Chapter 7
Aberrant Social Attention and Its Underlying Neural Correlates in Adults with Autism Spectrum Disorder

Jennifer C. Bush and Daniel P. Kennedy

Humans are highly social beings and display a strong early preference for seeking out and processing social information from their environment. For instance, newborn infants as young as 10 minutes old will show preference to face-like patterns over "scrambled" face patterns (Goren, Sarty, & Wu, 1975; Johnson, Dziuarawiec, Ellis, & Morton, 1991), 4-day-old infants have been shown to recognize their mothers (Field, Cohen, Garcia, & Greenberg, 1984), and 2–5-day-old newborns can discriminate between direct and averted eye gaze (Farroni, Csibra, Simion, & Johnson, 2002).

Social preferences and attention emerge early and spontaneously, and likely set the stage for the development and refinement of various social skills that are critical for successful social behaviors exhibited over one's lifetime (Chevallier, Kohls, Troiani, Brodkin, & Schultz, 2012). This includes the ability to infer the feelings of others, effectively communicating one's thoughts and feelings, and maintaining social relationships, among other important social functions. And, while beyond the scope of the present chapter, it is worth highlighting that social attention is a particularly far-reaching process, as it influences functional domains beyond those classically thought of as social. For instance, the development of language has been shown to rely critically on a highly social process—that of initiating and responding to the attention of others (i.e., joint attention) (Kuhl, 2007; see Bruinsma, Koegel, & Koegel, 2004 for review).

While social attention and early social preferences develop spontaneously for most people, this is not always the case. In particular, individuals with autism spectrum disorder (ASD) exhibit striking abnormalities in social attention. These abnormalities seem to emerge sometime within the first year of life and persist

J. C. Bush (✉) · D. P. Kennedy
Department of Psychological & Brain Sciences, Indiana University, 1101 East 10th Street, Bloomington, IN 47405, USA
e-mail: jencbush@indiana.edu

D. P. Kennedy
e-mail: dpk@indiana.edu

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over the life span, although they may manifest in more subtle ways in later childhood, adolescence, and adulthood, and therefore require more sensitive tools to quantify them. Shultz, Jones, and Klin (Chap. 6, this volume) covered the early development of social attentional abnormalities in autism. Here, we will review some of the social attentional abnormalities in older individuals with an ASD, and discuss the implications of these abnormalities on social functioning.

7.1 ASDs and Early Emergence of Abnormal Social Attention

Individuals diagnosed with ASD often have profound difficulties navigating the social world, exhibiting striking deficits in many areas that require complex social cognitive abilities. The term "spectrum" is crucial in understanding ASD, as there is a wide range of symptoms and degree of affectedness, resulting in incredible individual variation. This heterogeneity was first highlighted in Leo Kanner's (1943) original case studies of ASD, with individuals exhibiting a wide variation in language and social abilities. This heterogeneity no doubt contributes to the challenges associated with early identification and diagnosis, as well as the efforts to elucidate the mechanisms underlying ASD behavior and cognition. Though individuals with ASD exhibit impairment in several functional domains, Kanner highlighted the social and emotional disturbances as the core features of the disorder, arguing that "the outstanding, 'pathognomonic', fundamental disorder is that the children's inability to relate themselves in the ordinary way to people and situations of the beginning of life" (Kanner, 1943).

Numerous studies have documented the early emergence of social attentional abnormalities in young children with ASD. As a review and discussion of this topic can be found in the previous chapter (Shultz et al., Chap. 6, this volume), we do not intend to cover this topic in any depth. For context, however, we provide a brief description of several key findings.

Retrospective studies examining home videos report that by 12 months of age, children later diagnosed with ASD are less likely to look at faces of other people (Osterling & Dawson, 1994; Adrien et al., 1993), fail to orient to their names (Maestro et al., 2001; Osterling, Dawson, & Munson, 2002), show poor eye contact (Adrien et al., 1993; Clifford, Young, & Williamson, 2007), and show reduced initiation of, and response to, joint attention (Osterling & Dawson, 1994; Nadig et al., 2007; Sullivan et al., 2007)—a process that by definition involves social attention, and directing the attention of another social agent. Prospective longitudinal studies of infants at risk for an ASD, but whose young age precludes reliable diagnosis, also report similar differences. Infants later diagnosed with ASD show differences in visual attention, have reduced eye contact, look more at mothers' mouths than eyes, exhibit lower levels of social smiling, reduced bids for joint attention, and use significantly fewer gestures than typically developing infants

(Mitchell et al., 2006; Zwaigenbaum et al., 2005; Nadig et al., 2007; Ozonoff et al., 2010). These altered patterns of attention are generally thought to relate to the later emergence of autism, but how?

While the causes of abnormal social attention in ASD remain uncertain, early abnormalities in social attention are thought to have cascading downstream effects that affect typical neural, cognitive, and behavioral development (Mundy & Neal, 2000; Chevallier et al., 2012). In other words, abnormal social attention may result in diminished social expertise, which then impacts the behavioral, cognitive, and neural specialization for social processes generally, resulting in further atypical allocation of social attention, and so on. This continues on in a self-reinforcing cycle across development that ultimately results in atypical social functioning. Thus, understanding the earliest points within this developmental derailment may be crucial to understanding how autism emerges, and essential in informing early intervention.

However, social attention needs to also be studied beyond infancy and across developmental ages, as social attention as a process can profoundly shape social functioning at all ages across the life span. ASD is a life-long disorder, and it is well accepted that social attentional abnormalities do not simply disappear after infancy and childhood. As described in this chapter, social attentional abnormalities are present into adulthood in ASD.

Thus, it is essential to understand specifically how altered attention in adolescents and adults with ASD may continue to impact their behavior, cognition, and brain functioning. Some social abnormalities may be a consequence of early attentional abnormalities interacting over the course of development (e.g., lack of expertise for faces), but some of these differences may simply reflect persistent and atypical attentional processes at a particular moment in time (e.g., missing an important social cue about someone's emotional state).

There is evidence that at least some of what is claimed to be abnormal in terms of behavior, cognition, and brain activity in individuals with ASD may be accounted for by the latter. More specifically, altered patterns of attention in the moment may transiently influence behavior, cognition, and brain activity, but not reflect a more persistent dysfunction. Indeed, this is a major component of many interventions targeting social skills in adolescents and adults with autism—what are the important socially relevant cues/information in the immediate environment, and how should these cues be attended to and interpreted?

In this chapter, we focus on aberrant social attention as it is manifested in adolescents and adults diagnosed with ASD. We first review the literature detailing behavioral differences in social attention in individuals with ASD, and go on to discuss possible functional consequences (i.e., social deficits) that may be attributed to disrupted social attention. We also discuss some of the research on the neural correlates underlying atypical social attention in ASD, with a particular focus on several regions involved in social attentional processes. We conclude by highlighting some outstanding questions and discussing future promising directions for research on social attention in ASD.

7.2 Eye Tracking as an Indispensable Tool for Social Attention Research

Evidence for altered social attention was presented alongside the first clinical description of ASD. Kanner described children with ASD as appearing to be oblivious or indifferent to people, failing to look at both physicians and family members alike (Kanner, 1943). However, some of the first quantifiable experimental evidence of social attentional abnormalities came decades later. Langdell (1978) showed familiar faces to ASD and control individuals that were only party visible (i.e., just the eyes, nose, mouth, or hair). Participants were then asked to identify the familiar person from this impoverished image. Because the ability of individuals with ASD to recognize familiar individuals by their mouths alone was superior to controls, Langdell concluded that children with ASD must be looking at and attending to faces differently in their everyday lives (Langdell, 1978).

While measures of accuracy and reaction time can be used effectively to infer social attentional processes, as shown by Langdell, a perhaps more straightforward way to determine an individual's focus of attention is to measure where they choose to look. Eye tracking is a methodology/technology that can do just that, and has proven to be an extremely useful tool for investigating patterns of typical and atypical social attention. Most modern eye tracking relies on infrared light and high-speed sensors that can noninvasively track the movement of eyes at high sampling rates (ranging from 30 Hz to 2000 Hz).

Since we tend to look at what we are interested in and wish to attend to[1], eye tracking provides us with a near real-time insight into cognitive processes via a rapid, accurate, and largely automatic motor response. This is in contrast to other behavioral measures that often require explicit instructions, training, deliberate conscious motor control, or self-insight (e.g., responding via button presses or self-report measures). Furthermore, because eye tracking merely requires a participant to sit relatively still and look at a screen, it can and has been used across ages and levels of cognitive functioning, allowing scientists to test and draw comparisons across a wide range of participants. Given that social attentional abnormalities are thought to be a significant core feature of ASD, eye tracking has become an indispensible tool in this field of research. While the majority of the studies we describe in this chapter use eye tracking as a primary methodology, other non-eye tracking-based experiments will also be discussed because other approaches, like that used by Langdell, can also reveal novel and important insight into social attention in ASD.

[1] While we can dissociate our eye movements from the focus of our attention (von Helmholtz, 1909/1962; Posner et al., 1980), generally our attention corresponds to our overt eye movements.

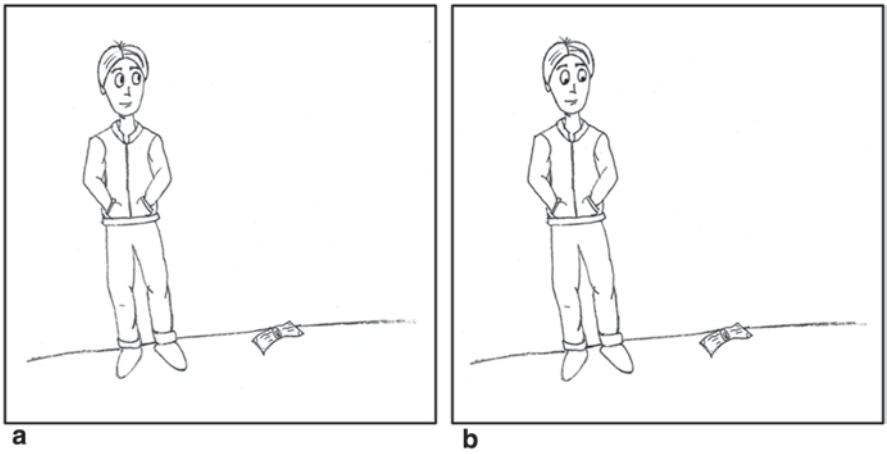

Fig. 7.1 *The eyes within a face can communicate one's attention and mental state.* The only difference between **a** and **b** is the location of the person's eye gaze. From drawing **b**, we might infer that the person is aware of the dollar on the ground, and from this we may predict that he will bend down to pick it up, a prediction we may not make based on drawing **a**. (Image courtesy of Susannah Burkholder)

7.2.1 Attention to Faces and Eyes in ASD

Faces are a key ingredient of social perception and interaction. A large amount of nonverbal social communicative information is conveyed by faces, and disproportionately so by the eyes in particular. Movement of muscles within the face can communicate complex emotional states, indicating a variety of emotional or mental states, including happiness, sadness, surprise, fear, and so on (Duchenne, 1862/1990; Darwin, 1872; Ekman, 1993). Beyond emotional expressions, the eyes within a face can communicate the target of one's attention, and give insight into what a person may be thinking about or planning to do next (see Frischen, Bayliss, & Tipper, 2007 for review). For example, if someone is looking at a dollar bill on the ground, we might infer something about their mental state—e.g., that they are aware that the dollar is on the ground—and predict something about their future behavior—e.g., that they may bend down to pick it up (Fig. 7.1).

