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Early Identification of Autism:

Early Characteristics, Onset of Symptoms, and Diagnostic Stability

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Abstract

In the first year of life, infants who later go on to develop autistic spectrum disorders (ASD) may exhibit subtle disruptions in social interest and attention, communication, temperament, and head circumference growth that occur prior to the onset of clinical symptoms. These disruptions may reflect the early course of ASD development and may also contribute to the later development of clinical symptoms through alterations in the child's experience of his or her environment. By age 2, developmental precursors of autism symptoms can be used to diagnose children reliably, and by age 3, the diagnosis is thought to be relatively stable. The downward extension of the autism diagnosis poses important questions for therapists in designing interventions that are applicable for infants who demonstrate early risk factors. We review current knowledge of the early signs of ASD in the infancy period (0–12 months) and the manifestation of symptoms in toddlerhood (12–36 months), noting the importance of considering the variability in onset and trajectory of ASD. Finally, we consider the implications of this emerging research for those who work or interact with young children, including the importance of early monitoring and the development and evaluation of age-appropriate interventions.

Keywords

autism; cognition; communication; development; infancy; psychopathology; risk; social

In 2000, the American Academy of Neurology and Child Neurology recommended that all children be screened for an autistic spectrum disorder (ASD) (Filipek et al., 2000). Autism spectrum disorders, including autistic disorder, pervasive developmental disorder not-otherwise-specified, and Asperger's disorder, are developmental disorders defined by impairments in social interaction and communication and the presence of a restricted repertoire of behavioral activities and interest (*Diagnostic and Statistical Manual of Mental Disorders* (4th ed) [*DSM-IV*]). Notably, boys are affected 3 to 4 times more often than girls (eg, Fombonne, 1999; Lord & Volkmar, 2002). Although ASD is not typically diagnosed until the age of 3–4 years, clear evidence for genetic involvement in the etiology of ASD (for review, see Bespalova & Buxbaum, 2003; Veenstra-Vanderweele, Christian, & Cook, 2004) indicates that risk factors are present from birth. Indeed, 30% of parents of children with

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ASD identify concerns prior to 1 year, and 80% identify problems by 2 years of age (Baghdadli, Pascal, Grisi, & Aussilloux, 2003; De Giacomo & Fombonne, 1998). Studying very early development may thus facilitate earlier detection and earlier intervention for children with ASD and their families. The following sections review what is known about the early characteristics of children with autism in the first year of life, the autism diagnosis in toddlers, and the stability of early diagnosis. We end by discussing the implications of this body of research for those working or interacting with infants and toddlers.

EARLY CHARACTERISTICS OF ASD IN INFANTS

Brain development is shaped not only by the interaction between the child's genes and his or her environment, but also by the way in which the child's behavior influences the environment he or she experiences (Westermann et al., 2007). Thus, early symptoms of ASD may reflect and contribute to abnormal trajectories of brain development (Dawson, Sterling, & Faja, in press; Mundy & Crowson, 1997). This theoretical position has led to intense focus on the early development of individuals with ASD, through complementary retrospective and prospective research strategies. Retrospective studies examine the early development of children who have already been diagnosed with ASD, using methods such as parental report, medical records, or early videotapes. Although these studies provide the majority of our current knowledge of the early development of ASD, their limitations include the possibility of parental bias, limits to the behaviors that can be studied and the precision of their measurement, and variability in the purpose of the original data collection. A more recent research strategy prospectively follows younger siblings of children with ASD, who experience a 2-to 50-fold increase in the risk for developing autism (eg, Landa & Garrett-Mayer, 2006; Volkmar, Lord, Bailey, Schultz, & Klin, 2004; Zwaigenbaum et al., 2005). Younger siblings who develop autism provide a specific opportunity for direct observation of the early development of autism symptoms. However, recent genetics findings suggest different pathways to developing autism in singleton versus multiplex families (Zhao et al., 2007), and little is known whether the course of autism differs in these 2 groups. Thus, it is important to recognize that information gleaned from current prospective studies may apply only to a subgroup of children with ASD. Combining the 2 methods provides the broadest picture of the early development of ASD, and so the following sections review evidence from both retrospective and prospective research methods.

