
Understanding the Social Nature of Autism: From Clinical Manifestations to Brain Mechanisms

2

Fred R. Volkmar and Brent van der Wyk

Beginning in the 1800s, reports of “wild” or feral children and other children we might now recognize as having autism began to appear (Donvan & Zucker, 2016), but it was not until the 1940s in reports by Leo Kanner (1943) and Hans Asperger (1944) that autism spectrum disorder (ASD) began to be clearly recognized. In their papers, Asperger and Kanner both used the word “autism” to suggest a special difficulty of social interaction exhibited in the cases they observed. As time has gone on, the unusual social nature of autism has been more clearly recognized, and much progress has been made in understanding, at the basic science level, potential mechanisms and best approaches to treatment (McPartland, Tillman, Yang, Bernier, & Pelphrey, 2014; Reichow & Barton, 2014; Voos et al., 2013).

Early controversy on the nature of autism began to shift in the 1970s, as it became clear that autism was a brain-based disorder (given the high rates of seizure disorders children exhibited in adolescence; Volkmar & Nelson, 1990) and had a very strong genetic basis given the much higher concordance rates in monozygotic twins (Folstein & Rutter, 1977). It also became clear that autism responded more positively to structured interventions rather than unstructured psychotherapy

(Bartak & Rutter, 1976). Since the official recognition of autism in 1980 (DSM III APA, 1980), research has exploded. With earlier detection and intervention, many individuals with autism and related conditions are now functioning independently in community settings (Howlin, Moss, Savage, Bolton, & Rutter, 2015) and indeed sometimes, as adults, appearing to no longer exhibit the condition (strictly defined; Fein et al., 2013).

In this chapter, we selectively review some aspects of this unique constellation of social difficulties noted in autism and related ASD both as they manifest clinically and as they may be related to brain mechanisms. In summarizing these results, we argue for a new conceptualization of autism that emphasizes these social learning problems as central. We should note that it is a testament to the field that this review cannot aim, in the space available, to be anything but selective, highlighting those aspects of social difficulties that appear to us most salient in terms of clinical or research relevance.

2.1 Autism as a Central Feature of the Condition

In his 1943 report, Kanner emphasized that autism (lack of interest in others) was one of two cardinal features of the condition (resistance to change and oversensitivity to the nonsocial

F.R. Volkmar, MD (✉) • B. van der Wyk, PhD
Yale University School of Medicine,
New Haven, CT, USA
e-mail: fred.volkmar@yale.edu

environment being the other). His insightful description served as the basis for subsequent refinements of the concept (Volkmar & Reichow, 2014). Kanner emphasized that the social disability in autism was marked and profound, and impacted the child's ability to understand and learn from others. This core disability helps us understand the impact of ASD on aspects of learning, communication, multitasking, and behavior.

Attempts to define autistic social dysfunction have evolved over time. In his very influential synthesis of Kanner's original report and subsequent work, Rutter (1978) emphasized delayed and deviant social development NOT simply the functioning of overall developmental delay. Other early investigators similarly identified social difficulties as a central diagnostic feature (Wing & Gould, 1979). This tradition continued in the DSM-III (Diagnostic and Statistical Manual 3rd Ed.) and its various successors (see Volkmar & McPartland, 2014).

Although there is general agreement that "autism" – that is the serious social disability – is the defining feature of ASD (Grossman, Carter, & Volkmar, 1997), attempts at more detailed characterization have been complicated. The complications arise given changes with age and developmental level, as well as major differences in approaches (e.g., should social problem be assessed relative to normative development or should specific abnormal features be identified). Quantification of social problems plagued the field for many years. On one side, attempts to estimate levels of severity are reflected in screening and diagnostic instruments looking at the "severity" of autism (e.g., on instruments like the ADOS (Autism Diagnostic Observation Scale) or CARS (Childhood Autism Rating Scale); see Lord, Corsello, & Grzadzinski, 2014). On the other hand, there are some approaches that assess social levels based on normative social processes such as normed tasks of memory for faces (Klin et al., 1999) or assess socialization skills based on instruments like the Vineland Adaptive Behavior Scales (Volkmar et al., 1987). As we note subsequently, a series of innovative measures have now been developed as part of the

study of brain processing social information and may offer alternative approaches. For example, work from the initial eye tracking studies conducted at Yale (Klin, Jones, Schultz, Volkmar, & Cohen, 2002) revealed major differences in the way more cognitively able individuals with autism viewed the social "panorama," as measured by infrared cameras, during observation of short clips from the movie classic *Who's Afraid of Virginia Wolff*. In that initial study, there was essentially no overlap between the groups in terms of focusing on eyes in the scenes (with a Cohen's d of 3.6) (Fig. 2.1).

Progress has also been made in the area of genetics. It has become clear that many genes are potentially involved with a broader autism phenotype likely exhibiting some, but not all, of the features of those individuals with more classic autism (Ingersoll et al., 2014; Rutter & Thapar, 2014). Although the relationship between social vulnerabilities to genetic vulnerabilities remains to be specified, work using more precise and sophisticated animal models is now underway (Thirtamara Rajamani, 2015).

2.2 A Selective Review of Specific Areas of Social Disturbance

2.2.1 Attachment

The process of attachment serves to give the developing infant opportunities for bonding and to develop complex social relationships with primary caregivers. In typically developing children, this process is quite robust, and while life long, becomes particularly noteworthy around 9–10 months as infants become quite comfortable with parents and quite wary of strangers. Attachment is manifested through a number of behaviors that include maintenance of proximity to caregivers as well as strong affective connections (Rutter, 2008). Early impressions of an absence of parental attachment in autism proved to be incorrect as a series of studies indicated some attachment to parents – albeit sometimes in atypical ways (Rogers, Ozonoff, & Maslin-Cole, 1991, 1993).



Fig. 2.1 Visual focus of a typically developing adult (top line) and a high functioning man with autism (bottom line) while moving a short movie clip from the class film *Who's Afraid of Virginia Woolf?* The typically developing individual focuses on the top portion of the face while

observing the interaction. The individual with autism focuses on the mouth region (losing much of the social-emotional information; Reprinted, with permission, from Klin et al., 2002).

For example, young children with autism are apparently more likely to exhibit disorganized patterns of attachment than typically developing children (Claussen, Mundy, Mallik, & Willoughby, 2002). In addition, they may exhibit unusual attachments to inanimate objects (i.e., in contrast to the usual “transitional objects” of children, typically soft and intensely specific) and to hard objects (bundles of twigs, cereal boxes, and toy trains); it is the category of objects rather than the special objects that is of paramount importance (Volkmar et al., 1994).

2.2.2 Perception of Faces

Faces have special salience for a newborn child. The ability to look at faces and derive meaning and information from facial expression is an important prerequisite for many other social skills. Clearly, there are alternate pathways, for example, blind infants usually develop social skills in typical ways albeit with understandable delays in some areas such as joint attention

(Bigelow, 2003). The interest in faces appears to be innate in a typically developing child with a very early preference for looking at faces or face-like stimuli, relatively rapid recognition of parent's faces, use of the top half of the face as a source of greater information (Farah, Wilson, Drain, & Tanaka, 1998), and activation of a specific areas of the brain (the fusiform gyrus) for aspects of facial recognition (Kanwisher, McDermott, & Chun, 1997). Sophistication in facial recognition and face processing continues to develop at a rapid pace during the first year of life. By the first birthday, the typically developing child is exquisitely sensitive to familiar and unfamiliar faces and has difficulties processing faces if the latter are presented upside down (the facial inversion effect). Face processing is smooth and rapid by this time and continues to develop over the next years of life (Fabio Falck-Ytter, 2008).

