methylaminoethanol 3:4 quinone has no effect on blood pressure.

It was impossible to oxidize the secondary hydroxyl group in the side chain of the epinephrine molecule without disrupting the benzene ring. The epinephrine product with 3 keto groups (monomethylamino aceto 3:4 quinone) can be obtained however by starting with adrenalone and then oxidizing the catechol group to quinone. Adrenalone was prepared by a method employed by Dziergowski.[3] This product was dissolved in the minimum of 0.1 N HCl and aqueous ammonia added until yellow crystals of adrenalone began to appear. Ammonia was added further while the solution was vigorously stirred. The solution first turned pink, then red and finally a dark red crystalline product separated out upon standing in the cold. The crystals were washed with cold water and dried. The product had no effect on blood pressure. (Found: C, 60.4; H, 5.4; N, 8.0. $C_9H_9NO_3$ requires C, 60.3; H, 5.0; H, 7.8). Monomethylamino aceto 3:4 quinone is more stable than monomethylaminoethanol 3:4 quinone. The same characteristic is shown by epinephrine and adrenalone.

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Effects of Ingested Fats and Sterols on Sterol Metabolism of the White Rat.

H. C. ECKSTEIN.

From the Department of Physiological Chemistry, Medical School, University of Michigan, Ann Arbor.

In a study of the effects of the ingestion of plant fats and sterols on the metabolism of the white rat, young litter mates were placed on plant diets consisting of a mixture of soy bean meal, corn oil, agar, starch and the Osborne and Mendel salt mixture. The 2 diets used were so prepared that the protein intake would be practically the same for both groups of rats. The fat content of one diet was 11% as compared with 34% for the other. Both were supplemented with a vitamin B yeast concentrate (Harris), carotene, and viosterol. A record of food intake was kept. The sterol contents of the liver, the remaining tissues, the diets, and the feces were determined gravimetrically by means of the digitonin method. The table shows that

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[3] Dziergowski, S. K., *Centralb.*, 1893, **2**, 861.

most of the rats on the high fat diet in series 1 and 3 had a higher percentage of liver sterols than those on the low fat diet. This is not true for series 2 in which not only poor growth was obtained but also in which the experimental period was increased by 3 weeks. The table also shows that the larger percentage of liver sterols cannot be due to sterol mobilization because of the similarity of the sterol contents of the rest of the tissues of all of the rats. Fats may therefore be the precursors of sterols. Such a view would be at variance with one held by Chanutin and Ludewig[1] who state that while carbohydrates cause a deposition of cholesterol in the white rat, fats inhibit that phenomenon. Further data showed that all of the rats synthesized sterols since the amounts excreted in the feces were always larger than those fed. In general this took place to a greater extent on the high fat diet. The source of the extra sterols has not been determined. According to Schönheimer[2] ergosterol and cholesterol are the only sterols absorbed, but Dam and Starrup[3] concluded that dietary "phytosterines" occasionally find their way to the livers of rats. The extra sterols may thus originate from ingested fat, from ingested sterols or from both.

TABLE I.
Sterol Contents of Livers and Remaining Tissues.

	Low fat-sterol diet			High fat-sterol diet		
	No. of rats	Liver sterols	Sterols in remaining tissues	No. of rats	Liver sterols	Sterols in remaining tissues
		%	%		%	%
Series 1	2	.41	.19	2	.85	.20
(7 wks.)		(.40-.42)*	(.19-.19)		(1.00-.69)	(.19-.20)
Series 2	3	.35	.19	3	.40	.20
(10 wks.)		(.28-.42)	(.18-.19)		(.39-.41)	(.20-.21)
Series 3	6	.39	.21	6	.61	.24
(7 wks.)		(.30-.46)	(.19-.23)		(.43-.72)	(.20-.29)

* The figures in parentheses represent the ranges of the values in the different experimental groups.

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[1] Chanutin, A., and Ludewig, S., *J. Biol. Chem.*, 1933, **102**, 57.

[2] Schönheimer, R., *Z. physiol. Chem.*, 1929, **180**, 1.

[3] Dam, H., and Starrup, U., *Biochem. Z.*, 1934, **274**, 117.