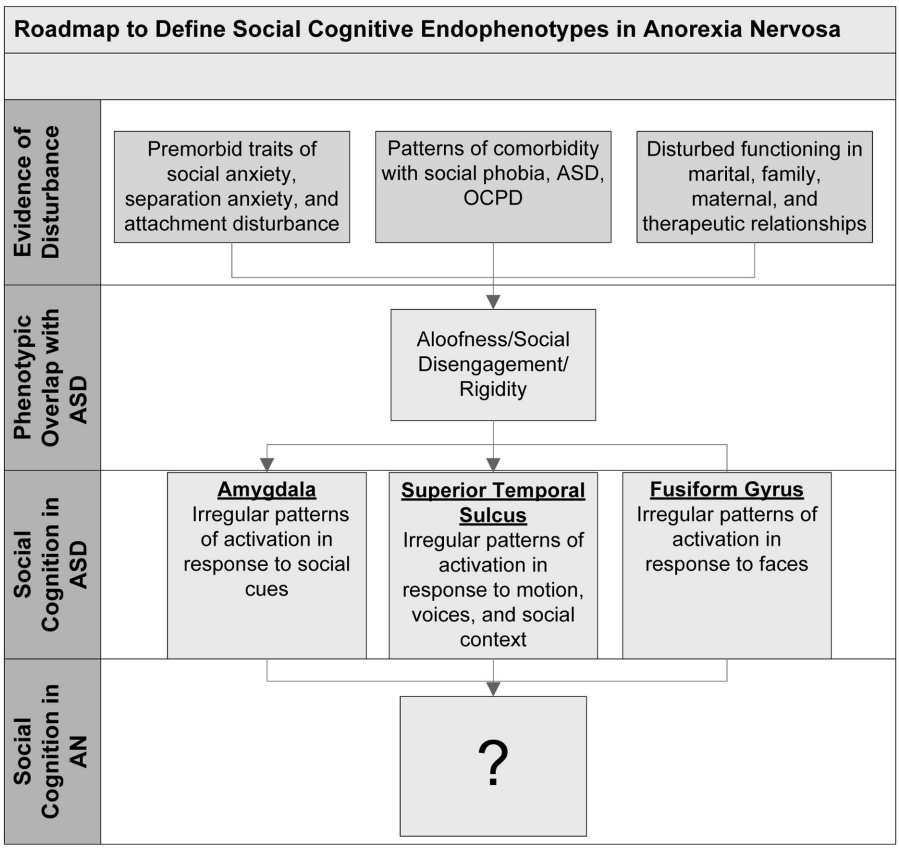

Figure 1. An overview of the strategy used to review the evidence of social cognitive disturbance in anorexia nervosa (AN) relative to autism spectrum disorders (ASDs). OCPD = obsessive-compulsive personality disorder.

leading causes of psychiatric mortality (Harris & Barraclough, 1998). Indeed, such strategies of treatment development may be sensitive to the phenomenology of individuals with AN and their families.

The Extent of Social Deficits in Anorexia

Our approach to this review is first to use the thesaurus in PsycINFO and MEDLINE to arrive at relevant keywords that were subsequently expanded in each search engine. We scanned reference lists for further relevant articles and then located articles in which interpersonal functioning was the central theme in Web of Knowledge, a database that provides links to articles that have subsequently cited the referent article. We reviewed method sections to determine the diagnosis of the sample and whether results were reported separately for individuals with AN relative to bulimia nervosa and whether differentiation of AN subtypes was noted. We excluded articles that did not differentiate by eating disorder diagnosis. We followed this approach for each topic area of the review.

Premorbid Social Functioning

Interpersonal difficulties have been described as part of the phenomenology of AN since its original appearance in the medical literature in the late 19th century (Pearce, 2004). Rather than being an artifact of the ill state or a manifestation of malnutrition, two strategies have been applied to support premorbid difficulties in interpersonal interaction: retrospective reports of childhood social functioning (Anderluh et al., 2003) and patterns of comorbidity with childhood onset diagnoses defined by social deficits, for example, separation anxiety disorder (Silberg & Bulik, 2005), social phobia (Melfsen, Walitza, & Warnke, 2006), and neurodevelopmental disorders of the autism spectrum (Connan, Campbell, Katzman, Lightman, & Treasure, 2003; Rastam et al., 2003). Both strategies have supported the presence of difficulties in social interaction prior to the onset of disturbed eating.

Increasingly, research on AN has included greater diagnostic specificity, not only distinguishing AN from bulimia nervosa but also distinguishing restrictive from binge/purge subtypes of AN. As delineated in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994), the *restrictive subtype* refers to individuals who engage in excessive food restriction and/or excessive exercise while the *binge/purge* subtype refers to those individuals with AN with a history of binge eating and purgative behavior via self-induced vomiting, misuse of laxatives, or other inappropriate compensatory behaviors. Although research is divided with some emphasizing similarities across subtypes and diagnoses (Fairburn, Cooper, &

Shafran, 2003), other research finds support for the distinction in epidemiological, genetic, and descriptive studies (e.g., Ribases et al., 2004, 2005). In this review, we indicate where subtypes are differentiated and highlight future directions for research with consideration of these subtypes.

Reports of childhood functioning prior to illness onset reflect long-standing patterns of interpersonal discomfort. Study of the developmental course of psychiatric comorbidity is methodologically complex (Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006). Notwithstanding, a number of rigorous investigations have helped to unravel the timing of disorder onset of psychiatric comorbidities germane to social functioning in individuals with AN (Godart, Flament, Perdereau, & Jeammet, 2003; Godart, Perdereau, Jeammet, & Flament, 2003; Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). Across these studies, findings have consistently revealed an onset of social anxiety prior to the onset of disordered eating (Kaye et al., 2004), an elevated presence of social anxiety relative to the general population (Godart, Flament, Perdereau, & Jeammet, 2002), and inconclusive evidence of higher rates of early social anxiety in individuals with a specific subtype of AN (Godart et al., 2006; Kaye et al., 2004). To illustrate, in a study of the developmental course of anxiety disorders in relation to eating symptomatology, Godart, Flament, Lecrubier, and Jeammet (2000) reported 75% of individuals with AN described the onset of an anxiety disorder prior to their eating disturbance, with 55% of the sample endorsing social phobia (Godart et al., 2000). Elevated prevalence was further confirmed in a later study that controlled for potential confounding variables such as age, duration of illness, and mode of treatment (Godart et al., 2006), findings replicated in several separate laboratories (Kaye et al., 2004; Melfsen et al., 2006). Kaye et al. (2004) reported numerically but not significantly higher prevalence of social anxiety in AN–purging versus AN–restrictive subtypes in a well-characterized sample of individuals with AN (Kaye et al., 2004). Thus, the course of social anxiety suggests that it predates the eating disturbance.

Evidence of persisting social disturbance is further supported by longitudinal, population-based investigations. Perhaps the most convincing body of work documenting premorbid and persisting social impairment emerged from the rigorously executed, population-based study of all adolescent onset AN cases born in 1970 in Göteborg, Sweden, conducted by Gillberg, Rastam, and colleagues (C. Gillberg & Rastam, 1992; C. Gillberg, Rastam, & Gillberg, 1994; I. C. Gillberg et al., 1995; I. C. Gillberg, Gillberg, Rastam, & Johansson, 1996; Ivarsson, Rastam, Wentz, Gillberg, & Gillberg, 2000; Nilsson, Gillberg, Gillberg, & Rastam, 1999; Wentz, Gillberg, Gillberg, & Rastam, 2001). These researchers identified a cohort of 51 individuals with AN with a mean age of 16 years at baseline and now have followed this sample in parallel with an age-, sex-, and school-matched sample over a 10-year period (Nilsson et al., 1999; Wentz et al., 2001; Wentz, Gillberg, Gillberg, & Rastam, 2000). Baseline methodology included a semi-structured family-genetic interview with the child's mother regarding psychiatric, neurodevelopmental, and physical symptoms among first degree relatives, city-wide physical exams of all adolescent students, and blind medical chart reviews (I. C. Gillberg, Rastam, & Gillberg, 1994). Five and 10 years later, follow-up assessments were conducted during which a comprehensive neurodevelopmental exam, structured clinical interviews, a neuropsychological battery, and assessment for neurodevelopmental disorders such as ASD were conducted. A longitudinal population-based study provides a unique window to disentangle enduring behavioral patterns from disease remnants.

Results from this investigation supported a sustained pattern of interpersonal deficit in a subset of individuals with AN. Before discussing findings regarding social functioning, it is noteworthy that after 10 years only 39% of the sample with AN at baseline were free of eating disturbance compared with 90% of the comparison group (Wentz et al., 2000, 2001). Limiting our discussion to findings of interpersonal difficulties, individuals with ASD were overrepresented in the group with AN (28% vs. 12%), and of those individuals with AN who exhibited a poor outcome, individuals with ASD were disproportionately present (I. C. Gillberg et al., 1994). Finally, in the group with AN, significantly more relatives had two or more of the four social impairment symptoms of autism (Rastam et al., 2003). As individuals with interpersonal deficits are disproportionately represented among individuals with AN who exhibit a chronic course of illness, and those individuals with interpersonal disturbance endorse symptoms of ASD, greater understanding of the nature and impact of ASD in AN is needed.

Research on attachment provides an alternative explanatory model for early disruptions in interpersonal relations in AN. A secure attachment between mother and child is a telling index of healthy parent–child interaction and is pertinent to future adaptive functioning (Okearney, 1996; Schieche & Spangler, 2005). As originally formulated by the contributions of Bowlby (Bowlby, 1969), Ainsworth (Ainsworth, Blehar, Waters, & Wall, 1978), and Harlow and colleagues (Seay, Hansen, & Harlow, 1962), among others, a secure attachment is a signal of perceived security on the part of the child (Bowlby, 1973). This felt security is hypothesized to result from reciprocal interactions in which the parent is responsive to the child's cues of physiological and emotional need. A test of this stability is Ainsworth's Strange Situation in which the infant or toddler is exposed to danger in a laboratory setting (Ainsworth et al., 1978). At such times, children who are securely attached will adapt to the parent's absence, seek the parent in danger, and become comforted with her reappearance. This emotional response on the part of the child is hypothesized to result from viewing the mother as the source of needs gratification and resolution of distress (Ainsworth et al., 1978).

A growing literature has supported early disruptions in attachment in AN that continue to manifest in adult relationships (Troisi, Massaroni, & Cuzzolaro, 2005; Ward, Ramsay, Turnbull, Benedettini, & Treasure, 2000). Given the adolescent age of onset, studies of attachment in AN have been necessarily based on retrospective reports. Notwithstanding the bias inherent in such recall, these reports have offered a window into the perceptions of the individual's childhood parental relations. This literature, combined with early reports of separation anxiety, has suggested early difficulties in establishing interpersonal security.

Before briefly highlighting relevant literature on attachment in AN, some considerations are necessary. Premature interpretation of the attachment and family functioning literature has opened an unfortunate chapter in research on eating disorders. Mimicking the legacy of the "schizophrenogenic mother" in schizophrenia (Hartwell, 1996) and the "refrigerator mother" in autism (Bettelheim, 1967), findings of attachment disturbance in AN do not imply the presence of maternal neglect or indifference. Rather, the ability to

read the cues of a young child varies within the population and is a complex interaction of factors such as, but not limited to, social cognitive capacities and interpersonal anxiety and is compounded by the child's ability to communicate need (Deater-Deckard & Petrill, 2004). Indeed, this latter point has been relatively neglected in the literature at the expense of the mother's ability to decipher cues. Thus, attachment disturbance provides an index of disruption in early parent–child communication and, for our purposes, provides evidence of early disturbances in social interaction only.

Several innovative research designs have minimized the methodological challenges inherent in attachment research. Shoebridge and Gowers (2000) performed a retrospective case-control study comparing the medical records of 40 individuals diagnosed with AN with a sample of age- and sex-matched nondiseased participants. Results indicated that children who later developed AN experienced greater distress at the first separation and had their first overnight experience away from home at an older age (Shoebridge & Gowers, 2000). A unique design by Waters, Beumont, Touyz, and Kennedy (1990) employed structured interviews with the mothers of twins, one of whom was diagnosed with AN. Reports of between-siblings functioning were compared, and additional comparisons were made with nontwin siblings of an individual with AN. Irrespective of twin status, mothers reported that their daughters with AN related less well to their healthy siblings than the siblings related to them. While not an index of attachment, the differences in interpersonal skill in this unique design deserve mention. A study by Troisi, Massaroni, and Cuzzolaro (2005) examined the relationship between early separation anxiety and current attachment disturbance and reported a higher correlation between childhood separation anxiety and adult attachment disturbance in adult women with AN relative to those with bulimia nervosa, despite equal prevalence of childhood separation anxiety. The association of retrospective reports with current attachment difficulties was supported in two other investigations (Ward et al., 2000; Ward et al., 2001).

Developmental pathways may also inform the etiology of interpersonal disturbance. Silberg and Bulik (2005) examined the distinct and shared gene–environment relationships in the development of overanxious disorder, separation anxiety, and eating disorders in early and middle adolescence. Pertinent to this review is that overanxious disorder was characterized by symptoms of self-consciousness and reassurance seeking in addition to worry and physical symptoms, thereby containing items with an inherent social component. Results indicated a shared genetic diathesis to separation anxiety, overanxious disorder, and eating disorders; distinct genetic pathways to early onset eating disorder symptoms; and shared environmental contributions to early and later onset separation anxiety. Such results highlight the need to understand the early onset of social discomfort and its relation to the early appearance of eating disorder symptoms and stress the importance of interventions to promote interactions that enhance interpersonal security. There has been limited study of friendship quality in AN as a focus of study, although this feature forms part of outcome measurement scales such as the Morgan–Russell scales (e.g., van der Ham, van Strien, & van Engeland, 1998) and thus may provide an opportunity to examine potential deficits on data already collected. In sum, patterns of early childhood social and separation anxiety, suggestive evidence of early attachment disturbance, and population-based studies describing elevated rates of ASDs combine to characterize the childhood of individuals with AN as fraught with social discomfort and insecurity.

