Review of Literature
II REVIEW OF LITERATURE

The literature pertaining to the study on “Promoting Nutritional Status and Behaviour Pattern of the Autistic Children through Dietary Intervention” is reviewed under the following headings:

A. Prevalence and etiology of autism
B. Characteristics of an autistic child
C. Food and dietary habits of children with autism
D. Diagnostic and treatment methods

A. Prevalence and etiology of autism

Autism is a neurobehavioural and cognitive disorder characterized by impaired development of interpersonal and communication skills, limited interests and repetitive behaviours (www.jisppd.com). There is widespread public concern about the apparent increase in autism based on prevalence studies during the last 20 yrs. Studies in the 1980s and early 1990s reported a prevalence of 4 to 10 per 10,000 children, whereas recent studies have reported a prevalence of 30 to 50 per 10,000 children (Wing and Potter 2002; Barbaresi et.al., 2005; Gernsbacher et.al., 2005 and Billstedt and Gillberg 2005).

The prevalence rate has increased due to heightened public awareness of autism, availability of more medical and educational resources, increased media coverage of affected children and families, and more training and information for physicians, psychologists and other service providers (Cowley 2000 and Committee on Children with Disabilities 2001). Its diagnosis is frequently missed as there is tremendous lack of awareness and knowledge about the disorder among health professionals (Singhi and Malhi 2001 and Kalra et.al., 2005). Debate continues about whether the overall prevalence of autism has increased or whether past rates underestimated true prevalence (Heussler et.al., 2001and Fombonne 2005).

Autism is increasing in proportion in India and as per statistics it is about one in every 200 persons. The alarming proportions by which it is rising can make India the most populous country in the world having such neurological disorder. As the
medical community is unable to find any suitable cause for its root, the only way available is early detection and intervention. Current epidemiologic studies estimate that there are approximately 1.7 million individuals with autism in India (Nair 2007 and www. autism-india.org).

Tables I and II present the prevalence of autism around the world and in selected nations.

### TABLE I
**AUTISM PREVALENCE AROUND THE WORLD (2000-2008)**

<table>
<thead>
<tr>
<th>Continent / region</th>
<th>Country</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>North America</td>
<td>US</td>
<td>1/152</td>
</tr>
<tr>
<td></td>
<td>Canada</td>
<td>1/54</td>
</tr>
<tr>
<td>Europe</td>
<td>UK</td>
<td>1/86</td>
</tr>
<tr>
<td></td>
<td>Sweden</td>
<td>1/188</td>
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<tr>
<td></td>
<td>Finland</td>
<td>1/833</td>
</tr>
<tr>
<td></td>
<td>Denmark</td>
<td>1/833</td>
</tr>
<tr>
<td></td>
<td>Iceland</td>
<td>1/769</td>
</tr>
<tr>
<td>Eastern Asia</td>
<td>Japan</td>
<td>1/112</td>
</tr>
<tr>
<td>Oceania</td>
<td>Australia</td>
<td>1/256</td>
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</tbody>
</table>

(www.autismspeaks.org/docs/sciencedocs/epidemiology_faq.pdf)

### TABLE II
**ESTIMATED NUMBER OF INDIVIDUALS WITH AUTISM IN SELECTED NATIONS**

<table>
<thead>
<tr>
<th>Country</th>
<th>Number in 10,000</th>
<th>Data Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>China</td>
<td>110</td>
<td>Peking Health Science Centre</td>
</tr>
<tr>
<td>India</td>
<td>200</td>
<td>Action for Autism India</td>
</tr>
<tr>
<td>USA</td>
<td>150</td>
<td>US Centers for Disease Control and Prevention and Autism Society of America</td>
</tr>
<tr>
<td>UK</td>
<td>65</td>
<td>National Autistic Society 2006</td>
</tr>
<tr>
<td>Mexico</td>
<td>15</td>
<td>Ministry of Health, Mexico</td>
</tr>
<tr>
<td>Philippines</td>
<td>50</td>
<td>Autism Society of Philippines</td>
</tr>
<tr>
<td>Thailand</td>
<td>18</td>
<td>Ministry of Mental Health, Thailand</td>
</tr>
</tbody>
</table>

(www.autism-society.org)

Studies that rely on administrative data for children who receive special education services have reported significant increases in prevalence from 1992 to 2001 (Newschaffer et al., 2005).

Since 1985, non-US studies have reported higher rates of autism, ranging from a prevalence of 7 – 10 per 10,000 children for autistic disorder and an
The estimated prevalence for autism spectrum disorders is 1.5 to 2.5 times higher (Fombonne 1999 and Gillberg et al., 1999).

