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Atypical Attentional Networks and the Emergence of Autism

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Abstract

The sociocommunicative impairments that define autism spectrum disorder (ASD) are not present at birth but emerge gradually over the first two years of life. In typical development, basic attentional processes may provide a critical foundation for sociocommunicative abilities. Therefore early attentional dysfunction in ASD may result in atypical development of social communication. Prior research has demonstrated that persons with ASD exhibit early and lifelong impairments in attention. The primary aim of this paper is to provide a review of the extant research on attention in ASD using a framework of functionally independent attentional networks as conceptualized by Posner and colleagues: the alerting, orienting and executive control networks (Posner and Petersen, 1990; Petersen & Posner, 2012). The neural substrates and typical development of each attentional network is briefly discussed, a review of the ASD attention literature is presented, and a hypothesis is proposed that links aberrant attentional mechanisms, specifically impaired disengagement of attention, with the emergence of core ASD symptoms.

Keywords

autism; attention; development; alerting; arousal; orienting; disengagement; executive control

1. Introduction

From birth, our senses are inundated with information from a diverse and ever-changing environment. Attentional mechanisms are responsible for the selection of a small portion of information from this deluge. What captures our attention automatically and what we choose to attend to influences the way we experience and perceive the world around us and impacts the course of brain and behavioral development. Attention can be broadly defined as information processing mechanisms that mediate perceptual selectivity. This selection is controlled by endogenous, goal-directed processes dependent on the desires, expectations,

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and/or knowledge of the observer (i.e. top-down control) and exogenous, stimulus-driven (bottom-up) factors may be orthogonal to goal-directed behaviors (Yantis, 1993) and are dependent on the physical characteristics of the stimuli (Wolfe, 1994). Attentional selection rarely consists of exclusively top-down or bottom-up mechanisms; rather, successful and adaptive information processing requires the integration of these two processes (see Pashler, Johnston, & Ruthruff, 2001, for review). Information selection may occur early with irrelevant information filtered from further processing or late with irrelevant information processed to a greater degree (see Pashler, 1999, for review), and is dependent on the perceptual load of the stimuli to be processed (Lavie, 2005).

Posner and Petersen (1990; 2012) conceptualized attention as consisting of three functionally independent attentional networks, which are responsible for a distinct set of cognitive processes: the alerting, orienting, and executive control networks. These three networks have been shown to have some degree of behavioral, neurophysiological, and neuroanatomical independence (Fan et al., 2007; Fan, McCandliss, Sommer, Raz, & Posner, 2002; Fan, McCandliss, Fossella, Flombaum, & Posner, 2005; see Raz & Buhle, 2006, for a recent review). More recently, Posner and Fan (2004) hypothesized that this model may assist in elucidating differences in attentional modulation between typically developing (TD) individuals and individuals with atypical attention. The current review will use this framework of attention to elucidate network-specific attentional abnormalities in autism spectrum disorder (ASD).

Autism spectrum disorder is a behaviorally-defined developmental disorder diagnosed on the basis of impairments and anomalies in the domains of social communication and repetitive and stereotyped behaviors (APA, 2000). While a diagnosis of ASD is based on these features, *attentional abnormalities* have been associated with the disorder since its first description (Kanner, 1943). Individuals with ASD exhibit early (Baranek, 1999; Elsabbagh et al., 2009; Osterling & Dawson, 1994; Osterling, Dawson, & Munson, 2002; Swettenham et al., 1998; Zwaigenbaum et al., 2005) and pervasive (see Allen & Courchesne, 2001; Burack, Enns, Stauder, Mottron, & Randolph, 1997, for earlier reviews) abnormalities of attention. Importantly, atypical attentional function has been shown in infants at-risk for ASD (at-risk due to having an older sibling diagnosed with ASD) (Elsabbagh et al., 2009; Zwaigenbaum et al., 2005), and may be one of the earliest characteristics that distinguish infants who later receive an ASD diagnosis (Zwaigenbaum et al., 2005). Further, non-social attentional strengths (e.g., visual search) and weaknesses (e.g., novelty detection) in ASD and their neurofunctional correlates have also been associated with increased autism symptomatology (Belmonte, Gomot, & Baron-Cohen, 2010; Gomot, Belmonte, Bullmore, Bernard, & Baron-Cohen, 2008; Joseph, Keehn, Connolly, Wolfe, & Horowitz, 2009; Keehn & Joseph, 2008; Keehn, Lincoln, Muller, & Townsend, 2010). In addition, findings from single gene disorders that have increased rates of ASD (e.g., fragile X syndrome) suggest an association between ASD symptomatology and attentional dysfunction (Cornish et al., 2012; Roberts et al., 2012; Scerif et al., 2011). Together, these findings suggest that attentional processes may impact the development of higher-level sociocommunicative functions. Consequently, understanding the development of attentional mechanisms in children with ASD may help elucidate the abnormal or delayed trajectories of attentional development in ASD, and furthermore, how these attentional abnormalities may contribute to the manifestation of the core ASD impairments.

The primary aim of this paper is to provide a review of attention research in ASD within the framework of neurotypical attentional networks. The first goal is to review the network-specific findings for investigations of attention in ASD after supplying a brief background of the brain regions and neurotypical development associated with each attention network. The second goal of this paper is to put forth a developmental framework regarding how aberrant

attentional mechanisms may function as a primary dysfunction with regard to the development of core ASD symptomatology. *Specifically, we posit that early deficits in disengaging attention result in cascade of impairments and ultimately contribute to the emergence of the ASD phenotype.* Theories of autism have postulated primary impairments in both social and non-social functions (see Happé, 2001, for more in-depth discussion). Although the focus of this section is the role of attention in the development of ASD, this is not meant to imply that attention networks are the only dysfunctional neural networks in ASD. As was aptly put by Goodman, “the very diversity of existing ‘unitary’ psychological and neurological explanations casts doubt on the hypothesis that infantile autism can potentially be explained by a fault in just one psychological or neurological system” (Goodman, 1989, p. 410). Rather, the goals of understanding whether dysfunctional attentional processes are of etiological significance in ASD is two-fold. If early attentional impairments play a causal role in the development of ASD, then 1) attentional deficits may be used as an early neuro-behavioral marker that can be used to identify infants at-risk for ASD and 2) the development of attention-targeted early interventions that may remediate abnormal developmental trajectories and improve outcomes in children with ASD.

2. Attention Networks

2.1 Alerting Network

The alerting network is responsible for achieving and maintaining a state of sensitivity to incoming information. Alertness has been divided into tonic and phasic components (see Sturm & Willmes, 2001, for review). Tonic alertness is a state of general wakefulness or arousal; endogenously-controlled tonic alertness, referred to as vigilance or sustained attention, is the voluntary maintenance of alertness at a certain level. Phasic alertness is a more transient alert state, commonly modified by a behavioral or experimental cue. These components of alertness parallel the tonic and phasic attributes of Sokolov’s orienting response (OR) theory (Sokolov, 1963; note that orienting here does not refer to spatial orienting, as discussed in section 2.2, but rather to a reflexive physiological reaction to a change in the environment). The alerting network is an interacting system of internal state-dependent attentional mechanisms that can increase or decrease information processing capacity (Kahneman, 1973) and influence the breadth of selective attention (Easterbrook, 1959). The efficiency and degree of phasic alerting is modulated by the level of tonic alertness, while goal-directed changes in levels of tonic alertness affect task performance. Thus, multiple factors are involved in typical and pathological alerting responses.

An additional process related to alerting is novelty detection. Novelty detection is a fundamental characteristic of Sokolov’s OR, and is dependent on a mismatch between an individual’s pre-existing representation and a novel stimulus; a phasic OR does not take place without the perception of a novel stimulus. Novelty detection is therefore an important prerequisite for an OR, which subsequently enables an individual to encode and process novel information.

2.1.1 Neuroanatomy of the alerting network—The neuroanatomy of alerting includes a network of subcortical and cortical regions (see Figure 1). The nuclei of the reticular formation in the brain stem, specifically the locus coeruleus-norepinephrine (LC-NE) system, is the core arousal center (see Robbins & Everitt, 1995, for review). The LC projects to the thalamic nuclei and terminates in cerebral cortex (Foote, Bloom, & Aston-Jones, 1983). In general, the LC-NE system supports appropriate levels of alertness in order to maintain efficient information processing. Norepinephrine, the neuromodulator associated with the alerting network, appears to inhibit spontaneous neural activity, permitting increased neural response to sensory stimulation (Foote, Freedman, & Oliver, 1975). The anterior cingulate cortex (ACC) and the right dorsolateral prefrontal cortex (DLPFC)

function to maintain endogenous alertness by modulating activity in the LC via the reticular nucleus of the thalamus (Sturm et al., 1999). Lastly, the alerting network is mediated by a right-lateralized ventral frontoparietal network, which is responsible for achieving and maintaining appropriate levels of alertness (Corbetta, Patel, & Shulman, 2008; Posner & Petersen, 1990).

2.1.2 Development of the alerting network—The development of tonic alertness and the waking state – e.g., increased periods with open eyes, toy manipulation, and babbling – in infants changes rapidly between 2 and 24 weeks (Dittrichova & Lapackova, 1964). Early infant attention functions to achieve an optimal state of arousal and is dependent on the amount of external stimulation and on the level of internal arousal (Karmel, Gardner, & Magnano, 1991). The autonomic nervous system and its physiological indices undergo considerable development across the lifespan (see Shields, 1983, for review). Stimulus orienting (i.e., a phasic OR), as measured by a deceleration of heart rate (HR), increases rapidly during the first year of life with no significant developmental changes occurring in infants between 14 and 26 weeks of age (see Reynolds & Richards, 2008, for review). However, sustained attention (i.e., the voluntary maintenance of alertness) undergoes a more protracted developmental time course, with the ability to sustain attention increasing rapidly from 2 to 6 months of age (Richards, 1995).

The development of phasic alertness has also been investigated using event-related potentials (ERP). Two examples include the Nc (negative central; Courchesne, 1977, 1978) and the P3 (see Polich, 2007, for review) components. The Nc component occurs between 350–800ms post-stimulus onset at central electrode sites. This component has been hypothesized to represent an OR that is insensitive to novelty and stimulus probability (Nelson & Collins, 1992; Richards, 2003), although stimulus novelty (Reynolds & Richards, 2005) and/or meaningfulness (de Haan & Nelson, 1999) may play a role in larger Nc responses. The Nc component appears in the first year of life during attentive states, increasing in amplitude while decreasing in latency with development (Richards, 2003). The amplitude of the Nc component decreases significantly from early childhood to early and late adolescence as the electrophysiological response transitions to the more mature P3 waveform (Courchesne, 1978).

