Table 1. Catch record (numbers) for Pogonophora collected from the New England continental slope on 18 June 1962. The combined density of *Siboglinum* sp. and *S. ekmani* at station 54 was 30 per square meter and at station 55 it was 25 per square meter.

Types	Station	
	54	55
Siboglinum ekmani		
Animals	1	3
Tubes	0	7 frags.
Siboglinum sp.		
Animals	5	2
Embryos	2	0
Tubes	6	0

is a new one and is now being studied by E. C. Southward.

The species *S. ekmani* has been reported from only two localities, both of which are in the eastern North Atlantic area—the Skagerrak (*3*) and southwest of Ireland (*4*). Thus, its occurrence in New England waters reveals a marked westward extension of its known geographic range. Catch records are much too scant to show geographic distributional patterns for individual species in this phylum. The records have so far disclosed little evidence of cosmopolitan or even ocean-wide distribution, which would generally be expected of bathyal species. Consequently, the records for *S. ekmani* from both sides of the Atlantic are an indication that future studies are likely to reveal widespread distribution for other species in this phylum.

Our specimens were collected from depths of 366 m and 567 m, which is somewhat shallower than the reported depth range of European collections of *S. ekmani*. The known depth range for this species in the Skagerrak is 487 to 650 m (*3*) and southwest of Ireland it is 620 to 1280 m (*4*). Since we did not sample deeper than 567 m, their maximum depth of occurrence in the New England region remains unknown. However, we have negative evidence indicating the minimum-depth boundary for both *Siboglinum* sp. and *S. ekmani* in this region is between 180 and 366 m. This evidence is based on the absence of pogonophorans in over 100 samples of benthic fauna from 64 stations uniformly spaced over an area of 12,000 square kilometers in the shallower water (35 to 180 m) adjacent to stations 54 and 55.

Quantitative information on members of this phylum is also sparse, and data given here are limited; however, the results from our samples are reasonably consistent. The catch records, as shown in Table 1, indicate a density of approximately 30 per square meter, both species combined, with *S. ekmani* only about half as abundant as *Siboglinum* sp. Total macrobenthos averaged 1700 specimens per square meter at station 54 and 700 at station 55. Although the numerical density of the pogonophorans was moderately low, these organisms were a significant component at the deeper station. Because of their moderately low density and small size (average diameter about 0.2 mm and length 10 cm), these species formed only a minor portion of the benthic biomass.

The principal organisms with which the New England pogonophorans were associated were generally the same at both localities. At station 54 the dominant organisms were: polychaete worms (Amphinomidae, Chaetopteridae, Maldanidae), foraminiferans (Astrorhizidae and Saccamminidae), and small sipunculoids. At station 55, which was deeper, the dominant organisms were: polychaete worms (Lumbrineridae, Capitellidae, Cirratulidae) and foraminiferans (Astrorhizidae and Saccamminidae).

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Epizootic Diarrhea of Infant Mice: Identification of the Etiologic Agent

Abstract. *Electron micrographs of intestinal epithelium of infant mice infected with epizootic diarrhea virus have demonstrated intracellular spherical structures measuring 65 to 75 mμ in diameter which have a complex morphology resembling several virus particles. They have ben interpreted as being the etiologic agent of this disease. The particles were present in association with, in some cases within, vesicles of the endoplasmic reticulum of intestinal epithelial cells. They were never seen in the nucleus.*

The disease which has come to be known as epizootic diarrhea of infant mice (EDIM) was first described in 1947 (*1*). Recent transmission experiments (*2*) have shown that this disease is infectious and communicable, and that the etiologic agent appears to be a fairly heat-resistant virus capable of serial transfer and susceptible of neutralization by specific hyperimmune rabbit antiserum. It is a highly contagious disease. It is widespread among mouse colonies and can thus be a potential cause of considerable difficulty in laboratories engaged in research on neoplasia and other viruses. Difficulty might be greatest where viral morphology is being studied in neoplastic tissues, for EDIM virus can infect adult mice without causing overt disease; viremia, however, may occur in such animals (*3*). It is thus possible that any organ may display EDIM virus particles when experimental mice come from a diarrheal colony.

In an attempt to obtain electron microscopic identification of the EDIM virus particle, 1- to 3-day-old mice were infected by oral administration of an appropriate dilution of purified infective intestinal filtrate. Within 1 day after onset of overt symptoms (3 to 5 days after exposure to the virus) the mice were killed, the small intestine was immediately exposed, and portions of the jejunum were fixed in osmium tetroxide. The tissues were then embedded in methacrylate, and sections were cut with a glass knife on a Porter-Blum type microtome for examination in an electron microscope (RCA EMU-2E). Normal tissue from uninfected mice of similar age was treated in identical fashion.

The technical methods used for the electron microscopy have been standard in this laboratory for the past several years and are described elsewhere (*4*). The methods of preparing, storing, and passaging the virus have been described in detail (*2*, *3*).