Social interest and attention

Retrospective studies reveal that some infants who go on to be diagnosed with ASD exhibit diminished social attention by 1 year, with poor eye contact, a lack of response to infant-directed speech, decreased reaction to attempts to engage the infant in play or interaction (eg, De Giacomo & Fombonne, 1998; Gillberg et al., 1990; Rogers & DiLalla, 1990; Volkmar, Stier, & Cohen, 1985), and less orienting, smiling, and vocalizing to people (Maestro et al., 2001, 2005; Maestro, Casella, Milone, Muratori, & Palacio-Espasa, 1999). Other social behaviors that may be disrupted include social smiling, expressiveness, and affective responses to social touch (eg, Adrien et al., 1992, 1993; Bernabei, Camaigni, & Levi, 1998; Mars, Mauk, & Dowrick, 1998; Osterling & Dawson, 1994; Werner, Dawson,

Osterling, & Dinno, 2000; Zakian, Malvy, Desombre, Roux, & Lenoir, 2000). Similarly, in prospective studies researchers have noted lower levels of orientation to social stimuli such as faces or voices, poorer imitation, poor eye contact, lack of social interest, less social smiling, and reduced expression of positive emotion (Bryson et al., 2007; Landa, Holman, & Garrett-Mayer, 2007; Sullivan et al., 2007; Wetherby, Watt, Morgan, & Shumway, 2007; Yirmiya et al., 2006; Zwaigenbaum et al., 2005). These disruptions in social attention and social enjoyment likely diminish infants' opportunity to learn about and from the people around them, and may indicate cause for concern when observed by parents or practitioners.

A particularly striking finding is the failure of 8- to 12-month-old infants who later develop autism to orient to verbalization and to their name, in comparison to both typically developing and developmentally delayed toddlers (eg, Baranek, 1999; Bernabei et al., 1998; Dawson, Meltzoff, Osterling, Rinaldi, & Brown, 1998; Maestro et al., 2001; Mars et al., 1998; Mundy & Neal, 2001; Tantam, 1992; Volkmar, Chawarska, & Klin, 2005; Werner et al., 2000; Zakian et al., 2000). Differences in responding to own name have also been observed in prospective studies (eg, Zwaigenbaum et al., 2005). However, Nadig et al. (2007) found that half of a group of children who went on to develop ASD showed a typical response to own name at 12 months, and similar proportions of children who failed to respond to their name at 12 months went on to develop other developmental delays as developed ASD. Failures to respond to own name may thus provide a "red flag"indicating the need for further evaluation, but may not be specifically related to a later diagnosis of ASD.

Communication

Although many typically developing children produce few words by the end of the first year, other forms of communication are more advanced, allowing delays or disruptions to be more clearly noted. Retrospective studies have found that infants who go on to develop ASD may exhibit delays in sound production (Maestro et al., 2002; Wetherby & Prizant, 1998), vocal quality (Sheinkopf, Mundy, Oller, & Steffens, 2000), and produce less simple and complex babbling at 12 months (Werner & Dawson, 2005). Early disruptions may also be seen in both the production of and response to communicative gestures like pointing to share interest (Adrien et al., 1993; Maestro et al., 2001, 2002; Osterling & Dawson, 1994; Osterling, Dawson, & Munson, 2002; Werner & Dawson, 2005). Communicative gazing and pointing are critical to the formation and maintenance of shared awareness of an event or object between the child and another individual, or "joint attention," which is critical to later language development (eg, Carpenter, Pennington, & Rogers, 2002; Charman, Baron-Cohen, et al., 2003; Mundy, Sigman, & Kasari, 1990; Stone & Yoder, 2001; Toth, Munson, Meltzoff, & Dawson, 2006). Prospective studies have also noted failures to respond to joint attention (Sullivan et al., 2007), and lower levels of language and gesture production (Landa & Garrett-Mayer, 2006; Mitchell et al., 2006) in infants who are later diagnosed with ASD. Early impairments in language precursors such as gesture or babbling may thus contribute to later language deficits in children with ASD and provide potential risk markers for further assessment.

Repetitive behaviors and other domains

Repetitive behavior may be less commonly observed in the early development of children with ASD than are social and communicative disruptions (Lord, 1995; Moore & Goodson, 2003; Stone et al., 1999; Werner & Dawson, 2005). Nonetheless, some indications of abnormal behaviors have been observed. For example, in retrospective studies, Baranek (1999) observed increased object mouthing and unusual postures in the first year of life in children with ASD, Maestro et al. (2002) observed a trend toward increased exploratory activities with objects between birth and 6 months, and Osterling and Dawson (1994) observed more self-stimulatory behaviors. Other noted motor abnormalities include decreased motility, abnormal muscle tone, and unusual posture or movement patterns (Adrien et al., 1992, 1993; Teitelbaum, Teitelbaum, Nye, Fryman, & Maurer, 1998; though, see Ozonoff et al., 2008). Prospective studies have also observed potential precursors of repetitive behavior, such as more limited toy play (Landa et al., 2007; Wetherby et al., 2004) and increased atypical motor mannerisms (such as arm waving) (Loh et al., 2007; Wetherby et al., 2004). Precursors of the repetitive behaviors observed in children with ASD may thus be apparent in the infancy period but are presently less useful as risk markers.

