produce renin and suggests a fetal source of renin in cord plasma; however, we cannot rule out that part of the renin in cord plasma; derives from uterine structures, especially from the placenta which contains a high amount of renin[12]. In this context it is of interest that PRC in amniotic fluid exceeds PRC in fetal and maternal plasma by far[3].

b) 3 to 48 h after delivery PRC in peripheral venous plasma of the infants was higher (27.0 ± 2.5) than in cord plasma. In 3 infants 2 determinations of PRC were done in the first 48 h after delivery; PRC between 5 to 7 h post partum was higher than between 23 to 48 h (mean decrease 17%). At the present time we have no explanation for the high PRC at delivery. Water loss[13] and reduction of extracellular volume[14] may account for the high PRC in the postnatal period; the 24 h fasting period to which the infants were submitted might intensify these effects and thus mediate the shortlasting further increase of PRC in the first 48 h after delivery; however, in our study the maximum of PRC did not coincide with the maximal weight loss of the newborns. 3 to 10 days post partum PRC was significantly lower (8.4 ± 2.8) than during the 3–48 h period. In infants between 3–24 months[15] PRC had further decreased (3.42 ± 0.50; $n = 46$; $p < 0.0005$ vs. 3–10 days period). It is of interest that in a recent report the aldosterone plasma level has been found extremely high in cord plasma and in peripheral venous plasma of infants in the first 3 days after delivery[16]. The high plasma aldosterone might be secondary to the increased PRC.

c) In mothers, PRC during labor was significantly higher (7.7 ± 1.0; $p < 0.0005$) than in normal subjects (recumbent 0.98 ± 0.12; upright 2.12 ± 0.22; $n = 22$; age 20–35 years; no significant differences between males and females). In 4 women studied 6 days after delivery PRC had decreased to 3.2 ± 1.5 ($p < 0.025$ vs. PRC during labor). Our findings are consistent with previous studies reporting elevated PRC[3,17] and PRA[4,6] during labor and immediately post partum[5]; PRC had returned to the normal range 2–4 days after delivery[17]; 7 days post partum PRA had normalized in one study[5] and was found still elevated in another report, returning to normal within 6 weeks[4]. It seems likely that, besides methodological reasons (PRC-PRA), small differences in sampling and postpartal conditions (diet, nursing) cause the differential results[18].

Zusammenfassung. Die Plasma-Renin-Konzentration (PRC) Neugeborener ist höher als die ihrer Mütter, verdoppelt sich innerhalb 48 h und sinkt bis zum 10. Tag unter den Geburtswert. Die PRC der Mütter ist bei der Entbindung sowie am 6. Tag post partum trotz Absinkens auf 50% des Ausgangswertes höher als bei nicht-graviden Kontrollen.

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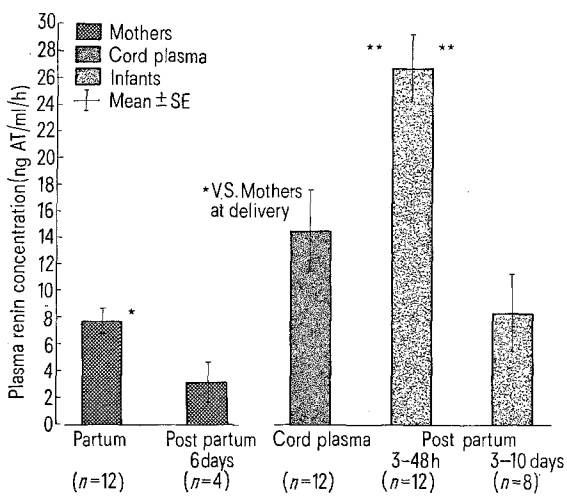

Plasma renin concentration (PRC) at delivery and during the newborn period in humans. $*p < 0.025$; $**p < 0.0005$.

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Oxidative Metabolism of the Limbic System in Prepuberal Rats

The limbic system presents variations in the consumption of oxygen in female rats in relation to the sexual cycle. SCHIAFFINI et al.[1] have shown that it is high during the estrous phase and that it decreases during the diestrous, whereas the contrary happens in the hippocampus. In males, the consumption of the amygdala is inferior to that of the hippocampus, and studies realized in vitro suggest a possible relationship between the oxidative metabolism of the limbic system and the pituitary gonadotrophins[2].

The oxidative metabolism of the limbic system in females is affected by the ovariectomy[3], experimental diabetes[4] and postnatal treatment with testosterone[5]. In this paper we have studied the oxidative metabolism in prepuberal rats of both sexes as well as their alterations after modifying those mechanisms that control the secretion of gonadotrophins by administering testosterone to the females or castrating the males in the first days of life[6].

