



Issue #2

Theoretical Perspectives on the Causes of Learning Disabilities

Introduction

Understandably, one of the first questions parents ask when they learn their child has a learning disability is Why? What went wrong? Mental health professionals stress that since no one knows what causes learning disabilities, it doesn't help parents to look backward to search for possible reasons. There are too many possibilities to pin down the cause of the disability with certainty. It is far more important for the family to move forward in finding ways to get the right help.

Scientists, however, do need to study causes in an effort to identify ways to prevent learning disabilities. Once, scientists thought that all learning disabilities were caused by a single neurological problem. But research has helped us see that the causes are more diverse and complex. New evidence seems to show that most learning disabilities do not stem from a single, specific area of the brain, but from difficulties in bringing together information from various brain regions. Therefore, causes of learning disabilities may be as diverse as the types of learning disabilities.

Clearly, the causes of learning disabilities are a nebulous area of research. There is lack of explicit cause and effect relationships, and studies have not been able to indicate any single factor directly responsible for causing learning disabilities. The condition is better understood by considering associated factors rather than cause and effect relationships. Research and practice in the field of learning disabilities have primarily focused on diagnosis and remedial education. To this end, it is necessary to explore the root cause of learning disabilities to be able to prevent them.

The field of learning disabilities has been plagued, almost since its inception, by fads and unproven theories. Little is actually known about the causes of learning disabilities, but we can presume that the students who exhibit them are as diverse as the indicators of the condition (Deutsch-Smith, 2004).

What is known about the etiology (cause) of learning disabilities is that abnormal brain structure and function play a significant role. Different abnormalities cause different types of learning disabilities. These neurological abnormalities can result from a variety of sources.

Genetic Links

The genetic basis for learning disabilities has been researched through twin studies, sibling analysis, and family pedigree analysis (Raskind, 2001). Twin studies in the field of LD have indicated that if one twin has a reading disability, the probability of the other twin also having a reading disability is 68 percent for identical twins (monozygotic) and 40 percent for fraternal twins (dizygotic). The research evidence generally supports the hypothesis that certain types of learning problems, including reading disabilities, are more common among identical twins than fraternal twins (DeFries, Gills, & Wadsworth, 1993). Similar findings are also observed in twins with speech and language disorders (Lewis & Thompson, 1992).

Familial transmission of learning disabilities has shown that if there is a family history (parents, siblings, and extended family) of reading disabilities, the probability of having a reading disability is significantly increased (Culbertson, 1998). Several modes of transmission have been investigated. Although there are, as yet, no definitive conclusions, a possible linkage to chromosomes 6 and 15 has been identified.

The fact that learning disabilities tend to run in families indicates that there may be a genetic link (Alarçon-Cazares, 1998). Researchers have found that about 35 to 45 percent of the first-degree relatives (parents and siblings) of persons with reading disabilities also have reading disabilities (Pennington, 1990; cited in Hallahan & Kauffman, 2003). Children who lack some of the skills needed for reading, such as hearing the separate sounds of words, are likely to have a parent with a related problem. However, a parent's learning disability may take a slightly different form in the child. A parent who has a writing disorder may have a child with an expressive language disorder. For this reason, it seems unlikely that specific LD are inherited directly. Possibly, what is inherited is a subtle brain dysfunction that can in turn lead to a learning disability. Similar evidence has been found in the area of speech and language disorders (Castles, Datta, Gayan, & Olson, 1999) and spelling disabilities (Schulte-Korne, Deimel, Muller, Gutenbrunner, & Remschmidt, 1996).

Note that there may be an alternative explanation for why learning disabilities might seem to run in families. Note that there may be an alternative explanation for why learning disabilities might seem to run in families. Family patterns do not clearly prove that heredity is a contributing factor. Some learning difficulties may actually stem from the family environment. For example, parents who have expressive language disorders might talk less to their children or the language they use may be distorted. In such cases, the child lacks a good model for acquiring language and, therefore, may seem to have learning disabilities.

Abnormalities in Fetal Brain Development

Throughout pregnancy, the fetal brain develops from a few all-purpose cells into a complex organ made of billions of specialized, interconnected nerve cells called neurons. During this amazing evolution, things can go wrong that may alter how the neurons form or interconnect.

In the early stages of pregnancy, the brain stem forms. It controls basic life functions such as breathing and digestion. Later, a deep ridge divides the cerebrum—the thinking part of the brain—into two halves, a right and left hemisphere. Finally, the areas involved with processing sight, sound, and other senses develop, as well as the areas associated with attention, thinking, and emotion.