The etiology of autism is still unclear but recent studies suggest that genetics plays a major role in conferring susceptibility. Autism is a polygenetic disorder with a heritability index of 0.90 (Karande 2006; Meyer et al., 2007; Paterson et al., 2007 and Mendelsohn and Schaefer 2008). Researchers suspect that there are a number of different genes that, when combined together increase the risk of getting autism. In families with one child with autism, the risk of having another child with autism is 3-8 per cent. A number of studies have found that first-degree relatives of children with autism also have an increased risk of autism spectrum disorders (Meyer et al., 2007; Mendelsohn and Schaefer 2008 and Caglayan 2010). Many studies have shown that parents from families with autistic members are more likely to have autistic children. It is also the case that many families with one autistic child are at increased risk of having another child with autism (Caglayan 2010 and http://autism.about.com).

Most plausible neurodevelopment theories of autism focus predominantly on genetic factors. However, studies of monozygotic twins indicate that less than 70 per cent of twin pairs are concordant for autism and approximately 90 per cent are concordant for a broader spectrum of related cognitive or social abnormalities (Paterson et al., 2007). Well known genetic disorders are linked with this disorder. Fragile X syndrome is found in an estimated 2.5 per cent of cases of autism (Bolton et al., 1997). A genetic disorder called ‘tuberous sclerosis’ is expected to cause 0.4 to 2.8 per cent of cases of autism (Dykens and Volkmar 1997).

Recent research suggests that more than ten genes contribute to the underlying genetic risk of developing autism (Meyer et al., 2007 and Mendelsohn et al., 2008). It has been documented that close relatives of children with autism, who themselves do not meet criteria of autism, can have autism-related symptoms, viz., milder social and communication deficits and stereotyped behaviours (Dalton et al., 2003 and Caglayan 2010). During the past two decades, family and twin studies have provided evidence for a significant genetic component in autism. The risk of autism in siblings of autistic probands is approximately 45 times greater than that in the general population (Lord et al., 2001).
Environmental factors and exposures may interact with genetic factors to cause an increased risk of autism. The California Department of Public Health found that women in the first eight weeks of pregnancy who live near farm fields sprayed with the organochlorine pesticides dicofol and endosulfan are several times more likely to give birth to children with autism. The association appeared to increase with dose and decrease with distance from field site to residence (Roberts et al., 2007).

D’Amelio et al., (2005) showed indirect evidence that prenatal exposure to organophosphate pesticides such as diazinon and chlorpyrifos may contribute to autism in genetically vulnerable children. Teratogens are environmental agents that cause birth defects. Some agents that are known to cause other birth defects have also been found to be related to autism risk (Szpir 2006). Busko (2008), suggested that exposure during pregnancy to pyrethrin, a common ingredient in antiflea and antitick pet shampoos can cause autism in the child.

Research indicates that many children with ASD have some type of immune system abnormality, have high amounts of yeast-induced compounds in their system and suffer from infections (Shaw 2002). Because these abnormalities are consistent with the effects of candidiasis, some researchers hypothesize that the two have a direct cause and effect relationship.

Pathogenic and opportunistic microbes can develop and grow into large colonies due to weaning off the breast capable of immune protection. Microbes such as Candida produce a host of toxic substances which go into the blood stream. The developing brain is particularly sensitive to these toxins. As a result, whatever skills the child has developed, while exclusively breast fed are gradually lost, there is no normal development of language, comprehension and behaviour (Gibson and Roberfroid 1995 and Vorobiev et al., 1998). Scientists in the United Kingdom studied gastrointestinal bacteria in 150 children with autism. They found a very high prevalence of the harmful bacteria Clostridia (Gibson and Roberfroid 1995).

Learning difficulties, poor coordination and even autism have been linked with ‘gut dysbiosis’, an imbalance of intestinal bacterial species. The original gut dysbiosis leads to a so called ‘leaky-gut’ where intestinal walls are damaged thus allowing toxins to pass through.
into the bloodstream, from where they may then penetrate the blood-brain barrier. Chief among these toxins are acetaldehyde, alcohol and opiate-like substances from undigested foods (Kiefer et al., 2004). A diet full of sugars, wheat, processed foods and soft drinks with colorants, flavourings and preservatives alters gut flora by feeding the pathogenic microbes and changes the whole body metabolism altering its pH towards acidic. This alters the brain chemistry and microbial flora in other body areas which predisposes children to eczema, asthma, ear infection and cold and has a direct damaging effect on the immune system (Srinivasan 2009 and http://www.dietarysupport.com). *Histotoxic clostridia* are well-known producers of toxins, including neurotoxins, which may contribute to gut dysfunction and have a systemic effect which could potentially be a contributory factor in the development of autism (www.foodsmatter.com).

