proteins on plasmin. HOWELL[10] is of the opinion that apart from inhibition of plasmin and activation of plasminogen, lipoproteins may also affect the level of the proactivator, adsorption of the activator and plasminogen on fibrin, and the diffusion of the activator into the clot. It is an interesting fact that, in all the cases investigated, euglobulin fibrinolysis inhibition depended on the amount of β-lipoproteins added.

The effects observed in vitro were confirmed by the further investigations on parturients and puerperants. A definite connection between the serum β-lipoprotein level and the plasma euglobulin fibrinolysis time in women during labour and confinement (Figure 2) was found. During labour, euglobulin fibrinolysis was inhibited and the β-lipoprotein concentration in the serum was raised compared to the mean control values. A similar lowering of fibrinolytic activity in parturients was also demonstrated by ELSNER[6]. BURSTEIN, on the other hand, observed a high level of β-lipoproteins in parturients and puerperants. HOWELL[10], in investigations on Europeans and Negroes, found that the latter had a higher fibrinolytic activity and a lower β-lipoprotein level.

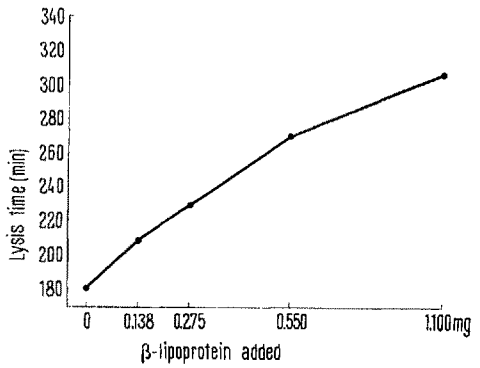

Fig. 1. The effect of β-lipoproteins on euglobulin lysis time in vitro.

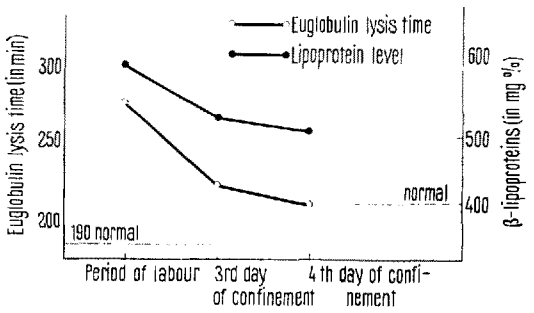

Fig. 2. Plasma euglobulin fibrinolysis time and the behaviour of β-lipoproteins in women during the period of labour and confinement.

It should be emphasized that, as the serum β-lipoprotein level in puerperants fell, the euglobulin fibrinolytic activity increased. This appears to have been substantiated by the results of our investigations in vitro. Our results concur with the observations of other investigators who have drawn attention to the relationship between β-lipoproteins and fibrinolysis[2,4,10,11]. In hyperlipaemia, fibrinolysis inhibition together with an increase in antiplasmin activity and β-lipoprotein concentration was observed[12,13]. It is also known that in alimentary lipaemia the fibrinolytic activity falls[14,15].

The investigations of the authors mentioned above and the results of our studies indicate that β-lipoproteins are to a certain extent responsible for the inhibition of fibrinolysis in vivo. Earlier investigations[3] proved that 10% of the antiplasmin activity in the plasma of healthy persons is due to β-lipoproteins. Though this is not a high % in physiological conditions, in pathological conditions, when it is combined with an increase in β-lipoproteins, it may be considerably higher and thus cause inhibition of the fibrinolytic system of the circulating blood. The plasminogen level fell slightly as the β-lipoprotein concentration decreased and the fibrinolytic activity increased in the euglobulin fraction.

Zusammenfassung. Es wurde festgestellt, dass aus Serum isolierte β-lipoproteiden in vitro die Fibrinolyse der Plasma-Euglobuline hemmen. Da weiter festgestellt werden konnte, dass in Puerperium das Niveau der Serum-Lipoproteide sinkt, während gleichzeitig die Aktivität des Fibrinolyse-Systems zunimmt, wird vermutet, dass auch in vivo ein ähnlicher Prozess abläuft.

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Influence of Epoxides of Androstane Series on Some Effects of Cortisol

In our previous work some antiglucocorticoid properties of androgenic-anabolic inactive 1,2α-oxido-5α-androstan-3,17-dione (I) affecting mostly the glycide metabolism, were described[1]. We were therefore interested to learn in which way some alterations of the molecule of this type of steroid would influence the effect reported.

