

Fetal Alcohol Syndrome (FAS) ^[1]

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The concept [Fetal Alcohol Syndrome](#) ^[3] (FAS) refers to a set of [birth defects](#) ^[4] that occur in children born to mothers who abused alcohol during [pregnancy](#) ^[5]. The alcohol-induced defects include pre- and post-natal growth deficiencies, minor facial abnormalities, and damage to the developing [central nervous system](#) ^[6] (CNS). FAS is the most serious condition physicians group under the heading of Fetal Alcohol Spectrum Disorders, which also includes Alcohol-Related Birth Defects, like alcohol-induced congenital cardiac defects that are unrelated to a diagnosis of FAS, and Alcohol-Related Neurodevelopmental Disorders, which occur in the absence of any facial [birth defects](#) ^[4] or growth delays. The severity of [birth defects](#) ^[4] associated with FAS can vary depending on the intensity, duration, and frequency of exposure to alcohol during [gestation](#) ^[7]. In addition to these dose-related concerns, maternal factors such as the mother's genetics or how quickly she metabolizes alcohol, and the timing of exposure during prenatal development also impact alcohol-induced abnormalities. As [birth defects](#) ^[4] and anomalies can arise when pregnant women consume alcohol, alcohol is a teratogen, an environmental agent that negatively impacts the course of normal embryonic or fetal development.

Given the broad range of symptoms that encompass FAS, the number of children impacted by maternal drinking during [pregnancy](#) ^[5] is difficult to determine. In 2012, the US Center for Disease Control and Prevention estimated that for every 1,000 children born in the US, between 0.2 and 1.5 are born with FAS. However, the number of infants born with a Fetal Alcohol Spectrum Disorder, which can include some but not all of the alcohol-induced [birth defects](#) ^[4] and anomalies required for a full FAS diagnosis, could be three times as prevalent.

Groups of French and American researchers concurrently observed the defects specific to FAS in the late 1960s and early 1970s. In 1968, Paul Lemoine and colleagues in Nantes, France, examined 127 children from 69 families that had at least one parent with chronic alcoholism. Among these children, researchers observed facial abnormalities and cognitive defects that manifested as low Intelligence Quotient (IQ) scores, hyperactivity, and developmental delays in motor coordination and language skills. Medical communities abroad largely dismissed the initial reports, indicating that many viewed alcohol as a benign agent into the late 1960s. Five years later US researchers observed a similar set of [birth defects](#) ^[4], and researchers recognized the potential of alcohol as a teratogen, legitimizing FAS.

Kenneth L. Jones and [David W. Smith](#) ^[8], pediatricians specializing in congenital [birth defects](#) ^[4] at the University of Washington School of Medicine in Seattle, Washington, catalogued a series of alcohol-induced [birth defects](#) ^[4] in 1973. They coined the term [Fetal Alcohol Syndrome](#) ^[3] in a 1973 article. Over the course of a year in several follow-up studies, Jones and Smith further refined the description of the defining defects and published their results. Although Jones and Smith catalogued the basic morphological defects in those articles, researchers and physicians later detailed alcohol-induced growth deficiencies, facial abnormalities, and [central nervous system](#) ^[6].

Whereas the majority of the alcohol-induced defects that occur with FAS affect a certain cell population, more systemic alcohol-induced defects are pre- and post-natal growth deficiencies. Fetuses and infants with FAS are small for their gestational age, and their growth deficiencies persist into childhood. They have difficulty gaining weight and are generally lower in weight and stature compared to non-affected children, a condition historically defined as failure to thrive. During [gestation](#) ^[7], these alcohol-induced growth defects increase the likelihood of [miscarriage](#) ^[9], stillbirth, and premature birth. Even when [a pregnancy](#) ^[5] is brought to term there is almost a two pound difference in the average weights between FAS-affected infants (five pounds, one ounce) and non-affected infants (seven pounds, seven ounces). These deficiencies are due in part to alcohol-induced defects to placentas, such as reduced thickness, poorly developed blood vessels, and the impaired transfer of nutrients to the developing [fetus](#) ^[10].

Compared to the broad pre- and post-natal growth deficiencies, the minor facial abnormalities in children with FAS are more readily identified. The pattern of facial defects alters the [morphology](#) ^[11] of midline features of the face, including the eyes, nose, and lips. These diagnostic characteristics include smaller eye openings (palpebral fissures) and folds of skin (epicanthal folds) at the corners of the eyes that stretch the upper lid taut to create a more oval shaped appearance of the eye than children who do not have FAS. The nose is generally shortened and accompanied by a lower nasal bridge. The ridges of the groove under the nose, called the philtrum, may be hypoplastic (underdeveloped) or absent. Facial defects occur as a result of alcohol-induced damage to cranial [neural crest cells](#) ^[12], which are responsible for the formation of the frontonasal process that gives rise to facial features.

Many FAS-related defects occur in the developing [central nervous system](#) ^[6] (CNS), and scientists have correlated them with gross morphological abnormalities of the brain and compared to normal children, an overall decrease in [white matter](#) ^[13], particularly in the cerebrum, or [forebrain](#) ^[14]. The human CNS is vulnerable to the teratogenic effects of alcohol from when the

[neural plate](#)^[15] begins to form in the third week through the rest of [gestation](#)^[7]. Certain areas of the developing CNS are particularly susceptible to alcohol-induced [birth defects](#)^[4], including the ocular system, [corpus callosum](#)^[16], basal ganglia, and [cerebellum](#)^[17].

