an antagonistic action at a concentration of 10 µg/ml. Aminopyrine in concentrations of 10 and 100 µg/ml reduced the rate-accelerant action of AAP in 3 out of 7 experiments, even in those instances in which AAP displayed intrinsic, positively chronotropic properties. This antagonistic action of aminopyrine was persistent: even when the vascular preparation was rinsed several times, the rate of contraction produced by subsequent applications of AAP no longer attained its original level. In the other 4 experiments aminopyrine proved virtually inactive. Indomethacin, applied in concentrations between 0.01 and 10 µg/ml, caused practically no modification of the rate-accelerating effect of AAP, even in cases in which it displayed an intrinsic negatively chronotropic action. In contrast, tribenoside[18] in concentrations of 0.1–1 µg/ml markedly intensified the rate-accelerating effect of AAP. Furthermore, preparations that had become largely insensitive to AAP through exposure to aminopyrine reacted again to AAP after being treated with tribenoside.

It is evident from the results of the experiments described above that substances possessing anti-inflammatory properties can be distinguished on the basis of quantitative and qualitative differences in their influence on the stimulant effect of AAP on the isolated murine portal vein[17].

Zusammenfassung. An der isolierten, autonom pulsierenden Portalvene der Maus zeigt Arachidonsäureperoxid einen ohne Tachyphylaxie-Erscheinungen wiederholbaren, vasotropen Effekt, der hauptsächlich in dosisabhängiger Steigerung der Kontraktionsfrequenz besteht. Dieser Effekt kann durch anti-inflammatorisch wirksame Substanzen entweder antagonistisch (Natriumsalicylat, Phenylbutazon, Aminopyrin) oder synergistisch (Tribenoside) beeinflusst werden. Die mit Arachidonsäureperoxid stimulierte Portalvene der Maus erscheint deshalb geeignet, Antiphlogistika zu differenzieren und Hinweise auf einen eventuellen Wirkungsmodus im Gebiet der terminalen Strombahn zu liefern.

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[18] Generic name of ethyl-3,5,6-tri-*O*-benzyl-D-glucofuranoside (Glyvenol®).

[19] The technical assistance of Mr. A. Blättler is gratefully acknowledged.

Influence of Erythorbic Acid on the Vitamin C Status in Guinea-Pigs

Humans as well as primates, the guinea-pig and some Indian native animal species, are not able to form L-ascorbic acid biosynthetically due to the lack of a special enzyme[1]. They, therefore, require a sufficient supply of vitamin C in the food in order to prevent a hypovitaminosis C or even scurvy.

Due to its reducing properties, L-ascorbic acid exhibits a high antioxidant potency. Thus, it is used, for instance, in fruit processing, as a curing aid in meat processing or in beer to prevent oxidative changes.

Erythorbic acid (D-isoascorbic acid, D-araboascorbic acid) is one of the stereoisomers of L-ascorbic acid with practically no biological activity (only one-twentieth of the biological activity of L-ascorbic acid)[2]. It possesses, however, similar antioxidative properties to L-ascorbic acid and is, therefore, used in some countries as an antioxidant in food[3].

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[2] J. Fabianek and H. Herp, Proc. Soc. exp. Biol., N.Y. *125*, 462 (1967).

[3] B. Borenstein, Food Techn. *19*, 115 (1965).

Effect of feeding various doses of D-erythorbic acid on the uptake of radioactivity after a single orally administered dose of L-(1-^{14}C)ascorbic acid in male guinea-pigs

Tissue	Radioactivity (dpm × 10^{-3}/g wet tissue)				
	Amount of D-erythorbic acid fed (mg/day)				
	0	20	50	100	400
Adrenal glands	177.0 ± 20.4	158.6 ± 25.0	79.9 ± 9.9[c]	108.4 ± 25.6[c]	99.8 ± 10.4[c]
Lungs	69.0 ± 8.0	64.3 ± 9.8	52.8 ± 15.3[a]	47.5 ± 13.0[c]	34.9 ± 4.4[c]
Kidneys	30.4 ± 6.3	30.5 ± 5.3	22.4 ± 3.9[b]	21.4 ± 3.4[c]	17.2 ± 2.2[c]
Testes	21.5 ± 3.2	26.0 ± 7.2	16.5 ± 3.2[b]	18.1 ± 4.4	12.4 ± 3.2[c]
Eyes	20.5 ± 4.3	19.4 ± 1.4	13.6 ± 0.3[c]	13.6 ± 2.1[c]	11.8 ± 1.2[c]
Pancreas	42.3 ± 4.3	47.0 ± 9.8	33.6 ± 6.8[b]	32.7 ± 8.9[a]	25.3 ± 3.6[c]
Cerebrum	6.5 ± 1.0	7.4 ± 0.7	5.8 ± 1.2	5.1 ± 1.0[a]	5.2 ± 0.7[b]
Cerebellum	7.4 ± 1.1	9.3 ± 1.2	7.1 ± 0.8	6.0 ± 0.6[b]	5.7 ± 1.0[b]
Liver	55.6 ± 9.8	62.7 ± 5.8	49.5 ± 12.5	33.9 ± 6.4[c]	40.6 ± 9.4[b]
Spleen	68.8 ± 21.7	83.5 ± 19.2	62.0 ± 12.2	55.8 ± 16.9	46.1 ± 5.8[b]

The experimental details are described in the text. 6 h after dosage of the radioactivity the animals were sacrificed and the tissues were quickly removed. The concentration of radioactivity in the tissues was determined by means of a Nuclear Chicago Mark II liquid scintillation spectrometer after solubilization of parts of the tissues with 1 *N* NaOH. Instagel® (Packard) was used as counting solution. The radioactivity is expressed as disintegrations per min (dpm per g of tissue) (± S.D.).

