prolactin[21]. Moreover the lack of 20α-HSD activity in the newly-formed set of C.L. observed in rats treated with estrogen alone (E_{15}) as well as with the association progesterone and estrogen (P + E) may be explained by the following facts: (a) estrogen alone, or combined with progesterone, more than progesterone alone, inhibit the secretion and/or the release of LH[18,22]; (b) estrogen explains a luteotropic action through the release of prolactin[14].

On the other hand, however, we could observe the appearance of the 20α-HSD activity in C.L. of T_{300} treated animals although testosterone inhibits the LH release[23].

The fact that the LH secretion and/or release is depressed by estrogen to a greater extent than by progesterone may be confirmed by the disappearance of both 3β-HSD and G-6-PD from the thecal cells and of G-6-PD from the interstitium in the E_{15} treated group. The dependence of these ovarian activities[3,8,9] and of the follicular and interstitial growth[24] on gonadotrophic stimulus is well known. Besides, interstitial cell inhibition has been morphologically noted to a greater extent in estrogen than in progesterone treated animals[17].

In our experiments, lack of both enzymes has been noted in the thecal cells of P + E and T_{300} treated animals. Moreover, in interstitial cells, G-6-PD activity was reduced or disappeared more frequently than the 3β-HSD activity. In this connection a direct correlation between the level of G-6-PD and the rate of steroidogenesis in the ovary has been noted[3].

Riassunto. La valutazione istochimica delle attività 3β-idrossisteroide deidrogenasica (3β-HSD), glucoso-6-fosfato deidrogenasica (G-6-PD) e 20α-idrossisteroide deidrogenasica (20α-HSD) a livello dell'ovaio di ratte trattate per 20 giorni con progesterone, testosterone, estradiolo o con l'associazione progesterone-estradiolo, permette di rilevare che, a dosi inibenti l'ovulazione, solo negli animali trattati con estradiolo o con progesterone ed estradiolo si inibisce la comparsa dell'attività 20α-HSD nei corpi lutei (C.L.) e si riduce quella 3β-HSD e G-6-PD nei follicoli e nella interstiziale.

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The Carbohydrate Metabolism Accompanying Intoxication by Aluminium Salts in the Rat

The effect of aluminium on mammal organism is not yet fully understood. It was found that intoxication with aluminium salts is accompanied by changes in metabolism of phosphorus compounds[1,2]. The common cause of serious disturbances is probably the formation of insoluble aluminium phosphate in the intestinal tract. This leads to the enhanced excretion of phosphorus from the organism and its negative balance. It was also found that, after peroral application of aluminium salts, the incorporation of intragastrically applied ^{32}P into the blood, liver, brain, kidney, spleen, muscle tissue and femur was decreased. Similarly the excretion of ^{32}P in the urine was lowered, while its excretion by the faeces was enhanced[3]. In the same paper it was confirmed that, during intoxication with aluminium salts, the levels of ATP decrease, while the levels of ADP and AMP increase.

It can therefore be supposed that application of increased doses of aluminium salts will influence the metabolism of carbohydrates. The findings of inhibition of glucose absorption in intestinal tract with aluminium salts[4] seems to confirm this view. We therefore studied some parameters of glycide metabolism in rats after the application of increased doses of aluminium chloride.

Two groups of white male rats, strain Wistar, weighing 175 ± 10 g were given the basal Larsen diet and water ad libitum, the control group received no aluminium, but the experimental group were given 200 mg aluminium/kg body weight incorporated into normal diet daily.

The Larsen diet consisted of: 622.6 g wheaten flower, 108.8 g dried milk, 163.3 g caseine, 32.7 g dried trefoil, 16.45 g calcium carbonate, 47.2 g margarine, 7.0 g fish liver oil, 2.4 g sodium chloride and tracer elements added.

Aluminium was added to the diet in the form of aluminium chloride. The extent of aluminium absorbed is about 10%. The experiment lasted 18 days. The weight of the rats under aluminium application decreased significantly ($P < 0.001$) compared with the control group, but neither the groups appeared sick. The last portion of diet and also of aluminium was given 24 h before decapitation of the animals.