Figure 1 illustrates the typical appearance of the spherical particles found in the cytoplasm of the epi-

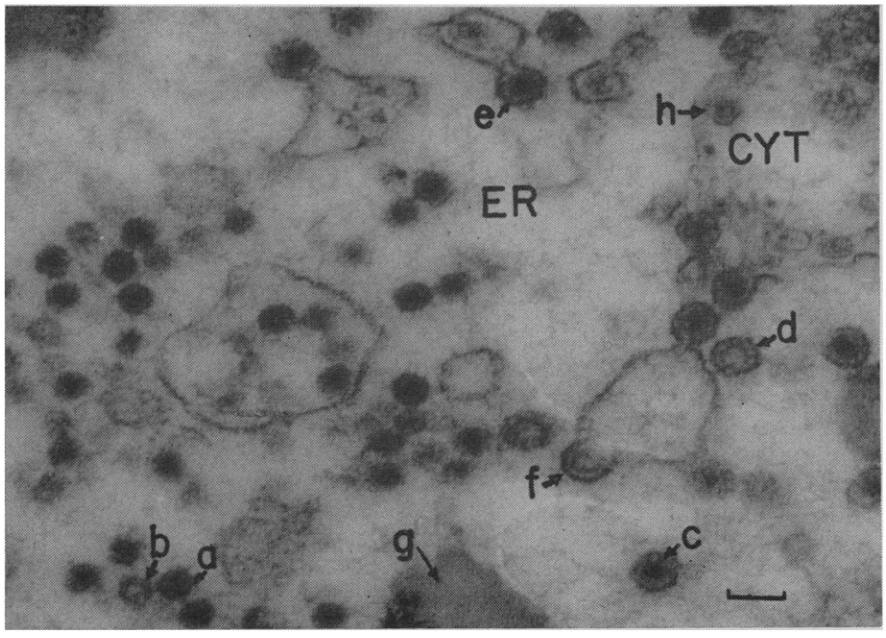

Fig. 1. Electron micrograph of cytoplasm of an epithelial cell from the jejunum of an infant mouse infected with EDIM virus, illustrating the different morphological forms of the virus described in the text. Most of the particles lie within a dilated vesicle of endoplasmic reticulum (*ER*), but some appear in the tongue of cytoplasm visible at upper right (*CYT*). The line at lower right represents 100 mμ.

thelial cells from the small intestine of infected mice. The particles varied in diameter from 65 to 75 mμ, and two distinct classes can be identified. In the smaller class (*a*, *b*) the particles measured about 65 mμ in diameter and were composed of a thick coat of medium density with an outer indistinct margin which did not have the sharply delimited characteristics of a typical membrane. This class of particle had an electron-dense center (*a*) or, less commonly, an electron-lucid center (*b*). The larger class (*c*, *d*) was similar to the first, with the exception that there was the addition of an outer sharply delimited membrane increasing the diameter to approximately 75 mμ. This class, like the first, had either an electron-dense center (*c*) or an electron-lucid center (*d*). Always associated with the virus particles within the dilated vesicles of endoplasmic reticulum were varying quantities of homogeneous, finely granular material of medium electron density, arranged in irregular masses (*g*).

The particles appeared to be most numerous within the vesicles of the endoplasmic reticulum and, in some cases, they appeared to be closely associated with the membrane of the endoplasmic reticulum in a manner suggesting possible formation at the membrane (*e*, *f*). Rarely, particles (*b*) were present outside the endoplasmic reticulum within the matrix of the cytoplasm at some stage in their development. In no case have the particles been found within the nucleus of the epithelial cells. No particles were seen in normal infant mouse intestine.

Transmission, infectivity, and purification studies (*2*) have yielded evidence that the agent of epidemic diarrhea of infant mice is a virus. The electron micrograph shown here reveals the presence of spherical particles that have the characteristic morphology of virus particles with dimensions compatible with filtration studies of EDIM virus. They have been tentatively identified as the EDIM virus particle (*5*).

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Complement-Fixing Properties of Pepsin-Treated Rabbit and Sheep Antibodies

Abstract. *Rabbit antibody with a sedimentation coefficient of 7S was digested with pepsin to yield the 5S fragment. Washed specific precipitates of 5S antibody and egg albumin, when added to guinea pig serum, fixed up to 40 percent of the total complement. This fixation could not be attributed to contamination with 7S antibody. Absorption of guinea pig serum with washed precipitates of 5S antibody and antigen removed that portion of complement which was fixed by fresh 5S antibody-antigen aggregates, but not that fixed by 7S antibody-antigen aggregates. The presence of 0.001M ethylenediaminetetraacetic acid prevented this absorption of complement by the 5S aggregates.*

Pepsin digestion of rabbit antibodies with a sedimentation coefficient of 7*S* yields a smaller 5*S* fragment, which precipitates antigen, but supposedly lacks all of Porter fragment III (*1*). Fragment III can fix complement (C′) when it is aggregated, but fragments I and II cannot (*2*). The conclusion has been drawn that it is fragment III in antibody that fixes complement and that the 5*S* fragment, though precipitable, does not fix complement (*3*).

Reinvestigation of this problem for other purposes led to the finding that 5*S* rabbit as well as 5*S* sheep antibody does retain certain complement-fixing activity. Because these results are important not only to the theories of antibody structure, but also in the interpretation of experiments testing the biologic activity of 5*S* antibody, they are briefly reported here.

Antisera were obtained from sheep and rabbits immunized with human serum albumin and egg albumin in complete Freund's adjuvant. The 7*S* gamma globulin was isolated by sodium sulfate precipitation (*4*) and then dialyzed against phosphate saline buffer (*p*H 6.9, 0.17*M*). It was digested with 2 to 3 percent pepsin in acetate chloride buffer (*p*H 4.1, 0.12*M*) for 18 to 24 hours at 37°C. The smaller 5*S* fragment was isolated by sodium sulfate precipitation, and then dialyzed against phosphate saline buffer. In the ultracentrifuge it gave a single symmetrical peak with a sedimentation coefficient of 5*S*. There was no evidence of any component with a higher sedimentation coefficient. Antibody and antigen nitrogen