Interpersonal Difficulties in the Family Context

Reports of family dysfunction have been part of the characterization of AN since its inception and continue to shape the development of treatment interventions (Ebeling et al., 2003). To be sure, the most extensive information regarding relational functioning in AN is in the area of family interactions (Dare, Le Grange, Eisler, & Rutherford, 1994). The influential early work of Minuchin (Minuchin, Rosman, & Baker, 1978) spurred hypotheses regarding specific transactional patterns that would be witnessed in families with a child with AN, that is, a psychosomatic family (Minuchin et al., 1978). These theoretical formulations greatly influenced the course of research in AN, that is, a search for certain styles of interaction relative to nondisordered families or alternative diagnoses. Findings to date have not supported a specific family pattern (Cook-Darzens, Doyen, Falissard, & Mouren, 2005; Whitney & Eisler, 2005). In fact, the literature can best be summarized as being "consistently inconsistent" (see Whitney & Eisler, 2005), with widely discrepant descriptions of patterns of interaction. Despite this, a notable finding is the elevated rates of dysfunction relative to control families across investigations (Cook-Darzens et al., 2005; Dare et al., 1994; Rastam & Gillberg, 1991).

Rather than styles of interaction, an alternative perspective to consider family functioning in AN is the frequency of discrepant reports among family members. If present, such discordance may be suggestive of differences in the manner family members view social information. In fact, such inconsistencies have been described regarding the family's level of affective expression (Casper & Troiani, 2001), emotional closeness (Cook-Darzens et al., 2005; Dare et al., 1994), organization (Dare et al., 1994; Karwautz, Nobis, et al., 2003), and conflict avoidance (Cook-Darzens et al., 2005; Karwautz, Nobis, et al., 2003). For example, Cook-Darzens et al. (2005) described the intrafamilial perceptions of 40 families with a child with AN as being significantly more conflicting than the perceptions of nondiseased control families across areas of functioning (Cook-Darzens et al., 2005). Also discordant are descriptions of family functioning between the clinician and various family members from patient to parent (Gowers & North, 1999; Moulds et al., 2000), both during the course of illness and following symptom remission. The studies cited above combined subtypes into a single group. To our knowledge, the study of intrafamilial perceptions in functioning across AN subtype has not been routinely conducted (Whitney & Eisler, 2005). Despite such contradictions in findings, recent suggestive evidence may provide a parsimonious explanation for these findings. As summarized in the following sections, individuals with AN, and potentially their family members, may have impairments in the processing of social information that could account for inconsistent reporting of family functioning.

The relatively extensive study of expressed emotion has provided compelling indirect evidence of intergenerational disturbance in social information processing in the family members of individuals with AN. Expressed emotion is an interpersonal construct whereby relatives react in a critical and blaming manner toward a family member with an illness, often criticizing and

blaming the individual for his or her ill state (Wearden, Tarrier, Barrowclough, Zastowny, & Rahill, 2000). This construct arose out of family studies with a member with schizophrenia, in which it was noted that the rate of relapse among individuals with schizophrenia differed sharply depending on the patient's living situation (Vaughn & Leff, 1976). Since the seminal work of Vaughn and Leff (1976), accumulating evidence highlights the relevance of this construct in relation to poor illness prognosis for the child (Wearden et al., 2000).

Given its prognostic significance, it is worth exploring the meaning of expressed emotion and its relevance to social cognition. To criticize or blame an individual for her illness bespeaks a lack of empathy or perspective taking on the part of the parent toward the child (Wearden et al., 2000). Of interest, the study of perspective taking, or theory of mind, has provided vital insights into the phenomenology of disorders such as ASD and schizophrenia (Pinkham et al., 2003), disorders in which social functioning is often profoundly impaired (Klin et al., 2002a; Pinkham et al., 2003). *Theory of mind* refers to an individual's ability to perceive and understand that other human beings have a mind, thereby giving others unique perspectives that guide their actions, beliefs, and emotional states (Bird, Castelli, Malick, Frith, & Husain, 2004). For example, impaired theory of mind is demonstrated via impairment in facial affect recognition (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001) and the appreciation of *faux pas* (Baron-Cohen, O'Riordan, Stone, Jones, & Plaisted, 1999). The importance of this construct is evidenced by the relationship of theory of mind to functional status in individuals with psychiatric illness (Hadwin, Baron-Cohen, Howlin, & Hill, 1997). Returning to expressed emotion, this behavioral pattern may be reconceptualized as a deficit in theory of mind. Indeed, individuals who convey criticism of an illness toward individuals with that mental illness—that is, expressed emotion—are possibly struggling to understand the experience of that person's illness on their behavior. Elevated rates of expressed emotion may reflect a social cognitive deficit in the parent that may be similarly present in their ill child, that is, impaired theory of mind. The negative synergy of expressed emotion and impaired theory of mind on the quality of parent–child interactions is in need of further study.

Turning to AN, in a meta-analysis of expressed emotion in psychiatric illness, Butzlaff and Hooley (1998) reported effect sizes in families with a child with an eating disorder exceeded those in families with a child with schizophrenia. Indeed, elevated levels of expressed emotion have been reported in the siblings of individuals with AN (Moulds et al., 2000), and the presence of elevated expressed emotion predicts poor treatment response in children whose family received the Maudsley Model of family therapy (Eisler et al., 1997). In a study of family therapy with adolescent AN, expressed emotion, particularly maternal criticism, was reported to explain approximately 30% of the variance in treatment response (van Furth et al., 1996). In total, if expressed emotion can be reconceptualized as a social cognitive deficit, such findings are suggestive of difficulties in social information processing that transcend the ill child to adversely impact parent–child interactions.

Marital Quality

Marriage provides another arena for the investigation of interpersonal functioning in AN. Unfortunately, there is limited information regarding the prevalence and quality of marriage in individuals with AN. However, what little is known is suggestive of difficulties in meaningful interaction. A population-based study of women with AN born in Sweden between 1968 and 1977 indicated that significantly more individuals with a history of AN lived with their parents as adults relative to the general population (Hjern, Lindberg, & Lindblad, 2006). While by no means a direct measure of marital status, this finding is certainly suggestive of less adult intimacy (Hjern et al., 2006). The presence of sexual problems, a construct that encapsulates anxiety and desire regarding sexual intimacy, was found to increase the likelihood of a poor outcome by a factor of five in a 12-year outcome study of AN (Fichter, Quadflieg, & Hedlund, 2006). A study of adolescent outcome confirmed these findings: Fears of sexual maturity and emancipation from parents were the strongest predictors of poor outcome (van der Ham et al., 1998). In a case-control study of the outcome of AN occurring approximately 12 years after illness onset, Sullivan, Bulik, Fear, and Pickering (1998) reported significantly more adult women with a history of AN were never married (45%) relative to an age- and gender-matched sample (16%).

Adult attachment patterns in AN provide additional evidence of difficulties establishing interpersonal intimacy. Ward, Ramsay, Turnbull, Benedettini, and Treasure (2000) examined attachment in adult AN. Individuals were instructed to complete the Reciprocal Attachment Questionnaire, a measure of adult attachment patterns. While distinct patterns were not discriminable by eating disorder (ED) subtype, ED patients, in general, exhibited patterns of greater self-reliance and compulsive care seeking, a pattern the authors characterized as a "push–pull" relationship dynamic (Ward et al., 2000). However, perhaps the most interesting aspect of this study was the difficulty individuals with AN had in completing this measure. Individuals were instructed to complete the measure in relation to (a) someone they were intimately or romantically involved with, (b) the person they would most likely turn to for comfort or advice, or (c) someone they would depend on. Individuals were further instructed the attachment figure should be someone outside of the immediate family. Despite these instructions, 22 patients chose a parent as the attachment figure (relative to 3 control participants), 16% of the patient sample reported no attachment figure (relative to 0% of control participants), and an additional 16% responded "unknown" to the attachment figure (relative to 0% of control participants) and left the measure blank. Given the relationship of attachment to meaningful, intimate, interpersonal interactions, findings from this study highlight significant deficits. Limited research into interpersonal processes in adult AN precludes the formation of meaningful hypothesis generation. However, the extant pattern of evidence implicates significant difficulty with interpersonal interaction, a pattern that persists throughout the lifespan.

Individuals With AN as Mothers

An area of particular concern is the limited data on motherhood in women with AN. Unfortunately, the study of women with AN as mothers is sparse (Mazzeo, Zucker, Gerke, Mitchell, & Bulik, 2005), and the majority of available evidence often combines mothers with differing ED diagnoses (S. Agras, Hammer, & McNicholas, 1999; Cooper, Whelan, Woolgar, Morrell, & Murray, 2004; Evans & le Grange, 1995). However, reports to date have

been suggestive of significant impairment, and the importance of the parenting domain warrants mention irrespective of this lack of breadth.

To place this literature in a meaningful context, a consideration of maternal responsivity is warranted. *Responsivity,* the ability of an individual to sense and respond to another's verbal and nonverbal cues (Dix, 1991), is a construct used to characterize parenting styles and, as such, may provide one index of social cognitive capacity (Pinkham et al., 2003). Indeed, maternal behaviors such as responsivity (Smith, Landry, & Swank, 2006; Wahler & Bellamy, 1997) and mental state talk (Ruffman, Slade, Devitt, & Crowe, 2006) have been reported to be predictive of the development of theory of mind in their children. Thus, a mother who models perspective taking, describes her own mental state, and references the mental state of others often has children with superior theory of mind skills (Ruffman, Perner, & Parkin, 1999). In infancy, the feeding relationship is one relevant domain for the assessment of responsivity given the demands placed on a mother to sense and respond to her child's signals of hunger and satiety. Thus, maternal responsivity within the feeding domain and in relation to mental state discourse may offer an index to gauge social cognitive capacities.

While the origin of these outcomes are unknown, children of women with AN have been documented to have impaired nutritional and growth patterns. Mazzeo et al. (2005) concluded that children whose mother was diagnosed with AN had an elevated risk for deleterious effects including malnutrition (Russell, Treasure, & Eisler, 1998) and preterm delivery (Sollid, Wisborg, Hjort, & Secher, 2004) and were more likely to be small for gestational age (Sollid et al., 2004). In a prospective study, Russell, Treasure, and Eisler (1998) studied 8 female mothers with AN. On the basis of a review of their children's growth records, 65% of the children (9/14) were reported to suffer from food deprivation as indexed by reduced weight for age at age 6 and height for age at age 8, whereas catch-up growth was dependent on the mother's engagement in treatment. van Wezel-Meijler and Wit (1989) described an early case series of 7 children with mothers with AN. All children were evidenced to exhibit delayed growth that improved when the children received medical supervision. Of importance, in each of the cases, the mother was able to follow the physician's prescriptions of a proper diet for her child once she had information. In other words, her illness did not preclude her from following a doctor's guidelines regarding her child's nutritional needs; rather, she seemed to be unable to determine what was needed on the basis of the child's signals. If evidence supports a lack of sensitivity to infant cues, one hypothesis regarding the origin of these deficits is the relative inability of the mother to sense such cues accurately rather than the intentional neglect of such cues due to illness-related factors (e.g., fear of her child's weight gain). Indeed, during infancy, the feeding relationship is often used as an exemplar of responsive parenting, as the ability of the parent to sense and respond to infant signals in this context is a pivotal foundation for the development of mutual trust and security (S. Agras et al., 1999; Park, Senior, & Stein, 2003; Whelan & Cooper, 2000). Thus, while the origin of reported deficits in responsive parenting is unknown, mother–child interaction in AN may offer an important platform to study social cognitive capacities.

Interpersonal Difficulties in the Treatment Context

Interpersonal difficulties have been reported to interfere with all stages of treatment progress in AN. An extensive literature has supported the difficulty in forming therapeutic alliances in individuals with AN (Vitousek, Watson, & Wilson, 1998). Although such difficulties have usually been ascribed to a lack of treatment motivation, in fact, researchers have yet to explore whether basic problems in social information processing interfere with such relational responding. Following treatment initiation, attachment disturbance predicts premature treatment termination (Tasca, Taylor, Ritchie, & Balfour, 2004) and negatively impacts treatment seeking (Goodwin & Fitzgibbon, 2002). During the illness, the challenging therapeutic milieu of specialized inpatient eating disorder programs has been described and contributes high rates of "burnout" among healthcare professionals who work in this treatment context (Newell, 2004). Given the inherently personal nature of psychotherapy, addressing social proficiency within the intervention itself may be crucial to ensure intervention success.

Summary: Implications of Social Impairment in AN

This cross-sectional glimpse of social disturbance may facilitate hypothesis generation into the pathophysiology of AN. Despite the extent of these deficits, we know little about the manner in which individuals with AN perceive their social environment and subsequently use these observations to guide their interactions, a construct known as *social cognition* (e.g., Flavell, 2004; Malle, 2003; Perner, 1991; Searle, 1983). Of importance, research on interpersonal patterns as distinguished by eating disorder subtype is sparse. Given advances in characterizing distinct characterological, behavioral, and genetic profiles between the purging and restrictive subtypes of AN, the study of interpersonal patterns across these diagnostic groupings may add an additional layer of precision to genetic and neurobiological research. Further, this focus of inquiry can facilitate greater understanding of the phenomenology of AN and spur a second generation of treatment development that may serve to potentiate existing interventions (Robin et al., 1999). To illustrate, as parents are increasingly being appreciated as crucial to the successful management of eating disorders (Lock, Le Grange, Agras, & Dare, 2001), deficits in social perception on either side of the relationship can impact the efficacy of treatment approaches. However, despite these pervasive discrepancies and the potential impact of impaired processing, our lack of knowledge in this area is pervasive. It is perhaps part of the functional significance and compelling nature of symptoms of malnutrition that, despite extensive interpersonal impairment that negatively impacts both treatment outcome and quality of life, research has largely focused on the symptoms of AN, which has led to limited investigation into processes that underlie social cognitive deficits, irrespective of their potential prognostic significance.

