fish extends to 51 and 51 sections, in the 4×10^{-7} T3 group to 87 and 83 sections and in the 8×10^{-7} T3 group to 99 and 94 sections on the LHS and RHS respectively. These results are summarized in the Table.

The most convincing point noted in this analysis was that among the 13 control fish and the 22 treated fish studied not a single case of overlaping results was found in the 2 groups. Comparing the responses to the two levels of the hormone, not a great deal of difference was noticed except at day 31 when the treatment of 8×10^{-7} T3 caused a small but an insignificant advanced growth of the bone. Figure a and b show the state of development of the lacrymal bone in a control and a T3 treated alevins respectively at day 31 after hatching.

These results provide further evidence in support of the finding that exogenous T4 promotes synthesis and further development of lacrymal bone in *Salmo trutta* alevin during the 31 days after hatching[2]. Moreover the results of T3 treatments are consistent as unlike those of T4 treatment; no overlaping cases were found in the T3 treatments. The results also indicate that the effect of higher concentration was slightly inhibitory as compared to the lower concentration one. It is also demonstrated here that T3 is more than 8 times as potent as T4 – a situation similar to that reported in the case of the rat[3,4]. The precise reason for the high potency of T3 are not known, however, it may be that T3 is metabolized faster than T4[5], thus the T3 treatment resulted in an efficient response[6].

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Free and Sulfoconjugated Dehydroepiandrosterone, Cyclic Adenosine-3′,5′-Monophosphate, and Free Estriol in Maternal and Cord Blood

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Summary. When free DHEA, its sulfatide, and sulfate were assayed in maternal plasma as well as in umbilical cord arterial and venous plasma, rather high concentrations were found in either fraction from cord arterial plasma, reflecting the fetal contribution not only of free DHEA and DHEA sulfate, but also of the lipophile steroid sulfatide. Since high DHEA levels were associated with elevated c-AMP concentrations, a certain interrelationship of both parameters is indicated. In the course of delivery, a rapid decrease of free estriol in maternal plasma was observed. Higher concentration of free estriol in umbilical venous plasma pointed at its placental biosynthesis from fetal precursors.

The role of sulfoconjugated DHEA (dehydroepiandrosterone, 3β-hydroxy-5-androsten-17-one) as a major precursor of estrogens in the fetoplacental unit has been established beyond any doubt[1,2]. Comparatively higher concentrations of sulfoconjugated DHEA in umbilical cord arterial blood suggested the biosynthesis of this precursor in fetal adrenal tissue[3]. Since, however, under physiological conditions the predominant portion of sulfoconjugated DHEA in adult human subjects apparently occurs as a lipophile compound, e.g. a diglyceride sulfate or 'sulfatide'[4,5], it seemed of particular interest to investigate whether also the fetal adrenal produces such lipophile sulfoconjugates. Especially in view of the fact that only DHEA sulfatide affects the activity of G-6-PDH (glucose-6-phosphate dehydrogenase)[6,7] or the concentration of c-AMP in plasma[8], whereas DHEA sulfate proved to be completely inactive in the G-6-PDH inhibition test.

Therefore, DHEA was determined in the fractions of free steroids, steroid sulfatides, and steroid sulfates from umbilical cord arterial and venous plasma as well as maternal plasma before, during, and after delivery. At the same time, c-AMP and free estriol (1, 3, 5(10)-estratriene-3, 16, 17β-triol) were measured in these samples.

Material and methods. In 20 normal pregnant women, blood samples were collected during the initial stage of labour, in the expulsion period, and 2 and 48 h after delivery. From the umbilical cord, arterial and venous blood were withdrawn immediately after delivery, yielding 0.8 to 3.7 ml of heparinized plasma.

All heparinized plasma samples were assayed for the above-mentioned parameters by standard procedures. Steroid sulfatides and sulfates were isolated by ion exchange chromatography on polyamide columns and subseuqnet thin layer chromatography[9]. Following solvolysis of sulfoconjugates DHEA was separated by repeated thin layer chromatography and quantitated by densitometry of its 2, 4-dinitrophenylhydrazone[10]. c-AMP was measured by the protein binding assay of Brown et al.[11], while free estriol was determined by radioimmunoassay[12].

