did, and thus never developed urea retention.

In either case, freshwater river rays now provide excellent subjects for the study of urea metabolism and elasmobranch osmoregulation in general.

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12. The first location is on the border between Peru and Brazil about 40 km from the confluence of the Yavarí (Javary) and the Amazon; the second is about 152 to 160 km south-southwest of Leticia, Colombia (by river), about 8 km from the confluence of the Tichito and the Itacuai. The latter flows into the Yavarí, which in turn joins the Amazon.
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Virus-Induced Peliosis Hepatis in Rats

Abstract. *Inoculation of newborn Fischer, W/Fu, or Lewis rats with the 9H virus recently isolated from leukemic rat tissues resulted in the development of peliosis hepatis. The virus was recoverable from the peliotic livers. The cystic lesions were of the parenchymal peliosis type and appeared to be the result of focal hepatic necrosis followed by hemorrhage.*

The entity peliosis hepatis is characterized by blood-containing cystic lesions in the liver. A number of cases occurring in man, most frequently in patients with pulmonary tuberculosis, have been described. It also has been observed in patients with other chronic wasting diseases, such as cancer, and in patients receiving androgens therapeutically (*1*). Several investigators have observed what appear to be peliotic lesions of the liver, spleen, and adrenal glands in mice after transplantation of ovarian or testicular tumors (*2*). A similar lesion also occurs spontaneously in animals, particularly cattle, and is referred to as "telangiectasis of the liver" (*3*).

The etiology and mechanisms of development of peliosis hepatis are unknown, although several theories have been proposed (*1*, *4*). To our knowledge, a viral cause of this disease has neither been previously demonstrated nor proposed in animals or humans. We now describe the induction of peliosis hepatis in rats with the 9H virus recently isolated from the same species (*5*).

The virus strain used in our experiments was extracted from leukemic liver and spleen cells maintained in vitro. The cells and supernatant of the culture were frozen and thawed three times and then centrifuged at 10,000*g* for 10 minutes. The resulting supernatant was filtered through a Millipore filter impermeable to bacteria (0.45 μ). The filtrate was centrifuged at 73,000*g* for 1 hour. The resulting pellet was again suspended in an amount of Hanks' salt solution containing 2 percent calf serum (HCS) which represented one-tenth of the original volume of the culture fluid. This material was negative when tested (*6*) for mycoplasma. Upon inoculation of a line of rat embryo cell cultures (REL) (*5*), hemagglutinins appeared in the supernatant, and cytopathic effects became evident within 5 to 7 days. Intraperitoneal inoculation into 15 newborn Fischer rats produced disease in four animals, with clinical symptoms as described below. Three more consecutive passages were made in Fischer rats with pooled liver-spleen extracts of the diseased rats.

To compare the incidence and pathology of the disease in different strains, as well as to attempt to recover the virus in vitro, we injected Fischer, W/Fu, and Lewis rats with liver extracts of the fourth Fischer rat passage. The extracts were prepared as follows: The tissue was weighed and homogenized with alundum and HCS, as a 20 percent (by weight) suspension. Coarse debris and alundum were removed by low-speed centrifugation. The extract was subsequently treated with high-frequency sound by means of a Raytheon 10-kc oscillator for 1 minute. It was partially purified by centrifugation at 2400*g* for 20 minutes and then centrifuged at 105,000*g* for 1 hour. The resulting pellet was again suspended in an amount of HCS equivalent to 0.5 g per milliliter of the original weight of tissue. This concentrated extract did not agglutinate guinea pig erythrocytes; however, the presence of the virus was ascertained when eight REL cell cultures, inoculated with the extract, developed typical cytopathology. Hemagglutinins also appeared in the culture supernatant within 7 days. Other samples of the extract were injected intraperitoneally (0.1 ml) into newborn rats. Thirty W/Fu rats (100 percent), 30 of the 36 Fischer rats (83 percent), and 11 of the 13 Lewis rats (85 percent) inoculated with the extract became ill and moribund within 12 to 30 days. Thirty control rats, consisting of untreated animals and rats inoculated with heated extract (86°C for 30

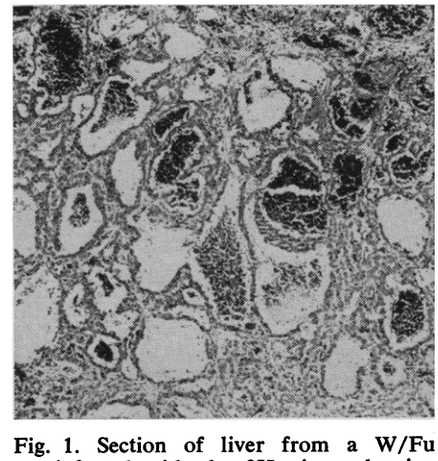

Fig. 1. Section of liver from a W/Fu rat infected with the 9H virus, showing multiple blood-containing cysts of various sizes lined by hepatic cells; hematoxylin-eosin stain. (× 48)

