susceptibility by IP injection. All groups were similar in sensitivity to the virus. The titer in channel catfish was $1\times10^{3.29}$ LD_{50}, blue catfish was $1\times10^{4.5}$ LD_{50}, channel female × blue male was $1\times10^{4.87}$ LD_{50}, and blue male × channel female was $1\times10^{4.08}$ LD_{50}.

In a CCV titration in blue catfish, the initial mortality of those injected with 3.16×10^{5} $TCID_{50}$ occurred 36 hours after injection, and, after 60 hours, 100 percent of these fish were dead. Blue catfish injected with 3.16×10^{4} $TCID_{50}$ had 100 percent mortality after 84 hours. Approximately 80 percent of those fish injected with 3.16×10^{3} or 3.16×10^{2} $TCID_{50}$ died; 70 percent of those injected with 3.2×10^{1} $TCID_{50}$ died; and 20 percent of the fish injected with 3.2×10^{0} $TCID_{50}$ died. No control fish were lost.

Intraperitoneal injection was the only successful method of infecting blue catfish. Feeding virus and cohabitation with infected channel catfish were unsuccessful in establishing infection in blue catfish, although these methods worked well for infected channel catfish. CCV replication reached a peak in blue catfish 42 hours PI when $1\times10^{5.8}$ $TCID_{50}$/gm of viscera were isolated. The channel female × blue male fingerlings reached a similar peak of replication 72 hours PI; however, the channel catfish fingerlings reached a peak of $1\times10^{2.7}$ $TCID_{50}$/gm of viscera 72 hours PI.

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MFR PAPER 1335

Viral Diseases of the Blue Crab, *Callinectes sapidus*

PHYLLIS T. JOHNSON

To date, 13 viruses have been found in crustaceans (Bang, 1971; Bazin et al., 1974; Bonami and Vago, 1971; Bonami et al., 1971; Chassard-Bouchaud et al., 1976; Couch, 1974; Federici and Hazard, 1975; Johnson, 1976a, b; Johnson and Bodammer, 1975; Vago, 1966). Eleven of them are from decapods, and 4 of the 11 occur in the blue crab.

Known viruses of the blue crab include: 1) a *Baculovirus* that infects the hepatopancreas (Fig. 1); 2) a reolike virus found in hemopoietic tissue, hemocytes, certain other mesodermal cells, and some ectodermal cells, including the general epidermis and probably the glia of the central nervous system (Fig. 2); 3) a herpeslike virus that is mainly confined to hemocytes (Fig. 3); and 4) a picornalike virus that attacks ectodermal elements, including neurosecretory cells, epidermis, and the bladder epithelium (Johnson[1]) (Fig. 4). Occasionally, the latter also infects hemocytes and hemopoietic tissues. A fifth entity which has many characteristics of the Rhabdoviridae has been found in crabs also infected with any of the last three viruses (Fig. 2), and it may represent a virus that manifests itself mainly in stressed animals (Johnson, footnote 1).

The *Baculovirus* apparently does not cause overt disease in its host. It usually infects only scattered cells, and, since tissue replacement is occurring constantly in the hepatopancreas, the damage caused usually must be minimal. The *Baculovirus* occurs in all

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[1]Johnson, P. T., Oxford Laboratory, Northeast Fisheries Center, National Marine Fisheries Service, NOAA, Oxford, MD 21654. Unpublished observations.