Given the richness of social information derived from faces, it should come as no surprise that individuals are drawn to faces, and disproportionately so to the eye region. This is seen at the earliest stages of life (Goren et al., 1975; Maurer & Salapatek, 1976) and persists over development into adulthood. However, this drive to attend to faces and eyes seems to be weaker in individuals with ASD, as noted in the clinical descriptions of ASD (Kanner, 1943; Adrien et al., 1993; Clifford, Young, & Williamson, 2007), and further detailed in experimental tasks described below.

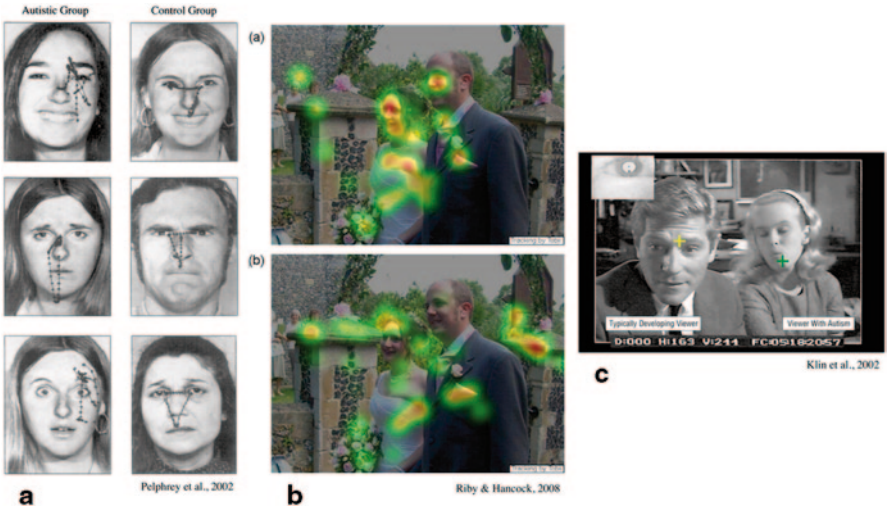

Fig. 7.2 *Differences in eye movements between ASD and NT individuals across several studies*. **a** Individuals with ASD spent a smaller proportion of time examining core features of the face (e.g., eyes, nose, and mouth; Pelphrey et al., 2002). **b** Compared to the controls, the ASD group spent less time looking at faces in complex scenes (warmer colors indicate longer fixation duration; Riby & Hancock, 2008). **c** Individuals with ASD fixated less on eyes and more on the mouth region compared with controls (Klin et al., 2002). The fixation point of an ASD viewer is in *green*, and an NT viewer is in *yellow*. Reprinted with permission

Pelphrey and colleagues (2002) were one of the first to address the question of how adults with ASD look at human faces. They used eye tracking to compare the locations of gaze between ASD and neurotypical (NT) adult controls when viewing pictures of faces portraying basic emotions (i.e., happy, sad, angry, disgusted, and afraid). Regardless of task demands (either freely viewing the image or identifying the emotion portrayed by the face), qualitative differences in scanpaths between groups were evident (Fig. 7.2). Individuals with ASD devoted less time to the core features of the face (i.e., nose, mouth, and eyes), and this effect was particularly pronounced for the time spent fixating the eyes. In addition to differences in gaze, the ASD group performed worse in the emotion identification task, more often confusing anger with fear. The link between emotion recognition and gaze to core features of the face was not assessed in their study, perhaps due to the relatively small sample size. Yet, a relationship between the two seems quite plausible, and this has been tested more directly in subsequent studies (e.g., Corden, Chilvers, & Skuse, 2008), which we discuss later on. It is important to note that all tested participants had IQs in the normal range, suggesting that the findings could not be attributed to a difference in general cognitive ability.

In another early study, differences in gaze were also found when adolescents and young adults with ASD viewed dynamic social stimuli (Klin, Jones, Schultz, Volkmar, & Cohen, 2002). Participants in this study watched clips from the movie *Who's Afraid of Virginia Woolf*, a black and white film characterized by intense emotions

and social interactions among the characters. The choice of using a dynamic video as a stimulus was a purposeful one and well motivated. The dynamic nature of videos and the associated visual and social complexity of this particular stimulus better reflect the demand characteristics of social situations encountered in the real world (and represents the type of stimuli that individuals with ASD often find challenging to understand). Klin and colleagues found that the groups showed quantifiable differences in how they looked at the video—adults with ASD spent less time looking at the eyes, and more on the mouth, body, and background/object regions, compared with age and verbal IQ matched controls. Consistent with the findings from Pelphrey et al. (2002), the amount of time spent fixating the eye region was the best predictor of group membership (Klin et al., 2002).

These atypical gaze patterns persist even when individuals with ASD are viewing images of familiar faces (Sterling et al., 2008). In this study, ASD and NT adults matched on age and IQ passively viewed familiar (e.g., family and friends) and unfamiliar faces. It was originally hypothesized that the ASD group would exhibit more normative gaze due to a heightened motivation to look at emotionally salient and familiar people. Overall, the NT group spent a greater proportion of time looking at the eyes regardless of familiarity, and made significantly more fixations to the eyes when looking at unfamiliar faces compared with familiar faces. In contrast, the ASD group did not display patterns of gaze that indicated any distinction between familiar and unfamiliar faces, and exhibited atypical attention regardless of familiarity.

It should be kept in mind that the majority of studies of visual attention in ASD, with several notable exceptions such as the study by Klin and colleagues (2002) described above, have utilized visually cropped and static faces in isolation, without a visible body or any other type of context. Thus it is possible that results derived from such studies are more informative specifically regarding face processing, rather than a broader reflection of the allocation of attention to social information more generally (Sasson, 2006). Faces are often considered to be a special category of stimuli (Kanwisher, McDermott, & Chun, 1997), and a face in the absence of any social context may not reflect how one might view that same face in a more embedded, naturalistic context in which they typically are encountered. This naturalistic context poses additional processing demands, notably including selection of the relevant parts of the scene, which we think perhaps better reflects the clinical descriptions of altered social attention in ASD. For example, Kanner (1943) did not note that the children failed to look at his eyes, but rather that they failed to look at his face. (As an aside, without eye tracking it is actually very difficult to know exactly where on your face someone is looking, whether it is at the eyes, nose, eyebrow, mouth, etc.) Therefore, instead of asking how an individual scans the features of a face, one can use eye tracking to ask the question of whether or not faces attract attention in the first place. While it is of course important to understand how one views the various internal and external features of a face, equally important, if not more relevant to ASD, is to also examine the *selection* of faces in the context of competing stimuli (e.g., background, objects, etc.).

Along with the work by Klin et al. (2002), some other studies have attempted to mirror the complexity of real social environments by presenting scenes depicting people within natural environments surrounded by competing visual stimuli. Here, one can ask whether individuals with ASD show attentional selection for faces, when faced with competing choices of what to attend to within a scene. Research measuring eye movements in NT adults viewing social and nonsocial scenes has found that they show a strong attentional preference for social scenes (Fletcher-Watson, Findlay, Leekam, & Benson, 2008). Fletcher-Watson et al. (2009) replicated this initial study with a group of high functioning ASD (HFA) and NT adolescents and adults. Participants were shown pairs of scenes, each consisting of a "person-present" scene and a "person-absent" scene. The results failed to show an overall group difference in attentional preference, with both groups showing a strong bias to look at the person-present scene and particularly the person in the scene. However, while the NT group exhibited this bias in the first fixation, the ASD group failed to show a preference for the social element of the scene in the first fixation, suggesting reduced social attentional priority. Thus, while faces still attracted the attention of individuals with ASD, faces were less salient, as indicated by the lack of social attentional bias in the first fixation. How this reduced saliency plays out in real-world contexts and affects social and emotional comprehension remains less clear. Additionally, while some of these above described effects appear to be subtle in adults, preference for nonsocial stimuli may be more pronounced in younger children with ASD (Klin, Lin, Gorrindo, Ramsay, & Jones, 2009; Pierce, Conant, Hazin, Stoner, & Desmond, 2011), suggesting differences in patterns of visual attention and preference may manifest in different ways over development. This suggests differences in patterns of visual attention and preference manifest in different ways across development, with the effects being more subtle but still detectable in older individuals with ASD.

Stimulus valence may also interact with attention in adults with HFA. There is an evolutionary advantage to rapidly processing threatening stimuli, as they provide relevant warning signs of imminent danger. They elicit an immediate fear response and direct attention toward the source of danger (Vuilleumier & Schwartz, 2001). This process has been termed the "threat detection advantage" (Eastwood, Smilek, & Merikle, 2001). Not surprisingly, attentional priority for threatening faces is evident beginning in childhood in typical development (Santos, Silva, Rosset, & Deruelle, 2010). Santos and colleagues (2012) measured spontaneous orienting behaviors in individuals with ASD to investigate whether they also show a threat detection advantage. Adolescents and adult participants were presented with pairs of emotional, either negative or positive, and neutral social scenes (i.e., positive emotional–neutral, negative emotional–neutral, or neutral–neutral pairs). While the NT groups showed a strong preference and displayed longer first fixation durations for the negative scenes over the positive or neutral scenes, the study failed to reveal initial preferential orienting toward socially threatening scenes in the HFA group. However, the HFA group did adapt their viewing behavior over time and reached typical patterns at later stages of viewing, reflected by a greater total number of

fixations on the negative emotional scene in comparison, similar to the patterns observed in the NT group.

It is worth emphasizing that both the Santos et al. (2012) and the Fletcher-Watson et al. (2008) studies found no overall group differences between ASD and NT when using average fixation duration as a measure of social attention. The differences in both studies are far subtler than the studies discussed previously, in that the differences only emerge in the first few moments following stimulus onset. Despite the differences being short lived, these still may translate into social difficulties in the real world—perhaps an individual with ASD misses a fleeting emotional expression, or misses a quick glance indicative of a person's interest. Given that social information in the real world can be very brief, any impairment in initially orienting to or paying attention to these social cues could have debilitating effects on one's success in social interactions. However, how these small differences in social attention relate to real-world social impairment has yet to be demonstrated.

