Chapter I

INTRODUCTION
Autism is a disorder occurring in childhood that interferes with the normal course of social, communicative, and cognitive development. Autism is necessarily a psychological disorder of very early childhood because of the diagnosis of autism is ruled out if the disorder is first manifested later than the third year of life. The other serious psychological disorders of childhood, attention deficit disorder, anxiety, and depression, begin later in life, although there may be precursors earlier on. Because autism begins so early, be disentangled from the predetermined systematic disturbances. Another characteristic of autism is its heterogeneity in terms of both symptoms and developmental trajectory. Because of this heterogeneity and presence of milder symptoms in relatives of individuals with autism and general population, autism is often described as a syndrome. A major variation in the population of individuals with autism is the co-occurrence or lack of co-occurrence of mental retardation (Sigman et al., 2006).

Acknowledgement of this variation occurred early in the history of the definition of the syndrome. Autism was described in the 1940s by two clinicians who were initially unaware of each other’s work because of the disruption caused by World War II. Leo Kanner in 1943 identified the disorder in early childhood in children whose communicative development was mostly compromised; while a year later Hans Asperger described a clinical picture of less severity and with fewer communicative deficits (Sigman et al., 2006).
Autism spectrum disorders include the three diagnoses: autistic disorder, Asperger’s disorder, and pervasive developmental disorder not otherwise specified (PDD-NOS). Here, the term autism refers to this group of diagnosis. No diagnostically informative biological test for autism exists. The diagnostic criteria are behavioural, including specific number and levels of impairment in the three core domains: social interaction, communication, and repetitive or stereotypic behaviour (Newschaffer et al., 2007).

Individuals who have some of these difficulties but whose behaviour does not reach criterion for the full syndrome are diagnosed with pervasive developmental disorders. The latter is diagnosed with no history of language delay, although the differentiation of Asperger disorder and high-functioning autism is often difficult to make. Children with autism are sometimes described as high-functioning or low functioning depending on the presence or absence of mental retardation as reflected in an IQ higher or lower than 70. Between 50% and 70% of children with autism have an IQ lower than 70. The growth in agreement on diagnostic criteria progressed along with research on core deficits and with the creation and standardization of instruments to measure and score the presence or absence of these deficits (Sigman et al., 2006).

Other developmental, behavioural, psychiatric, and medical conditions commonly co-occur with autism. Mental retardation has historically been an associated diagnosis in children with autism. Behavioral difficulties may be related to core features (e.g., perseveration, hyperactivity, self-injury), or sensory abnormalities. Psychiatric symptoms (e.g., anxiety, and depression) may be influenced by severity of core deficits, cognitive impairments, and/or
comorbid medical disorders. In children with autism, specific genetic, neurologic, or metabolic disorders are identified as etiologic factors. Many other medical symptoms or disorders are commonly reported in children with autism: seizures, immune system dysregulation, gastrointestinal symptoms, feeding difficulties like refusal, selectivity, selectivity to textures, and sleep disruption (Newschaffer et al., 2007).

DEVELOPMENTAL FEATURES OF AUTISM

The developmental psychopathological perspective on autism explains the empirical findings concerning atypical behaviour and development within the context of normal principals of development. From this perspective, the concepts of normal development highlight the specific types of normal deviations, abnormalities, rates, and patterns of development of individuals and groups with perspective disorders.

SOCIAL FUNCTIONING IN AUTISM

According to Volkmar et al. (2005) social difficulties are the most powerful predictor of diagnosis for older individuals with autism; this likely is true for infant as well. Preschool children with autism often fail to demonstrate social skills typically present in the first months of life. Rutter (1978) emphasized that the unusual social development observed in autism was one of the essential features for definition; it was distinctive and was not just a function of associated mental retardation.

As cited by Berger (2006) Kanner emphasized, social characteristics of the normally developing neonate and infants are aberrant in autism - the individual has difficulty forming emotional ties with parents. Lord and Rutter
(1994) characterize the difficulties as being in ‘reciprocal social interaction and ability to form relationships’, including failure to use mutual gaze, facial expression, joint attention, and negative reactions to physical contact. By the age of 2 years, the typically developing toddler has skills in social awareness and interaction, imitation, symbolic play, and communication through gestures and language. Many of these aspects have been found to be disturbed in children with autism.

**Gaze**

According to Skuse (2003) eye contact ‘avoidance’ is sometimes as a feature of autism. Normally developing infants spend a significant proportion of their waking time in eye contact with their caregivers; persons with autism, however, fail to establish this pattern of mutual gaze.

Dawson et al. (2000) reported that eye contact is limited as is overall social engagement and responsivity. Studies with preschool-age and older children reveal that the human face holds little interest for children with autism; similarly they appear to lack a preference for speech sounds. Gaze behaviour also varies as a function of developmental level; more developmentally advanced children exhibit an increased frequency of eye contact (Sigman., 1992).

**Joint Attention**

Joint attention skills involve attention with others through pointing, showing, and coordinated look between object and people. It is one of the earliest emerging social behaviour and deficits in joint attention are apparent
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prior to language acquisition. Kasari et al. (2006) cited theoretically that joint attention represent beginning understanding of the mental representations of others and understanding others leads to better social-cognitive and language abilities. According to Charman et al. (2003) significant associations are found between early joint attention skills and later language abilities.

Overtime, initiation and response to bids for joint attention do increase in children with autism, but are severely compromised in natural contexts. The pattern of these skills in autism may be atypical (Volkmar et al., 2004).

Imitation

For typically developing children, imitation and play are important for symbolic and social-cognitive development. Difficulty with imitation of others people movements appear to be particularly affected in autism. Problems with imitation discriminate children with autism from those with other developmental disorders (Rogers et al., 2003).

