News
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March 19, 1984

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*****Biochemist discovers metabolic key to respiratory distress syndrome.

DALLAS -- Babies of mothers with diabetes are often born with respiratory distress syndrome (hyaline membrane disease).

Until recently it was thought that the babies' problem, usually seen in premature babies, was due to an imbalance in insulin and glucose in the mother. But Dr. John Bleasdale, assistant professor of Biochemistry and Obstetrics and Gynecology at The University of Texas Health Science Center at Dallas, has recently discovered that the condition may be caused by the mother's abnormal metabolism of inositol. Bleasdale recently presented his findings at the international Chilton Conference on Inositol and Phosphoinositides held at the health science center.

Inositol is a sugar produced from glucose by the body's cells and also found in fruits, vegetables and wheat in the diet. Pregnant women with diabetes do not metabolize inositol normally. And this results in an excess of the sugar supplied to the fetus, which in turn causes the fetus to produce an immature form of surfactant, the substance that allows the lungs to open normally.

"The alveoli (air sacs in the lungs), used for gas exchange, are by their nature unstable structures," says Bleasdale. "They have a tendency to collapse on expiration due to surface tension. This is overcome by lung surfactant, which acts as a detergent and allows the air sacs to remain open on expiration."

Surfactant is a lipoprotein, containing more than 90 percent fat, which is responsible for the surface tension-lowering property. The major fat (lipid) component is phosphatidylcholine, formerly called "lecithin."

"The lipids of the first surfactant produced in the fetus at about 32 weeks are mostly phosphatidylcholine and phosphatidylinositol (a lipid containing inositol)," says Bleasdale. Normally as the fetus develops, the inositol-containing fat is replaced by one with glycerol -- phosphatidylglycerol. This begins at the 35th week. Normal term for a fetus is 40 weeks.

A baby delivered before the phosphatidylglycerol-rich surfactant is produced is at greater risk for respiratory distress syndrome (RDS). "Normally after week 35 of gestation, the fetal surfactant becomes rich in phosphatidylglycerol, but this is delayed in babies of mothers with mild diabetes," says Bleasdale. The mild form of diabetes causes big babies, probably because they get extra glucose from the mother, but like premature babies they have an immature form of surfactant.

Babies of women with severe diabetes surprisingly do not have a greater risk for RDS. The severe form causes stress on the fetus, which accelerates lung maturation. So the babies are small but have mature surfactant.

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Developing, rapidly growing cells require large amounts of inositol. The fetal lung cells synthesize inositol from glucose and also take inositol from the blood. Normally the supply of inositol from both sources decreases in late gestation. This decrease causes the fetal lung cells to start producing a different lipid, the phosphatidylglycerol necessary for mature surfactant.

Because the diabetic mother supplies her fetus with abnormally large amounts of glucose and inositol, the availability of inositol to fetal lung cells remains high. So they continue to make phosphatidylinositol, resulting in immature surfactant.

"Failure of nerve cells to take up inositol has been implicated in diabetic neuropathy," says Bleasdale. "Some physicians advocate supplementing the diet with inositol to treat diabetic neuropathy. But we think that would be detrimental for a pregnant woman. It would further impair the synthesis of mature fetal surfactant."

Bleasdale's work was made possible by funds from the National Institutes of Health, the Robert A. Welch Foundation and the Cecil H. and Ida Green Center for Reproductive Biology Sciences.

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