Findings that demonstrate atypical social orientation as presented thus far may seem robust and largely consistent. However, they have not gone unchallenged, and replication across studies has been an issue. For example, van der Geest and colleagues (2002a) were not able to replicate the findings of Pelphrey et al. (2002). HFA and typically developing children matched on age and IQ were shown faces depicting basic emotions while eye movements were recorded. Unlike previously discussed findings, the results showed both groups behaving very similarly, fixating on the eye and mouth regions longer than other parts of the face, with the first fixations of both groups directed to the eyes. In another study, children with ASD also showed similar preferences to human figures over objects in a complex cartoon scene (van der Geest, Kemner, Verbaten, & Van Engeland, 2002b). Furthermore, Fletcher-Watson et al. (2008) found no evidence of overall group differences in fixation time on the eyes between young adults with ASD and controls. And, individuals with ASD have been found to be just as effective in detecting nonsocial changes as NT controls in a change blindness task (Fletcher-Watson et al., 2006). It was hypothesized that NT individuals would show reduced reaction times in detecting changes in eye-gaze direction given their superior attentional bias toward social information. Contrary to their hypotheses, however, individuals with ASD showed very similar abilities in detecting eye-gaze changes as controls.

What conclusions can we draw about social attention in ASD based on these discrepancies? There are a number of possibilities here, including differences in participant demographics, differences in the nature of the task and its demands, and differences in the nature of the stimuli themselves. For example, while the participants in the Pelphrey et al. (2002) and Klin et al. (2002) studies consisted of male adolescents and young adults (mean ages of 25.2 and 15.4 years, respectively), the two van der Geest et al. (2002a, b) studies tested school-aged male children (mean age of 10.6 years). It is possible that discrepant findings could indicate developmental changes in gaze behavior, not only in individuals with ASD but also in NT individuals. It has been demonstrated, for instance, that qualitative differences in

patterns of attention can emerge during development. For example, Nakano et al. (2010) found that typical infants prefer to watch mouths over eyes during speech, but that this preference reverses with development (see also Schulz et al., Chap. 6, this volume). The same study also found the time spent viewing the mouth relative to face to be significantly longer in NT children compared with NT adults and adults with ASD, but no differences were found between NT and children with ASD, suggesting these differences only emerge later in development. In addition, control participants showed more standardized gaze behaviors, more likely exhibiting similar gaze patterns, compared with ASD participants. Given the fact that the studies discussed above had relatively small sample sizes, the results may also be strongly influenced by the heterogeneity in gaze behaviors among individuals with ASD.

In addition to differences in subject characteristics, some of the studies discussed used static photos of faces (Pelphrey et al., 2002; van der Geest et al., 2002a), while others used complex and/or dynamic stimuli containing multiple people (Klin et al., 2002), or static cartoon depictions of people and objects as stimuli (van der Geest et al., 2002b) to test differences in social attention. It is possible that differences in the type of stimuli used contribute to these discrepant findings across studies. To address this possibility directly, Speer and colleagues (2007) compared the effects of stimulus content and stimulus type on gaze patterns. Each ASD and NT participant was shown four different types of stimuli: social dynamic, social static, isolated dynamic, and isolated static. The social dynamic stimuli consisted of video clips of highly emotional interactions among two or more characters. The isolated dynamic stimuli were also videos, but only depicted one individual. The social static and isolated static stimuli were static images of either two or more people, or just one person, respectively. Interestingly, group differences were only statistically different between the two groups in the social dynamic condition. Though it seems that statistical power issues may account for the lack of differences in these other conditions, individuals with ASD spent less time looking at the eyes than the NT group and more time looking at the body, replicating previous results (Klin et al., 2002). The authors concluded that differences in social orienting and face processing associated with ASD might at least be partially dependent on the stimuli being both realistic and social, since when either one variable was missing, the ASD group did not perform significantly differently from those in the control group. However, it is worth raising the caution here that it seems that statistical power issues could have accounted for lack of differences in these other conditions. A more recent study also found that spontaneous allocation of attention to faces, and more specifically the eyes, is influenced by the way in which the faces are presented (Hanley, McPhillips, Mulhern, & Riby, 2013). This study varied stimuli based on the degree of social context presented, whether faces were presented in isolation or within a social scene, as well as whether the social interaction depicted was acted or naturally occurring. Their results demonstrated that both adolescent and young adults with ASD and NT controls attend similarly to isolated faces. However, when faces were presented within a social context, with additional information competing for attention, ASD participants showed abnormally reduced fixations to the eyes, while

the NT group prioritized the visual information from the eyes. Therefore, atypicalities in the ASD group were more evident when viewing more visually and socially complex images, highlighting ecological validity as an important experimental parameter to consider in research involving this population.

7.2.2 Attention to Eye Gaze in ASD

The studies discussed thus far highlight the importance of the eyes in faces, and how salient they seem to be to NT individuals compared to those with ASD (Klin et al., 2002; Pelphrey et al., 2002). Eyes, in addition to providing valuable information about the target's emotions or identity, can also help identify another person's intentions or alert of potential threats in our environment (Ristic et al., 2005; Hadjikhani, Hoge, Snyder, & de Gelder, 2008). For instance, eyes making direct eye contact might signal anything from aggression to romantic interest, while eyes directed toward a particular location might indicate the focus of a person's attention and signal that something is of interest in that location. Given the communicative and social importance of the direction of one's eye gaze, it comes as no surprise that NT individuals are highly attentive and sensitive to changes in gaze direction. Following one's direction of gaze (i.e., gaze following) can be a largely automatic and spontaneous process. For example, 9-month-old infants are sensitive to the relationship between gaze direction and location of objects, looking longer at a face that gazes toward an object compared with those looking in the opposite direction (Senju, Csibra, & Johnson, 2008). In fact, as discussed in other chapters (Bertenthal & Boyer, Chap. 2, this volume), gaze following emerges at the earliest stages of development (2–5-day-old newborns) (Farroni, Massaccesi, Pividori, & Johnson, 2004).

Given the findings that individuals with ASD exhibit reduced fixation on the eyes, one might predict that these atypicalities translate into difficulties in utilizing gaze information in the typical manner (see Frischen et al., 2007 for a review on visual attention to gaze in NT individuals). Being able to follow the gaze of an individual is thought to be a foundational behavior for a number of important domains of functioning. For example, gaze following behaviors at 10–11 months are strongly and positively correlated with subsequent language development at 18 months in typically developing infants (Brooks & Meltzoff, 2005). Furthermore, joint attention skills at 20 months have been positively correlated with the ability to understand the minds of others (i.e., mentalizing or theory of mind (Baron-Cohen, 1995)) at 44 months in typically developing infants (Charman et al., 2000). Joint attention, language, social communication, and theory of mind are all domains that are affected in ASD (American Psychiatric Association, 2013; Baron-Cohen, Leslie, & Frith, 1985; Happé, 1995), further leading researchers to suspect altered attention to, and utilization of, eye gaze of others in ASD.

Many researchers have argued that children with ASD indeed show deficits in the processing of gaze. For example, children diagnosed with ASD show reduced gaze-monitoring (i.e., looking in the same direction that an adult is looking in) at

18 months of age (Baron-Cohen et al., 1996) and show impairments in joint attention (Leekam, Baron-Cohen, Perrett, Milders, & Brown, 1997), suggesting a relative insensitivity to direction of gaze. Despite these early differences, other studies have found that children with ASD do show reflexive orienting to gaze direction (Swettenham, Condie, Campbell, Milne, & Coleman, 2003; Chawarska, Klin, & Volkmar, 2003). Using a modified spatial cueing paradigm (Posner, Snyder, & Davidson, 1980), Swettenham et al. (2003) asked HFA and chronological age-matched typically developing (TD) children to press a key as quickly as possible when they detected a target on the screen. Prior to the target presentation, participants would see a person's face whose eyes would look either to the left or to the right. The target would then appear randomly on either side of the face, congruent or incongruent to the gaze direction of the person. Despite the fact that the gaze direction did not predict the target's location and that participants were explicitly told this, neither group was able to ignore the gaze cues, as demonstrated through faster responding to the congruent cues compared with the incongruent cues. Chawarska et al. (2003) found comparable results in an experiment using a similar paradigm in 2-year-old children. Both toddlers with ASD and age-matched TD children exhibited shortened saccadic reaction times to the congruent target, suggesting reflexive eye movements brought on by the direction cue of the person's gaze.

However, there is one important consideration to keep in mind when interpreting these findings—the stimuli used in these studies included faces without bodies or any other visual information in the background competing for the viewer's attention. This situation is vastly different from social stimuli that individuals normally encounter, which are embedded within complex, dynamic, multimodal, and less predictable environments. Given the data suggesting individuals with ASD tend to increasingly display abnormal gaze patterns when viewing more ecologically valid stimuli (Speer et al., 2007; Hanley et al., 2013), it is possible we may discover differences between groups as we move away from isolated social stimuli.

To address this possibility, Freeth et al. (2010) investigated whether fixation patterns of individuals with ASD are affected in the same manner as NT controls by the eye-gaze direction of an observed person when viewing a complex social scene. Adolescents with HFA and adults and NT controls, matched on IQ, viewed photos of an everyday setting containing one person and multiple objects. The person in the photo looked either straight ahead or at an object on the opposite side of the scene. The objects present in the photos were located either at the exact location of gaze, on the same side of the photo as the gaze but not at its actual location, or on the opposite direction of the gaze. Similar to previous findings (Fletcher-Watson et al., 2009), no overall group differences were found in the proportion of time spent fixating on the face compared with objects or bodies. However, faces seemed to be more highly prioritized by the NT group compared with ASD, as indicated by quicker first fixations to the face when the scene was initially presented. In contrast, individuals with ASD were faster to first fixate on objects, but later adjusted their gaze to look at faces. Both groups were cued by the gaze direction of the person,

replicating studies of younger children and toddlers (Swettenham et al., 2003; Chawarska et al., 2003).

However, another way of looking at the influence of gaze direction of visual attention is also to examine the consequences of such gaze, not just on how viewers saccade toward the object, but how they prioritize that object once fixated. In the same study by Freeth and colleagues (2010), NT individuals showed an immediate increase in fixation duration on the object that the person in the photo was looking at, while this pattern was not observed in the ASD group. This result suggests that while adolescents with HFA are able to follow gaze direction, the significance of the gaze cue in influencing their processing of the scene is different. Riby et al. (2013) further tested this idea by additionally asking participants to explicitly identify the object being gazed upon. Using a similar paradigm, ASD and NT individuals looked at pictures including one actor gazing at a target object among three different possible objects, and were asked to name the target object. Compared with a condition when participants freely viewed the stimuli, individuals with ASD increased their gaze at the person's face and eyes when asked to identify the target, indicating that they understood where to look for this information. However, while the NT group shifted their attention from the face to the correct target, the ASD group failed to follow the gaze of the person and continued to look at implausible objects, rather than the target, which resulted in more incorrect responses. These data suggest that the ability to use gaze reflexively is spared when individuals with ASD are not required to select gaze information within very simplified viewing situations. However, the deficit becomes clearer when gaze is embedded in a complex scene, as reflected by the inability to effectively use the information conveyed by gaze direction.