Social Cognition: The Roadmap Provided by ASDs

Systematic exploration of the relation between AN and ASD may facilitate novel hypothesis generation. Observed overlap may help place these disorders within a broad nosology associating disorders on the basis of temperament and neurocognitive communalities that may help transcend boundaries placed by diagnos-

tic classification systems and may help propel meaningful investigations into overlapping and distinct pathophysiological processes. Further, relative to that in AN, social information processing in ASD has been characterized extensively. The methodologies used to inform social processes in ASD can provide a framework for a similar system of inquiry in AN.

Social Endophenotypes: Elucidating Behavioral and Trait Similarities in AN and ASD

ASDs are characterized by dysfunction in three pivotal domains: (a) restricted, repetitive behaviors; (b) impairment in verbal and nonverbal communicative abilities; and (c) deficits in social reciprocity (American Psychiatric Association, 1994). Despite the pervasive impact of these domains on widespread areas of functioning, the deficits in social interaction, in particular, are crucial in understanding the phenomenology of these disorders (Klin et al., 2002a). To elaborate, the social phenotype of autism can be construed on two levels: intrinsic and observable. Intrinsic manifestations include the internal neurocognitive mechanisms that subserve social information processing. Observable features include the manifest interpersonal styles, behavioral patterns, and personality features that characterize affected individuals and, often in more subtle form, family members of affected probands (M. Murphy et al., 2000; Piven, 2001). As will be demonstrated, consideration of the observable phenotypic expression of both ASD and AN provides provocative evidence regarding potential overlap in behavioral deficits and trait features. In addition, comparisons of intrinsic cognitive processes highlight areas of overlap but, more importantly, also highlight novel areas for exploration in AN.

Figure 1 presents the roadmap for our review. First, although numerous diagnostic categories have been established to differentiate elements of the autism spectrum, for simplicity, we will consistently use the term *ASD* to distinguish the high-functioning end of this spectrum. Specifically, we refer to individuals diagnosed with high-functioning autism or Asperger's syndrome in accordance with criteria of *DSM–IV* (American Psychiatric Association, 1994). The choice of high-functioning individuals was intended to facilitate comparisons across these diagnoses. Intellectual functioning complicates the clinical picture in ASD, while research conducted in individuals with AN has been almost exclusively conducted on individuals of average to high intellectual functioning (see Gravestock, 2003, for a review of intellectual impairment in eating disorders). The term *autism* will be used whenever these criteria are not met.

Overview of the Phenotypic Expression of ASD

Characterization of the phenotypic expression of ASD can help to advance hypotheses into underlying social neurocognitive deficits and facilitate comparisons with AN (Baron-Cohen, Jolliffe, Mortimore, & Robertson, 1997; Baron-Cohen, O'Riordan, et al., 1999; Baron-Cohen & Wheelwright, 2004; Craig & Baron-Cohen, 2000; Klin, Pauls, Schultz, & Volkmar, 2005; Lawson, Baron-Cohen, & Wheelwright, 2004; Stein et al., 2004; Travis, Sigman, & Ruskin, 2001; Wentz et al., 2005). Research by Lawson et al. (2004) characterized individuals with ASD as logical systemizers with impaired empathetic understanding. Constricted in their range of expression (Kanner, 1943), lacking in imagination (Craig & Baron-Cohen, 2000), and struggling with the nuances of social interaction (Baron-Cohen, O'Riordan, et al., 1999), these individuals frequently withdraw from such interactions (Baron-Cohen & Wheelwright, 2003; Prior et al., 1998). Rather, individuals with ASD seek order, predictability, and rule-based systems, perhaps even in the context of interpersonal relationships (Baron-Cohen, Richler, Bisarya, Gurunathan, & Wheelwright, 2003; Hill, Sally, & Frith, 2004; Sally & Hill, 2006). In fact, as highlighted in a later section, theories of social information processing in ASD may offer insights regarding how individuals with AN approach social interaction (Dapretto et al., 2006; Hobson, Chidambi, Lee, & Meyer, 2006; Kanner, 1943; Klin, Jones, Schultz, & Volkmar, 2003; Klin et al., 2002a; Lawson et al., 2004).

While the tendency to find order and create systems can be highly adaptive in certain contexts, in the social realm, this tendency does not promote adaptive functioning. For example, on the Autism Spectrum Quotient, a self-report measure of subtle autism symptomatology, the score of mathematics students exceeded those of students from social science disciplines, while the scores of 6% of mathematic Olympiads exceeded the clinical cutoff. Notably, on interview, 100% (11/11) of these math champions met three of the diagnostic criteria for an ASD (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). Despite this adaptive use of rule-governed information processing in the field of mathematics, the complexity of human social interaction belies the creation of a coherent system. Given the nuances, subtleties of expression, and incongruence between actions and words that may present in social interaction (e.g., *faux pas*, humor; Baron-Cohen, 1997; Baron-Cohen, O'Riordan, et al., 1999), such situations may be experienced as both overwhelming and confusing by individuals with ASD. The drive to systematize, while exceedingly adaptive in certain contexts, may be insufficient for intimate social interaction (Baron-Cohen, 2004a). We shall return to this strategy of information processing when we present a social model of AN, as the concept of systematizing social interaction is a pivotal aspect of this model.

Studies of personality features may help further characterize the phenotype of ASD. For example, Ozonoff, Garcia, Clark, and Lainhart (2005) reported individuals with ASD exhibited elevations on content subscales of the Minnesota Multiphasic Personality Inventory–2 measuring social discomfort and on clinical scales measuring social introversion, a finding replicated on the Introversion scale of the Personality Pathology Five scale. On Cloninger's Temperament and Character Inventory, individuals with ASD demonstrated high levels of harm avoidance, low levels of self-directedness, and low levels of cooperativeness (Soderstrom, Rastam, & Gillberg, 2002). More recently, a study of 112 individuals with ASD replicated this profile but demonstrated low levels of novelty seeking and reward dependence as well as the aforementioned traits (Anckarsater et al., 2006). These character traits are further substantiated by high rates of social anxiety in individuals with ASD (Melfsen et al., 2006). These results paint a picture of rigid, socially introverted individuals who strive for order and sameness and have difficulty apprehending interpersonal interactions. However, there is great diversity in the manner in which individuals with ASD interact. If patterns of social interaction are to help advance study into the pathophysiology of ASD, precise delineation and measurement of specific features is essen-

tial. We devote the followings sections to the delineation of specific, measurable features.

Overview of the Phenotypic Expression of AN

Striking parallels emerge when this clinical picture is compared with what is known of the phenotypic expression of AN. With the intention of exploring transdiagnostic similarities, we first consider personality functioning in AN. Relative to the paucity of information on social information processing in AN, the study of personality features in AN is extensive and dates back to early writings on this disorder (e.g., Gull, 1868).

The frequent convergence in reports of personality features in the clinical presentation of the restricting subtype of AN has prompted proposals that personality functioning be included as part of diagnostic criteria for this disorder (Westen & Harnden-Fischer, 2001). Indeed, reviewing the personality profile of the restricting subtype of AN, Wonderlich, Lilenfeld, Riso, Engel, & Mitchell (2005) stated "the personality of an individual with the restricting subtype of AN is well known to any clinician in the field. Someone with this diagnosis is frequently constrained, conforming, obsessional, rigid, and perfectionistic" (p. S69). To demonstrate, we present a vignette of an individual with AN (Figure 2) drawn from an historical account from 1866 (cited in Brumberg, 1988) to highlight features extensively documented in AN both premorbidly and following weight restoration: cognitive and behavioral rigidity (Anderluh et al., 2003; Tchanturia, Morris, et al., 2004), perfectionism (Bastiani, Rao, Weltzin, & Kaye, 1995; Bulik et al., 2003; Halmi et al., 2000; Woodside et al., 2002), social withdrawal (Diaz-Marsa, Carrasco, & Saiz, 2000; I. C. Gillberg et al., 1995; Godart et al., 2004; Holliday, Uher, Landau, Collier, & Treasure, 2006; Karwautz, Troop, Rabe-Hesketh, Collier, & Treasure, 2003; Kaye et al., 2004), constriction (Geller, Cockell, Hewitt, Goldner, & Flett, 2000), and harm avoidance (Diaz-Marsa et al., 2000; Klump et al., 2000, 2004). In fact, the reliable presentation of this behavioral and personality cluster has been demonstrated to add to the incremental validity of diagnostic accuracy (Westen & Harnden-Fischer, 2001). Worthy of note is a Q-sort study by Westen and Harnden-Fischer (2001) that classified eating disorder diagnoses on the basis of personality type. Notably, a constricted/overcontrolled type emerged characterized by behavioral inhibition, limited psychological insight, constricted emotional expression, shyness in social situations, anxiousness, and distortions in body image. This convergence, however, contrasts sharply with rates of categorically based personality disorder diagnoses in the group for which estimates range from 21% to 97% (Skodol et al., 1993; Vitousek & Manke, 1994). Despite this inconsistency, there is consensus that the anxious/fearful personality cluster predominates in AN (Wonderlich et al., 2005).

Case of Molly Fancher, 16 Clinical Report of AN, 1866, Brooklyn Daily Eagle

"Her books were her delight…she neglected all for them, and would arise late in the morning in the consequence of weakness, hasten away to school, fearful of being tardy, and in the evening, in her anxiety to learn her lessons, again neglect a meal for which she felt no inclination."

Figure 2. A late-19th century description of a young woman with anorexia nervosa (AN) from Brumberg, 1988 (p. 79).

Clues to Social Cognitive Endophenotypes for Investigation

Interpersonal patterns and personality traits in ASD offer potential clues to defining a social endophenotype that may transverse diagnostic boundaries. We focus on two personality traits and their defined behavioral manifestations: aloofness with accompanying social withdrawal and insistence on sameness with accompanying perceptual and behavioral rigidity. Several methodological challenges emerge with these comparisons. The measurement of personality features, specifically, and personality disorders, more generally, is plagued by changes in diagnostic classification systems, the use of different assessment tools, and inconsistencies between subjective and informant reports (McConaughy, Stanger, & Achenbach, 1992). Further, as highlighted by Lilenfeld et al. (2006), the relationship of personality features to psychiatric illness is often difficult to discern from the literature because of vaguely articulated causal models. These challenges become further complicated when comparing across diagnoses, as constructs familiar within one diagnostic class may utilize different semantics for similar features. Our goal is to define these features clearly such that researchers in AN and ASD can consider novel perspectives from which to conceptualize familiar concepts.

Aloof/Social Withdrawal in ASD

Vast heterogeneity exists in the presentation of socioemotional deficits across the autism spectrum (Volkmar, Lord, Bailey, Schultz, & Klin, 2004). To illustrate, Wing (1997) characterized subtypes of ASD on the basis of styles of social interactions. These groupings reflect great variation, as evidenced by characterization of aloof, low-functioning individuals notable for total disengagement from social interaction and failure to engage in interpersonal reciprocity to a loner subtype, which is characterized by individuals with high or superior functioning who may navigate the social realm by gravitating toward occupations that condone an isolated style and by mastering social nuances by rote. An active but odd cluster is characterized by individuals with ASD who actively seek social interaction but do so in an inappropriate and one-sided fashion (Borden & Ollendick, 1994; Waterhouse et al., 1996). Finally, a passive subgroup comprises individuals who fail to initiate social interaction but who accept the advances of others and potentially enjoy such advances (Prior et al., 1998). The vast diverse manifestations of these deficits have prompted work on more precise delineation and measurement of interpersonal styles (Baron-Cohen & Wheelwright, 2003; Hartman, Luteijn, Serra, & Minderaa, 2006; Verte et al., 2006).

Subtle expressions of interpersonal patterns described in ASD have been documented at elevated rates in family members and are

believed to reflect genetic liability to autism (Piven, 2001). For example, mild manifestations of the aloof and withdrawn subtype characterized by Wing and colleagues (Waterhouse et al., 1996) have been described in parents of diseased probands (M. Murphy et al., 2000). Piven, Palmer, Jacobi, Childress, and Arndt (1997) described elevated rates of rigidity, withdrawal, anxiety, and aloofness among parents with multiple children with autism. More recently, work by M. Murphy et al. (2000) characterized a withdrawn personality cluster obtained by factor analysis and encompassing features such as undemonstrative, shy, aloof, unresponsive, and self-conscious that was found to best discriminate families with an autistic child. Importantly, personality features were defined behaviorally without reference to underlying motivations and were assessed by structured interview (the modified Personality Assessment Schedule, Tyrer, 1988). As defined, the aloof feature is denoted by choice of lifestyle (e.g., choosing a solitary job), avoidance of interpersonal engagements (e.g., failure to attend parties related to employment), and a dearth of intimate friendships (e.g., lacking close confidants). This body of work supports the value of studying subtle expressions of interpersonal styles in family members, a strategy that may help specify neurocognitive and genetic contributions to more pathological variants of the behaviors of interest.

Indeed, clearly delineated personality features can highlight domains of neurocognitive dysfunction. As a case in point, the aloof feature defined by M. Murphy et al. (2000) discriminated parents of children with ASD who exhibited disrupted social cognitive capacities from parents of children with ASD without this degree of dysfunction (Losh & Piven, 2007). Such features may offer clues into underlying cognitive dysfunction and, when compared with those in individuals with AN, may facilitate hypotheses regarding unique and common developmental pathways of these disorders, ultimately leading to the specification of social cognitive endophenotypes.