The modality-independent P3 response is generally elicited using an odd-ball paradigm (analogous to a continuous performance task; see Figure 2C), which requires a manual response to a prespecified target displayed infrequently amongst standard stimuli (some odd-ball paradigms also include a third novel non-target stimuli). Briefly, the P3 component consists of two subcomponents: 1) a frontocentral P3a (or “novelty P3”; Friedman, Cycowicz, & Gaeta, 2001) component that habituates rapidly and is associated with the redirection of attention monitoring during stimulus discrimination, and 2) a parietal P3b component that reflects a memory comparison and facilitates context maintenance. Previous research has demonstrated that these components reflect activity of separate neural generators and neurotransmitter systems (Polich, 2007) and involve regions such as the temporal-parietal junction, lateral prefrontal cortex, and the locus coeruleus (see Nieuwenhuis, Aston-Jones, & Cohen, 2005, for discussion). Developmental changes of the P3 component from early child- to adulthood include decreased latency (Courchesne, 1978; Cycowicz, Friedman, & Rothstein, 1996; Zenker & Barajas, 1999) and changes in scalp distribution (Courchesne, 1978; Cycowicz et al., 1996). Behaviorally, phasic alerting mechanisms continue to develop until age 8 years, at which point they may have matured to adult levels (Morrison, 1982). These findings are in accord with conclusions from a cross-sectional study of P3a development (Cycowicz et al., 1996), which suggests that despite changes in latency and scalp topography, by age 7–8 years phasic orienting may be functioning at adult levels.

The development of sustained attention across childhood to adolescence has often been measured using Continuous Performance Tests (CPT; see Figure 2C), in which participants are asked to respond to a pre-defined target stimulus presented randomly within a stream of non-target stimuli. Sustained attention abilities, as measured by the CPT, increase significantly from 3 to 6 years of age (Akshoomoff, 2002; Kerns & Rondeau, 1998; Levy, 1980) and continue to develop into late childhood and adolescence, reaching adult-like levels around the age of 12 years (Lin, Hsiao, & Chen, 1999).

The Attention Network Test (ANT), developed by Fan and colleagues (2002), examines the efficiency of each attentional network (see Figure 2A). The ANT consists of both a cued reaction time task (Posner, 1980) and a flanker paradigm (Eriksen & Eriksen, 1974). Alerting, orienting, and executive control scores are calculated by series of cognitive subtractions. The alerting score is calculated by subtracting response times in conditions that maximize and minimize alerting (i.e., double cue vs. no cue). The alerting score which reflects both phasic and tonic components of alertness (Posner, 2008), increases between the ages of four and seven years (Mezzacappa, 2004), but remains similar between the ages of six to ten years (Rueda et al., 2004). However, the process continues to mature as 10-year-old children show significantly higher alerting scores than adults (Rueda et al., 2004). Developmental decreases in alerting scores are likely due to changes in the level of tonic alertness; younger children have difficulty maintaining appropriate levels of tonic alertness. Additionally, the network mediating both phasic and tonic levels of alertness likely continue to develop into adulthood (Konrad et al., 2005).

2.1.3 Alerting network in ASD—Levels of tonic arousal and the modulation of phasic alertness have been areas of intense speculation but modest empirical consensus in ASD-related research (see Bryson, Wainwright-Sharp, & Smith, 1990; Rogers & Ozonoff, 2005, for reviews). Previous authors have argued for hyperarousal (Hutt, Hutt, Lee, & Ounsted, 1964), hypoarousal (Rimland, 1964), and dysfunctional arousal modulation (Ornitz & Ritvo, 1976). Others have proposed that “nonresponsiveness” (i.e. hypoarousal) may develop as a result of sensory overload due to early chronic hyperarousal (van Engeland, 1984). Liss and colleagues (2006) have hypothesized that hyperarousal in ASD may lead to the development of overselective attention, and, similar to van Engeland (1984), that this may result in reduced or absent OR to stimuli may be outside an atypically narrower focus of attention. Alternatively, the varying hypotheses and inconsistent findings of hypo- and hyper-arousal may reflect heterogeneity within the ASD population, with subtypes exhibiting different arousal states (Hirstein, Iversen, & Ramachandran, 2001; Schoen, Miller, Brett-Green, & Hepburn, 2008).

Evidence for hyperarousal in ASD comes from investigations employing multiple modalities. Studies examining skin conductance have demonstrated increased skin conductance levels (SCL) and frequency of skin conductance responses (SCR) (Palkovitz & Wiesenfeld, 1980). Heart rate measures have shown decreased HR deceleration (or a relative acceleration) (Palkovitz & Wiesenfeld, 1980) and increased baseline HR (Ming, Julu, Brimacombe, Connor, & Daniels, 2005) in ASD as compared to TD individuals. More recently, evidence of larger tonic pupil size in children with ASD has also been found (Anderson & Colombo, 2009). Furthermore, children with ASD show atypical HR response between rest and task states (Althaus, Mulder, Mulder, Aarnoudse, & Minderaa, 1999; Toichi & Kamio, 2003), indicative of dysfunctional modulation of arousal.

Contrary to evidence of hyperarousal in ASD, equivalent or reduced levels of arousal have also been shown. Prior studies have reported no difference between low-functioning children and adolescents with ASD and chronological age (CA) and mental age (MA) matched TD individuals in electrodermal response to an auditory habituation paradigm

(Stevens & Gruzelier, 1984). Normal SCL, spontaneous SCR, and HR have also been shown in high-functioning adults with ASD during rest (Zahn, Rumsey, & Van Kammen, 1987); however, Zahn and colleagues (1987) also report *reduced* reactivity for task versus baseline and decreased phasic SCR activity to imperative stimulus in a simple response time (RT) task, which may also be indicative of abnormal task-related modulation of arousal.

A previous study has also demonstrated no difference in spontaneous fluctuations in skin conductance to auditory stimuli between ASD and TD and developmentally delayed (DD) children (van Engeland, 1984). Interestingly, van Engeland (1984) interpreted the findings in a high-functioning subgroup to represent a “paradoxical reaction,” in which participants were atypically open to environmental stimuli, and, thus, potentially overwhelmed with sensory information. The results of the atypically increased openness to sensory information may lead to previously discussed hyperarousal states, and, subsequently, to the development of sensory non-responsiveness to novel stimuli. In support of this hypothesis, van Engeland and colleagues (1991) demonstrated significantly reduced SCR and fixation times to novel visual stimuli in high-functioning children with ASD.

Although behavioral evidence of equivalent phasic alerting in TD and ASD children exists (Raymaekers, van der Meere, & Roeyers, 2006), electrophysiological measures have demonstrated atypical phasic alerting in ASD. The N1c component, which reflects automatic attentional capture resulting from an auditory stimulus, is reduced in amplitude in ASD as compared to TD children (Bruneau, Bonnet-Brilhault, Gomot, Adrien, & Barthelemy, 2003; Orekhova et al., 2009). Reduced (Courchesne et al., 1985) or absent (Ciesielski, Courchesne, & Elmasian, 1990) Nc response has also been shown in adolescents and adults with ASD. More recently, McCleery and colleagues (2009) reported reduced Nc amplitudes in 10-month-old infants at-risk for ASD compared to TD infants during passive viewing of faces and objects. These findings suggest the modulation of phasic alerting mechanisms may be dysfunctional in ASD.

In accord with these findings on phasic alertness, individuals with ASD also display robust insensitivity to novel information. Behaviorally, this has been shown in two separate visual search studies (Greenaway & Plaisted, 2005; Keehn & Joseph, 2008). Multiple studies have shown reduced P3 amplitude in response to novel auditory (Ciesielski et al., 1990; Courchesne, Kilman, Galambos, & Lincoln, 1984; Courchesne, Lincoln, Kilman, & Galambos, 1985; Courchesne, Lincoln, Yeung-Courchesne, Elmasian, & Grillon, 1989; Dawson, Finley, Phillips, Galpert, & Lewy, 1988; Lincoln, Courchesne, Harms, & Allen, 1993; Novick, Kurtzberg, & Vaughn, 1979; Novick, Vaughan, Kurtzberg, & Simson, 1980) and visual targets (Ciesielski et al., 1990; Courchesne et al., 1989; Pritchard, Raz, & August, 1987; Townsend et al., 2001) in ASD. More specifically, Courchesne et al. (1984; 1985) reported decreased P3a amplitudes to novel auditory stimuli, as well as reduced P3b to target auditory compared to standard stimuli in ASD as compared to TD adolescents. Townsend and colleagues (2001) reported abnormalities in both P3a and P3b in adults with ASD to peripheral visual targets.

In fMRI studies, Gomot and colleagues (2008; 2006) examined passive and active detection of auditory oddball stimuli. Passive detection of novel auditory stimuli resulted in greater activation in bilateral temporal parietal junction (TPJ) and right inferior and middle frontal regions in TD compared to ASD participants. In contrast, *active* novelty detection task (requiring behavioral response) resulted in significantly greater activation in the right prefrontal cortex and left inferior parietal lobule in the ASD compared to the TD group. Thus, atypical response to novel stimuli may be dependent on the *context* of the experiment. Reduced ASD activation for passive novelty detection may result from a predisposition to ignore novel information. However, the authors suggest that increased ASD activation to

active novelty detection may reflect over-focusing in the ASD group, which could result in maladaptive allocation of attention. This is in accord with electrophysiological findings demonstrating that the allocation of attention (in an active task) results in enhanced responses (Kemner et al., 1994) or – in the case where passive tasks have shown reduced amplitudes – equivalent responses (Dunn et al., 2008; Whitehouse and Bishop, 2008) in individuals with ASD.

Keehn and colleagues (2010) recently employed the ANT to investigate attentional networks in ASD. The authors demonstrated similar alerting scores in children and adolescents with ASD and TD children. Interestingly, whereas there were no correlations between network scores for TD children (as reported previously; see Rueda et al., 2004), children with ASD exhibited significant correlations between alerting and executive control networks suggesting that these two networks may not function as independently in children with ASD. Similarly, Raymaecker and colleagues (2004) demonstrated that increased arousal was related to poorer response inhibition performance in ASD but not TD individuals. These findings suggest that alerting and executive networks may interact to a greater degree in individuals with ASD, and as a result difficulty controlling the level of arousal may produce impaired executive control abilities (discussed in greater detail in Section 3.1).

In contrast to studies of phasic alertness and novelty processing, previous studies have demonstrated equivalent sustained attention abilities in ASD and TD individuals (Garretson, Fein, & Waterhouse, 1990; K. A. Johnson et al., 2007; Noterdaeme, Amorosa, Mildenerger, Sitter, & Minow, 2001; Pascualvaca, Fantie, Papageorgiou, & Mirsky, 1998). These findings suggest that endogenous maintenance of tonic alertness may be intact in individuals with ASD.

2.1.4 Summary—The arousal-state and phasic alerting mechanisms develop rapidly during the first year of life. The efficiency and speed of phasic alerting may continue to develop into the early school-age years (Morrison, 1982), while endogenous maintenance of alertness has a more protracted course of development not reaching adult-like levels until early adolescence (Lin et al., 1999).

Inconsistent results have been observed in studies investigating tonic levels and phasic responsiveness of the alerting network in ASD, and limit unequivocal conclusions regarding dysfunction within this attentional network. The alerting network reflects a complicated interaction between the internal state of the individual, their responsiveness to external stimuli, and their task- or goal-related endogenous modulation of alertness. Therefore, elucidating underlying alerting abnormalities in ASD has proven difficult. Further complicating the study of the alerting network is the fact there may be separate subgroups of hyper- and hypo-aroused individuals with ASD.

