Table IV. Effect of crystalline insulin on glycogen content of diaphragm and $^{14}CO_2$ production from ^{14}C-U-glucose by diaphragms of control and 'meal fed' rats

Experimental group	Glycogen content of diaphragm [b]			$^{14}CO_2$ production from ^{14}C-U-glucose in vitro [c]		
	Without hormone mg/100 g wet weight	Insulin		Without hormone counts/min 100 mg wet weight	Insulin	
		mg/100 g wet weight	% of mean of groups without hormone		counts/min 100 mg wet weight	% of mean of groups without hormone
Control	274 ± 9.8 [a] (5)	745 ± 16.5 (5)	272	948.8 ± 91.4 (6)	1991.8 ± 177.6 (6)	210
'Meal fed'	298 ± 19.6 (6)	617 ± 32.3 [e] (6)	207	1241.7 ± 58.2 [d] (6)	1959.9 ± 151.0 (6)	158

[a] Mean values ± standard error of mean; number of rats in parentheses. [b] Diaphragms removed 45 min after injection of saline or insulin (0.2 U/kg). [c] Animals treated as above, diaphragms incubated for 60 min in 5 ml KRP buffer, pH 7.4, containing 25 μmoles glucose and 0.25 μC ^{14}C-U-glucose (no insulin added). Symbols for statistical differences between compared group averages: [d] ($P = 0.02$); [e] ($P < 0.01$).

has similarly been reported recently for hereditary obese[16], but not for goldthioglucose-treated obese mice[17]. It therefore appears that in the 'meal fed' rats a nutritionally induced regulatory mechanism is brought into play which influences the sensitivity of target organs to insulin and directs the hormone's metabolic effect towards adipose tissue. The nature of this regulatory mechanism is as yet unknown.

The increased sensitivity of adipose tissue to insulin may be an important factor in the mechanism of the greater synthetic capacity found in adipose tissue of meal fed' rats[18,19].

Zusammenfassung. Intraperitoneale Verabreichung von Insulin führte bei Ratten, die täglich 2 h gefüttert wurden, im Vergleich mit ad libitum gefütterten Kontrolltieren, zu einer grösseren Herabsetzung des Blutzuckerspiegels, einer höheren Fett- und Glykogensynthese im Fettgewebe, jedoch zu einer verminderten Glykogensynthese im Zwerchfell.

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The Effect of Endotoxin on the Lactic Acid Production in Pregnant and Non-Pregnant Rats

The effect of endotoxin on aerobic glycolysis has been examined in certain tumours[1,2], leucocytes[3–5], peritoneal macrophages, spleen[6], brain[2], kidney, mucosa of small intestine, thymocytes[7] and placenta[8,9]. Generally the action was found to be biphasic[5,10].

After an initial enhancement of aerobic glycolysis, a transitory notable inhibition set in, followed again by a hyperfunction. The course of these events is remarkably dose-dependent. The K-2 carcinoma[6] thymocytes, kidney, mucosa of small intestine[7], however, were endotoxin insensitive. Finally we found in placentas nothing but a permanent inhibition of aerobic glycolysis[8,9].

This report concerns a comparative study of lactic acid production of some tissues of pregnant and non-pregnant rats after endotoxin treatment.

Method. Rats of mixed breed (National Institute of Public Health) in day $17^1/_2$ to day $18^1/_2$ of pregnancy were used throughout. The endotoxin was extracted from *Serratia marcescens* by the method of BOIVIN and MESROBEANU[11]. In preliminary titrations in non-pregnant rats, the preparation was adjusted to contain one LD_{10}/ml. This dose was inoculated i.p. into pregnant and non-pregnant rats. Similarly pregnant and non-pregnant rats were given 1 ml saline by the same route for control purposes. The animals were killed by decapitation 24 h later. Leucocytes were obtained from peritoneal exudates produced by the i.p. injection of 20 ml sterile broth (bouillon) into each rat 6 h prior to decapitation. The peritoneal exudates contained $8–12 \cdot 10^7$ cells/ml. Bone

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Lactic acid production (μg/mg dry material/h for spleen, bone marrow, placenta and μg/10^7 cells/h for leucocytes) in the tested tissues of endotoxin- and saline-treated pregnant and non-pregnant rats 24 h after treatment. The numbers of samples are in brackets