Aloof/Social Withdrawal in AN

Social withdrawal in AN presents an interesting paradox. A logical supposition that has long been held in the field is that individuals with AN strive for social acceptance, a facet of their vulnerability, and that social isolation occurs with the onset of AN and abates with illness remission (Keys, 1950). Two patterns of evidence suggest this latter formulation does not fit the phenotype of AN: trait features and chosen venues for social engagement. First, studies of personality features in AN have noted elevated levels of interpersonal constriction and social withdrawal that persist with recovery (C. Gillberg & Rastam, 1992; Nilsson et al., 1999). For example, Holliday et al. (2006) studied the chronicity of personality features in both currently ill and recovered individuals with AN. Individuals with AN in the ill state demonstrated extreme social isolation relative to healthy control participants, with effect sizes greater than 1.5. While such social introversion improved with recovery and was significantly different from the ill state, scores of recovered individuals with AN remained significantly lower than those of nondiseased control participants, continuing to demonstrate large effect sizes (Holliday et al., 2006). No differences were reported between AN subtypes. Elevated rates of social discomfort provide further substantiation. Premorbidly, fears of negative evaluation have been reported to increase the risk of restrictive eating patterns (Gilbert & Meyer, 2005). Further, not only do symptoms of social anxiety persist beyond conservative definitions of illness recovery (Holtkamp, Muller, Heussen, Remschmidt, & Herpertz-Dahlmann, 2005), but also these persisting symptoms predict ongoing social impairment (Godart et al., 2004). Godart et al. (2002) found social disability was frequent in AN with symptoms of social anxiety increasing social dysfunction (Godart et al., 2004). Kaye et al. (2004) reported that 20% of 293 individuals with AN had a comorbid diagnosis of social anxiety that predated eating disorder symptom onset. Importantly, social anxiety in individuals with AN indicates that fears in social settings are independent of concerns about body weight or eating and relate more to a fear of negative evaluation. Thus, there is growing evidence that social fears and related social withdrawal are frequent, pernicious symptoms in AN. However, unlike ASD, the desire for social acceptance is more discernable.

The second line of evidence is more speculative. We propose that individuals with AN seek out organized activities as a venue for social engagement because the systematic structure of these activities offers a more predictable and rule-governed domain for social interaction than does nonstructured play, a forum necessary for more intimate interpersonal engagement (Schuler, 2003). To clarify, while there may not be an overt difference in rates of participation in extracurricular activities, there may be distinctions in the intensity or manner of participation (i.e., frenetic engagement in exercise, seeking out leadership rather than cooperative roles in groups, etc.). There has been extensive study related to rates of eating disturbance in athletes (e.g., Ringham et al., 2006) and the tendency to seek out athletics related to aesthetic concerns (e.g., Sundgot-Borgen & Torstveit, 2004). Of interest, an incidental note is that athletics related to aesthetic concerns (e.g., ballet, ice skating, gymnastics) are also solitary activities, an observation that may be incidental. However, a further line of research is needed to explore the quality of team membership and cooperation in individuals with eating disturbance. To our knowledge, research examining excessive participation in organized activities in AN has also not been conducted. If true, given that time after school is limited, excessive engagement in organized activities necessarily means there is less time to foster intimate friendships in unstructured venues. As highlighted by the model of AN we propose, research into social functioning in AN will need to examine more nuanced definitions of social competence to capture the nature of deficits.

Interim Summary: Aloofness and Social Withdrawal

Topographically similar behaviors may have different underlying motivations. We have deliberately focused on overt behaviors to minimize the methodological challenges of comparing personality features across diagnoses (Wonderlich, Joiner, Keel, Williamson, & Crosby, 2007). However, while combining what is known about social withdrawal in ASD and AN highlights intriguing areas for exploration, such comparisons need to take into account several developmental, psychological, and neurocognitive factors that may lead to alternative instigating and maintaining factors for social isolation. Further complicating researchers' understanding of the phenomenology of social withdrawal is that one's stated motivations for withdrawal may differ from the actual drives or, similarly, that an individual may have limited insight into the true

motivations for such withdrawal. Understanding contextual factors that propagate and perpetuate social withdrawal may aid in the specification of subtypes based on interpersonal style that may further our understanding of the nature of these disorders.

Developmental Considerations

Individuals with ASD or AN may change in interpersonal capacities over time, resulting in differing motivations for isolation across the developmental trajectory. For example, Wing (1997) noted that high-functioning individuals with ASD are often not detected until high school when difficulties interacting with peers and instructors becomes more evident, particularly those in the passive subtype. Individuals with AN have a typical age of onset in adolescence. Thus, although early social discomfort has been documented, it is conceivable that supportive strategies developed in youth to compensate for potential deficits no longer function during the complexity of adolescence or, alternatively, that disrupted social cognitive capacities come online in adolescence, which may be among the many genetically influenced changes that occur during this developmental period (e.g., Klump et al., 2006), factors potentially compounded by the challenging unstructured social demands of this stage (Bruch, 1973). Thus, the study of social withdrawal needs to take a contextual approach, exploring not only social cognitive deficits but also social and developmental factors that influence this pattern.

Psychological Considerations

Certain individuals with ASD may desire more social affiliation than may be evident from patterns of avoidance. First, social support appears to be essential to improve the quality of life in individuals with ASD (Renty & Roeyers, 2006). In a study of 58 individuals with high-functioning ASD, perceived social support accounted for significant variance in quality of life measures after taking into account demographic and disability-related information. Indeed, ratings of quality of life and patterns of comorbidity suggest dissatisfaction with one's well-being. A recent review documents elevated rates of depressive symptoms in individuals with ASD, although the developmental trajectory of these symptoms is unclear (Stewart, Barnard, Pearson, Hasan, & O'Brien, 2006). Other studies have noted the relation of depressive symptoms in ASD to processes of social comparison, suggesting a possible unmet desire for greater social affiliation (Hedley & Young, 2006). Finally, rates of social anxiety are elevated both in individuals with ASD and their family members (Melfsen et al., 2006; Piven & Palmer, 1999). These findings are worthy of consideration and present some challenging observations to reconcile. An individual who withdraws from social intimacy may do so for a variety of reasons: lack of interest, fear of negative evaluation, feeling overwhelmed by sensory stimuli, and low desire for novelty, to name several well-documented patterns (Dalton et al., 2005). Taking a broad perspective on the pattern of and motivation for social withdrawal in ASD can deepen our understanding of further sources of phenotypic overlap in ASD and AN. Thus, in interpreting features indicative of social withdrawal, researchers need to be cautious in drawing conclusions about the motivation underlying this withdrawal. The overlap in strategies and motivations across these entities is unstudied but may provide insight into the phenomenology of both disorders.

Rigidity in ASD

While repetitive and restrictive behaviors are defining criteria of ASD, symptom heterogeneity exists, complicating study both within and across individuals (South, Ozonoff, & McMahon, 2005). Repetitive behaviors in ASD are delineated in the *DSM–IV* (American Psychiatric Association, 1994) by such diverse features as stereotyped motor mannerisms; preoccupation with objects or parts of objects; patterns of interest that are unusual in their narrowness or intensity of pursuit; and/or extreme rigidity or insistence on sameness. To achieve better parsimony, researchers have conducted factor analyses of behavior profiles (e.g., Georgiades et al., 2007), examined developmental features (e.g., Bodfish, Symons, Parker, & Lewis, 2000), hypothesized common neurobiological mechanisms (e.g., Lewis & Bodfish, 1998), and studied accompanying clinical features (Gabriels, Cuccaro, Hill, Ivers, & Goldson, 2005), among other strategies. Such distinctions are valuable in that they can help to focus neurobiological and genetic research (Silverman et al., 2002). We examine these typologies for overlap with AN and discuss the relationship of ASD and AN with the disorders most robustly associated with AN: obsessive-compulsive personality disorder (OCPD) and obsessive-compulsive disorder (OCD), which are also notable for rigidity.

Why are we including a discussion of behavioral rigidity in an analysis of social cognitive processes? First, because irrespective of the role of social cognitive disturbances in the etiology or exacerbation of behavioral rigidity, rigidity in behavior is interpersonally oriented in that such patterns are often experienced as the most intrusive feature by carers of individuals with AN or ASD (Williams, Dalrymple, & Neal, 2000). Investigating the role of social cognitive deficits in propagating such rigidity is needed. Second, behavioral and cognitive rigidity are part of the core experience of AN, while OCPD and OCD, disorders defined by rigidity, are the most frequently reported comorbidities in AN (Halmi et al., 2005). Thus, a comprehensive discussion of rigidity across ASD and AN would ideally accommodate the relationship to OCD and OCPD to establish a framework by which all three diagnostic classes can be considered. Third, individuals with OCPD and OCD likewise have poor interpersonal outcomes (C. Gillberg & Rastam, 1992). Thus, rigidity in behavior does impede interpersonal intimacy, although, importantly, the contribution of social cognitive deficits to this inflexibility is unclear. Study of behavioral and cognitive rigidity in conjunction with study of social cognitive deficits may facilitate the delineation of patterns of interpersonal deficit in AN. Finally, semantics inhibit progress in psychiatric research. Equating constructs in the realm of behavioral rigidity is a needed exercise to facilitate more intense study. Understanding the unique and overlapping manifestations of rigidity across these diagnostic classes may provide an organizational structure that propels investigations into disorder pathogenesis, helps delineate patterns of interpersonal interaction, and targets genetic research.

Insistence on Sameness in ASD

Cross-diagnostic comparisons of the feature *insistence on sameness* is a logical starting point given reports of this feature in

parents of individuals with ASD and the association of this feature with the high-functioning end of the ASD spectrum (Abramson et al., 2005). As described in the *DSM–IV* (American Psychiatric Association, 1994), insistence on sameness is defined as the "apparent inflexible adherence to specific, nonfunctional routines or rituals" (p. 71). This behavioral rigidity has been described across multiple areas of functioning including daily routines (Carrington & Graham, 2001), food type and texture (Ahearn, Castine, Nault, & Green, 2001; Schreck, Williams, & Smith, 2004; Williams et al., 2000), and environmental features (Greaves, Prince, Evans, & Charman, 2006). In parents, more subtle expressions of this feature have been described (Piven et al., 1997). For example, as operationalized in the Personality Assessment Schedule (a key instrument used to define such features in family studies of autism, e.g., M. Murphy et al., 2000; Piven et al., 1997), a rigid individual may go to inconvenient extremes to find a particular brand of food or may avoid employment that would interfere with the individual's schedule. Importantly, the determination of severity on the Personality Assessment Schedule is predicated on the developmental and contextual inappropriateness of the behavior. Thus, as defined, individuals with ASD exhibit an aversion to novelty across domains of functioning that significantly impacts relationships, employment, and well-being.

Rigidity in AN

While evidence of behavioral and cognitive rigidity has been reported to both predate and persist in AN (Anderluh et al., 2003; Kaye et al., 2004; Lilenfeld et al., 1998), the construct of rigidity has been an increasingly broad and underspecified term. Thus, we consider rigidity as defined by Kanner's original description of the feature in ASD, that is, an inability to experience something as complete if any of its detailed constituents was lacking or altered (Kanner, 1943). To capture the nuances of behavioral and cognitive rigidity seen in individuals with OCD or OCPD, comparisons with specific features within these diagnoses will more usefully propel cross-diagnostic comparisons than will consideration of full clinical syndromes. From this perspective, there are several likely arenas of comparison in AN and OCD, notably the endorsement of themes of order/symmetry within the OCD diagnosis and the personality feature of low novelty seeking. Thus, the following two sections summarize these areas (themes of order/symmetry and low novelty seeking) as evidence of behavioral rigidity in AN. The robust links to OCPD are more difficult to accommodate as the majority of studies highlight diagnosis comorbidity rather than deconstructing specific features of the OCPD diagnosis. Thus, we merely highlight the robust concordance.

Order and Symmetry

Themes of order and symmetry in AN exhibit chronicity, as such patterns have been endorsed in the recovered state in several studies (Halmi et al., 2003; Srinivasagam et al., 1995). This obsessional content seems to be preferred in individuals with AN relative to other forms of symptom expression in OCD (Halmi et al., 2003; Matsunaga, Kiriike, et al., 1999; Srinivasagam et al., 1995). In comparisons of individuals with AN and comorbid OCD relative to individuals with OCD only, the AN–OCD group endorsed more frequent themes of symmetry and orderliness along with higher prevalence of OCPD relative to the OCD-only group (Matsunaga, Kiriike, et al., 1999). However, a separate study reported concordance in rates of symmetry concerns relative to individuals with OCD (Halmi et al., 2003). Further, obsessive themes demonstrate evidence of diagnostic specificity: Individuals with bulimia nervosa were more likely to demonstrate aggressive themes, while women with AN endorsed more frequent themes of symmetry and exactness (Matsunaga, Miyata, et al., 1999). While the data are limited, these suggestive findings highlight the need for semantic equivalence of the insistence on sameness construct and concern with symmetry and order across diagnoses.

Novelty Seeking

Novelty seeking is defined as the active pursuit of new experiences with unknown outcome (Cloninger, Przybeck, Svrakic, & Wetzel, 1994). Thus, low levels of this construct appear closely related, although different from, insistence on sameness. To elaborate, an individual who insists on sameness theoretically may be willing to try a novel experience for which he has no previous associate. However, once experienced, it may be that the experience must always replicate the first experience. Such distinctions may prove important at the level of neurobiology. Lacking this evidence, we assume semantic equivalence. Individuals with AN have endorsed low novelty seeking in several investigations (Karwautz, Troop, et al., 2003; Klump et al., 2000), although Klump et al. (2004) reported improvement with remission. Several reports indicate that individuals with the purging subtype of AN exhibit higher rates of novelty seeking relative to those with the restricting subtype (Reba et al., 2005). Other studies have confirmed low levels of novelty seeking that were independent of body mass index but did not differ by AN subtype (Klump et al., 2000). Karwautz, Troop, Rabe-Hesketh, Collier, and Treasure (2003) examined the relationship of personality features and their relationship to personality diagnoses in individuals with AN. A cluster analysis identified a group of patients characterized by elevated levels of harm avoidance, low novelty seeking, low self-directedness, and low cooperativeness, a subgroup of features associated with the anxious, avoidant, and dependent personality disorders (Karwautz, Nobis, et al., 2003). At best, the study of novelty seeking highlights the need for studies that examine the equivalence of trait features such as harm avoidance, novelty seeking, and behavioral rigidity for cross-diagnostic research to advance.

