

Pre- and Post-natal Growth Deficiencies and Fetal Alcohol Syndrome ^[1]

By: O'Neil, Erica Keywords: [Fetal alcohol syndrome](#) ^[2] [Human development](#) ^[3]

Maternal consumption of alcohol (ethanol) during [pregnancy](#) ^[5] can inhibit prenatal growth, resulting in fetuses that are small for gestational age. Those prenatal growth deficiencies can have lasting consequences for early childhood development and are often reflected by low weight and stature. Those alcohol-induced pre- and post-natal growth deficiencies (failure to thrive) are among the abnormal developmental criteria used to identify [Fetal Alcohol Syndrome](#) ^[6] (FAS). FAS is characterized by minor facial abnormalities and deficiencies of the [central nervous system](#) ^[7] as well. A deficiency in prenatal growth is often referred to as an intrauterine growth restriction (IUGR), a general term that refers to stunted fetal growth that may be a result of genetic or environmental factors.

IUGR is estimated to occur in approximately 5 to 7 percent of infants born in the United States, a statistic that includes both alcohol-induced growth deficiencies and those that are due to other causes. Those impacts on gestational growth can be fetal, maternal, or placental in origin, and can result from chromosomal abnormalities in the [fetus](#) ^[8], smoking during [pregnancy](#) ^[5], or a mother contracting rubella while pregnant. Most often, IUGR occurs due to placental abnormalities that impede nutrient exchange between the mother and [fetus](#) ^[8] and results in malnutrition and growth deficiencies. Those growth deficiencies are generally classified when birth weights or the estimated weight of a developing [fetus](#) ^[8] is in the lowest tenth percentile for gestational age. The average birth weight for a full-term child born with FAS is almost two pounds less than non-affected children (5 pounds, 1 ounce, to 7 pounds, 7 ounces, respectively). Measurements of body parts such as the circumference of the head and abdomen are also used to help classify IUGR.

IUGR is symmetric or asymmetric, depending on when growth disruptions occur during development. If growth is affected during the first and second [trimester](#) ^[9], or throughout the entire [pregnancy](#) ^[5], then the IUGR is referred to as symmetric due to a body-wide decrease in cellular proliferation and [differentiation](#) ^[10]. Symmetric IUGR is often observed in children with FAS, as it coincides with the developmental period during which alcohol results in widespread defects. Asymmetric IUGR occurs when growth disruptions occur only in the third [trimester](#) ^[9], and the pattern is generally characterized by a normal-sized head and a smaller than normal abdominal cavity. The head retains its normal size because by the third [trimester](#) ^[9] the [fetus](#) ^[8] can redistribute cardiac resources to the command centers of the body, such as the brain and heart, at the expense of other less vital organs like the digestive system.

In addition to reduced cellular proliferation, ethanol has the ability to affect the proper development of the [placenta](#) ^[11]. Ethanol-induced placental defects include a general [reduction](#) ^[12] in thickness and poorly developed blood vessels that impair nutrient exchange between the mother and [fetus](#) ^[8] that can result in fetal malnutrition. Furthermore, insulin-like growth factors mediate the population of cells responsible for the maintenance of the [placenta](#) ^[11] and blood vessels. As ethanol inhibits the expression of those factors, poor placental development can occur that further exacerbates malnutrition and contributes to growth deficiencies.

The effects of fetal growth deficiencies can persist well after birth. Maternal and fetal iron deficiency [anemia](#) ^[13] has been cited as a potential factor contributing to initial low birth weights and sustained delays in childhood growth. Pregnant mothers who are heavy consumers of alcohol are at a higher risk for [anemia](#) ^[13], which means that less dietary iron is available to the [fetus](#) ^[8] during development. There is also a greater risk for placental defects that may affect the delivery and absorption of iron to the [fetus](#) ^[8]. A [fetus](#) ^[8] relies on maternal iron for support throughout much of the [pregnancy](#) ^[5] and during the third [trimester](#) ^[9] the [fetus](#) ^[8] begins to store iron in preparation for the demands of early infancy. The fetal iron stored is generally sufficient to last six months after birth, at which point iron must be obtained from the infant's diet. Impairments to iron absorption and delivery may result in general growth deficiencies due to [anemia](#) ^[13] in early childhood.

Although there are no clinical methods for preventing or mitigating the effects of IUGR, preliminary laboratory studies in mice have shown the potential for two proteins, ADNF-9 and NAPVSIPQ, to reverse IUGR and prevent fetal deaths that would otherwise result from those growth deficiencies. It has been proposed that the proteins may confer protection against the oxidative stress generated as a byproduct of ethanol metabolism.

Further laboratory analyses of the causes of IUGR have shown the potential to mitigate those growth deficiencies in animal models but not [humans](#) ^[14]. The issue is further complicated by recognizing that ethanol-induced IUGR results as a combination of environmental and biological mechanisms, including a complex interplay between maternal, fetal, and placental resources. Heavy maternal alcohol consumption while pregnant has often resulted in small children that have difficulty gaining weight and are often abnormally small for their developmental age, historically noted as a general failure to thrive. While the mechanisms of

those growth deficiencies are not fully understood, it is recognized that pre- and post-natal growth defects are important criteria by which FAS can be identified.

Sources

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[Fetal alcohol syndrome](#)^[15] [Fetal Alcohol Syndrome](#)^[16]

Topic

[Disorders](#)^[17]

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- [1] <https://embryo.asu.edu/pages/pre-and-post-natal-growth-deficiencies-and-fetal-alcohol-syndrome>
- [2] <https://embryo.asu.edu/keywords/fetal-alcohol-syndrome>
- [3] <https://embryo.asu.edu/keywords/human-development>
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- [5] <https://embryo.asu.edu/search?text=pregnancy>
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- [9] <https://embryo.asu.edu/search?text=trimester>
- [10] <https://embryo.asu.edu/search?text=differentiation>
- [11] <https://embryo.asu.edu/search?text=placenta>
- [12] <https://embryo.asu.edu/search?text=reduction>
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