FAS defects that impact eye development can lead to visual impairment or blindness in the embryo. The vulnerable time period for damage to the eyes in [humans](#)^[18] begins in the third week following [fertilization](#)^[19], when the precursor to what will become the eyes, called the optic cup begins to develop. Microphthalmia, or abnormally small eyes, occurs in ninety percent of FAS-affected individuals. Developmentally small eyes occur when alcohol affects the retina as it begins to form. A smaller retina results in a smaller than normal eye, which can further impair vision. Alcohol's impact on ocular development can also lead to optic nerve [hypoplasia](#)^[20], the leading cause of developmental blindness

Alcohol also impacts the Corpus callosums of fetuses exposed to alcohol between weeks five and six after [fertilization](#)^[19]. The [corpus callosum](#)^[16] is a dense band of [white matter](#)^[13] that separates the right and left hemispheres of the brain and is responsible for coordinating communication between hemispheres. Some individuals with FAS exhibit defects which include complete non-development (agenesis), underdevelopment, or spatial displacement of the [corpus callosum](#)^[16]. Corpus callosum defects can lead to poor bimanual motor coordination or motor-visual coordination, and issues with faculties like abstract thought and decision making.

The basal ganglia, a cluster of nuclei deep within the brain, also act as a center of communication between the cerebrum, thalamus, and surrounding areas of the brain. As nuclei, the basal ganglia are clusters of specialized, densely-compacted neurons, and when exposed to alcohol, can decrease in volume just like the [white matter](#)^[13] that composes the CNS. Defects to the basal ganglia can impact motor control, spatial awareness, memory and verbal learning. Defects can also lead to behavioral issues in children, such as hyperactivity and impulsivity that characterize attention deficit disorders, and the perseverative behaviors that characterize obsessive compulsive disorders and autism spectrum disorders.

The [cerebellum](#)^[17] begins differentiating in the third [trimester](#)^[21] and it is the last structure in the brain to do so. During the third [trimester](#)^[21], the fetal brain undergoes an intense period of [neuron](#)^[22] creation (neurogenesis and gliogenesis) as well as development of the sites where chemicals transmit signals between neurons ([synaptogenesis](#)^[23]). That time period is called the [brain growth spurt](#)^[24]. Defects to the [cerebellum](#)^[17] can result in issues with physical dexterity, coordination, and visuospatial processing, as well as problems with learning and memory.

Scientists studied animal models, primarily [chick](#)^[25], [mouse](#)^[26], [rat](#)^[27], and [zebrafish](#)^[28] to define the mechanisms and developmental timeline of alcohol's teratogenic effects on developing embryos and fetuses. Alcohol's impact on developing neurons include an increase in programmed cell death ([apoptosis](#)^[29]), as well as oxidative stress and damage to [radial glia](#)^[30] which are the cellular populations that later in the process of development become neurons. All of those mechanisms impair the proliferation and migration of cells, and damage to cell populations has been proposed as a process for how alcohol causes [birth defects](#)^[4] and CNS damage. The exact mechanism of alcohol's teratogenicity in the destruction of certain cell populations is debated, and the above mechanisms may work separately or together.

Scientists have debated whether or not the alcohol-induced [apoptosis](#)^[29] prematurely eliminates specific cell populations during development, such as cranial [neural crest cells](#)^[12], retinal cells, [cerebellum](#)^[17] cells and other vulnerable populations. Numerous studies confirm that the biomechanical mechanism occurring during this cell death as being a [capsase-3](#)^[31] enzyme activation cascade, which is a component of programmed cell death by [apoptosis](#)^[29]. Scientists have also hypothesized that this programmed cell death may be triggered by the metabolic breakdown of alcohol into acetaldehyde, which can inhibit the formation of retinoic acid. Retinoic acid is the metabolized product of Vitamin A, (retinol) a part of many developmental processes.

In addition to those proposed apoptotic mechanisms, alcohol damages the neural stem cell progenitor pools, like [radial glia](#)^[30], that give rise to neurons and the supporting glial cells in the CNS. In addition to forming cells in the CNS, [radial glia](#)^[30] provide a physical scaffolding for, and chemically guide the neurons they form. When alcohol affects embryos or fetuses, it can impact the development and migration of these [radial glia](#)^[30] progenitor pools and it can result in the creation of fewer neurons and [glia](#)^[32], morphological abnormalities in those that are produced, and confounded [cell migration](#)^[33]. Damage to these cell populations can decrease their volumes and it can cause structural abnormalities, which can impact the CNS from its initial development through to the development of neural networks.

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The concept Fetal Alcohol Syndrome (FAS) refers to a set of birth defects that occur in children born to mothers who abused alcohol during pregnancy. The alcohol-induced defects include pre- and post-natal growth deficiencies, minor facial abnormalities, and damage to the developing central nervous system (CNS). FAS is the most serious condition physicians group under the heading of Fetal Alcohol Spectrum Disorders, which also includes Alcohol-Related Birth Defects, like alcohol-induced congenital cardiac defects that are unrelated to a diagnosis of FAS, and Alcohol-Related Neurodevelopmental Disorders, which occur in the absence of any facial birth defects or growth delays. The severity of birth defects associated with FAS can vary depending on the intensity, duration, and frequency of exposure to alcohol during gestation. In addition to these dose-related concerns, maternal factors such as the mother's genetics or how quickly she metabolizes alcohol, and the timing of exposure during prenatal development also impact alcohol-induced abnormalities. As birth defects and anomalies can arise when pregnant women consume alcohol, alcohol is a teratogen, an environmental agent that negatively impacts the course of normal embryonic or fetal development.

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