Obsessive-Compulsive Personality Disorder (OCPD)

By definition, the expression of OCPD appears synonymous with behavioral rigidity. The limitations of considering the combined features of OCPD notwithstanding, the body of evidence associating OCPD with AN is significant and has received extensive attention in the research literature (e.g., see Halmi et al., 2005; Lilenfeld et al., 2006; Tchanturia, Morris, et al., 2004). Thus, a comprehensive presentation of this literature is beyond the scope of this work, and we merely highlight findings germane to the current discussion. OCPD is a maladaptive personality pattern characterized by excessive rigidity, need for order/control, preoccupation with details, and excessive perfectionism (American Psychiatric Association, 1994). Of note, in AN, symptoms of OCPD

are long standing, appearing in childhood (Anderluh et al., 2003), persisting with recovery (Tchanturia, Morris, et al., 2004), and, in some reports, are associated with poor treatment outcome (Nilsson et al., 1999). For example, Anderluh, Tchanturia, Rabe-Hesketh, and Treasure (2003) conducted a retrospective interview assessing childhood traits of obsessionality. They reported elevated rates of childhood rigidity and perfectionism in individuals with a history of AN with each additional trait increasing the odds of developing an eating disorder by a factor of 6.9. Importantly, rigidity was defined as difficulty in adjusting to change (e.g., household moves, changes in family schedule or structure, changes in planned family activities), that is, in a manner very similar to the insistence on sameness feature in ASD.

Parents of individuals with AN evidence elevated levels of rigidity and associated perfectionism. In a family-based study of AN, parents of anorexic probands evidenced a lifetime rate of OCPD of 19% relative to 6% of parents of healthy control participants, while the probands themselves evidenced a rate of 46 per 100 relative to 5 per 100 in control participants (Lilenfeld et al., 1998). Indeed, the high prevalence rates of maladaptive and pervasive personality features supports the view of Westen and Harnden-Fischer (2001), among others (Wonderlich et al., 2005), that, in fact, such features may form part of the core phenomenology of AN. If so, the overlap of such traits across diagnostic entities such as ASD and the inclusion of patterns of social functioning, as addressed in this review, can enrich the phenotypic characterizations of these disorders.

Information Processing in AN, ASD, and OCPD

Patterns of rigidity may be seen in strategies of information processing, overlap that may help focus research into the neurocognitive commonalities of AN, ASD, and OCPD. To facilitate hypothesis generation regarding relationships of AN, ASD, and OCPD, we provide a cursory overview of styles of information processing. We only briefly summarize this literature so as not to get too far afield from our focus on social cognition; however, this review highlights areas needed for future research and will facilitate novel questions regarding developmental trajectories. This overview further emphasizes a challenge in conducting neuropsychological investigations in high-functioning individuals. Rather than focus on the outcome of neuropsychological assessments, there is a need to examine the processes by which high-functioning individuals arrive at test results. These cognitive strategies may direct attention to compensatory mechanisms individuals have adopted to adapt to neurological deficits (Klin et al., 2002a). With this consideration, we present findings indicating overlap on tasks of information processing that document a bias for local versus global configurations and on executive tasks examining cognitive flexibility.

Preference for Detail Over Global Configurations

Styles of information processing may contribute to or exacerbate the insistence on sameness in ASD. As reviewed by Happe and Frith (2006), Kanner's original conceptualization of insistence on sameness linked it to a feature referred to as *central coherence,* a robust style of information processing in ASD in which superior focused processing on detail at the expense of global configurations has been repeatedly documented (we highlight this feature in later sections on neurocognitive functioning). This focus on detail with failure to integrate context, that is, failure to understand the gist of a situation, leads to a sense of incompleteness: A situation is not complete until all details are present (Kanner, 1943). Deficits in coherence highlight a key aspect of the experience of an individual with ASD. By missing the overarching meaning of a situation, a focus on detail becomes the way in which an individual with ASD organizes their experience and thus may underlie the drive to systemize described in this population (Lawson et al., 2004). Importantly, such behavioral and cognitive rigidity is not merely a preference; individuals with ASD often become distressed when such routines or rule-based systems are disrupted (Greaves et al., 2006).

Unfortunately, while preoccupation with detail is part of the diagnostic criteria of OCPD, the study of central coherence in both AN and OCPD is limited but suggestive. Several studies have documented deficits in various forms of memory and learning in AN (Fowler et al., 2006; A. D. Lawrence et al., 2003; R. Murphy, Nutzinger, Paul, & Leplow, 2004; Tchanturia, Morris, Surguladze, & Treasure, 2002). Examination of the pattern of results indicates that findings can be interpreted as a bias for detailed processing at the expense of more efficient organizational strategies. For example, A. D. Lawrence et al. (2003) investigated visual associative learning in AN. Of note, individuals with AN exhibited deficits in a dynamic categorization task; however, they exhibited spared performance on a pattern recognition task (A. D. Lawrence et al., 2003). Such results are supportive of a bias for a processing of details (i.e., patterns) at the expense of associating figures based on global categorizations. Deficits in both strategic (Sherman et al., 2006) and autobiographical memory have been noted (Nandrino, Doba, Lesne, Christophe, & Pezard, 2006). Importantly, memory deficits may be a result of inefficient strategies to encode information, incurring difficulty on subsequent ability to recall (Happe & Frith, 2006). For example, a study conducted with the Rey–Osterrieth Complex Figure documented impairments in the ability to recall a complex figure in individuals with AN (Sherman et al., 2006). The Rey figure is intricate, containing both global organizing configurations as well as minute details within each configuration. Thus, the order and manner in which an individual copies the figure provide insight into the strategies used for organization (Bennett-Levy, 1984). Consistent with predictions on preferences for detail at the expense of global processing, that is, poor coherence, individuals with AN were reported to display less use of global configurations than were control participants, a strategy that negatively impacted both immediate and delayed recall (Sherman et al., 2006).

Set-Shifting Deficits

Cognitive rigidity, the inability to shift cognitive strategy to changing contingencies, may be related to an insistence on sameness. Indeed, this feature has been documented in AN, OCD, OCPD, and ASD. In AN, compelling findings from the work of Tchanturia, Treasure, and colleagues (Holliday, Tchanturia, Landau, Collier, & Treasure, 2005; Tchanturia, Anderluh, et al., 2004; Tchanturia, Morris, et al., 2004) revealed difficulty in the shifting of cognitive sets across several domains, a deficit that persists with symptom recovery (Holliday et al., 2005; Tchanturia,

Morris, et al., 2004). For example, set-shifting deficits were reported in individuals with AN on neuropsychological tasks that required them to integrate verbal context to help in choosing a word form in an alternating sequence. This deficit persisted with weight restoration (Tchanturia, Morris, et al., 2004). Similarly, deficits in haptic set shifting, a measure of tactile sensitivity, have been shown as well as impaired cognitive shifting on the Wisconsin Card Sorting Test, a finding replicated in a separate laboratory (Steinglass, Walsh, & Stern, 2006). In combination, these studies document patterns of cognitive and sensory rigidity. In fact, the report of similar patterns in unaffected siblings of individuals with AN has led Holliday et al. (2005) to propose cognitive set shifting as an endophenotype of AN. Also of note is a study examining the relationship of set-shifting difficulties in individuals with AN with accompanying features of OCPD (Tchanturia, Morris, et al., 2004). Thus, as demonstrated via cognitive, experiential, and behavioral evidence, individuals with AN exhibit rigidity, a feature that may also be present in unaffected family members.

Reports of cognitive rigidity have been documented in individuals with OCD (e.g., Schmidtke, Schorb, Winkelmann, & Hohagen, 1998; Veale, Sahakian, Owen, & Marks, 1996; Watkins et al., 2005). Of interest, N. S. Lawrence et al. (2006) reported that deficits in set shifting were distinctly related to concerns about symmetry and exactness in an adult OCD sample. Set-shifting deficits were reported to discriminate individuals with OCD from those with Tourette's syndrome (also in the OCD spectrum). Radomsky and Rachman (2004) also reported that individuals with OCD and concerns regarding symmetry have difficulty concentrating on a cognitive task when their surroundings are in disarray, another example of difficulty shifting cognitive sets.

Executive deficits in cognitive flexibility (e.g., set shifting) are among the most frequently reported deficits in executive functioning in ASD, and such difficulties appear correlated with autism symptomatology within the ritualistic/repetitive behavior domain (Hill & Bird, 2006; Lopez, Lincoln, Ozonoff, & Lai, 2005). However, findings in the high-functioning end of the spectrum have been less consistent, with some authors reporting superior functioning on executive functioning tasks requiring an extradimensional conceptual shift (Landa & Goldberg, 2005), with a trend for individuals with ASD to have greater difficulty on intradimensional shifts, that is, a perceptual task. Studies of family members have also been conflicting with set-shifting deficits reported in fathers of ASD probands (Wong, Maybery, Bishop, Maley, & Hallmayer, 2006) but not in siblings (Wong et al., 2006). Diverse results on set-shifting tasks highlight the importance of delineating the nature of the set-shifting task as well as the neuroanatomic processes employed to achieve task results. For example, Schmitz et al. (2006) reported that despite equivalent performance in individuals with ASD relative to that in healthy control participants, the brain regions activated differed across groups. Thus, in working with high-functioning individuals, an emphasis on the cognitive strategies employed rather than merely on task outcome is essential for meaningful cross-diagnostic comparisons.

Interim Summary: Cross-Diagnostic Comparisons of Rigidity

Consideration of the behavioral deficits in ASD in isolation may assist in delineating commonalities across ASD, OCPD, and AN. Happe, Ronald, and Plomin (2006) suggested that research into the pathogenesis of ASD may be impeded by the joint consideration of deficits in communication, social interaction, and repetitive behaviors, when, in fact, accumulating community evidence suggested the independent transmission of these features. Alternatively, it may be that these behavioral domains co-occur in individuals but evidence different developmental trajectories. For example, neurocognitive processes that facilitate biased processing may promote rigidity as a way to cope with a seemingly ever-changing environment. Indeed, the inability to categorize situations with similar meanings but diverse contexts may lead to an overreliance on rigid verbal rules as a way to navigate this diversity. Such considerations highlight the importance of a transactional perspective when considering distinct behavioral domains.

Other commonalities not yet explored, as well as important differences between ASD and AN, will be critical to consider in carving out phenotypes relevant to investigations of common biological pathways. For instance, whereas language impairment is a hallmark of autism, language deficits have not been an area of systematic investigation in AN or OCPD. Alternatively, more subtle deficits in receptive and expressive communication may prove fruitful in future investigations. Importantly, consideration of the expression of symptoms across diagnostic classes highlights the importance of a spectrum approach. It may be that while overlap is evidenced in one symptom domain such as reciprocal social interaction, other domains evidence more commonality across the three diagnostic classes (e.g., rigidity). Thus, our emphasis is not on the relative position of these disorders on a specific continuum of the obsessive-compulsive spectrum, since such dimensions remain to be adequately adjudicated (Hollander & Benzaquen, 1997; Lochner & Stein, 2006), but rather, we support the importance of a spectrum approach in considering social deficits. Loosening the constraints of these diagnostic labels and considering neurocognitive profiles specific to social functioning may add an additional layer of phenotypic precision to genetic and behavioral research.

Summary: Phenotypic Expression of ASD and AN

Returning to ASD and AN, the considerations outlined above provide evidence regarding observable trait similarities between individuals with ASD, individuals with AN, and unaffected relatives across both groups. As summarized, personality overlap, particularly features of aloofness and rigidity, is suggestive. In regard to social functioning, both groups demonstrate significant impairment, and, as will be reviewed in the next section, preliminary work with AN shows similarities in social information processing. However, information on social cognitive processes in AN is limited. This review attempts to address this lapse by using the systematic study of social cognition in ASD as a framework for a similar process of study in AN. The study of eating behavior shows the reverse pattern. We know a great deal about the eating behaviors in individuals with AN as that symptom cluster has been the most compelling for researchers and clinicians, perhaps at the detriment of other needed areas of inquiry. In contrast, eating disturbance, although certainly not manifesting as AN, is widely acknowledged but is rarely a source of systematic inquiry in ASD. Of importance, eating problems in ASD contribute significantly to caregiver burden (Williams et al., 2000) and have been related to

features of rigidity (Ahearn et al., 2001; Schreck et al., 2004). Of further significance is our decision to limit this review to the high-functioning end of the autism spectrum. Disrupted eating in low-functioning individuals with ASD in comparison with that in individuals with AN who exhibit intellectual disability is a complex issue and an important area necessitating a separate review (Zucker & Losh, 2007). Perhaps in a parallel process, communication difficulties and symptoms of social impairment are so compelling that researchers have focused on these areas at the expense of focusing on eating patterns and behaviors. Certainly, the most favorable outcome of the current review would be for our conceptualization of broad phenotypes to be mutually beneficial to researchers and clinicians in both fields, leading to innovations in treatment development.

Teachings From Autism: The Neural Basis of Social Behavior

Social cognition is defined as the ability to construct representations of others, oneself, and interactions between oneself and others (Adolphs, Sears, & Piven, 2001). Interpersonal interaction in ASD is profoundly impaired across several domains of functioning and via multiple channels of communication (Volkmar et al., 2004). Despite this complexity, emerging work in ASD has demonstrated how systematic descriptions of the neuropsychological and behavioral features of the social phenotype of ASD have informed theories of social information processing (Hill & Bird, 2006; Klin et al., 2002a; Lawson et al., 2004). In turn, these theories have promoted guided inquiry into the neural circuitry underlying behavioral disturbances in ASD (Baron-Cohen, 2004b; Pelphrey, Adolphs, & Morris, 2004). Together, these findings have prompted the development of innovative treatment approaches (e.g., Drew et al., 2002). The systematic study of social cognition in ASD can influence similar work in AN, irrespective of proposed similarities between the phenotypes of these disorders. In this next section, we explore disruptions in social neurocognitive functioning in ASD and relate this pattern of evidence to research on these processes in AN. Further, we integrate these findings with theories of social information processing across both disorders to derive a new framework for the study of AN.