As new cells form, they move into place to create various brain structures. Nerve cells rapidly grow to form networks with other parts of the brain. The networks enable information to be shared among various regions of the brain. Throughout pregnancy, brain development is vulnerable to disruptions. If the disruption occurs early, the fetus may die, or the infant may be born with widespread disabilities and possibly mental retardation. If the disruption occurs later, when the cells are becoming specialized and moving into place, the result may be errors in the cell makeup, location, or connections. Some scientists believe that these errors may later show up as learning disabilities.

Maturation Delay

Another theory to explain learning disabilities suggests that they occur because there is maturational delay (rather than a permanent dysfunction) within the neurological system (Samango-Sprouse, 1999). Some children develop and mature at a slower rate than others in the same age group. As a result, they may not be able to do the expected school work. This kind of learning disability is called maturational lag. Here are some typical symptoms of maturational delay:

- Slow maturation of language skills, especially those of reading
- Delayed development of motor skills
- Uneven performance patterns on measures of intellectual development
- Visual–motor problems
- Incomplete or mixed dominance
- Right–left confusion
- Social immaturity
- Tendency for members within a family to show similar symptoms.

Brain Structure and Learning Disabilities

In comparing people with and without learning disabilities, scientists have observed certain differences in the structure and functioning of the brain (Richards, 2001). For example, new research indicates that there may be variations in the brain structure called the planum temporale, a language-related area found in both sides of the brain. In people with dyslexia, the two structures were found to be equal in size. In people without dyslexia, however, the left planum temporale was noticeably larger. Some scientists believe reading problems may be related to such differences (Leonard, 2001; Raskind, 2001).

It is now widely accepted that the brain structure or function of a person with LD is different from that of a person who does not have learning disabilities. There is a view that the language area in the brain of an individual is well developed in the left hemisphere and is tiny, and hence, dysfunctional in the right hemisphere. So, in the normal course of information processing, the nerve impulses set up in the visual cortices travel for interpretation to the left hemisphere of the

brain. In the brains of individuals with LD, the language areas are well developed in both the hemispheres. In this case, the nerve impulses travel to both hemispheres simultaneously. Thus, the corpus callosum becomes “jammed” with nerve impulses as the two language areas refer the messages they receive from the visual cortices back and forth for comparison and analysis. This confusion caused by criss-crossing of the nerve impulses may be why a child with learning disabilities often reads b as a d and vice versa. With more research, scientists hope to learn precisely how differences in the structures and processes of the brain contribute to learning disabilities, and how these differences might be treated or prevented.

Measuring the Brain and Brain Function

A variety of methods are now available to measure the physical structure as well as the function of the brain. Neuroanatomical techniques include autopsy studies; neuro-imaging techniques include CT scan, MRI, PET, rCBF, and SPECT; electrophysiological measures include EEG, ERP, and AEP; and neuropsychological assessments evaluate brain/behavior relationships.

A number of studies of brain structure and function have been carried out on individuals with learning disabilities (Silver, 1999). One method of looking at structural differences in the brain is through postmortem or autopsy studies. Postmortem findings have indicated that the normal brain has asymmetries: one side of the brain is not a perfect mirror image of the other. These asymmetries are expected and considered normal (just as it is quite ordinary or typical for one foot to be longer than the other).

Important research efforts have focused on reading disabilities, since they represent the most common and frequently identified type of learning disability. Studies have shown that people with reading disabilities have symmetry in brain structures where there should be asymmetry. For example, in people without LD, the temporal lobe (planum temporale area) in the left hemisphere is often larger than the same area in the right hemisphere. However, in subjects with LD, this area in the left hemisphere has been found to be the same size as in the right hemisphere.

Another technique for studying the brain is the CT scan (computed tomography (roentgen-ray)). With this technique, a beam of X rays is aimed through the brain, identifying bone, grey matter, and fluid. A computer then reconstructs an image of each slice or brain section, allowing abnormalities in structure to be detected. CT scans of the occipital lobe for example, have shown asymmetry of the occipital pole in subjects without LD and symmetry in subjects with LD.

Magnetic resonance imaging (MRI) is a technique that involves detecting the electromagnetic energy of brain protons and constructing an image by superimposing magnetic fields. Recent advances in MRI technology have enabled researchers to discover that specific regions of the brains of some individuals with reading and language disabilities show activation patterns during phonological processing tasks that are different from the patterns found in the brains of persons without disabilities (Simos et al., 2000). MRI research has shown that individuals without LD showed leftward asymmetry in the angular gyrus of the parietal lobe, whereas people with LD did not show the expected asymmetry.