The capacity to imitate also appears to be a prerequisite for subsequent symbolic activities; here too, children with autism display serious deficits (Prior et al, 1979). Studies have revealed that younger children with autism consistently have problems in imitating simple body movements and those that involve objects (Stone, Ouslely, & Littleford, 1995., Charman et al, 1997)

Older children with autism consistently have difficulties in imitation (Hobson & Lee, 1999., Loveland et al., 1994, Smith & Bryson, 1994), and these difficulties are present by at least second year of life (Charman et al.,

**Play**

Play activities in the typical child progress from simple object exploration to functional object to pretend play. Although the evidence on this issue is limited, in the first months of life strong differences are not observed between infants with autism and typical or delayed peers. But by nine to twelve months, abnormalities are evident and become progressively more deviant due to higher levels of perseveration (Osterling et al., 2002). Although early functional play routines may be observed, by around age 2 years, differences from typically developing peers are striking: play is less purposeful, less symbolic, and less developmentally complex.

Volkmar et al. (1997) found lack of symbolic play in autism may emerge from social difficulties or may be part of a more general problem in achieving symbolic thoughts and language. Parents reporting on the play of children with autism say it lacks social engagement and is characterized by repetitive and stereotyped object manipulation and non-functional use of objects.

**Attachment**

The relative absence or oddness of attachment behaviours in children with autism indicates their failure to forge basic social-affective connections with the important figures in their lives (Lord, 1993). By the end of the first years of life, typical infants have developed strong pattern of attachments;
suck behaviours maintain the proximity of the child to the caregiver while also facilitating exploration (Rutter, 1995). Children with autism do form attachment to parents (Capps et al., 1994) and differentially respond to familiar and unfamiliar individuals. However, the quality of attachment behaviours may be unusual. Among younger children with autism, attachments to unusual objects are relatively common, but the significance of this is unclear (Volkmar et al., 2005).

Rutgers et al. (2004) also report that children with autism display attachment behaviour to their attachment figure when distressed, and that they discriminate between parents and stranger by directing more social behaviour to the caregiver than to the stranger.

**LANGUAGE AND COMMUNICATION IN AUTISM**

According to Bailely et al. (1996) serious abnormalities in language and communication are a fundamental feature of autism. As well as language delay, there are marked qualitative abnormalities. Language level is a good predictor of social educational outcome, and is strongly associated with severity of behavioural symptoms, social cognitive performance and familial loading. The pivotal role of language features, together with the finding that some halves of individuals with autism do not acquire useful language at all, means that language impairment must be accounted for in many psychological theories. Current psychological approaches to autism have attempted to explain the language features as one consequence of specific cognitive deficits.
Even before they begin to produce spoken language, pattern of sound production in children with autism are abnormal (Witherby et al., 2000), as is vocal quality (Sheikopf et al., 2000), a likely precursor of the noteworthy deficits in information and vocal quality seen later (Shriberg et al., 2001). The development of non-verbal communicative abilities is intimately involved for typical children in the development of conventional communicative abilities and marks the beginning of intentional communication (Bates et al., 1979). However, very young children with autism communicate less frequently than matched developmentally delayed children. They are less likely to use contact and conventional gestures but are more likely to use highly unconventional gestures such as manipulating their conversational partner’s hand to obtain objects (Stone et al., 1997). Children with autism have difficulties with both the expression and production of affective responses, and the range, frequency, and integration of affective display are unusual (Yirmia et al., 1989). Children with autism are less likely to look at an apparently distressed adult (Sigman et al., 1992) and they have difficulties imitating facial display of emotion (Loveland et al., 1994).

According to Rutherford (2005) some people with autism exhibits echolalia or immediate repetition of both words and intonations in lieu of communicative speech. Language acquisition in autism is delayed compared to typical development and comprehension of words is delayed relative to production of words. In contrast, articulation skills seem to be spared.

Osterling et al. (2001) reports verbal children with autism do not readily use language to share information have posited that this phenomenon is attributable to a lack of curiosity. Children with autism have difficulties
with initiating and maintaining topics in conversation, conversational turn taking, and maintaining an appropriate level of detail as well as having difficulties with speaker-listener relations and pronominal reversal. Thus many language impairments in people with autism pertain to the social use of language. This fact has led some investigators to relate at least some of the language problems found in autism to impairments in social relatedness or lack of comprehension of speaker-listener conversational rules.

**COGNITIVE FUNCTIONING IN AUTISM**

The earliest formulation of the syndrome, emphasizing the social and emotional aspects of the disorder (Bettelheim, 1967; Kanner, 1943) was succeeded by two major conceptualizations. One group of investigators viewed the autistic person’s deviations in perceptual processing as the primary disorder (Ornitz & Ritvo, 1976); other theorists focused on the individuals problems in language development and usage (Churchill, 1972).

One consistent observation made first by Kanner (1943) concerns the emergence of noteworthy discrepancies across developmental areas. This developmental decalage has its origin in early childhood (Sigman & Ungerer, 1981), and it not only persists overtime but it often becomes more striking (Klin et al., 1995). Differences in sensori-motor development do not seem to be syndrome-specific (Cox et al., 1999., Dawson et al. 2002). Although studies of older children have demonstrated difficulties in executive functioning (Pennington & Ozonoff 1996), studies with younger children generally have no syndrome specific differences; in some instances preschool children with autism perform better than matched controls on tasks.
The significance of cognitive dysfunctions in autism become clear from research studies of perceptual and language deficits (Rutter, 1983). These studies showed that profound and syndrome specific impairments were found only in higher-level perceptual processing. Thus, careful investigation of language and perceptions laid the foundation for studies of cognition in autistic populations (Sigman et al, 1997).

Bailey et al. (1996) reports that the notion of low level sensory perceptual deficits, such as stimulus over-selectivity or sensory dominance, was replaced by the concept of a general high level cognitive dysfunctions in deriving meaning from structured or semantic information. Burack et al. (1997) observed that a multiplicity of mechanisms and processes are attributed to attention which is considered essential to both typical and atypical development. Attention is viewed as a potential candidate in research for a core deficit association with autism.