There is evidence for increased plasma-levels of norepinephrine (Lam, Aman, & Arnold, 2006), increased pupil diameter, aberrant skin conductance responses and levels, atypical heart rate, abnormal electrophysiological alerting response (Nc, N1c) and novelty detection (P3), and atypical neurofunctional activation of the alerting network in persons with ASD. Although some studies report unimpaired phasic and endogenous tonic alertness in ASD, the evidence reviewed here suggests that under some conditions or in some individuals, there are impairments of tonic (arousal) and phasic components of the alerting network in ASD.

Importantly, divergent results on passive and active tasks may reflect atypical control of alertness. Specifically, increased activation in ASD (relative to TD peers) in right frontoparietal regions has been interpreted as over-focusing (Gomot et al., 2008), while atypically decreased activation to novel information in ASD during passive tasks may be

indicative of a default processing state that rejects or ignores novelty (Gomot et al., 2006). Likewise, evidence from studies of HR variability suggests that between-group differences arise when examining *changes* in autonomic state from rest to task conditions (Althaus et al., 1999; Toichi & Kamio, 2003). Thus, alerting abnormalities in ASD cannot be characterized simply by atypical tonic levels of arousal, but may also include the impairments in regulating levels of alertness across different situations and differences in active and passive tasks.

2.2 Orienting Network

The orienting network is responsible for the selection of information from sensory input. Posner and colleagues (1984) have defined visuospatial orienting as disengaging, shifting, and reengaging attention. In contrast to the phasic alerting mechanisms that respond homogeneously across the visual field, orienting visual attention facilitates processing over a localized area (e.g., Mangun & Hillyard, 1991). Although anatomically distinct and functionally independent, alerting and orienting mechanisms do interact (Callejas, Lupianez, Funes, & Tudela, 2005; Callejas, Lupianez, & Tudela, 2004; Fan et al., 2009; Fuentes & Campoy, 2008). For example, non-spatial cues (that result in increased phasic alertness) can facilitate attention orienting (Callejas et al., 2004; 2005), while disengaging and re-orienting attention may serve to attenuate arousal levels (Derryberry & Rothbart, 1988). Thus, alerting (both phasic and tonic components) may be bi-directionally related to orienting abilities.

Orienting visual attention can occur overtly, with concurrent head/eye-movements, or covertly, without corresponding head/eye-movements. Attention may also be directed reflexively (automatically) or voluntarily to a spatial location based on central (endogenous) or peripheral (exogenous) cues. In commonly used spatial attention testing paradigms, reflexive orienting occurs to both peripheral and central (arrows, gaze) non-predictive (i.e., 50% trials validly cued) cues. Predictive (e.g. 80% of trials validly cued) peripheral and central (arrows, gaze) cues engage voluntary as well as reflexive orienting mechanisms, which interact in a super-additive manner (Olk, Cameron, & Kingstone, 2008).

2.2.1 Neuroanatomy of the orienting network—The network of areas responsible for directing attention include superior parietal lobe, intraparietal sulcus, temporal-parietal junction and dorsofrontal (frontal eye fields; FEF) cortices, thalamus, and superior colliculus (Corbetta & Shulman, 2002; Mesulam, 1990; Posner & Petersen, 1990). Furthermore, the cerebellum may also play a role in both covert and overt orienting of attention (Akshoomoff, Courchesne, & Townsend, 1997; Pelisson, Goffart, Guillaume, & Quinet, 2003). Evidence from individuals with cortical and subcortical lesions suggests that reflexive orienting is likely mediated by subcortical and more posterior cortical regions, while a network of frontal-parietal regions underlie voluntary orienting (Rafal, 1998). Acetylcholine (ACh) is the neuromodulator associated with the orienting network, with increased levels of ACh resulting in more rapid attention reorienting (Thiel, Zilles, & Fink, 2005; Witte, Davidson, & Marrocco, 1997). Specifically, ACh may act to expand the attentional spotlight so that attentional misdirection does not elicit a reorienting response (Thiel et al., 2005).

2.2.2 Development of the orienting network—Johnson (1990) proposed a model for the development of overt attention in infants based on the maturation of subcortico-cortical and cortico-cortical pathways as outlined by Schiller (1985, 1998). Briefly, this model proposes that in newborns a subcortical pathway mediates early reflexive shifts of attention; the development of an inhibitory pathway, which is responsible for suppressing visuospatial orienting, leads to “obligatory looking” (difficulty disengaging visual attention) around 1 month. Maturation of cortical layer 4 at two months and subsequently layers 2 and 3 of

primary visual cortex at three months allows for development of pathways responsible for smooth pursuit and anticipatory eye-movements, respectively.

Overt orienting to exogenous cues is limited in 2-month-old infants; however, clear orienting including facilitation and inhibition by peripheral cues appears by 4 months of age, with speed of spatial attention shifting increasing from 4 to 7 months (M. H. Johnson & Tucker, 1996). Similarly, the development of covert attention appears to be present by 4 months of age (M. H. Johnson, Posner, & Rothbart, 1994). By the age of 3 months, infants are also sensitive to direction of gaze and direct their attention towards gaze-cued locations (Hood, Willen, & Driver, 1998). However, if the gaze-cue remained onscreen (as opposed to being removed prior to the appearance of the target) 3-month-olds did not orient attention to the gaze-cued location as frequently, perhaps due to difficulties disengaging visual attention (Hood et al., 1998).

Following the period of reduced disengagement efficiency at around 1 month (i.e., obligatory looking), an infant's ability to disengage attention increases rapidly. Results from investigations employing the gap-overlap eye-movement paradigm have demonstrated that 4-month-old infants exhibit significant improvements in disengaging their attention during an overlap (competing information) condition relative to 3-month-old infants (Frick, Colombo, & Saxon, 1999). In 2-, 4-, and 6-month-old infants, saccadic RT for overlap trials decreased significantly at each age group ($6 < 4 < 2$ months) (McConnell & Bryson, 2005).

The development of orienting abilities during early childhood has been examined using the ANT. Orienting efficiency increases between the ages of 4 to 7 years (Mezzacappa, 2004); however, no change in orienting score was exhibited from six years of age to adulthood (Rueda et al., 2004). This finding may be task-dependent as the ANT did not include invalid trials, thus reducing the amount of attentional disengagement necessary to complete the task. An fMRI study using a modified ANT (which included invalid trials), demonstrated that children exhibited reduced activity in the right TPJ during attentional reorienting and exhibited increased activation relative to adults in superior frontal gyrus and insula (Konrad et al., 2005).

During the school-age period and adolescence, the ability to disengage (Wainwright & Bryson, 2002) and shift (Schul, Townsend, & Stiles, 2003) attention to predictive exogenous cues continue to develop, perhaps reaching adult-like levels at approximately 10 years (Wainwright & Bryson, 2002; although see Schul et al., 2003, for evidence of age-related changes after 10 years) as the speed of attentional movements continues to increase during this period (Pearson & Lane, 1990). This is consistent with research using endogenous cuing paradigms, which suggests that the development of orienting may reach adult-like levels by 8 to 9 years of age (Goldberg, Maurer, & Lewis, 2001).

2.2.3 The orienting network in ASD—Deficits in orienting visual attention have been consistently observed in individuals with ASD. Evidence in support of early orienting deficits comes from observational and retrospective video analysis. Swettenham and colleagues (1998) measured spontaneous shifts of attention while 20-month-old ASD, TD, and DD infants participated in a five-minute free play session. In general, infants with ASD showed less attention shifting than did the two comparison groups. Studies using retrospective home video analysis have also reported that ASD infants orient to visual stimuli less (Baranek, 1999), do not orient towards people or human voices as frequently as TD infants (Maestro et al., 2002), and orient to name significantly less as compared to TD and DD infants (Osterling & Dawson, 1994; Osterling et al., 2002).

Dawson and colleagues (1998b) examined orienting to a variety of social and non-social stimuli in 5-year-old children with ASD, children with Down syndrome, and MA-matched TD children. They found that children with ASD failed to orient to both social and non-social sounds as frequently as the comparison groups. Furthermore, those children with ASD who were able to orient attention demonstrated delayed orienting to social stimuli. In a follow-up study, Dawson and colleagues (2004) compared a larger sample of 4-year-old children with ASD to DD and MA-matched TD toddlers. Replicating previous results, children with ASD failed to orient as frequently to social, and to a lesser extent non-social, sounds.

In older children, adolescents, and adults with ASD orienting abilities have been measured using various spatial cuing paradigms (see Figure 2B). Following an attention-directing cue, the response to information at a cued location assesses visuospatial orienting and comparing responses to information at a validly cued versus an invalidly cued location provides a measure of time for attention disengagement and re-orienting. This is typically referred to as a “validity effect” (RT for invalid minus valid cue condition) and represents the behavioral cost of misdirecting attention with an invalid cue. Townsend and colleagues (1996) found slower orienting in adults with ASD compared to TD individuals. A comparison of TD, ASD, and cerebellar lesion participants (Townsend et al., 1999) replicated these findings and suggested that slower orienting was related to decreased size of cerebellar vermis VI-VII. A similar study that included children with ASD reported no difference in RT validity effect; however, similar to adults, children with ASD showed a significant correlation between the orienting speed and vermis size (Harris, Courchesne, Townsend, Carper, & Lord, 1999). A more recent study using the ANT showed that children and adolescents with ASD were slower to shift attention (Keehn et al., 2010).

Ristic et al. (2005) examined predictive and non-predictive orienting to endogenous gaze-cues adolescents with ASD. Individuals with ASD showed similar validity effects for predictive, but not for non-predictive, cues compared to TD individuals. These findings suggest that individuals with ASD do not reflexively orient attention to shifts in gaze, but can use volitional control of attention (but see Wainwright-Sharp & Bryson, 1993, for example of impaired volitional orienting). In agreement, Goldberg et al. (2008) reported that a validity effect to non-predictive endogenous gaze-cues seen in TD children was absent in children with ASD. However, equivalent orienting in ASD and TD children to non-predictive gaze cues has also been reported (Kylliainen & Hietanen, 2004; Senju, Tojo, Dairoku, & Hasegawa, 2004; Swettenham, Condie, Campbell, Milne, & Coleman, 2003), although some evidence suggests that in ASD gaze cues may be processed differently (Vlamings, Stauder, van Son, & Mottron, 2005). In accord with this view, an fMRI study of social and non-social orienting showed that while TD individuals exhibited greater activation to social versus non-social cues, individuals with ASD did not (Greene et al., 2011).

Two studies have investigated exogenous and endogenous cues in the same cohort of individuals. Renner and colleagues (2006) showed intact endogenous, but impaired exogenous orienting in children and adolescents with ASD. These results are in agreement with previous neuroimaging findings that suggest abnormal function of the network underlying reflexive orienting and relatively spared, though still atypical, network underlying more voluntary orienting in ASD (Haist, Adamo, Westerfield, Courchesne, & Townsend, 2005). However, a more recent study by Pruett et al. (2010) demonstrated similar performance on predictive and non-predictive, exogenous and endogenous paradigms in ASD and TD children.

2.2.3.1 Disengagement of attention: Attentional disengagement can also be studied by examining eye-movements. Disengagement efficiency is often determined by examining saccadic RT to targets appearing when the fixation cross remains on the screen (overlap condition) compared to when the cross disappears prior to the target onset (gap condition), i.e., the gap effect (see Figure 2D). The gap effect results from two separate mechanisms: 1) a generalized warning effect (i.e. phasic alerting) as a consequence of fixation offset, and 2) the release of ocular inhibition due to a) the disappearance of a foveal stimulus, and b) the top-down preparation of a saccadic response (Kingstone & Klein, 1993; Taylor, Kingstone, & Klein, 1998).