The autistic children have at least one immune-related problem: thyroid disease, Crohn’s disease, rheumatoid arthritis, chronic fatigue syndrome, fibromyalgia or allergies (Seroussi 2002). Children of mothers who have autoimmune diseases such as type I diabetes, rheumatoid arthritis and celiac disease have up to three times greater risk for autism (Gardener et al., 2009).

Many of the parents swore that their child’s autistic behaviour began at 15 months, shortly after the child received the MMR vaccine (Seroussi 2002). Vaccine ingredients such as mercury and aluminium are related to neurological problems. During the late 1980’s and 1990’s the preservative thimersol, a compound that is half mercury, was routinely added to vaccines contributed to autism (Rimland 2000; Bernard et al., 2001 and DeLong 2011). Some believe that mercury in the vaccines (BCG, MMR and DPT) may result in autism spectrum disorder. Recent studies reveal that Hep-B vaccines (thimerosal containing vaccines) is one of the important causes for higher prevalence rate (Shaw 2002).

Using chelation, Lonsdale et al., (2002) had found elevated arsenic, mercury, cadmium and lead in the urine of children with autism. Woods (1996) supported that, precoproporphyrin is a biomarker for mercury toxicity; suggesting people with autism have higher levels of mercury than controls. Nataf et al., (2006) found that children with autism have significantly higher levels of atypical molecule, precoproporphyrin than neurotypical children. James et al., (2004) asserted that children with autism
tend to exhibit high levels of oxidative stress and poor methylation, the process by which the body detoxifies itself.

Although the mercury preservative used in some vaccines is known to be neurotoxic, the most recent research on this subject does not suggest a specific link between vaccines and autism (Halsey et al., 2000 and Parker et al., 2004). In 2009, the US Federal Vaccine Court rejected “speculative and unpersuasive” claims that vaccine caused autism (CNN 2011). Increased exposure to mercury through thimerosal containing vaccines is one of the important issues on hand. The Centers for Disease Control and Prevention, the Institute of Medicine of the National Academy of Sciences and the U.K National Health Service have all concluded that there is no evidence of a link between the MMR vaccine and autism (www.cdc.gov).

Waldman et al., (2006) explored the possibility of television as the trigger for autism. Since children watch more television during rain or snow, the authors suggest that increased television viewing combined with genetic predisposition leads to increased autism. Baron – Cohen (2006) observes that autistic children are often the offspring of two “highly systematizing” parents, people with occupations such as statistics.

Three parental characteristics and two obstetric conditions emerge as potential risk factors for autism viz., paternal age, maternal age, maternal immigration, growth restriction and new born hypoxia. However, low apgar score, fetal distress, cesarean delivery, maternal hypertension and bleeding during pregnancy could also be the cause (Kolevzon et al., 2007). Other possible causes include prenatal or intrapartum use of medications and parental preconception chemical exposures (Newschaffer et al., 2002 and Tendon, 2004).

A study by Campbell et al., (1996) on premature infants found that those who survived cerebellar haemorrhagic injury were significantly more likely to show symptoms of autism than controls without the injury. Prenatal rubella and influenza, LBW and breech delivery may result in autism. Autism has been reported to be associated with prenatal stress such as job loss and family discord, storms etc. Prenatal stress can disrupt brain development and produce behaviours resembling symptoms of autism (Campbell et al., 1996 and Kinney et al., 2008). Roman (2007) reported that thyroid problems that lead to thyroxin deficiency in the mother in weeks
8-12 of pregnancy has been postulated to produce changes in the fetal brain leading to autism.

It has been hypothesized that folic acid taken during pregnancy could play a role in causing autism by modulating gene expression through epigenetic mechanism (Muskei et al., 2006) and sustained exposure to ultrasound waves can also cause autism (Ang et al., 2006).

More than 30 per cent of autistic children have elevated levels of serotonin in their platelets (Mc Dougle et al., 2005). It has been postulated that at early stages of development, when the blood-brain barrier is not yet fully formed, the high levels of serotonin in the blood enter the brain of a developing foetus and cause loss of serotonin terminals. These results in damaged neurocircuitry which persists throughout subsequent development and eventually the symptoms of autism appear (Whitaker-Azmitia 2005).

There is some evidence that allergies to certain foods could contribute to autistic symptoms. Most people who hold to this theory feel that gluten and casein are the most significant culprits (Seroussi 2002). Food additives and sugars seem to have an underlying effect on children who suffer from autism (Strickland 2009). The inability to properly metabolise the proteins results in a peptide. If the peptides are left undigested, they can function as opioids (endorphin like substances) within a person’s system. Some, instead of passing out of the system through the urine, enter the blood stream and cross the blood-brain barrier. This process may cause serious neurological problem (Knivsberg et al., 1995 and Shattock et al., 2002). Endorphins are internally produced opioids that have effects similar to those of externally administered opiate drugs like morphine. One endorphin theory of autism asserts that, in essence, individuals with autism behave like addicts high on heroin (Bauman 1996).

