This study and previously cited literature suggest that analysis of blood for chlorinated hydrocarbon insecticides might be used as a clinical tool as well. The data of Dale *et al.* (*3*), however, suggest that, in mammals, it may not be possible to distinguish between a dose causing death and a dose producing only tremors.

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Adenovirus in Blood Clots from Cases of Infectious Hepatitis

Abstract. *Adenovirus type 5 was isolated from blood clots from 27 of 30 sporadic cases of infectious hepatitis. Only one isolation of virus, also adenovirus 5, was made from blood clots from 70 persons with no known contact with infectious hepatitis.*

Investigation of the etiology of infectious hepatitis in Arizona included efforts to isolate viruses from blood clots. Blood was collected in vacuum tubes without preservative or anticoagulant from sporadic cases and from family contacts. Specimens were refrigerated at approximately 8°C until processing, which was completed within 10 hours of collection. Serums were centrifuged at 1500 rev/min for 20 minutes and decanted from the clots. The clots were frozen and thawed rapidly three times in dry ice–alcohol bath, and 0.2 ml of the lysed clots was inoculated into each of two or four tubes of tissue culture of human embryonic lung cells (*1*). Tissue culture was grown in M-199 medium containing 15 percent calf serum and maintained in this medium containing 2 percent calf serum. Cultures were incubated in stationary racks at 37° to 39°C. On the initial passages, medium was changed 24 hours after inoculation to avoid toxic effects. A minimum of five blind passages was made at 5-day intervals before specimens were considered free of virus. For each passage, cultures were frozen and thawed rapidly three times, and 0.2 ml of the pooled material was inoculated into each tube of tissue culture. Cytopathogenic effects frequently were observed on the second passage, but usually complete destruction of the cell sheet was not obtained until fourth or fifth passage.

From 8 June to 29 December 1965, viruses were isolated during the acute phase of disease from 27 of 30 sporadic cases of infectious hepatitis. Age of patients ranged from 2 to 45 years. Viruses also were recovered from all of 12 family contacts of two cases, three of whom subsequently developed infectious hepatitis. These patients and contacts all lived in the vicinity of Phoenix, Arizona, but they were not concentrated in any particular areas.

Because of the regularity with which viruses were isolated from persons with infectious hepatitis, efforts were made to determine the prevalence of virus among people without signs or symptoms of infectious hepatitis. Blood specimens were obtained between 3 December and 29 December 1965 from 70 persons with no known contact with infectious hepatitis. These persons were matched with the age of patients as closely as possible. Examination of the specimens as described above resulted in one isolation of virus. During the same interval, viruses were isolated from all of the five sporadic cases available for study.

The viruses isolated from cases or contacts and the single virus isolated from the control group produced adenovirus type cytopathology and appeared to be adenovirus type 5 on the basis of serum neutralization tests made in human embryonic lung tissue culture. For these tests, 100 $TCID_{50}$ (tissue culture infective dose, 50 percent effective) was neutralized with dilutions of commercially prepared type-specific antiserums containing at least ten antibody units.

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Peroxidation of Liver Lipids in the Pathogenesis of the Ethanol-Induced Fatty Liver

Abstract. *Administration of an acutely intoxicating dose of ethanol produced significant increases in the concentration of liver triglyceride and enhanced the peroxidation of liver lipids in rats. Adipose triglyceride and lipid peroxide concentrations were unaltered. Coenzyme Q_4, an effective antioxidant, significantly inhibited accumulation of liver triglyceride following ethanol intoxication and prevented the peroxidation of liver lipids. These results, which demonstrate the selective ability of ethanol to induce peroxidation of liver lipids, together with the effectiveness of antioxidants, support the previously proposed hypothesis that peroxidation of liver lipids following ethanol intoxication is a factor in the pathogenesis of ethanol-induced liver injury.*

Previous studies have demonstrated the development of actute fatty infiltration of the liver, caused by an accumulation of triglyceride following oral administration of a single intoxicating dose of ethanol (*1–3*) or alcoholic beverages (*3*) to normal rats. Administration of an antioxidant prior to, or simultaneous with, ethanol was associated with inhibition of acute fatty liver induced by ethanol (*4–5*). Hypertriglyceridemia induced by simultaneous administration of ethanol and triglyceride was prevented by antioxidants (*4*); and accumulation of triglyceride in liver, as well as necrosis and mortality, following a lethal dose of carbon tetrachloride was also inhibited by either the intraperitoneal, oral, or intravenous administration of antioxidants (*5–7*). Results of these studies suggested the hypothesis that antioxidants protected ethanol-treated and carbon tetrachloride–exposed rats by inhibiting the formation of lipid peroxides, lipohydroperoxides, or other complexes (*4–7*).

To test this hypothesis, total lipids, triglycerides, and peroxide concentrations of liver and adipose tissue were measured following administration of an acutely intoxicating dose of ethanol.