Landry and Bryson (2004) examined gap-overlap performance in children with ASD, CA-matched children with Downs syndrome, and MA-matched TD children, and demonstrated that the ASD group showed significantly increased latencies to disengage visual attention (on overlap trials), but similar response latencies to shift visual attention on gap trials compared to both comparison groups. Additionally, the authors report that the frequency of fast attentional shifts (i.e., the number of shifts with latency between 100 and 300ms) for the gap condition was significantly reduced in the ASD group, suggesting that in addition to difficulty disengaging attention on overlap trials, children with ASD did not efficiently shift attention to the target even when disengagement mechanisms were not competing with the central stimulus.

Impaired disengagement has also been demonstrated in low-functioning adults with ASD (Kawakubo et al., 2007) and at-risk infants (Elsabbagh et al., 2009; Zwaigenbaum et al., 2005). For at-risk infants, there was no difference between high-risk (i.e., infant with an ASD sibling) and low-risk (i.e., infant with a TD sibling) infants at six months in their ability to shift or disengage visual attention. Importantly, when re-tested at 12 months the high-risk group showed poorer performance in disengaging visual attention compared to performance at 6 months. Specifically, 25% of high-risk infants demonstrated *longer* latencies to disengage attention. Interestingly, every single child that exhibited increased difficulties disengaging attention between 6 and 12 months received an ASD diagnosis at 24 months (Zwaigenbaum et al., 2005).

While evidence for impaired disengagement of attention in ASD exists across the lifespan, it should be noted that some studies report typical attention disengagement in ASD (Kawakubo, Maekawa, Itoh, Hashimoto, & Iwanami, 2004; Leekam, Lopez, & Moore, 2000; Mosconi et al., 2009). However, conflicting results may in some cases be due to low statistical power and cohort effects in very small sample studies (ASD $n = 7$ in Kawabuko et al., 2004), or large differences in gap-overlap paradigms (e.g., Leekam et al., 2000). Additionally, although Goldberg and colleagues (2002) found no differences in disengagement or facilitation of visual attention in adolescents with ASD, individuals with ASD made fewer express saccades (short latency saccades occurring between 80 – 140 ms) to the target.

2.2.4 Summary—Subcomponents of the orienting network are established by the middle of the first year of life, but continue to develop at least into the school-age years. Orienting visual attention relies on a distributed network of brain areas, including a dorsal frontal-parietal cortical network as well as subcortical structures (thalamus, superior colliculus, and cerebellum) for shifting and disengaging attention.

In ASD, orienting deficits appear to be present within the first year of life. Retrospective analysis of home videos and prospective analysis of at-risk infant siblings have shown impairments in disengaging and shifting attention to both social *and* non-social auditory and visual stimuli before the first birthday in infants later diagnosed with ASD. Furthermore,

investigations of children, adolescents, and adults with ASD have revealed slower, less efficient visual orienting abilities and fewer express saccades; reflexive orienting may be more impaired than volitional shifts of attention, although there is some conflicting evidence.

2.3 Executive Control Network

The executive control network is a multidimensional attentional system, responsible for inhibition, planning, error monitoring, set shifting, working memory, and cognitive flexibility. Recent studies have shown that executive control is not mediated by a unitary mechanism, but can be dissociated into *at least* three separate but associated functions – set shifting, working memory, and inhibition, – in TD children and adults (Huizinga, Dolan, & van der Molen, 2006; Miyake et al., 2000). Although typically referred to in the singular, the executive control “network” is undoubtedly comprised of multiple overlapping systems. Set shifting refers not to visuospatial orienting, but rather to shifting between multiple mental sets (also referred to as “task switching”; Monsell, 2003). Working memory corresponds to active monitoring, updating, and maintenance of task-relevant information. Finally, inhibition refers to an individual’s ability to prevent pre-potent or automatic responses. These three domains of executive function will be the focus of the following section; however, it should be noted that separate taxonomies of executive function exist (e.g., Anderson, 2002). Omnibus tests including the Wisconsin Card Sorting Task (WCST) and the Tower of Hanoi or the similar Tower of London have been employed to test executive control in ASD. However, because these tasks tap multiple executive functions, their explanatory significance with regard to unique executive functions is limited. For the purpose of this section, only tasks that attempt to isolate set shifting, working memory, or inhibitory executive control functions will be considered.

Two important considerations should be noted when comparing different populations on executive tasks (Roberts & Pennington, 1996). First, although tasks may be designed to isolate and test a discrete executive function, subtle lapses in a separate function may ultimately result in poorer performance. For example, inhibitory deficits in clinical populations may result from dysfunctional working memory processes (i.e., forgetting task instructions) and not abnormal inhibitory control. Second, the degree of prepotencies may differ between groups resulting in the erroneous appearance of superior/inferior inhibitory abilities (e.g., Adams and Jarrold, 2009).

2.3.1 Neuroanatomy of the executive control network—The neural substrates of executive control include regions within the prefrontal cortex – orbitofrontal, ventrolateral prefrontal, and dorsolateral prefrontal cortex (DLPFC) – as well as medial frontal regions (anterior cingulate cortex; ACC) and subcortical regions such as the basal ganglia and cerebellum (Heyder, Suchan, & Daum, 2004). Additionally, more posterior areas (mainly located in the parietal lobe) may also be important for executive processes (Collette, Hogge, Salmon, & Van der Linden, 2006; Wager, Jonides, & Reading, 2004; Wager & Smith, 2003).

More specifically, the right inferior frontal cortex may mediate inhibitory processes (Aron, Robbins, & Poldrack, 2004), while bilateral dorsal frontal regions (superior frontal sulci) are important for updating working memory and right ventral frontal regions are important for manipulating information in working memory (Wager & Smith, 2003). Finally, shifting may be mediated by bilateral medial frontal cortex (ACC), intraparietal sulci, and to a lesser degree anterior insula and DLPFC (Wager et al., 2004). Dopamine is the primary neuromodulator associated with prefrontal cortex and working memory function; however,

norepinephrine also plays an important role in working memory, aside from its potential role in inhibition and set shifting (Robbins, 2000; Robbins & Arnsten, 2009).

2.3.2 Development of the executive control network—Relative to the alerting and orienting networks, the executive control network undergoes the most protracted development (see Diamond, 2002, for review), with differing developmental time courses for each executive component.

2.3.2.1 Set Shifting: The currently limited literature suggests that this executive component continues to develop between 8 and 13 years of age (Lehto, Juujarvi, Kooistra, & Pulkkinen, 2003), and does not reach adult levels until approximately 15 years of age (Huizinga et al., 2006). Surprisingly, performance on the Intradimensional/Extradimensional (ID/ED) subtest of the CANTAB appears to reach adult levels by approximately 8 years of age (De Luca et al., 2003; Luciana & Nelson, 1998). However, this early maturation may be task-dependent. Switching tasks with larger inhibitory and/or working memory demands may result in a slower, more prolonged developmental trajectory.

2.3.2.1 Working memory: The ability to hold information across a delay is present in the first year of life. Working memory functions, such as capacity and the ability to manipulate online information, develop gradually during the preschool period (see Garon, Bryson, & Smith, 2008, for review), and continue to develop between 8 and 13 years of age (Lehto et al., 2003), reaching adult-like levels after age 15 years (Huizinga et al., 2006). The verbal and non-verbal components of working memory appear to follow similar linear performance increases between 4 and 15 years of age (Gathercole, Pickering, Ambridge, & Wearing, 2004).

Results from cross-sectional studies of performance on the Spatial Working Memory CANTAB subtest are consistent with this developmental trajectory. Specifically, adult-like performance is achieved around the age of 15 years (De Luca et al., 2003; Luciana, Conklin, Hooper, & Yarger, 2005). Furthermore, results from cross-sectional studies of memory-guided saccade paradigms have revealed that the latency and accuracy of initial saccades mature by approximately 14–15 years of age (Luna, Velanova, & Geier, 2008).

2.3.2.3 Inhibition: Simple inhibitory processes come online within the first year of life and develop during preschool years, while more complex inhibitory processes appear later in preschool years and continue to develop through school age (Garon et al., 2008). By age 11 years, children perform at adult levels on the Eriksen Flanker task and Stop-Signal task (Huizinga et al., 2006). Inhibitory abilities, as measured by the ANT (identical to the Eriksen flanker task), increase between four and seven years (Mezzacappa, 2004; Rueda et al., 2004), and remain static from the age of seven into adulthood (Rueda et al., 2004). Similarly, there appears to be no relationship between age and inhibition between the ages of 8 and 13 years (Lehto et al., 2003). Yet, performance on the anti-saccade task does not reach adult levels until 14–15 years of age (Luna et al., 2008).

2.3.3 Executive control network in ASD—Executive dysfunction in ASD has been extensively reviewed (Geurts, Corbett, & Solomon, 2009; Hill, 2004a, 2004b; O’Hearn, Asato, Ordaz, & Luna, 2008; Ozonoff, South, & Provençal, 2005; Russo et al., 2007). Ozonoff and colleagues (2005) concluded that individuals with ASD demonstrate relatively intact inhibitory and working memory processes, but impaired cognitive flexibility/set shifting abilities (although see Geurts, Corbett et al., 2009, for discussion of intact cognitive flexibility). Although previously thought to be a primary deficit in ASD, absence of early executive control impairments in 3- to 4-year-old children with ASD (Dawson et al., 2002; Griffith, Pennington, Wehner, & Rogers, 1999; Yerys, Hepburn, Pennington, & Rogers,

2007) suggests that dysfunctional executive control processes may be a secondary to the development of ASD.

Two caveats should be mentioned: First, approximately 30% of children and adolescents with ASD receive a co-morbid diagnosis of ADHD (Leyfer et al., 2006; Simonoff et al., 2008), and profiles of executive impairment may differ between ASD individuals with and without ADHD (Sinzig, Morsch, Bruning, Schmidt, & Lehmkuhl, 2008; Yerys et al., 2009). Thus, differential criteria as to inclusion (or exclusion) of participants with a co-morbid diagnosis of ADHD across studies may contribute to the conflicting findings within the ASD executive control literature. Second, as is likely the case for many of the findings discussed here and above, the intellectual level (high- versus low-functioning) of the sample is an important factor as lower IQ is generally associated with reduced executive control (Keehn et al., 2010; Liss et al., 2001; Lopez, Lincoln, Ozonoff, & Lai, 2005; Steele, Minshew, Luna, & Sweeney, 2007; Williams, Goldstein, Carpenter, & Minshew, 2005).

2.3.3.1 Set Shifting: Initial evidence of impaired set shifting in ASD was established with the robust and well-replicated finding of perseverative deficits on the WCST (see Hill, 2004a; Ozonoff et al., 2005, for review of findings); however, the WCST is not a pure measure of set shifting abilities, and thus impaired performance in ASD may be due to a number of alternative factors (see Geurts, Corbett et al., 2009, for a more detailed discussion).