Social Perception

Social perception requires the viewer to use social cues to make inferences about individuals engaged in interpersonal interactions, such as their status, motivation, mood state, trustworthiness, and so on (Sergi et al., 2006). The interpretation of such complex stimuli requires the integration of information from multiple sensory channels (Wright, Pelphrey, Allison, McKeown, & McCarthy, 2003). For example, the auditory channel includes, but is not limited to, vocal information such as voice prosody, pitch, nonverbal emotional utterances (e.g., cries), and affective vocal tonality. In the visual domain, the ability to perceive motion plays a crucial role in the ability to interpret the intentions of actions from eye gaze direction, facial expression, body gestures, postural adjustments, and overall directionality and speed of body movement. Such multisensory analysis is further complicated by the differential meaning imparted to these cues by the social context in which the signals arise (Baron-Cohen, O'Riordan, et al., 1999; Stone, Baron-Cohen, & Knight, 1998). To isolate these discrete processes, functional magnetic resonance imaging (fMRI) in healthy adults has identified key brain regions involved in social perception, consistently implicating the superior temporal sulcus region, amygdala, and the fusiform gyrus. Each of these regions, along with related findings in ASD and AN, is discussed below.

Amygdala

The amygdala is a complex subcortical structure that is composed of at least thirteen different nuclei and is highly interconnected with other brain structures, including the thalamus, hippocampus, brainstem, basal forebrain, claustrum, and neocortex (Amaral & Insausti, 1992; Amaral, Veazey, & Cowan, 1982). As an integrative center of highly processed sensory information, emotional circuits, and outputs that influence somatomotor and autonomic activity, the amygdala is considered a key region for processing information of motivational salience for an organism (Anderson & Phelps, 2001). Accordingly, Brothers (1990) proposed that the amygdala is a key component of a neural circuit that forms the neurobiological basis of social perception, along with the superior temporal sulcus and the orbitofrontal cortices.

Lesions of the amygdala are associated with impaired social functioning in animal research (Emery et al., 2001; Freese & Amaral, 2005; Nishijo, Ono, & Nishino, 1988; Prather et al., 2001), and research in humans has increasingly supported the importance of this region in processing motivationally and emotionally salient stimuli of various kinds, including faces. Lesion (Adolphs, Tranel, Damasio, & Damasio, 1994; Anderson & Phelps, 2001) and functional neuroimaging studies (Breiter et al., 1996; J. S. Morris et al., 1996; Whalen et al., 1998) have demonstrated that the human amygdala is critical for normal judgments about the internal states of others from viewing pictures of their facial expressions. Adolphs and colleagues (1994) have previously reported that bilateral damage to the amygdala can result in a disproportionate impairment in recognizing the intensity of fear from faces alone. In functional neuroimaging studies, amygdala activation is consistently detected in response to viewing facial expressions of fear, and this finding persists when fearful stimuli are presented in a subliminal fashion (Whalen et al., 1998) or when fearful eye sclera serve as the only stimuli (Whalen et al., 2004), indicating the minimal amount of visual information needed for amygdala activation in the presence of fear cues. Although this initial work has emphasized the role of the amygdala in processing fear relative to other emotions, recent fMRI studies have shown that the amygdala responds to a variety of emotions (Fitzgerald, Angstadt, Jelsone, Nathan, & Phan, 2006; Yang et al., 2002), and that, in some circumstances, amygdala-lesioned patients can compensate for their facial processing deficits by using cognitive strategies (Adolphs et al., 2005; Graham, Devinsky, & LaBar, 2006).

The amygdala's role is not limited to making judgments about basic emotions but also includes a role in making social judgments (Adolphs, Tranel, & Damasio, 1998). Adolphs and colleagues have found that the amygdala is important for recognizing complex mental states and social emotions from faces (Adolphs, Baron-Cohen, & Tranel, 2002) and for judging the trustworthiness of people from viewing faces (Adolphs et al., 1998), which has been supported by fMRI studies in healthy adults (Winston,

Strange, O'Doherty, & Dolan, 2002). Studies also have suggested a more general role for the amygdala in theory of mind abilities (Stone, Baron-Cohen, Calder, Keane, & Young, 2003). Extensive work supports the pivotal role and sensitivity of the amygdala to social information processing: from the detection of basic emotional state from limited facial cues to the ability to make complex social judgments regarding human intentions.

Amygdala in ASD

The importance of the amygdala in ASD has been suggested by postmortem, structural, and functional imaging studies (e.g., Aylward et al., 1999; Kemper & Bauman, 1993; Schumann et al., 2004). After demonstrating poor performance by high-functioning individuals with ASD in judging mental or emotional states from viewing a photograph of only the eyes of the face (the Eyes Task), Baron-Cohen, Ring, et al. (1999) observed reduced activation of the left amygdala during performance of this task by individuals with ASD relative to that of typically developing individuals. Critchley et al. (2000) found that participants with ASD showed no left amygdala activation in a sex judgment task involving covert processing of facial emotions, and Wang, McCarthy, Song, and Labar (2005) reported no evidence of normal amygdala modulation in individuals with ASD performing a face matching task. Taken together, these studies provide converging evidence that the functioning of the amygdala is abnormal in ASD during social perception.

Neuropsychological studies also support the role of the amygdala in ASD. Adolphs et al. (2001) compared individuals with ASDs with a woman with bilateral focal amygdala lesions (patient SM). The individuals with ASD and patient SM were remarkably similar in exhibiting abnormal social judgments regarding heightened evaluations of trustworthiness and approachability relative to those of neurologically normal individuals. The results of a later study (Adolphs et al., 2005) of patient SM's visual scan paths, combined with data concerning abnormal scan paths for faces in individuals with ASD (Adolphs et al., 2005; Klin, Jones, Schultz, Volkmar, & Cohen, 2002b; Pelphrey et al., 2002) suggest a potential mechanism underlying these shared social perception abnormalities. The eye tracking data demonstrated that patient SM failed to fixate on the eyes when viewing facial expressions of emotion. Similarly, Pelphrey and colleagues (2002) observed that individuals with autism scanned the eyes less when judging facial expressions of emotion. Notably, when Adolphs et al. (2005) instructed patient SM to look at the eyes, her deficit in recognizing fear temporarily disappeared, but it returned after the instruction was forgotten. Results were interpreted as indicating that the amygdala is normally involved in directing the visual system to seek out and attend to the eyes as a socially salient stimulus. Consistent with this hypothesis, Dalton et al. (2005) combined eye tracking and fMRI and found a strong positive correlation in individuals with autism between the level of amygdala activity and gaze fixation on the eye region. Such findings confirm the vital role of the amygdala in facilitating processing of socially salient stimuli.

Amygdala in AN

Unfortunately, a limited body of research has explored the neuroanatomy and corresponding neurocircuitry in individuals with AN in the realm of social cognition. Rather, work conducted to date has largely focused on the symptoms of AN related to eating and body image at the expense of more global considerations of pervasive deficits in functioning (Lask et al., 2005). Nonetheless, findings to date indicate that exploration of the social brain in AN is warranted. As preliminary support, investigations of gross neuroanatomy using single photon emission computed tomography (SPECT) have most frequently implicated the temporal lobe, a neuroanatomic region that includes the amygdala, superior temporal sulcus, and fusiform gyrus, as a region of hypometabolic functioning that persists after weight restoration (Lask et al., 2005). Thus, although the relationship of these regularities to interpersonal functioning has not yet been explored, this region is, indeed, an area in need of investigation.

Studies of the amygdala in AN have largely focused on activation in this region as validating the salience of body weight and food stimuli. Abnormal amygdala activation has been demonstrated reliably in response to disease-related cues (e.g., enhanced activation of the amygdala to the presentation of food- or weight-related stimuli relative to control participants; Frank, Bailer, Henry, Wagner, & Kaye, 2004). Similarly, Uher et al. (2005) reported greater activation in the right amygdala of test participants relative to that in control participants when overweight female silhouettes were viewed. Evidence of irregularities in the serotonergic binding in the mesial temporal cortex, including the amygdala, particularly an underexpression of the 5-HT2A receptor, has been reported, and importantly, these irregularities are seen in recovered individuals (Frank et al., 2002). Recent work has documented an association of this receptor irregularity with the trait of harm avoidance, a reliably reported feature in AN connoting inhibited behavior in anticipation of frustrated nonreward or potential punishment (Cloninger, Przybeck, Svrakic, & Wetzel, 1994). Thus, the association of irregularities in the mesial temporal cortex with harm avoidance is in keeping with the role of this neural region for determining motivational salience (O'Doherty, Critchley, Deichmann, & Dolan, 2003; O'Doherty et al., 2004). Unfortunately, to our knowledge, despite pervasive deficits in interpersonal functioning, no study has examined the role of the amygdala in relation to social cognition in AN. The study of social stimuli will enable more direct comparisons with findings from the literature in ASD.

Superior Temporal Sulcus

The superior temporal sulcus is increasingly being regarded as a higher-order processing region for the integration of multisensory stimuli and motion perception (Grossman & Blake, 2002; Jellema, Maassen, & Perrett, 2004; LaBar, Crupain, Voyvodic, & McCarthy, 2003; J. P. Morris, Pelphrey, & McCarthy, 2005; Pasternak & Merigan, 1994; Pelphrey, Mitchell, et al., 2003). To separate unique brain activation to motion cues from other contextual factors, researchers have frequently used highly degraded visual stimuli such as point-light displays (Heberlein, Adolphs, Tranel, & Damasio, 2004). In such arrays, a point of light is placed at several joints of the human body and the movement of these dots conveys either biological motion (e.g., points of light jumping) or random motion. Investigations using both degraded stimuli and investigating goal-directed action relative to random action have demonstrated that while the superior temporal sulcus is sensitive to

motion, this region is differentially sensitive to motion arrayed in biologically relevant sequences (J. P. Morris et al., 2005; Pelphrey, Mitchell, et al., 2003). Further, the superior temporal sulcus demonstrates sensitivity to the context of biological motion, becoming more active when an action and its context are incongruent (Pelphrey et al., 2004). In a study of hand grasping, Pelphrey et al. (2004) demonstrated greater activation when a figure's reach to grasp motion was incongruent with its surrounding context than when this action was congruent.

Similarly, the superior temporal sulcus is also sensitive to the context of facial movement. The superior temporal sulcus region has demonstrated acute sensitivity to subtle changes in facial movement associated with facial affect. LaBar, Crupain, Voyvodic, and McCarthy (2003) utilized morphing technology to gradually morph a neutral expression into one of fear or anger, and the stimuli with such motion changes elicited greater activity in the superior temporal sulcus, amygdala, and fusiform gyrus than did static portrayals of the same emotions (LaBar et al., 2003). Consistent with the sensitivity of the superior temporal sulcus to incongruent contextual information, Mosconi, Mack, McCarthy, and Pelphrey (2005) reported greater activation in the superior temporal sulcus when a figure's eye gaze was incongruent with the location of an object placed in the figure's periphery (Mosconi et al., 2005). This body demonstrates the sensitivity of the superior temporal sulcus to biologically relevant motion, potentially particularly in ambiguous contexts.

Similarly, the superior temporal sulcus region becomes increasingly active when auditory stimuli is associated with socially relevant features (Wright et al., 2003). In healthy developing individuals, the superior temporal sulcus exhibits greater activation in response to angry prosody of human voices relative to neutral prosody (Sander et al., 2005). Further, this process was demonstrated to occur independently of attentional resources devoted toward detecting a social input (Sander et al., 2005). An alternative line of inquiry has examined the role of the superior temporal sulcus in integrating visual and auditory inputs (Barraclough, Xiao, Baker, Oram, & Perrett, 2005). Barraclough et al. (2005) demonstrated that the superior temporal sulcus had unique projection zones for visual and auditory input but also specialized areas in which these inputs overlapped. Thus, evidence to date highlights the important role of the superior temporal sulcus in several necessary processes for social perception: the detection of biologically relevant motion, the integration of motion with social context, the convergence of visual and auditory inputs, and the integration of visual and auditory stimuli with their affective salience.

Given the complex role of the superior temporal sulcus for social perception, this brain region has been increasingly implicated as an essential part of the human mirror neuron system (Iacoboni & Lenzi, 2002). There has been surging interest in the study of this putative system, a system that describes the correspondence in neuroanatomic activation that occurs in both the perception and execution of behavior, that is, the correspondence in activation when scratching your head relative to when watching someone scratch his or her head (Iacoboni & Lenzi, 2002). The superior temporal sulcus has been hypothesized as a key structure within this system given its relevance in integrating inputs from diverse sensory channels including biological motion (Arbib, Billard, Iacoboni, & Oztop, 2000). An increasing body of evidence supports the association of activation in the putative mirror system with empathetic responding (Bodini, Iacoboni, & Lenzi, 2004; Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003; Iacoboni & Lenzi, 2002). Thus, the mirror system may be a particularly compelling region of study in disorders associated with deficits in reciprocal social interaction such as ASD. Notably, systematic study of the mirror neuron system is relatively recent. Conclusive statements regarding the role of implicated neural structures in the mirror neuron system await further investigation. In sum, this body of work has provided convincing evidence of the crucial role of the superior temporal sulcus in processing diverse forms of biological motion, an essential component of socially salient stimuli. As such, this region may be a fascinating area for examination in AN.

Superior Temporal Sulcus in ASD

Several lines of evidence support impairment in the superior temporal sulcus in ASD. Boddaert et al. (2003) reported abnormal laterality and hypoactivation of the left superior temporal sulcus region during speech perception using positron emission tomography, and Gervais et al. (2004) reported abnormal responses to human voices in the superior temporal sulcus region by using fMRI. A region of the superior temporal sulcus that had previously been shown to respond selectively to vocal sounds in typically developing people (Belin, Zatorre, Lafaille, Ahad, & Pike, 2000) failed to activate in response to this stimulus category in individuals with autism (Gervais et al., 2004). These participants with autism showed normal auditory cortex activation to nonvocal sounds.

