matings along with the concentration of potassium in each type are given in the Table.

The results show that all HK \times HK matings resulted in HK progeny, whereas HK \times LK and LK \times LK resulted in both HK and LK types. This indicates that

Potassium type of progeny from matings of rams and ewes of various phenotypes

Ram		Ewes			
Identifi- cation No.	Potas- sium type	LK (9.27)		HK (25.20)	
		Progeny			
		LK	нк	LK	нк
G 75	HK (26.24)	2 (9.60)	2 (29.22)	_	11 (29.65)
G 423	HK (32.64)	1 (9.60)	1 (26.24)		10 (30.78)
G 475	HK (32.64)	2 (9.20)	4 (34.08)	_	3 (30.29)
G 988	LK (9.60)	2 (10.21)	2 (35.84)	6 (10.62)	3 (31.36)

Figures in parenthesis represent blood potassium concentration in mEq/l. The concentration in LK varies from 6.40-12.80 and in HK from 24.97-37.12.

HK is inherited as a simple recessive character and LK animals may be either homozygous or heterozygous. HK \times HK mating is essentially required to confirm whether HK is a recessive character, and since the number of this type of mating is fairly high in this experiment, the deficiency of an earlier study 2 on an American breed is, therefore, removed. This promotes confidence in the hypothesis that HK is a recessive character.

Résumé. Par le photomètre Flame la concentration du potassium dans le sang de 102 moutons marvaris (4 mâles, 49 femelles et leurs 49 descendants) a été mesuré. Sur ce nombre, 45 appartiennent au type HK (à forte concentration, moyenne 30,34 mEq/l – de 24,97 à 37,12) et 27 au type LK (à faible concentration, moyenne 9,72 mEq/l, – de 6,40 à 12,80). Le caractère HK est donc héréditaire récessif tandis que les individus de type LK sont peut-être homozygotes ou héterozygotes.

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Investigations on the Relation Between β -Lipoproteins and Plasma Euglobulin Fibrinolysis

Pappenhagen and co-workers 1 found that chylomicrons and lipoproteins of low density inhibit the fibrinolysis of plasma euglobulin. The elimination of chylomicrons from the serum of patients suffering from atherosclerosis causes, as was demonstrated by Sarkar in his investigations 2, an increase in the fibrinolytic activity of these sera. It has recently been reported that the β -lipoproteins isolated from human blood plasma inhibit the action of plasmin in pure systems 3.

RIDING and Ellis showed that a parallel rise in β -lipoprotein antiplasmin activity in the serum and in the cholesterol level occurs as pregnancy progresses. Burstein, however, found that there is a considerable rise in β -lipoproteins in the serum of parturients and puerperants. Some authors 6,7 have also reported that during pregnancy the fibrinolytic activity is lowered.

These data suggest that there is a connection between fibrinolytic activity and the β -lipoprotein level in the blood serum.

The aim of the investigations presented here was to determine the effect of β -lipoproteins on the fibrinolysis of the plasma euglobulin fraction in vitro, and to study the fibrinolytic activity in the euglobulin fraction and the β -lipoprotein level in the serum of women during and after labour.

Material and methods. β-lipoproteins obtained from the sera of 10 healthy women, aged 20-40 years, by the Burstein method⁵ were dissolved in a borate buffer at pH 7.4 in the initial volume of the serum. From the plasma of the same women, euglobulins were obtained by the Kowalski method⁸. Some of these euglobulins were dissolved in a borate buffer at pH 7.4 (controls) and the

remainder were dissolved in a buffer to which various concentrations of β -lipoprotein were added (1.1, 0.55, 0.275, 0.138 mg).

The euglobulins were coagulated with calcium chloride and the lysis time was determined. The homogeneity of the β -lipoproteins was confirmed by paper electrophoresis. Investigations on the euglobulin time, the level of β -lipoproteins during labour and confinement, and the values of plasminogen, were carried out on 49 women in the first stage of labour and the third and fourth day of puerperium.

Discussion. As Figure 1 shows, β -lipoproteins isolated from the serum and dissolved in a borate buffer, when added to the euglobulin fraction of the plasma, definitely lengthen the fibrinolysis time of the clot formed from that fraction. The results obtained in these investigations indicate an inhibition of the fibrinolysis by the β -lipoproteins. Our results confirm those of Pappenhagen and coworkers¹. Riding and Ellis⁴ and one of the authors of this paper³ demonstrated the inhibitive action of β -lipoproteins.

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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®. BURSTEIN, on the other hand, ob-

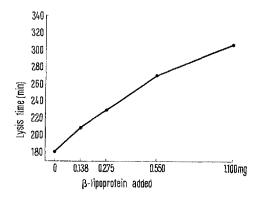


Fig. 1. The effect of β -lipoproteins on euglobulin lysis time in vitro.

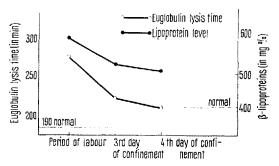


Fig. 2. Plasma euglobulin fibrinolysis time and the behaviour of β -lipoproteins in women during the period of labour and confinement.

served a high level of β -lipoproteins in parturients and puerperants. Howell¹⁰, in investigations on Europeans and Negroes, found that the latter had a higher fibrinolytic activity and a lower β -lipoprotein level.

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 14,16 .

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 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. 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; 1,2 α -oxido-5 α -androstan-17 β -ol-3-one 2 ; 1,2 α -oxido-4,6-androstadien-3,17-dione 3 (II); 4,5 β -oxidoandrostan-3,17-dione 4 ; 2,3 α -oxido-5 α -andro-

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