In addition to the WCST, investigators have begun to use the CANTAB ID/ED subtest as a measure of set shifting in ASD. For example, Hughes and colleagues (1994) found that low-functioning children with ASD performed poorly compared to DD children and MA-matched TD children. Similarly, Ozonoff et al. (2004) observed poorer performance on set shifts in a large ASD sample, ranging in age from 6 to 47 years. However, other studies investigations have failed to find set shift impairments in children and adolescents with ASD (Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009; Goldberg et al., 2005; Happé, Booth, Charlton, & Hughes, 2006; Ozonoff, South, & Miller, 2000). The absence of set shift impairments on the ID/ED subtest is surprising given the prior findings of impaired shifting on the WCST. However, similar ASD and TD performance may result from ceiling effects, given that TD children reach adult-like performance levels on the ID/ED test around age 8 years (Goldberg et al., 2005). Nevertheless, the absence of set shifting deficits in ASD in these studies suggests that children and adolescents with ASD are capable of shifting mental sets. This appears consistent with a neuroimaging study showing similar behavioral performance and brain activation for set switching in ASD and TD (Shafritz, Dichter, Baranek, & Belger, 2008).

Shifting attention has also been examined with the cross-modal shifting paradigm. Courchesne and colleagues (1994) found impaired attention shifting between auditory and visual modalities in adults with ASD, similar to patients with cerebellar lesions. These deficits reflect slowed rather than absent shifts as both groups were able to shift attention when given more time (>2.5s). Electrophysiological results from the same paradigm suggest poor performance by individuals with ASD may result from aberrant distribution of attentional resources (as indexed by the slow negative wave) (Ciesielski, Knight, Prince, Harris, & Handmaker, 1995).

2.3.3.2 Working Memory: The results of studies examining working memory in ASD are inconsistent, possibly due to the varying degree of capacity, maintenance, and manipulation demands of each task. Prior studies using the A-not-B task have demonstrated intact working memory in 3 year-old children with ASD compared to MA-matched TD and DD children (Dawson et al., 2002; Griffith et al., 1999; Yerys et al., 2007). However, by the age

of 5, separate studies have shown that children with ASD perform worse compared to TD comparison children on tasks requiring working memory processes (Dawson, Meltzoff, Osterling, & Rinaldi, 1998a; McEvoy, Rogers, & Pennington, 1993).

Bennetto and colleagues (1996) report impairments on verbal working memory tasks for adolescents with ASD compared to TD individuals. However, poorer performance on these tasks, sentence and counting spans, may be due to the use of a dual-task rather working memory impairment per se. This is supported by Garcia-Villamizar and Della Sala (2002) who found performance to be normal for a single-task working memory condition, but impaired for dual-task working memory in adults with ASD.

Russel and colleagues (1996) reported no difference in working memory between low-functioning children with ASD and DD children. Similarly, Ozonoff and Strayer (2001) tested children on N-back, spatial memory-span, and box search tasks and found no working memory impairment on any task for ASD relative to Tourette syndrome and TD comparison groups.

Williams and colleagues (2005) reported equivalent performance of verbal working memory (N-back, Letter-Number Sequencing), but impaired spatial working memory performance (Spatial Span, Finger-Window subtests) in children and adults with ASD. More recently, impaired spatial working memory performance in children and adults with ASD has been reported using the CANTAB Spatial Working Memory subtest (Corbett et al., 2009; Goldberg et al., 2005; Happé et al., 2006; Steele et al., 2007). However, Happé et al. (2006) reported impairments in children, but not adults, with ASD compared to TD individuals.

Studies employing memory-guided saccades tasks have reported deficits in saccade accuracy in children and adults with ASD (Luna, Doll, Hegedus, Minshew, & Sweeney, 2007; Minshew, Luna, & Sweeney, 1999) and slower saccade latency but similar accuracy (Goldberg et al., 2002), compared to TD individuals. Additionally, adults with ASD showed reduced activation of DLPFC during a memory-guided saccade task compared to TD adults (Luna et al., 2002).

At first glance, the results of the working memory studies reviewed appear inconsistent. Yet, close inspection suggests that the working memory deficits may be due to load (Garcia-Villamizar & Della Sala, 2002) and/or poor or inefficient use of strategies (Corbett et al., 2009; Steele et al., 2007). Neuroimaging evidence is in accord with these hypotheses, indicating that decreased functional connectivity between regions may, in part, play a role in poorer ASD performance during more complex or dual-task conditions (Kana et al., 2007). In addition, atypical activity during working memory performance suggests that individuals with ASD may rely on less efficient strategies, which may result in impaired performance during more difficult working memory tasks (Koshino et al., 2005).

2.3.3.3 Inhibition: Inhibitory processes seem to function similarly in young children with ASD and in TD children (Dawson et al., 2002; Griffith et al., 1999; Yerys et al., 2007). However, typical developmental improvement in inhibitory abilities may be reduced in ASD (Luna et al., 2007; Ozonoff & McEvoy, 1994; Solomon, Ozonoff, Cummings, & Carter, 2008; although see Happé et al., 2006, for evidence on developmental improvement)

On tasks that tend to isolate inhibitory processing from other executive functions, individuals with ASD have shown typical levels of performance (e.g. Ozonoff & Strayer, 1997); although when measures of inhibition are paired with other executive components (e.g. set switching), deficits in inhibitory abilities may be observed in ASD (e.g. Ozonoff, Strayer, McMahon, & Filloux, 1994). Similarly, individuals with ASD tend to have more

difficulty in inhibition tasks that require more cognitive resources (Rinehart, Bradshaw, Tonge, Brereton, & Bellgrove, 2002)

The majority of previous studies have demonstrated *intact* inhibitory abilities in ASD for Go-NoGo (Geurts, Begeer, & Stockmann, 2009; Happé et al., 2006; Kana, Keller, Minshew, & Just, 2007; Ozonoff et al., 1994; Raymaekers, Antrop, van der Meere, Wiersema, & Roeyers, 2007; Raymaekers et al., 2004, 2006), Eriksen flanker (Henderson et al., 2006; Keehn et al., 2010), Start-Signal (Ozonoff & Strayer, 1997), Negative Priming (Brian, Tipper, Weaver, & Bryson, 2003; Ozonoff & Strayer, 1997), and Stroop (Adams & Jarrold, 2009; Ambery, Russell, Perry, Morris, & Murphy, 2006; Bryson, 1983; Christ, Holt, White, & Green, 2007; Eskes, Bryson, & McCormick, 1990; Goldberg et al., 2005; Ozonoff & Jensen, 1999; Russell, Jarrold, & Hood, 1999) paradigms. However, results from anti-saccade tasks have consistently shown inhibitory impairment in children and adults with ASD (Goldberg et al., 2002; Luna et al., 2007; Minshew et al., 1999; Mosconi et al., 2009; Thakkar et al., 2008); however, performance on the anti-saccade task reaches adult-like levels at much later age compared to other inhibitory tasks. This indicates that the prepotency to saccade towards a peripheral target may be one of the more difficult actions to inhibit, and therefore requires an extended period in order to mature.

2.3.4 Summary—Executive control is not a unitary construct, but instead consists of at least three independent, but associated components: set shifting, working memory, and inhibition. These components are mediated primarily by prefrontal cortex and undergo more protracted development compared to the other attentional networks. Moreover, each executive component follows a distinct developmental trajectory, reaching adults levels at varying times between early and late adolescence.

Although previously thought to be a primary deficit in ASD, the absence of early executive control deficits in preschool-aged children with ASD (Dawson et al., 2002; Griffith et al., 1999; Yerys et al., 2007) suggests that executive control deficits may be secondary to the development of ASD. The degree to which executive control abilities improve with development remains unclear. The trajectory of development appears similar in ASD and TD individuals (Luna et al., 2007); however, there are conflicting reports of age-related improvement (Happé et al., 2006) or decline (Solomon et al., 2008), which may be related to the executive component tested (Ozonoff et al., 2004).

Coordinated communication within the distributed network of brain regions responsible for these processes results in efficient and effective modulation of executive control. Functional neuroimaging studies have reported reduced functional connectivity between regions associated with the executive control network (Just, Cherkassky, Keller, Kana, & Minshew, 2007; Kana et al., 2007; Koshino et al., 2005), although these reductions in functional connectivity may be age-dependent (Lee et al., 2009). Just and colleagues (2007) have hypothesized that executive dysfunction in ASD may result from poor coordination between brain regions necessary to complete complex executive tasks. This hypothesis is supported by behavioral results that demonstrate poorer performance on tasks that necessitate multiple executive components (e.g. WCST; see Hill, 2004a. for review).

3. The Role of Attention in the Development of ASD: A Developmental Framework

Attention has often been considered an associated or secondary deficit within the domain of ASD research; instead, the findings laid out above overwhelmingly indicate early and lifelong impairments and abnormalities in efficiently modulating each attentional network. When viewed from a developmental perspective, the relationship between atypical

attentional processes and social and communicative deficits may merge into a coherent model. The following section outlines a developmental framework for understanding the distinct pattern of attentional strengths and weaknesses in ASD, and how these may be related to the emergence of ASD phenotype (Figure 3). Although, much of the following framework is based on inference rather than direct evidence, the model is consistent with the relevant evidence. While ASD is a developmental disorder, very little longitudinal research is available. Therefore many of the links proposed in the current model are suggested, based in part on TD literature, and have yet to be tested in infants and children with ASD.

As discussed in the Introduction, lower-level attentional functions may operate as essential elements for the development of higher-level sociocommunicative processes. Such a model has been used by Karmiloff-Smith (2009) to explain delayed language development in Williams syndrome. What follows is a developmental framework that explores the role that atypical attentional processes may have on the emergence of the ASD phenotype. Because impaired disengagement is the earliest attentional deficit reported in infants at-risk for ASD (Elsabbagh et al., 2009; Zwaigenbaum et al., 2005) and is associated with a later autism diagnosis (Zwaigenbaum et al., 2005), the current framework makes the initial assumption that abnormal disengagement of attention represents a primary disturbance in ASD. Efficient disengagement of visual attention has significant influence on two early skills: arousal regulation and joint attention. Additionally, and more speculatively, disengagement of attention may also be associated with the development of perceptual biases (see Colombo, 1995, for discussion). Thus, dysfunctional attentional disengagement may have sequelae that, in combination with other primary disturbances, result in the heterogeneous phenotypic end-state associated with ASD. Secondary impairments and anomalies in arousal modulation, joint attention, and visual-perceptual processes produce more global deficits in a variety of domains. Each will be discussed in turn.

3.1 Atypical arousal regulation

Shifting attention to distracting stimuli temporally suspends distress in infants (Harman, Rothbart, & Posner, 1997). For example, during face-to-face interactions, infants shift attention away from faces in order to regulate arousal levels (Field, 1981). This suggests that early deficits in disengaging attention may result in the development of atypical arousal regulation. The process of arousal modulation in early infancy involves a dynamic interplay between the internal state of the infant and the level of external stimulation, with a purpose of maintaining a homeostatic state (Gardner, Karmel, & Magnano, 1992).