Results and discussion. As shown in the Table, the maternal plasma levels of DHEA varied considerably in the course of delivery. Whereas in the initial stage of labour, the concentration of total DHEA averaged 83.9 μg/100 ml, a total of 97.2 μg DHEA/100 ml were found during the expulsion period. As compared to 42.3 $\pm$ 18.9 μg/100 ml, measured in peripheral plasma of 10 normal preg-

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Concentration of DHEA, c-AMP, and free estriol in maternal and cord plasma

Parameter	Initial labour	During expulsion	2 h after delivery	48 h after delivery	Cord artery	Cord vein
DHEA (µg/100 ml)						
Free	2.7± 1.1	3.5± 0.9	3.3± 0.7	2.6± 1.5	8.7± 4.4	5.7± 2.2
Sulfatide	48.3± 20.4	51.3± 19.7	38.2± 14.5	40.8± 18.0	112.8± 47.5	73.4± 34.2
Sulfate	32.9± 8.5	42.4± 14.4	19.0± 7.2	26.4± 8.3	80.4± 46.2	45.2± 6.7
c-AMP (pMol/ml)	11.2± 2.8	21.0± 6.4	16.0± 4.1	14.1± 4.7	31.4± 11.5	30.3± 12.2
Estriol (ng/ml)	10.2± 4.3	4.0± 1.9	2.0± 1.1	0.2± 0.09	5.7± 2.7	12.1± 6.7

nant women near term, the above values indicate a distinct increase in adrenocortical activity. The enhanced biosynthesis of primarily sulfoconjugated DHEA subsided after delivery, the levels of total DHEA falling to 60.5 µg/100 ml and 69.8 µg/100 ml 2 and 48 h resp. later. That the fetal adrenal provides a substantial portion of DHEA for placental biosynthesis of estriol[3] may be gathered from comparatively high concentrations of total DHEA in umbilical cord arterial plasma (201.9 µg/100 ml), the levels agreeing with those reported by SIMMER et al.[3]. Concerning the distribution of DHEA upon the fractions of free and sulfoconjugated steroids, the fraction of steroid sulfatides contained between 52.7% and 63.1% of assayed DHEA, followed by 31.4–43.6% in the fraction of steroid sulfates, and 3.2–5.5% in the fraction of free steroids. Although the instability of steroid sulfatides[4,5] may have affected these figures – in previous experiments the fraction of steroid sulfatides comprised roughly 80% of total DHEA in peripheral plasma of normal subjects – it can be concluded that the fetal adrenal also secretes DHEA sulfatide.

With regard to the assumed interrelationship between DHEA and c-AMP[8], the present results reveal a substantial rise of plasma c-AMP during the expulsion period, followed by a rapid decrease after delivery. Concomitant with the high concentrations of DHEA in umbilical cord arterial (or venous) plasma also elevated levels of c-AMP were obtained, supporting the contention that DHEA – especially as the sulfatide – exerts a definite influence upon plasma c-AMP. Regarding the nucleotide levels reported here, it should be pointed out that heparinized plasma was used throughout as in preceding investigations[8]. Hence, these concentrations cannot be compared with those gained with EDTA-treated plasma, which yields higher levels of c-AMP due to immediate inhibition of phosphodiesterase, as will be seen in a forthcoming communication.

When free estriol was determined in the various samples, a decline was observed in the expulsion period, which may be attributed to a reduced biosynthesis of placental estrogens. Whether an insufficient supply of C_{19}-steroid precursors is responsible for this decrease, or an impaired metabolic activity of placental tissue, remains to be seen. Within 48 h the maternal plasma concentration of free estriol had returned to non-pregnancy levels. In contrast to SCHILD et al.[13], who could not verify significant differences in the estriol content of umbilical cord arterial and venous blood, the higher levels of free estriol in the venous blood, shown in the Table, might very well hint at its placental origin.

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Sexual Steroids and Monoamine Metabolism During Gestation[1]

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Summary. The experiments show influence of progesterone and estradiol on regulation of enzymes of monoamine metabolism, MAO and COMT during pregnancy. Both the hormones inhibit enzymes MAO and COMT in the adrenals when determined at 0 h parturition. Estradiol has stronger inhibitory effect than progesterone. The results provide evidence for important endocrine implication during pregnancy for processes of monoamine regulation.

The role of sexual steroids in regulation of monoamine metabolism has been a subject of great interest recently. Hormones can modify the physiological disposition of labelled amines in rat uterus[2,3]. Progesterone increases monoamine oxidase (MAO) in the uterus during estrus[4,5]. Cyclic variations in progesterone production by adrenals and ovaries directly affect monoamine metabolism[6,7]. In the rat, estradiol inhibits MAO and progesterone seems to stimulate the enzyme[8–11]. The studies in the past have been mainly devoted to normal or cyclic animals, but hardly any study has been performed during pregnancy. The present study reports the effects of estradiol and progesterone on enzymes MAO and catechol-*O*-methyltransferase (COMT) during late pregnancy and parturition.

Materials and methods. White Sherman rats were used in all the study. The females were made pregnant as described previously and kept at 21 °C with natural night