Material and methods. We have studied the oxidative metabolism of the amygdala, hippocampus, and the hypothalamus of 21-day-old Wistar rats, pertaining to the following experimental groups: a) control females; b) control males; c) females, 5 days old, treated with 100 µg of testosterone propionate, in oil solution; d) males castrated 2 or 7 days after birth.

Table I. Oxidative activity of amygdala, hippocampus and hypothalamus in prepuberal rats

Groups	Amygdala		Hippocampus		P [c]	Hypothalamus	
Female	0.862 ± 0.29 [a]	17 [b]	1.152 ± 0.28	16	< 0.01	1.238 ± 0.26	18
Male	1.335 ± 0.43	18	1.288 ± 0.58	20	N.S.	1.322 ± 0.39	21
Female + TP on day 5	1.024 ± 0.47	18	1.348 ± 0.17	16	< 0.02	1.145 ± 0.43	18
Male castrated on day 2	0.936 ± 0.24	10	1.267 ± 0.30	13	< 0.01	1.103 ± 0.27	14

[a] Mean $\pm$ standard deviation expressed in μl O_2/mg wet tissue/h. [b] Number of determinations. [c] P between amygdala and hippocampus.

Table II. Pairwise test.

	Female (0.862)	Male (1.335)	Female + TP on day 5 (1.024)	Male castrated on day 2 (0.936)
Female	—			
Male	0.473 [a]	—		
Female + TP on day 5.	0.162	0.311 [a]	—	
Male castrated on day 2.	0.074	0.399 [a]	0.088	—

[a] $P < 0.01$. Differences in the oxidative metabolism of the amygdala between the different groups.

When the animals were 21 days old, they were killed by decapitation and the amygdala, hippocampus and hypothalamus were dissected. The consumption of oxygen was determined by Warburg manometry in vessels of 12–15 ml capacity, containing 3 ml of Krebs Ringer phosphate buffer pH 7.4 and 7.7. mM glucose. The central wall of the vessel contained 0.2 ml of saturated NaOH solution. The vessels were gased for 5 min with 100% O_2; after 10 min, for the equilibrium of the system, the study was executed at 37 °C, 120 beats per min for 1 h.

The results are expressed as μl O_2/mg wt tissue/h. The analysis of the data was carried out using Student's t-test, the analysis of the variance and the Pairwise test[7].

Results. Table I expresses the oxygen consumption per amygdala, hippocampus, and hypothalamus in the different groups of animals. The consumption of oxygen is greater in the hippocampus than in the amydgala in female rats ($P < 0.01$), in females treated with testosterone ($P < 0.02$) and in the males that had been castrated 2 days after birth ($P < 0.01$). The differences between the amygdala and the hippocampus do not appear in the males.

The analysis of variance of the consumption of oxygen by the different tissues of the 4 experimental groups shows that differences appear only for the amygdala ($P < 0.01$) and not for the hippocampus or hypothalamus.

The differences in the consumption of oxygen by the amygdala in the 4 experimental groups were analyzed by the Pairwise test and are expressed in Table II. The consumption is greater in the males than in the females, the females treated with testosterone, or males castrated 2 days after birth. Between these last 3 groups, there are no significant differences. We have also studied the effects of castration on the 7th day in males. In this group there is also a decrease in the oxidative metabolism of the amygdala ($X + D.S. = 1.000 \pm 0.28$) in relation to the control animals.

Discussion. In the prepuberal males, the consumption of oyxgen by the amygdala and hippocampus is similar, while in the adult animals the consumption is lower in the amygdala than in the hipopcampus[2]. The difference between adult males and prepuberal males might be due to the different plasmatic levels of LH and FSH[8].

In prepuberal intact females, androgenized females and males castrated 2 days after birth, the consumption of oxygen by the amygdala is less than by the hippocampus, similar to what happens in adult females in diestrus[1].

The consumption of oxygen by the amygdala is greater in males than in females at 21 days. Castration in males (days 2–7) produces a decline in the consumption of oxygen by the amygdala, which reaches a rate similar to that of the females. On the contrary, in adult males[2] as well as in females[3] castration is followed by an increase in the consumption of oxygen by the amygdala. The different effects of castration on the oxidative metabolism of amygdala cannot be explained by a different pituitary response[9,10].

Resumen. El consumo de oxígeno por amigdala, en ratas prepúberes, es mayor en machos que en hembras, hembras tratadas en el día 5° con propionato de testosterona y machos castrados postnatalmente. En los machos prepúberes, la castración origina una disminución en el consumo de oxígeno por amígdala.

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22 March 1972.

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