7.2.3 Additional Evidence for Social Attention Abnormalities in ASD

Eye tracking is an incredibly versatile methodological tool to study social attention in ASD, as evidenced by the numerous studies and diverse findings across differing experimental paradigms. However, there are other behavioral measures of social attention that have provided further evidence for disruption in social attention and information processing among older children and adults with ASD. One paradigm in particular that has been widely used to study attentional differences in ASD is the change detection paradigm. In a change detection study, participants are presented with pairs of images that are nearly identical, with the exception of one part of the scene that is altered (e.g., color, presence or absence of an object, gaze direction, etc.) (Simons, 2000). The change is made especially difficult to detect by presenting the images in temporal succession, separated by a brief blank screen, rather than showing them both simultaneously (Rensink, O'Reagan, & Clark, 1997). This paradigm provides additional information beyond what eye tracking can show since it evaluates not only where, but also what specific features the participant is attend-

ing to (Simons & Rensick, 2005). For instance, an individual may be looking at an object, but not processing its color, size, orientation, smoothness, and so on. In the social realm, an individual may be looking at a face, but perhaps does not notice the eye-gaze direction, facial expression, identity, etc. Thus, compared with eye tracking, change blindness paradigms can, in certain cases, be better suited to address some questions regarding social (and nonsocial) attention.

As one example, Kikuchi et al. (2009) tested social and nonsocial change detection in ASD and TD school-aged children who were presented with pairs of photographs depicting multiple faces and objects in a naturalistic setting. A change was introduced in one of the photographs, replacing either a face or object within the image, and participants were asked to press a key when the change was detected. A previous study had shown a social change advantage in TD children (Humphreys, Hodsoll, & Campbell, 2005). However, given the eye tracking data pointing to reduced attention to faces in ASD, it was hypothesized the ASD group would not show this advantage. This was borne out in the results: TD individuals detected changes to faces faster than changes to objects, while children with ASD did not.

It is of course expected that faces would not be preferentially processed if attention and gaze were not preferentially allocated to them. However, NT adults have additionally been shown to have difficulties ignoring faces, suggesting that faces are not only processed preferentially, but also perhaps automatically, in a mandatory fashion (Farah, Wilson, Drain, & Tanaka, 1995). Remington, Campbell, & Swettenham (2012) examined the degree to which faces capture attention in ASD and NT adults by simultaneously imposing a perceptual load. The strong, automatic attentional bias for faces in NT individuals would predict that they would continue to be distracted by faces despite the increased perceptual load, whereas individuals with ASD would not show signs of interference. Participants were instructed to decide whether a name shown on the screen was male or female. To manipulate the perceptual load, the name was presented alone or among a list of a varying number of distractor nonwords. Additionally, a face was presented next to the list of words, which was either congruent or incongruent (same or opposite gender as the target name). Participants were explicitly told to ignore the faces. If the faces are being attended to, one would expect them to interfere with the name task, as indicated by a faster reaction time for congruent versus incongruent trials. Regardless of perceptual load, the NT group showed congruency effects, suggesting they were unable to ignore the faces despite their irrelevance to the task at hand. While this effect was evident during lower perceptual load conditions in the ASD group, it was not found during higher load conditions. The differing congruency effect with regard to load in ASD is intriguing, as it indicates that individuals with ASD only processed the faces when task demands were low. In other words, the face processing bias seems comparatively weak in ASD, especially in certain situations, though not necessarily absent.

In addition to attention to faces in general, evidence of altered attention to eye gaze in ASD was found in a recent study by Pellicano, Rhodes, and Calder

(2013). This study examined the ability of the gaze perception system to adapt to prolonged exposure to a particular gaze direction. A similar study conducted with NT adults showed that prolonged exposure to faces with eyes averted 25° leftward resulted in a tendency to judge subsequent faces with leftward gaze as looking forward (Jenkins, Beaver, & Calder, 2006), which is referred to as a perceptual aftereffect (i.e., distortions following exposure to a stimulus). Children with ASD and NT controls (9–14 years of age) were asked to first complete a gaze acuity task where they categorized the direction of gaze of faces with eyes averted 10° or 5° left, straight ahead, and 10° or 5° right. Next during the adaptation phase, participants observed a series of faces showing gaze averted 25° in a single direction (left or right) for 1.5 minutes. During the post-adaptation phase, participants completed a second gaze acuity task, with the adapting image showing gaze in the adapted direction preceding the test image to maintain adaptation effects. Controls displayed an increased tendency to perceive gazed averted in the adapted direction as looking straight-ahead, replicating previous findings (Jenkins et al., 2006). While the ASD group also showed these aftereffects, the effects were significantly reduced relative to controls, which the authors interpret to suggest an attenuated calibration of others' gaze. Furthermore, the ASD group showed reduced accuracy in categorizing subtle deviations in gaze direction compared with controls during the pre-adaptation phase, and accuracy was related to the magnitude of gaze aftereffects in ASD. The results suggest not only subtle atypicalities in gaze perception in general, but also that the mechanisms underlying gaze processing may be less flexible in ASD.

7.3 Functional Consequences of Aberrant Social Attention in ASD

So far we have focused on behavioral studies characterizing atypical social attention among individuals with ASD. While there are some discrepancies in findings (e.g., fixation time to mouth versus eyes), generally individuals with ASD appear to show differences in attention to various social stimuli. But why does this matter? There is an important question that we have not yet addressed: Namely, how do abnormalities or deficiencies of social attention contribute to the ASD phenotype? In other words, what are the functional consequences? As described earlier, it has been hypothesized that a disruption of early social attentional mechanisms constitutes a primary deficit in ASD, with detrimental downstream consequences for social and language development (Chevallier et al., 2012). Thus, one might expect to see reduced or abnormal attention to faces together with impaired processing of faces. Below, we discuss separately how individuals with ASD process two types of social information; namely, facial identity and emotional expression.

When taking verbal IQ into account, individuals with ASD are relatively able to recognize highly familiar faces (Wilson, Pascalis, & Blades, 2007). Impair-

ment in face recognition becomes more evident when remembering and discriminating unfamiliar faces (Boucher & Lewis, 1992; Klin et al., 1999; Blair, Frith, Smith, Abell, & Cipolotti, 2002). Blair et al. (2002), for example, examined the facial recognition memory of adults with ASD and two control groups matched on either age or verbal IQ. Participants were administered a standardized memory recognition test, involving previously unfamiliar pictures of faces and buildings. The ASD group exhibited significantly poorer visual recognition memory for unknown faces compared with both comparison groups. Yet, the ASD group showed intact memory for buildings, suggesting a domain-specific memory impairment for faces. Findings of a circumscribed facial recognition impairment for unfamiliar faces have also been observed across ages, not attributable to general cognitive deficits, memory deficits, or task demands (Klin et al., 1999; Boucher & Lewis, 1992).

Many have also reported reduced accuracy in identifying emotions in adults with ASD (Ashwin, Chapman, Colle, & Baron-Cohen, 2006; Wallace, Coleman, & Bailey, 2008; Corden et al., 2008; Harms, Martin, & Wallace, 2010; Kennedy & Adolphs, 2012). Adults with HFA when compared with matched controls performed significantly worse overall in a task involving the recognition of basic emotions (happiness, fearful, disgust, anger, sadness, and surprise) (Ashwin et al., 2006). In this particular study, the emotion recognition deficits, however, were limited to negative emotions—fear, disgust, anger, and sadness—with no group differences in identifying happiness, surprise, or neutral expressions. These results were also replicated by Wallace, Coleman, and Bailey (2008), who found that adults with HFA showed a general impairment in recognizing basic expressions, with a particular impairment in recognizing fear, disgust, and sadness. This group further examined emotion recognition by gradually revealing the number of facial features available to the participant. Here, the participants viewed either the mouth or eyes alone, a nose, or all three features (eyes, nose, and mouth) during the task. While the NT group was significantly more accurate in recognizing fear from the eyes than the mouth, the ASD group showed no advantage in the eyes-only condition, frequently misidentifying fear as anger.

Eye tracking data simultaneously collected during a similar emotion recognition task further revealed the importance of the eyes in the recognition of fear in particular (Corden et al., 2008). This study found adults with ASD to display difficulties in recognizing fearful and sad faces, with a trend toward an effect for angry faces. The ASD group generally spent less time fixating the eye region compared with controls, consistent with the previous findings reviewed earlier (Klin et al., 2002; Pelphrey et al., 2002). The degree of fear recognition impairment in the ASD group was also predicted by their time fixating away from the eyes throughout the experiment. In other words, time spent fixating on the eyes was positively correlated with fear recognition accuracy.

Adding further support for different strategies of emotion recognition, Spezio et al. (2007a) found individuals with ASD gaze at, and utilize, information from faces differently from controls. This study used the “Bubbles” method (Gosselin

& Schyns, 2001) to further understand the relationship between how people use information from different regions of the face when making emotion judgments. With this method, faces are largely masked except the parts of the face underlying the location of randomly placed and different sized circles (i.e., Bubbles), with each size of bubble corresponding to a different spatial frequency. The participant is then shown this degraded, bubbled image and the viewer is instructed to make a judgment based on what they see. Using reverse correlation, one can then determine which parts of the image were shown when participants were able to make a correct compared with an incorrect response in order to determine what information was used for successful task performance. Spezio et al. (2007a) tested adults with HFA and controls matched on IQ and revealed that individuals with ASD are strongly distinguishable from controls in the features they rely on while making emotion judgments. The HFA group utilized the information from the eye regions significantly less while also showing a marked increased reliance on information from the mouth compared with controls. This difference in strategy is particularly interesting given that, in this study, the groups showed no differences in overall performance in emotion recognition.

This was not the only study, however, to fail to find performance differences in recognizing facial expressions in ASD—in fact, many other studies have also failed to find such differences in the recognition of basic emotions in both children and adults with ASD (Baron-Cohen, Spitz, & Cross, 1993; Grossman, Klin, Carter, & Volkmar, 2000; Adolphs, Sears, & Piven 2001). Other studies have found that even if such differences exist, they can be somewhat subtle and require more sensitive methods and measures to detect any abnormality (Kennedy & Adolphs, 2012). In particular, it seems that discrepancies across studies may at least partially depend on task demands, with the lack of effects reflecting possible ceiling effects. Grossman et al. (2000), for example, found similar performance (both in accuracy and in reaction time) between HFA and NT adults when participants were simply asked to identify the emotion depicted in a photograph. However, differences were observed in these same participants during a more complex task where they viewed emotional faces paired with an emotional label that either matched the expression (e.g., happy face with the word "happy") or mismatched (e.g., happy face with the word "afraid") and were asked to identify the emotion of the person. In this condition, only the ASD group's accuracy was negatively affected by the mismatched verbal label. These results provide evidence for a qualitative difference in the processing of emotions by individuals with ASD.