A substantial body of work now exists on difficulties in facial perception in individuals with ASD. This work uses various methods, based on behavioral responses, neurophysiological correlates (electroencephalogram [EEG]), and

neuroanatomy (e.g., functional magnetic resonance imaging [fMRI]). These difficulties (some of which are discussed in greater detail in subsequent portions of the chapter) manifest clinically by the early onset of difficulties with gaze, difficulty using the face to regulate and derive meaning in social interaction, and lack of eye contact (Chawarska et al., 2014a; Chawarska, Macari, Volkmar, Kim, and Shic, 2014b). Behaviorally, these difficulties are also expressed in diminished social-emotional responses to others. These impairments include reduced eye contact, joint attention, social orienting, deficits in the imitation of faces and in the face recognition, attenuated responses to emotional displays of others and in gaze behaviors (McPartland, Webb, Keehn, & Dawson, 2011), and may have implications for imitation in general (Rogers, Cook, & Meryl, 2014).

Some of the earliest work with young children relied on retrospective analysis of videotapes (e.g., made at the child's first birthday – often a rather social event albeit one in a familiar environmental context). For example, Osterling and Dawson (1994) revealed that children subsequently diagnosed with ASD paid significantly less attention to faces than typically developing controls. Other work with older individuals has used a range of methods. For example, behavioral studies have revealed difficulties with tasks that involve face processing (Dawson, Webb, & McPartland, 2005), some differences in processing inverted faces (Fabio Falck-Ytter, 2008), and lack of some of the usual early markers of evolved facial perception such as the lack of the facial inversion effect in autism as well as facial memory (Klin et al., 1999). These difficulties also involve problems with the use of emotional information provided by the face to guide accurate perception and awareness of others (Tanaka et al., 2012). Studies have also shown differences in the process of face recognition. For example, typically developing children employ a piece-meal processing strategy in looking at objects, but not at faces. While in ASD, face processing is more like that of a typical child in looking at objects (Chawarska & Shic, 2009).

As we discuss subsequently, a growing body of work has used EEG and fMRI methods to clarify neurophysiological and neuroanatomical correlates of these abilities, and new experimental procedures (e.g., eye tracking) have been used to clarify, in much greater detail, specific differences in processes such as face perception. As a practical matter for the developing child with autism, the nonsocial world has much greater salience (relative to the typically developing children) – a phenomenon strongly emphasized by Kanner (1943) in his original report on the condition.

2.2.3 Gaze Behavior and Eye Contact

Unusual aspects of gaze and limited or poor eye contact are frequently noted by parents as some of the first signs of autism. These processes are intimately related to other aspects of social development including facial processing (Chawarska et al., 2014a, 2014b). The ability to engage in mutual gaze is important for both perception of feelings and attentional focus of others, as well as the ability to engage in the back and forth of communication in social interactions (Mundy, 2016). As with face processing, the interest in others is expressed early on in the typically developing infants ability to focus on the parent, while in infants with ASD, reduced levels of mutual gaze are striking (McPartland et al., 2011). Data are somewhat conflicting on the age at which this can first be demonstrated, with some evidence suggesting it can be observed as early as 6 months and other work suggesting this is less clear until about 18 months (Chawarska, Volkmar, & Klin, 2010; Ozonoff & South, 2001; Young, Merin, Rogers, & Ozonoff, 2009). In this and other studies, differences in methods are likely critical. Data from eye tracking have been, perhaps, the most convincing demonstration of early differences (Chawarska, Macari, & Shic, 2013).

2.2.4 Imitation

The desire to imitate emerges very early in typical development. Imitation is important in laying

the foundation for many other skills including communication. A considerable amount of research on the emergence of imitation in autism has revealed substantial impairments in imitation, in all its various forms, and these difficulties are interrelated with other core social abilities such as joint attention, play, and emerging communication abilities (for a review, see Rogers, 2014). It has been argued that difficulties in this area are a core aspect of autism (Rogers & Pennington, 1991). Although this issue remains a topic of theoretical debate, there is widespread agreement that intervention focused on enhancing imitation and social attention is essential for young children with autism (Rogers et al., 2006).

Intervention for imitation difficulties has focused on the various forms of imitative activities, for example, object imitation (Ingersoll, Lewis, & Kroman, 2007), motor imitation (Ingersoll & Schreibman, 2006), gestural imitation (Ingersoll et al., 2007; Ingersoll & Schreibman, 2006), and overall imitation (Ozonoff & Cathcart, 1998). Although it is generally assumed that enhanced abilities in imitation will be related to gains in other areas, few studies have directly addressed the issue, but the work available suggests important gains in related skills such as communication, play, joint attention, and overall cognitive abilities (Ozonoff & Cathcart, 1998; Rogers & Vismara, 2014). A number of the model programs developed for young children with autism target imitation as a critical developmental skill, for example, the Early Start Denver Model of Rogers, Dawson, and Vismara (2012), Pivotal Response Training (Koegel & Frey, 1993), and applied behavior analytic models (Ingersoll et al., 2007). Fostering imitation is also naturally involved in interventions targeting other abilities such as joint attention and play (Kasari, Freeman, & Paparella, 2001; Kasari, Huynh, & Gulsrud, 2011).

2.2.5 Play Skills

Although difficult to define, play is a central activity of childhood. It involves important cognitive, motor, and social aspects and becomes symbolic,

an important foundation for other skills (Piaget, 1952). Play begins with simple object manipulation (sensorimotor play) and then progresses to functional play (e.g., using materials in conventional ways) and finally becomes much more symbolic and complex play (where the activity of play is much less constrained by the actual materials present). For example, a small cup might be used initially for banging or smelling, but then becomes used for functional play such as feeding a doll, to then becoming rather unconstrained so that the cup might assume any number of symbolic functions. There are important cultural and contextual factors in understanding play, and play is a central focus of children's lives during preschool (and beyond) and serves as a practice ground both for pretense and as a pleasurable shared joint experience with parents, siblings, and with peers. Piaget (1952) emphasized the importance of play for cognitive development.

Difficulties in symbolic play in ASD have been well documented since the 1970s (Ricks & Wing, 1975). Subsequent work confirmed the lack of symbolic play in young children with autism (Wing, Gould, Yeates, & Brierley, 1977) with play tending to be stereotypic and repetitive with difficulties extending to both nonsymbolic and symbolic play (Ungerer & Sigman, 1981). These difficulties extend to the easiest forms of play so that both sensorimotor and functional play skills are delayed and/or deviant in their development even when overall cognitive ability is controlled.

Intervention studies have shown that a range of methods can be used to improve play skills. These include both direct, as well as, more naturalistic teaching and the use of peers (particularly in the preschool period) to facilitate play abilities (Kasari, Freeman, & Paparella, 2006; Rogers, 2005).

