was modified after birth, bringing about a decrease in palmitic acid and an increase in stearic acid (fig. 2).
Contrary to TG, the linoleic acid content increased progressively during the 1st post-natal week, despite a large supply from the maternal milk. On the contrary, arachidonic acid content was doubled during the 1st day (fig. 2). As the latter acid is derived from the former, it can be deduced that strong stimulation of its synthesis occurs just after birth. Arachidonic acid is the precursor of prostaglandin E_2 whose tissue level has been found to increase 10-fold on the 2nd post-natal day[8]. As PGE_2 has an antilipolytic effect on rat brown adipocytes by inhibiting adenylate cyclase[9], this phenomenon, combined with an increase in lipoprotein lipase activity[10] could favour TG accumulation in the tissue during the first postnatal days.
Except for an increase in fatty acid unsaturation up to day 3 (fig. 1), cold exposure had no significant effect on PL fatty acid composition during the first 2 post-natal weeks. However, at the end of the 3rd week, a higher level of linoleic acid was observed, whereas the levels of palmitic and oleic acids were lower (fig. 2). Similar variations have been observed following cold-acclimation of the adult rat[4].
Conclusion. It can be concluded that in the early postnatal period, the large increase in lipid content of BAT, which is reduced in cold-exposed rats, would mainly be due to the uptake of fatty acids provided by maternal milk. Cold exposure has only a weak effect on the fatty acid composition of both triglycerides and phospholipids up till the end of the 2nd week. However, during the 3rd week, cold-dependent differences are observed, showing that the involutional process occurring in the brown fat of a growing rat could be stopped by cold-exposure.

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Low ambient oxygen tolerance in some freshwater teleosts[1]

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Summary. Measurements of the asphyxial oxygen level for *Rhinomugil corsula, Tilapia mossambica, Puntius sarana* and *Carassius auratus* at 30 and 35 °C revealed that at 35 °C the lethal oxygen level was higher for *T. mossambica* and *P. sarana* and lower for *R. corsula*, but it remained the same for *C. auratus* at 30 and 35 °C.

The importance of adequate ambient oxygen in an aquatic environment cannot be over-emphasised. Studies of the dissolved oxygen requirement of several fishes at different ambient oxygen concentrations have shown that most of the fishes have the ability to sustain complete or partial lack of oxygen[3-9]. But information on low ambient oxygen tolerance in fishes is lacking especially in the case of tropical species which are more likely to be exposed to conditions of low oxygen and high temperatures, in view of their ecophysiological characteristics[10].
Four freshwater teleosts viz. mullet, *Rhinomugil corsula* (Hamilton), cichlid, *Tilapia mossambica* (Peters), minor carp, *Puntius sarana* (Cuvier and Valenciennes) and goldfish, *Carassius auratus* (Linnaeus) were acclimated to freshwater at 30±0.5 °C or 35±0.5 °C for at least 15 days before the experiment. A modification of Fry's respirometer (capacity 3 l) was used and the design of the respirometer was such that the diffusion of gases into and out of water in the respirometer was minimized[11]. The experimental fish was exposed to oxygenated water (air saturated) and the dissolved oxygen was reduced by the respiration of the fish until it was asphixiated (loss of equilibrium). Thus, asphyxial oxygen concentration is the low lethal level of oxygen, below which fish cannot survive. Winkler's technique[12] was followed for the estimation of dissolved oxygen in the water samples.
The asphyxial oxygen concentration for the 4 species tested are given in the table. The species tested can be arranged in the decreasing order of tolerance of low oxygen: *C. auratus, T. mossambica, P. sarana* and *R. corsula* at 30 °C. At 35 °C, the order of tolerance changed slightly, the position of *T. mossambica* and *P. sarana* being reversed. It is significant that the order of hypoxic tolerance is the same as that of the anaerobic abilities as judged from the magnitude of the respiratory quotients[7-9].
The temperature effect (30-35 °C) on the asphyxial oxygen level is not evident. But it is noted that the lethal oxygen level is higher at 35 °C for *T. mossambica* and *P. sarana*, though the difference is only statistically significant ($p < 0.05$) for *T. mossambica*. The mean value for *R. corsula* is lower at 35 °C (table) but it is statistically not significant ($p < 0.05$). Since in all the experiments, after asphyxiation,

Asphyxial oxygen concentration (mg O_2/l) of 4 freshwater teleosts acclimated to 30 and 35 °C and tested at the acclimation temperature