Early disruptions potentially associated with ASD have also been observed in the domains of temperament and sensory responsivity. Both retrospective and prospective studies have revealed that infants who go on to develop ASD may display increased passivity, being almost “too good,”or conversely increased irritability (Adrien et al., 1993; Bryson et al., 2007; Stone & Lemanek, 1990; Zwaigenbaum et al., 2005). Retrospective studies have reported altered sensory responses, with observations of both *hyper*reactivity to sound (Dahlgren & Gillberg, 1989; Ornitz, Guthrie, & Farley, 1977) and *hypo*reactivity to visual stimulation (Baranek, 1999; Baranek, David, Poe, Stone, & Watson, 2006). However, disruptions in motor abilities and sensory response are also common in children with developmental delay (eg, Baranek et al., 2006; Osterling et al., 2002; Ozonoff et al., 2008), indicating that disruptions in these domains may provide less specific early markers of ASD. An important finding from recent prospective studies is that visual attention may be “stickier” in infants who develop ASD, with atypically prolonged fixation or slowed orientation (Bryson et al., 2007; Wetherby et al., 2004; Zwaigenbaum et al., 2005). Alterations in visual attention may alter the way in which infants experience their environment, potentially impacting later development.

Both retrospective and prospective studies have indicated that one of the first early biological risk indices for ASD may be atypical growth in head circumference, which is likely to reflect atypical brain growth (Bartholomeusz, Courchesne, & Karns, 2002) and may overlap the onset of symptoms (Dawson et al., 2007). Although at birth, children who are later diagnosed with ASD have been shown to have small to normal head circumferences (eg, Courchesne, Carper, & Akshoomoff, 2003; Dawson et al., 2007; Redcay & Courchesne, 2005; Webb et al., 2007; but see Lainhart et al., 1997; Torrey, Dhavale, Lawlor, & Yolken, 2004), at age 2–4 years, both head circumference and brain volume may be enlarged (Bloss & Courchesne, 2007; Redcay & Courchesne, 2005; Sparks et al., 2002). Growth may be particularly accelerated in the infancy period: Webb et al. (2007) identified a 7- to 9-month time window during which head growth was particularly accelerated in comparison to the

Centers for Disease Control and Prevention norms (see also Courchesne et al., 2003). Growth rate may slow down after 12 months, correlated to a slowing in acquisition or loss in skills in infants with autism (Elder, Dawson, Toth, Fein, & Munson, 2008). Careful monitoring of head circumference may thus provide additional information concerning risk for ASD during infancy.

Although the research reviewed in this section clearly indicates that signs of ASD are present during infancy for some children, it is notable that an estimated 1 of 5 children with ASD appears to display a normal developmental trajectory during the first year (Filipek et al., 1999) and 20% to 49% of children with ASD also exhibit significant regression or skill loss (Davidovitch, Glick, Holtzman, Tirosh, & Safir, 2000; Goldberg et al., 2003; Luyster et al., 2005; Ozonoff, Williams, & Landa, 2005; Tuchman & Rapin, 1997). It is thus important to bear in mind that the absence of early risk markers does not rule out the later development of ASD symptoms.

CHARACTERISTICS OF ASD IN TODDLERS

Although research on ASD during infancy is currently relatively scarce, there is a wealth of evidence concerning the manifestation of ASD in the toddler years (between 12 months and 3 years). This is partly due to increased parental recognition of ASD in this time period; by 24 months, 90% of parents recognize that their children's development is abnormal (De Giacomo & Fombonne, 1998), and most families initially express concern to a pediatrician by 18 months (Howlin & Moore, 1997; Siegel, Pliner, Eschler, & Elliot, 1988). The following sections briefly review what this research has revealed about the symptoms of ASD in the toddler years.

Social interaction

Social impairments seen in toddlers with ASD resemble those considered core components of the disorder in older children and adults. For example, toddlers with ASD may show reduced attention to social stimuli such as faces and voices (Dahlgren & Gillberg, 1989; Dawson et al., 1998; Klin, Jones, Schultz, Volkmar, & Cohen, 2002a, 2002b; Lord, 1995; Swettenham et al., 1998) and may show marked deficits in orienting to social stimuli that exceed those seen in orienting to nonsocial stimuli, or those observed in children with developmental delay (Dawson et al., 1998, 2004). Diagnostic symptoms found in older children, such as limited eye contact, limited overall social engagement and responsivity, and increased tendency for isolation, are also seen in toddlers with autism (Adrien et al., 1993; Dawson, Osterling, Meltzoff, & Kuhl, 2000; Maestro et al., 1999; Sparling, 1991). The presence of many of the social impairments that are central to an ASD diagnosis is one contributing factor to the increase in the reliability of an ASD diagnosis made during toddlerhood.