2.3 Neural Aspects of Social Information Processing

2.3.1 Early Studies of Social Cognition

Social and affective processing has been, until recently, a rather neglected branch of cognitive

psychology. The prevailing attitude was that complex social behaviors could be explained as the aggregation of simpler and more foundational cognitive components: attention, memory, etc. However, concerted research efforts during the 1970s and 1980s led to a set of findings that challenged this established view and argued for dedicated cognitive mechanisms for social information processing. Behavioral studies of face processing (Valentine, 1988; Yin, 1969) and biological motion (Johansson, 1973; Kozlowski & Cutting, 1977) suggested specialized mechanisms for this kind of social perception. Similarly, Cosmides and Tooby (1992) pursued a research program that argued convincingly for specialized mechanisms for social reasoning. At the time, investigation of the neural bases of such function in healthy subjects was beyond the ability of science, but studies of patients with brain damage, developmental disorders, and electrophysiological studies of nonhuman primates all began to point to dedicated neural mechanisms for a number of social processes (Bauer, 1984; Brothers, 1990; Signer, 1987). This work came together in a seminal article by Brothers (2002), in which the term “social brain” was coined. Brothers argued that the evidence suggested that not only were there good reasons to suspect that specializations for social information processing is evolutionarily adaptive, but that by the evidentiary standards of the day, social processing was a cognitive module – an innately specialized and encapsulated neurocognitive mechanism (Fodor, 1986). The elevation of social cognition to “modular” status placed it on par with language in the pantheon of cognitive psychology and jump started a research program that continues to this day into the neural bases of social cognition.

At the time, the awareness that ASD was primarily a disorder of social cognition led researchers to study the condition. Indeed, the field of social cognition has benefited from the growing awareness of and attention to ASD and lifted it from a niche research area to one with profound public health significance. This work also benefited from coincident developments in human brain imaging methods, most notably, fMRI.

fMRI uses powerful magnetic fields to image regional changes in blood oxygenation. These changes are thought to be driven by the metabolic needs of neurons that fluctuate as a function of their activity. A standard strategy in an fMRI experiment is to present stimuli to individuals and measure the resulting blood oxygenation-dependent signal (BOLD). Differences in the BOLD signal, as a function of stimuli or task, are interpreted as differences in the underlying brain activation to those signals. As fMRI is noninvasive, it provided the opportunity to study aspects of brain function in healthy controls. An advantage that fMRI had over other noninvasive methods, such as EEG and ERP, was an incredibly high degree of spatial accuracy. It was this spatial resolution that was particularly attractive to researchers interested in finding and cataloging the neural correlates of various modules of cognition, and in this respect, social cognition was no different.

2.3.2 Key Nodes of Social Cognition in Healthy Individuals

Brothers (1990, 2002) identified several cortical and subcortical nodes of the social brain, namely the orbitofrontal cortex, the temporal cortex, and the amygdala, (Kling & Brothers, 1992; Perrett, Hietanen, Oram, Benson, & Rolls, 1992; Raleigh & Steklis, 1981). Much of the evidence implicating these regions came from the study of nonhuman primates, using electrophysiology and experimentally induced lesions. In humans, early fMRI studies supported the initial delineation of the social brain with only minor revisions. In a set of early studies of face processing, two subsets of the temporal cortex were identified on the fusiform gyrus and the super temporal sulcus (Haxby, Hoffman, & Gobbini, 2000; Kanwisher et al., 1997; McCarthy, Puce, Gore, & Allison, 1997). Simple contrasts of static face versus non-face stimuli activated a region on the fusiform gyrus on the ventral surface of the temporal lobe, termed the fusiform face area (FFA). More complex facial stimuli, which captured dynamic aspects of a face in motion, tended to activate a

lateral and posterior surface of the temporal lobe in the superior temporal sulcus (STS; Beauchamp, Lee, Haxby, & Martin, 2003; Pelphrey, Singerman, Allison, & McCarthy, 2003b). Emotion in faces, especially negative affect, tended to activate the amygdala, consistent with social brain theory (Breiter et al., 1996). The orbitofrontal cortex, extending dorsally along the medial surface of the prefrontal cortex and the dorsal medial prefrontal cortex (dmPFC), was associated with mentalization (Happé et al., 1996) and affective arousal (Ketter, George, Kimbrell, Benson, & Post, 1996).

However, new regions and functions were also being discovered. For example, while fearful faces activated the amygdala, in addition to the FFA, disgusted faces activated the insula that was also activated during the experience of disgust (Phillips et al., 1997). The perception of bodies and actions also required the extension of initial theory. Regions in the posterior temporal cortex were activated by the perception of bodies (Chan, Peelen, & Downing, 2004; Downing, Jiang, Shuman, & Kanwisher, 2001). The STS itself was also found to be active to the perceptions of bodily movement (Bonda, Petrides, Ostry, & Evans, 1996; Carter & Pelphrey, 2006; Pelphrey et al., 2003; Puce & Perrett, 2003) and were modulated by the social and mental significance of those actions (Beauchamp et al., 2003; Vander Wyk, Hudac, Carter, Sobel, & Pelphrey, 2009; Vander Wyk, Voos, & Pelphrey, 2012). Finally, with respect to mentalization and theory of mind, regions in the temporoparietal junction (TPJ) were activated during such tasks (Castelli, Happé, Frith, & Frith, 2000; Gallagher & Frith, 2003; Saxe & Kanwisher, 2003; Völlm et al., 2006) and have spawned a lively and ongoing debate as to how functional roles are partitioned across these regions (Buccino et al., 2007; see also Farah et al., 1998).

A surprising addition to the set of social regions came from electrophysiological studies in nonhuman primates. While studying the response properties of motor neurons, researchers identified neurons, now dubbed “mirror neurons,” that fired in response to both the performance and observation of specific actions

(Rizzolatti, Fogassi, & Gallese, 2001). The response properties of mirror neurons are intriguing because they might represent a mechanism by which the affective or cognitive state, which is hidden from direct observation, could be related to detailed knowledge about one’s own affect and cognition. For example, during action perception, merely representing the ongoing details of a bodily motion misses a great deal of useful and predictive information about why the action was being taken. Thus, the additional computational challenge for the observer is to decode from those actions potential motivating goals, beliefs, and affect. As mirror neurons appeared to play a role in the self-generation of actions, they were proposed to have a more direct association with the goals, beliefs, and actions that the observer themselves would have when activating that action plan (Rizzolatti, 2005; Uddin, Iacoboni, Lange, & Keenan, 2007). Their activation during observation, thus, would permit the activation of mental states that the observer could infer the actor as having (Meltzoff & Brooks, 2001). The evidence for a mirror neuron system in human neuroimaging is complex. Many studies reported mirror neuron-like patterns of activation (e.g., Gazzola & Keysers, 2009; Iacoboni et al., 1999). But other studies note, or have argued, that any cognitive inferences from action to underlying mental intention can be performed without reference to mirror neurons (Kilner & Frith, 2008; Lingnau, Gesierich, & Caramazza, 2009; Turella, Pierno, Tubaldi, & Castiello, 2009).

Nevertheless, the notion of social representations of others rising out of self-representation has a long history in developmental and social psychology (Iacoboni, 2009; Meltzoff, 1990). For example, physical imitation, which can be observed even in very young infants (Meltzoff & Moore, 1977), was thought to be a precursor to more sophisticated social learning and even abstract social cognitive phenomena divorced from purely physical embodiment, such as empathy and theory of mind (Bandura, 1962). Mirror neurons presented as potential mechanism for at least the early stages of this development and their discovery energized the investigation into the neural bases of these relatively high-level social phe-

nomena (Gallese, 2001; Kaplan & Lacoboni, 2006; Lacoboni, 2009; Lacoboni et al., 1999; Molnar-Szakacs & Overy, 2006; Williams, Whiten, Suddendorf, & Perrett, 2001). As with many efforts in social cognitive neuroscience, from a relatively circumscribed list, the number of potential regions identified as part of the “mirror neuron system” has expanded (for a review, see Molenberghs, Cunnington, & Mattingley, 2012).