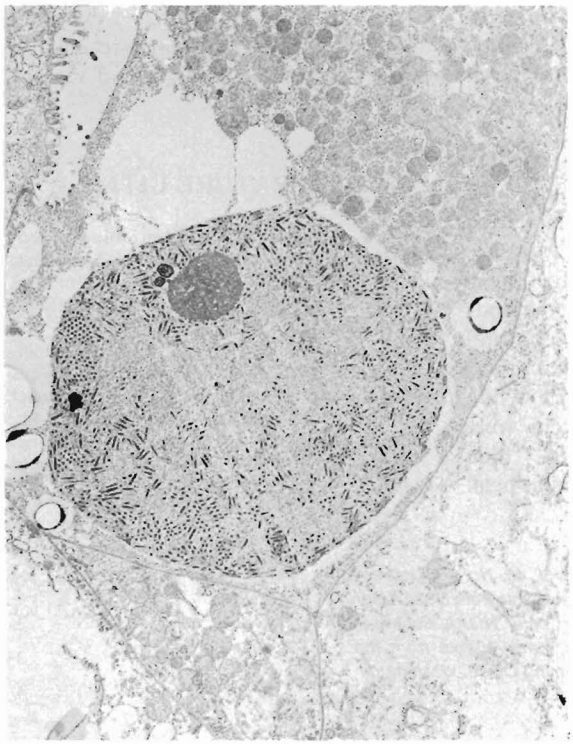

Figure 1.—*Baculovirus* in a nucleus of the epithelium of the hepatopancreas. The rodlike particles often occur in paracrystalline arrays. 5,500×.

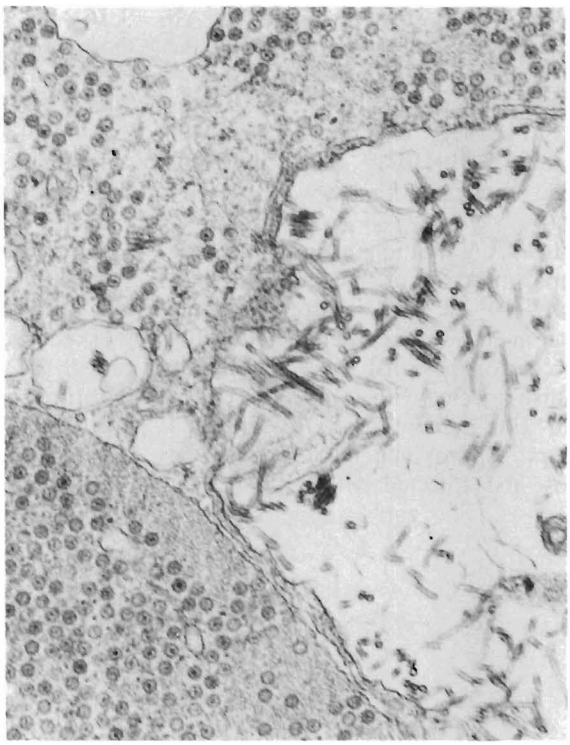

Figure 2.—Reolike virus and rhabdolike virus infecting the cytoplasm of hemocytes. The icosahedral particles of the reolike virus are in the cytoplasm, while the rhabdolike virus appears as sinuous tubules within the enlarged endoplasmic reticulum. 27,700×.

populations of the blue crabs that I have sampled.

The reolike virus (RLV) causes a fatal infection. If infected hemolymph is injected into the body cavity of a crab, the virus may kill within 3 days, while crabs fed infected tissues usually take 12-32 days to die. There is massive destruction of the hemopoietic tissue and young hemocytes. Marked necrosis of the neuroglia, possibly caused by RLV, also occurs. Neurological damage may be the actual cause of death in such cases. Symptoms include lack of molting, sluggishness, trembling of the appendages, paralysis, and disorientation (Johnson, 1977). These symptoms may be largely dependent on destruction of the nervous tissues. RLV has been found in crabs from both Chincoteague and Chesapeake Bays.

The herpeslike virus (HLV), which infects mainly hemocytes, causes fatalities in injected crabs only after 30-40 days, and natural infections may take much longer before causing death. Symptoms are not manifested until infection is terminal, and death is probably due to the massive destruction of hemocytes. The hemolymph of moribund animals is chalky white, due to the presence of virus particles and fragments from lysed cells. HLV has been found only in crabs taken from Assawoman Bay, Del., and Chincoteague Bay, Va.