Attention

According to Burack et al. (1997) intentional abnormalities in older children with autism are well documented. These included fixating on apparently irrelevant stimuli, to the exclusion of other information in the environment, perseverating on self-initiated tasks and foci of interest, and difficulties in focusing on educational tasks. Difficulties in autism have to do with selective attention, with problems in attending to multiple aspects of stimuli and with auditory stimuli in particular. Sustained attention is considered relevant to autism. Casey et al., (1993) found minor differences in sustained attention in high functioning male adult savants with autism. Intact visual sustained attention was also found among lower functioning persons.
with autism. There is preliminary evidence that abilities in auditory sustained
attention may be deficient. Reflexive visual orienting of both the physical eye
and mind’s eye is of interest because of common reports that person with
autism perseverate in looking at and acting on idiosyncratic stimuli, are
immune from distractions when so engaged, and appear to have problems in
voluntarily shifting attention from one stimulus to another.

Infants later diagnosed with autism showed that they attend less to
people than to objects as compared to both typically developing and delayed
controls (Baranek, 1999; Mastero et al., 2002; Osterling et al., 2002), this also
true after the second year of life (Dawson et al., 1998). Selective social
attention is particularly impaired, for example; during free play 20-months-
olds with autism were more likely to look at objects than at people than were
either typical or delayed peers (Swettenham et al., 1998). Recent work
employing new approaches to the study of social attention has shown that
older individuals with autism exhibit markedly deviant pattern of attention to
people and, particularly, to faces as compared to developmentally matched
controls (Klin et al., 2002). Similar findings are emerging with very young
children with autism.

Various attempts have been made to account for early differences in
social-visual attention. The problem might have to do with avoiding complex
visual stimuli, for example, faces (Swettenham et al., 1998), or with avoiding
unpredictable and variable social stimuli (Dawson & levy, 1989). A third
hypothesis posits the fundamental problem as a lack of social motivation and
salience. Chawarska et al. (2003) in a study of automatic intentional cueing in
2-year-olds focused that the visual attention of the children with autism could
be cued by directional changes in eye movement, although cue-specific differences also suggested different underlying strategies.

**Memory**

According to Mottron et al. (2001) memory deficits such as poor recall of recent events have been reported in individuals with autism. Abnormal memory functioning is consistent with ‘executive’ models of autism that predict deficiencies in the ability to use efficient encoding and retrieval strategies.

An alternative explanation would relate these impairments to either an episode or amnesic-like deficit, or to a semantic deficit. However the prediction of an amnesic deficit was only partially supported by empirical research on memory in individuals with autism. Impairments in immediate recall of words list (Boucher & Warrington, 1987) were reported in low-functioning persons with autism. Bennetto et al. (1996) reported that high-functioning individuals with autism display typical immediate and delayed recognition of visually presented material; delayed matching to sample tasks, long-term recognition, cued recall, or new learning ability.

**Discrimination learning**

Hermelin and O’Connor, (1970); Maltz (1981) and Prior, (1979) suggests that autism is not associated with pervasive deficits in the capacity to discriminate stimuli. Autistic individuals easily learn simple discriminations of line position, length of line, and dimensionality. Deficiencies in discrimination learning occur only when the stimuli to be differentiated are symbolic (Minshew, Goldstern, Munez & Payton, 1992), and these difficulties
can be surmounted when the material is made concrete (Fein, Tinder, & Waterhouse, 1979). Charlop and Carlson (1983) found that autistic children are also able to learn both reversal and non-verbal shifts and do so at the appropriate ages.

Frith (1972) reported the scientific evidences proving the limitations in autistic individual’s abilities to sequence information in both auditory and visual modalities. Hermelin and O’Connor, (1970) found that greater difficulties are demonstrated in temporal sequencing than in spatial sequencing. Deficits in cross-modal learning have been reported in some studies but not in others.

**Intelligence**

Autistic children have deficits both in cognitive processes and in stored knowledge, with the major impairments occurring in the verbal domain. About 75% of autistic children have IQs below 70, the cut off for the administrative definition of mental retardation. However, intelligence is not uniformly impaired across all the domains usually tested. Individuals with autism, regardless of overall intelligence, have an almost unvarying characteristic cognitive strength in common: the Block Design subtest on the Performance Subscale of the Wechsler Intelligence Scales (Seigel et al., 1996). Almost all autistic individuals do very poorly on another Wechsler sub test, Picture Arrangement. This illustrates the great difficulty that autistic individuals have with narrative logic (Folstein, 1999). Rutter (1983) has interpreted the pattern of scores shown by autistic children on standardized intelligence testes and in other testing situations as indicating special difficulties in sequencing and abstraction. Autistic children show few specific
deficits in spatial performance, perceptual organization, and attentional short-term memory skills (Sigman et al., 1997)

Bailey et al. (1996) report outside of standardized or experimental measures, there are frequent reports of so-called splinter skills or islets of ability. These talents may be in reading, spelling, mathematics, music or drawing. These relatively preserved abilities, in the context of overall intellectual disability, do not seem to be especially rare. A small proportion of individuals develop an isolated talent to a level that is in excess of that found in normal people—the “idiot savant” phenomenon. Although the savant phenomenon is not limited to autism, the great majorities are autistic, and their spectacular abilities in memory, music, calculation or drawing sometimes wane as their autistic symptoms improve.

RESTRICTED AND REPETITIVE BEHAVIOURS IN AUTISM

According to Osterling et al. (2001) approximately half of children with autism are reported to display stereotypic motor movements, the most common of which are rocking; toe walking; arm, hand, and finger flapping; and spinning. These stereotypes are more prevalent in children with autism who are young or low-functioning, with higher-functioning children with autism exhibiting more complex repetitive behaviours. These ritualistic activities may include the repeated arrangement or ordering of objects, engagement in a complicated yet seemingly arbitrary sequence of motor movements, or an insistence on sameness in terms of physical environment, sequence of events in the course of a day or a given procedure, or eating habits. Individuals with autism may tend to exhibit restricted behaviour in terms of particularly intense, circumscribed interests or preoccupations with
unusual subject matter. These individuals may perseverate on these topics and are likely to exhibit encyclopedic knowledge of pertinent factual information.