[a] $p < 0.05$; [b] $p < 0.01$; [c] $p < 0.005$; as compared with 0 mg D-erythorbic acid

Another effect, which has not yet been further investigated, concerns the influence of erythorbic acid on L-ascorbic acid uptake by the tissues. Preliminary results obtained in a study on male guinea-pigs (220–250 g, 7 animals per group), administered orally erythorbic acid (0, 20, 50, 100 or 400 mg per day) in addition to L-ascorbic acid (20 mg per day, vitamin C-deficient diet) for three days and a single oral ^{14}C-labelled dose of L-ascorbic acid (10 µCi, specific activity 4.78 mCi/mmole) on the last day of the experiment, together with erythorbic acid, indicate a significant impairment in the uptake of the labelled vitamin C, already following administration of 50 mg of erythorbic acid per day, in the adrenal glands, the lungs, the kidneys, the testes, the eyes and in the pancreas (Student's t-test, $p < 0.01$). Administration of 100 or 400 mg of erythorbic acid caused an even further decrease in the uptake of the labelled ascorbic acid by these tissues. In addition, uptake of ascorbic acid was also significantly reduced in cerebrum, cerebellum, liver and spleen ($p < 0.01$). The percentage reduction in accumulation of the vitamin caused by administration of erythorbic acid was approximately 50% in the adrenal glands, testes, kidneys, lungs and eyes (Table).

From these preliminary results it is to be concluded that the availability of L-ascorbic acid (vitamin C) is diminished, if erythorbic acid is administered together with L-ascorbic acid.

Zusammenfassung. Eine Verabreichung von Erythorbinsäure (D-Isoascorbinsäure, D-Araboascorbinsäure) vermindert die Aufnahme von Ascorbinsäure (Vitamin C) in verschiedenen Organen des Meerschweinchens. Da Erythorbinsäure nur eine sehr geringe Vitamin-C-Aktivität besitzt, wird bei einer gleichzeitigen Einnahme von Erythorbinsäure und Ascorbinsäure die Verfügbarkeit des Vitamin C für den Tierorganismus signifikant reduziert.

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Synthesis of Ochratoxins T_A and T_C, Analogs of Ochratoxins A and C

Ochratoxins are toxic metabolites produced by several species of *Aspergillus* and *Penicillium*[1–4]. Ochratoxin A (OA), 7-carboxy-5-chloro-3,4-dihydro-8-hydroxy-3-methyl isocoumarin moiety (ochratoxin α) linked by an amide bond to L-β-phenylalanine through the 7-carboxyl group, and ochratoxin C (OC), the ethyl ester of OA, are the most toxic metabolites within this series. In contrast, ochratoxin α (Oα) has not proven toxic to test animals[5,6]. In order to pinpoint the influence of the side chain in the Oα moiety on the toxicity of OA, we have investigated the effect of substituting tyrosine for phenylalanine. This paper presents a method for the synthesis of ochratoxin T_A (OT_A) and T_C (OT_C), and describes the physicochemical properties of these analogs.

Materials and method. Ochratoxin A was produced in rice by *Aspergillus ochraceus* 3174 as described[7,8]. Ochratoxin α was prepared by acid hydrolysis of natural crystalline OA[5], and was purified by Adsorbosil chromatography[5].

Thin layer chromatography (TLC). Silica gel G and silica gel H (both from Brinkmann Instruments Co.), coated to the glass plate to a thickness of 0.25 mm and 0.5 mm respectively, were used for analytical and preparative TLC. Benzene: acetic acid (3:1) was used as a developing reagent. The fluorescent spots or bands were detected under a longwave u.v. light or charred with H_2SO_4.

Preparation of OT_C and OT_A. 25 mg tyrosine ethyl ester and 27 mg of ethoxy-1-ethoxy carbonyl-1,2-dihydroquinoline (EEDQ) were added to a tetrahydrofuan solution containing 27 mg of Oα. The mixture was then stirred at room temperature overnight, filtered, and evaporated. Crystals were formed after addition of Skellysolve B to the oily residue, to which a small amount of ethyl acetate was added. The crystals were redissolved in EtOH,

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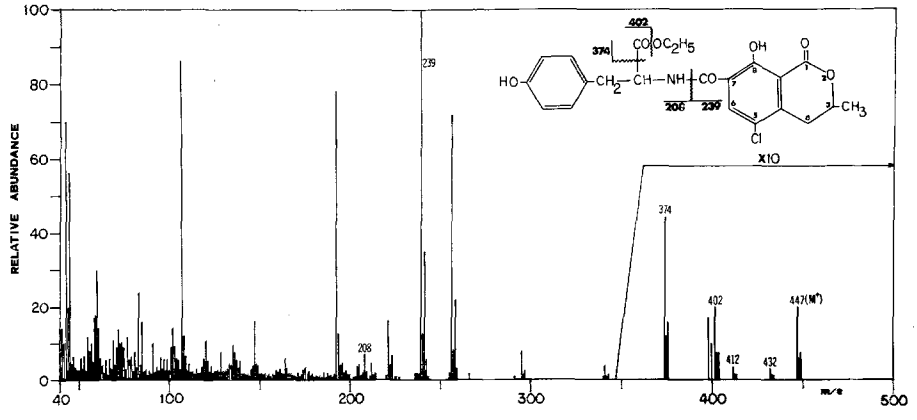

Mass spectrum and structure of ochratoxin T_C.