Recent functional brain imaging studies indicate that autism may be caused by atypical functioning in the temporal lobes and an abnormal interaction between frontal and parietal brain areas (Boddaert and Ziebovicius 2002 and Schultz 2005). It has been postulated that early development failure in autism involving the amygdale has a cascading influence on the development of cortical areas, specifically the fusiform “face area” of the ventral temporal lobe. Development of face perception
and social cognitive skills are supported by the amygdale-fusiform system and deficits in this network are instrumental in causing autism (Tendon 2004 and Schultz 2005). Figure 1 presents the contributory factors to ASD.

(www.healthfullivingsf.com/autism/)

Underlying causes and contributors to ASD
Figure 1

B. Characteristics of an autistic child

Autism is a complex neurodevelopment disorder characterized by restricted, repetitive, stereotyped patterns of behaviour, interests and activities. The triad of impairments includes impairment in social communication, social interaction and imagination (Hanbury 2006). The impaired behaviours manifest along a wide spectrum and commence before 36 months of age. In a third of autistic children, loss of language and social skills occurs during the second year of life, usually between
15 and 21 months of age. Disruptive behaviours and learning difficulty is not uncommon (Singhi and Malhi 2001 and Karande 2006).

In addition to core symptoms, children with autism frequently have serious behavioural disturbances, such as self-injurious behaviour, aggression, hyperactivity and temper tantrums in response to routine environmental demands (Lahiri et al., 2006).

**Clinical features of autism**

- Spinning
- No speech
- Flapping hands
- Walking on tip toes
- Lack of eye contact
- Self injurious behaviour
- Lack of interest in toys
- Dislike of being touched
- Non-speech vocalizations
- Preoccupation with hands
- Lack of response
- Dislike of certain foods
- Repetitive behaviour
- Balancing e.g., standing on a fence
- Behaviour that is aggressive to others
- Lack of interaction with other children
- Extreme dislike to touching certain textures
- Desire to keep objects in a certain physical pattern
- Desire to follow set pattern of behaviour/interaction
- Treating other people as if they were inanimate objects
- Confusion between the pronouns “I” and “you”
- Echolalia: speech consisting of literally repeating something heard
- Either extremely passive behaviour or extremely nervous active behaviour
- When picked up offering no “help” (feels like lifting a sack of potatoes)
Some of the symptoms related to autism include abnormally accelerated rate of growth in head size between 6-14 mon of age which may serve as an early warning signal of risk for autism. Atypical brain development is present by at least 4-6 mon of age (Courchesne et al., 2003; Powell 2004 and Dawson et al., 2006). Head circumference studies in children and adults with autism have consistently identified a subset of approximately 20 per cent of persons with autism with macrocephaly and two postmortem studies have shown a high proportion of individuals with autism who have increased brain weight (Bailey et al., 1995 and Lainhart et al., 2006).

The onset of brain overgrowth coincided with the onset of the signs and symptoms of autism, indicating that the overgrowth was part of a pathologic process that disrupted the development of normal brain structure and function in autism (Minshew et al., 2007). It appears that brain development characterized by abnormal growth of neurons, excessive tissue in some areas, and too little tissue in other areas, affects several cognitive functions including those involved in emotional and social interaction (Bolton et al., 1997). Seventy five per cent of children with autism have associated mental retardation and seizures in 30 per cent of cases (Karande 2006).

Children with ASD might be very good at things like putting puzzles together or solving computer problems, but not very good at some things most people think are easy, like talking or making friends. Some autistic children are mute; others acquire language skills with some degree of success but still cannot communicate i.e., carry on a true conversation (Tager-Flusberg et al., 2009). As infants, autistic children often do not babble, gesture, or speak single words at the normal ages. When they do speak, they may use a flat, robot like tone (Filipek et al., 2000). Autistic individuals are relatively unable to understand the intentions of other person’s actions (Gallese 2007 and Zalla et al., 2011).

More specifically, one of the deficits in the social domain is the inability to share enjoyment, interests or achievements with other people through non verbal behaviours (Kenworthy et al., 2010).

Of the children with autism, eight per cent had epilepsy, five per cent had cerebral palsy, one per cent had vision impairment and one per cent had hearing
loss (Wong 1993 and Yeargin-Allsop *et al.*, 2003). Most (above 85-90%) autistic children are mentally retarded with below normal IQ. But about 10 per cent autistic children are extraordinarily intelligent, having the ability to memorize long lists of information, the ability to make lightning-fast mathematical calculations and artistic abilities (Heaton *et al.*, 2004). Autistics have enhanced perceptual processing (Dawson *et al.*, 2006). Autistic children have “Islets of competence” areas where the child has normal or even advanced competence. One of the most intriguing characteristics is the occasional child who shows savant performance- an exceptional ability in a highly specialized area of functioning. Typical examples include drawing skill, musical skill, arithmetic, calendar arithmetic, memory skills and perfect pitch (Volkmar *et al.*, 1994).