Language and communication

Although it is important to recognize that there is extensive variability in normative language development (Fenson et al., 1994), language delays are some of the most commonly reported parental concerns in the early development of children with ASD (Chawarska et al.,

2007; De Giacomo & Fombonne, 1998; DeMyer, 1979; McConkey, Truesdale-Kennedy, & Cassidy, 2008). In addition to the estimated 20% to 25% of children with ASD who remain nonverbal (Lord, Shulman, & DiLavore, 2004; Lord, Risi, & Pickles, 2004; Sigman, 1998; Sigman & McGovern, 2005), others may not produce their first word until 18 months or older (Lord, Risi, et al., 2004; Lord, Shulman, et al., 2004; Ornitz et al., 1977; Rogers & DiLalla, 1990). Toddlers with ASD may also show reduced babbling, complex vocalization, and less vocal imitation, and use fewer single words and phrases (eg, Goldberg et al., 2005; Landa & Garrett-Mayer, 2006; Mitchell et al., 2006; Werner & Dawson, 2005; Wetherby et al., 2007; Zwaigenbaum et al., 2005), although use of single words becomes a relative strength over time for many individuals with ASD (Lord & Paul, 1997). Where speech is present, it may be stereotyped or echolalic (Maestro et al., 1999), or favor syllables with atypical phonation (Sheinkopf et al., 2000). Receptive language, and pragmatic aspects of language, may be particularly impaired (Charman, Drew, Baird, & Baird, 2003; Landa & Garret-Mayer, 2006; Rapin & Dunn, 2003), and toddlers may show unusual preferences for listening to mechanical signals rather than infant-directed speech (Kuhl, Coffey-Corina, Padden, & Dawson, 2004).

Other aspects of communication may also be impaired. Although 2- to 4-year-old toddlers with ASD may be equally likely to communicate to request or protest, they may be less likely than typically developing toddlers to coordinate gestures with vocalizations and eye gaze, or to communicate for joint attention (eg, Mundy et al., 1990; Stone, Ousley, & Littleford, 1997; Wetherby & Prizant, 1998; Wetherby et al., 2004). Indeed, difficulties in joint attention and imitation have been proposed as core components of ASD (eg, Dawson & Adams, 1984; Mundy, 2003; Rogers, Bennetto, McEvoy, & Pennington, 1996; Sigman, Dijamco, Gratier, & Rozga, 2004; Williams, Whiten, Suddendorf, & Perrett, 2001). Joint attention difficulties (such as following a point or giving an object) are commonly reported by parents (Wimpory, Hobson, Williams, & Nash, 2000), and have been identified in many observational studies (eg, Baron-Cohen, Allen, & Gillberg, 1992; Maestro et al., 2001; Mundy & Neal, 2001; Sigman & Ruskin, 1999; Werner & Dawson, 2005; Wetherby, Prizant, & Schuler, 2000). Toddlers with ASD are also less likely to show pleasure during joint attention episodes (Baron-Cohen et al., 1992; Hoshino et al., 1982; Lord, 1995; Wetherby et al., 2004) and are less likely to initiate joint attention to request assistance from others (Charman et al., 1997; Landa et al., 2007; Wetherby et al., 2004, 2007). Toddlers with ASD are less likely to imitate others, reducing their opportunities to learn from social situations (Rogers, Hepburn, Stackhouse, & Wehner, 2003; Stone et al., 1997; Stone, Lemanek, Fishel, Fernandez, & Altemeier, 1990). Finally, differences in play have been noted, with play being less complex, less purposeful, and less symbolic in toddlers with ASD (Baron-Cohen et al., 1996; Dawson et al., 1998; McDonough, Stahmer, Schreibman, & Thompson, 1997; Mundy et al., 1986; Sigman & Ruskin, 1999; Stone et al., 1990). Abnormalities in social aspects of communication may negatively impact engagement with the social environment.

