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News
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******Major investigation underway on Respiratory Distress Syndrome in premature infants.

DALLAS--Floating inside the mother's womb, the developing fetus exists with only rudimentary lungs until the final stages of development.

For some infants, forced into the world prematurely, the lungs are critically immature and have a decreased capacity for exchanging carbon dioxide for oxygen.

A research team at Southwestern Medical School, led by Dr. John M. Johnston, professor of biochemistry and obstetrics and gynecology, is involved in a major investigative project aimed at the prevention of a condition which claims the lives of 40,000 premature babies each year in the U.S.

Called Respiratory Distress Syndrome (or Hyaline Membrane Disease), the disease is the leading cause of mortality in prematurely born infants. The more premature the infant, the higher the risk of the disease.

The condition is characterized by a collapse and sticking together of the baby's lungs on expiration. In its most serious form, membranes form where the lungs stick together. And by X-ray the lungs have a "ground glass" pattern made up of collapsed alveolar spaces (the fiber-lined air sacs within the lungs).

The disease received public attention in the early '60s with the death of young Patrick Kennedy.

Symptoms of RDS are often easily recognizable by obstetricians. With the first expiration of air, the child grunts with exertion--nostrils flaring in an excruciating attempt to get oxygen into the lungs. All the child's energy goes into each breath to fill the lungs with air. Breathing becomes almost impossible. Emergency treatment with use of a respirator is needed to pop the lungs open while sustaining a constant pressure inside the lungs to prevent the collapse of the alveoli.

And then comes the anxious waiting period by parents to see if their child can develop its lungs while its life is being sustained by a respirator. Hospital costs can become staggering.

Dr. Johnston explains that the chemical key to the lung maturation process is a substance called "surfactant." Surfactant is a protein and lipid (fatty material) complex. The lipid component is chemically similar to the non-stick spray used to coat frying pans. One of the chief components of surfactant is lecithin. And the lecithin part of surfactant begins to be produced by the lungs as early as the 24th week of pregnancy (the normal length of gestation is 40 weeks). It's not until around the 35th week, however, that lecithin is produced in adquate amounts to facilitate breathing after delivery.

Working under a grant from the National Institute of Child Health and Human Development, Johnston and the team of investigators are researching the regulation of surfactant production. With researchers in biochemistry, obstetrics and gynecology, physiology, pediatrics and cell biology--all working within the Cecil H. and Ida Green Center for Reproductive Biology Sciences--the ultimate goal is to understand the biochemical basis for the

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mechanism by which surfactant is made during lung maturation. The doctors think that perhaps by administering hormones which regulate surfactant formation, they can cause the lungs of the fetus to mature more rapidly in utero. This would be done in high risk pregnancies after taking samples of the amniotic fluid from within the uterus and testing the fluid for surfactant levels. The purpose would be to stimulate the lungs to produce the necessary surfactant components.

Averting RDS becomes a necessary consideration in certain classes of diabetes in the mother, Johnston explains. Here, even if the mother carries the baby close to term, the child may still have RDS. This is because the surfactant in this case is produced in adequate amounts but is deficient in one of the lipids, phosphatidylglycerol.

"Recently we've been able to find a biochemical mechanism by which we can relate the fact that an increase in lecithin causes an increase in phosphatidylglycerol," Johnston says. Yet he adds that this doesn't hold true in the diabetic mother since the two lipids become disassociated.

Two percent of all pregnant women develop gestational diabetes, a form of diabetes in which the mother is only diabetic during pregnancy and then 24 hours after delivery she returns to normal.

RDS is also a problem for the mother with vascular disease.

For cases of diabetes and vascular disease, the doctor is faced with a formidable problem. Since the fetus in both cases can often die in utero, the doctor must deliver the child before it dies in the uterus but must wait as long as possible so that the lungs can mature. The problem is compounded in the diabetic mother since testing for the lecithin of surfactant is less reliable.

Johnston and the other Southwestern researchers are looking at how surfactant is made and how it's secreted by the lung's alveolar type II cells. They also are investigating the hormonal influences involved in the regulation of the biochemical processes. Earlier they predicted which regulatory enzymes would aid in the making of surfactant and successfully showed that the activity of certain key enzymes was increased as a function of gestational age. "We also found that the enzyme PAPase was secreted into the amniotic fluid with surfactant," says Johnston.

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