The blood glucose was estimated according to SOMOGYI[5], liver and muscle glycogen according to GOOD[6], pyruvic acid in blood and liver following FRIEDEMANN and HAUGEN[7], lactic acid in blood, liver and muscle according to BARKER and SUMMERSON[8] and coenzyme A levels in liver according to HANDSCHUMACHER et al[9].

The results are summarized in the Table. The most pronounced change was the decrease of glycogen concen-

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Influence of aluminium intoxication on experimental rats

Values estimated	Control group	Experimental group	Significance
No. of rats	8	8	
Blood glucose mg%	109.60 ± 4.25	103.30 ± 2.23	$P > 0.05$
Glycogen			
Liver g%	2.530 ± 0.085	0.309 ± 0.030	$P < 0.001$
Muscle g%	0.442 ± 0.028	0.271 ± 0.023	$P < 0.001$
Lactic acid			
Blood mg%	13.72 ± 0.76	13.55 ± 0.66	$P > 0.05$
Liver mg%	11.89 ± 0.75	15.36 ± 0.72	$P < 0.01$
Muscle mg%	32.36 ± 3.50	51.61 ± 2.80	$P < 0.001$
Pyruvic acid			
Blood mg%	0.265 ± 0.012	0.366 ± 0.019	$P < 0.001$
Liver mg%	0.296 ± 0.002	0.400 ± 0.021	$P < 0.001$
Coenzyme A liver	17.27 ± 1.03	5.05 ± 0.27	$P < 0.001$

tration in liver, while its level in muscle was decreased only slightly though still significantly ($P < 0.001$). The glucose and lactate level in blood did not change significantly. However, the level of lactic acid in liver and muscle was increased. The level of pyruvic acid increased too, mostly in liver. The coenzyme A values are expressed in mg of acetylated 4-aminoazo-benzene related to 100 g of fresh liver tissue. These levels are strongly decreased in the experimental group ($P < 0.001$).

These results seem to point to a disturbance of glycide metabolism accompanying increased supply of aluminium salts. The most pronounced changes were the decrease of glycogen concentration in liver and increase of pyruvic and lactic acids levels in the same tissue. These changes have perhaps a common cause, i.e. the disturbance in phosphorus metabolism provoked by excess doses of aluminium salts. Therefore it can be assumed that there is decreased glucose absorption from the gut. The results correlate well with the other experiments in which the incorporation of ^{32}P into various phosphate fractions under the influence of aluminium was studied. The incorporation of ^{32}P into phospholipids, RNA and DNA in experimental animals was found to be significantly lowered[3].

Zusammenfassung. Bei Intoxikation von Versuchsratten durch Aluminiumsalze wurden Störungen im Glyzid-Metabolismus festgestellt. Er ergab sich ein Glykogenrückfall in Leber und Muskelgewebe und eine Erhöhung des Brenztraubensäure- und Milchsäurespiegels. Es wird angenommen, dass die beobachteten Veränderungen mit Störungen des Phosphormetabolismus und der Phosphorilationsreaktionen durch die Aluminiumtoxikation zusammenhängen.

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The Mode of Timing of DNA Replication and of Mitosis in Cultured Animal Cells[1]

It has been postulated repeatedly, with little or no support from experimental evidence, that the DNA-synthesis period (*S*-period), the *G*2-period and the period of observable mitosis (*M*-period) of the mitotic cycle have each fixed durations, and that therefore, differences in generation time are due to expansion or contraction of the *G*1-period alone[2–5]. Evidence in favour of an alternative mode of timing of DNA replication and of mitosis has been obtained in carefully planned and executed experiments with 2 established (heteroploid) mouse cell strains, P815Y and L929, grown in fluid suspension at many different exponential rates in chemostat and cytogenerator[6–8]. Observations indicating that exposure of animal cells to exogenous thymidine alters the pre-established rate of DNA synthesis and subsequently modifies the normal chronology and duration of the cell-division-cycle, forced me to abandon the standard pro-

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