It has been demonstrated through autopsy, CT scan, and MRI studies that there are structural differences in the brains of subjects with LD in comparison to subjects without LD. It has also

been demonstrated that in the subjects with LD, there are differences in brain function—that is, how the brain works. Functional neuroimaging techniques, including PET (positron emission tomography), rCBF (regional cerebral blood flow), fMRI (functional magnetic resonance imaging), and SPECT (single photon emission computed tomography), are used to measure brain activity while subjects are engaged in a task such as reading. An fMRI is a noninvasive method that measures blood flow, while PET and SPECT methods involve the injection of radioactive materials. SPECT scan results have indicated that subjects with LD show under-functioning in the occipital lobe while reading, in comparison to subjects without LD.

Electroencephalograms (EEGs), event-related potentials (ERPs), and averaged evoked potentials (AEPs) record electrical activity of the brain through electrodes. Research has shown that subjects with dyslexia showed less electrical activity in the parietal lobe, in comparison to subjects without dyslexia.

Neuropsychological assessments include a variety of tests of cognitive/intellectual, language, visual-perceptual, academic, motor, sensory, and emotional/behavioral abilities and functions. A profile of strengths and weaknesses is then correlated with known brain functions. The neuropsychological research has indicated significant findings as well. Deficiencies in language/verbal learning, reading, written language, verbal reasoning, verbal memory, arithmetic computation, and processing speed have been associated with left hemispheric dysfunction. Deficiencies in spatial function, nonverbal reasoning, nonverbal cues, social skills, and social/emotional information have been associated with right hemispheric dysfunction. Phonological processing deficits have been identified as a primary difficulty in persons with language and reading disabilities, and structural and functional abnormalities in the medial geniculate nuclei have been associated with these findings.

It is important to emphasize that individuals with LD can learn, but the process may be inefficient as a result of the specific differences in brain structure and function. Inefficiency refers to either low accuracy or low speed in learning or performing a task and is quite distinct from inability or incapacity. Information can be processed, but at a slower rate and/or by different methods as compared to individuals without learning disabilities. The educational process, learning strategies, compensatory techniques, and remedial intervention can significantly affect the learning process. Therefore, effective and efficient learning and teaching methods are needed to specifically meet the needs of individuals with learning disabilities.

Biological Basis for Reading Disabilities

Recent research has found convincing evidence that dyslexia is caused by a functional disruption in the brain (Gilger, 2001). The research, led by Dr. Sally Shaywitz, a Professor of Pediatrics at the Yale University School of Medicine, was published in the March 3, 1998 Proceedings of the National Academy of Sciences (NAS). These findings represent a critical new piece of evidence that builds on the already solid research in the area of reading disability.

The researchers used functional magnetic resonance imaging (fMRI), which enables researchers to look into the brain as it is working. The research used fMRI to image the brains of 32 adults with dyslexia and 29 adults without dyslexia while they attempted to perform a progressively complex series of reading tasks. The tasks included letter recognition, rhyming letters and words,

and finally, categorizing words. The findings showed that brain activation patterns of readers with dyslexia were significantly different from those of readers without dyslexia.

Reading requires an ability to recognize that spoken words can be segmented into smaller units of sound (phonological awareness) and that the letters in the printed word represent these sounds. Individuals with dyslexia do not recognize these smaller sound units and have difficulty mapping alphabetic characters onto the spoken word.

The results of the study indicated that readers without dyslexia systematically increased their brain activation as the difficulty of mapping print into phonological structures increased. The readers with dyslexia did not systematically increase their brain activity. The demonstrated disruption in brain function among readers with dyslexia occurred in a part of the brain involving traditional visual and language regions. During reading, people with dyslexia showed a pattern of under activation in a large posterior brain region, an area which connects the visual areas with the language areas.

These findings reconcile seemingly contradictory evidence from previous imaging studies which were not able to map out the full extent of the disruption. Of particular importance was the finding that the angular gyrus, a brain region considered pivotal in carrying out cross-modal (e.g., vision and language) associations necessary for reading, is involved. The current findings of under activation in the angular gyrus of readers with dyslexia coincide with earlier studies of people who lost the ability to read due to brain damage centered in that same area of the brain. According to the authors, it is no coincidence that both the acquired and developmental disorders affecting reading have in common a disruption within the neural systems serving to link the visual representation of the letters to the phonological structures they represent.