Group	Treatment	Spleen		Bone marrow		Leucocyte		Placenta	
		$\bar{X}$	$s_{\bar{x}}$	$\bar{X}$	$s_{\bar{x}}$	$\bar{X}$	$s_{\bar{x}}$	$\bar{X}$	$s_{\bar{x}}$
Non-pregnant (NP.)	Saline (S.)	8.3 (11)	0.50	10.3 (5)	0.64	4.1 (5)	0.3	–	–
	Endotoxin (E.)	10.3 (15)	0.38	16.8 (5)	0.49	5.1 (5)	0.2	–	–
Pregnant (P.)	Saline (S.)	8.6 (8)	0.60	12.5 (5)	0.73	4.7 (5)	0.2	11.6 (20)	0.41
	Endotoxin (E.)	11.4 (10)	0.55	19.7 (5)	0.64	7.1 (5)	0.6	8.7 (14)	0.42
Difference between	NP.S. and NP.E.		$p < 0.01$		$p < 0.001$		$p < 0.05$		
	P.S. and P.E.		$p < 0.01$		$p < 0.001$		$p < 0.01$		$p \ll 0.001$
	NP.E. and P.E.		$p > 0.1$		$p < 0.01$		$p < 0.05$		
	NP.S. and P.S.				$p > 0.05$				

marrow samples were taken from the tibia and femur. Leucocytes and bone marrow cells from 2 animals were pooled. Spleens were studied separately for each individual animal. 3 pooled placentas per animal served as individual samples. Tissue samples were weighed and minced prior to their transfer into an adequate volume of Krebs-Henseleit solution containing 200 mg% glucose. The lactic acid production was measured by the method of DIESCHE and LÁSZLO[12]. Lactic acid production is expressed as μg/mg dry material/h and μg/10^7 cell/h for spleen, bone marrow, placenta and leucocytes, respectively.

Results. Lactic acid production of spleens, bone marrow cells, leucocytes and placentas of endotoxin treated pregnant and non-pregnant rats is shown in the Table.

When tested after 24 h following treatment with endotoxin, the lactic acid production of the placentas and of other tissues (spleen, bone marrow and leucocytes) exhibited a considerable difference. Aerobic glycolysis was inhibited in placentas, while stimulated in all tissues tested.

In endotoxin-treated animals, the rate of lactic acid production was significantly more enhanced in bone marrow cells and leucocytes of pregnant rats than in the non-pregnant ones.

Zusammenfassung. Die Milchsäureproduktion ist im Knochenmark und in den Leukozyten trächtiger Tiere infolge Endotoxineinwirkung signifikant höher als bei Kontrolltieren. Während sich die Milchsäureproduktion endotoxinbehandelter Plazenten infolge Dauerhemmung der aeroben Glykolyse völlig von allen untersuchten Geweben unterscheidet, bleibt der Sauerstoffverbrauch unverändert.

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Clotting Factors of the Primary Aqueous Humor of the Rabbit's Eye

According to several authors, primary aqueous humor (PAH) accelerates the clotting time of the whole blood and of the plasma[1,2] and contains various clotting factors, e.g. prothrombin[2], factor V and factor VII[2,3]. PANDOLFI and NILSSON[4] found that PAH contains plasminogen and proactivator but no measurable amounts of other clotting factors. The purpose of the present study was to characterize more precisely the nature of the clot-promoting substances of this fluid.

PAH was drawn from the anterior chamber of the rabbit's eye by means of a puncture with a s.c. needle. About 0.1–0.3 ml of the transparent fluid was obtained from each eye. Samples contaminated with blood were discarded.

The following investigations were performed:

(1) The influence of PAH on the recalcification time of fresh, human, platelet-poor plasma (PPP) was studied in the system: 0.1 ml of PPP + 0.1 ml of PAH (or 0.9% NaCl) + 0.1 ml of 0.025 *M* $CaCl_2$. The recalcification time of 99 control samples amounted on an average to 214 sec (range: 120–360 sec). The recalcification time of 124 samples containing PAH amounted on an average to 128 sec (range: 35–240 sec). All but 18 fluid samples significantly shortened the clotting time of the recalcified

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