The relation of abnormalities in the superior temporal sulcus to styles of information processing in individuals with ASD is currently an active area of study. As mentioned in the previous discussion of central coherence (Happe & Frith, 2006), individuals with ASD have consistently been reported to exhibit perplexing patterns of visual information processing, exhibiting superior or even savant capacities for tasks that require the detection of patterns or sequences, that is, attention to detail, but impaired capacities on tasks that demand integration of meaningful structures from details (O'Riordan, Plaisted, Driver, & Baron-Cohen, 2001). Thus, individuals with ASD exhibit enhanced processing on neuropsychological tasks in which the ability to inhibit global structure confers an advantage, such as embedded figure tasks (Jolliffe & Baron-Cohen, 1997; Plaisted, O'Riordan, & Baron-Cohen, 1998). Although evidence of these capacities is being increasingly documented, the neurocognitive correlates of these processes remain an open area of investigation (Klin et al., 2002a).

Dakin and Frith (2005) discussed the deficits in central coherence reported in ASD in relation to the neuroanatomical functioning of the visual system. They noted that the nature of the visual stimuli used in numerous research investigations does not permit a clear distinction of whether individuals with ASD exhibit superior local processing that interferes with the detection of global configurations or, rather, whether genuine impairment in the detection of global structure exists. They do note, however, that in every report in which the perception of motion has been conducted in ASD, there is evidence, either direct or indirect, of atypical activation in the superior temporal sulcus (Dakin & Frith, 2005). Given the essential role of motion in complex social stimuli (i.e., body language, facial muscles, gaze direction), combined with the sensitivity of the superior temporal sulcus to incongruent contex-

tual factors, further study of the superior temporal sulcus may help to inform information processing capacities in ASD and, potentially, in AN.

Superior Temporal Sulcus in AN

Although systematic inquiry exploring the superior temporal sulcus has not been conducted, neuropsychological findings in AN highlight dysfunction in tasks implicating the superior temporal sulcus. First, individuals with AN have been reported to exhibit deficits in identifying affective prosody in language (Kucharska-Pietura, Nikolaou, Masiak, & Treasure, 2004), while, to our knowledge, motion perception has not been investigated. However, most notable is burgeoning evidence supporting poor perceptual organization capacities in AN, particularly in regard to central coherence (Chowdhury, Gordon, & Lask, 2001; I. C. Gillberg et al., 1996; Lask et al., 2005; Wentz et al., 2000). Importantly, these visual–perceptual deficits have been demonstrated in both adolescent and adult patients and have been reported to persist with recovery (Lask et al., 2005).

Exploration of the superior temporal sulcus could also shed light on a perplexing and poorly understood symptom in AN, namely body image disturbance. Body image disturbance, part of the core phenomenology and diagnostic criteria of AN, refers not only to perceptual aspects (i.e., the accuracy with which the body is perceived) or cognitive aspects (i.e., the evaluation one gives to one's image) but also to dysfunction in the way in which the body is experienced (Uher et al., 2005). The plausibility of the role of the superior temporal sulcus in body image disturbance is supported by studies describing distinct regions in the occipitotemporal cortex that respond selectively to the human body and project to the superior temporal sulcus (Downing, Jiang, Shuman, & Kanwisher, 2001). Further, the detection of motion is necessarily postural. Perceiving motion permits an individual to shift and maintain posture in the presence of changing environmental features (Dalton et al., 2005). Thus, motion sensing involves the unification of perceptual and experiential dimensions, two facets of body image.

Traditionally, visual assessments of body image disturbance in individuals with AN have yielded limited findings (Skrzypek, Wehmeier, & Remschmidt, 2001). However, the paradigms employed, such as estimation of the whole body or body parts in isolation by using various methodologies from video morphing to light beam adjustment, have not been designed to assess the role of impaired coherence in body image disturbance. The implementation of novel paradigms that incorporate what is known of the information processing styles in AN may lead to more fruitful findings in the arena of body image disturbance.

AN presents perplexing paradoxes that investigation of the superior temporal sulcus and other components of the mirror neuron system may help to resolve. Individuals with AN have been reported to engage in elevated levels of social comparison (Mitzman, Slade, & Dewey, 1994; Tsiantas & King, 2001), competitiveness (Shafran, Cooper, & Fairburn, 2002), and conformity (Casper, Hedeker, & McClough, 1992; Strober, 1981). Notably, far from being adaptive, such tendency to mimic is often quite maladaptive; that is, individuals with AN often mimic the behavior of others (e.g., dieting) in circumstances in which doing so is detrimental to their physical and mental health. As indirect support, an extensive body of research documents the role of social pressures from family, peers, and the media on symptom exacerbation, and individuals with AN, by definition, take these standards to a dangerous extreme (Stice & Whitenton, 2002). Thus, researchers are left with the perplexing circumstance of individuals who imitate and conform and yet struggle to form meaningful attachments. One manner in which to interpret these behaviors is that individuals with AN may have difficulty interpreting the meaning of an action within its situational context. For example, one possibility is that deficits in decoding the nuances of social interaction may result from both bottom up (i.e., integration of conflicting or ambiguous sensory information) and top down (i.e., integration of somatic markers of emotion into decision making). Study of brain regions implicated in social perception will be pivotal in isolating the key neural circuits involved in these deficits.

Fusiform Gyrus

The specificity of the fusiform gyrus in the processing of static facial stimuli is well established (McCarthy, Puce, Gore, & Allison, 1997). Some of the clearest evidence for face specificity in the fusiform gyrus comes from studies during which electrodes inserted in the ventral occipitotemporal cortical surface during brain surgery display face-specific activity within 200 ms of the presentation of a face (Allison, Puce, Spencer, & McCarthy, 1999; McCarthy, Puce, Belger, & Allison, 1999; Puce, Allison, & McCarthy, 1999). Further, while the fusiform gyrus is activated in response to both previously viewed and novel faces, relative activity in the fusiform gyrus discriminates between faces that have been actively viewed versus covertly viewed (Lehmann et al., 2004). Caldara et al. (2006) reported the fusiform gyrus was specifically sensitive to face-shaped geometric shapes that exhibited a top-heavy pattern relative to other configurations. Together, these findings support the specific sensitivity of the fusiform gyrus for the detection of familiar and unfamiliar conspecific faces.

Fusiform gyrus in ASD. Disorientation to faces is a well-documented behavioral feature of individuals with ASD, recognized as early as the 1st year of life (Osterling, Dawson, & Munson, 2002). Given the specificity of the fusiform gyrus for processing of facial features, this region has been a likely target of exploration in ASD. In studies of individuals with ASD, Schultz et al. (2003) found that there was less face-evoked activation in the fusiform gyrus in combination with activation in regions recruited for nonface object perception. Additional fMRI studies have replicated the finding of fusiform gyrus hypoactivation (Critchley et al., 2000; Hubl et al., 2003; Pierce, Haist, Sedaghat, & Courchesne, 2004). Given the crucial role the amygdala plays in driving enhanced processing of the eye region, the study of interconnectivity between the amygdala and fusiform gyrus has enabled researchers to propose several developmental theories regarding social processing deficits (Dakin & Frith, 2005; Dalton et al., 2005). These are briefly overviewed in the following section. While it has been supported that individuals with ASD have hypoactivation in brain regions specific for face processing, the developmental significance of this region to social cognition deficits is an important area of exploration.

Fusiform gyrus in AN. Unfortunately, work on the fusiform gyrus in AN is too sparse for meaningful comparison. In a study of

body image, Uher et al. (2005) reported that the fusiform gyrus was less activated bilaterally in individuals with AN relative to individuals with bulimia nervosa or to healthy control participants. Of significance, this study presented the participants with silhouettes of underweight, normal weight, and overweight women. Individuals with AN evidenced decreased activation in the fusiform gyrus irrespective of the type of stimulus, indicating the pattern of activation is not merely an artifact of the body weight of the stimuli but may reflect a more pervasive deficit. Functioning of the fusiform gyrus may help to inform the development of social information processing deficits in AN.

Integrating Affective and Social Processing

We emphasize above the unique contributions of three brain structures to social perception: the amygdala, superior temporal sulcus, and fusiform gyrus. This analytic perspective is helpful in providing a framework for organizing the emerging understanding of the social brain but does not fully reflect the complexity of interactions among these and other brain regions during social perception. How do the amygdala, superior temporal sulcus, and fusiform gyrus interact as components of a neural system to jointly process socially relevant information? How is the integration of emotional relevance achieved? The available data support the idea that higher-order association cortices in the temporal lobes, such as the fusiform gyrus and superior temporal sulcus, first encode the visual properties of socially relevant stimuli, and this information is then subsequently passed to neurons within the amygdala that associate the visual percept with its emotional meaning (Adolphs, 2003). However, there is now also clear evidence for the converse: top-down modulation of occipitotemporal cortices by structures such as the amygdala. Such a scheme is supported by the anatomical evidence for feedback projections from the amygdala to nearly all levels of the visual cortex hierarchy (Vuilleumier, Richardson, Armony, Driver, & Dolan, 2004). It is therefore likely that an initial stage of visual processing draws on the fusiform gyrus (for representing static features of faces and their spatial configuration) and the superior temporal sulcus (for representing dynamic features of socially relevant visual stimuli, including faces and biological motion stimuli; Anderson & Phelps, 2001). However, at the same time, some of this visual information from occipitotemporal cortical regions, as well as coarser visual information via subcortical routes, is being conveyed to the amygdala and associated with its social and emotional meaning. At later processing times, the amygdala can then influence the perceptual representation of the stimulus in the fusiform gyrus and the superior temporal sulcus region (McCarthy et al., 1999; Pelphrey, Mack, Song, Guzeldere, & McCarthy, 2003). Such interactions implicate not only that what we see can affect an individual's emotional state but, also, that an individual's emotional state can influence what is seen. Furthermore, these brain regions are anatomically connected to sectors of the orbitofrontal and ventromedial prefrontal cortex, where social evaluative processes become associated with motivational goals and executive control of behavior. Such interdependent circuitry results ultimately in the choice and execution of adaptive or maladaptive emotion regulation strategies (Hinshaw, 2003; Figure 3). Understanding the interactions of these neural circuits can enhance our knowledge of the pathophysiology of AN and may provide a vital resource for the investigation of social perception in other psychiatric disorders.

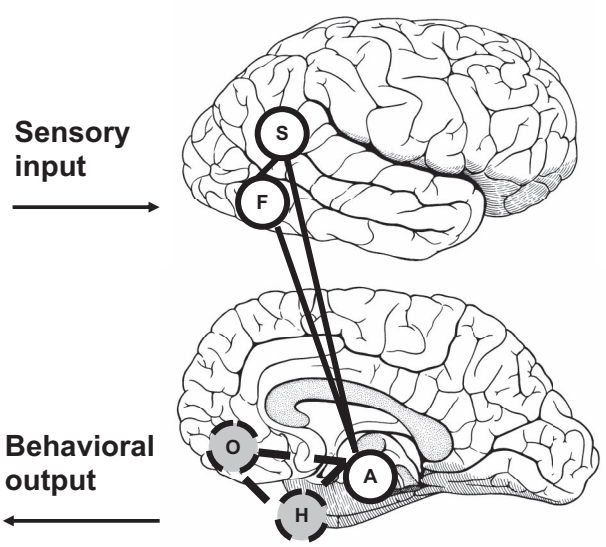

Figure 3. Social brain network in anorexia nervosa. Sensory input is evaluated for social information in the superior temporal sulcus (S), fusiform gyrus (F), and amygdala (A; S, F, and A are presented in white circles with solid lines). Projections from the A to frontolimbic brain regions, including orbitofrontal cortex (O) and hypothalamus (H) link social information processing with motivational goals and control of feeding behavior (O and H are presented with grey circles and dashed lines). Feedback projections from frontolimbic regions, in turn, can influence representation of sensory input.

Developmental Models of Social Deficit

Understanding of the interrelationship of functional neural circuits related to social perception can facilitate the design of developmental models of social information processing. For example, several competing theories have been proposed to explain hypoactivation in the fusiform gyrus in ASD (Dakin & Frith, 2005; Dalton et al., 2005; Schultz et al., 2003). In an oversimplification (see Schultz, 2005; and Dalton et al., 2005, for thorough accounts), Schultz (2005) has argued that early amygdala disruption in autism leads to a lack of interest in faces. This corresponding lack of attention to faces throughout development leads to abnormalities in the functional pathways between the amygdala and the fusiform gyrus, which are dependent on ongoing input from the amygdala to develop fusiform gyrus face specificity. Thus, avoidance of faces is a passive, developmental process that inhibits the development of the face specificity of this region. Alternatively, Dalton et al. (2005) proposed that amygdala dysfunction and associated oversensitivity to fear result in an active avoidance of face processing. Thus, according to these authors, avoidance of emotionally salient cues is an active process that results in dysfunction due to a lack of experience in processing faces.

Other authors explored the role of the superior temporal sulcus in contributing to social cognitive deficits. According to reports by Pelphrey and Morris (2006) and Dakin and Frith (2005), disruption in the superior temporal sulcus affects the manner in which indi-

viduals with ASD process faces. There is evidence that individuals with ASD selectively process noncrucial facial regions (e.g., the chin, lips, and mouth) to make emotional judgments rather than processing key regions (e.g., eyes). Such processing is proposed to be congruent with deficits in central coherence, the difficulties individuals with ASD have in integrating parts with wholes. In summary, the workings of the social brain can provide novel hypotheses into the pathophysiology of social functioning in disease.

Could these models have relevance for AN? Unfortunately, knowledge of the workings of the social brain in AN is too premature to hypothesize developmental models. Notwithstanding, several patterns of evidence provide interesting arenas for hypothesis generation. In accordance with the models of Dalton et al. (2005) and Schultz (2005) implicating that amygdala dysfunction either passively or actively promotes fusiform gyrus hypoactivation, the avoidance of emotional experience is a frequently cited feature of AN. As codified in the features of harm avoidance and in deficits such as impaired interoceptive awareness, the avoidance of emotional experience has been consistently documented in AN (Fassino et al., 2004; Klump et al., 2000). These features describe the tendency to anticipate and avoid anxiety-provoking situations and the difficulty distinguishing somatic from emotional internal cues. As an emotionally provocative stimulus, the degree to which individuals with AN actively fail to process facial regions has not been studied. This hypothesis would imply that individuals with AN have the capacity to read the subjective experience of others accurately; however, elevated levels of anxiety lead to prolonged avoidance of salient facial cues promoting inaccurate interpretation. In accordance with the developmental models proposed by Schultz (2005) and Dalton (Dalton et al., 2005) for ASD, such chronic avoidance, whatever its biological substrate, would lead to decreased experience in the processing of faces and increased dysfunction over time.