Species	30 °C	35 °C
Rhinomugil corsula	0.86±0.018 (16)	0.84±0.015 (10)
Tilapia mossambica	0.36±0.071 (9)	0.56±0.015 (11)
Puntius sarana	0.41±0.025 (10)	0.49±0.250 (10)
Carassius auratus	0.28±0.015 (8)	0.28±0.236 (9)

The values in parentheses indicate the number of determinations. In each case ± SE is indicated.

the fish revived subsequently in air-saturated water, it became evident that the anaerobic ability to survive hypoxia is greater in *C. auratus* than in *T. mossambica, P. sarana* and *R. corsula.* However, only observations at other temperatures can give the full extent of temperature influence on the hypoxic tolerance of the fish. When oxygen was being reduced in a respirometer, some fish were seen to become more passive and some more active, thereby establishing a dichotomy in behavior of fishes at low oxygen levels[10,13]. The passive fish (e.g. *T. mossambica*[10] and *Chanos chanos*[13]) are also likely to escape hypoxic waters and reach oxygen-rich water if available. The full capacity for low oxygen tolerance can perhaps be known only if the fishes concerned are studied after acclimating them to low oxygen. But in the few known cases, low oxygen acclimation increased blood oxygen capacity and also efficiency in the utilization of oxygen, but not oxygen tolerance and anaerobic capacity[6,14,15].

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Voltage transients during ionic substitution in renal cortical tubules[1]

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Summary. A voltage transient is described which is found during proximal tubular perfusion with impermeant cation or anion salt solutions in the rat. It was shown that the magnitude of transepithelial diffusion potentials depended on luminal hydrostatic pressure, suggesting that the observed transients might be the consequence of the enlargement of ionic pathways by tubular dilatation. Thus, when reporting PD values, care should be taken to define the pressure levels at which measurements were performed.

When studying the transepithelial PD (potential difference) in the mammalian proximal tubule, it is important to ascertain that the microelectrode tip is within the lumen when measurements are made[3]. One of the methods used for this localization is luminal perfusion with impermeant cations. Since the paracellular pathway is the main passive ion path across the epithelium, ion concentration gradients across the epithelium generate diffusion potentials. The proximal tubule is highly permeable both to Na and Cl[4]. When Na is replaced by a chloride salt of a less permeant cation, like choline or magnesium, the tubular lumen is rendered positive due to the Na concentration gradient that is established. This potential shift is obviously detected only when the microelectrode tip is correctly localized in the tubular lumen. In an earlier paper we studied proximal transtubular PD in the rat using this localization method[5]. In these studies, we noted a positive voltage transient at the start of perfusion with isosmotic choline chloride solutions that has not been reported before. The present paper analyses the origin of this voltage transient.

Material and methods. Studies were performed on male rats weighing between 250 and 350 g. Anesthesia was induced by i.p. injection of pentobarbitone (40 mg/kg b.wt). The rats received an infusion of 3% mannitol in saline at 0.08 ml/min during the experiments. The animal preparation as well as the micropuncture and electrical measurement techniques used have been described previously[5]. The microelectrodes used were: a) Single and double-barrelled Ling-Gerard microelectrodes with tip diameter less than 0.5 µm filled by boiling with 3 M KCl solution to which

Transepithelial PD and voltage transients during luminal microperfusion of rat proximal tubule

Perfusion	Microelectrodes	PD max (mV)	PD st (mV)	△PD (mV)	n
Choline Cl iso	Ling-Gerard	16.8±0.79	9.9±0.79	6.9±0.50*	61
Choline Cl iso	Ringer-agar	14.3±0.41	9.4±0.42	4.9±0.23*	117
Choline Cl 15 mM	Ringer-agar	-	0.66±0.21	-	19
$MgCl_2$	Ringer-agar	8.0±0.44	4.4±0.43	3.6±0.23*	38
Na_2SO_4	Ringer-agar	−5.8±0.19	−1.6±0.13	−4.2±0.22*	58
Sodium cyclamate	Ringer-agar	−7.2±0.50	−4.7±0.38	−2.5±0.26*	18

PD max, peak value of transepithelial voltage including transient; PD st, PD during continuous perfusions; △PD, value of the voltage transient (PD max - PD st); Sign (+/−) denotes luminal polarity. *$p < 0.01$ (difference with $\Delta = 0$). n, number of perfusions.