haben wir die P/O-Quotienten der Leber- und Tumormitochondrien und ihre Streubreite aufgeführt.

Vergleicht man die P/O-Quotienten, so kann man bemerken, dass bei zunehmender Konzentration von 4-Benzylhydroxy-3,5-dijodbenzoesäure die P/O-Quotienten der Tumormitochondrien sich wesentlich schneller verringern als die der Lebermitochondrien. Die P/O-Quotienten der Tumormitochondrien betragen bei einer Konzentration von $5 \cdot 10^{-5} M$ nur noch $0{,}1 M$ und bei $10^{-4} M$ sind sie gleich Null, dagegen liegen die P/O-Quotienten der Lebermitochondrien bei einer Konzentration von $5 \cdot 10^{-5} M$ und bei $10^{-4} M$ noch über bzw. knapp unter 1.

Diese Ergebnisse, die die früher mit Chlorpromazin gemachten Erfahrungen prinzipiell bestätigen, lassen den Schluss zu, dass der oxydative Energiestoffwechsel von Tumorgeweben durch Chemotherapeutika selektiv beeinflusst werden kann.

*Summary.* The effects of 4-benzylhydroxy-3,5-diiodobenzoic acid on isolated mitochondria of WALKER carcinosarcoma 256 and liver tissue of the same animals were compared. It was shown that, by the same concentrations of the uncoupling halo-phenol, the oxidative phosphorylation of tumor mitochondria was impaired in a higher degree than that of liver mitochondria.

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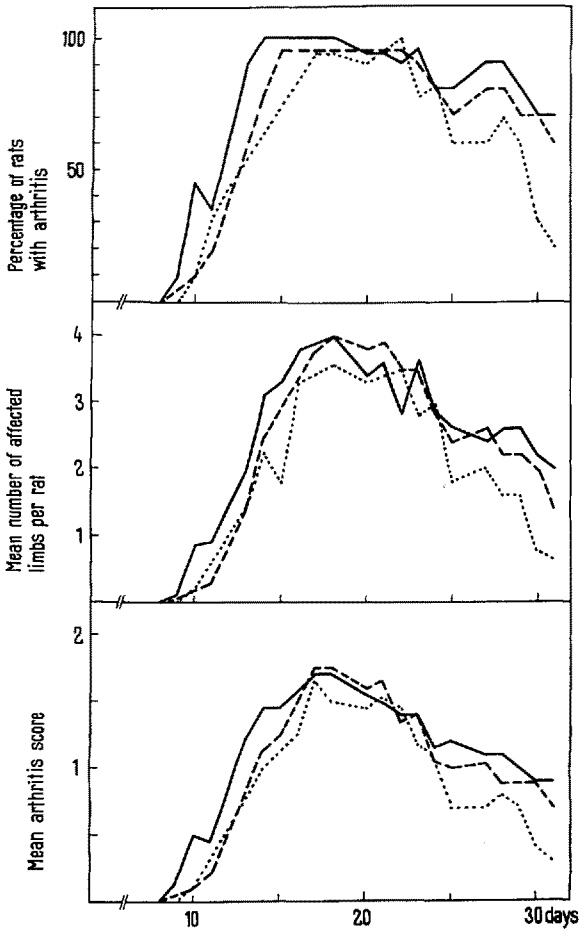
## The Effect of ε-Aminocaproic Acid on Adjuvant Arthritis in Rats

ε-Aminocaproic acid (EACA) is known, and clinically used, as an inhibitor of the activation of profibrinolysin[1-4], but its immunologic properties have hitherto received only slight attention. EACA inhibits the tuberculin reaction of the skin[5] and lengthens the survival time of homografts[6,7]. Antigen-antibody reactions *in vitro* may also be inhibited by EACA[8], and the acid suppresses experimental allergic encephalomyelitis in rabbits[9]. The present study is concerned with the effect of EACA on adjuvant arthritis.

White adult male laboratory rats weighing 240–300 g were used as the test animals. Arthritis was induced by two simultaneous injections (0.05+0.05 ml) of heat-killed, dried *Mycobacterium phlei* suspended in paraffin oil at a concentration of 6 mg/ml. Injections were made intradermally in the tail, one-third from the proximum. Daily subcutaneous injections of EACA (Epsikapron®, Kabi, 100 mg/ml), were started three days before the administration of the adjuvant, and continued to the end of the experiment. The control rats were injected with 1.0 ml of physiologic saline. The development of arthritis was observed from day to day, and the degree of arthritis was scored from 0 to 3 according to the swelling and erythema in the joints, each limb separately. The mean score for each day was calculated from the scores of the maximally affected limb in every rat. The mean number of affected limbs per rat, and the mean number of rats with arthritis were also calculated. Rats which failed to develop arthritis were included in the calculations. Half the animals were bled and killed on the 24th day after the injection of the adjuvant, and the rest on the 31st day. Blood specimens were centrifuged, the sera removed and stored at −20°C for serologic studies. These will be reported later.

