FETAL PROLACTIN LEVELS AND RESPIRATORY DISTRESS SYNDROME

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SUMMARY

Prolactin levels were measured by radioimmunoassay in cord blood of infants between 28 and 40 weeks of gestation. Also infants, whose mothers received betamethasone prior to delivery, were examined.

Between 33 and 36 weeks, the mean plasma cord prolactin levels in infants who developed RDS were significantly lower than HPRL levels in those infants who did not develop RDS.

These findings suggest that betamethasone did not alter HPRL levels and point out the possibility of a role of prolactin in fetal lung maturation.

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The development of the respiratory distress syndrome (RDS) in the newborns depends on the deficient production of pulmonary surfactants which are rich of lecithin and phospholipids produced by fetal alveolar cells.

The mechanism that regulates fetal lung maturation is not clear.

Some hormones have been utilized to accelerate surfactant production: corticosteroids to the fetus ([2]), administration of betamethasone to the pregnant women ([6]), tiroxine ([3]), growth hormone ([9]), insulin ([8]).

Hamosh and Smith ([4, 8]) report that prolactin administered in utero increases the lung's lecithin content.

The aim of this paper is to explore the possibility that fetal prolactin might influence lung maturation.

MATERIAL AND METHODS

This study comprehends 77 infants between 28 and 40 weeks of gestation. Their mothers had received no glucocorticoids prior to delivery.

Samples of umbilical blood cord were obtained at delivery of the newborns. The plasma was separated and frozen until assay.

A further 28 newborns, between 28 and 36 weeks of gestation, whose mothers received betamethasone prior to delivery as prophylaxis against the development of RDS in the infants, were considered separately.

The development of RDS was assessed by clinical and radiologic signs. All obstetric and neonatal data were used to assess gestational age.

Prolactin levels were measured by radioimmunoassay.

RESULTS

Table 1 represents the subdivision of 77 infants according to gestational age, prolactin levels, and the presence or absence of RDS in the untreated group.

Of the 77 infants, 31 developed RDS.

The mean HPRL levels in infants between 33 and 36 weeks who developed RDS (94.8 ± 5.6 ng/ml) were significantly lower ($p < 0.025$) than the HPRL levels

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Table 1. — *Plasma prolactin levels in umbilical cord in the untreated group.*

Weeks of pregnancy	Cord prolactin levels (ng/ml) no-RDS	RDS	Statistical significant association
28-32	44.6 ± 19.8 (5)	18.4 ± 7.5 (6)	n.s.
33-36	112.7 ± 11.4 (21)	94.8 ± 5.6 (18)	< 0.025
37-40	101.3 ± 9.9 (20)	99.4 ± 10.4 (7)	n.s.

in infants of the same age who did not develop RDS (112.7±11.4 ng/ml).

There was a similar trend in newborns between 28 and 32 (18.4±7.5 vs 44.6± 19.8 ng/ml), but it did not reach statistical significance.

No difference was observed in the infants between 37 and 40 weeks (99.4± 10.4 vs 101.3±9.9 ng/ml).

Table 2 represents the subdivision of 28 infants between 28 and 36 weeks according to gestational age, prolactin levels, and the presence or absence of RDS. Their mothers were treated prior to delivery with betamethasone.

In this group fetal prolactin levels were similar to those observed in the first group.

DISCUSSION

We found lower cord prolactin levels in those infants who developed RDS compared to the others. This difference was significant between 33 and 36 weeks of gestation.

Similar results were observed in the infants whose mothers received betamethasone prior to delivery as prophylaxis against the development of RDS.

Liggins and Howie ([6]) observed an acceleration of fetal lung maturation after administration of betamethasone.

Studies on the temporal relationship between the rise of plasma corticosteroids and the appearance of surfactant in the amniotic fluid showed that the rise in fetal lung surfactant precedes the rise in fetal plasma cortisol ([7]).

Aubert ([1]) observed that fetal prolactin levels rise prior to the increase in surfactant synthesis.

Hamosh ([4]) suggests the possibility of a role of prolactin as a trigger of surfactant synthesis because he observed rapid increase in lung phospholipids in rabbit fetuses injected with prolactin.

Josimovich ([5]) demonstrated membrane receptors of HPRL in fetal monkey lungs.

Winters ([12]) suggested that fetal HPRL might exert an adrenocorticotropic role in utero, in view of the temporal relationship between fetal HPRL levels and adrenals weight.

Ballard ([2]) suggests that any effect of HPRL upon the fetal lung could be mediated by the fetal adrenal.

However, in the anencephalic fetus, cord HPRL levels are high while the fetal adrenal is hypoplastic.

Table 2. — *Plasma prolactin levels in umbilical cord of infants whose mothers were treated with betamethasone.*

Weeks of pregnancy	Cord prolactin levels (ng/ml) no-RDS	RDS	Statistical significant association
28-32	48.3 ± 20.2 (5)	20.6 ± 8.2 (2)	n.s.
33-36	115.2 ± 16.5 (16)	96.7 ± 7.4 (5)	<0.025

Fetal prolactin levels have been correlated with fetal estrogen levels ([1]). The estrogens might influence fetal lung maturation and the association between HPRL and RDS could reflect the influence of estrogens on prolactin secretion and lung maturation.

Our data show a significant association between low fetal plasma prolactin levels and the development of RDS in the newborns. These findings, accordingly to other investigations ([1, 4]), raise the possibility of a role of prolactin in fetal lung maturation.

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