The picornalike virus (CBV) was found in a group of about 100 young crabs that had been collected for use in nutrition studies. Over a 2-month period, most of the crabs died of their infection. CBV has a predilection for ectodermal tissues; apparently hemopoietic tissue and hemocytes are only secondarily infected, and sometimes they are not attacked at all. Many of the symptoms displayed are probably due to destruction of neurosecretory cells. The molt pattern is interfered with; often the animals are blinded; and disorientation is evident. It may take a month after the first symptoms are manifested before death occurs. The tissues attacked differ according to the particular crab, and the leisurely course of the disease in some crabs is probably due to the fact that vital centers were not attacked until late in the infection. As well as disrupting the neurosecretory systems, some infections involve extensive destruction of the gill epithelia, thus greatly reducing respiration and interfering with osmotic control.

The impact of these viruses in a natural population has not been investigated. They can cause serious mortalities in captive populations, as has been shown. Holding under artificial conditions causes stresses probably not usually encountered in the wild, such as crowding and inadequate

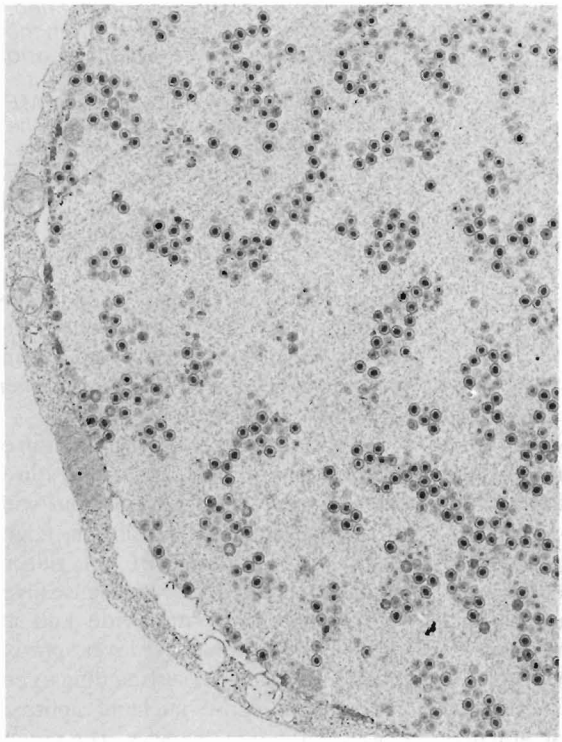

Figure 3.—Herpeslike virus in the greatly enlarged nucleus of a hemocyte. The cytoplasm is reduced to a thin rim surrounding the nucleus. 5,500×.

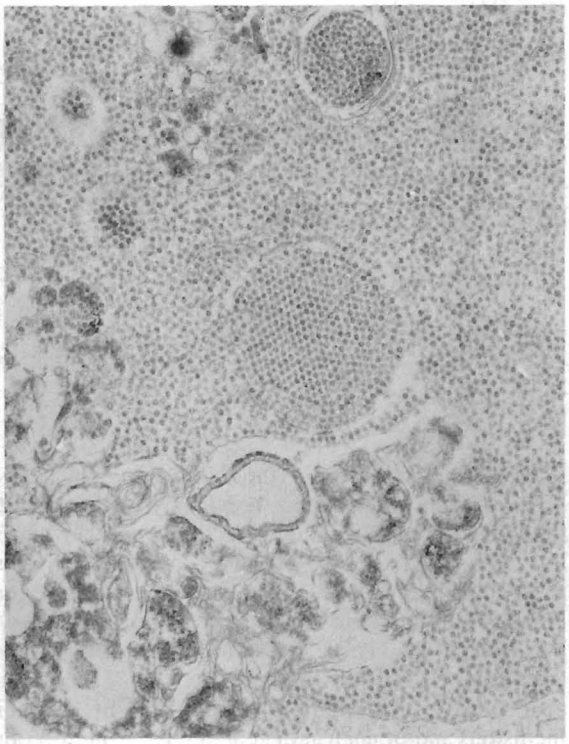

Figure 4.—Picornalike virus infecting a cell of the bladder epithelium. Note the association of the icosahedral particles with membranes. 27,700×.

diet. To date, histopathological examination has been the only sure method of determining presence of virus infection. Other more rapid means must be devised to assess properly the extent of virus infection and of virus-induced mortality in wild populations.

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