TABLE 3

AMOUNTS OF DDT OR DDT METABOLITES PRESENT IN VARIOUS PARTS OF FLIES SURVIVING AN 11.25 μG DOSAGE OF RADIOACTIVE DDT (6 TO 10 FLIES EXAMINED ON EACH DATE). ONE μG WAS EQUIVALENT TO 180 CPM

Dissected parts	After 2 days		After 6 days		After 10 days	
	μg	Percentage of total	μg	Percentage of total	μg	Percentage of total
Body fluids	—	—	0.23	9.7	0.30	9.6
Reproductive system	0.24	20.2	.33	13.8	.50	17.4
Intestinal tract	.03	2.8	.12	5.0	.10	3.3
Thoracic ganglion	.05	4.3	.12	5.2	.07	2.4
Thoracic muscle	.04	3.8	.14	6.1	.16	5.2
Abdominal and thoracic cuticle-hypoderm	.44	37.0	.70	29.6	1.03	34.3
Wings, legs, head	.38	31.9	.72	30.6	.84	27.8
Total	1.19	100.0	2.36	100.0	3.00	100.0

hypoderm. Sternburg and Kearns (*1*) found by chemical analysis that very little, if any, DDT or DDE (1,1,dichloro-2,2-bis(*p*-chlorophenyl) ethylene) was present in the interior parts of resistant houseflies treated with DDT. The penetration and distribution of DDT in flies over a long period of time appear to be physical phenomena. Richards and Cutkomps (*5*) showed that cuticle and purified chitin absorbed part or all of the DDT when placed in moderately dilute suspensions of DDT in distilled water.

In order to obtain information on the rate of absorption over a period of several days, Orlando resistant flies were treated on the thorax with 5.9 μg radioactive DDT/fly and held at room temperature (60°–75° F). In 24 hr the mortality was 25%. From 6 to 10 survivors were killed at intervals, and a radioassay was made of the amount of DDT absorbed. Table 2 shows that the amount absorbed increased from 0.39 μg after 1 day to 1.26 μg after 9 days. The flies that died within 24 hr showed a similar trend.

The increase in amount of DDT absorbed suggested further tests to determine whether the absorbed radioactive DDT was concentrated in certain organs. Flies were treated with 11.25 μg DDT each, and the mortality was 54% in 24 hr. As shown in Table 3, the total internal radioactivity of these flies increased with time, up to 10 days, but the morphological distribution did not change. From 31 to 40% of the total DDT applied was found in the internal systems, and the remainder was in the cuticle. The amount absorbed per fly was considerably greater than that resulting from treatments with 5.9 μg (Table 2). This is in agreement with the results of Sternburg *et al.* (*1*), who had shown that absorption of DDT was greater with larger applications.

These experiments demonstrate the importance of timing the analysis of flies in DDT-absorption studies. A 5-day delay in the analysis of flies succumbing within 24 hr after treatment increased the amount of DDT absorbed as much as 45%, and 9-day delay 62%. About the same increase was obtained for flies that survived the DDT treatment for 5 days, indicating that absorption of DDT proceeds at about the same rate in dead as in living flies. Treated flies radioassayed after a lapse of more than a year showed a large increase in the amount of DDT penetrating the integument.

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The Accumulation of Serum Cholate and its Relationship to Hypercholesteremia[1]

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Hypercholesteremia can be produced in the rat by such different procedures as (1) ligation of the bile duct (*1, 2*); (2) production of experimental nephrosis (*3*); and (3) injection of Triton W 1339 (*6*). Recently we have obtained evidence (*4–6*) which indicates that each of these procedures also induces a rise in the cholate content of the animal's blood. It is the accumulation, moreover, of this latter steroid that appears responsible for the ensuing hypercholesteremia observed in the above conditions. In other words, the rise in cholesterol content of plasma seems to be a phenomenon *secondary* to the "hypercholatemia" effected by these particular procedures. The mechanism, however, by which excess accumulation of cholate in blood induces hypercholesteremia is still to be determined.

In view of the above findings concerning the primacy of cholate accumulation in the pathogenesis of

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[2] With the technical assistance of Barbara Gunning.