2.3.3 Social Neuroscience of ASDs

ASDs have been at the heart of many discussions in social neuroscience from the very beginning. In Brothers' (1990) article, autism was presented as evidence for the modularity of the social processing system. Since then, virtually every aspect of social cognition has been examined in ASD samples in some way or another (for a recent review, see Philip et al., 2012). With respect to social perception and face/body motion perception, differences in brain activation between individuals on the spectrum and healthy controls observed are generally consistent with behavioral difficulties seen in these individuals. For example, early studies report less selective activation of the fusiform in ASD relative to healthy controls during face processing (Schultz et al., 2000; Wang, Dapretto, Hariri, Sigman, & Bookheimer, 2004; although for other perspectives, see Hadjikhani et al., 2004; Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2007) as well as in the STS during action perception (Ahmed & Vander Wyk, 2013; Blake, Turner, Smoski, Pozdol, & Stone, 2003; Freitag et al., 2008). Similarly, both the dmPFC and the TPJ showed a similar lack of selective activation during mentalization tasks in individuals with ASD (Happé et al., 1996; Lombardo, Chakrabarti, Bullmore, Baron-Cohen, & Consortium, 2011; Silani et al., 2008). Similar findings can be found in other domains of social cognition. Thus, one of the most consistent results from this work has been a relative lack of specialization in the core circuits for social cognition. However, other findings are also worth considering, and here we take the ongoing study of face processing as an example.

Even in the early fMRI studies of face processing, there were hints that there was more than simply a lack of specialization in the ASD samples. Studies found that individuals on the spectrum recruited other brain regions outside of the fusiform to a greater degree than healthy controls (Pierce, Müller, Ambrose, Allen, & Courchesne, 2001; Schultz et al., 2000). This suggests two, not necessarily distinct, possibilities. First, it may be that ASD is associated with an intrinsically large-scale reorganization of cortical circuitry, which includes both regional increases and decreases in activation during social cognition tasks. Recent studies of large-scale cortical networks are consistent with this account showing a relatively broad difference in the pattern of connectivity in ASD (Minshew & Williams, 2007; Monk et al., 2009). These data are often collected while the individual is at rest, reducing the chances that the differences are due to differences in explicit strategies.

A second possibility is that individuals with ASD selectively or strategically engage with social tasks differently than typically developing controls. For instance, individuals with ASD tend not to look directly at eyes when looking at faces (Pelphrey et al., 2002). This differential engagement would be expected to have consequences for the resulting brain activation. In one study, input selectivity was experimentally manipulated by having controls and individuals with ASD attend to locations on a face that corresponded to greater or lesser amounts of eye contact (Perlman, Hudac, Pegors, Minshew, & Pelphrey, 2011). In high eye contact conditions, individuals with ASD showed relatively normal levels of fusiform activation. Results such as these suggest preserved function in socially selective regions that may be accessible through top-down strategic manipulation of behaviors. However, it remains to be seen whether such findings are robust or predict social function in individuals.

2.3.4 Key Limitations in Research

The study of the brain function in ASD extends well-beyond face processing, but this subset of

the literature is illustrative of key strengths and limitations of neuroimaging applied to the study of ASD. First, few results have been consistently replicated. For example, studies have found hypo-, hyper-, and normal activation in the fusiform gyrus during different face processing tasks in individuals with ASD relative to typically developing individuals. This variability is due in part to the enormous heterogeneity intrinsic to the disorder. But it is also due to the small sample sizes typical of neuroimaging studies and methodological variation across labs in the choice of task and condition contrast.

2.3.5 Theoretical Models

A number of different overarching theoretical models have been proposed to account for the social difficulties in autism. At the more global level, these have posited deficits in theory of mind (Baron-Cohen, Leslie, & Frith, 1985), difficulties in executive functioning (Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009), central coherence (Happé, Briskman, & Frith, 2001), the extreme male brain (Baron-Cohen, 2002), and enactive mind (Klin, Jones, Schultz, & Volkmar, 2014). As a practical matter, these theories have been helpful in stimulating research, even though they have their limitations and often significantly overlap to some degree (South, Ozonoff, & McMahon, 2007; for a comprehensive review, see Chown, 2016).

At a more granulate theoretical level, a range of models have been proposed. Understanding differences in face perception and eye gaze has been a central focus of this effort with a number of competing models proposed. For example, (1) it has been proposed that eye contact and gaze are actively avoided because it is experienced as aversive (Hutt, Hutt, Lee, & Ounsted, 1964), or (2) that due to hypoactivation of structures, like the amygdala, eye contact is not associated with a positive social experience (Shultz, Klin, & Jones, 2011), or (3) that eye contact is much less salient and thus interferes with social communication (Senju, Southgate, White, & Frith, 2009), or (4) that it is disturbance related to difficulties in

subcortical and cortical brain systems pathways that process social information (Kliemann, Dziobek, Hatri, Baudewig, & Heekeren, 2012).

2.3.6 Implications for Treatment

It has long been recognized that the social learning problems of autism were rightly a major focus of treatment. The highly influential report from the National Research Council on Educating Young Children with Autism (2001) highlighted the importance of enhanced social engagement for learning and developmental progress. Over time, a number of different approaches for enhancing social skills in general, and relative to specific social processes in particular, have been made (for a comprehensive review of evidence-based approaches, see Ferraioli & Harris, 2011).

Overall, methods for teaching social skills take several forms. At the more general level, approaches for enhancing social skills, vary somewhat, with age. For example, peer inclusion (with some degree of peer training) is helpful particularly in preschool and early school aged children. As children become older, social skills groups (of various types) are used, and with adolescence and adulthood, individual work becomes more common. Various evidence-based methods have been developed for processes as diverse as joint attention, video modeling, imitation, theory of mind skills, etc. and have been utilized with a growing body of work. Some comprehensive models (e.g., the Early Start Denver Model and Pivotal Response Training) emphasize acquisition of important social skills.

The advances made are important, but unfortunately so are the limitations of the available research. Peer inclusion models, adoptions of existent treatment approaches (e.g., like cognitive behavioral therapy), and individualized approaches to teaching social skills and social communication skills continue to be important. Unfortunately, the available literature relative to adolescents and adults (where social differences are often acutely experienced) remains quite limited indeed.

2.4 Summary

Researchers have demonstrated that ASD is an early onset neurodevelopmental disorder characterized from the beginning by a profound social disability; this impacts the developing child's motivation and ability to undemand others and develop truly reciprocal relationships. It has far reaching impact on styles of learning and information processing. Although progress has been made in identifying potential neural mechanisms, the brain basis of the condition remains complex and relatively poorly understood. On the other hand, the work available has demonstrated some of the potential "downstream" impacts of this social disability. In some respects, it may, perhaps, be better to regard autism and related conditions as disorders of social learning. From a practical standpoint, it is clear that the fundamental principles of treatment are to minimize negative aspects of autism and their impact on learning, and maximize, to the extent possible, both more normative developmental processes and alternative pathways to social leaning. As with basic research on brain mechanisms, a body of intervention research has now developed (Reichow & Barton, 2014; Reichow, Doehring, Cicchetti, & Volkmar, 2011) and, for many, but sadly not all, individual outcome appears to be improving (Fein et al., 2013). In this chapter, we have reviewed the clinical manifestations of social dysfunction in autism as well as potential neural mechanisms. We have also summarized some aspects of intervention and theory as they relate to this issue. Clearly, a major theme of this entire body of work has been to more precisely delineate what is likely to be a highly heterogeneous social phenotype. Hopefully, with changes in improved methods of characterization and the use of more ecologically valid methods for examining specific mechanisms of social dysfunction, progress will continue and lead to more truly comprehensive theories. Future work must be truly interdisciplinary and transdisciplinary for this to be achieved.