These children also suffer from multiple ear infections, acid reflux, and food intolerances. They have been very small for age and had constant diarrhea. Some battled not only thrush, but also constant, chronic congestion, eczema and food sensitivities (Lewis 2003; Savage 2003 and Vest 2003).

Children with autism also commonly have damaging oral habits such as bruxism, tongue thrusting, picking at the gingival, lip biting and pica. Bruxism, by definition is a nonfunctional, involuntary, forceful grinding or gnashing of teeth that affects 10 - 20 per cent of the population.

The red blood cells of children with autism have low levels of a fatty acid linked to cognitive function. The low fatty acid levels may trigger biochemical changes in the brain linked to autism. The levels of docosahexanoic acid and total omega-3 fatty acids are significantly lower in the red blood cells of autistic children than in normally developing children (Zimmer 2009).

Autistic children are known for their poor food repertoire, eating a very limited diet, difficult behaviour during meal times, having to be fed, not sitting to eat, eating only junk food and eating untidily (Dawson 1997). The strong preference kids with autism have for certain foods leads to nutritional deficiencies because their diets lack sufficient variety (Zimmer 2009).

Physical health profile of the autistic child strongly tends toward the following observations:
Gastrointestinal abnormality

- Malabsorption (Horvath et al., 1999)
  - Frequent reports of acholic stools, undigested fibers, positive Sudomonas
  - 85% of autistics meet criteria for malabsorption (Horvath et al., 1999)
- Maldigestion-elevated urinary peptides (Shattock 1990; Reichelt et al., 1994 and Sun and Cade 1999)
- Microbial overgrowth-fungal, bacterial and viral (Shaw et al., 1997 and Bolte 1998)
  - IgA elevations (Bull et al., 2003)
  - Pyrrole elevations (Wakefield et al., 1998 and Mc Ginnis 2003)
- Abnormal intestinal permeability (D'Eufemia 1995)
- G.I symptoms reported by parents - diarrhoea, constipation, gas, belching, probing, visibly undigested food

Compromised immunity

- Recurrent infections (Stern et al., 2005)
- Abnormal indices
  - T-cell deficiency (Cohly et al., 2005), reduced NK cell activity (Vojdani et al., 2008), low or absent IgA (Zimmerman 2000) and low C4B levels (Warren et al., 1991)
  - Skewed (“elevated”) viral titers

Detoxification weakness

- Phase II depression (Edelson and Contor 1998)
  - Low sulphation, low glutathione conjugation, low glucuronidation, and low glycine conjugation
- Sulphation deficit
- Peroxisomal malfunction (Kane et al., 1997)
- Higher blood lead levels in autism and documented response to EDTA chelation (Green 2006)
- Apparent temporal association between autism onset and lead exposure (Eppright et al., 1996)
Abnormal nutritional profile in children with autism

- Lower serum magnesium (Strambi 2006)
- Lower RBC magnesium
- Low activated B6 (P5P), higher serum copper in 42 per cent
- Low EGOT (functional B6) (Kotsanis 1996)
- B6 and magnesium therapeutic efficacy—multiple positive studies (Pfeiffer et.al., 1995)
- Low derivative omega-6 RBC membrane levels had GLA and DGLA below mean (Kane et.al., 1997)
- Low methionine levels not uncommon (Pangborn 1995)
- Below normal glutamine and high glutamate (Moreno-Fuenmayor et.al., 1996)
- Higher copper / zinc ratios (Isaacson et.al., 1996)
- Reduced sulphate conjugation and lower plasma sulphate (Waring 1997)
- B12 deficiency suggested by elevated urinary ethylmalonic acid (Wakefield et.al., 1998)
- Hypocalcinurics - improve with calcium supplementation lower hair calcium (Coleman 1994)

C. Food and dietary habits of children with autism

Anecdotally, it has long been noted that many children with autism have feeding difficulties and unusual eating patterns. Many of these youngsters have an extremely limited food repertoire, which is likely related to sensory regulatory difficulties, desire for sameness or other issues. Twenty per cent of parents believed health problems in their child affected their eating patterns, 67 per cent of respondents described their child as a picky eater, the most frequently reported problem behaviours were trying new foods (69%), taking medicine (62%), eating few foods (60%), mouthing objects (56%) and rituals surrounding eating (46%) (Williams et.al., 2000).