As reviewed above, individuals with ASD may demonstrate both hyper- and hypo-aroused states, which may be a reflection of either impaired modulation of arousal or separate subgroups of individuals with ASD. While prior studies of older children, adolescents, and adults with ASD have shown atypical arousal regulation, a more recent study by Anderson and Colombo (2009) reported that compared to TD children, 4-year-old children with ASD exhibit increased tonic pupil size, which is indicative of increased arousal. Furthermore, prospective studies investigating temperament in infants at risk for ASD have also demonstrated characteristics of over-reactivity and poor arousal modulation (Bryson et al., 2007; Garon et al., 2009). As hypothesized by previous investigators (Dawson & Lewy, 1989a; Gold & Gold, 1975), abnormal arousal levels would have developmental consequences in a variety of domains including: 1) abnormal perception of novel information, 2) reduced attention to social information, 3) restricted and repetitive behaviors, 4) over-focused attention, and 5) reduced efficiency of executive control abilities.

Arousal levels impact novelty processing (Barry, Clarke, McCarthy, Selikowitz, & Rushby, 2005). In TD infants, decreased efficiency of attentional disengagement is related to greater aversion to novelty (Johnson, Posner, & Rothbart, 1991; McConnell & Bryson, 2005). As a

result of poor regulation of arousal (perhaps due in part to impaired disengagement) novel stimuli and environments may be over-arousing, such that new information is perceived as aversive. Multiple studies using a variety of experimental modalities have shown that individuals with ASD have dysfunctional novelty processing (see section 2.1.3). Furthermore, prior studies have demonstrated that an inefficient alerting system (Keehn et al., 2010) and decreased sensitivity to new information (Keehn & Joseph, 2008) are both related to increased sociocommunicative deficits in ASD.

A largely consistent finding across multiple studies has been reduced amplitude of the frontal (novelty) P3 or P3a component in ASD (Jeste and Nelson, 2009). The P3a, may reflect inhibition of ongoing processing in order to attend and evaluate novel stimuli (Polich, 2007). One model of ASD has hypothesized that the disorder may reflect an abnormally increased ratio of excitation to inhibition (Hussman, 2001; Rubenstein & Merzenich, 2003). Thus, decreased response to novelty in ASD (as reflected by reduced P3a amplitudes) may reflect a primary disturbance in the balance of excitation and inhibition, which is necessary to stop ongoing activity.

Alternatively, the locus coeruleus-P3 hypothesis suggests that the cortical sources of the P3 component (lateral prefrontal cortex and temporal-parietal junction) are modulated by the LC-NE system (Nieuwenhuis, Aston-Jones, & Cohen, 2005). Reduced amplitude of the P3 component in ASD could also reflect atypical subcortical modulation of the LC-NE system, which as discussed in section 2.1.1, is important for modulation of tonic and phasic alertness. The idea that atypical subcortical-cortical anatomical connectivity could result in reduced P3a amplitudes in ASD has been previously hypothesized (Dawson & Lewy, 1989b). More recently, increased tonic pupil size in young children with ASD has been observed (Anderson & Colombo, 2009). Pupil diameter has been shown to be correlated with LC tonic activity (Aston-Jones & Cohen, 2005), thus increased pupil diameter in ASD may indirectly reflect increased tonic activity of the LC-NE system, which could result in reduced phasic responsiveness to novel stimuli.

Due to an unstable arousal system, the novel, dynamic, and complex features of social stimuli and unpredictable nature of social interactions may be overarousing to individuals with ASD (Dawson & Lewy, 1989a). Because infants use attentional shifts to regulate arousal during early face-to-face interactions (Field, 1981), impaired disengagement during these interactions could lead to hyperarousal in infants and toddlers with ASD. Following repeated instances of overarousal during early social interactions, social information may become aversive and individuals with ASD may not perceive social interactions as intrinsically rewarding. This would be in agreement with a previous theory of ASD that suggests that social dysfunction may result from an abnormal reward system associated with social stimuli (Dawson et al., 2002). This appears consistent with the surprising finding that attention to faces in 6 month old at-risk infants is not associated with later ASD diagnosis (Young, Merin, Rogers, & Ozonoff, 2009), which may in turn suggest that inattention to faces and atypical face processing are secondary to impaired attention disengagement and develop later due to earlier overarousal during face-to-face exchanges.

In accord with this idea, Dalton et al. (2005) showed that increased attention to eyes was associated with hyperactivation of the amygdala in ASD, indicative of overarousal resulting from direct gaze. These authors hypothesized that the avoidance of eye contact may be a strategy used by individuals with ASD in order to reduce arousal, and may result in dysfunctional face and gaze processing strategies. These results were replicated by Kliemann et al. (2012), who also report increased amygdala activity in ASD in response to attention to eyes, and, moreover, subsequent gaze avoidance as measured by the frequency of shifting attention away from the eye region. Likewise, previous studies have also shown

that children with ASD exhibit increased arousal (as measured by SCR) to direct gaze compared to TD children (Joseph, Ehrman, McNally, & Keehn, 2008; Kylliainen & Hietanen, 2006), and that increased arousal to direct gaze is related to poorer face recognition skills in ASD (Joseph et al., 2008).

Also in agreement with the hypothesis that the complex nature of social stimuli may be overarousing, Pierce and colleagues (1997) found that the social perception abilities of children with ASD was more impaired with an increasing number of social cues. As argued by the authors, if dysfunctional social cognition was due to impaired perception of social information in ASD, trials with redundant social information (i.e. multiple cues) should improve task performance; however, if ASD social deficits are related to an attentional impairment, then task performance should improve in conditions with reduced attentional requirements. In accord with the latter hypothesis, the authors reported similar performance between ASD and comparison groups for the single cue condition, but impaired performance for the ASD group for the multiple cue conditions, and suggest that dysfunctional arousal modulation may influence attentional capacity for social information, resulting in poorer social perception in situations with redundant social information.

In addition to deficits in communication and reciprocal social interaction, ASD is defined by the presence of restricted and repetitive behaviors (see Turner, 1999, for review). Hutt and colleagues (1964) hypothesized that chronically hyperaroused individuals with ASD may engage in repetitive behaviors (e.g., hand flapping) in order to reduce levels of arousal. Furthermore, they hypothesized that aversive response to novel objects or events may be exacerbated by increased arousal levels, resulting in rigid patterns of behavior and insistence on sameness. A more recent study showing an association between restricted and repetitive behaviors and atypical sensory responsiveness (Gabriels et al., 2008) is in agreement with findings relating arousal level to increased repetitive behaviors (Colman, Frankel, Ritvo, & Freeman, 1976). Interestingly, Garon and colleagues (2009) recently demonstrated that an observational measure of restricted and repetitive behaviors was related to measures of temperament, including attentional shifting and activity level. Because impaired disengagement of attention may result in overarousal, the persistent use of repetitive movements may reflect an alternative means of “self-soothing” (Liss et al., 2006, p. 167) for individuals with ASD. These findings suggest that atypical disengagement and hyperarousal may play a part in the presence of these behaviors.

Liss and colleagues (2006) have hypothesized that over-focused attentional style in ASD may be the result of hyperarousal. Additionally, the authors hypothesize that over-focused attention in these individuals would result in an amplification of sensory information at the locus of attention (and reduced processing of information outside the atypically narrow attentional focus). This is in agreement with Townsend and Courchesne (1994) who showed enhanced sensory processing within a *narrower* attentional spotlight specifically in adult ASD participants with parietal lobe abnormalities. Prior studies with TD adults suggest that increased arousal may result in a narrowed attentional focus and increased suppression of peripheral information (Britton & Delay, 1989; Tracy et al., 2000). Over-focused attention may help explain the superior performance of individuals with ASD (as compared to their TD peers) on a variety of visuospatial tasks (see Dakin & Frith, 2005, for review). Eye-tracking studies have shown reduced fixation durations in children and adolescents with ASD (Joseph et al., 2009; Keehn et al., 2009), indicative of enhanced perceptual discrimination at the locus of attention. Lastly, Liss and colleagues (2006) found that a subgroup of individuals with ASD who exhibited this over-focused profile had greater social impairment, which is congruent with the findings by Joseph et al. (2009) of enhanced visual search ability being associated with increased sociocommunicative impairments in children with ASD. This is consistent with the hypothesis of early increases in arousal resulting in a

narrowed visual field, i.e., enhanced processing within and reduced orienting to stimuli outside a restricted attentional spotlight.

Abnormal modulation of arousal could also have important consequences for the development of efficient executive control abilities. For example, Raymaekers et al. (2004) and Geurts et al. (2009) found that stimulus presentation rate (which modulates arousal levels) differentially affects inhibitory control abilities in individuals with ASD. In addition, children and adolescents with ASD show atypical interdependence between alerting and executive control networks (Keehn et al., 2010). Although speculative, dysmodulation of arousal in ASD may result in differential task-related increases in arousal and subsequent decreases in performance according to the Yerkes-Dodson law (Yerkes & Dodson, 1908). Previous studies have shown that executive tasks result in changes in arousal levels (Hoshikawa & Yamamoto, 1997). The dysmodulation of arousal observed in ASD may be associated with an abnormal task-related autonomic response, which may in turn result in poorer executive performance (because in ASD regulation of autonomic response taxes executive resources). This drain on attentional resources may be especially challenging as children with ASD may have generally reduced attentional capacity (Ming et al., 2005; Vaughan Van Hecke et al., 2009).

Previous research has demonstrated that early measures of executive function are related to novelty processing (Sheese, Rothbart, Posner, White, & Fraundorf, 2008) and self-regulation (Gerardi-Caulton, 2000). Due to early impairments in arousal regulation in ASD, individuals with ASD may recruit or rely on effortful control or self-regulation in order to mediate the states of both hypo- and hyperarousal. Alternatively, Posner et al. (2011) have proposed that efficient executive control abilities may arise from earlier reliance on orienting abilities. They suggest that orienting to novel information may activate executive network functions necessary for self-regulation and facilitating further development of that network. Therefore, early impairments in the orienting network (i.e. impaired disengagement) may have implications for the development of efficient executive control processes.

As a consequence of early-onset abnormalities in the orienting network and dysfunctional response to novelty, networks associated with arousal and executive control would be expected to show atypical interdependence in ASD. This hypothesis is supported by findings of executive deficits being related to arousal modulation (Geurts, Begeer et al., 2009; Keehn et al., 2010; Raymaekers et al., 2004). High levels of arousal (or stress) result in increased release of norepinephrine and dopamine in prefrontal cortex, and have been found to produce impairments in working memory (Robbins & Arnsten, 2009). Children and adolescents with ASD show increased variability of cortisol levels and elevated cortisol after exposure to novel, non-social stimuli (Corbett, Mendoza, Abdullah, Wegelin, & Levine, 2006). Therefore increased reactivity and variability of stress response may contribute to poorer performance in difficult executive tasks (Geurts, Corbett et al., 2009).

Although potential links between impaired disengagement and aberrant arousal regulation in ASD have not been systematically investigated, some studies have shown the presence of both types of deficit in the same individuals (Bryson et al., 2007; Watson et al., 2007). Disengagement of attention has an important regulatory function early in development (e.g., Harman, Rothbart, & Posner, 1997). If this function is impaired, atypical arousal and responsivity to environmental stimuli are a possible developmental consequence. As outlined above, deficits within these functions could have implications for a broad array of domains associated with ASD, including novelty processing, social attention, over-focused attention, restricted and repetitive behaviors, and executive function.