The results of the current study (Figure and Table) showed that EACA could not completely inhibit the




—— Controls. - - - - 50 mg EACA/rat daily. ····· 100 mg EACA/rat daily.

Development of adjuvant arthritis in rats during treatment with EACA.

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development of adjuvant arthritis, but in the rats treated with EACA the disease was slighter and of shorter duration than in the controls.

It seems that adjuvant arthritis is not inhibited by EACA so greatly as by whole body irradiation[10], 6-mercaptopurine[11], steroids or some other clinically used drugs[12], but it is possible that the effect of EACA or its analogues on adjuvant arthritis might have some clinical implications.

Effect of EACA on the duration of adjuvant arthritis in rats

| Treatment | Duration of arthritis (days) in rats observed | |
|---|---|---|
| | 24 days[a] | 31 days[a] |
| Controls | $12.7 \pm 1.9$ (20) | $18.5 \pm 4.8$ (10) |
| 50 mg EACA/rat daily | $11.2 \pm 3.3$ (20) | $15.6 \pm 6.7$ (10) |
| 100 mg EACA/rat daily | $10.6 \pm 2.1$[b] (22) | $12.6 \pm 4.9$[c] (12) |

[a] Mean $\pm$ standard deviation is given. The figures in parenthesis refer to the number of rats in the group. [b] Compared with the controls by Student's $t$-test, $0.01 > P > 0.001$. [c] $0.02 > P > 0.01$. Other differences are statistically insignificant; $P > 0.05$.

It is held that EACA suppresses immunologic processes of the delayed type in general[9]. The present findings agree with this, since strong evidence has been presented suggesting that adjuvant arthritis is an immunologic phenomenon of the delayed type[13]. The exact functional mechanism of EACA in this respect is, however, difficult to assess, especially since EACA probably has no significant effect on the production of antibodies[9].

*Zusammenfassung.* Die experimentelle Arthritis bei der Ratte, verursacht durch Injektion von mycobacterialem Adjuvans, wird von ε-Aminocapronsäure zu einem geringen Grade gehemmt, indem das Krankheitsbild milder und die Dauer der Arthritis kürzer wird.

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## Lipoid Bodies under Different Conditions of Diet in the Intestinal Epithelium of a Mammal, *Funambulus pennanti* Wroughton

During the course of studies on the lipoid bodies in the epithelial cells of small intestine of *Funambulus pennanti*, the author has investigated the effect of different diets on the above-mentioned inclusion and its histochemistry. The results given in the paper are with particular reference to the conditions, namely normal, starved and overdose of phospholipines in the food, for which a few squirrels collected from the field were immediately chloroformed, another set of squirrels was starved for three days and a third set was fed with the yolk of hens' eggs for three days. The material was fixed in calcium formal[1] and McManus fluid, and was cut into sections of 6 $\mu$ and stained with Sudan black B. The lipoid bodies are fairly large, granular and spherical in shape (Figure 1) but some of them look irregular, also, in normal condition. In the villi of the starving squirrels, the lipoid bodies had decreased to a considerable extent (Figure 3), but in the villi of the other set which was given an extra dose of phospholipines (yolk), the lipoid bodies had greatly increased in number and size (Figure 2).

Frozen section technique was followed for the histochemical studies of lipoids. The sections were tested for triglycerides, phospholipines and lipo-proteins, The main scheme employed for testing the various lipoids consists of three steps[2].

(1) The frozen sections were first tested for triglycerides with nile blue sulphate[3].

(2) Another set of sections was treated with acetone and then tested for phospholipines with Sudan black B and acid haematein[4–6]. Acetone dissolved out the triglycerides from the frozen sections.

(3) Another set of sections was treated with ether and alcohol. This treatment dissolved both triglycerides and

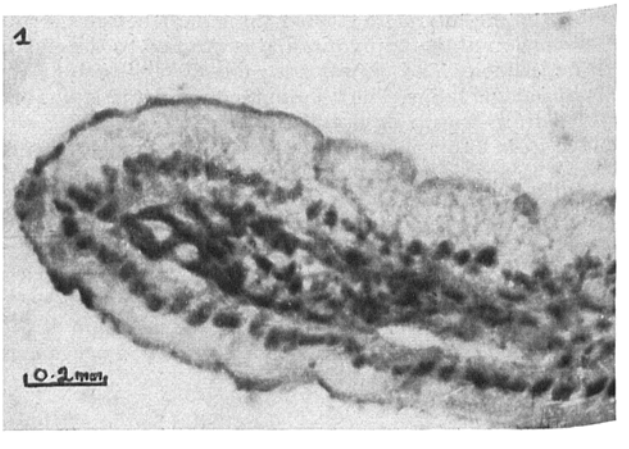


Fig. 1. A villus of small intestine of squirrel showing lipoid bodies in normal condition.

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