Repetitive behavior and other domains

Although social and communication impairments are the clearest features of ASD in toddlerhood, impairments in other domains have been noted. For example, repetitive or unusual motor behaviors, such as repeated play with an object or repetitive body

movements, may become more apparent between 12 months and 3 years (Maestro et al., 1999; Richler, Bishop, Kleinke, & Lord, 2007; Watson et al., 2007; Watt, Wetherby, Barber, & Morgan, 2008; Werner, Dawson, Munson, & Osterling, 2005; Wetherby et al., 2004). There is some evidence that motor skills in general may be delayed (Landa & Garett-Mayer, 2006), although possibly not more than in those with other developmental disabilities (Provost, Lopez, & Heimerl, 2007; Rogers et al., 2003). Repetitive behaviors around the age of 2 years may predict later diagnosis (Lord et al., 2006; Mooney, Gray, & Tonge, 2006; Morgan, Wetherby, & Barber, 2008) but may have limited sensitivity and specificity until after the age of 3 years (Baron-Cohen et al., 1992; Charman et al., 2005; Lord, 1995; Richler et al., 2007; Stone et al., 1999; Werner & Dawson, 2005). Finally, temperament-related behavioral problems may also be observed, with parents reporting these as some of their earliest concerns about their toddlers (McConkey et al., 2008). Thus, toddlers with ASD may exhibit deficits in a range of domains that extend beyond social and communicative problems.

STABILITY OF EARLY DIAGNOSIS

Diagnoses are made through clinical judgment in combination with comprehensive behavioral assessments using developmentally appropriate versions of the clinically standardized measures: the Autism Diagnostic Interview–Revised (Lord, Rutter, & Le Couteur, 1994); the ADOS (Autism Diagnostic Observation Scale; Lord, Rutter, DiLavore, & Risi, 1999), and the *DSM-IV* criteria (American Psychological Association, 1994). However, clinicians have questioned the applicability of the *DSM-IV* criteria to toddlers with ASD, as young children were underrepresented in the initial field trial of the instrument (Volkmar et al., 1994), and the utility of the restricted behaviors criteria have been questioned for younger age groups (Lord, 1995; Stone et al., 1999). The Autism Diagnostic Interview– Revised may be less accurate for children with a mental age of less than 24 months (Lord et al., 1994) and may underidentify ASD in higher functioning toddlers (Lord et al., 1997). A recent modification of the ADOS for younger children (Gotham, Risi, Pickles, & Lord, 2007) produced sensitivity and specificity scores (when compared to consensus clinician judgment) of between 78% and 97% and 53% and 92%, respectively (Gray, Tonge, & Sweeney, 2008; Gotham et al., 2007). Thus, it is important to recognize that the gold standards of ASD diagnosis may have limitations when used with infants and young toddlers. Increased knowledge of symptom manifestation in very young children has led to the development of new measures such as the AOSI (Autism Observational Scale for Infants; Zwaigenbaum et al., 2005) and the ADOS-Toddler (Lord, Luyster, Gotham, & Guthrie, in press), which may begin to address these concerns. However, it is particularly critical for very young children that an experienced clinician couples judgment with standardized test scores in coming to a diagnostic decision, and diagnoses before the age of 3 years may be more appropriately treated as a “provisional diagnosis.”

Despite the challenges to making reliable early diagnoses, several studies have found that 90% to 100% of children diagnosed with an ASD using standardized measures in combination with clinical judgment between 2 and 3 years received the same diagnosis at a 1- or 2-year follow-up (Charman et al., 2005; Cox et al., 1999; Gillberg et al., 1990; Lord, 1995; Moore & Goodson, 2003; Stone et al., 1999). In a longer-term follow-up, 88% of a

sample of children diagnosed at the age of 2 years remained on the autism spectrum at age 9 years (Turner et al., 2006; see also Charman et al., 2005; Lord et al., 2006). Stability for more specific diagnoses within the spectrum is lower (eg, Charman et al., 2005; Lord et al., 2006), diagnoses are less stable if they are made closer to 2 years (Charman et al., 2005; Turner & Stone, 2007), and it is currently difficult to predict diagnostic stability for individual children (Sutera et al., 2007). Thus, it is important that children who receive an early diagnosis of ASD continue to be closely monitored.