Eating disorders such as failure to thrive, rumination, pica, obesity and anorexia nervosa can affect children with autism spectrum disorders. Children experiencing these problems are at risk for serious health and growth problems that can lead to life threatening consequences (Kedesdy and Budd 1998).
Pica, the ingesting of non-nutritious substance, is a behaviour that can start at any point in life under various circumstances, if a child is persistently eating non-edible items such as paper, dirt or craft items and chewing on plaster or wood. There are multiple causes and treatments for pica. Nutritional deficiencies, sensory stimulation, lack of ability to discriminate non-edible items and relief of anxiety are all possible factors that can lead to pica. Rumination is the persistent regurgitation, re-chewing, re-swallowing or occasionally vomiting of previously eaten food and is a second behavioural problem of eating that can have serious health consequences (Wheeler 2004).

Selective eating can have significant development and health consequences. Some children with autism spectrum disorders will eat mostly foods that fit into only one of these four categories; sweet, sour, bitter or salty. A child may choose to eat mostly foods which are salty, the feel or touch of the food, the temperature and the texture of the food. It is common for children with autism spectrum disorders to have a strong preference for one particular texture of foods such as crunchy or smooth. Some children with an autism spectrum disorder are much more affected by the smell of the food. The smell of foods that are not familiar and comfortable may affect their ability to eat. The way food “look” is another issue. Some children only eat foods of one colour such as white or orange foods. Many children on the autism spectrum will only eat something if it is presented each time in the same plate. Some extremely selective kids will want “perfect” uniformity of their food and will refuse to eat if they detect even the slightest change (Dawson 1997 and Kedesdy and Budd 1998). In many situations mealtimes have become a negative experience where children often feel forced and may resist more and more, until it becomes virtually impossible to feed them anything (Dawson 1997).

There is much overlap between hyperactivity and autism. So for autistic children who show signs of hyperactivity, improving blood sugar balance is a must. When a child is regularly snacking on refined carbohydrates, sweets, chocolate, fizzy drinks, juices and little or no fibre to slow the glucose absorption, the levels of glucose in their blood will seesaw continually and trigger wild fluctuations in their levels of activity, concentration, focus and behaviour. These of course will not help the child’s brain function (Dawson 1997).
There have always been attempts to connect abnormal behaviour directly to a person’s diet. Such behaviour includes hyperactivity, autism, sleep disorders and mood swing disturbances in children. Limit trigger foods in the diet, some trigger foods to eliminate would be caffeine, sodas, refined and processed foods, all junk foods and fast foods, artificial colours and preservatives.

There are many anecdotal reports of dramatic improvements in children with autism from parents who removed casein and gluten from their diets. It has observed that as levels of peptides in the blood decrease, the symptoms of autism decrease. If the peptides are reduced to normal range, there would be typically dramatic improvements (Cade, http://www.panix.com).

Gluten – free diet is an eating plan in which gluten is eliminated from the diet. Foods and drinks containing wheat, barley, rye, oats or anything made from these grains are avoided. Gluten – free diet is often used for children with autism in combination with casein – free diet. The casein – free diet calls for the elimination of milk protein (Whitely et.al., 1999).

Gluten is broken down in the intestines into several by – products including one called gluteomorphine. These by–products are much more common in the urine of children with autism than in children without autism. Scientists have concluded that they are leaking from the intestines into the blood of these children. If gluteomorphines are being absorbed into the blood circulation in children, it could affect their behaviour, reduce their desire for social interaction, block pain messages, and increase confusion. Recent evidence of a genetic mutation common among children with autism has been traced to a gene involved in gastrointestinal function (Le Breton and Kessick 2001; Lewis 2005 and Crosthwaite 2006).

Exorphin peptides are derived from incompletely digested proteins, particularly food containing gluten and casein. One of these, called IgA and derived from gluten in wheat, has been detected in 80 per cent of autistic subjects. So the first problem is the poor digestion of proteins. A lack of sufficient zinc and vitamin B6 could contribute to this, as both are essential for proper stomach acid production and protein digestion, yet are often deficient in autistic children with pyroluria (Whitely et.al., 1999).
A recent report showed that the protein and nutrient intakes of children with autism on standard diets was adequate while there was a trend towards lower calcium and copper intake in children on elimination diets. It’s not completely clear that foods do worsen autism, although there are many theories about how this could occur. It has been suggested that autism could result from a loss of regulation of the immune system, causing an increase in inflammatory – causing chemical signals from white blood cells. It is felt that these chemicals (cytokines) may be responsible for the neurological abnormalities seen in children with autism. The cytokines from the autistic children are much higher than those from normal children after being exposed to gluten or casein (www.foodforthebrain.org).