3.2 Impaired joint attention

Joint attention is the coordinated attention between an individual and his/her social partner. In the most common scenario, it simply implies “looking where someone else is looking” (Butterworth & Jarrett, 1991, p. 223, as cited in Moore & Corkum, 1994). Prior research has shown impaired joint attention in children with ASD (see Bruinsma, Koegel, & Koegel, 2004, for review). Early joint attention may depend on the attention-capturing characteristics of an environmental stimulus and on changes in head/gaze direction of the caregiver (Butterworth & Grover, 1990). These early joint attention abilities rely on more basic attentional mechanisms (Frischen, Bayliss, & Tipper, 2007) and begin to develop between 6 and 12 months, continuing to mature into the second year of life.

One such mechanism necessary for successful joint attention is the ability to efficiently disengage attention from the current focus in order to shift and engage the object or event at the locus of the caregiver’s attention (Hood, Willen, & Driver, 1998). Early difficulties disengaging attention could have important implications for successfully responding and initiating joint attention in infants with ASD. In accord with this idea, Charman referred to joint attention abilities as “not a starting point but merely a staging post in early sociocommunicative development” (Charman, 2003, p. 321). Perhaps early disengagement difficulties reflect a possible origin for joint attention difficulties and its developmental consequences. Impaired disengagement of attention in at-risk infants has been observed within the second half of the first year of life (Elsabbagh et al., 2009; Zwaigenbaum et al., 2005), a period in which joint attention abilities begin to appear. Recently, Schietecatte and colleagues (2011) reported a relationship between attention disengagement and joint attention abilities in ASD, i.e., faster disengagement was associated with more joint attention initiations.

Importantly, joint attention abilities in TD infants and toddlers have been linked to both language development and understanding the psychological states of others (Carpenter, Nagell, & Tomasello, 1998). Multiple studies have also linked early joint attention skills to later language (Charman, 2003; Dawson et al., 2004; Presmanes, Walden, Stone, & Yoder, 2007; Thurm, Lord, Lee, & Newschaffer, 2007) and social functioning (Charman, 2003; Presmanes et al., 2007; Schietecatte et al., 2011) in children with ASD. Thus, deficits in disengaging attention may lead to failure to initiate and respond to joint attention, which in turn, could result in delayed or abnormal language acquisition and the impaired mental state attribution skills.

3.3 Atypical perceptual processes

The role of disengagement on the development of perceptual processing biases is currently the most speculative consequence of impaired disengagement (Elsabbagh et al., 2009), and evidence in support of this hypothesis is limited. Individuals with ASD have been shown to excel at a variety of visuospatial tasks relative to their TD peers (see Dakin & Frith, 2005, for review). Two theoretical models, Weak Central Coherence (Happé & Frith, 2006) and Enhanced Perceptual Functioning (Mottron, Dawson, Soulieres, Hubert, & Burack, 2006), have been proposed to explain these “islets of ability.” Both theories posit that enhanced ASD performance is due, at least in part, to a local processing bias. That is, individuals with ASD are biased towards local or featural information rather than global properties of a stimulus. Previous research investigating the mechanisms underlying fixation durations in TD infants has demonstrated that “long lookers” tend to show a local processing bias (Colombo, Freeseaman, Coldren, & Frick, 1995). Colombo (1995) hypothesized that one possible explanation for increased fixation durations (i.e., “long lookers”) may be impaired disengagement of attention associated with immature development of the brain regions associated with the orienting network. These infants may find it difficult to shift attention

away from salient local features of stimuli. However, at this point, whether impaired disengagement leads to the development of local processing bias and enhanced abilities in ASD remains conjecture. Future research investigating the relationship between disengagement abilities and local-global processing biases in both TD and ASD populations is necessary in order to establish this link.

In addition to disengagement abilities, Gardner and Karmel (1995) have shown that level of arousal also interacts with infants' preference for certain stimulus characteristics; higher arousal results in increased looking to less intense stimuli, lower arousal in increased looking to more intense stimuli. Therefore, level of arousal (which, again, is influenced by the efficiency of attentional disengagement) may have important implications for the development of visual-perceptual preferences or sensitivities, and may contribute to the development of atypical perceptual processes in ASD.

3.2 Future Research

Typical development of attentional systems undergoes rapid change during the first years of life. The maturation of alerting, orienting, and executive control functions continues into the school-age and adolescence. Fifty years of attention research on ASD has largely given us static pictures of a developmental disorder after these attentional mechanisms have generally reached adult-like levels in TD individuals. While these studies have provided important information regarding the attentional strengths and weaknesses in ASD, the question whether these impairments are a cause or a consequence of ASD remains to be resolved. More recently, prospective studies of infants at-risk for ASD have provided researchers with a glimpse of early attentional function and its relationship to the development of the disorder. These studies have provided a nascent understanding of the emergence of the autistic phenotype. However, important questions remain; these include, but are not limited to:

1. *How does attention set (i.e., top-down modulation of attention) affect the processing of new information in ASD?* Results from neurofunctional assays of novelty processing show intact or enhanced processing of novel information when individuals with ASD are given an active task, but deficient response to new information during passive tasks. Future research investigating novelty processing in ASD should include conditions with and without a top-down attentional set to examine how task-relevant and task-irrelevant novel information captures attention in ASD.
2. *Why do individuals with ASD take longer to disengage attention?*
Although many studies using the gap-overlap task have reported impaired disengagement, specifically slower saccadic RT to the overlap condition, these studies have yet to determine whether these effects are due to 1) differences in tonic alertness (i.e. increased saccadic RT when target onset is not preceded by fixation offset [phasic warning]), and 2) abnormal or inefficient release of ocular inhibition. Future studies should attempt to elucidate which of these mechanisms results in impaired disengagement in ASD.
3. *Do early arousal levels influence sensitivity to new information and does this impact sociocommunicative development?* A consistent finding across multiple experimental methodologies indicates that individuals with ASD demonstrate dysfunctional novelty detection. However, the question that remains to be fully answered is *why* individuals with ASD do not respond in a typical fashion to the onset of novel information and how this aberrant response develops. Prior studies have shown that inefficient alerting and insensitivity to new non-social information

are both related to greater sociocommunicative dysfunction. Future prospective studies of at-risk infants should investigate how early arousal levels influence sensitivity to new information later in life to determine if atypical arousal regulation results in abnormal novelty processing and deficits in sociocommunicative functioning

4. *How might visuospatial processing strengths in autism develop, do these superior abilities arise from early impairments in disengaging attention or dysregulation of arousal resulting in over-focused attention, and how are they related to sociocommunicative dysfunction?* Future studies should examine the relationship between early arousal regulation and attentional disengagement and the development of visuospatial processing biases, and how over-focused attention and/or a local processing bias may impact the development of social information processing skills in ASD.
5. *Are social and communicative impairments in autism developmental sequelae of abnormal disengagement of attention?* Resolving this question has important implications for both the early identification of infants at-risk for ASD and for developing targeted early interventions. Evidence reviewed above suggests that attentional abnormalities are present and that they are related to impairments in higher-level sociocommunicative processes. However, longitudinal behavioral and physiological research with young children at risk for ASD will help to resolve the question of causality. Such research can tell us whether impairments in attentional disengagement play a role in the development of sociocommunicative deficits associated with ASD.

Lastly, and more generally, future research endeavors would also benefit from a cross-syndrome perspective (see Cornish, Scerif, & Karmiloff-Smith, 2007, for example). Tracking the developmental trajectories of attentional functions in variety of developmental disorders may help to understand how attention impairments may ultimately result in unique phenotypic outcomes. Importantly, understanding the maturation of attentional function in single gene disorders with a high prevalence of ASD (e.g., fragile X, Tuberous Sclerosis) may provide unique insights into the role of attentional dysfunction in those with and without ASD.

3.3 Clinical Implications

If early attentional impairments play a causal role in the development of ASD, then 1) attentional deficits may be used as an early neuro-behavioral marker to identify infants at-risk for ASD and 2) the development of attention-targeted early interventions may remediate abnormal developmental trajectories and improve outcomes in children with ASD.

Early identification of infants at risk for ASD is of paramount importance for successful early intervention. Thus far, prospective research investigating socioemotional function in infants at-risk for ASD has not revealed significant patterns of early social dysfunction (see Rogers, 2009, for review). Thus, current diagnostic tools may rely on abnormal behaviors that appear on a more consistent basis *later* in development. If attentional abnormalities are one of the first characteristics that distinguish infants who are later diagnosed with ASD, diagnostic tools should employ an approach that emphasizes these attentional deficits. These tools may result in earlier diagnosis, which has important implications for successful early intervention that may help reduce the atypical development trajectories in ASD.

Furthermore, if early attentional dysfunction is a primary impairment in ASD, the development of early attention-targeted interventions may help ameliorate the development of higher-level sociocommunicative deficits. Posner and Rothbart (2005) have suggested

that early attentional interventions may be a useful tool to promote cognitive and social development. Because children with ASD evidence early attentional impairments, interventions targeted at atypical attentional networks may produce generalized improvement across multiple domains. For example, previous research has demonstrated that early interventions have been successful in improving joint attention abilities in children with ASD (Kasari, Freeman, & Paparella, 2006; Whalen & Schreibman, 2003). Perhaps training the constituent functions of joint attention earlier in development (disengaging, shifting) may have important implications for both the development of joint attention and arousal regulation abilities. A recent study indicates that these abilities (e.g. disengagement) can be trained in TD infants, and that training may make significant impact on attentional function (e.g. more shifts of attention between individuals and objects during free play) (Wass, Porayska-Pomsta, & Johnson, 2011). Furthermore, the distribution of infant attention seems to be malleable (Jankowski, Rose, & Feldman, 2001) and subtle changes may have a large impact on information processing strategies. Finally, regardless of whether atypical attention plays a causal role in the development of sociocommunicative deficits, integrating knowledge associated with attentional strengths and weakness may improve current intervention strategies (see Koegel, Shirotova, & Koegel, 2009, for example).

3.4 Conclusion

This paper reviews attentional function in ASD within the framework of three attentional networks. Autism spectrum disorder is characterized by attentional dysfunction of the alerting, orienting, and executive control networks. The current body of research has only begun to describe the onset and development of attentional impairments in ASD. Nevertheless, ASD may be characterized by dysmodulation of arousal (perhaps with subgroups of hyper- and hypoarousal) and impaired novelty processing, slowed attentional disengagement and shifting, and poorer performance on complex executive control tasks (i.e. requiring more than one executive function).

As was said by Gold and Gold, “using attentional mechanisms as our fulcrum, we may be able to understand the global nature of autism and appreciate the clinical manifestations of this disease” (Gold & Gold, 1975, p. 76). The proposed developmental framework attempts to explain the diverse and heterogeneous nature of ASD by exploring the hypothesis that atypical attentional disengagement may be *one of many* primary impairments associated with the disorder. This deficit in attentional disengagement results in a cascade of impairment, including poor joint attention abilities that lead to delayed language acquisition and atypical arousal modulation that influences novelty and social-information processing. Ultimately, one goal of future research should be to describe the development of attentional networks in ASD, which may be used to understand the emergence of the ASD phenotype and to develop efficacious early interventions that will aid children with ASD and their families.