References

- Ahmed, A. A., & Vander Wyk, B. C. (2013). Neural processing of intentional biological motion in unaffected siblings of children with autism spectrum disorder: An fMRI study. *Brain and Cognition*, 83(3), 297–306. doi:10.1016/j.bandc.2013.09.007
- American Psychiatric Association. (1980). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Asperger, H. (1944). Die "autistischen Psychopathen" im Kindersalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Bandura, A. (1962). Social learning through imitation. In M. R. Jones (Ed.), *Nebraska symposium on motivation*. Lincoln, NE: University of Nebraska Press.
- Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences*, 6, 248–254.
- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a "theory of mind?". *Cognition*, 21(1), 37–46.
- Bartak, L., & Rutter, M. (1976). Differences between mentally retarded and normally intelligent autistic children. *Journal of Autism and Childhood Schizophrenia*, 6(2), 109–120.
- Bauer, R. M. (1984). Autonomic recognition of names and faces in prosopagnosia: A neuropsychological application of the guilty knowledge test. *Neuropsychologia*, 22(4), 457–469.
- Beauchamp, M. S., Lee, K. E., Haxby, J. V., & Martin, A. (2003). fMRI responses to video and point-light displays of moving humans and manipulable objects. *Journal of Cognitive Neuroscience*, 15(7), 991–1001.
- Bigelow, A. E. (2003). The development of joint attention in blind infants. *Development and Psychopathology*, 15(2), 259–275.
- Blake, R., Turner, L. M., Smoski, M. J., Pozdol, S. L., & Stone, W. L. (2003). Visual recognition of biological motion is impaired in children with autism. *Psychological Science*, 14(2), 151–157.
- Bonda, E., Petrides, M., Ostry, D., & Evans, A. (1996). Specific involvement of human parietal systems and the amygdala in the perception of biological motion. *The Journal of Neuroscience*, 16(11), 3737–3744.
- Breiter, H. C., Etcoff, N. L., Whalen, P. J., Kennedy, W. A., Rauch, S. L., Buckner, R. L., & Rosen, B. R. (1996). Response and habituation of the human amygdala during visual processing of facial expression. *Neuron*, 17(5), 875–887.
- Brothers, L. (1990). The neural basis of primate social communication. *Motivation and Emotion*, 14(2), 81–91.
- Brothers, L. (2002). The social brain: A project for integrating primate behavior and neurophysiology in a new domain. In J. T. Cacioppo (Ed.), *Foundations in social neuroscience* (pp. 367–385). Cambridge: MIT Press.
- Buccino, G., Binkofski, F., Fink, G. R., Fadiga, L., Fogassi, L., Gallese, V., ... Henson, R. N. (2007).

- Separate coding of different gaze directions in the superior temporal sulcus and inferior parietal lobule. *Current Biology*, 17(1), 20–25.
- Carter, E. J., & Pelphrey, K. A. (2006). School-aged children exhibit domain-specific responses to biological motion. *Social Neuroscience*, 1(3–4), 396–411.
- Castelli, F., Happé, F., Frith, U., & Frith, C. (2000). Movement and mind: A functional imaging study of perception and interpretation of complex intentional movement patterns. *NeuroImage*, 12(3), 314–325.
- Chan, A. W., Peelen, M. V., & Downing, P. E. (2004). The effect of viewpoint on body representation in the extrastriate body area. *Neuroreport*, 15(15), 2407–2410.
- Chawarska, K., Macari, S., & Shic, F. (2013). Decreased spontaneous attention to social scenes in 6-month-old infants later diagnosed with autism spectrum disorders. *Biological Psychiatry*, 74(3), 195–203.
- Chawarska, K., Macari, S., Volkmar, F. R., Kim, S. H., & Shic, F. (2014a). ASD in infants and toddlers. In *Handbook of autism and pervasive developmental disorders, Volume 1: Diagnosis, development, and brain mechanisms* (pp. 121–147). Hoboken, NJ: John Wiley & Sons Inc.
- Chawarska, K., Macari, S. L., Volkmar, F. R., Kim, S. H., Shic, F., Paul, R., ... Pelphrey, K. A. (2014b). ASD in infants and toddlers. In *Handbook of autism and pervasive developmental disorders* (4th ed.). Hoboken, NJ: John Wiley & Sons, Inc.
- Chawarska, K., & Shic, F. (2009). Looking but not seeing: Atypical visual scanning and recognition of faces in 2 and 4-year-old children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 39(12), 1663–1672.
- Chawarska, K., Volkmar, F. R., & Klin, A. (2010). Limited attentional bias for faces in toddlers with autism spectrum. *Archives of General Psychiatry*, 67(2), 178–185.
- Chown, N. (2016). *Understanding and evaluating autism theory*. London, UK: Jessica Kingsley.
- Claussen, A. H., Mundy, P. C., Mallik, S. A., & Willoughby, J. C. (2002). Joint attention and disorganized attachment status in infants at risk. *Development and Psychopathology*, 14(2), 279–291.
- Corbett, B. A., Constantine, L. J., Hendren, R., Rocke, D., & Ozonoff, S. (2009). Examining executive functioning in children with autism spectrum disorder, attention deficit hyperactivity disorder and typical development. *Psychiatry Research*, 166(2–3), 210–222.
- Cosmides, L., & Tooby, J. (1992). Cognitive adaptations for social exchange. In J. H. Barkow, L. Cosmides, & J. Tooby (Eds.), *The adapted mind: Evolutionary psychology and the generation of culture* (pp. 163–228). Oxford: Oxford University Press.
- Dawson, G., Webb, S. J., & McPartland, J. (2005). Understanding the nature of face processing impairment in autism: Insights from behavioral and electrophysiological studies. *Developmental Neuropsychology*, 27(3), 403–424.
- Donvan, J., & Zucker, K. (2016). *In a different key: The story of autism*. New York, NY: Penguin.
- Downing, P. E., Jiang, Y., Shuman, M., & Kanwisher, N. (2001). A cortical area selective for visual processing of the human body. *Science*, 293(5539), 2470–2473.
- Fabio Falck-Ytter, T. (2008). Face inversion effects in autism: A combined looking time and pupillometric study. *Autism Research: Official Journal of the International Society for Autism Research*, 1(5), 297–306.
- Farah, M. J., Wilson, K. D., Drain, M., & Tanaka, J. N. (1998). What is “special” about face perception? *Psychological Review*, 105(3), 482–498.
- Fein, D., Barton, M., Eigsti, I. M., Kelley, E., Naigles, L., Schultz, R. T., ... Tyson, K. (2013). Optimal outcome in individuals with a history of autism. *Journal of Child Psychology and Psychiatry*, 54(2), 195–205.
- Ferraioli, B., & Harris, S. (2011). Treatments to increase social awareness and social skills. In B. Reichow, P. Doehring, D. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-based practices and treatments for children with autism* (pp. 171–196). New York, NY: Springer.
- Fodor, J. A. (1986). *The modularity of mind*. Cambridge, MA: MIT press.
- Folstein, S., & Rutter, M. (1977). Infantile autism: A genetic study of 21 twin pairs. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 18(4), 297–321.
- Freitag, C. M., Konrad, C., Häberlen, M., Kleser, C., von Gontard, A., Reith, W., ... Krick, C. (2008). Perception of biological motion in autism spectrum disorders. *Neuropsychologia*, 46(5), 1480–1494.
- Gallagher, H. L., & Frith, C. D. (2003). Functional imaging of ‘theory of mind’. *Trends in Cognitive Sciences*, 7(2), 77–83.
- Gallese, V. (2001). The shared manifold hypothesis. From mirror neurons to empathy. *Journal of Consciousness Studies*, 8(5–6), 33–50.
- Gazzola, V., & Keysers, C. (2009). The observation and execution of actions share motor and somatosensory voxels in all tested subjects: Single-subject analyses of unsmoothed fMRI data. *Cerebral Cortex*, 19(6), 1239–1255.
- Grossman, J. B., Carter, A., & Volkmar, F. R. (1997). Social behavior in autism. *Annals of the New York Academy of Sciences*, 807, 440–454.
- Hadjikhani, N., Joseph, R. M., Snyder, J., Chabris, C. F., Clark, J., Steele, S., ... Feczko, E. (2004). Activation of the fusiform gyrus when individuals with autism spectrum disorder view faces. *NeuroImage*, 22(3), 1141–1150.
- Hadjikhani, N., Joseph, R. M., Snyder, J., & Tager-Flusberg, H. (2007). Abnormal activation of the social brain during face perception in autism. *Human Brain Mapping*, 28(5), 441–449.
- Happé, F., Briskman, J., & Frith, U. (2001). Exploring the cognitive phenotype of autism: Weak “central coherence” in parents and siblings of children with autism: I. Experimental tests. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 42(3), 299–307.
- Happé, F., Ehlers, S., Fletcher, P., Frith, U., Johansson, M., Gillberg, C., ... Frith, C. (1996). Theory of mind in the

