to approximately 180 to 190 mg% by the family diet pattern.

Also, it was shown earlier(6) that the dietary regimen influenced serum lipid fatty acid composition. After a year of the dietary regimen the effect is even more pronounced, particularly in respect to the large increase in percentage of linoleic acid in the serum cholesterol esters and triglycerides. While this suggests an inverse relationship between serum cholesterol level and percentage of linoleic acid in serum cholesterol esters and triglycerides, it should be pointed out that the initial marked drop (10 weeks) in the level of the serum cholesterol was accompanied by a significant change in the fatty acid spectrum of the cholesterol esters(6); further changes in the spectrum (as indicated here) occurred during the latter part of the dietary period when the serum cholesterol level was stabilized.

Summary. Clinically healthy subjects with elevated serum cholesterol levels were placed on a family diet pattern high in linoleic acid for a one-year period. The diet produced a highly significant decrease in serum cholesterol level (from 293 to 230 mg%). The greatest decrease occurred during the first several months; thereafter the serum cholesterol level fluctuated around 230 mg%. The diet produced a marked increase in percentage of linoleic acid in the serum cholesterol esters and triglycerides, with concomitant drops in percentages of saturated and oleic acids. It is concluded that such a dietary regimen for lowering the serum cholesterol level is practical for long periods.

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Liver Lipid Peroxide Levels in Carbon Tetrachloride Poisoning.* (27703)

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The protective action of Vit. E in carbon tetrachloride poisoning has been shown by histological means in rat liver(1). Vit. E is also known to be a lipid antioxidant. On these bases it was suggested that Vit. E functioned in this form of poisoning to protect the cellular lipid from oxidation "hypothetically" called forth by carbon tetrachloride or its metabolites. It has also been shown that early in carbon tetrachloride poisoning there is a dislocation of ribosomes from lipoprotein membranes, and impairment of protein synthesis by the liver(2,3). The possibility that formation of lipid peroxides in the lipoprotein membranes may play a part in these changes was submitted to test.

Methods and materials. Male Sprague-Dawley rats were fasted for 16-18 hours prior to experimentation. The animals were intubated without anesthesia and 0.50 cc carbon tetrachloride (C.P.) per 100 grams body weight dissolved in an equal volume of mineral oil was administered by gastric tube. Control animals received only mineral oil. At

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TABLE I.

	Wt	Liver wt	Peroxide content per unit wt of liver
Control animals	222.0	6.84	100.1*
	315	8.90	100.0*
Treated "	219	6.80	102.0
	212	7.20	97.0
	384	10.8	97.4
	282	8.4	100.1
			Avg 99.1%

* Measured against an unfasted, untreated control rat of similar weight.

3 hours the animals were dispatched by a sharp blow to the head and the liver quickly isolated and weighed in ice cold distilled water. The tissue was minced and homogenized in a teflon pestled apparatus in 4 volumes of distilled water at 0°C. The homogenates were quickly frozen in an acetone dry-ice mixture and stored at –30°C before analysis. Aliquots of homogenate were analyzed for lipid peroxide by the thiobarbituric acid method(4).

Results and discussion. The results appear in Table I. The peroxide content of livers of untreated, non-fasted animals is arbitrarily set at 100%. There is no difference in control and experimental animals in regard to the amount of lipid peroxides in hepatic tissue at the 3-hour time period, when there are demonstrable morphologic alterations in the ergastoplasm and when there is clearly demonstrable depression of protein synthesis. It appears, therefore, that lipid peroxides do not play a significant role in the early phase of carbon tetrachloride intoxication.

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Isolation of a Virus from Infectious Bovine Kerato-Conjunctivitis. (27704)

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Infectious bovine kerato-conjunctivitis ("pink-eye") has a world-wide distribution. Outbreaks of the disease have been reported in India, South Africa, Holland, Canada, England and the United States(1). In one of the earliest detailed investigations of the disease, a classical description of its symptoms was presented(2). In this investigation, isolation of a pleomorphic bacillus from affected eyes was reported, but cultures of the organism failed to reproduce the disease in eyes of healthy cattle(2). Failure to induce the disease in cattle by inoculation of cultures of various types of organisms isolated from cases of the disease has been described(1). The possibility of Rickettsiae being associated with the disease has also been suggested(3). Infectious bovine rhinotracheitis virus (IBR) may cause changes of the ocular and palpebral conjunctiva, whereas symptoms of infectious bovine kerato-conjunctivitis (IBKC) are associated with changes of conjunctiva and cornea, which sometimes lead to loss of eye(2, 4,5). The rapid spread of IBKC through herds of susceptible cattle suggested a viral etiology of the disease. The present study was undertaken in an attempt to determine whether a virus was involved in the origin of this disease.

Materials and methods. Eye tissue obtained from cattle with acute infectious kerato-conjunctivitis was used as starting material.* Affected tissue was removed from eyes of infected cattle and shipped by a method previously described(6). Twelve le-

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