in the maternal bones. (6) These observations illustrate the likelihood of introducing serious errors in theoretical calculations of radiation dosages to fetal tissues by estimating fetal tissue distributions from data obtained from adult animals. For example, in the case of ^{95}Zr, bone is the target tissue of greatest concern in both the maternal and fetal body, since concentrations were 5-20 times those in the other tissues, at least at 24 hours following contamination. For ^{95}Nb the liver and kidneys receive the greatest burden in the maternal body, but in the fetus on the other hand, liver and bone attain even higher concentrations. (7) Rigorous conclusions regarding the relative ease of transport of ^{95}Zr *vs* ^{95}Nb across the placenta cannot be drawn from this data. For example, although there was more ^{95}Nb than ^{95}Zr in the fetal bone and liver, the reverse was true for fetal muscle and kidney. Thus the large differences in avidity for these radionuclides among the various fetal tissues obscured any possible differences in placental transport, *per se*. Information concerning relative turnover rates in the tissues and the fate of ^{95}Nb which "grows in" from the decay of deposited ^{95}Zr must await further experimentation.

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Plasma and Tissue Lipids in Hypercholesteremic Pigeons. (30123)

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We reported earlier that feeding vegetable oils, especially sesame oil, along with cholesterol increased total cholesterol, phospholipids, β-lipoprotein cholesterol, triglycerides and free fatty acids of plasma in monkeys(1) and chicks(2). There was also disturbances in the distribution of lipids in the tissues of the monkeys(3). As pigeons are susceptible to natural atherosclerosis, it was our interest to study the effect of feeding sesame oil with or without simultaneous administration of cholesterol on the different fractions of plasma and tissue lipids in these birds.

Materials and methods. Three-months-old pigeons *(Columbia livia)* were fed the basal diet(1) for one week, to make them accustomed to the diet, and divided into 3 groups. Group A birds received only the basal diet. Group B birds received the basal diet mixed with sesame oil at 10% level and birds of Group C received the basal diet mixed with sesame oil (10%) and cholesterol (1%). All birds were fed 5 mg ascorbic acid per bird and 2 drops of a concentrate of vit. A once a week. Each bird consumed daily about 30 g of the diet.

After 8 weeks on these diets, the pigeons were fasted overnight and blood collected by cardiac puncture in heparinized tubes. They were killed subsequently by decapitation, different tissues were removed, freed from adherent blood and weighed. Weighed portions of liver, brain, heart, skin and carcass were cut into small pieces, dried at 60°, Soxhleted

TABLE I. Plasma Lipids of Pigeons Fed Different Diets for 8 Weeks.

Plasma lipids (mg %)	Pigeons fed basal diet (10)	Pigeons fed basal diet + sesame oil (7)	Pigeons fed basal diet + sesame oil + cholesterol (12)
Total cholesterol	146 ± 3.7	142 ± 5.6	702 ± 16.2
β-lipoprotein cholesterol	70 ± 3.7	75 ± 3.5	310 ± 27.1
Phospholipids	274 ± 6.7	342 ± 10.4	749 ± 19.3
Triglycerides	195 ± 11.6	225 ± 16.6	284 ± 9.7
Lipoproteins: (%) α	66 ± 2.1	66 ± 2.4	35 ± 1.9
β	34 ± 2.1	34 ± .6	65 ± .9

Figures in parentheses indicate number of birds. Values = Mean ± S.E.

with petroleum ether and the extract used for estimation of cholesterol, phospholipid and total lipid(3). Blood plasma was used for estimation of the different fractions of lipids(2).

Results. Plasma lipids. Plasma cholesterol level did not change when sesame oil alone was mixed with the basal diet, but when cholesterol was added to the diet along with sesame oil, plasma cholesterol increased markedly. Plasma phospholipids increased after feeding sesame oil alone or with cholesterol but the increase was more marked when sesame oil was fed along with cholesterol. The rise in the plasma phospholipid was less as compared with the rise in plasma cholesterol. This resulted in an increase in the cholesterol:phospholipid ratio. Plasma triglycerides increased after administration of sesame oil with cholesterol. Administration of sesame oil alone did not produce any change. Feeding of sesame oil with cholesterol led to an increase in the β-lipoprotein percentage with a concomitant decrease in the α-lipoprotein percentage resulting in an increase in the β:α ratio (Table I).