These findings have important implications for the large numbers of intelligent men, women and children with dyslexia. . . . If you have a broken arm, you can hold up an x-ray as evidence. Up to now, individuals with dyslexia were often doubted and there was little concrete evidence they could show to support the neurobiologic nature of their reading difficulty. These brain activation patterns, by revealing a functional disruption in those neural systems responsible for reading, now provide neurobiologic evidence for what, up to now, has been a hidden disability. (Gilger, 2001, pp. 490–491)

Biochemical Abnormalities

Chemicals play an important role in brain activity, controlling and releasing electrical impulses between neurons. The absence or excessive presence of biochemical substances can cause abnormal electrical activity in the brain.

Endocrine Problems

The endocrine glands, located in the various parts of the body, secrete hormones or strong chemical substances directly into the blood stream. Hormones influence the functions of tissues and organs and thus help to determine behavior. There seems to be some relationship between these chemicals and hyperactivity and learning disorders.

Thyroxine Imbalance - Thyroxine, the hormone secreted by the thyroid gland, controls the basal metabolic rate of the body, that is, the rate of consumption of oxygen and energy output. A low level of thyroxine can result in poor memory, low I.Q. and a lack of energy. Excessive thyroxine can result in nervous hyperactivity, irritability, and difficulty in concentration. A minimal lowering of blood sugar levels can also result in word-finding problems and increased spelling errors.

Thyroid Dysfunction - Children born without a functioning thyroid system may be at risk for learning disabilities. Babies who are screened at birth for congenital hypothyroid syndrome receive a prompt and lifelong program of thyroid hormone therapy, which prevents serious developmental delays. However, longitudinal follow-up reveals that, though IQs may be in the normal range, there are usually profiles of deficits similar to learning disabilities. Research is underway on relationships between optimal levels of maternal thyroid during the entire period of gestation and optimal fetal development.

Nutritional Problems

Poor Nutrition. There seems to be a link between nutritional deprivation (either the child's or the mother's when she was pregnant) and poor biochemical functioning in the brain. A poor diet and severe malnutrition can reduce the child's ability to learn by damaging intersensory abilities and delaying development.

Recent studies and clinical trials conducted at Purdue University in the United States and Surrey and Oxford in the United Kingdom indicate that some learning disabilities, such as dyslexia and dyspraxia, may have a nutritional basis. And, as previously stated, researchers such as Feingold believe that some learning disabilities might be caused by allergies to certain foods, food additives, and dyes, or by environmental allergies.

Dietary Sensitivities - In the early 1970s, Feingold (1975) proposed that much of the hyperactivity involved with LD could be attributed to food additives. This untested idea, based on testimonials, gained wide public acceptance. He believed that removing synthetic colors and flavors, as well as certain fruits and vegetables containing salicylates (natural pesticides), from the diet could treat behavioral disturbances. Feingold believed that as many as 10 to 25% of all children may be sensitive to salicylates. The success of the diet may depend on the degree of a person's sensitivity to salicylates and food additives, and the amount of additives present in foods. Feingold also speculated that certain foods, such as sugar, caused behavior changes.

Over the years, dozens of scientists put Dr. Feingold's theories to the test, but the evidence they gathered failed to support the theory that additives, sugar, or other substances in food cause or contribute to hyperactivity. Most professionals in learning disabilities and the scientific community give little credence to biochemical imbalance as a significant cause of children's learning problems (Heward, 2003; Kavale & Forness, 1983).

Vitamin Deficiency - Another popular approach of the 1970s was megavitamin therapy. The chief advocate was Alan Cott, who theorized (1972) that learning disabilities can be caused by the inability of a person's blood to synthesize a normal amount of vitamins. In an effort to treat LD, large daily doses of certain vitamins were recommended to counteract the suspected vitamin

deficiency. Again, scientific research (Arnold, Christopher, Huestis, & Smeltzer, 1978) has failed to substantiate the benefit of this treatment (Gargiulo, 2004).

Complications of Pregnancy and Birth

Other possible causes of learning disabilities involve complications during pregnancy. Damage may be inflicted on the neurological system at birth by conditions such as abnormal fetal positioning during delivery, anoxia (a lack of oxygen), or chemicals in the blood. When a baby's brain is given certain kinds of chemicals or does not get enough blood or oxygen, permanent brain damage can occur. Many students with learning disabilities have had some sort of trauma either before or during their birth.

In some cases, the mother's immune system reacts to the fetus and attacks it as if it were an infection. This type of disruption seems to cause newly formed brain cells to settle in the wrong part of the brain. Or during delivery, the umbilical cord may become twisted and temporarily cut off oxygen to the fetus. This, too, can impair brain functions and lead to learning disabilities.