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Highlights

- Attention in ASD is reviewed in comparison with neurotypical attentional networks
- ASD is characterized by deficits in alerting, orienting, and executive networks
- A model proposes that deficits in disengagement contribute to emergence of ASD
- Atypical attention may be a useful behavioral marker for early ASD identification
- Efficacious early interventions may benefit from knowledge of attentional function

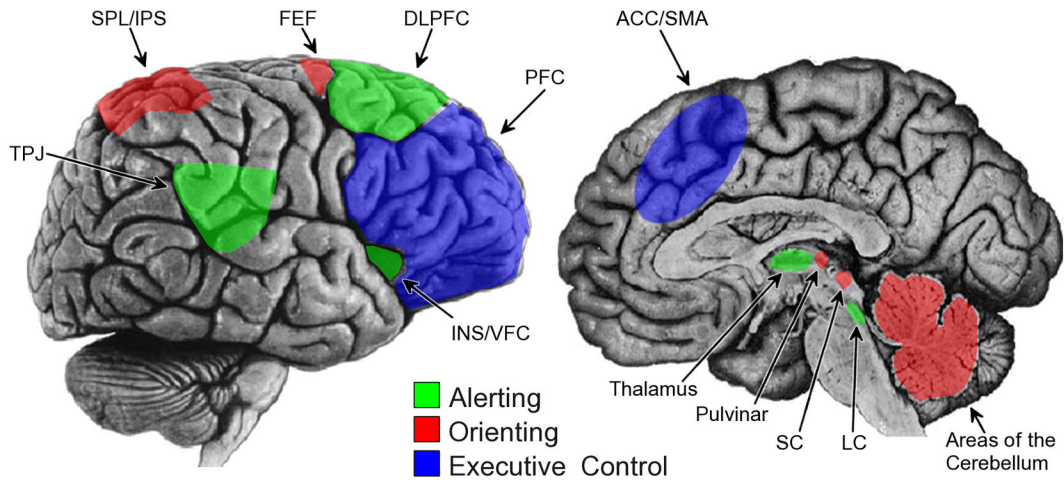


Figure 1.

Illustration of cortical and subcortical regions involved in each attentional network. The alerting network (green) is comprised of right lateralized ventral frontoparietal cortical regions, including the temporal-parietal junction (TPJ), dorsolateral prefrontal cortex (DLPFC), and the insula/ventral frontal cortex (INS/VFC), as well as the thalamus and locus coeruleus (LC). The orienting network (red) includes bilateral dorsal frontoparietal areas (superior parietal lobe/intraparietal sulci, SPL/IPS; frontal eye fields, FEF) as well as the superior colliculus (SC), pulvinar, and areas of the cerebellum. The executive control network includes rostral brain locations, including prefrontal cortex (PFC) and anterior cingulate gyrus/supplementary motor area (ACC/SMA).

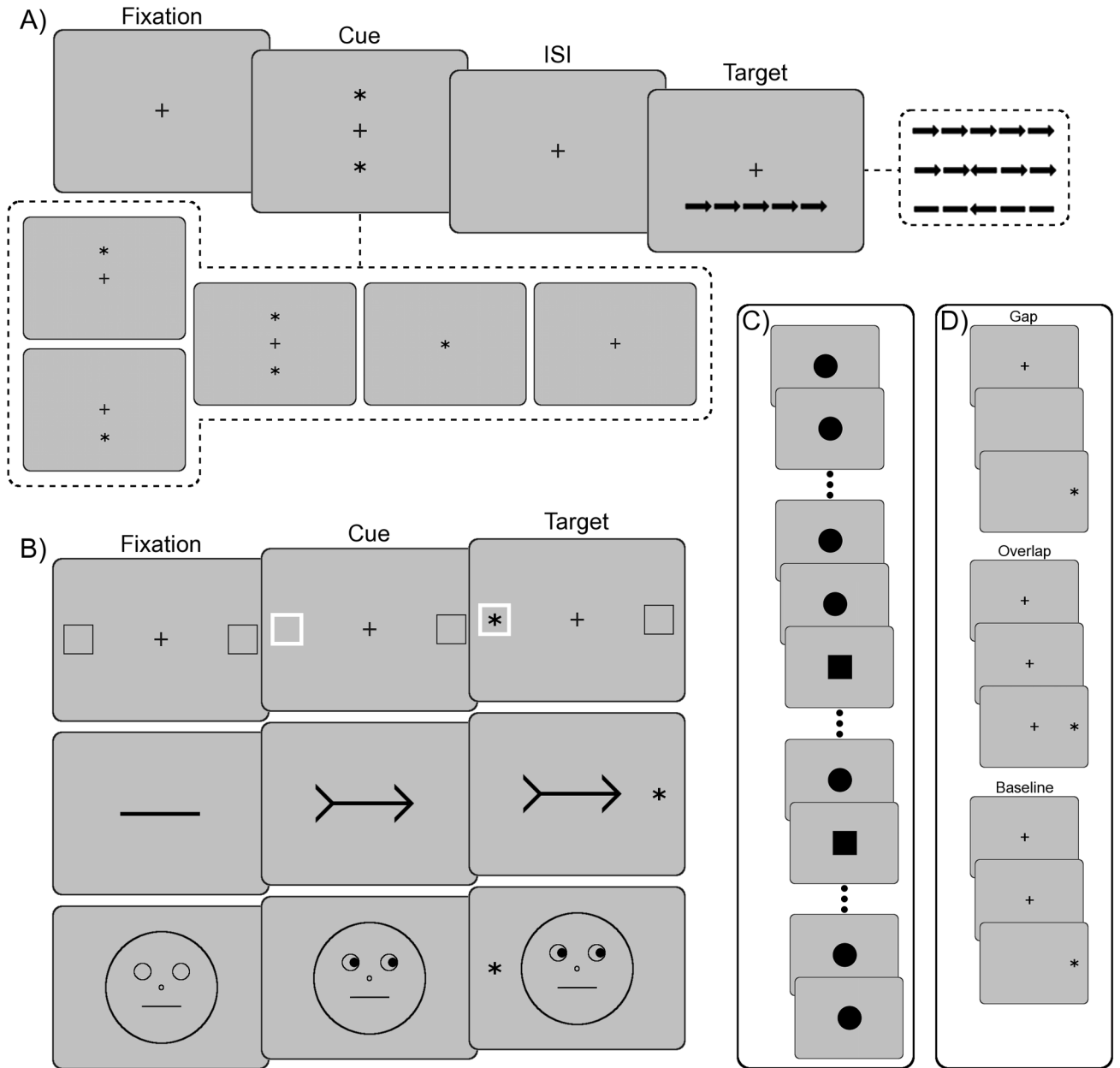


Figure 2. Paradigms used to study discrete attentional processes: (A) The attention network test (ANT) begins with a fixation cross displayed for a variable duration, then a cue (spatial, double, central, or no cue) is presented, followed by an inter-stimulus interval, and a target (congruent, incongruent, or neutral). The task is to indicate whether the central arrow points left or right. (B) Variations of the Posner cuing paradigm including valid flash (exogenous), valid arrow (endogenous), and invalid gaze (endogenous). (C) A continuous performance test (CPT) or oddball paradigm where a target (square) is presented randomly in a series of standards (circles). (D) A gap-overlap task where a target can occur after fixation offset (gap), with the fixation remaining on screen (overlap), or with the simultaneous offset of the fixation (baseline).

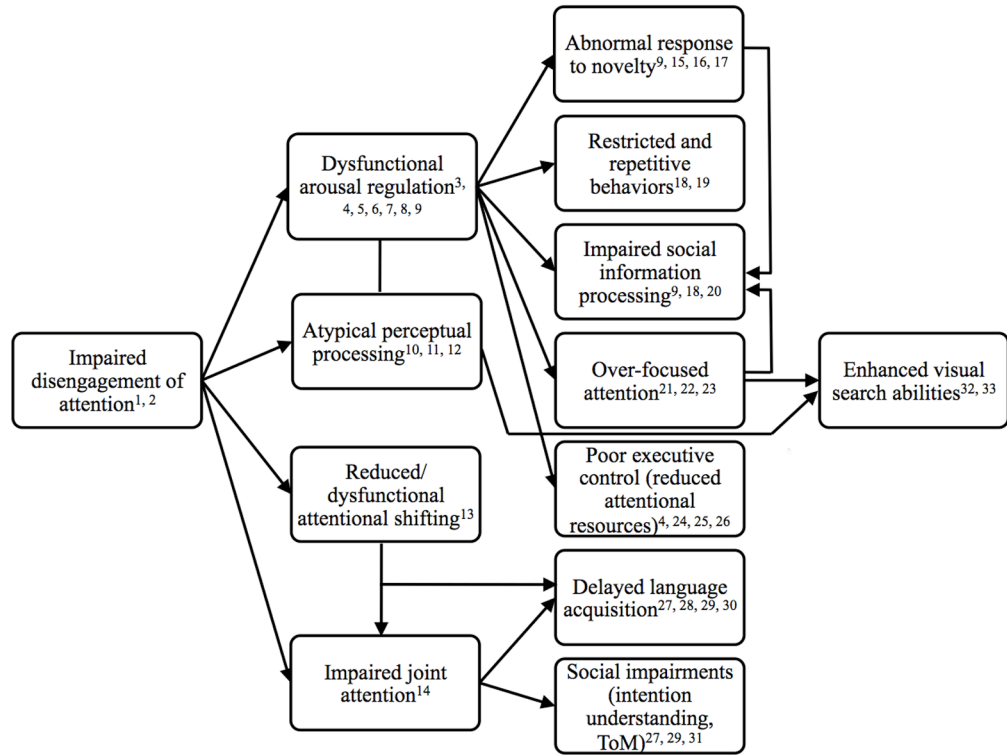


Figure 3. Outline of developmental framework. Bold citations represent longitudinal or correlational findings supporting link or association with ASD symptomatology; Italicized citations represent previous theories that have hypothesized link; Underlined citations represent finding from typically developing literature supporting link. ¹Elsabbagh et al., 2009; ²Zwaigenbaum et al., 2005; ³Anderson & Colombo, 2009; ⁴Keehn et al., 2010; ⁵Field, 1981; ⁶Harman et al., 1997; ⁷Johnson et al., 1991; ⁸McConnell & Bryson, 2005; ⁹*Dawson & Lewy, 1989a*; ¹⁰Colombo et al., 1995; ¹¹Gardner et al., 1993; ¹²*Colombo, 1995*; ¹³*Courchesne et al., 1994*; ¹⁴Schietecatte et al., 2011; ¹⁵Gomot et al., 2008; ¹⁶Keehn & Joseph, 2008; ¹⁷*Gold & Gold, 1979*; ¹⁸Garon et al., 2009; ¹⁹*Hutt et al., 1964*; ²⁰Pierce et al., 1997; ²¹*Liss et al., 2006*; ²²Britton & Delay, 1989; ²³Tracy et al., 2000; ²⁴Ciesielski et al., 1995; ²⁵Geurts et al., 2009; ²⁶Raymaekers et al., 2004; ²⁷Charman, 2003; ²⁸**Dawson et al., 2004**; ²⁹Presmanes et al., 2007; ³⁰Thrum et al., 2006; ³¹Schietecatte et al., 2011; ³²Joseph et al., 2009; ³³Keehn et al., in press.