THEORIES ABOUT DEFICITS IN AUTISM

The neuropsychological level of explanation is intermediate between brain and behavior. It attempts to link these two levels of analysis by mapping relationships between brain structures and the psychological and behavioral functions they serve. The theories of autism, each of which postulate a core neuropsychological impairment that is argued to account for defining behavioural features of autism. There are three classes of theories postulated to explain the developmental deficits in autism.

Social-Cognitive Theories

The first class of theories suggests that the principle disorders in autism stems from an innate lack of responsiveness to social stimuli, and that these disorders give rise to subsequent social and cognitive impairments. These social-cognitive theories stem from Kanner’s original formulation of the autistic syndrome and are based on and supported by extensive research in the developmental tradition.

Research on the development of typical children shows that neonates prefer facial representations to object representations (Reddy et al., 1997) and are capable of imitating the facial gestures of social partners (Meltzoff & Moore, 1983). By 3-4 months of age, infant differentiate familiar from unfamiliar people and are sensitive to variations in the emotional and social responsiveness of their caregivers (Tronick et al., 1978). Triadic interactions, in which the infant and a social partner share their experience of an object or
another person, emerge in the second half of the first year of life (Bates et al., 1979; Bruner, 1983; Carpenter et al., 1998). By the second half of the second year of life, typical infants engage in episodes of pretend play in which they represent elaborate social interactions from point of view of different social partners.

Many studies have been conducted to assess the social capacities and behavioural tendencies on developmental level. Children with autism are less likely to imitate others (Rogers, 1999), to follow the gaze and pointing of others (Loveland & Landry, 1986; Mundy et al., 1986; Sigman et al., 1999), to imitate shared looking with others, and to engage in pretended play than are typical or mentally retarded children without autism.

Failure to attend to crucial social stimuli from early on could lead to the deficits observed in older children and adults with autism on a variety of face and voice-processing tasks. Perhaps not surprisingly then, neuroimaging studies provide evidence for abnormalities in the neural systems underling face and voice processing in individuals with ASD. In typical individuals, the lateral aspect of the fusiform gyrus responds preferentially to face over other objects (Kanwisher et al., 1997). In contrast, several functional magnetic resonance imaging studies have now observed reduced activity in the FG both in children and adult with autism spectrum disorders.

A related area of research in both typical and autistic development has focused on the concept of “theory mind,” Based on studies of Premack and Woodruff (1978) and Wimmer and Perner (1983), this concept refers to the ability of children to take the perspective of others so that they understand that other people have intentions, knowledge, and beliefs that may differ from
their own. Extensive research has corroborated the original observations of Baron-Cohen et al. (1985) that children with autism have great difficulty with theory of mind. Some studies have found that autistic children with high levels of intelligence are able to take the perspective of others sufficiently well. In fact, some high-functioning children with autism are even able to solve second-order theory of mind problems, in which they have, reasoning capacities (Bowler, 1992). However, even those individuals who perform successfully on second-order false belief tasks often have difficulty providing appropriate justifications for their responses and are impaired on advanced tasks that are more naturalistic story materials involving the comprehension of white lies, double beliefs, and irony, for example (Happe, 1994).

In the past decade, neuroimaging researchers have used a wide variety of mentalising tasks and converged up on a network of brain regions associated understanding the mental states of others. This network includes the medial prefrontal cortex, the STS, and the temporal poles. Three studies have now examined the neural basis of impairments in theory of mind in autism and all have found abnormalities in the activation of the medial prefrontal cortex (Happe et al., 1996; Castelli et al., 2002; Nieminen-von wendt et al., 2003). One of the difficulties with studies of children with autism stems from the fact that diagnosis of autism is often not made until children reach 3-5 years of age. This means that many other processes may have gone awry in the early development of the children. Therefore, the lack of social responsiveness may stem from earlier deficits in attention or cognitive processes that have resulted in the children being isolated socially and emotionally from other people.
One conundrum in understanding these social deficits is that there is little information about why the children with autism do not engage socially. One theory is that typical children are born with a tendency for social interactions are aversive to children with autism could also be born unresponsive to social interaction, which then becomes aversive to them. Another way to consider the social-cognitive theories of autism is from the point of view of specificity, uniqueness, and universality of the deficits. Social deficits are universal in autism and are required for the diagnosis. However, social difficulties are not unique to autism, although the severity and kind of social deficits experienced by individuals with autism are unusual in most other syndromes. Finally, it has been difficult to prove that the social interaction deficits are specific rather than general, in that individuals with autism manifest a variety of other problems such as limitations in shifting attention and possibly, some disturbances in their capacity to create cognitive categories.

**Executive Function Theory**

The fact that the social cognitive theories of autism do not explain all of the symptoms manifested by children with autism has led to the postulation of other theories. Although older, high-functioning individuals with autism have social difficulties of various kinds, they have other problems that seem to interfere with their functioning, particularly in the academic and professional arenas. Typically developing individuals use various executive functions to go beyond automatic activities, such as creating strategies for behaviour, making plans, shifting topics, maintaining a representation in working memory, and solving tasks requiring the ability to
be flexibly innovative (Shallice, 1988). The dorsolateral prefrontal cortex is known to play an important role in working memory and executive functioning. Two recent neuroimaging studies have found that high-functioning individuals with autism show reduced activity in working memory tasks (Koshino et al., 2005; Luna et al., 2002).

Children with autism have more problems with executive functions than do matched groups of comparison children, and children with autism demonstrate these difficulties in their real-life activities (Russell, 1997). Even the most able individuals may not be able to achieve at a level appropriate for their cognitive skills because they cannot plan or carry out an integrated course of action. Therefore, there is much evidence for theory that a core deficit in autism is in executive functions.

One problem in making executive functions central to the understanding of autism is that children with a number of other syndromes, such as attention deficit disorder and schizophrenia, are reported to have similar problems with executive functions. This suggests that executive function deficits are not unique to autism. However, a comparison of performance of different forms of executive function, such as flexibility, planning, and inhibition, shows that children with autism are particularly weak in some of these executive functions, such as flexibility and planning, and relatively strong in inhibition (Ozonoff & Jenson, 1999). In contrast, children with attention deficit hyperactivity disorder have the most problems with inhibition and can more flexibly create strategies and plans. Thus, there is uniqueness in the pattern of
executive function shown by children with autism relative to children with other disorders.