Some autistic children have an enzymatic defect that removes essential fats from brain cell membranes more quickly than it should. This means that an autistic child is likely to need a higher intake of essential fats than the average. And it has been found that supplementation with eicosapentanoic acid, which can slow the activity of the defective enzyme, has clinically improved behaviour, mood, imagination, spontaneous speech, sleep patterns and focus of autistic children (Bell et.al., 2000 and Bell 2001).

The autistic children had significantly greater intake of all nutrients with the exception of vitamins A and C and fat. Parents of autistic children reported a more positive belief in the relationship between diet and behaviour and a more positive attitude about the importance of nutrition (Cermak et.al., 2010). Diet on its own is a very powerful tool in helping an autistic child. But to normalize the gut flora a strong probiotic is needed.

D. Diagnostic and treatment methods

Studies have shown that autism can be diagnosed between 2 and 3 yrs of age (Stone et.al., 1999 and Charman and Baird 2002). More severely affected children usually present in the preschool years with language delay (Rapin 1997). Early identification of autism is important because early intervention services may be more effective in children with autism than in children with other developmental disabilities (Lipkin and Schertz 2006).

The criteria for diagnosing autism are entirely clinical. Semi-structured interview schedules and rating scales have been developed to assist the clinician in
increasing the objectivity and reliability of assessment (Singhi and Malhi 2001). Table III presents the screening tools used for autism.

**TABLE III**

**AUTISM SPECIFIC SCREENING TOOLS**

<table>
<thead>
<tr>
<th>Screening tool</th>
<th>Characteristics</th>
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<tbody>
<tr>
<td>Checklist for Autism in Toddlers (CHAT)</td>
<td>For use in children aged 18 mon; 14 items; 9 derived from parent history and 5 from direct observation. Specificity 98%, sensitivity 38%. Does not discriminate children with autism and children with mental retardation.</td>
</tr>
<tr>
<td>Social Communication Questionnaire (formerly called Autism Screening Questionnaire)</td>
<td>For use in children aged &gt; 4 yrs.</td>
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</table>

(Siegel 2004 and Stone et.al., 2004)

Since there are no definitive diagnostic tests, a clinical diagnosis by an expert, based on Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV, TR criteria, remains the gold standard for diagnosis (Lord et.al., 2000; Volkmar et.al., 2004 and Simpson 2005). Table IV presents the diagnostic tools available for autism.

**TABLE IV**

**AUTISM – SPECIFIC DIAGNOSTIC TOOLS**

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Characteristics</th>
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<tbody>
<tr>
<td>Autism Diagnostic Interview - Revised (ADI-R)</td>
<td>Semi structured interview that is reliable, valid and differentiates autism from other developmental disorders; takes 11/2 hrs to administer limiting clinical use.</td>
</tr>
<tr>
<td>Autism Diagnostic Observation Schedule- Generic (ADOS-G)</td>
<td>Reliable, valid, direct assessment that helps to differentiate ASDs from other developmental disorders; requires specific training; takes 30 min to administer making it practical for clinical use.</td>
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<tr>
<td>Gilliam Autism Rating Scale</td>
<td>Checklist that may be used by parents and teachers and other professionals to quantify autism symptoms.</td>
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</tbody>
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Table continued
According to Campbell *et al.*, (1996) Childhood Autism Rating Scale (CARS), Autism Behaviour Checklist (ABC) and Autism Diagnostic Observation Schedule (ADOS) are also used to diagnose autism.

Early detection and early intervention is crucially important in the life of the child with autism. It is universally acknowledged that autism can be detected as early as 18 months. Early and appropriate intervention can ensure that many children with autism can be mainstreamed (Poon *et al.*, 2010).

Various types of therapies are available, including applied behaviour analysis, auditory integration training, dietary interventions, discrete trial teaching, medications, music therapy, occupational therapy, physical therapy, sensory integration, speech/ language therapy and vision therapy (www.aarogya.com).

Autism has appropriately been called “an extreme challenge to integrative medicine”. At the same time, integrative medicine and nutritional approaches offer tremendous promise for the future of this heartbreaking group of disorders (www.guelphnaturopath.com).

The classes of medication used are anticonvulsant, antidepressant, antihistamines, antihypertensives, antipsychotics, anxiolytics, β-adrenergic antagonists (β - blockers), mood stabilizers, narcotic antagonists, sedatives and stimulants (Sweeney *et al.*, 1998 and Tsai 1999). Short-term use of drugs for target symptoms like aggression, hyperactivity, stereotypic behaviour, insomnia, and short attention span is usually warranted. Long term use has to be carefully evaluated because all groups of drugs have side effects like extra pyramidal symptoms, seizures, tardive syndromes and metabolic complications (Shrinath 2006).