TABLE 1

THE SERUM CHOLATE IN NORMOCHOLESTEREMIC AND HYPERCHOLESTEREMIC SUBJECTS

Type of subject	No.	Av age	Total serum cholesterol (Mg/100 ml)			Total serum cholate (Mg/100 ml)			Cholesterol/ cholate ratio
			Mean	S. E. mean	Range	Mean	S. E. mean	Range	
Normal	25	29	228	± 8.7	(110–280)	5.3	± .26	(1.7–7.2)	43
Patients with nephrosis	12	6	660	±56.0	(390–1000)	19.9	±3.0	(9.8–44.0)	33
Patients with xanthoma	6	47	456	—	(330–600)	15.4	—	(9.0–32.0)	30
Patients with diabetes	5	54	445	—	(410–490)	15.0	—	(12.0–17.0)	30
Patients with myocardial infarct	8	50	388	±14.7	(320–465)	12.8	±1.4	(10.5–16.0)	30
Patients with hypothyroidism	2	46	352	—	(310–395)	12.0	—	(12.0–12.0)	29

various experimental forms of hypercholesteremia, it appeared important to investigate whether hypercholatemia is present in clinical states of hypercholesteremia. The results of such a study are reported in this communication.

Blood samples[3] obtained from normal subjects and patients suffering from disorders frequently associated with hypercholesteremia were analyzed for their serum cholesterol content according to methods previously described (*2*). Sera containing less than 300 mg of cholesterol/100 ml were considered normocholesteremic, and those containing more, hypercholesteremic. A number of the normocholesteremic samples obtained from normal subjects and also a group of samples selected from known hypercholesteremic patients were analyzed for their cholate content by absorption photometry according to a combination of the methods of Minibeck (*7*) and Wilken (*8*). The chemical analysis for cholate was not found to be influenced by variations in the cholesterol or lipid content of the sample.

As Table 1 indicates, the average serum cholate of 25 young normal subjects (having an average serum cholesterol of 228 mg/100 ml) was 5.3 mg/100 ml (range: 1.7–7.2; S. E. mean: ± 0.26).

Hypercholesteremia, on the other hand (Table 1) was invariably associated with an elevation of serum cholate (hypercholatemia). Moreover, the amount of cholate accumulation appeared to be more closely correlated with the degree of hypercholesteremia than with any particular disease entity. Thus, the 12 patients with nephrosis whose average serum cholesterol was 660 mg/100 ml had an average cholate of 19.9 mg/100 ml (range: 9.8–44.0; S. E. mean: ± 3.0). The 8 patients with myocardial infarction, on the other hand, had a much lower serum cholesterol (388 mg/100 ml) and a corresponding lower cholate (12.8 mg/100 ml). In both groups, however, the cholesterol/cholate ratio remained about the same. The same relationships between cholesterol and cholate also were observed in the patients with xanthoma, diabetes, and hypothyroidism (Fig. 1).

The present study indicates that an elevated blood cholate level occurs in practically all cases of hypercholesteremia, irrespective of the latter's seeming etiology. The degree of hypercholesteremia moreover appears to bear a close relationship to the extent of hypercholatemia.

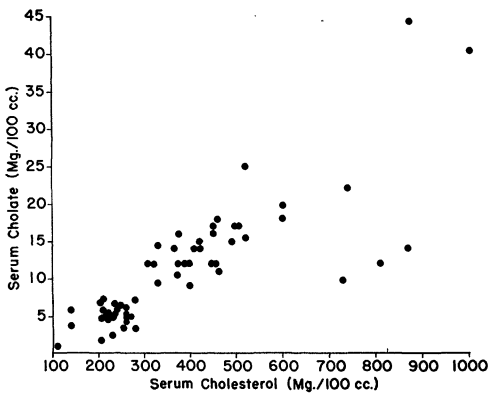

FIG. 1. The relationship of serum cholate to serum cholesterol in the normocholesteremic and hypercholesteremic subject.

These findings appear important because experimental accumulation of cholate itself in plasma has been found by us (*5*) to produce hypercholesteremia. Moreover, such diverse states as biliary obstruction and experimental nephrosis lead to hypercholatemia, which in turn evokes hypercholesteremia.

