

Binding of C<sup>14</sup>-L-tryptophan by human serum albumine and prealbumine

Protein	Concentration	Amino acid *	dpm in the undialyzed sample	dpm in the dialysate (mM)
Albumine	1.4 × 10 <sup>-3</sup>	C <sup>14</sup> -L-tryptophan	5293	4580
Albumine	7.0 × 10 <sup>-3</sup>	C <sup>14</sup> -L-tryptophan	5468	3062
Albumine	1.4 × 10 <sup>-2</sup>	C <sup>14</sup> -L-tryptophan	5814	2252
Prealbumine	1.4 × 10 <sup>-3</sup>	C <sup>14</sup> -L-tryptophan	5625	4856
γ-Globulin	1.4 × 10 <sup>-3</sup>	C <sup>14</sup> -L-tryptophan	5794	5772
Albumine	1.4 × 10 <sup>-3</sup>	C <sup>14</sup> -L-leucine	5559	5547

Each value is the average (and range) of 3 experiments. \* C<sup>14</sup>-L-tryptophan or C<sup>14</sup>-L-leucine were added at a molar concentration of 1.4 × 10<sup>-3</sup> with a specific activity of 50 μCi/mM.

The results of the ultrafiltration dialysis with purified human albumine and prealbumine have confirmed that L-tryptophan binds to both albumine and prealbumine fractions and have indicated that it has the same affinity for either fraction.

Since albumine is present in plasma at a much higher concentration than the other proteins, the results indicate that most of the tryptophan present in plasma is bound to albumine and suggest that the significance of the binding of tryptophan to the other protein fractions is limited under physiological conditions.

**Riassunto.** Sieri di uomo e di ratto sono stati incubati con C<sup>14</sup>-L-triptofano e sottoposti a elettroforesi su gel di poliaccrilamide. Nel siero umano la radioattività si è localizzata nelle frazioni albuminica, prealbuminica e

macroglobulinica. Nel siero di ratto il C<sup>14</sup>-L-triptofano si è localizzato nella zona delle albumine, α-globuline e macroglobuline.

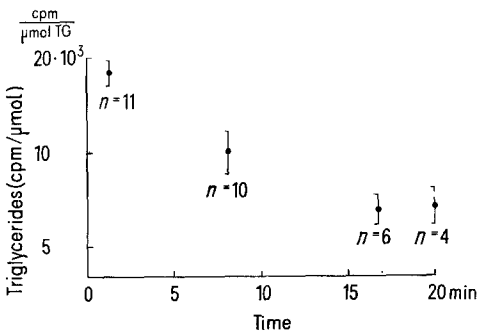
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Studies on the Turn-Over of Plasma Triglycerides using Triglyceride <sup>14</sup>C-labelled Chyle in Pregnant Rats

Several animal species, including man and the rat, display a hypertriglyceridaemia during the last week of pregnancy<sup>1</sup>. The rise in plasma triglycerides could result either from a rise in the output, or from a fall in the rate of removal from circulation. Some evidence for the latter hypothesis was given by OTWAY<sup>2</sup> and HAMOSH<sup>3</sup>, because they noted a reduction in the activity of the plasma-clearing lipase after the 19th day of pregnancy.



The specific activity of plasma triglycerides as a function of time after the i.v. injection of <sup>14</sup>C-1-palmitate labelled chyle. At zero time, 1 × 10<sup>6</sup> counts/min (recovered in the triglycerides of the injected chyle) per 300 g body weight were injected. Data are mean values ± standard errors for several estimations (The number of estimations is indicated by n). The turn-over time was calculated by regression analysis to be 17.3 ± 2.7 min.

The purpose of the present study was to evaluate the rate of plasma triglyceride removal from the circulation on the 21st day of pregnancy.

**Methods.** A thoracic duct cannulation had been performed on male Wistar rats according to the method of BOLLMAN<sup>4</sup>. The rats were then given 100–200 μC of <sup>14</sup>C-1-palmitate dissolved in 1 ml corn oil. The chyle was collected for 12 h in a flask containing citrate. The chyle was concentrated by centrifugation for 60 min at 70,000 ×g. The top layer was aspirated and resuspended in 0.9% NaCl. The chyle was kept at 4°C. All injection procedures were completed within 10 days after collection of chyle. The chyle was injected into the tail vein of pregnant Wistar rats (21 days) weighing 320 ± 10 g. At appropriate times, they were anesthetized with ether and blood drawn from the vena cava inferior. The blood and an aliquot of the injected chyle were extracted for lipids according to the method of FOLCH.<sup>5</sup> The triglycerides were solated by thin layer chromatography and assayed for quantity and radioactivity, and the specific activity was calculated. The disappearance rate of labelled chyle was calculated by linear regression analysis. The plasma volume of pregnant rats was measured by dye dilution technique, according to HAMILTON<sup>6</sup>. Details of the methods used have already been described<sup>7</sup>.

