News
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******Dallas scientists search for a way to prevent premature births.

DALLAS--Three hundred thousand babies are born prematurely each year in the United States. Those that survive often suffer permanent physical and mental disabilities. For example, it has been estimated that 50 percent of the patients in mental institutions in the U.S. are victims of an untimely birth, according to an internationally known researcher in obstetrics.

Prematurity may be this country's major health hazard, says Dr. Paul MacDonald, director of the Cecil H. and Ida Green Center for Reproductive Biology Sciences at The University of Texas Southwestern Medical School at Dallas.

The problems of cancer and cardiovascular disease--which mainly strike adults--seem small compared with the astronomical costs and the suffering caused by prematurity, he says. "The child that is born prematurely and survives may face 70 years or more of living with a serious and costly disability such as brain damage and cerebral palsy."

Dr. MacDonald recently began a two-million-dollar, five-year study titled "The Initiation of Labor: Prevention of Prematurity." The goal of the federally funded study is to find the mechanism that triggers the contractions of the uterus in labor and thus ejects the fetus from the womb. "If we fully understood what causes labor to begin, we almost certainly could find a way to prevent or arrest premature labor," Dr. MacDonald says.

A number of theories have been proposed to explain the onset of labor, but none of them have been proven. One theory suggested that labor begins when the wall of the uterus is stretched to a certain degree. Another theory is that a hormone called "oxytocin" formed in the mother's brain is responsible for labor's beginning.

But now Dr. MacDonald and his associates have come up with new evidence that the fetus itself generates the biochemical signals that lead to its journey down the birth canal. "Our hypothesis is based on the concept that the fetus controls its own destiny," he says.

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According to the Dallas team's hypothetical model of labor, the triggering mechanism is a complex series of biochemical events involving the fetus' brain and endocrine glands as well as the fetal membranes. (The fetal membranes, along with the placenta, make up the "afterbirth," which is expelled from the uterus after childbirth.)

One of the key discoveries made by the UT Southwestern researchers is that a fatty acid known as "arachidonic acid" is present in large quantities in the fetal membranes. "Dr. Jack Johnston in the biochemistry department here measured the content of arachidonic acid in human fetal membranes and found they are one of the richest sources of that substance ever studied," Dr. MacDonald explains.

Arachidonic acid is the substance from which a compound called a "prostaglandin" is formed, he continues, and that prostaglandin will induce labor at any stage of pregnancy. "The prostaglandin levels increase in the pregnant women at the time of labor, so it seems likely that the prostaglandin is involved in the initiation of labor."

The arachidonic acid is stored in the fetal membranes as a chemical compound known as an "ester." Dr. MacDonald adds:

"In order to make prostaglandin, you have to liberate arachidonic acid from the ester. There is an enzyme found in snake venom that will do this, so the biochemists here at Southwestern tested the fetal membranes for that enzyme, and they found that it is present in the membranes. Moreover, they found that there is a lot of free arachidonic acid in the amniotic fluid of women in labor, which is consistent with our theory that the rate of release of arachidonic acid determines the onset of labor."

However, each theory explaining labor still seems deficient to some extent, Dr. MacDonald says. "Our inability to identify conclusively the events involved in labor only emphasizes the need for further studies."

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