However, some studies by the researchers who were among the strongest proponents of executive function disorders have failed to find a difference between the executive function skills of young children with autism and matched groups of typically developing groups and groups with other disorders (Dawson et al., 2002; Griffith et al., 1999). Obviously, it may be that the children with autism can handle simple, lower-level strategies and behaviour and that impairment is only demonstrated with more sophisticated tasks. If this is the explanation, then this suggests the executive function deficits are central to autism but are not primary, so they only appear as children become older and smarter.

Central Coherence and Information-Processing Theories

Although the social-cognitive and executive function theories of autism go far in explaining the patterns of weakness in the behaviours and abilities of individuals with autism, neither of these theories explains the patterns of strengths in at least some of the children. About 20% of children with autism have special islets of abilities with numbers, music, arts, calendars, and even poetry (Hermelin, 2001). Moreover, as a group, children with autism are quite able to find figures that are embedded in larger forms, complete jigsaw puzzles, and reproduce patterns with blocks as in the block design tasks included in most intelligence tests (Shah & Frith, 1993). In order to explain these strengths and related deficits in using context and top-down approaches to problems, Frith (2003) has proposed that individuals with autism have a limited drive for “central coherence,” and consequently they
focus on details and overlook broader contexts. Thus, the individuals with autistic skills are able to reproduce visual scenes with great precision despite their cognitive limitations. Besides explaining these strengths, the central coherence theory may be applicable to repetitive and stereotyped behaviours as well as the narrow interests and over selectivity demonstrated by many high-functioning individuals with autism.

Related to the central coherence theories are theories that attribute cognitive problems to a disorder of information processing. Recent evidence has narrowed this hypothesis to complex information processing based on the finding that high-functioning individuals with autism have difficulties with memory for complex information as well as for high-order interpretation aspect of language and concept formation (Minshew et al. 1999). In contrast, the performance of the high-functioning adolescents and adults with autism on attention, sensory perception, elementary motor abilities, simple associative memory, formal language, and the rule-learning aspects of abstraction did not differ from the performance of adolescents and adults without autism. Thus, the cognitive profile in autism involved selective deficits in complex abilities in the domains demonstrating deficits. It has been proposed that the basis of this pattern is the under development of neural connectivity between neural systems (Just et al., 2004).

**Brain Mechanism in Autism**

Regarding the significance of brain in autism different areas have emerged as related to the problem.

**(a) Size**
Kanner’s seminal paper noted that children with autism had enlarged heads. Although subsequent studies of external head circumference confirmed this observation, it did not receive much attention until the past decade when support for this finding began to accumulate via MRI, postmortem studies, and additional head circumference studies. The size increase appears to be a shifting of the entire autism brain and head size distribution rather than merely an excess of megalencephaly among a minority of cases that elevates what would otherwise be a normal autism population mean size (Volkmar et al., 2004).

According to Courchesne et al. (2003) both MRI volumetric analysis and simple measures of head circumference indicate that autism involves transient postnatal macrencephaly. Aylward et al. (2002) found that neonates later diagnosed with autism or PDD-NOS have normal head circumference; but by 2-4 years of age, 90% of these have larger-than-average brain volumes. Recent findings suggest that the brain may be enlarged by as much as 10% in volume in toddlers with autism. However, the magnitude of the effect appears to diminish with age. In adolescence and adulthood the effect is less consistently found across studies and the size of the effect is diminished to a few percent increases at most. Several studies have shown the head is not significantly enlarged at birth leading to the proposal that there are specific events in the first months of life that are responsible for the brain enlargement.
(b) Corpus Callosum

Although different studies have noted various segments of the corpus callosum to be abnormal, they consistently find a reduced callosal size. Indeed, the posterior corpus callosum in autism is actually smaller than normal and the degree of this callosal hypoplasia correlates with the degree of frontal hyperplasia. This compartmental specificity of white matter hyperplasia is consistent with the idea of differential effects on local and long-range connections (Baron-Cohen & Belmonte, 2005). Reductions in white matter may also be consistent with PET study that showed reduced inter-regional correlations in persons with autism, suggesting reduced functional integration and connectivity. One consequence of reduced interconnectivity in the autistic brain might be increased modularity and reduced integration of functions. Such a reduction in neural integration would be consistent with one influential theory that attributes autistic symptoms to a lack of ‘central coherence’ a cognitive processing style that makes integration of parts into wholes problematic (Volkmar et al., 2004).

Given the corpus callosum importance in the lateralization of cerebral functions, its reduced size in autism patients, particularly in the context of an enlarged brain suggests the possibility of aberrant lateralization of brain functioning in the disorder. Many studies have provided clinical evidence of abnormal motor and language lateralization in autism patients, including increased left-and mixed-handedness and an unusual pattern of cerebral dominance for language.
(c) Cerebellum

Evidence implicating the cerebellum in the pathophysiology of autism was originally put forward by Ornitz and colleagues in the 1960s and 1970s; their studies showed that autistic children exhibited clinical and neurophysiologic deficits indicative of cerebellar dysfunction (cited by Ciaranello & Ciaranello, 1995). The cerebellum, in particular, is one of the most consistently abnormal structures in the autistic brain. Post mortem neuropathology studies have revealed that very consistent reductions in the number of Purkinje neurons in the cerebellum. The precise nature of these abnormalities, including a lack of gliosis, suggests a prenatal origin. Although classical neuropsychological models see the cerebellum as strictly a motor system, some evidence finds a role for it in cognition, sensation and attention especially as it participates as part of larger cerebellar-cortical systems. In autism, dysfunctions of cerebellar-cortical serotonergic pathways have been noted and fMRI evidence implicates the cerebellum in attention difficulties seen in autism (Volkmar et al., 2004).