Infants born prematurely and with low birth weights have increased in numbers over the last 20 years; however, in that same period, infant and neonatal mortality rates have dramatically improved. Being born too small or too soon entails high risk of serious morbidity contributing to long-term neurologic impairment. Premature infants have an increased risk for cerebral palsy, mental retardation, sensory impairment, developmental delays, and learning and school problems. Nutrition plays a key role in the prevention of prematurity and in neonatal care during hospitalization and in the follow-up period.

Low birth weight babies are at risk for learning disabilities. According to some studies, children whose birth weight was less than five pounds lagged behind their peers academically and displayed other subtle behavioral characteristics that undermined their efforts at school. During their preschool years, many of these children exhibited poor motor skills and neurological immaturity.

Prenatal Exposure to Harmful Substances

Drugs prescribed or otherwise, taken by the mother, pass directly to the fetus - Research shows that use of cigarettes, alcohol, or other drugs during pregnancy may have damaging effects on the unborn child (Codina, Yin, Katims, & Zapata, 1998). Scientists have found that mothers who smoke during pregnancy may be more likely to bear smaller babies. This is a concern because small newborns, usually those weighing less than five pounds, tend to be at a risk for a variety of problems, including learning disabilities.

Cocaine - Cocaine, especially in its smokable form, known as crack—seems to affect the normal development of brain receptors that help transmit incoming signals from our skin, eyes, and ears, and help regulate our physical response to the environment. Because children with certain learning disabilities have difficulty understanding speech sounds or letters, some researchers believe that LD may be related to faulty receptors (Murphy-Brennan & Oei, 1999).

Marijuana. The main psychoactive ingredient in marijuana, THC, crosses the placenta and has the potential for harming pregnancy outcome. Some recent epidemiological studies suggest that maternal marijuana use during pregnancy may result in perinatal hypoxia (low oxygen to the

baby), premature labor, low birth weight, and physical and behavior anomalies in the offspring. However, marijuana use has been difficult to measure in these reports, since it is an illegal drug, and therefore prone to under-reporting, and many of the women studied also used other drugs, raising the possibility of drug interactions with marijuana.

Six-year-old children are more likely to show signs of Attention-Deficit Hyperactivity Disorder if their mothers smoked six or more marijuana cigarettes (joints) per week. This was the conclusion after testing 126 children at the Department of Psychology, Carleton University, Canada. Fourteen of the children had mothers who admitted smoking between one and six joints per week and 19 had mothers who admitted smoking at least six marijuana joints per week during pregnancy.

The data pertaining to maternal use of marijuana, are suggestive of an association between that drug and particular aspects of attentional behavior—possibly sustained attention. Cognitive psychologists have frequently divided the attentional process into three sub-systems that perform different but interrelated functions. These include orienting toward sensory events, detecting signals to be focused on—including information stored in memory, and maintaining a vigilant state. The present findings suggest that prenatal exposure to cigarettes and marijuana may be associated differentially with subsystems within the attentional process. . . . In addition, Discriminant Function Analysis revealed a dose–response relationship between prenatal marijuana use and a higher rating by the mothers on an impulsive/hyperactive scale. (Fried & Watkinson, 1992, p. 1)

Prescription Heart Medication - Recent research suggests that warnings should be given to pregnant women taking the heart arrhythmia drug Amiodarone (AMD). Researchers at the Department of Medicine, Mount Sinai Hospital (1999), Toronto, Canada have found that when women took this drug during pregnancy there was an observed increase in language disorders for their children who were exposed to the drug during pregnancy. In the research project, the offspring of twelve mothers were followed after giving birth while taking the drug.

Early speech delay and difficulties with written language and arithmetic are part of the Nonverbal Learning Disability Syndrome. An association between AMD exposure and such a syndrome is plausible. Firstly, language skills may be more sensitive to neurotoxic damage than measure of global cognitive function. Secondly, another antenatal exposure, congenital hypothyroidism, has also been associated with this syndrome. Thirdly, AMD may have a direct neurotoxic effect on the developing fetal brain. (p. 5)

Alcohol - Alcohol also may be dangerous to the fetus' developing brain. It appears that alcohol may distort the developing neurons. Heavy alcohol use during pregnancy has been linked to fetal alcohol syndrome, a condition that can lead to low birth weight, intellectual impairment, hyperactivity, and certain physical defects. Any alcohol use during pregnancy, however, may influence the child's development and lead to problems with learning, attention, memory, or problem solving. Because scientists have not yet identified safe levels, alcohol should be used cautiously or avoided entirely by women who are pregnant or who may soon become pregnant. Research has also indicated:

- Third trimester exposure may affect the developing hippocampus or allied structures, leading to deficits in the ability to encode visual or auditory information. (Coles, 1991)

- Alcohol-exposed children are likely to experience academic difficulties, and it is possible that some of these children will develop specific learning disabilities. (Coles, 1991)
- Lower verbal comprehension and spoken language scores were found among 84 children at 13 months of age whose mothers drank an average of .24 ounces of absolute alcohol per day—about one-half drink per day. (Gusella & Fried, 1984)

Nicotine - According to Pressinger (1999), although the percentage of smoking in the general population is declining, the rate of this [decline] is slowest among women of childbearing age. The recent National Household Survey on Drug Abuse reported that among women of reproductive age, approximately one-third smoke cigarettes on a regular basis. These figures for the United States are within one or two percentages of those noted in Canada and Sweden. In five surveys throughout the U.S., the extent of cigarette use by women during pregnancy in non-ghetto, urban regions has been reported to be between 22% and 30%. An additional recent statistic that bears upon the issue of smoking habits and pregnancy is that the proportion of heavy smokers has increased in the past decade, particularly among women. In Sweden, the proportion of heavy smokers has almost doubled, while in Canada, the increase of heavy smokers was 57% among females versus 31% among males. This has important implications because the relationship between the consequences of maternal smoking and effects on the offspring appears to be dose related. Also of concern is that it is estimated by the Office of Smoking and Health that one-third to one-half of nonsmoking pregnant women are exposed to significant levels of involuntary or second-hand smoke. Demonstrating the increased toxic insults today's developing child has from cigarette smoke, figures show smoking has increased 3 to 4-fold from 1940 to the beginning of the 1980s, although it has since then decreased somewhat. (p. 1)

Smoking cigarettes has been found to have a possible causal effect on learning disabilities in children. Research suggests the following:

- In pregnant women who smoke, accumulation of carbon monoxide in the fetal blood stream could lead to serious reductions in oxygen to the developing infant. Carboxyhemoglobin levels (hemoglobin that is carrying carbon monoxide instead of oxygen) can concentrate in the developing fetus reaching twice the levels of that in the mother. (Denson, Nanson, & McWatters, 1994)
- Children of mothers who smoked 10 or more cigarettes a day are between three and five months behind in reading, mathematics, and general ability when compared to the offspring of non-smokers, after allowing for associated social and biological factors. (Makin & Fried, 1991)
- McCartney (1994) found overall poorer performance on central auditory processing tasks (SCAN) among 110, six- to eleven-year-old children exposed to prenatal cigarette smoke. Maternal smoking during pregnancy was linearly associated with the poorer performance on listening skills in a noisy background and attending to simultaneous information in both ears. Children exposed to passive cigarette smoke performed more poorly than children of non-smokers and equal to that found in children exposed to light prenatal smoking.
- Human reports as well as animal studies have recorded accelerated motor activity, learning and memory deficits in offsprings of mothers exposed to nicotine during pregnancy. (Roy, 1994)
- Research has links increased hyperactivity, attention deficits, lower IQ, and learning disabilities in children with parents who smoked during pregnancy (Roy, 1994)

- According to the Finland Department of Public Health (1994), the more cigarettes a mother smoked during pregnancy, the greater the likelihood her child would demonstrate severe behavior problems as the child became older.

Women who smoked at least a pack a day had children with twice the rate of extreme behavior problems such as anxiety, conflict with others, or disobedience, when compared with children of non-smokers. School performance of the smokers' children was poorer than that of their controls when measured in terms of their mean ability on theoretical subjects. The children of the smokers were more prone to respiratory diseases than the others. They were also shorter in length by nearly 1 centimeter (a little less than a half an inch) and their mean ability at school was poorer than among the controls for mothers who smoked 10 cigarettes and 20 cigarettes per day.

Pesticide Exposure during Pregnancy - Exposure among pregnant women to pesticides generates considerable concern, as these chemicals are intentionally designed to damage the nervous system. This concern increases when we realize the total extent to which all of us are exposed to pesticides. In fact, evidence now shows that everyone is exposed to some level of pesticides every second of the day. In June, 1993, the National Academy of Sciences released its long-awaited report on the health hazards posed to infants and young children from exposure to pesticides in the food supply. The NAS stated that any pesticides are harmful to the environment and are known or suspected to be toxic to humans. Pesticides can produce a wide range of adverse effects on human health that include acute neurologic toxicity, cancer, reproductive dysfunction, and possible dysfunction of the immune and endocrine systems.

