



as already described by me. 30 mgm. of the dry triturated matter were taken for analysis.

In Fig. 1 the results of analyses are expressed as percentage of elastin in the dry weight of the sample and plotted against age at time of death. Statistical analysis of this graph shows a highly significant increase in elastin content of the human aorta with age between 0 and 20 years (correlation coefficient: $r = 0.877$; Student's $t = 6.81$; $P < 0.001$). The equation of the regression line is $y = 17.5 + 1.65x$.

Further investigations in progress in this laboratory seem, on the other hand, to demonstrate a significant decrease of elastin content in the aorta after 20 years until old age.

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Lack of Effect of Coenzyme Q_{10} on Coumarin-induced Hypoprothrombinæmia

SEVERAL coumarin and indanedione drugs are capable of inducing hypoprothrombinæmia, presumably by interfering with the cofactor role of vitamin K in prothrombin synthesis. This role of vitamin K, probably involving electron transport, can be competitively inhibited by coumarins, and the inhibition competitively overcome by large doses of vitamin K (ref. 1).

Recently² another group of quinones, the coenzymes Q , has been shown to be involved in electron transport. Numerous similarities of coenzymes Q to the vitamin K group³ have led to the following comparison of vitamin K₁ and coenzyme Q_{10} as regards inhibition

Table 1. EFFECT OF COENZYME Q_{10} ON PROTHROMBIN RESPONSE TO ACENOCOUMARIN IN NORMAL GUINEA PIG*

	Prothrombin time (sec.)	
	Range	Average
Q_{10} vehicle (1 or 2 c.c.)	31-34	33
Q_{10} (10 or 20 mgm.)	31-36	34
Acenocoumarin (1 dose) 4.5 mgm. in 1 c.c. H_2O	49-62	55
Acenocoumarin (1 dose) + Q_{10} 10 mgm.	53-130	79
Acenocoumarin (2 doses 24 hr. apart) each 4.5 mgm.	99->180	>149
Acenocoumarin (2 doses 24 hr. apart) + Q_{10} 10 mgm.	78-177	134
Acenocoumarin (2 doses 24 hr. apart) + vitamin K ₁ ('Mephyton') 10 mgm.	35-50	43

* All drugs were given intraperitoneally. Each group consists of at least four animals.

of the prothrombin response to a coumarin anti-coagulant.

Guinea pigs were treated with sodium acenocoumarin, vitamin K₁, coenzyme Q_{10} (10 mgm./c.c.) in solvent vehicle (consisting of 'Emulphor' and dimethylacetamide, and the co-enzyme Q_{10} kindly provided by Dr. Karl Folkers of Merck, Sharp and Dohme, Rahway, New Jersey) and solvent vehicle as indicated in Table 1. Blood specimens were obtained 24 hr. after the last dosage, and the prothrombin time estimated by techniques previously reported⁴. The relatively long normal prothrombin time by the method employed is characteristic of the species. The results in Table 1 demonstrate that coenzyme Q_{10} has no vitamin K-like protective effect against acenocoumarin-induced hypoprothrombinæmia under the experimental conditions used.

Table 2. EFFECT OF COENZYME Q_{10} ON RESPONSE TO ACENOCOUMARIN IN ACUTELY STARVED GUINEA PIGS

	Prothrombin time (sec.)	
	Range	Average
Acenocoumarin 4.5 mgm. + 1 c.c. saline	40-55	48
Acenocoumarin 4.5 mgm. + co-enzyme Q_{10} 10 mgm.	60-75	68
Acenocoumarin 4.5 mgm. + 1 c.c. vehicle	50-87	63

To test this observation further, the experiment was repeated in 12 animals deprived of food the day prior to and after dosage. The results are presented in Table 2. It is evident that coenzyme Q_{10} had no protective effect.

It is concluded that in the guinea pig, exogenous coenzyme Q_{10} has no vitamin K-like antagonistic action against the hypoprothrombinæmia induced by acenocoumarin.

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