Clark, Winkielman, & McIntosh (2008) utilized a different method to increase task demands—namely, by reducing the exposure duration of the face stimuli dramatically. HFA and NT adolescents and adults were briefly shown pairs of emotional faces, neutral faces, and non-faces. In the Emotion condition, participants decided which face was happy or angry, in the Neutral condition they decided on the gender, and in the Non-Face condition they identified which image was an object or an animal. The stimuli were only presented for 15 ms or 30 ms, which previous studies have shown is long enough for NT adults to extract valence of faces (e.g.,

Murphy & Zajonc, 1993). While both groups performed similarly in the Neutral and Non-Face conditions, the ASD group performed worse during the Emotion condition, suggesting a specific impairment in the ability to rapidly extract emotion information from faces.

Together, these findings point to a possibility that higher-functioning individuals with ASD perhaps utilize compensatory mechanisms to decode emotions—one that is less automatic and slower. When faced with more demanding situations, these compensatory strategies fail, thus exposing their impairments. This may also explain the discrepancy researchers often observe—surprisingly intact performance on various laboratory tests assessing emotion perception, but clear deficits in real-world interactions. Given the studies reviewed above, one explanation may be that real-world interactions qualitatively differ from typical laboratory tests in a number of ways; for instance, the real world is highly dynamic, less structured and less predictable, more interactive (i.e., may require a response), and highly context dependent, with fleeting social cues, etc. In essence, identifying facial expressions in the real world is perhaps more similar in some ways to the more demanding experiments described above, precisely where the deficits in individuals with ASD become more apparent.

Furthermore, assessing emotion recognition abilities in ASD should extend beyond the study of the basic emotions, because our repertoire of generating and decoding facial expressions extends well beyond the basic emotions (happy, sad, fear, surprised, disgust, anger, contempt, along with neutral). Often times we are faced with the need to identify complex expressions that may reflect more of a mental state (e.g., troubled) and/or social emotion (e.g., embarrassment, flirtatiousness), both of which are subtler than the basic emotions. One widely used task is the "Reading the Mind in the Eyes" task (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001), where a participant is shown a picture of a person's eyes and asked to identify the social emotion that best describes their perceived mental state (e.g., annoyed, ashamed, distrustful, and interested). Multiple studies utilizing this task have shown individuals with ASD perform worse than matched controls (Baron-Cohen et al., 2001; Kaland, Callesen, Møller-Nielsen, Mortensen, Smith, 2008; Holt et al., 2014; Schuwerk, Vuori, & Sodian, 2014). More recently, tasks using complex videos rather than static, isolated images of eyes have been used to assess the recognition of complex social emotions in ASD (Dziobek et al., 2006; Golan, Baron-Cohen, Hill, & Golan, 2006). Golan et al. (2006) had adults with HFA and matched controls view clips of videos depicting dramatic emotional interactions between multiple people and asked participants to identify the complex emotion (e.g., smugness, awkwardness) of a character in the scene. Consistent with previous studies utilizing static images, the ASD group performed significantly worse than controls.

Taken together, these results suggest that while recognition of basic emotions may be preserved in some individuals with ASD, adults with ASD may continue to

exhibit difficulties with recognizing more subtle and complex social emotions (see Harms et al., 2010 for review; Golan & Baron-Cohen, 2006).

7.4 Neural Correlates of Altered Social Attention

The broad range of behaviors and affected domains that characterize ASD suggests there perhaps is a diverse set of neural systems that is responsible for the ASD behavioral phenotype (Schultz, 2005; Amaral, Schumann, & Nordahl, 2008). Beyond visual attention differences to social stimuli, individuals with ASD, not surprisingly, have difficulties making judgments based on social stimuli (e.g., face processing, theory of mind). It is not difficult to see that the ability to efficiently and accurately make various social judgments is crucial to successfully function in our vastly social world. In particular, the fundamental role of face processing in guiding social interactions has led to the initial hypothesis that abnormalities in the neural circuitry involved in face processing contribute to social dysfunction in ASD (Schultz et al., 2003). Within the social attentional domain specifically, researchers have studied multiple regions that may be involved in the atypical behavioral findings we have discussed. In this section we review the current literature examining the possible neural correlates of atypical social attention in ASD, with a focus on three brain regions known to play important roles in processing facial identity, facial expressions, and gaze direction.

However, we should at the outset make it very explicit that although we are focusing on single brain regions in the following sections, this is largely due to the fact that until recently, research on ASD (and in cognitive neuroscience more generally) has been very brain region- and location-centric (Sporns, 2011). Yet, it is undeniable that any single brain region acts within a larger network context, and its specialization comes from the input it receives, the local computations it performs, and the output it sends—in other words, a brain region does not act alone and cannot be fully understood independently from other regions. Yet, the majority of research on autism to date that has examined neural correlates of social attention and emotional processing has focused on a few select regions that have been implicated in these processes. Methods to describe and make sense of network-level properties are being actively and rapidly developed (e.g., Bullmore and Sporns, 2009; Sporns, 2013; Bassett & Lynall, 2013), while at the same time behavioral links to network-level functioning are still quite sparse. Therefore, in the section that follows, we focus on the fusiform face area (FFA), the amygdala, and the superior temporal sulcus (STS), while we fully acknowledge the essential role that brain networks will play in ultimately understanding complex social behaviors and psychopathology (e.g., Uddin, Supekar, & Menon, 2010; Kennedy & Adolphs, 2012; Happé & Frith, 2014) (Fig. 7.3).

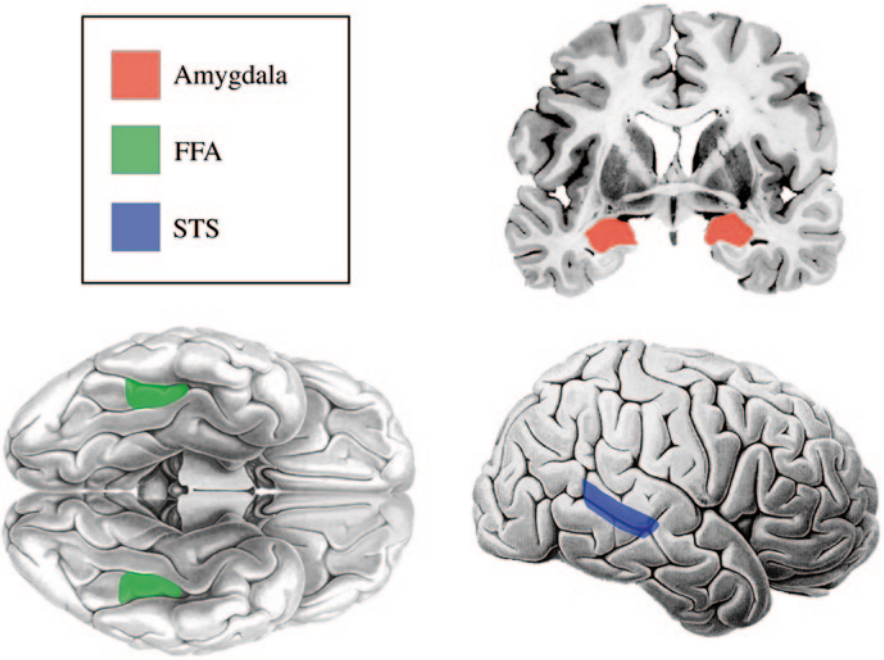

Fig. 7.3 *Three brain regions associated with atypical social attention in ASD.* Amygdala *(red)*, fusiform face area (FFA; *green)*, superior temporal sulcus (STS; *blue)*. Adapted from Kennedy and Adolphs (2012). Reprinted with permission

7.4.1 Fusiform Face Area (FFA)

Functional magnetic resonance (fMRI) studies with NT controls have identified a small region of the fusiform gyrus (FG) that is more strongly activated in response to face stimuli than other visual stimuli (Puce, Allison, Gore, & McCarthy, 1995; Kanwisher et al., 1997; Haxby, Hoffman and Gobbini, 2002). This area is known as the fusiform face area (FFA), and is identified functionally by contrasting FG activation when subjects are viewing faces versus non-face objects (e.g., houses). Its putative role in face perception (Kanwisher et al., 1997), has been further supported by convergent studies showing that lesions in this area result in prosopagnosia, or the inability to recognize faces (Damasio, Tranel and Damasio, 1990). However, whether or not the FFA is truly face-specific has been challenged by Gauthier and Nelson (2001), who argued that the region is responsive to categories of objects with which one has visual expertise. Support for this comes from studies showing this region to be engaged during bird or car viewing in individuals who are bird or car experts (Gauthier, Skudlarski, Gore and Anderson, 2000). This suggests that the functional specialization of the FFA may have evolved to discriminate between individual objects within a broader category, which are most likely faces for the majority of individuals. FFA activa-

tion in NT individuals is automatic in response to any face regardless of expression (Winston, Henson, Fine-Goulden, & Dolan, 2004) and lesions to this area do not appear to cause deficits in emotional expression recognition (Damasio et al., 1990), suggesting a specific role in processing facial identity. Regardless of whether the FFA is specialized for faces uniquely or more broadly sensitive to category expertise, it is undeniable that the FFA is a region critically involved in face processing in NT individuals. Given the abnormal gaze patterns to faces in ASD and difficulties with facial identity recognition, the FFA has been widely studied as one possible neural substrate that may be responsible for abnormal social attention (see Schultz, 2005 for review).

Schultz et al. (2000) conducted the first fMRI study to test whether ASD involves abnormal neural activation during face processing. First, regions of interest (ROIs) that activated in response to viewing objects and faces were identified in the right inferior temporal gyrus (ITG) and FFA, respectively, in a separate group of NT controls. The patterns of activations in adults with HFA and IQ-matched controls in these brain regions were then compared while participants were asked to decide if two pictures simultaneously presented were the same or different from each other. The pairs of stimuli included neutral faces (without hair or ears), various objects (e.g., cars, boats, chairs), or nonsense patterns. They found differences in the pattern of brain activation during face discrimination between ASD and NT groups. Specifically, consistent with prior studies with NT adults (Kanwisher et al., 1997; Haxby et al., 2002), the NT group showed activation in the right FFA during face discrimination, but this pattern was not seen in the ASD group. However, the ASD group showed abnormally increased activity in the ITG during face discrimination compared with the NT group, an area that was most strongly associated with object discrimination in NT controls. The authors suggested that this might mean that the ASD group processed faces more like objects, though such an interpretation based on reverse inference has to be considered with due caution. Thus, the results indicated that individuals with ASD exhibited not only reduced activity to faces in the expected brain region (i.e., FFA/FG), but also increased activation in an unexpected location (i.e., ITG) that is more specialized for processing objects. This finding is important because it demonstrates not just hypoactivation, but an altered pattern of responding.

Pierce et al. (2001) reported similar findings: HFA and NT adults matched on gender and age performed a face perception task, pressing a button in response to female faces, alternating with a shape perception task, during fMRI acquisition. The investigators chose three cortical ROIs: FFA, ITG, and the middle temporal gyrus (MTG), which has also been previously shown to consistently respond to objects more than faces (Allison et al., 1994). The ASD group did not differ from the NT group in terms of accuracy and response times on either face or object perception task. However, the ASD group showed either abnormally weak or no activation in the FFA in response to faces. But, this did not mean that the brain was simply unresponsive to faces. Rather, the ASD subjects in this study exhibited unique and non-overlapping functional maps in response to faces, some showing maximal response to faces in the frontal cortex, temporal cortex, occipital cortex, or the cerebellum.