Among the NAS's critical findings were that existing pesticide policies do not protect the young adequately, instead treating kids as little adults. Unique dietary patterns common among children (such as eating only a few favorite foods almost exclusively) are ignored in existing calculations, although such habits result in children's far greater exposure to multiple pesticides in food, by body weight, than occur in the adult population. The NAS expressed particular concern over children's dietary exposure to neurotoxic pesticides, stating that children tend to retain a greater portion of a given dose of certain toxins than adults and are not as capable of detoxifying them in their bodies because their livers are still developing. Children also are at greater risk from neurotoxins because the nervous system in an infant or young child has not yet developed fully.

Toxins in the Child's Environment

Toxic Metals - Researchers are looking into environmental toxins that may lead to learning disabilities, possibly by disrupting childhood brain development or brain processes. According to studies reviewed, more than 20% of the children in the United States have had their health or learning significantly adversely affected by toxic metals such as mercury, lead, and cadmium; and more than 50% of children in some urban areas have been adversely affected. Significant behavioral effects were also documented. Adults can be similarly affected. Many epidemiologists believe the evidence demonstrates that over 50% of all U.S. children have had their learning ability or mental state significantly adversely affected by prenatal or postnatal exposure to toxic substances. Toxic metals have been documented to be reproductive and developmental toxins, causing birth defects and damaging fetal development, as well as creating or contributing to neurological effects, developmental delays, learning disabilities, depression, and behavioral abnormalities in many children.

Lead Poisoning - Approximately 434,000 U.S. children aged 1 to 5 years have blood lead levels greater than the Center for Disease Control's (2004) recommended level of 10 micrograms of lead per deciliter of blood. Lead poisoning can affect nearly every system in the body. Because lead poisoning often occurs with no obvious symptoms, it frequently goes unrecognized. Lead poisoning can cause learning disabilities, behavioral problems, and, at very high levels, seizures, coma, and even death.

Lead is a metal that does not belong in the human body. Today, lead-based paint is the most common source of lead poisoning in children. Over many years, painted surfaces crumble and become common household dust. This dust coats the objects that curious children put in their mouths. Adults can also ingest lead in this way. It is the most common way for the lead in paint to get into a person. Children will also chew on windowsills or other painted surfaces. Sometimes they eat old paint chips.

The major source of lead exposure among U.S. children is lead-based paint and lead-contaminated dust found in deteriorating buildings. Lead-based paints were banned for use in housing in 1978. However, approximately 24 million housing units in the United States have deteriorated leaded paint and elevated levels of lead-contaminated house dust. More than 4 million of these dwellings are homes to one or more young children.

Other sources of lead poisoning are related to hobbies (making stained-glass windows), work (recycling or making automobile batteries), drinking water (lead pipes, solder, brass fixtures, valves can all leach lead), and home health remedies.

Certain children are at greater risk for lead poisoning. These children include the following:

- Children under the age of 6 years because they are growing so rapidly and because they tend to put their hands or other objects into their mouths.
- Children from all social and economic levels can be affected by lead poisoning, although children living at or below the poverty line (who generally live in older housing) are at greatest risk.
- Children of some racial and ethnic groups living in older housing are disproportionately affected by lead. For example, 22% of black children and 13% of Mexican-American children living in housing built before 1946 have elevated blood lead levels, compared with 6% of white children living in comparable types of housing.

Exposure to lead can have a wide range of effects on a child's development and behavior. Even when exposed to small amounts of lead levels, children may appear inattentive, hyperactive, and irritable. Children with greater lead levels may also have problems with learning and reading, delayed growth, and hearing loss. At high levels, lead can cause permanent brain damage and even death (American Academy of Pediatrics, 2000).

Cadmium - There are some researchers that suggest that cadmium exposure is related to learning disabilities in children. Cadmium is a natural element in the earth's crust. It is usually found as a mineral combined with other elements such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulfur (cadmium sulfate, cadmium sulfide) (U.S. Department of Labor, 2004).

All soils and rocks, including coal and mineral fertilizers, contain some cadmium. Most cadmium used in the United States is extracted during the production of other metals such as zinc, lead, and copper. Cadmium does not corrode easily and has many uses, including batteries, pigments, metal coatings, and plastics. The health effects in children are expected to be similar to those in adults (kidney, lung, and intestinal damage). It is not known if cadmium causes birth defects in people. Cadmium does not readily pass from a pregnant woman's body into the developing child, but some portion can cross the placenta.