**Results.** All the values given were normalized to a constant injected dose (1 × 10<sup>6</sup> counts/min) of radioactivity (recovered in the triglyceride fraction of the chyle), and a body weight of 300 g. The Figure represents the specific activities of plasma triglycerides as a function of

time after the injection of labelled chyle at zero time. The turn-over time of plasma triglycerides was calculated to be  $17.3 \pm 2.7$  min. The concentration of plasma triglycerides is  $3.7 \pm 0.2$   $\mu$ moles triglyceride per ml plasma. The plasma volume is  $4.3 \pm 0.3$  ml per 100 g body weight. The turn-over rate of plasma triglycerides was computed to be  $2.8 \pm 0.7$   $\mu$ moles triglycerides per min. The injected chyle contained about 2  $\mu$ moles triglyceride per animal.

**Discussion.** The estimation of the turn-over rate of plasma triglycerides by the tracer technique is based upon the following assumptions: first, that the plasma triglycerides are regarded as a homogenous metabolic pool, second, that the turn-over of the labelled chyle triglyceride is representative of the entire plasma triglyceride fraction. These assumptions are surely not strictly correct, but are in accordance with the results obtained by other investigators<sup>8-10</sup>.

We estimated the turn-over rate of plasma triglycerides of pregnant rats to be 2.8  $\mu$ moles triglyceride per min per 300 g body weight. In non-pregnant rats the values for the turn-over rate of plasma triglycerides range from 0.5 (HAUDE<sup>6</sup>) to 0.9 (BAKER<sup>8</sup>) to 2.1 (LAURELL<sup>9</sup>) to 3.5 (BELFRAGE<sup>7</sup>); the data are expressed as  $\mu$ moles triglyceride per min standardized to 300 g body weight.

If we compare these data, we see that the rate for pregnant rats exceeds the rates for non-pregnant rats, except the rate determined by BELFRAGE<sup>7</sup>, which is slightly higher. We must consider, however, that the rate determined by BELFRAGE represents the maximal removing capacity of plasma triglycerides, and that this rate was found under unsteady state conditions, because 124 mg chylomicron lipid was injected. The injection of such an amount of triglycerides increased the pool by more than one magnitude of order!

It seemed to us, therefore, that a fall (compared with non-pregnant rats) in the rate of plasma triglyceride

removal from the circulation could not explain the hypertriglyceridaemia of pregnancy. Our results support the hypothesis of NAISMITH<sup>11</sup> and RICHARDSON<sup>1</sup> that a rise in hepatic lipogenesis is responsible for the hypertriglyceridaemia of pregnancy.

**Zusammenfassung.** Die Umsatzrate der Plasmatriglyceride trächtiger Ratten beträgt 2,8  $\mu$ Mol/min/300 g Körpergewicht. Da die Umsatzrate nichtträchtiger Tiere kleiner ist, sprechen die Befunde gegen eine Störung des Abbaues der Plasmatriglyceride während der Gravidität.

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## Respiratory Alkalosis in a Panting Lizard (*Sauromalus obesus*)

When the desert lizard *Sauromalus obesus* is exposed to temperatures between 43°C and 45°C, respiratory ventilation and respiratory evaporative water loss increase dramatically<sup>1</sup>. This respiratory response appears to be thermoregulatory (panting) and is apparently mediated by both peripheral and central components of the nervous system<sup>2</sup>. The increase in evaporative cooling during panting is sufficient to keep deep body temperature and brain temperature below an ambient temperature of 45°C for extended periods of time and has a greater cooling effect on the brain than on the remainder of the body<sup>3</sup>.

The ventilation necessary to support such significant evaporative cooling is greater than that required to satisfy metabolic demands for oxygen. If the additional ventilation passes over the gas exchange surface of the lungs, a reduction in the  $P_{CO_2}$  of the blood should occur, resulting in respiratory alkalosis. This situation is complicated by the fact that lizards, as well as other poikilotherms, regulate pH at a temperature-dependent set point. It appears that they regulate towards a constant alkalinity with respect to the neutral point of water which changes with temperature<sup>4-11</sup>. The purpose of this note is to establish whether the drive for temperature regulation through evaporative cooling or the maintenance of acid-base balance is the dominant regulatory mechanism during panting in the desert lizard *Sauromalus obesus*.

**Materials and methods.** The animals employed in these experiments were two specimens of a group of lizards which had been previously used to establish the non-panting relationship between pH and  $P_{CO_2}$  of carotid blood and body temperature<sup>11</sup>. Polyethylene catheters were inserted into the right carotid artery and the animals were maintained at 40°C for 3 days. Carotid blood was collected in heparinized capillary tubes directly from the catheter. Carotid blood  $P_{CO_2}$  and pH

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