The lack of spatial overlap between subjects resulted in an overall image showing no positive functional activity, suggesting individuals with ASD may process faces via unique neural circuitry. Hypoactivation of the FFA was subsequently replicated by multiple studies (e.g., Critchley et al., 2000; Hall, Szechtman, & Nahmias, 2003; Wang, Dapretto, Hariri, Sigman, & Bookheimer, 2004) and together these findings have led to suggestions that individuals with ASD fail to develop cortical face specialization, possibly due to reduced social interest or attentional deficits to faces (Pierce et al., 2001; Grelotti, Gauthier, & Schultz, 2002). Yet, we should keep in mind that ASD individuals do not exhibit profound face perception deficits to the degree that is found in prosopagnosia—i.e., most individuals with ASD are not prosopagnosic. So, what might these neural differences mean?

Neural responses to faces in NT individuals have also been found to be dependent on factors such as familiarity and emotional valence. For example, FFA activity is greater in NT individuals when looking at familiar faces compared with non-familiar faces (Henson et al., 2003), and greater in response to emotional faces than neutral faces (Vuilleumier, Armony, Driver, & Dolan 2001). To study if functional activation in the FFA is modulated by familiarity of faces, Pierce et al. (2004) presented ASD and NT adolescents and adults matched on gender and age with familiar faces (i.e., family and friends) and faces of strangers during fMRI data acquisition. The results were consistent with previous studies of NT individuals (Vuilleumier et al., 2001; Winston et al., 2004) showing greater FFA activation in response to familiar faces, compared with faces of strangers. And, while the NT group did show quantitatively greater FFA activation to unfamiliar faces than the ASD group, these differences were not statistically significant, in contrast with previous studies (Schultz et al., 2000; Pierce et al., 2001). The authors speculate that these findings indicate an enhanced motivation overall to attend to the faces due to familiar faces being dispersed throughout the task. Thus, previous findings of FFA hypoactivation may not reflect a true neurofunctional abnormality, but rather a social attentional difference that may be more apparent in some tasks (Schultz et al., 2000; Pierce et al., 2001), but not others (Pierce et al., 2004).

Given the eye tracking findings discussed in the previous section, one possibility is that the abnormalities in the way individuals with ASD visually attend to faces (Klin et al., 2002; Pelphrey et al., 2002) may very well be contributing to abnormal FFA activity. Hadjikhani et al. (2004) assessed cortical activation in response to face and non-face objects in ASD and NT adults matched on age and IQ. Participants were shown pictures of faces, objects, and scrambled pictures. Each stimulus had a red fixation cross in the center and participants were instructed to focus on the fixation cross to maximize the possibility that they would attend to the central area of the face. Individuals with ASD showed bilateral FFA, as well as inferior occipital gyri (IOG) activation in response to faces, similar to controls. Another area of the FG, medial to the FFA, which is referred as the fusiform object area (FOA) was activated in response to objects in both groups. In contrast to previously discussed studies (Schultz et al., 2000; Pierce et al., 2001) there was no evidence of areas abnormally recruited to process faces in the ASD participants. These results suggest

that findings of FFA hypoactivation in individuals with ASD may reflect a failure to attend appropriately to faces rather than a lack of cortical specialization. Introducing a fixation cross as well as instructions to maintain focus on it throughout scanning may have facilitated the participants' attending to the core features of the face. In addition, this study used passive viewing without an active task (i.e., same/different and gender discrimination), which the authors suggest may have prevented participants from using particular strategies to support task performance (e.g., focusing on peripheral features of the face in their efforts to discriminate faces). However, while this study was suggestive of gaze differences accounting for FFA hypoactivation in ASD, it did not directly test this hypothesis.

A subsequent study by Dalton et al. (2005) directly examined the hypothesized relationship between patterns of gaze fixation and brain activation during face processing, predicting that diminished gaze to the eye regions of faces may underlie FFA hypoactivation. ASD and NT adolescents and adults matched on age participated in two tasks during fMRI acquisition, a facial emotion discrimination task and a facial identity recognition task (familiar vs. unfamiliar faces), while being simultaneously eye tracked. Consistent with other work, the ASD group spent significantly less time fixating on the eyes than controls across tasks. Furthermore, for both tasks, the NT group showed significantly greater activation in the bilateral FFA in response to faces compared with the ASD group. Importantly, and as hypothesized, FFA activity was strongly and positively correlated with the amount of fixation to the eye region in the ASD group, suggesting that diminished fixation to the eyes in faces may account for this FFA hypoactivation to faces.

All together, the evidence regarding hypoactivation of the FFA points to the following: Individuals with ASD do not appear to exhibit a pure functional abnormality of the FFA. Rather, the FFA may be hypoactive in some situations due to reduced attention and/or altered gaze. These results, however, still do not provide us with answers regarding the question of what regions are then responsible for abnormal social attention in ASD. For this, we next move on to consider another region known to be important for social attention—the amygdala.

7.4.2 Amygdala

While the FFA is important in facial identity perception, the amygdala has been shown to be critical in early stage processing of facial expressions (Calder, Lawrence and Young, 2001; Morris et al., 1996), among other functions. The amygdala responds quickly to emotionally potent stimuli and its activity varies with valence of the stimuli (Morris et al., 1996; Schultz et al., 2000). Studies have shown that the amygdala is activated by fearful faces (Morris et al., 1996; Hariri, Mattay, Tessitore, Fera and Weinberger, 2003), but this activation appears to be particularly sensitive to the presence of fearfully widened eyes (Morris, DeBonis, & Dolan, 2002), even when processed without conscious awareness (Whalen et al., 2004). A patient with bilateral amygdala damage has also been found to have difficulties

in identifying fearful expressions (Adolphs, Tranel, Damasio, & Damasio, 1994). However, this emotion recognition deficit was subsequently found to be due to a lack of spontaneous gaze fixation to the eye region, as explicit instruction to look at the eyes completely restored the patient's ability to identify fearful facial expressions (Adolphs et al., 2005). In NT adults, the magnitude of gaze preference for the eye region of fearful faces is correlated with amygdala activation, suggesting that it may be involved in reflexive gaze orienting toward eyes (Gamer and Büchel, 2009; Adolphs et al., 2005).

The amygdala's responsiveness to eyes and facial expressions makes it a strong candidate region underlying abnormal social attention in ASD (i.e., amygdala hypothesis of ASD; Baron-Cohen et al., 2000), and consequently, its anatomy and functioning in ASD has been highly investigated. However, neuroimaging studies of the amygdala in ASD have reported highly discrepant results, with some reporting hyperactivation to faces (Dalton et al., 2005; Monk et al., 2010; Weng et al., 2011), some reporting hypoactivation (Baron-Cohen, Ring, Wheelwright, Bullmore, Brammer, Simmons, & Williams, 1999; Critchley et al., 2000; Ashwin, Baron-Cohen, Wheelwright, O'Riordan, & Bullmore, 2007; Pelphrey, Morris, & McCarthy, 2007), and others finding initially typical levels of activity but reduced levels with habituation over time (Kleinhans et al., 2009). Anatomically, older adolescents and adults show no differences in amygdala volume (Haznedar et al., 2000), but a longitudinal study of 8–18-year-old boys with and without ASD shows an altered developmental trajectory (Schumann et al., 2004). In this particular study the amygdala was enlarged in 8–12-year-old boys with ASD relative to controls, but this difference was not found in 13–18-year olds due to differing growth trajectories between groups.

Baron-Cohen et al. (1999) were the first group to provide evidence for abnormal amygdala activation in ASD. ASD and NT adults were presented with photographs of eyes and asked to either discriminate gender or indicate the mental state of the person presented. The NT group was more accurate in both tasks and demonstrated significantly greater activation of the left amygdala throughout the tasks, while the ASD group did not. Critchley et al. (2000) also compared ASD and NT individuals on two different facial discrimination tasks—one involving the emotion (explicit emotion processing) and another involving the gender (implicit emotion processing) of faces depicting different emotions. Individuals with ASD exhibited hypoactivation of the left amygdala compared with NT controls during the implicit but not the explicit task. Furthermore, Ashwin et al. (2006) found NT adults to show greater left amygdala and left OFC activation to emotional faces, with only NT individuals displaying varying degrees of amygdala activation with regard to the intensity of fearful expressions. Hypoactivation of the amygdala has also been found in individuals with ASD when viewing dynamic facial expressions (Pelphrey et al., 2007).

However, when gaze was monitored along with fMRI data acquisition, Dalton et al. (2005) again found highly revealing results regarding amygdala activity in ASD. Like FFA activity, amygdala activation in the ASD group was strongly and positively correlated with the amount of time spent fixating the eye region, but this

was not the case in the control group. All of these observations, combined with behavioral data indicating abnormal social attention in ASD, highlight the importance of monitoring visual attention in conjunction with brain activation.

Yet even with attention carefully monitored and equated across groups, neural differences of the amgydala have still been found. For example, rather than recording eye movements, one fMRI study implemented an attention-cuing paradigm to provide a measure of a participant's attentional bias (Monk et al., 2010). ASD and NT adults viewed emotional and neutral face pairs, after which a target appeared in the congruent (emotional face) or incongruent (neutral face) location. Participants then pressed a button to indicate the location of the target. The reaction times varied based on which facial expression draws more attention, with bias scores reflecting the difference between reaction times of incongruent and congruent trials. There were no differences between groups in attention bias, but the ASD group showed abnormal brain activity nonetheless, with greater right amygdala activation in response to happy and sad faces compared with controls. A separate experiment by this same group (Weng et al., 2011) attempted to control for possible group differences in attention in a different way; in this case, by presenting face stimuli for only a short amount of time (250 ms) to preclude gaze away from faces. Here, they also found that individuals with ASD exhibited greater amygdala activation compared with controls. Together these findings suggest that hypoactivation of the amygdala in ASD may simply be due to attentional differences between groups, and when attentional differences are considered, individuals with ASD may rather show increased amygdala activation to faces. Despite these discrepancies, these studies collectively provide evidence for abnormal amygdala activity in ASD, though more research is needed.

Comparing individuals with ASD to those with amygdala lesions can provide further insight into whether amygdala dysfunction may underlie the type of social attentional abnormalities seen in ASD. Much of this insight has so far come from studies of a single patient, known as SM, whose rare genetic condition (Urbach-Weithe disease) caused bilateral calcification of her amygdala (Adolphs, 2010). Like in ASD, SM exhibits reduced gaze to the eye region of faces (Adolphs et al., 2005; Spezio, Huang, Castelli, & Adolphs, 2007b), with this effect being particularly pronounced in the first few fixations of a face. A study by one of us (Kennedy and Adolphs, 2010) found that when SM was shown a picture of a face to which she had to saccade, she only initially fixated the eyes on 15% of the trials, in contrast to controls who first fixated eyes 78% of the time. However, when this same patient was explicitly instructed to fixate the eye region, she was able to do so, and her previously deficient ability to identify fearful facial expressions became normal (Adolphs et al., 2005). This points toward a role of the amygdala in directing one's gaze to important social information.