- brain. Evidence from a PET scan study of Asperger syndrome. *Neuroreport*, 8(1), 197–201.
- Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2000). The distributed human neural system for face perception. *Trends in Cognitive Sciences*, 4(6), 223–233.
- Howlin, P., Moss, P., Savage, S., Bolton, P., & Rutter, M. (2015). Outcomes in adult life among siblings of individuals with autism. *Journal of Autism and Developmental Disorders*, 45(3), 707–718.
- Hutt, C., Hutt, S. J., Lee, D., & Ounsted, C. (1964). Arousal and childhood autism. *Nature*, 204, 908–909.
- Ingersoll, B., Lewis, E., & Kroman, E. (2007). Teaching the imitation and spontaneous use of descriptive gestures in young children with autism using a naturalistic behavioral intervention. *Journal of Autism and Developmental Disorders*, 37(8), 1446–1456.
- Ingersoll, B., & Schreibman, L. (2006). Teaching reciprocal imitation skills to young children with autism using a naturalistic behavioral approach: Effects on language, pretend play, and joint attention. *Journal of Autism and Developmental Disorders*, 36(4), 487–505.
- Ingersoll, B., Wainer, A., Volkmar, F. R., Paul, R., Rogers, S. J., & Pelphrey, K. A. (2014). The broader autism phenotype. In *Handbook of autism and pervasive developmental disorders* (4th ed.). Hoboken, NJ: John Wiley & Sons.
- Johansson, G. (1973). Visual perception of biological motion and a model for its analysis. *Perception & Psychophysics*, 14(2), 201–211.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kanwisher, N., McDermott, J., & Chun, M. M. (1997). The fusiform face area: A module in human extrastriate cortex specialized for face perception. *The Journal of Neuroscience*, 17(11), 4302–4311.
- Kaplan, J. T., & Lacoboni, M. (2006). Getting a grip on other minds: Mirror neurons, intention understanding, and cognitive empathy. *Social Neuroscience*, 1(3–4), 175–183.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in young children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 47(6), 611–620.
- Kasari, C., Freeman, S. F. N., & Paparella, T. (2001). Early intervention in autism: Joint attention and symbolic play. *International Review of Research in Mental Retardation: Autism*, 23, 207–237.
- Kasari, C., Huynh, L., & Gulsrud, A. C. (2011). Play interventions for children with autism. In S. W. Russ & L. Niec (Eds.), *Play in clinical practice: Evidence-based approaches* (pp. 201–217). New York, NY: Guilford Press.
- Ketter, T. A., George, M. S., Kimbrell, T. A., Benson, B. E., & Post, R. M. (1996). Functional brain imaging, limbic function, and affective disorders. *The Neuroscientist*, 2(1), 55–65.
- Kilner, J. M., & Frith, C. D. (2008). Action observation: Inferring intentions without mirror neurons. *Current Biology*, 18(1), R32–R33.
- Kliemann, D., Dziobek, I., Hatri, A., Baudewig, J., & Heekeren, H. R. (2012). The role of the amygdala in atypical gaze on emotional faces in autism spectrum disorders. *The Journal of Neuroscience*, 32(28), 9469–9476.
- Klin, A., Jones, W., Schultz, R. T., & Volkmar, F. R. (2014). The enactive mind—from actions to cognition: Lessons from autism. In F. R. Volkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (vol. 1, pp. 682–703). Hoboken, NJ: Wiley.
- Klin, A., Jones, W., Schultz, R. T., Volkmar, F. R., & Cohen, D. (2002). Defining and quantifying the social phenotype in autism. *American Journal of Psychiatry*, 159(6), 895–908.
- Klin, A., Sparrow, S. S., de Bildt, A., Cicchetti, D. V., Cohen, D. J., & Volkmar, F. R. (1999). A normed study of face recognition in autism and related disorders. *Journal of Autism and Developmental Disorders*, 29(6), 499–508.
- Klin, A., Volkmar, F. R., & Sparrow, S. S. (1992). Autistic social dysfunction: Some limitations of the theory of mind hypothesis. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, 33(5), 861–876.
- Kling, A. S., & Brothers, L. A. (1992). The amygdala and social behavior. In J. P. Aggleton (Ed.), *The amygdala: Neurobiological aspects of emotion, memory and mental dysfunction* (pp. 353–378). New York, NY: Wiley-Liss.
- Koegel, R. L., & Frea, W. D. (1993). Treatment of social behavior in autism through the modification of pivotal social skills. *Journal of Applied Behavior Analysis*, 26(3), 369–377.
- Kozlowski, L. T., & Cutting, J. E. (1977). Recognizing the sex of a walker from a dynamic point-light display. *Perception & Psychophysics*, 21(6), 575–580.
- Lacoboni, M. (2009). Imitation, empathy, and mirror neurons. *Annual Review of Psychology*, 60, 653–670.
- Lacoboni, M., Woods, R. P., Brass, M., Bekkering, H., Mazziotta, J. C., & Rizzolatti, G. (1999). Cortical mechanisms of human imitation. *Science*, 286(5449), 2526–2528.
- Lingnau, A., Gesierich, B., & Caramazza, A. (2009). Asymmetric fMRI adaptation reveals no evidence for mirror neurons in humans. *Proceedings of the National Academy of Sciences*, 106(24), 9925–9930.
- Lombardo, M. V., Chakrabarti, B., Bullmore, E. T., Baron-Cohen, S., & Consortium, M. A. (2011). Specialization of right temporo-parietal junction for mentalizing and its relation to social impairments in autism. *NeuroImage*, 56(3), 1832–1838.
- Lord, C., Corsello, C., & Grzadzinski, R. (2014). Diagnostic instruments in autistic spectrum disorders. In F. Volkmar, R. Pauls, S. Rogers, & K. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (4th ed. pp. 609–660). Hoboken, NJ: John Wiley & Sons.
- McCarthy, G., Puce, A., Gore, J. C., & Allison, T. (1997). Face-specific processing in the human fusiform gyrus. *Journal of Cognitive Neuroscience*, 9(5), 605–610.