Tissue lipids. Total cholesterol increased in liver and carcass, diminished in brain and did not change in the heart and skin of pigeons fed cholesterol along with sesame oil. Feeding only the oil increased cholesterol of liver without producing any change in the other tissues studied. Phospholipids of liver only increased after feeding oil with cholesterol. Total lipids diminished in the heart and skin, increased in liver and remained unaltered in the brain of pigeons fed cholesterol along with sesame oil. Feeding only the oil increased the lipid content of the carcass (Table II).

Discussion. The increase of plasma cholesterol observed in pigeons fed sesame oil along with cholesterol indicated that sesame oil facilitated the absorption of cholesterol. Similar observations were reported in case of monkeys(1) and chicks(2). Atheromatous aorta is capable of greater extraction of cholesterol from the circulating blood stream than the normal aorta(4). The increase of circulating cholesterol might play an important role in initiation of the atherogenic process. The increase in plasma phospholipids after feeding sesame oil indicated that the oil helped in the synthesis of phospholipids by providing the fatty acids necessary for their synthesis. The marked increase in plasma phospholipids after feeding the oil along with cholesterol indicated that a rise in plasma cholesterol stimulated the synthesis of phospholipids so as to maintain a normal cholesterol:phospholipid ratio. The rise in plasma phospholipids, however, failed to keep pace with the hypercholesteremia and as a result an increase in cholesterol:phospholipid ratio was observed. The increase in the β-lipoprotein of plasma was probably due to greater need in the body to transport the cholesterol that was being added continuously as a result of the increased absorption of cholesterol due to the presence of fat. The effect of feeding cholesterol on β-lipoprotein of plasma reflected the extent to which the rates of biosynthesis and degradation of cholesterol could be altered to accommodate the influx of preformed cholesterol(5). Increase of plasma triglycerides seemed to be related

TABLE II. Tissue Lipids of Pigeons Fed Different Diets for Eight Weeks.

Lipids of tissues		Pigeons fed basal diet (7)	Pigeons fed basal diet + sesame oil (7)	Pigeons fed basal diet + sesame oil + cholesterol (7)
Total cholesterol, mg/100 g wet tissue:	Liver	325 ± 13	490 ± 31	1578 ± 232
	Brain	2578 ± 517	2482 ± 291	1481 ± 238
	Heart	168 ± 42	153 ± 9	145 ± 14
	Skin	137 ± 29	118 ± 20	124 ± 23
	Carcass	98 ± 16	120 ± 12	155 ± 10
Total phospholipids, mg/100 g wet tissue:	Liver	1221 ± 314	1717 ± 238	1910 ± 173
	Brain	6464±1310	5088 ± 518	5372 ± 975
	Heart	1144 ± 202	1048 ± 131	1030 ± 101
	Skin	454 ± 86	288 ± 30	305 ± 20
	Carcass	588 ± 64	654 ± 61	668 ± 52
Total lipids, g/100 g wet tissue:	Liver	6.48 ± 1.04	5.74 ± .84	8.43 ± 1.02
	Brain	14.62 ± 2.32	12.51 ± 1.41	12.64 ± 1.50
	Heart	8.16 ± 1.71	9.98 ± .95	6.71 ± .59
	Skin	51.56 ± 5.01	61.57 ± 2.13	40.83 ± 3.02
	Carcass	8.51 ± .86	13.01 ± .88	10.49 ± .95

Figures in parentheses indicate number of birds. Values = Mean ± S.E.

to the increase of β-lipoprotein since the latter normally acts as a vehicle for transport of the former. The hyperlipemia observed seemed to be associated with the increased content of triglycerides which probably could not be utilized in the body. Increased formation of β-lipoprotein might be partly due to the increase of triglycerides as an attempt to transport them from liver to adipose tissue which acts as the storage depot for triglycerides.