REGRESSION

Regression (or skill loss) was first noted by parents and medical providers (eg, Davidovitch et al., 2000; Goldberg et al., 2003; Kurita, 1985; Lord, 1995; Rogers & DiLalla, 1990; Tuchman & Rapin, 1997) and has since been validated as a phenomenon through the analysis of home-videotapes (Osterling et al., 2002; Werner & Dawson, 2005) and prospective studies (Bryson et al., 2007; Landa et al., 2007). Regression occurs in approximately 10% to 50% of the children with ASD (eg, Baird et al., 2008; Fombonne & Chakrabati, 2001; Kobayashi & Murata, 1998; Lingam et al., 2003; Lord, Shulman, & DiLavore, 2004; Luyster et al., 2005; Rogers & DiLalla, 1990; Tuchman & Rapin, 1997), although notably some retrospective reports of regression may be driven by skill “stagnation” or a failure to progress, rather than absolute skill loss. Mean reported age of regression is around 19 to 21 months, with a range from less than 1 year to greater than 3 years (Davidovitch et al., 2000; Kobayashi & Murata, 1998; Shinnar et al., 2001; Tuchman & Rapin, 1997). Regression may be seen in both early-onset and late-onset ASD and it may be rare for children to be completely developmentally typical before the regression occurred (Lord et al., 2004; Ozonoff et al., 2005; Siperstein & Volkmar, 2004; Werner et al., 2005). Regression has not been linked to family characteristics such as socioeconomic status, ethnicity, birth order, gender, family risk for ASD (Lainhart et al., 2002), or vaccination (Fombonne & Chakrabati, 2001; Richler et al., 2007; Taylor et al., 2002). However, families often report that regression was preceded by a precipitating factor such as a physical illness or psychosocial stressor (Goldberg et al., 2003; Ozonoff et al., 2005; Shinnar et al., 2001).

Regression typically involves some loss of spoken language (Cox et al., 1999; Landa et al., 2007; Lord et al., 1994, 2004; Luyster et al., 2005; Tuchman & Rapin, 1997), although loss of socioemotional reciprocity alone is also observed (Goldberg et al., 2003; Landa et al., 2007; Lord et al., 2004; Luyster et al., 2005; Ozonoff et al., 2005). For example, in a recent study, 17% of 333 children lost both language and social skills, and 41% lost either language or social skills (Hansen et al., 2008). Other symptoms such as sensory or temperamental dysregulation or repetitive and restrictive behaviors may emerge around this time (Bryson et al., 2007; Landa et al., 2007). Evidence is currently mixed with respect to the effect of regression on developmental outcome, with some studies finding poorer outcomes in language, IQ, adaptive skills, and/or severity of ASD symptoms (Bernabei, Cerquiglini, Cortesi, & D’Ardia, 2007; Brown & Prelock, 1995; Kobayashi & Murata, 1998; Kurita, 1985; Richler et al., 2007; Rogers & DiLalla, 1990; Tuchman & Rapin, 1997), whereas other studies find no differences between children who did and did not experience regression (Baird et al., 2008; Fombonne & Chakrabati, 2001; Lord et al., 2004; Short & Shopler, 1988; Tolbert, Brown, Fowler, & Parsons, 2001; Werner et al., 2005). Variations between studies

are likely influenced by fluctuation in the diagnostic criteria and sample construction over time, in addition to factors influencing parent recognition of symptoms or participation in studies (Hansen et al., 2008). Thus, although regression is a clear “red flag” for ASD, the implications of regressive versus nonregressive ASD for the developmental trajectory of individual children is presently unclear. It is however important for practitioners and parents to recognize that although some children never regain skills lost during regression (Lord et al., 2004), language loss does not necessarily indicate poorer language skills later in life, or preclude later language acquisition (Goldberg et al., 2003). For example, a recent report found that at age 9 to 14 years, only 1 child out of 26 who experienced language regression had failed to gain single words (Baird et al., 2008).

IMPLICATIONS FOR PRACTICE

Although much remains unclear about the early development of ASD, current research has several implications for those working or interacting with infants and toddlers. In this section, we discuss 3 specific recommendations for parents, researchers, practitioners, and the wider community.

Monitoring socioemotional development and screening for ASD is critical

The relatively high prevalence of ASD and the existence of identifiable symptoms in the early development of some infants and toddlers indicate that monitoring infants and children for these signs is a critical public health measure. To facilitate developmental monitoring, a number of research-based screeners have been developed to identify children at risk for autism from the general population (eg, Quantitative Checklist for Autism in Toddlers, Developmental Behavior Checklist— Early Screen, Infant Toddler Checklist, First Year Inventory) or to identify children at risk for ASD from a population of children exhibiting general developmental delays (eg, Autism Behavior Checklist, Gilliam Autism Rating Scale, Screening Tool for Autism, Pervasive Developmental Disorders Screening Test–II, Childhood Autism Rating Scale). Indeed, in July 2006, the American Academy of Pediatrics issued a policy statement stating that developmental surveillance be incorporated at every well-child preventive care visit from 9 to 30 months by using a standardized developmental screening test. However, while screeners are a critical tool for detecting signs of ASD in large groups of children, the significant variability in the onset, symptoms, and course of ASD has made it difficult to achieve acceptable sensitivity and specificity in screeners aimed at very young children (see Table 1). A further challenge to the efficacy of widespread screening is parent follow-up; a recent study found that 14% of parents of children who tested positive using a population screener waited for 6 months before seeking further evaluation, and 18% did not seek any further evaluation (Dietz, Swinkels, van Daalen, Van Engeland, & Buitelaar, 2006). Delayed follow-up and lack of follow-up were higher for parents with younger children, children with higher cognitive skill, and children with fewer symptoms. In addition to improving the current battery of screeners, steps may need to be taken to maximize the likelihood that screen-positive children access further evaluation, including parental support and education, increased service availability, and the reduction of financial barriers.