Risperidone, another atypical anti- psychotic medication is found to reduce aggression, over activity and stereotypes across all ages (Nicolson *et al.*, 1998). Autism in itself is not an indication for using psychotropic medications as drugs

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Characteristics</th>
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<tbody>
<tr>
<td>Childhood Autism Rating Scale</td>
<td>Structured interview and observational tool designed to be used by experienced clinicians or other professionals to identify symptoms consistent with ASD.</td>
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(Lord *et al.*, 2000)
cannot be a substitute for behavioural and educational interventions. These medications are used to reduce interfering behaviours to make the child more amenable to interventions. Psychotropic medications should never be used in isolation but used only in conjunction with behavioural, educational and habitative therapies (Hollander et.al., 2003).

There is currently no known “cure” for autism. The only treatment that has been shown to make a positive impact in ameliorating the core behavioural deficits in autistic children is early intensive behavioural and educational intervention therapy (National Research Council 2001).

Treatments of the digestive system abnormalities have led to varying degrees of improvements in the core symptoms of ASD, including behaviour, communication and social skills. The main treatments for this abnormal inflammatory bowel disease include diet, treatment of gut yeast and supplemental enzymes (Wheeler 2004).

While nutritional therapy and dietary restrictions may not cure autism or its core symptoms, they can be a helpful complementary treatment. Pfeiffer et.al., (1995) suggest that B6 - magnesium treatment may be a promising adjunct in the treatment of autism, as it may influence reactions affecting several neurotransmitter systems. Megson (2000) presented two case studies in which eye contact and behaviour of children with ASD improved after being treated with cod liver oil supplements, which are rich in vitamin A.

Over the last two decades there has been mounting evidence suggesting a link between zinc deficiency and clinical depression (Levenson 2006). Research suggests that tetrahydrobiopterin (BH4), a folate derivative and an essential co-factor in the synthesis of the neurotransmitters dopamine, epinephrine and serotonin, may play a role in autism. Iron deficiency has also been implicated in autism. A recent study revealed that 77 per cent of a group of children with ASD were deficient in iron. Iron supplementation significantly improved sleep disturbances in these children (www.autism.com).

Deficiencies in essential fats are common in people with autism. Some autistic children have an enzymatic defect that removes essential fats from brain cell membranes more quickly than it should. This means that an autistic child is likely to
need a higher intake of essential fats than the average. And it has been found that supplementing eicosapentanoic acid, which can slow the activity of the defective enzyme, has clinically improved behaviour, mood, imagination, spontaneous speech, sleep patterns and focus of autistic children (Bell et.al., 2000; Bell 2001 and Vancassel et.al., 2001).

Flax seeds (*Linum usitassimum*) are rich in alphanlinolenic acid, an omega 3 fat that is a precursor to eicosapentanoic acid. Several studies have established that EFA is deficient in the autistic children (www.whfoods.com). Apart from this flax seeds are a very good source of dietary fibre and manganese. They are also a good source of folate and vitamin B6 and minerals such as magnesium, phosphorus and copper. In addition, flax seeds are concentrated in lignin phytonutreints, that possess anti-tumour, antibacterial, antifungal and antiviral properties.

Many studies have shown that dimethyl glycine (DMG) enhances the effectiveness of the immune system, improves the physical and athletic performance in humans (www.autism.com). DMG increased antibody production by more than 400 per cent. It also acts as a detoxifier within the human body, all of which are beneficial to the autistic children (Reap and Lawson 1990). Brown rice is a rich source of DMG. Brown rice often referred to as whole rice or cargo rice, is the whole grain with only its inedible outer hull removed. Brown rice retains its nutrient rich bran and germ (www.whfoods.com).

Supplementing the autistic child with DMG led to improvement in the child’s behaviour, produced better eye contact, increased frustration tolerance, improved the child’s speech and created more interest in speaking (www.autism.com). Among the effective natural treatments for yeast infection, use of probiotics is considered as an effective one. The strategy behind this natural *Candida* cure is a simple one- flood the intestines with friendly, beneficial bacteria to thrive (www.autism-nutrition.com).

Dunne et.al., (1999) report that when an efficient probiotic is introduced to the gut, over time, it clears out the “bad” microbes together with old putrefaction products
and reestablishes the normal gut flora. Probiotic foods have diverse health benefits and are claimed to be increasingly used in neutraceuticals and functional foods. Probiotic modulation of host immunity is a very promising area for research (Gill et al., 2001). Human trials suggest that certain lactic acid bacteria strains exert a positive effect on the immune system, through an induced increase in certain immunoglobulins such as serum IgA, increased non-specific phagocytose activity or increased formation of certain non-proinflammatory cytokines (http://www.bfr.bund.de/cm/245/probiotika_en.pdf).