Kennedy and Adolphs (2010) further studied the relationship between the amygdala and gaze by having SM and controls explore faces using a gaze-contingent eye tracking paradigm that only revealed a small region of the face in real time at the location being fixated. This task eliminates the competition between facial features

and forces the participant to deliberately seek out features to fixate using top-down attentional control, as opposed to bottom-up attentional processes. Remarkably, SM's fixation patterns to the face normalized; the time she spent fixating the eye region was statistically indistinguishable from that of controls. This suggests that during unrestricted viewing, her fixations were perhaps influenced by attentional competition with other aspects of the face. This is consistent with fMRI studies of amygdala functioning in NT adults. Gamer and Büchel (2009) found that the amygdala is activated when individuals fixate the mouth and then subsequently make a saccade toward the eyes, but not the other way around (i.e., when one fixates the eyes, and then saccades toward the mouth). This implies that gaze to the eyes is driven by the amygdala. Taken together, these results suggest that the amygdala is not necessary for processing emotion information from the eyes, but rather required for spontaneous attention to socially or emotionally salient emotion within the face (Adolphs et al., 2005).

Despite some similarities in how people with ASD and amygdala lesions visually explore faces, Birmingham, Cerf, and Adolphs (2011) argue that these two groups may do so for different reasons. While SM appears to look less at eyes due to an exaggerated sensitivity to the bottom-up saliency of the mouth, individuals with ASD may be showing an abnormal top-down bias away from the eyes and toward the mouth (Neumann et al., 2006). To test this theory further, SM, adults with ASD, and NT controls were shown ecologically valid photos of complex social scenes while being presented with three conditions—Neutral (determine what kind of room the scene depicts), Description (describe the scene), and a Social Attention task (describe where the people in the scene are directing their attention). Previous research found NT observers to increase their fixations to the eyes of the people during the Social Attention condition relative to other conditions (Birmingham, Bischof and Kingstone, 2008). As expected, NT controls showed an overall greater proportion of fixations to faces and particularly the eyes, while also looking more at the eyes when the task required greater social attention. SM and the ASD groups were similar in that both looked less at the eyes, but the results showed more differences between these groups than similarities. For example, SM showed significantly more fixations to the mouth especially in the earliest fixations, while the ASD groups did not show this effect. SM also showed intact top-down modulation of gaze by task, increasing her gaze to eyes during the social attention condition. However, the ASD group failed to increase their fixations to eyes for this condition, suggesting an impaired top-down modulation of gaze in response to task demands. The authors argued that the amygdala is critical for stimulus-driven social attention, but not endogenous, top-down control of social attention.

Together, the studies reviewed in this section indicate that the amygdala is a key node implicated in social attentional processes in NT individuals, and whose dysfunction is likely to contribute to certain aspects of the social attentional phenotype in ASD.

7.4.3 Superior Temporal Sulcus (STS)

In addition to abnormal attention to social stimuli, individuals with ASD often exhibit gaze-processing deficits, having particular difficulties with following and utilizing information derived from gaze shifts as described earlier (Freeth, Chapman, Ropar, &Mitchell, 2010; Riby, Hancock, Jones, & Hanley, 2013). Many studies have demonstrated the role of the STS region in processing eye movements of others (Puce et al., 1998; Wicker, Michel, Henaff, & Decety, 1998; Allison, Puce, & McCarthy, 2000). For example, dynamic eye-gaze changes of both direct and averted gaze produce strong activation in the bilateral STS of NT individuals (Wicker, Michel, Henaff, & Decety, 1998). Attention to gaze elicits stronger STS response than attention to identity (Hoffman & Haxby, 2000), and lesions of this region have been shown to impair gaze direction judgments in monkeys (Campbell, Heywood, Cowey, Regard, & Landis, 1990). The STS is also sensitive to the social context in which the gaze shift occurs, with differential activation based on whether the gaze shift is perceived as consistent or inconsistent with the subject's expectations regarding the observed person's intentions (Pelphrey, Singerman, Allison, & McCarthy, 2003). It is thought that the STS is involved in processing social information conveyed by gaze direction (Allison et al., 2000), a skill in which ASD individuals show difficulties (Riby et al., 2013).

Behavioral studies have demonstrated that individuals with ASD are able to perceive gaze direction cues (Swettenham et al., 2003), though perhaps less reliably (Freeth et al., 2010), and yet show impairments in their ability to link the direction of gaze with the observed person's intentions (Riby et al., 2013). These impairments have been attributed to a theory of mind process; namely, that a person with ASD specifically misses the mentalistic significance of the gaze (Baron-Cohen, 1995). If this were true, we could expect brain regions normally involved in eye-gaze processing to be insensitive to the intentions that are conveyed by gaze shifts. Pelphrey, Morris, & McCarthy (2005) tested this hypothesis by presenting ASD and matched NT controls with an animated virtual avatar that shifted their gaze to look either toward a target (congruent condition) or toward an empty location away from a target (incongruent condition). Throughout the fMRI task, participants also pressed a button in response to a gaze shift, which both groups were able to do equally well. Similarly, both groups demonstrated STS activation in response to viewing eye-gaze shifts. However, only the NT group showed differential activity in response to congruent compared with incongruent gaze shifts in this region.

Direction of eye gaze, in addition to indicating intentions, can inform us of potentially harmful situations in our environment (Olsson, Nearing, & Phelps, 2007). Humans are generally more sensitive to direct compared with averted gaze of neutral faces (Senju and Johnson, 2009). However, a person with averted gaze and a fearful facial expression is not only socially and emotionally engaging, but also alerts the observer to potential environmental danger (Hadjikhani et al., 2008). NT individuals detect averted gaze in a fearful face faster (Adams & Kleck, 2003) and rate the expression as more intense than the same expression with direct gaze

(Sander, Grandjean, Kaiser, Wehrle, & Scherer, 2007). A recent study by Zürcher et al. (2013) investigated the neural responses to fearful averted versus direct gaze in young adults with ASD. While the NT group exhibited increased activation in posterior STS, FFA, and anterior insula, the ASD group failed to demonstrate increased activation in response to fearful faces with averted gaze. Furthermore, eye tracking data collected throughout the study showed no differences in the time spent on the eyes between gaze conditions or groups, strongly suggesting fixation differences did not underlie these observed differences between groups. The absence of activation has been suggested to reflect the inability to grasp the increased emotional valence of averted gaze in the fearful face. Taken together, these results thus support the theory that gaze processing deficits in ASD are not based on a general deficit in gaze discrimination, but rather due to a failure to use the social meaning of gaze.

7.4.4 Electrophysiological Measures of Social Attention

In addition to fMRI, electroencephalography (EEG) is a widely used method in the field of social neuroscience to measure neurophysiological responses to social stimuli. While fMRI provides excellent spatial resolution of brain activity, EEG provides superior temporal resolution in comparison. EEG measures electrical potential differences across the scalp that reflect underlying neuronal activity of the brain. Neuronal activity is often measured as event-related potentials (ERPs), i.e., changes in brain activity that are phase locked to the stimulus. Since ERPs reflect a response to a specific stimulus, researchers can use it to quantify the speed at which the stimulus is processed. Furthermore, EEG does not require participants to be lying down, which makes it ideal for testing individuals across age and functioning levels that otherwise may not be suitable for fMRI. On the other hand, EEG has relatively poor spatial localization compared with fMRI, underscoring the importance of utilizing both methodologies to fully understand brain activity.

As discussed extensively by Puce et al. (Chap. 4, this volume), ERP studies involving NT individuals have found robustly different patterns of brain activity in response to faces compared with non-face objects. In particular, the N170 is a right-lateralized, negative ERP peak occurring approximately 170 ms after stimulus presentation over the lateral posterior region of the scalp (Bentin, Allison, Puce, Perez, & McCarthy, 1996). Though it may be evoked by non-face stimuli, faces and even eyes alone evoke shorter N170 latencies and larger amplitudes compared with objects (Bentin & Deoull, 2000). Various manipulations to the face stimuli such as inversion, decomposition, as well as movements in eyes and mouth all influence N170 activity (Eimer, 2000; Itier & Batty, 2009).

Given the fMRI findings of aberrant activity in regions associated with face processing in ASD, it is not surprising that many have observed similarly atypical ERP responses (see Feuerriegel, Churches, Hofmann, & Keage, 2014 for review). For example, McPartland et al. (2004) carried out a study assessing

the temporal aspects of the neurophysiological response to faces in individuals with ASD, predicting the ASD group to exhibit impaired temporal processing of faces (i.e., slower neurophysiological response), as indicated by longer N170 latencies in response to faces. Age and IQ matched ASD and NT adolescents and adults viewed upright and inverted pictures of faces and furniture during EEG recording. In addition, participants were assessed for their facial recognition memory. As hypothesized, the ASD group exhibited abnormal temporal processing of faces, with significant differences in N170 latency. More specifically, the ASD group showed significantly longer N170 latencies to faces compared with controls. While the NT group showed significantly longer latencies to inverted rather than upright faces, the ASD group did not, suggesting less sensitivity to the configural alteration of the faces. Furthermore, the ASD group failed to exhibit the standard right lateralization in N170 amplitude to faces, suggesting atypical cortical specialization. Individual differences in the latency of the N170 were correlated with performance on the face recognition task in the ASD group. Slower left hemisphere N170 latency to both upright and inverted faces was associated with better face recognition performance in individuals with ASD, yet faster latencies were associated with improved face recognition in the NT group. Investigators interpreted these findings as evidence of not only atypical cortical specialization, but also different face processing strategies from those of NT individuals.

Differential N170 activity has also been found in response to emotionally salient face stimuli in this population. A recent study by Wagner et al. (2013) examined both the visual scanning patterns and ERPs to emotional faces compared with nonsocial stimuli (houses) to examine how these neural measures of face processing differ across stimuli. ASD and NT adolescents and adults viewed faces conveying angry, fearful, or neutral expressions and houses during simultaneous eye tracking and EEG recording. While the eye tracking measures revealed very similar scanning of faces between groups, ERP measures revealed significant differences. The ASD group in particular showed a lack of neural differentiation between emotion types, while NT participants showed significantly larger N170 amplitudes to fearful faces compared with angry faces. In response to houses, individuals with ASD showed a larger amplitude P1 component (also known as P100, which is a positive ERP component at 100 ms after stimulus onset) compared with controls. Increased P1 responses are thought to indicate enhanced early visual processing and attention (Heinze, Luck, Mangun, & Hillard, 1990), which suggests the ASD group was allocating increased resources for processing nonsocial compared with social stimuli. Lastly, this study revealed significant correlations between visual fixation patterns and neural processing of faces in the NT group, but these relationships were not found in the ASD group. Greater proportion of time spent scanning the eye region was associated with faster N170 responses to faces in NT individuals, which is thought to indicate efficient face processing. In contrast, those who spent a greater proportion of time scanning the mouth showed slower N170 responses. These results were in line with McPartland et al. (2004)—faster N170 latencies associated with better facial recognition

in the NT group, but not the ASD group. The lack of association between visual scanning and neural responses further suggests that the ASD group utilized different strategies of face processing.