- McPartland, J. C., Tillman, R. M., Yang, D. Y. J., Bernier, R. A., & Pelphrey, K. A. (2014). The social neuroscience of autism spectrum disorder. In *Handbook of autism and pervasive developmental disorders, Volume 1: Diagnosis, development, and brain mechanisms* (pp. 482–496). Hoboken, NJ: John Wiley & Sons Inc.
- McPartland, J. C., Webb, S. J., Keehn, B., & Dawson, G. (2011). Patterns of visual attention to faces and objects in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41(2), 148–157.
- Meltzoff, A. N. (1990). Foundations for developing a concept of self: The role of imitation in relating self to other and the value of social mirroring, social modeling, and self practice in infancy. In D. Cicchetti & M. Beeghly (Eds.), *The self in transition: Infancy to childhood* (pp. 139–164). Chicago, IL: University of Chicago Press.
- Meltzoff, A. N., & Brooks, R. (2001). “like me” as a building block for understanding other minds: Bodily acts, attention, and intention. In B. Malle, L. J. Moses, & D. Baldwin (Eds.), *Intentions and intentionality: Foundations of social cognition* (pp. 171–191). Cambridge: MIT Press.
- Meltzoff, A. N., & Moore, M. K. (1977). Imitation of facial and manual gestures by human neonates. *Science*, 198(4312), 75–78.
- Minshew, N. J., & Williams, D. L. (2007). The new neurobiology of autism: Cortex, connectivity, and neuronal organization. *Archives of Neurology*, 64(7), 945–950.
- Molenberghs, P., Cunnington, R., & Mattingley, J. B. (2012). Brain regions with mirror properties: A meta-analysis of 125 human fMRI studies. *Neuroscience and Biobehavioral Reviews*, 36(1), 341–349.
- Molnar-Szakacs, I., & Overy, K. (2006). Music and mirror neurons: From motion to ‘e’motion. *Social Cognitive and Affective Neuroscience*, 1(3), 235–241.
- Monk, C. S., Peltier, S. J., Wiggins, J. L., Weng, S. J., Carrasco, M., Risi, S., & Lord, C. (2009). Abnormalities of intrinsic functional connectivity in autism spectrum disorders. *NeuroImage*, 47(2), 764–772.
- Mundy, P. (2016). *Autism and joint attention: Development, neuroscience, and clinical fundamentals*. New York: Guilford Press.
- National Research Council (NRC). (2001). *Educating young children with autism*. Washington, D.C: National Academy Press.
- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders*, 24(3), 247–257.
- Ozonoff, S., & Cathcart, K. (1998). Effectiveness of a home program intervention for young children with autism. *Journal of Autism and Developmental Disorders*, 28(1), 25–32.
- Ozonoff, S., & South, M. (2001). Early social development in young children with autism: Theoretical and clinical implications. In G. Bremner & A. Fogel (Eds.), *Blackwell handbook of infant development handbooks of developmental psychology* (pp. 565–588). Malden, MA: Blackwell Publishers.
- Pelphrey, K. A., Mitchell, T. V., McKeown, M. J., Goldstein, J., Allison, T., & McCarthy, G. (2003). Brain activity evoked by the perception of human walking: Controlling for meaningful coherent motion. *Journal of Neuroscience*, 23(17), 6819–6825.
- Pelphrey, K. A., Sasson, N. J., Reznick, J. S., Paul, G., Goldman, B. D., & Piven, J. (2002). Visual scanning of faces in autism. *Journal of Autism and Developmental Disorders*, 32(4), 249–261.
- Pelphrey, K. A., Singerman, J. D., Allison, T., & McCarthy, G. (2003). Brain activation evoked by perception of gaze shifts: The influence of context. *Neuropsychologia*, 41(2), 156–170.
- Perlman, S. B., Hudac, C. M., Pegors, T., Minshew, N. J., & Pelphrey, K. A. (2011). Experimental manipulation of face-evoked activity in the fusiform gyrus of individuals with autism. *Social Neuroscience*, 6(1), 22–30.
- Perrett, D. I., Hietanen, J. K., Oram, M. W., Benson, P. J., & Rolls, E. (1992). Organization and functions of cells responsive to faces in the temporal cortex [and discussion]. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 335(1273), 23–30.
- Philip, R. C., Dauvermann, M. R., Whalley, H. C., Baynham, K., Lawrie, S. M., & Stanfield, A. C. (2012). A systematic review and meta-analysis of the fMRI investigation of autism spectrum disorders. *Neuroscience and Biobehavioral Reviews*, 36(2), 901–942.
- Phillips, M. L., Young, A. W., Senior, C., Brammer, M., Andrew, C., Calder, A. J., ... Williams, S. (1997). A specific neural substrate for perceiving facial expressions of disgust. *Nature*, 389(6650), 495–498.
- Piaget, J. (1952). *Play, dreams and imitation in childhood*. London, UK: Routledge.
- Pierce, K., Müller, R.-A., Ambrose, J., Allen, G., & Courchesne, E. (2001). Face processing occurs outside the fusiform ‘face area’ in autism: Evidence from functional MRI. *Brain*, 124(10), 2059–2073.
- Puce, A., & Perrett, D. (2003). Electrophysiology and brain imaging of biological motion. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, 358(1431), 435–445.
- Raleigh, M. J., & Steklis, H. D. (1981). Effects of orbitofrontal and temporal neocortical lesions on the affiliative behavior of vervet monkeys (*Cercopithecus Aethiops Sabaeus*). *Experimental Neurology*, 73(2), 378–389.
- Reichow, B., & Barton, E. E. (2014). Evidence-based psychosocial interventions for individuals with autism spectrum disorders. In *Handbook of autism and pervasive developmental disorders, Volume 2: Assessment, interventions, and policy* (pp. 969–992). Hoboken, NJ: John Wiley & Sons Inc.
- Reichow, B., Doehring, P., Cicchetti, D., & Volkmar, F. R. (Eds.). (2011). *Evidence-based practices and treatments for children with autism*. New York, NY: Springer.
- Ricks, D. M., & Wing, L. (1975). Language, communication, and the use of symbols in normal and autistic chil-