Alternatively, or additively, deficits in the superior temporal sulcus could lead to impairment in the way multiple sources of information were integrated (Pelphrey & Morris, 2006). What evidence is there to support these hypotheses in AN? The only indirect evidence are deficits in visuospatial organization that persist with recovery and are identified in both adult and child patients, a finding that has been repeated by several laboratories (Lask et al., 2005; Treasure, Tchanturia, & Lopez, 2006). The mechanisms driving these deficits have yet to be elucidated; however, the superior temporal sulcus, in relation to other regions of the social brain, could provide novel insights. Together, the transaction of social cognitive processes can help researchers understand the nature of the mature phenotype in psychiatric illness. From these considerations, we pose a social model of AN.

A Social Model of AN

To understand the interpersonal challenges of individuals with AN, a model is needed that integrates cognitive neuroscience with the known social phenotype of this disorder. In this section, we merge this extensive body of literature by combining models of information processing drawn from both research literatures—in AN, the habit-learning model of Steinglass and Walsh (2006) and the pathologic fear conditioning model of AN of Strober (2004); and in ASD, the empathizing/systemizing dimensional system of Lawson et al. (2004). Linking these models provides a pathway to study social cognition in AN, perhaps bringing new hypotheses for a social cognitive endophenotype that bridges diagnostic classifications.

The empathizing/systemizing system characterizes individual strengths in analyzing information to find order and build systems relative to searching for human understanding (Lawson et al., 2004). The tendency of individuals with AN for prediction or control in combination with their desire for social acceptance provides an interesting interpretation of the empathizing/systemizing system (Lawson et al., 2004). Consideration of the presentation of AN lends readily to the description of these individuals as systemizers. Although not often a term used in the eating disorder literature, the search for order, control, and predictability is part of the core phenomenology of this illness and is the essence of systemizing (Lee, Chan, Kwok, & Hsu, 2005; Surgenor, Horn, & Hudson, 2003; Westen & Harnden-Fischer, 2001). In fact, weight control is but one manifestation of this systemizing. As a case in point, an oft-cited theme that initially motivated the desire for extreme weight loss in individuals with AN is a need for control; that is, the workings of their physical body become the obsessive focus of these systemizing pursuits (Shafran et al., 2002). Thus, there is similarity in the subjective style of information processing across these diagnostic categories.

However, there is a seeming difference between individuals with ASD and individuals with AN in their ostensible desire for affiliation. Notably, the social subtypes described by Wing (1997) are notable for patterns of withdrawal but differ, potentially, in the desire for affiliation. Social discomfort, in general, is an oft-cited feature in ASD. In contrast, despite evidence of elevated social anxiety and associated social avoidance (Bulik, 1995; Flament, Godart, Fermanian, & Jeammet, 2001), the desire for social acceptance in AN is compellingly strong as evidenced by a significant body of work documenting self-consciousness (Shafran et al., 2002), conformity (Huon et al., 2002; Stice, Maxfield, & Wells, 2003; Stice & Whitenton, 2002), and interpersonal events as features that confer risk of eating disorder development (Schmidt, Tiller, Blanchard, Andrews, & Treasure, 1997). Notably, this desire occurs against a backdrop of insecure attachments (Ward et al., 2001). This disjunction highlights a paradox of AN: Although individuals with AN may desire affiliation, they may not be very skilled at such a goal (Stice, 2002; Ward, Ramsay, Turnbell, Steele, et al., 2000). In AN, the impairment in social processing and desire for interaction may have prompted an adaptive strategy: They may be systemizers of social information, that is, mirroring the behavior of others as a way to adapt to their social environment to compensate for deficits in social cognition.

Differences in the sex ratios of these disorders are notable. In ASD, the male to female ratio is 10:1 (C. Gillberg, 1989). An "extreme male brain" theory of Asperger's syndrome has been proposed in which individuals are described to exhibit extreme forms of prototypically male strengths in cognitive processes: for example, spatial organization and mathematics (Baron-Cohen et al., 2003). In AN, the sex ratio is the opposite with a male to female ratio of 1:9. Thus, although individuals with AN may exhibit a similar pattern of information processing, that is, a relative strength in systemizing relative to empathizing, the stronger desire for affiliation in female individuals may promote the use of systematizing in a social context (Connellan, Baron-Cohen,

Wheelwright, Ba'tki, & Ahluwalia, 2001; Luxen, 2005). Of note, individuals with AN in the diseased state have pathologically low levels of estrogen, an artifact of starvation that may differentially impact individuals with AN relative to nondisordered individuals of low weight (Brambilla et al., 2003). The sex differential across these disorders is offered only as an arena for speculation and to demonstrate that similar styles of information processing may manifest differently in interaction with sex or, in a related fashion, that a genetic liability may differentially manifest in men relative to women.

A systematizing view of social interaction maps onto what is known of the neuroanatomic and neuropsychological findings in AN. Recently, two theories of information processing have been proposed to explain the pathophysiology of AN. Steinglass and Walsh (2006) proposed dysfunction in learning strategies in AN. By using evidence from neuropsychological studies (Steinglass & Walsh, 2006; Tchanturia, Anderluh, et al., 2004), these authors provided data that individuals with AN have difficulty adjusting behavior to changing contingencies. In other words, these individuals have difficulty adjusting their behavioral response despite feedback that their response is not promoting goal acquisition (Tchanturia, Anderluh, et al., 2004). As reviewed, such deficits manifest on neuropsychological tests as impaired set shifting, that is, a delay in switching from one strategy to another (Tchanturia, Anderluh, et al., 2004). Further evidence is provided by studies documenting deficits in response flexibility and problem solving strategies (Fassino et al., 2002). The previously reviewed literature on behavioral and cognitive rigidity provides further indirect evidence of disruptions in learning processes. While the type of learning that is disrupted in individuals with AN remains unclear, further research in functional neurocircuits that distinguish learning in individuals with AN from healthy individuals can inform this theoretical perspective.

A failure to learn from experience may promote greater use of models as a learning source. Individuals learn via a variety of strategies, and one powerful mode of learning is via models, or imitative learning (Bandura, Ross, & Ross, 1963). While the neuroanatomic correlates of mirroring or imitative learning have yet to be conclusively elucidated (Iacoboni, 2005), phenotypic evidence is provocative regarding the overreliance of this strategy as a source of learning in individuals with AN (le Grange, Tibbs, & Noakes, 1994; Strober, 1981). Thus, to cope adaptively with their social environment, individuals with AN may use guides and develop systems and rules. Such strategies are highly adaptive in certain contexts. For example, research with individuals with ASD has demonstrated that when the social context lends itself to systemizing, the performance of individuals with ASD equals that of healthy comparison participants (Sally & Hill, 2006). However, these strategies have boundaries on their usefulness, as interpersonal interaction is limited in its predictability. The habit-learning model of Steinglass and Walsh (2006), combined with further study of disrupted forms of learning in individuals with AN, may help to substantiate both whether individuals with AN have to overrely on patterning as a coping strategy and why they do not benefit as quickly from interpersonal cues.

However, such a model does not address the choice of models individuals with AN choose to mimic. As illustrated in Figure 4, individuals with AN often mimic the unhealthiest member of a social group with symptoms that may be triggered by the dieting behavior of others. Further, they stare at emaciated individuals and have difficulty disengaging from those around them who engage in unhealthy behaviors. It is here that Strober's (2004) model of pathologic fear conditioning in AN is relevant. Strober (2004) proposed individuals with AN have a neural fear network that mimics that seen in phobic responses: overresponsive to fear cues and resistant to fear extinction. While an organized structure of

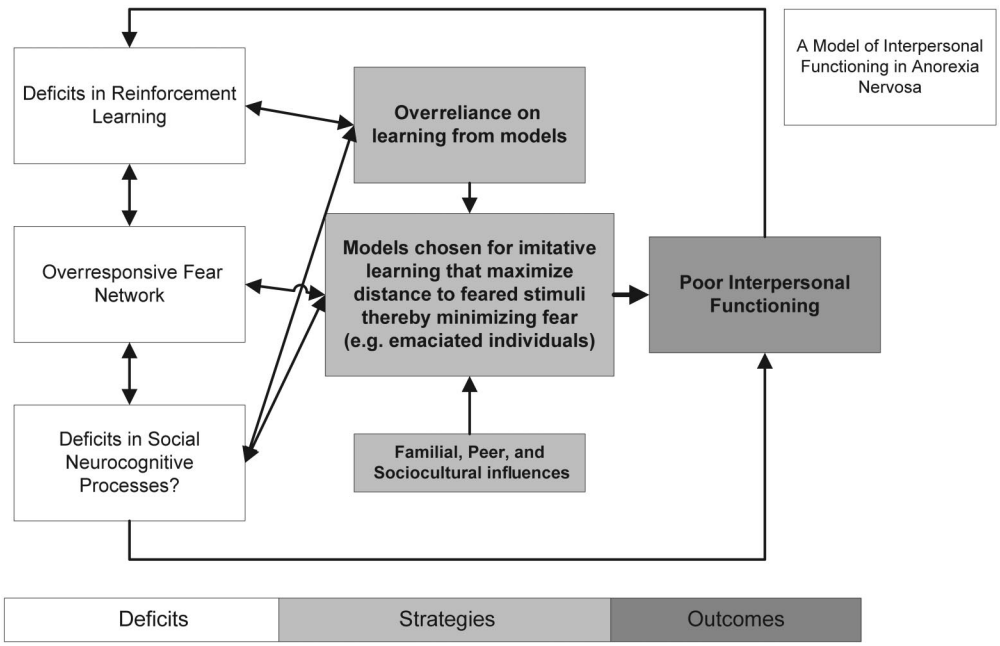

Figure 4. A social model of anorexia nervosa.

cues to danger and subsequent automatic responding is adaptive for survival, when this system overresponds, that is, when it perceives danger in the absence of real threat as in the case of anxiety disorders, maladaptive avoidance can occur. Strober noted this model of pathologic fear conditioning may represent a broad endophenotype of anxiety disorders, in general, and, thus, posed the question, "If a common liability to extreme fear conditioning exists among persons with diverse anxiety disorders, why does a fear of weight gain develop in some persons and phobic states in others?" (p. 508; Strober, 2004). Notably, a consideration of computational neuroscience in conjunction with Lawson, Baron-Cohen, and Wheelwright's model (2004) may provide a tentative hypothesis to address this question.

The prototypical, and arguably adaptive, response of anxiety is escape or avoidance (Derryberry & Reed, 2002). More precisely, an individual will strive to maximize the distance between oneself and that which is feared. Thus, in individuals with high levels of trait anxiety, the safest model to mimic is the one that minimizes the risk to the largest degree. In a society in which overweight is feared, the choice is clear: Individuals with AN who fear overweight will mimic the most emaciated model. Unfortunately, Steinglass and Walsh's (2006) theory of habit learning combined with Strober's theory of a "pathologic" fear network would complicate the process of disengagement from the practices from an unhealthy model. Individuals with AN would overrely on maladaptive models, would demonstrate impairment in their ability to learn when a model was being maladaptive and thus adapt their behavior accordingly, and ultimately, would have difficulty disengaging from this model.

Indeed, there is substantial evidence of both high levels of trait anxiety and the trait of harm avoidance, which is a feature associated with behavioral inhibition, in individuals with AN and their parents. It is of further interest that the trait of harm avoidance is associated with dysfunction in the ventral striatum (Frank et al., 2005). Recent research in computational neuroscience has begun to elucidate the functions of the ventral striatum in instrumental conditioning, a type of learning requiring the organism to make a behavioral response and subsequently learn from the results of that response (O'Doherty et al., 2004). The striatum is heavily interconnected with structures in the limbic system, including the amygdala. The amygdala, in turn, provides a bridge from the striatum to social cognitive structures and the prefrontal cortex (Figure 3). Thus, computations in the striatum may link maladaptive learning to social cognitive deficits, a process that may be potentiated by emotional valence and may result, ultimately, in maladaptive behavioral responses. The study of social cognitive processes thus should accommodate potential disruptions in learning and reward mechanisms posited in AN.

Summary

In summary, the investigation of social cognitive processes and manifest interpersonal patterns may propel new hypotheses regarding the pathophysiology of AN and, ultimately, lead to novel intervention and prevention efforts. Understanding the nature of social cognitive processes in AN can aid in the delineation of a social cognitive endophenotype that may help bridge ostensible barriers of diagnostic classifications and facilitate genetic research by helping to define precise behavioral endophenotypes. Given the heterogeneity seen across the subtypes of AN, it would be ill informed to presume that there exists one interpersonal style in AN. Rather, study of patterns of interpersonal interaction most likely exists that may help focus genetic and neurobiological research. Furthermore, the study of social cognition provides a new focus to help understand barriers to treatment in AN, barriers such as poor therapeutic and patient–carer alliances. Such findings are particularly crucial given the strong interpersonal focus in treatments for AN. Thus, identified deficits in social cognitive processes would highlight neglected areas in treatment design that, if addressed, could greatly potentiate existing interventions while providing new avenues for treatment development. Ultimately, treatments that incorporate social cognitive deficits may be sensitive to the phenomenology of individuals with AN while addressing existing treatment barriers and thus may result in particularly efficacious interventions. Finally, in the case of ASD and AN, it is our intention that collaboration across these fields can not only facilitate the development of interventions to promote social competence in AN but, perhaps, can also facilitate better identification and treatment of individuals with ASD and their family members with eating disturbance (Zucker & Losh, 2007). Our hope is that this review provides several new avenues for hypothesis generation, a process that can be facilitated by interdisciplinary collaboration. Now is not the time to be socially isolated.

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Received August 22, 2006
Revision received May 29, 2007
Accepted June 4, 2007 ■