Courchesne (2003) found that cerebellar white matter volumes, combined with vermis size, can discriminate 95% of toddlers with autism from normal controls and can predict whether a child with autism will be high or low functioning. Interestingly, people with autism show abnormally low activation of cerebellar cortex in a visual vigilance task, but abnormally high activation during a purely motor task of self-paced button-pressing and the degree of abnormally high cerebellar motor activation correlates with the anatomical deficits in cerebellar volume (Baron-Cohen & Belmonte, 2005).
(d) **Limbic system**

There is also significant interest in the role of limbic system circuitry, particularly the amygdala and hippocampus, in causal models of autism. Postmortem studies have repeatedly noted abnormalities in these areas, including reduced density, cell size, and dendritic arborization in structures such as the amygdala, hippocampus, septum, anterior cingulated and mammillary bodies. The amygdala, in particular, plays a critical role in emotional arousal, assigning significance to environmental stimuli and mediating emotional learning, and thus it is often highlighted as a core structure in models of autism pathobiology. Interest in the amygdala also stems from its role in perceptual processing of social stimuli. Several studies have now found hypoactivation of the amygdala in autism during tasks involving the perception of facial expressions and during theory of mind type tasks (Volkmar et al., 2004). Anderson and Sobel (2003) suggest that the amygdala’s role in the social cognitive and perceptual process might largely be one of mediating physiological arousal. According to Dawson et al. (1998) and Klin et al. (2003) hypoactivation of the amygdala in autism may reflect nonspecific tasks effects, i.e., less interest in or emotional arousal by task stimuli. This view provides good support for social motivation hypothesis of autism pathobiology.

Baron-Cohen et al. (2000) point out that four lines of evidence converge on the hypothesis of an amygdala deficit in autism. Histopathologically, cell packing density in the amygdala increased in autism. Behaviorally, people with autism show a similar pattern of deficits to those seen in patients with amygdala lesions. In terms of gross anatomy, magnetic resonance imaging
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morphometry suggests that’s abnormal volume of the amygdala, although there is disagreement as to whether amygdala volume in autism may be reduced or enlarged. Physiologically adults with autism spectrum conditions manifest abnormally low activation of the amygdala during tasks of inferring emotion from pictures of the eyes or of the whole face and during passive processing of facial expressions of unfamiliar faces but not familiar faces.

(f) Cerebral Cortex

Regionally, frontal lobes show the greatest degree of enlargement, and occipital lobes show the least (Carper et al., 2002; Piven, 2004); within the frontal lobe, the dorsolateral convexity and medial frontal gyrus-areas that figure prominently in the social brain show significant overgrowth, whereas precentral gyrus and orbital cortex are not robustly affected (Carper & Courchesne, 2005). Thus the cortical areas most affected are precisely those broadly projecting, phylogenetically and ontogenetically late developing regions that are essential to complex cognitive functions such as social behaviour and language (Baron- Cohen & Belmonte, 2005).

In the areas of the brain, reduced activity has been found in left medial frontal cortex during an empathizing task in orbitofrontal cortex during recognition of mental state words and in superior temporal sulcus during passive listening to speech sounds as compared with non speech sound (Gervais et al. 2004).

A more complete picture of brain function and dysfunction in autism can be constructed if one examines not only the deficits associated with impaired empathizing but also the more subtle cognitive differences and even
superiorities associated with strong systemizing. Ring et al. (1999) conducted functional imaging during performance of the Embedded Figures Test-on which behavioural performance of people with autism is superior to normal reveals unusually high activation in ventral occipital areas and abnormally low activation in prefrontal and parietal areas. This theme of abnormally high activation in unimodal or low level processing regions alongside low activation in frontal and other integrative regions recurs in finding of heightened activity during face processing in peristraite cortex (Critchely et al., 2000), inferior temporal gyrus and superior parietal lobule (Hubel et al., 2003), precuneus (Wang et al., 2004), and other areas outside the fusiform face area (Pierce et al., 2001); by comparison, fusiform activity is abnormally low.

Belmonte and Yurigeun-Todd (2003) found that a visual attention task evokes heightened activity in ventral occipital cortex and abnormally low activations in parietal and prefrontal cortices. Just et al. (2004) report that in a sentence processing task, activation is greater than normal in Wernicke’s area and less than normal in Broca’s area, which suggests that processing is enhanced at the level of single words and impoverished at the level of sentential context. In addition, activity in superior temporal gyrus during inference of mental state from pictures of eyes is heightened and connectivity between extrastriate and prefrontal and temporal cortices during attribution of mental states from movements of animated shapes is weakened (Castelli et al., 2002), while prefrontal and medial temporal activations are abnormally low (Baron-Cohen & Belmonte, 2005).
CAUSES OF AUTISM

Despite a large body of literature in a variety of disciplines, the etiology of autism is still unknown. State-of-the-science technique have been used to investigate possible abnormalities in genetics, brain structure, neurochemistry, immunologic systems, and neurophysiology, but no one defect has been identified.

Evidence of Genetic Determinants

Ultimately the cognitive and neural abnormalities in autism spectrum conditions are likely to be due to genetic factors. The sibling recurrence risk for autism is approximately 4.5% or a tenfold increase over general population rates (Jorde et al., 1991). An epidemiological study of same sex autistic twins found that 60% of monozygotic pairs were concordant for autism versus 0% of dizygotic pairs (Baily et al., 1995). When a broader phenotype was considered, 92% of monozygotic pairs were concordant versus 10% of dizygotic pairs. The high concordance in monozygotic twins indicates a high degree of genetic influence, and the risk to a monozygotic co-twin can be estimated at more than 200 times the general population rate (Baron-Cohen & Belmonte, 2005).