Cholesterol of liver increased in sesame oil-fed pigeons. This was further augmented when cholesterol was also fed. Similar observations were recorded in studies with monkeys(3). It might be possible that sesame oil, though an unsaturated fat, accelerates biosynthesis of cholesterol in the liver. Corn oil fed animals also had increased cholesterol in the liver and heart(6). Cholesterol formation in brain seemed to be depressed after the oil was fed with cholesterol. Hypercholesteremia did not produce any change in the cholesterol of heart and skin. Increased liver phospholipids associated with hypercholesteremia were probably due to the body's tendency to maintain the cholesterol:phospholipid ratio in the liver for normal functioning of the organ. Diminished total lipids in heart and skin of hypercholesteremic pigeons was probably due to mobilization of fat from these tissues. Similar observations of mobilization of fat after cholesterol administration had been noted(7). It was of interest to note that heart could utilize or mobilize the fat more efficiently than other tissues. There had been no interrelationships between the plasma levels and tissue distribution of the lipids.

Summary. Different fractions of plasma lipids and lipid content of tissues were determined in hypercholesteremic pigeons. Hypercholesteremia was produced by feeding sesame oil and cholesterol. Hypercholesteremic pigeons had significantly higher levels of plasma β-lipoprotein cholesterol, phospholipids, triglycerides, β-lipoprotein percentage and increased β:α lipoprotein ratio; increased cholesterol in the liver and carcass; increased phospholipid in the liver; increased total lipid in the liver and diminished total lipid in the heart and skin. There had been no interrelationships between plasma levels and tissue distribution of lipids. Hypercholesteremia disturbed the lipid metabolism in pigeons.

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Effects of Cortisone on *in vitro* Viral Infection. I. Inhibition of Cytopathogenesis by Polio, Rabies and Yellow Fever Viruses in Human Embryonic Cells.* (30124)

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The action of steroid hormones on *in vitro* infection of cells by viruses has been studied with different systems(1-8). Contrasting results have been reported for action of cortisone, including protection against cytopathic effect coupled with increased virus production (4), enhanced cytopathic effect accompanying increased virus release(5), prolonged survival of chick embryos associated with initial inhibition of viral synthesis but final increase in virus yield(2,3), and reduction of virus titers revealed by cortisone-treated cells(6-8). It was therefore of interest to reexamine the effects of cortisone on *in vitro* virus infection with the human diploid cell strains now available(9). This paper reports some effects on cytopathogenesis induced by 3 different viruses.

Materials and methods. Cell-culture systems. The following strains of normal human fibroblasts were used in the experiments: the WI-38 human diploid cell strain(9) derived from lung tissue of normal human embryo and used at the 20th culture passage level, the 418 strain derived from lung tissue of normal human female embryo and used at the level of the 9th culture passage, and the HLT strain from normal human embryo, which was used at the 30th subculture level. The 418 strain was started by the procedure described by Hayflick and Moorhead(9). For purpose of this study, the HLT strain was initiated from tissue fragments explanted in medium with and without 2.5 μg/ml cortisone acetate (Vitamix Corp., Philadelphia, Pa.). Methods employed for the subculture of cell strains, or for preparation of monolayer cultures in tubes or in Petri dishes, followed exactly the techniques described in detail elsewhere(9), except that the 418 strain was subcultured by 1:3 rather than 1:2 splits. Cells were dispersed for subculture by treatment with 0.25 mg/ml trypsin in Earle's solution. For treatment of cells during subculture, cortisone was added to medium to a concentration of 2.5 μg/ml, except as noted.

Viruses. The 17 D strain of yellow fever virus, 230th chick-embryo passage in the form of lyophilized suspension of chick embryonic tissues, was used as inoculum to start the cell-culture passage series described here. The CHAT strain of type 1 poliovirus (11) adapted to grow in human diploid cell strains was used for plaque-count experiments. The CVS (received from Nat. Inst. Health, Bethesda, Md.) and HEP-Flury [chick embryo adapted(10)] strains of fixed rabies virus were used in the form of infected mouse brain and infected chick embryo suspensions, respectively.

Effects of virus infection on cells. Cytopathic effects were determined by daily microscopic examination of cultures until division for subculture or observation of complete lytic destruction. Some cultures were stained by May-Grünwald Giemsa stain be-

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