These findings suggest that clinical hypercholesteremia may be a phenomenon secondary to an initial derangement of cholate metabolism. Such a relationship cannot be considered too surprising when it is recalled that considerable data have been amassed (*9–11*) that suggest some sort of metabolic relationship between bile salts and cholesterol.

The present findings also indicate that the role of the liver (either as a primary or a secondary agent)

[3] The authors wish to express their thanks to John Gofman for his kindness in supplying a number of the blood samples analyzed in this study.

must be kept in mind in any evaluation of the factors involved in the pathogenesis of human hypercholesteremia.

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Growth of the Scutellum of Maize in Culture

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The discovery (*1*) that maize embryos of small size upon excision gave rise to small plants, and that those of larger size at time of excision gave larger ultimate sizes of embryos, and Merry's (*2*) evidence of the same situation in barley embryos suggest that if embryos could be held in an embryonic condition until they had grown beyond the normal size of embryos in mature seeds, and then planted, they should give rise to seedlings and mature plants of abnormally large size. Kent and Brink (*3*) had considerable success in preventing germination of barley embryos and in continuing their embryonic growth by the use of tomato juice in culture.

In trials with maize, tomato juice had little or no effect on embryos, although it did serve to expedite the growth of corn endosperm in culture (*4*). Any check in germination of the embryos was so slight as to be of no importance in producing embryos of abnormal size.

Since the root and the shoot could not be kept from growing out, thus producing germination and setting up seedling growth, rather than the desired embryonic growth, it seemed that the scutellum at least might be led to continue embryonic growth by the removal of the root-stem axis. Following this idea, numbers of embryos of the variety Evergreen, which were nearing mature size, were excised and the root-stem axis was removed from each. At the same time embryos of Golden Bantam variety were treated in the same way, but these were in an earlier stage of growth than the Evergreen. One set of each variety was grown on White's medium and one on White's medium plus tomato juice, as used by Kent and Brink.

A set of Evergreen embryos was weighed and measured to serve as controls, then dried and weighed to give the dry weight. Golden Bantam embryos were treated in the same way. It is obvious from Table 1 that the Golden Bantam embryos were a great deal younger than those of the Evergreen. The cultures were continued until growth ceased. Wet and dry weights and measurements of length and width of the scutelli were obtained.

TABLE 1

WEIGHTS OF CULTURED, CONTROL, AND RIPE SCUTELLI OF MAIZE

Variety	Treatment	Av wt (mg)	
		Wet	Dry
Evergreen	Cultured	977.3	224.2
"	Control	171.0	126.8
"	Ripe	393.9	154.3
Golden Bantam	Cultured	90.0	27.3
" "	Control	18.9	13.3
" "	Ripe	252.6	150.0

TABLE 2

LENGTHS AND WIDTHS OF CULTURED, CONTROL, AND RIPE SCUTELLI OF MAIZE

Variety	Treatment	Av length (mm)	Av width (mm)
Evergreen	Cultured	12	9.0
"	Control	5	4.5
"	Ripe	6.5	6.0
Golden Bantam	Cultured	5.5	3.0
" "	Control	3.0	2.0
" "	Ripe	6.0	5.0

For comparison with the cultures and controls, ripe grains of the two varieties were soaked and germinated. When the embryos were fully expanded the root-stem axes were removed from them, and wet and dry weights and length and width measurements were taken.

In the cultures a number of scutelli formed outgrowths that were partial roots or partial stems. These growths were apparently due to incomplete excision of the root-stem axis rather than to regeneration. All such scutelli were removed from the experiment. No true regeneration of scutelli was seen, but some scutelli did develop papillate or even calluslike outgrowths on their surfaces. More outgrowths were seen on the medium with tomato juice than on the plain White's medium, but the differences were not great and both sets were thrown together in the tables.

The scutelli of Golden Bantam corn grew fairly well and showed nearly a fivefold increase in net weight and more than a doubling of the dry weight. They never, however, approached the size or weight of the scutelli from ripe seeds—a result, probably, of excision at a stage definitely too young.

The Evergreen scutelli made a much better showing and at the end were nearly twice as large and as heavy as those from normally ripened seeds.

Tables 1 and 2 show that one can take scutelli from