Although single-gene disorder and chromosomal abnormalities are likely to be responsible for only a fraction of all autism cases, the co-occurrence of autistic behaviour and certain neurogenetic disorders such as Fragile X syndrome, Tuberous sclerosis complex, Rett syndrome, Chromosome 15q duplication syndrome, and many others is more evidence for a genetic etiology (Sigman et al., 2006).
Immunological Mechanism

The possible involvement of the system in the pathogenesis of autism is another area of intense research. Various mechanisms have been postulated, including exposure to infectious agents and / or specific immune dysfunction. Studies demonstrating increased risk of autism in certain geographical regions (Gillberg et al., 1991) or for particular seasons of implicate exposure to various infectious agents during early pre-or postnatal development. The best studied of these is the increased risk of autism with congenital rubella syndrome (Chess 1971). Others have invoked this mechanism for the putative causal role of the measles, mumps, and rubella vaccine (Wakefield et al., 1998), although this has largely been refuted by epidemiological studies (Madsen et al., 2002; Taylor et al., 1999).

It has also been suggested that individuals with autism have a disordered immunity with both decreased and increased function of different segments of the immune system having been reported (Horing & Lipken, 2001). Recent evidence shows subtle signs of inflammation in the brains and cerebrospinal fluid of individuals with autism (Vargas et al., 2005). The immunological mechanism could be part of a complex cascade of events that are involved with the etiology of autism related to and modulated by genetic predisposition and / or environmental triggers during critical periods in development.

Possible Environmental Influences

Despite the strong genetic influences, some scientists believe there are environmental factors that are likely to autism. Because autism is a
developmental disability in which symptoms must be present before age 3, any causality related exposure must occur at a very early stage in development. Most research has focused on prenatal factors or those occurring in the first year or two of life. Early gestational exposures are especially important because of corresponding anatomical evidence of anomalies occurring very early in fetal brain development. Autism risk is documented to be increased with chemical exposures to thalidomide, valproate, and misoprostal (Arndt et al., 2005). Various perinatal or obstetrical factors have also been implicated such as low birth weight, premature birth, low APGAR scores, abnormal presentation, but data have been conflicting (Larsson et al., 2005).

Major focus is now being turned to a number of chemicals, such as pesticides, heavy metals, or polychlorinated biphenyls (PCBs) and polybrominated diphenylethers (PBDEs), which are known to have neurotoxicity. These are compounds to which humans are commonly exposed in food, air water, or other products and that are being explored as a possible cause of autism and other developmental disabilities through prenatal / or postnatal exposures. The most controversial of these is mercury, which can be found in routine environmental exposures (water, air), dental amalgams, food, and a specific form of which has been administered to babies in increasing amounts over time through a compound used as a preservative in childhood vaccines (thiomorosal). Regarding vaccine toxicity, many scientists pointed to evidence that ethyl mercury is fundamentally a different compound than methyl mercury (Clements, 2004) and that several epidemiological
studies showed no association with increased autism risk. (Andrews et al., 2004).

**INTERVENTIONS**

The driving force for research on autism and pervasive developmental disorders is the provision of increasingly effective treatment and interventions. At the same time the planning of an intervention strategy must be carefully related to the assessment of a child’s or adult’s current level of functioning, an understanding of the individual’s strengths and difficulties, a theoretical and pragmatic model for planning and delivering long term, stage-by-stage programming; and a vision of the individual’s potential future.

**Theoretical Background of Autism Interventions**

One of the reasons why there has been less progress on intervention approaches have not grown out of knowledge and theory about the core deficits in autism. Treatment approaches to autism have been based on the “grand” psychological theories that prevailed when the treatments were originated. The earliest treatment approaches were psychodynamic, in keeping with the mid-twentieth century understanding of autism and of typical development and functioning. The paradigmatic example of this approach was the Orthogenic School founded by Bruno Bettelheim and dedicated to undoing the psychic harm caused by “refrigerator” mothering.

Psychodynamic approaches were succeeded by interventions based on the theories of learning that increasingly dominated in psychology. These treatments, which were much more appropriate for intervention with children with autism, were used to address the deficits in communication and social
behaviour that characterized children with this diagnosis and reduce the manifestation of behaviour problems that interfered with the children’s development. Lovass (1987) pioneered an approach to intervention that was built on the principles of reinforcement that were shown to govern the behaviour of animals and human being by such behavioural theorists of the time. Moreover, Lovass recognized that interventions with children with autism required intense involvement, so interventions were designed for 40-hour-a-week carried out over several years.

The early behaviorists also created methods to assess the gains of individual children over time. These methods were adaptations of assessment techniques used in learning studies with animals and typical children. The creation of these methods allowed for a comparison of the effectiveness of different teaching methods.

Behavioural interventions known as discrete trials continue to dominate the treatment approaches used in many parts of the world. This intensive, individual involvement of therapist with children clearly shapes some of the behaviour of the children in treatment in ways that expands their skills and minimizes their behaviour problems. At the same time, students and followers of Lovass have recognized that some of the gains of the children in treatment do not generalize to situations outside the treatment milieu. This is especially true for skills developed in the communication and social domains. Thus, current behavioural treatments often are carried out in more naturalistic settings to encourage the generalization of skills. In addition, the content of behavioural interventions has changed to focus on
pivotal skills, such as social motivation and self-initiations that form the basis for social engagement (Koegel & Koegel, 1995).

During the same years that behavioural approaches were being designed, an alternative strategy to intervention was constructed based on a community approach to intervention. The strategy used by Eric Schopler in the TEACCH programme was to insure that families were furnished with high-quality diagnostic and treatment services and providing consultation at the level of the school and clinic.

Recently, the trend has been to use more developmental approach to interventions, targeting the areas that have been shown to be at deficit in children with autism. Given that young children with autism show less social orienting and imitation, less capacity to follow the gaze and pointing gestures of others, less referencing of others in ambiguous situations and sharing of interesting experiences, and less pretended play, interventions have been developed that focus on increasing all of these social skills and behaviours. An increasing number of interventions also attempt to foster peer interaction both in mainstreamed and special school programmes. Most of these developmental interventions substitute relationship-based approaches for the more didactic form of teaching that characterizes the behaviourally based interventions.

**The Goals of Intervention**

The aims of intervention vary a great deal depending on the theoretical understanding of autism held by the intervention personnel. At the same time, because of the evidence that individuals with autism with better language score have better outcome later in life; many interventions have been focused
on improving communication skills. In addition, interventions also targets the social behaviours that are so frequently either missing or aberrant in individuals with autism.