Cadmium can also be found in breast milk. The offspring of animals exposed to high levels of cadmium during pregnancy had changes in behavior and learning ability. Cadmium may also affect birth weight and the skeleton in developing animals.

Animal studies also indicate that more cadmium is absorbed into the body if the diet is low in calcium, protein, or iron, or is high in fat. A few studies show that younger animals absorb more cadmium and are more likely to lose bone and bone strength than adults.

Severe Head Injuries

The signs of severe head injuries can be very different, depending on where the brain is injured and how severely. Spivak (1986) estimated that as many as 20% of children identified with a learning disability have had a prior brain injury. Children with brain injuries will often have LD, as well as other difficulties, such as those listed below.

Physical disabilities - Individuals with severe head injuries may have problems speaking, seeing, hearing, and using their other senses. They may have headaches and often feel tired. They may also have trouble with skills such as writing or drawing. Their muscles may suddenly contract or tighten (called spasticity). They may have seizures. Their balance and walking may also be affected. They may be partly or completely paralyzed on one side of the body, or both sides.

Difficulties with thinking - Because the brain has been injured, it is common that the person's ability to use the brain changes. For example, children with head injuries may have trouble with short-term memory (being able to remember something from one minute to the next, such as what the teacher just said). They may also have trouble with their long-term memory (being able to remember information from a while ago, such as facts learned last month). People with severe head injuries may have trouble concentrating and only be able to focus their attention for a short time. They may think slowly. They may have trouble talking and listening to others. They may also have difficulty with reading and writing, planning, understanding the order in which events happen (called sequencing), and judgment.

Social, behavioral, or emotional problems - These difficulties may include sudden changes in mood, anxiety, and depression. Children with head injuries may have trouble relating to others. They may be restless and may laugh or cry a lot. They may not have much motivation or much control over their emotions.

Most individuals with severe head injuries display attention or concentration deficits. Attention or concentration refers to the amount of time an individual stays on task. It might mean the short

amount of time it takes to hear information, or the length of time it takes to process that information.

An individual's ability to perceive and understand information seen, heard, or touched is considered comprehension. An individual with a brain injury will display difficulties with comprehension, which is evidenced in the following ways: difficulty understanding written and pictorial directions, difficulty following conversations (due to vocabulary problems), misinterpretation of auditory and visual information, literal interpretations of jokes and proverbs, and decreased understanding of questions.

Social–Environmental Causes

Low Socio-Economic Status and Learning Disabilities - According to Blair and Scott (2002; cited in Deutsch-Smith, 2004, p. 119), “a strong relationship exists between learning disabilities and low socio-economic status (SES) Whether factors associated with poverty (such as limited access to health care) or the lack of supportive environment puts these children at great risk for learning disabilities is not known, but the relationship is clear.” Although the IDEA definition and others specifically exclude SES conditions as etiological possibilities, many educators believe that this risk factor indirectly contributes to the learning and behavioral difficulties of some pupils.

The Relationship between Poor Instruction and Learning Disabilities - Another environmental variable that is likely to contribute to children's learning problems is the quality of instruction that they receive. As noted by Heward (2003), many special educators believe that Engelmann (1977) was correct when he claimed more than 25 years ago that the vast majority of “children who are labeled ‘learning disabled’ exhibit a disability not because of anything wrong with their perception, synapses, or memory, but because they have been seriously mistaught. Learning disabilities are made, not born.” Lovitt (1978) also contends that learning disabilities may result from poor teachers and inadequate instruction. While implying that the poor quality of some learning environments contributes to learning disabilities, researchers also note that learning problems can be remediated by direct, systematic instruction (Gersten, Carnine, & Woodward, 1987; cited in Gargiulo, 2004).

Conclusion

Since no one knows for sure what causes learning disabilities, mental health professionals stress that it does not help to look backward to search for possible reasons. Despite substantial work related to this field, determining precise causation has been difficult, and the effort to do so still continues (Hardman, Drew, & Egan, 2003). As should be evident after reading this chapter, there are likely many causes of learning disabilities, and in some cases, a specific type of learning disability may have multiple causes. Teachers need to recognize that it is not certain what causes learning disabilities and to not make assumptions about the students they teach (Deutsch-Smith, 2004).

Consequently, parents and teachers must adopt an eclectic, multidisciplinary approach and seek advice from professionals in the field. It is far more important to move forward in finding ways to cope effectively and overcome the difficulties.