Monitoring and screening should be more intensive when working with high-risk groups

Several groups of infants and young children may be at particularly high risk for ASD, and it is therefore critical that their development is closely monitored. One group at particularly high risk is the younger siblings of children with ASD, who not only are at risk for developing ASD themselves but may also display subtle developmental delays that are characteristic of a "broader autism phenotype". "Broader autism phenotype" usually refers to cognitive or neural characteristics of family members that resemble those found in individuals with ASD. Although the precise nature of the broader autism phenotype is currently undefined for infants and young toddlers, more than 10% of toddler siblings who themselves do not have ASD nonetheless show social and communication deficits (Landa & Garrett-Mayer, 2006; Zwaigenbaum et al., 2005). Particular risk areas for siblings of children with ASD in the infant and toddler years are joint attention (Cassel et al., 2007; Goldberg et al., 2005; Presmanes, Walden, Stone, & Yoder, 2007; Stone, McMahon, Yoder, & Walden, 2007; Yirmiya et al., 2006) and language or gesture production (Iverson & Wozniak, 2007; Mitchell et al., 2006; Stone et al., 2007; Toth, Dawson, Meltzoff, Greenson, & Fein, 2007; Yirmiya et al., 2006; Yirmiya, Gamliel, Shaked, & Sigman, 2007). In addition to an increased risk for ASD, it is thus important to recognize that siblings may face other developmental challenges that require consideration and treatment, and may have more variability in developmental trajectories, although some delays may spontaneously resolve in infancy or early childhood (Gamliel, Yirmiya, & Sigman, 2007).

In addition to infants at familial risk for ASD, other groups may be more vulnerable due to their medical status. For example, extreme prematurity has recently been associated with increased risk for ASD (Hultman, Sparen, & Cnattingius, 2002; Schendel & Bhasin, 2008), and early signs of ASD (Limperopoulos et al., 2008). General neurological vulnerability may also underlie the association between infantile seizure disorders and ASD, with some evidence that more prolonged seizures during infancy are associated with greater deficits later in development (Saemundsen, Ludvigsson, Hilmarsdottir, & Rafnsson, 2007; Saemundsen, Ludvisson, & Rafnsson, 2007). There are also several genetic or congenital disorders in which rates of ASD are thought to be elevated, including tuberous sclerosis, Fragile X syndrome, Down syndrome, and neurofibromatosis (for review see Abrahams & Geschwind, 2008; Zafeiriou, Ververi, & Vargiami, 2007). Children with these known genetic conditions should be routinely screened for the presence of ASD symptoms such that treatment programs can be targeted appropriately.

Advances in early detection require advances in early treatment

As early symptoms of ASD may contribute to abnormal trajectories of brain development (Dawson et al., in press; Mundy & Crowson, 1997), it is critical that children showing these symptoms receive appropriate treatment. In 2001, the National Research Council recommended that preschool-aged children with ASD should receive at least 25 hours of structured intervention weekly. This should include a comprehensive curriculum, generalizability training, predictability and routine of intervention activities, prevention and reduction of problem behaviors, academic preparation, family involvement, and high intensity (for further details of effective intervention characteristics, see Dawson & Osterling, 1997; Dawson & Zanolli, 2003; Faja & Dawson, 2006; and for publicized

guidelines for parents and practitioners, see National Research Council, 2001; *Journal of Autism and Developmental Disorders* special issue, 2002). However, although many interventions have been specifically developed for young children with ASD (eg, Chandler, Christie, Newson, & Prevezer, 2002; Drew et al., 2002; Green, Brennan, & Fein, 2002; Mahoney & Perales, 2003; McGee, Morrier, & Daly, 1999; Rogers & Dawson, 2007; see Faja & Dawson, 2006, for review), the challenges of conducting systematic evaluations of their efficacy with a developing population has led to little knowledge of necessary components, general effectiveness, or the impact of individual differences on treatment outcome (Lord et al., 2005; Smith et al., 2007). Families with young children who receive an ASD diagnosis should be guided through treatment options by a specialist clinician. As early intervention for ASD appears more effective than later intervention (Fenske, Zalenski, Krantz, & McClanahan, 1985; Harris & Handleman, 2000; Rogers, 1996; for review, see Rogers, 1998; Rogers & Vismara, 2008), it is clearly critical to accompany research on early diagnosis with the development and evaluation of interventions suitable for infants and toddlers with identified or suspected ASD.