According to Bergman and Gerdtz (1997) behavioural interventions have the predominant treatment approach for promoting the social, adaptive, and behavioural functions of children and adults with autism. Behavioural approaches have been adopted increasingly for enhancing personal independence and responsible choice through skill development and habilitative training, increasingly repertoires of prosocial behaviour and leisure activities, and teaching methods of self-control and relaxation. In addition, behavioural interventions have been employed for reinforcing adaptive responses and suppressing maladaptive ones.

Autism is lifelong neurodevelopmental disorder affecting sociability and communication for which no etiological based treatment has been developed. Nevertheless, as there is no cure at present, the word ‘treatment’ should be used only in a very limited sense, reflecting interventions aimed at helping people with autism to adjust more effectively to their environment (Francis, 2005).

Interventions for autistic children cover different psycho educational, behavioural techniques, alternative/augmentative communication, social skill teaching, parental involvement, sensory integration and auditory integration therapy.
Psycho educational

The Treatment and Education of Autistic and Related Communication Handicapped Children (TEACCH) are a comprehensive model of intervention from childhood through to adulthood. TEACCH focuses on understanding the culture of autism; i.e., the differences in the ways in which the people with autism spectrum think, learn, and experience the world, arguing that these cognitive differences underpin autistic symptoms and explain the behavioural problems exhibited. The goal of the approach is to obtain maximum autonomy for the person at all levels of functioning, depending upon their abilities. TEACCH intervention activities includes diagnosis, parent training, education, social leisure skill development, communication, vocational training and supported employment placements.

Behavioural Techniques

Behavioural components are incorporated in many other educational approaches in autism, such as TEACCH. Thus, with the term ‘behavioral treatments refer to those interventions where the Skinnerian-based techniques from the predominant feature of this approach. Although behavioral methodology has been introduced, applied behaviour analysis and discrete trial learning still remains the core feature of behavioural intervention in autism.

Skills in receptive /expressive language, attending to social stimuli imitation, pre-academics and self-help that are deficient are broken into discrete components. They are then taught on a one-to one basis, in school and / or at home, using reveals for the successful completion of each step.
Behavioral techniques of reinforcement, backward chaining, shaping, and prompt fading are used.

In Applied Behavioural Analysis (ABA) approach the focus is on the use of rewards of reinforcement to encourage desired behaviors and the elimination or reduction of unwanted behavior by removing their positive consequences by means of ‘time out’, ‘extinction’ or punishment (Francis, 2005).

In conclusion, the literature shows that intensive behavioral therapy clearly benefits children with autism and yields a high degree of parental satisfaction: however, the original effectiveness claim was overstated and its cost-effectiveness, in terms of time, effort, and money, has been adequately assessed (Howlin, 1997).

Alternative/Augmentative Communication

As interest turned away from enhancing speech to enhancing communication alternative and augmentative communication approaches emerged. These methods are used in conjunction with other interventions, acting as a compliment to the communication domain. As the vast majority of persons with autism are visual thinkers and learners, visual support can help them to make sense of the process of communication, regardless of their level of speech. Picture Exchange Communication (PECS) was developed by Andrew Bondy and Lori Frost, as an augmentative alternative training package, based on Skinnerian rationale, for teaching functional communication to children and adults with autism and other communication deficits. PECS has been shown to facilitate the development of spoken words its principal advantage is the teaching of communication intent and initiation,
a constant setback of the majority of the other alternative/augmentative communication approaches (Bondy & Frost, 2001).

Facilitated communication claims to offer an alternative means of communication to people with speech problems through a facilitator who support their hands, wrist, or arm to help them to use a communicator board or to type words, phrases, or sentences, even if they do not have communicative speech (Francis, 2005).

Social Skills Teaching

Another interesting area of intervention, especially for higher functioning people with autism, is that of teaching social skills. Carol Gray’s Social Stories is a noteworthy complimentary intervention aimed at improving the social understanding of people with autism. The stories are produced in responses to a troubling situation, to explain the how and why of its social context, and for praising the positive achievements of the child. After gathering relevant information about the topic and discussing it with the person, so that it can be tailored to their perspective, a short script is customized to their needs, interests, and abilities, and the specific guidelines of the technique.

According to Gray (1998) Social Stories include factual information regarding the social situation, positive reactions of others in that social situation, and directive statements of appropriate or desired social responses. It is then taught to the person with a relevant title comparing the core information of the social story and using descriptive, perspective directive and control sentence in a specific rate. The incorporation of visual cues in social stories adds to their effectiveness.
**Parental Intervention**

Despite the lack of methodologically adequate studies supporting parent-mediated interventions, both the existing literature and clinical experience suggest that the use of parents as co-therapists provides an economical method of increasing the number of hours a child receives treatment in a constant and consistent way. Diggle et al (2003) found that it offers children the possibility of generalizing what they have learned at school or from the specialists and above all, empowers the parents and makes them feel in control of their child. This results in a better parenting style and the avoidance of distress and disappointment.

**The Efficacy of Intervention**

Although there has been some progress in the design of interventions, the examination of the efficacy of intervention is neglected. There is some suggestion that the age at which treatment begins may be an important factor. Available treatment studies have generally focused on preschool or school-age children, and few studies have directly addressed the issues of intervention in infancy. This problem will become more critical as early diagnosis improves (Volkmar et al., 2004).

Various factors appear to be central in successful intervention programs. Children with less classical autism may respond better than those with more strictly defined autism, and children with better cognitive abilities or higher levels of engagement may respond more positively. For all treatments, generalization of skills learned across setting is critical. Unfortunately, even though earlier detection and intensive intervention have failed to do well even
with excellent intervention; the study of such children may help to clarify how treatments can be more individually tailored to the child. Although there has been a growing interest in teaching approaches for social skills, much of this literature has focused on somewhat older children. In addition, much of information on effective treatments has emerged though single-subject designs. There is a critical need for research that addresses issues of treatment mechanisms and moderators as well as individual differences in response to treatments.

A review of the empirical literature on the various aspects of autism further clarifies the issues.