- dren. *Journal of Autism and Childhood Schizophrenia*, 5(3), 191–221.
- Rizzolatti, G. (2005). The mirror neuron system and its function in humans. *Anatomy and Embryology*, 210(5), 419–421.
- Rizzolatti, G., Fogassi, L., & Gallese, V. (2001). Neurophysiological mechanisms underlying the understanding and imitation of action. *Nature Reviews Neuroscience*, 2(9), 661–670.
- Rogers, S. J. (2005). Play interventions for young children with autism spectrum disorders. In L. Reddy, T. Files-Hall, & Schaefer (Eds.), *Empirically based play interventions for children* (pp. 215–239). Washington, DC: American Psychological Association.
- Rogers, S. J., Cook, I., & Meryl, A. (2014). Imitation and play in autism. In F. R. Volkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (vol. 1, pp. 382–405). Hoboken, NJ: Wiley.
- Rogers, S. J., Dawson, G., & Vismara, L. A. (2012). *An early start for your child with autism: Using everyday activities to help kids connect, communicate, and learn*. New York, NY: Guilford Press.
- Rogers, S. J., Hayden, D., Hepburn, S., Charlifue-Smith, R., Hall, T., & Hayes, A. (2006). Teaching young nonverbal children with autism useful speech: A pilot study of the Denver model and PROMPT interventions. *Journal of Autism and Developmental Disorders*, 36(8), 1007–1024.
- Rogers, S. J., Ozonoff, S., & Maslin-Cole, C. (1991). A comparative study of attachment behavior in young children with autism or other psychiatric disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 30(3), 483–488.
- Rogers, S. J., Ozonoff, S., & Maslin-Cole, C. (1993). Developmental aspects of attachment behavior in young children with pervasive developmental disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32(6), 1274–1282.
- Rogers, S. J., & Pennington, B. F. (1991). A theoretical approach to the deficits in infantile autism. *Development and Psychopathology*, 3(2), 137–162.
- Rogers, S. J., & Vismara, L. (2014). Interventions for infants and toddlers at risk for autism spectrum disorder. In *Handbook of autism and pervasive developmental disorders, Volume 2: Assessment, interventions, and policy* (pp. 739–769). Hoboken, NJ: John Wiley & Sons Inc.
- Rutter, M. (1978). Diagnosis and definitions of childhood autism. *Journal of Autism and Developmental Disorders*, 8(2), 139–161.
- Rutter, M. (2008). Implications of attachment theory and research for child care policies. In *Handbook of attachment: Theory, research, and clinical applications* (2nd ed. pp. 958–974). New York, NY: Guilford Press.
- Rutter, M., & Thapar, A. (2014). Genetics of autism spectrum disorders. In *Handbook of autism and pervasive developmental disorders, Volume 1: Diagnosis, development, and brain mechanisms* (pp. 411–423). Hoboken, NJ: John Wiley & Sons Inc.
- Saxe, R., & Kanwisher, N. (2003). People thinking about thinking people: The role of the temporo-parietal junction in “theory of mind”. *NeuroImage*, 19(4), 1835–1842.
- Schultz, R. T., Gauthier, I., Klin, A., Fulbright, R. K., Anderson, A. W., Volkmar, F., ... Gore, J. C. (2000). Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and Asperger syndrome. *Archives of General Psychiatry*, 57(4), 331–340.
- Senju, A., Southgate, V., White, S., & Frith, U. (2009). Mindblind eyes: An absence of spontaneous theory of mind in Asperger syndrome. *Science*, 325(5942), 883–885.
- Shultz, S., Klin, A., & Jones, W. (2011). Inhibition of eye blinking reveals subjective perceptions of stimulus salience. *PNAS Proceedings of the National Academy of Sciences of the United States of America*, 108(52), 21270–21275.
- Signer, S. F. (1987). Capgras’ syndrome: The delusion of substitution. *Journal of Clinical Psychiatry*, 15, 402–440.
- Silani, G., Bird, G., Brindley, R., Singer, T., Frith, C., & Frith, U. (2008). Levels of emotional awareness and autism: An fMRI study. *Social Neuroscience*, 3(2), 97–112. 15:402-440.
- South, M., Ozonoff, S., & McMahon, W. M. (2007). The relationship between executive functioning, central coherence, and repetitive behaviors in the high-functioning autism spectrum. *Autism*, 11(5), 437–451.
- Tanaka, J. W., Wolf, J. M., Klaiman, C., Koenig, K., Cockburn, J., Herlihy, L., ... Schultz, R. T. (2012). The perception and identification of facial emotions in individuals with autism spectrum disorders using the let's face it! Emotion skills battery. *Journal of Child Psychology and Psychiatry*, 53(12), 1259–1267.
- Thirtamara Rajamani, K. K. (2015). Animal models of drug addiction and autism spectrum disorders. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 76(5-B(E)), No Pagination Specified.
- Turella, L., Pierno, A. C., Tubaldi, F., & Castiello, U. (2009). Mirror neurons in humans: Consisting or confounding evidence? *Brain and Language*, 108(1), 10–21.
- Uddin, L. Q., Iacoboni, M., Lange, C., & Keenan, J. P. (2007). The self and social cognition: The role of cortical midline structures and mirror neurons. *Trends in Cognitive Sciences*, 11(4), 153–157.
- Ungerer, J. A., & Sigman, M. (1981). Symbolic play and language comprehension in autistic children. *Journal of the American Academy of Child Psychiatry*, 20(2), 318–337.
- Valentine, T. (1988). Upside-down faces: A review of the effect of inversion upon face recognition. *British Journal of Psychology*, 79(4), 471–491.
- Vander Wyk, B. C., Hudac, C. M., Carter, E. J., Sobel, D. M., & Pelphrey, K. A. (2009). Action understanding in the superior temporal sulcus region. *Psychological Science*, 20(6), 771–777.

- Vander Wyk, B. C., Voos, A., & Pelphrey, K. A. (2012). Action representation in the superior temporal sulcus in children and adults: An fMRI study. *Developmental Cognitive Neuroscience*, 2(4), 409–416. doi:[10.1016/j.dcn.2012.04.004](https://doi.org/10.1016/j.dcn.2012.04.004)
- Volkmar, F. R., Klin, A., Siegel, B., Szatmari, P., Lord, C., Campbell, M., . . . et al. (1994). Field trial for autistic disorder in DSM-IV. *American Journal of Psychiatry*, 151(9), 1361–1367.
- Volkmar, F. R., & McPartland, J. C. (2014). From Kanner to DSM-5: Autism as an evolving diagnostic concept. *Annual Review of Clinical Psychology*, 10, 193–212.
- Volkmar, F. R., & Nelson, D. S. (1990). Seizure disorders in autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 29(1), 127–129.
- Volkmar, F. R., & Reichow, B. (2014). The evolution of autism as a diagnostic concept: From Kanner to DSM-5: A commentary. In *Handbook of autism and anxiety* (pp. 217–230). Cham, Switzerland: Springer International Publishing.
- Volkmar, F. R., Sparrow, S. S., Goudreau, D., Cicchetti, D. V., Paul, R., & Cohen, D. J. (1987). Social deficits in autism: An operational approach using the vineland adaptive behavior scales. *Journal of the American Academy of Child and Adolescent Psychiatry*, 26(2), 156–161.
- Völlm, B. A., Taylor, A. N., Richardson, P., Corcoran, R., Stirling, J., McKie, S., . . . Elliott, R. (2006). Neuronal correlates of theory of mind and empathy: A functional magnetic resonance imaging study in a nonverbal task. *NeuroImage*, 29(1), 90–98.
- Voos, A. C., Pelphrey, K. A., Tirrell, J., Bolling, D. Z., Vander Wyk, B., Kaiser, M. D., . . . Ventola, P. (2013). Neural mechanisms of improvements in social motivation after pivotal response treatment: Two case studies. *Journal of Autism and Developmental Disorders*, 43(1), 1–10.
- Wang, A. T., Dapretto, M., Hariri, A. R., Sigman, M., & Bookheimer, S. Y. (2004). Neural correlates of facial affect processing in children and adolescents with autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43(4), 481–490.
- Williams, J. H., Whiten, A., Suddendorf, T., & Perrett, D. I. (2001). Imitation, mirror neurons and autism. *Neuroscience and Biobehavioral Reviews*, 25(4), 287–295.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9(1), 11–29.
- Wing, L., Gould, J., Yeates, S. R., & Brierley, L. M. (1977). Symbolic play in severely mentally retarded and in autistic children. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 18(2), 167–178.
- Yin, R. K. (1969). Looking at upside-down faces. *Journal of Experimental Psychology*, 81(1), 141.
- Young, G. S., Merin, N., Rogers, S. J., & Ozonoff, S. (2009). Gaze behavior and affect at 6 months: Predicting clinical outcomes and language development in typically developing infants and infants at risk for autism. *Developmental Science*, 12(5), 798–814.

Handbook of Social Skills and Autism Spectrum
Disorder

Assessment, Curricula, and Intervention

Leaf, J.B. (Ed.)

2017, XVII, 445 p. 7 illus., Hardcover

ISBN: 978-3-319-62994-0