These results are consistent with many of the fMRI studies indicating abnormal face processing as implied by reduced FFA and abnormal amygdala activity (e.g., Schultz et al., 2000; Pierce et al., 2001; Baron-Cohen et al., 2010; Critchley et al., 2000). However, it is important to keep in mind that when attention is explicitly directed at the eye region with fixation points at the center of the face, individuals with ASD show more normative neural activation (Hadjikhani et al., 2004). Furthermore, simultaneous eye tracking during free viewing of faces suggests that diminished gaze fixation to eyes in particular may account for aberrant activation of areas involved in face processing (Dalton et al., 2005). In light of these findings, Webb et al. (2012) conducted a similar experiment to that of McPartland et al. (2004), but attempted to manipulate visual attention to the stimuli. ERP responses of ASD and NT adults matched on age and IQ were collected while participants viewed images of upright and inverted faces and houses. However, attention was directed to the center of the stimuli by a cross hair that appeared prior to stimulus onset. When attention was cued to the appropriate area around the core features of the face, the two groups demonstrated similar P1 and N170 responses, with greater amplitude and faster latency to faces than houses. Some have proposed that the N170 is mediated by the eye region (Doi, Sawada, & Masataka, 2007; Nemrodov, Anderson, Preston, & Itier, 2014), thus the cued attention to the eyes may have facilitated the normative N170 and P1 response in the ASD group in this study. However, the ASD group failed to exhibit differential ERP responses to upright versus inverted faces unlike the NT group, consistent with McPartland et al. (2004). These results together with the previously discussed fMRI studies that have taken visual attention into consideration provide further evidence for abnormal social attention underlying differential neural activation in ASD.

Yet, despite all of this research, the neural origins of abnormal social attention remain largely unknown, as none of these regions alone are able to provide a sufficient explanation for the social attentional differences observed in ASD. As briefly mentioned in the beginning of this section, recently researchers have argued that abnormal activation in areas like the FFA, amygdala, and STS is not driven by a primary (i.e., causal) neural dysfunction within any of these regions, but rather neural abnormalities in brain circuitry (Schultz, 2005; Dalton et al., 2005). Consequently, there has been a shift in the field toward taking a systems-level approach to understand the functional interactions within and between different brain networks, and further research efforts with this network perspective will be important in elucidating the origins of atypical social attention in ASD.

7.5 Summary

This chapter reviewed the current literature exploring social attentional differences in ASD, resulting social cognitive deficits, and possible neural correlates. The literature is vast and many studies report discrepant findings. However, in general, researchers seem to agree that individuals with ASD show a wide range of differences in social attention as observed in the orienting to (Clark et al., 2008) and visual scanning of faces (Pelphrey et al. 2002; Klin et al., 2002; Speer et al., 2007), scanning of social scenes more generally (Fletcher-Watson et al., 2009; Santos et al., 2012), and responding to eye gaze (Freeth et al., 2010; Riby et al., 2013). The functional consequences we have discussed include deficits in judging facial identity (Blair et al., 2002) and recognizing emotional facial expressions (Wallace et al., 2008; Corden et al., 2008), and there are likely more downstream consequences on social cognition more broadly. And, while some studies have found that some adults with ASD show intact abilities in these domains, it has been suggested that those individuals may be utilizing different strategies. Thus, compensatory mechanisms may be playing a significant role in their success on tests of social processing (Grossman et al., 2000; Spezio et al., 2007a; Baez et al., 2012), as can sometimes be revealed with more sensitive behavioral and neural measures.

We focused on three brain regions (the FFA, the amygdala, and the STS) that may underlie particular aspects of abnormal social attention in ASD. While some studies have found robust differences in neural activation in these regions (Schultz et al., 2000; Baron-Cohen et al., 2010; Dalton et al., 2005; Pelphrey et al., 2005), many have also reported no differences. We suggest that one possible factor underlying these discrepancies is due to differences in experimental tasks used, and whether or not they accounted for possible social attentional differences between groups. For example, while the FFA has been shown to be significantly less active in ASD when viewing faces, its activity is strongly and positively correlated with the amount of fixation to the eye region for individuals with ASD, which suggests that social attentional abnormalities may underlie the FFA hypoactivation to faces in other studies (Hadjikhani et al., 2004; Dalton et al., 2005). Individuals with ASD, while able to identify gaze shifts in others, fail to show modulated STS activity to gaze in conjunction with particular expressions or target locations (Zürcher et al., 2013). These results suggest that gaze processing deficits in ASD are not due to a general deficiency in perceptual discrimination, but rather a failure to derive appropriate social information from the gaze shift observed. Lastly, ERP studies have also showed abnormal N170 responses to faces in ASD (McPartland et al., 2004; Wagner et al., 2013), but additional studies have found more normalized responses when attention is cued to the eyes of the face stimuli (Webb et al., 2012). Such findings further support the idea that aberrant visual social attention strongly impacts neural differences in ASD, in terms of both regional activation and the temporal dynamics of brain activity.

7.6 Outstanding Questions and Issues for the Field

Thus, while these studies have contributed importantly to our understanding of the neural underpinnings of ASD, perhaps their most important contribution has been to highlight the critical role of social attention. With altered social attention comes altered neural responding, and with normative social attention comes more normative neural activity. This suggests that social attention profoundly shapes how individuals not only seek out information from their environment, but also that it has downstream consequences on how they process that information. Like other modulatory systems (arousal, reward, etc.), attention influences nearly every aspect of human cognition.

When considered from a developmental context, the implications of altered social attention become clear—individuals with an ASD may not be extracting the relevant social information from their environment, their brain thus fails to respond in a normal way to social information, and they fail to develop expertise within the social domain. This lack of expertise likely has self-reinforcing properties, such that social information becomes less and less prioritized, and social competencies lag behind their typically developing peers. In order to function in our highly social world, compensatory processes are likely developed, but these processes seem to lack the fluidity, spontaneity, and ease that characterize social processes for typically developing individuals.

Some have argued that social attentional differences are the downstream effects of social motivational deficits which, when combined together, lead to social cognitive deficits (Chevallier et al., 2012). Yet, additional experimental paradigms to sensitively quantify social motivation are needed. While questionnaires such as the Social Responsiveness Scale (SRS; Constantino, & Gruber, 2002) provide subscales of social motivation, these questionnaires require either the individual's or their caregiver's insight. There is thus a need for the development of experimental manipulations that can directly measure social motivation, and examine how motivational factors relate to the abnormal allocation of attention and subsequent social functioning. The ability to sensitively quantify social motivation may impact the development of individualized treatments for ASD. For example, if a particular individual has low social motivation and correspondingly altered social attention, interventions aimed to increase social motivation (e.g., Floortime (Greenspan & Wieder, 1997)) may result in more normative patterns of social attention, and may be more impactful than other types of interventions (e.g., social skills training) for that particular individual. The apparent normalization of brain activity when social attention is taken into account bodes well for the development of interventions for children and adults. These findings suggest that individuals with ASD may not engage their brains in the typical ways, but also suggest that they may be capable of doing so, given particular instructions, conditions, and/or motivation.

Beyond behavioral interventions, the neuropeptide oxytocin has been pointed to as a possible point of therapeutic intervention of social attention and motivation,

as it is hypothesized that aberrant oxytocin systems contribute to social deficits in ASD (Waterhouse, Fein, & Modahl, 1996). In mammals, oxytocin has been associated with the development of prosocial behavior, such as maternal attachment, and abnormal oxytocin levels have been reported in children with ASD (Modahl et al., 1998). Furthermore, oxytocin concentrations are positively predictive of theory of mind and social communication performance in both ASD and non-ASD cohorts, highlighting its role in social functioning in humans (Parker et al., 2014). While more studies need to be conducted to verify its role in social attention, intranasal oxytocin administration has been shown to increase fixation time on core areas of faces (particularly the eyes) and promote social approach behaviors (Andari et al., 2010), improve emotion recognition (Guastella et al., 2010) and affective speech comprehension (Hollander et al., 2007) in individuals with ASD. It is, however, implausible that there will be a single treatment that will work for all individuals with ASD, given its heterogeneous biological underpinnings.

This heterogeneity, in terms of both etiology and behavioral expression, also likely underlies some of the discrepant findings across studies and laboratories. Heterogeneity is perhaps the least disputed feature of the condition (Geschwind & Levitt, 2007), and not only impacts replication across study samples, but also suggests that methods and analyses need be sensitive to individual differences. Rather than increasing sample sizes in an attempt to identify a statistically significant group difference between ASD and NT individuals, perhaps researchers should begin focusing on individual, idiosyncratic patterns of abnormal social attention and neural activity, and begin to ask how these individual patterns relate to particular deficits or strengths in social functioning within that individual (e.g., Byrge, Dubois, Tyszka, Adolphs, & Kennedy, 2015). In other words, researchers may be better served by embracing the heterogeneity characteristic of ASD, rather than fighting against it.

So, where do we go from here? While we have come a long way in our understanding of ASD, many questions still remain in understanding the etiology and structure of atypical social attention in ASD:

- Although social attention and social cognition seem to be associated, the causal relationship between the two is unknown. Does abnormal attention produce abnormal cognition, or does abnormal cognition result in altered attention? In other words, is altered gaze a cause or side effect of altered cognition? The development of new experimental approaches to address these questions will be required (Bush, Pantelis, Morin, Duchesne, Kagemann, & Kennedy, in press).
- Does normalizing social attention normalize social perception and social responding, like it seems to do for brain activity? For example, if individuals with ASD are instructed to look at the eyes in faces, are they able to use this information effectively to make correct social judgments?
- What aspects of altered eye movements in ASD can be attributed to bottom-up attentional differences, top-down attentional differences, or a combination of the two?

- Can eye tracking measures of social attention be used for diagnostic purposes? While findings from infants point toward this possibility (Jones & Klin, 2013), it is less clear whether this approach will be effective in older children and adults.
- To what degree is abnormal attention in ASD domain-specific? Can we dissociate basic attentional processes from abnormal social attentional processes in ASD?

7.7 Conclusions

Studies using stimuli and experimental paradigms with greater ecological validity, along with a focus on individual differences, will be crucial for a complete understanding of social attention in ASD. Furthermore, going beyond region- and location-centric studies of potential neural mechanisms, and toward a systems-level approach to link behavior to network-level functioning, will be critical in illuminating the origins of atypical social attention in ASD.

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