SUMMARY

The rapid rate of research into the early development of ASD has revealed that some children who go on to receive a diagnosis of ASD exhibit disruptions in the development of key social and communication skills in the first year of life. Early screening may identify children who are at elevated risk for developing ASD, allowing the possibility of targeted intervention or more careful monitoring. Increasing awareness of these early signs among parents and practitioners is critical in ensuring that symptoms of ASD are recognized and evaluated as soon as they appear, particularly for groups of children who may be at increased risk. Notably, a proportion of children develop signs of ASD after a period of regression or developmental “stagnation” in the second year, mandating continued screening and further evaluation of children exhibiting any loss of skills. Development and evaluation of treatment options for young children with ASD, and ensuring prompt access to currently available interventions for children with a new diagnosis, are critical goals as the field moves forward.

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Table 1

Screeners for detection of autistic spectrum disorders in children younger than 3 years[a]

Measure	Age	Population	Items	Sensitivity	Specificity	Authors
CHAT	~18 mo	1. *N* = 91 2. *N* = 16000 3. *N* = 16235	14 Items, parent report and professional observations	1. 100% (c) 2. 3. 20–38% (p) (6.2 per 100000)	1. 100% (c) 2. 83% 3. 97.6–99.8% (p)	1. Baron-Cohen et al. (1992) 2. Baron-Cohen et al. (1996) 3. Baird et al. (2000)
M-CHAT	18–24 mo	4. *N* = 1293 5. *N* = 1293	23 Items Parent report (yes/no)	4. 97% (c) 5. 100% (c)	4. 99% (c) 5. 98% (c)	4. Robins, Fein, Barton, and Green (2001) 5. Robins and Lee (2003)
Q-CHAT	18–24 mo		25 Items, parent report (5-point scale)	Not yet published		Allison et al. (2008)
DBC-ES	18–48 mo	*N* = 120	17 Parent report items	88% (c)	69% (c)	Gray and Tonge (2005)
FYI	12 mo		63-Item parent report, multiple choice	None published	None published	Reznick, Baranek, Reavis, Watson, and Crais (2007)
ITC/CSBS DP	9–24 mo	1. *N* = 3026 2. *N* = 5385	24 Items	1. 89% (c,p) 2. 93% (p)	1. 89%(c,p)	Wetherby and Prizant (2002) 1. Wetherby, Woods et al. (2004); 2. Wetherby et al. (2008)
ABC	18 mo to 35 y		57-Item interviewer checklist	38–58% (c)	76–97% (c)	Krug et al. (1980)
CARS	>2 y	*N* = 774	15 Items, trained observer	94% (c)	85% (c)	Schopler, Reichler, DeVellis, and Daly (1980), Perry et al. (2005)
STAT	1. 2–3 y 2. 2–3 y 3. 2–3 y 4. 12–23 mo	1. *N* = 33 2. *N* = 52 3. *N* = 50 4. *N* = 71	12 Play-based items	1. 83%(c) 2. 92% (c,p) 3. 100% (c) 4. 95%	1. 86% (c) 2. 85% (c,p) 3. 90% (c) 4. 73%	1. Stone, Coonrod, and Ousley (2000) 2,3. Stone, Coonrod, Turner, and Pozdol (2004) 4. Stone, McMahon, and Henderson (2008)
PDDST-II Levels 1,2, and 3	Under 6 y	*N* = 687	Parent questionnaire	1. 92%(c) 2. 73% (c) 3. 58% (c)	1. 91% (c) 2. 49% (c) 3. 60% (c)	Siegel (2004)
ESAT	14 mo	*N* = 31724	4 Parent questions plus follow-up	Detected 5.7 per 10000 (p)[b]	25%	Dietz et al. (2006)

Abbreviations: C = clinic sample, p = population sample.

[a]Other screeners for more general developmental concerns are reviewed by Filipek et al. (1999)

[b]estimated prevalence = 30 to 60 per 10 000.