The effect of the following compounds was studied in 268 male rats of Wistar-Konárovice strain (180–220 g): androst-4-en-3,17-dione; 1,2α-oxido-5α-androstan-17β-ol-3-one[2]; 1,2α-oxido-4,6-androstadien-3,17-dione[3] (II); 4,5β-oxidoandrostan-3,17-dione[4]; 2,3α-oxido-5α-andro-

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The glycogen content and its fraction in rat liver after 10 days' administration of compound II, cortisol-acetate, and their combination. The differences are significant at $P < 0.05$

Group	No. of animals	Total glycogen µg/100 mg tissue	Significance	Insoluble glycogen µg/100 mg tissue	Significance	Soluble glycogen µg/100 mg tissue	Significance
A – oil	9	452 ± 103	A < C	131 ± 48		325 ± 85	A < C
B – compound II	8	396 ± 111	B < C and D	125 ± 32	∅	279 ± 94	B < C and D
C – cortisol	9	1959 ± 666	C > D	165 ± 34	∅	1794 ± 634	C > D
D – cortisol and compound II	9	757 ± 243		128 ± 29	∅	634 ± 237	

stan-17-one[6]; 1,2α-oxido-2β-brom-5α-androstan-3,17-dione; 1,2α-oxido-2β-chlor-5α-androstan-3,17-dione; and 1,2α-oxido-2β-chlorandrosta-4,6-dien-3,17-dione[6].

The 4 groups of rats received daily doses (A) of 0.4 ml olive oil, (B) 2 mg of tested substance p.o, (C) 2 mg of cortisol s.c., and (D) 2 mg both of compound studied and cortisol (all per 100 g body weight) for 10 consecutive days. The compounds were dissolved in oil, the used volume of which was equal for all the groups including controls (A). 24 h after the last injection and after 24 h of starvation, the animals were decapitated. Cholesterol[7], total lipemia and glucose level in the serum, and total glycogen and its fractions in the liver were determined as before[8]. The statistical evaluation was accomplished as previously[8]. The fiducial limits of the means are always mentioned.

From the above-mentioned steroids only compound II produced a positive effect. The Table shows that cortisol caused a marked increase in the total glycogen in the liver, mostly in its soluble fraction. This increase was inhibited by a simultaneous administration of compound II. In repeated experiments (4 times) a decrease in hypercholesterolemia, hyperglycemia and hyperlipemia in serum was observed in the groups treated with cortisol and compound II (D), which was not always statistically significant. Compound II alone did not affect the parameters followed. In the test according to HERSBERGER et al.[9], this steroid did not produce any androgenic-anabolic effect. The results reveal that compound II appears to be less active than compound I. Compound I alone decreased the soluble glycogen fraction in the liver, and in interaction with cortisol even the insoluble fraction was decreased[1].

On the basis of the fact that from the steroids studied only 2 compounds were active in interaction with cortisol, one may presume that this effect is connected with a rather specific structure and that it does not depend on the androgenic-anabolic activity. The transfer of oxide from position 1,2α or the introduction of chlorine and bromine in position 2β adversely affect this effect[10].

Zusammenfassung. Auf der Basis von Struktur-Aktivitätsuntersuchungen verschiedener Epoxyden der Androstanreihe konnte gezeigt werden, dass die Antiglucocorticoid-Eigenschaften solcher Verbindungen von einer besonders spezifischen Struktur abhängig sind.

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An Effect of DMSO on Post-Irradiation Saccharin Avoidance in Mice

Some interesting results were reported recently by Moos[1] regarding the protective effect of dimethyl sulfoxide (DMSO) against X-radiation in mice. The animals were treated with DMSO 5–10 min before exposure by immersing the major part of their tails in anhydrous DMSO for various lengths of time. One observed that when the subjects were exposed to total body irradiation with doses ranging between 700 and 760 R, 75–95% of the experimental animals survived over 30 days compared to 25–45% survivors in the water-treated control group. We also studied different aspects of the post-irradiation aversion to sodium saccharin in mice[2,3] during the time of this experiment. Though numerous experimental results have been published regarding this avoidance behavior[4–8], none has offered any satisfactory answers regarding the mechanism of these changes. LEVAN treated a group of mice with DMSO as described above, and then, subjecting these animals to the post-irradiation saccharin-water preference test, surprisingly found that the animals continued to prefer sodium saccharin solution to water after whole-body exposure to a total dosage of 450 R. This