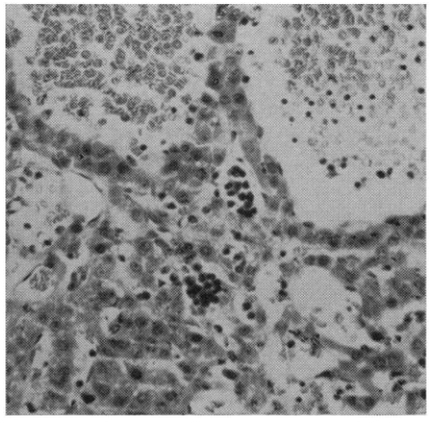

Fig. 2. Details of portions of two fully developed peliotic cavities (top) with degeneration, necrosis, and dissociation of adjacent hepatic cells. Peliotic cavity in process of formation is shown in lower right area. Two foci of extramedullary hematopoiesis are in center. (× 214)

minutes) or extracts of normal rat livers, were not affected.

The clinical and pathological features were identical in all three strains of rats inoculated with the virus; however, there was variation in the degree of severity among individual animals. At the time of onset of clinical symptoms, the animals were pale and inactive. They died a few days later. At autopsy, the livers were pale consistently, but, beneath their capsules and on the cut surfaces, there were scattered, minute, dark, hemorrhagic foci. The livers adhered to the diaphragms. The spleens were usually considerably smaller than those of the control rats and often adherent to the stomach. Hemorrhagic ascites was observed in some of the animals. All other organs appeared normal.

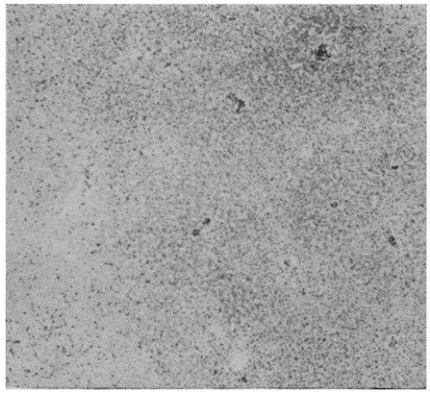

Fig. 3. Section of liver from a W/Fu control rat, showing intact architecture for comparison with Fig. 1. (× 48)

In the sections of liver obtained from rats that either died or were killed, numerous cystic spaces containing blood were observed (Figs. 1 and 2). The spaces had no relation to any particular zone within the liver lobules and were lined by hepatic cells. Mitotic figures were evident in some of the lining cells.

In addition to the blood cells, macrophages and hepatic cells, showing evidence of degeneration or necrosis, were seen in the lumens of some of the cysts. Some of the cystic lesions were separated from adjacent ones by only a single row of hepatic cells. Frequently, cysts communicated with each other in the areas where only remnants of the original partition, consisting of hepatic cells showing varying degrees of degeneration, were seen. In some sections, hemorrhagic cysts extended up to the surface of the liver causing an elevation of the capsule. Areas of fatty metamorphosis, foci of necrosis, and areas of dissociation of hepatic cells were observed in the rest of the hepatic parenchyma. Inflammation was not evident, but there were foci of extramedullary hematopoiesis. As a rule, the spleens of the infected animals showed some evidence of atrophy of the lymphoid tissue. No prominent changes were evident in other tissues (brain was not examined) except foci of extramedullary hematopoiesis. Except for the presence of slight congestion, there was no lesion in the livers of the control animals (Fig. 3). Also, foci of extramedullary hematopoiesis seemed to be less conspicuous than in the infected rats.

The cystic liver lesions in the infected animals of these experiments are characteristic of peliosis hepatis of the parenchymal type in humans (*1*). They are apparently the result of focal hepatic necrosis and hemorrhage after infection with the 9H virus. These findings suggest a possible new etiological factor for consideration in further investigations on peliosis hepatis in other species. They also provide a model system in rats for studies on the pathogenesis of this disease and the nature of the inducing virus.

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Potato Spindle Tuber Virus: A Plant Virus with Properties of a Free Nucleic Acid

Abstract. *Infectious entities, extractable, with phosphate buffer, from tissue infected with potato spindle tuber virus and inciting symptoms on tomato that are typical of this virus, have properties incompatible with those of conventional virus particles. The infectious particles sediment in sucrose density gradients at approximately the same rate as particles with a sedimentation coefficient of 10S, are insensitive to treatment with organic solvents, and can be concentrated by ethanol precipitation. Treatment with phenol changes neither their infectivity nor their sedimentation properties. Infectivity is insensitive to deoxyribonuclease, but at low ionic strength it is sensitive to ribonuclease. At high ionic strength, infectivity partially survives incubation with ribonuclease. These properties, as well as elution patterns from columns of methylated serum albumin, suggest that the extractable infectious agent may be a double-stranded RNA.*

Potato spindle tuber has been recognized as an important disease of potatoes for many years (*1*). In nature, the potato spindle tuber virus (PSTV) is spread primarily by mechanical means almost as readily as potato virus X (PVX) is (*2*, *3*). Symptoms incited by PSTV in potato foliage are often very difficult to detect, and study of the causal virus was hampered by the lack of a suitable indicator plant until Raymer and O'Brien (*4*) discovered that this virus also causes distinctive symptoms on tomato plants. In 1964, Allington *et al.* (*5*) reported that potato