The work described here was carried out in part at the Applied Physics Laboratory of the Johns Hopkins University and the Catholic University of America.

S. D. BRUCK

National Heart and Lung Institute,
National Institutes of Health,
Bethesda,
Maryland 20014

Received February 27; revised March 30, 1973.

1 Bruck, S. D., *J. Biomed. Mater. Res.*, **6**, 173 (1972).
2 Bruck, S. D., *Trans. Amer. Soc. Artif. Intern. Organs*, **18**, 1 (1972).
3 Bruck, S. D., *Biomat., Med. Dev., Art. Org.*, **1**, 79 (1973).
4 Foster, E. D., Smith, M. R., and Dobell, A. R. C., *Can. J. Surg.*, **15**, 339 (1972).
5 Salzman, E. W., *Blood*, **38**, 509 (1971).
6 Sawyer, P. N., and Pate, J. W., *Amer. J. Physiol.*, **175**, 103 (1953).
7 Szent-Györgyi, A., *Nature*, **157**, 875 (1946).
8 Szent-Györgyi, A., *Bioenergetics* (Academic, New York, 1957).
9 Evans, M. G., and Gergely, J., *Biochim. Biophys. Acta*, **3**, 188 (1949).
10 Eley, D. D., Parfitt, G. D., Perry, M. J., and Taysum, D. H., *Trans. Faraday Soc.*, **49**, 79 (1953).
11 Taylor, B. C., Sharp, W. V., Wright, J. I., Ewing, K. L., and Wilson, C. L., *Trans. Amer. Soc. Artif. Intern. Organs*, **17**, 22 (1971).
12 Bruck, S. D., *Polymer*, **6**, 319 (1965).
13 Bruck, S. D., *J. Polymer Sci.*, Part C, **17**, 169 (1967).
14 Bruck, S. D., *Ind. Eng. Chem.*, **59**, 18 (1967).
15 Bruck, S. D., *Polymeric Organic Semiconductor*, US Patent No. 3,362,917 (January 9, 1968).
16 Bruck, S. D., *Polymeric Organic Semiconductor*, Canadian Patent No. 764,338 (August 1, 1967).
17 Bruck, S. D., *J. Polymer Sci.*, part A-1, **8**, 771 (1970).
18 Frankel, S., Reitman, S., and Sonnenwirth, A. C. (eds.), *Gradwohl's Clinical Laboratory Methods and Diagnosis*, 1 (Mosby, Saint Louis, 1970).
19 Packham, M. A., Warrior, E. S., Glynn, M. F., Senyi, A. S., and Mustard, J. F., *J. Exp. Med.*, **126**, 171 (1967).

Intestinal Adenomatosis in the Pig: Occurrence of a Bacterium in Affected Cells

ENTEROPATHY of the weaned piglet, originally described by Biester and Schwarte[1], has been further reported by Rowland and Rowntree[2]. Affected pigs show adenomatous proliferations of the intestinal mucosa, principally of the lower ileum and caecum. The affected tissue consists of glands lined by vigorously proliferating immature epithelial cells throughout the entire depth of the mucosa, with loss of villi and often the development of frankly polypoid masses. Goblet cells do not form. In the absence of secondary infection, most animals recover within about 6 weeks and the intestine is normal at slaughter. Histochemical studies have shown the adenomatous cells to be lacking the more common enzymic activities of normal mature villous epithelium.

Immunofluorescent studies, using a fluorescein isothiocyanate (FITC) conjugated serum prepared from an affected pig, appropriately absorbed with pig tissue, have demonstrated, in the cases so far examined, particulate fluorescence in the apical cytoplasm of affected cells throughout the depth of the mucosa. These animals originated from several farms.

Further proof of the specificity of this reaction is afforded by the removal of fluorescent activity from the serum after absorption with affected intestinal mucosa but not normal intestinal mucosa from the same animal.

Electron microscopic examination of conventionally prepared and cryostat material has consistently identified an irregularly curved bacterium within abnormal cells situated in the same areas in which fluorescence has been observed. Attempts to identify the presence of these organisms by the usual bacterial stains, either in section or in smears of affected intestinal mucosa, have been unsuccessful. The ultrastructure of the intracellular bacterium is simple and we have been unable to observe any specialized cell structures, such as axial fibres, which have been present in intracellularly located spirochaetes in swine[3].

Cultural examination of affected intestinal mucosa has resulted in the isolation of an organism with a similar morphology to that seen by the electron microscope in the adenomatous lesions. These bacteria have the characteristics of a vibrio, but initial studies suggest that the organisms differ biochemically from most vibrio species isolated from the pig by other workers[4-7]. The vibrios obtained from adenomatous lesions show a degree of antigenic homogeneity, although preliminary work indicates some antigenic differences between isolates. In contrast, none of these organisms is agglutinated significantly by a series of five antisera (unpublished results of G. H. K. L. and J. Hannah) prepared against whole cells of isolates considered to be *V. coli*, recovered from the large intestine of pigs. These latter have characteristics similar to those described by Lussier[7]. The vibrio occurring in the proliferative intestinal lesions has been demonstrated to be present in three cases, from which it has been isolated in numbers approximating to 15×10^6 per g weight wet tissue.

Hyperimmune serum against the vibrio isolates has demonstrated, using a "sandwich" technique, specific intracellular fluorescence at a similar site to that observed using conjugated porcine immune serum.

Within the cell, the organisms are not contained within vacuoles or other membrane-bound structures. This relationship seems unusual, although bacteria have recently been described free in the cytoplasm of affected hepatic cells of the Mongolian gerbil in Tyzzer's disease[8].

One must be extremely cautious at this stage in drawing conclusions regarding any interaction between the intracellular bacteria and the proliferative intestinal changes observed, especially in view of the long and controversial history of the possible pathogenicity of various vibrio species in the pig. Even in the absence of specific cause and effect, however, the host-parasite relation in these circumstances may prove of considerable interest and possible significance.

A. C. ROWLAND
G. H. K. LAWSON
A. MAXWELL

Department of Veterinary Pathology,
Royal (Dick) School of Veterinary Studies,
Veterinary Field Station,
Easter Bush, Roslin,
Midlothian

Received March 1, 1973.

1 Biester, H. E., and Schwarte, L. H., *Amer. J. Pathol.*, **7**, 175 (1931).
2 Rowland, A. C., and Rowntree, P. G. M., *Vet. Rec.*, **91**, 235 (1972).
3 Taylor, D. J., and Blakemore, F. W., *Res. Vet. Sci.*, **12**, 177 (1971).
4 Doyle, L. P., *Amer. J. Vet. Res.*, **9**, 50 (1948).
5 Roberts, D. S., *Austral. Vet. J.*, **32**, 116 (1956).
6 Deas, D. W., *Vet. Rec.*, **72**, 65 (1960).
7 Lussier, G., *Canad. Vet. J.*, **3**, 267 (1962).
8 Port, C. D., Richter, W. R., and Moize, S. M., *Lab. Invest.*, **25**, 81 (1971).

Pepsin Inhibitory Activity amongst Activation Peptides of Pepsinogen

THE concept of the pepsin inhibitor (PI) and a method for its isolation in crystalline form from the acid-activated pepsinogen (Pg) system, in which pepsin (P) and other peptides (pp)