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Interaction of Microorganisms with Enterocytes After Oral Administration of Pesticides

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Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 123, No. 4, pp. 467-470, April, 1997
Original article submitted November 22, 1996

Intragastral administration of the pesticides Sumi-alpha and Omait to rats significantly increases the number of parietal microorganisms in the jejunum, ileum, and particularly in the cecum. Electron microscopy shows that parietal microorganisms invade goblet cells during secretion and then enter prismatic cells via the lateral plasma membrane. The number of parietal microorganisms entering enterocytes after Sumi-alpha is higher than after less toxic Omait, reaching the maximum 5 h after administration.

Key Words: *parietal microorganisms; enterocytes; pesticides*

Pesticides produce various adverse effects on animals and humans. Worldwide, about 4 mln tons of pesticides are used in each year, but only about 1% of them reach the target. Annual record of pesticide poisoning is almost 500,000 cases [5,7,8]. However, pesticides are indispensable for modern agriculture.

A vast majority of pesticides have adverse effects after ingestion with water or food. Ingested pesticides directly affect the epithelial lining of mucous membranes and so-called parietal microorganisms (PM) of the digestive tract [1-3]. Interactions of PM with mucosal structures of the small intestine, from which most nutrients [8] and pesticides are absorbed, are analyzed in the present study.

MATERIALS AND METHODS

Using optical and transmission electron microscopy, interactions of PM with enterocytes of the jejunum, ileum, and cecum in Wistar rats (>100 g) were studied after oral administration of the moderately toxic pesticide Omait (Juniroll Chemicals; LD_{50}=815 mg/kg for rats) or the highly toxic pesticide Sumi-alpha (Sumimoto Chemicals; LD_{50}=75 mg/kg for rats) in a dose of 1.0 ml/100 g body weight [4]. Both pesticides are supplied as a concentrated emulsion.

The pesticides were administered intragastrally via a gastric tube. Intact rats served as controls. Five hours, and 1, 3, 7, 15, and 30 days after administration the rats were decapitated, and intestinal specimens were collected. They were fixed in 2.5% glutaraldehyde, postfixed 1% osmium tetroxide, double stained, embedded in Epon—Araldite, and processed for electron microscopy (Hitachi H-600). Semithin sections stained with methylene blue—fuchsin were examined by light microscopy, with measurement of the relative volumes of PM (expressed in %) in areas located 40-45 μ from the plasma membranes of enterocytes [1,2].

RESULTS

In the control rats, the occurrence of PM was the highest in iliac crypts (2.5±0.1%) and cecum (5.2±0.1%). In pesticide-treated rats, PM were present in

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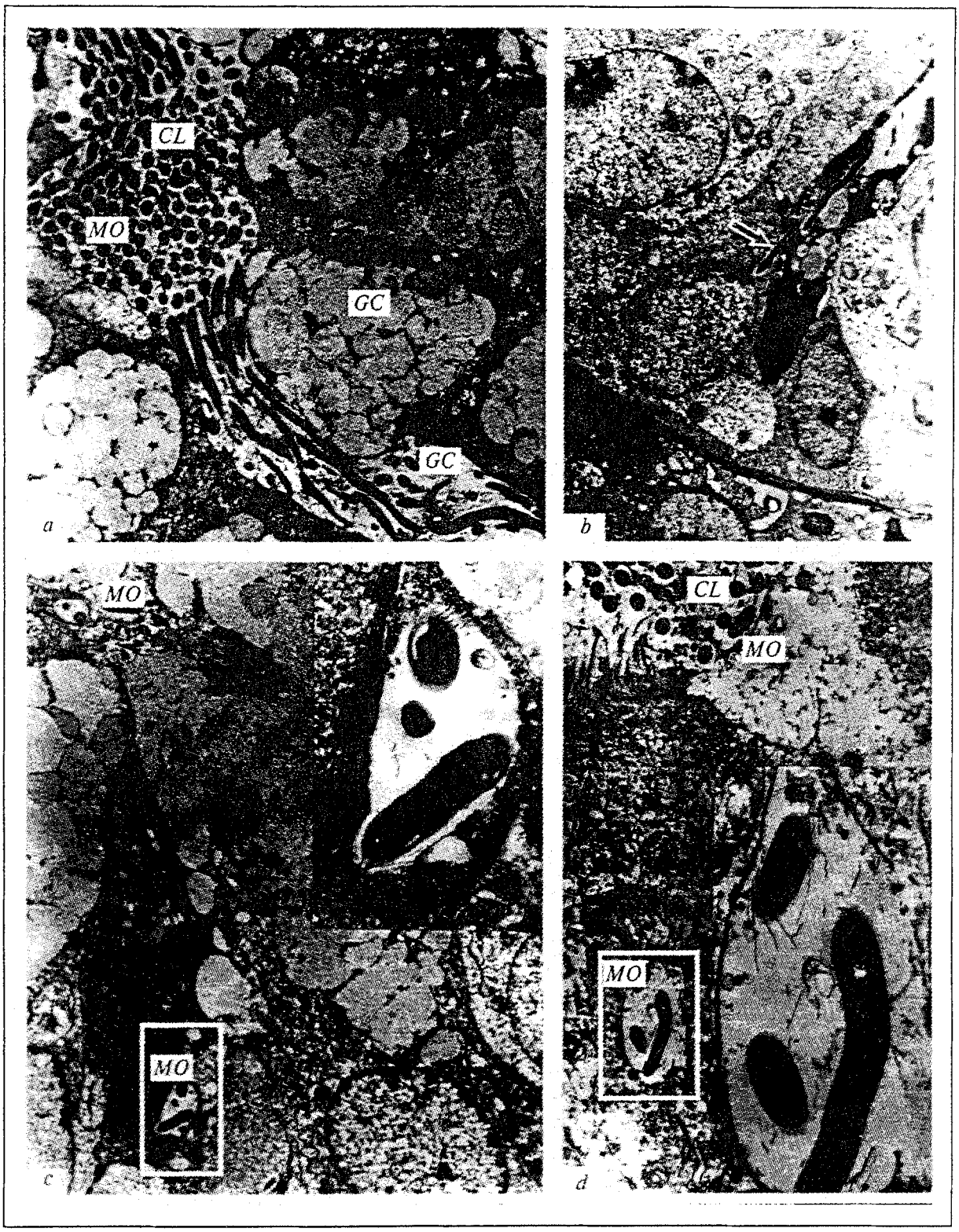

Fig. 1. Interaction of microorganisms with cecal enterocytes 5 h after oral administration of the pesticide Sumi-alpha.
a) numerous microorganisms (*MO*) are present in cryptal lumen (*CL*) and have invaded a goblet cell (*GC*), ×7500);
b) microorganisms invading plasma cell (arrow) through the lateral membrane, ×5500;
c) microorganisms in the prismatic cell, ×9000 (inset: ×40,000);
d) microorganisms in the basal part of prismatic cell, ×7500 (inset: ×40,000).

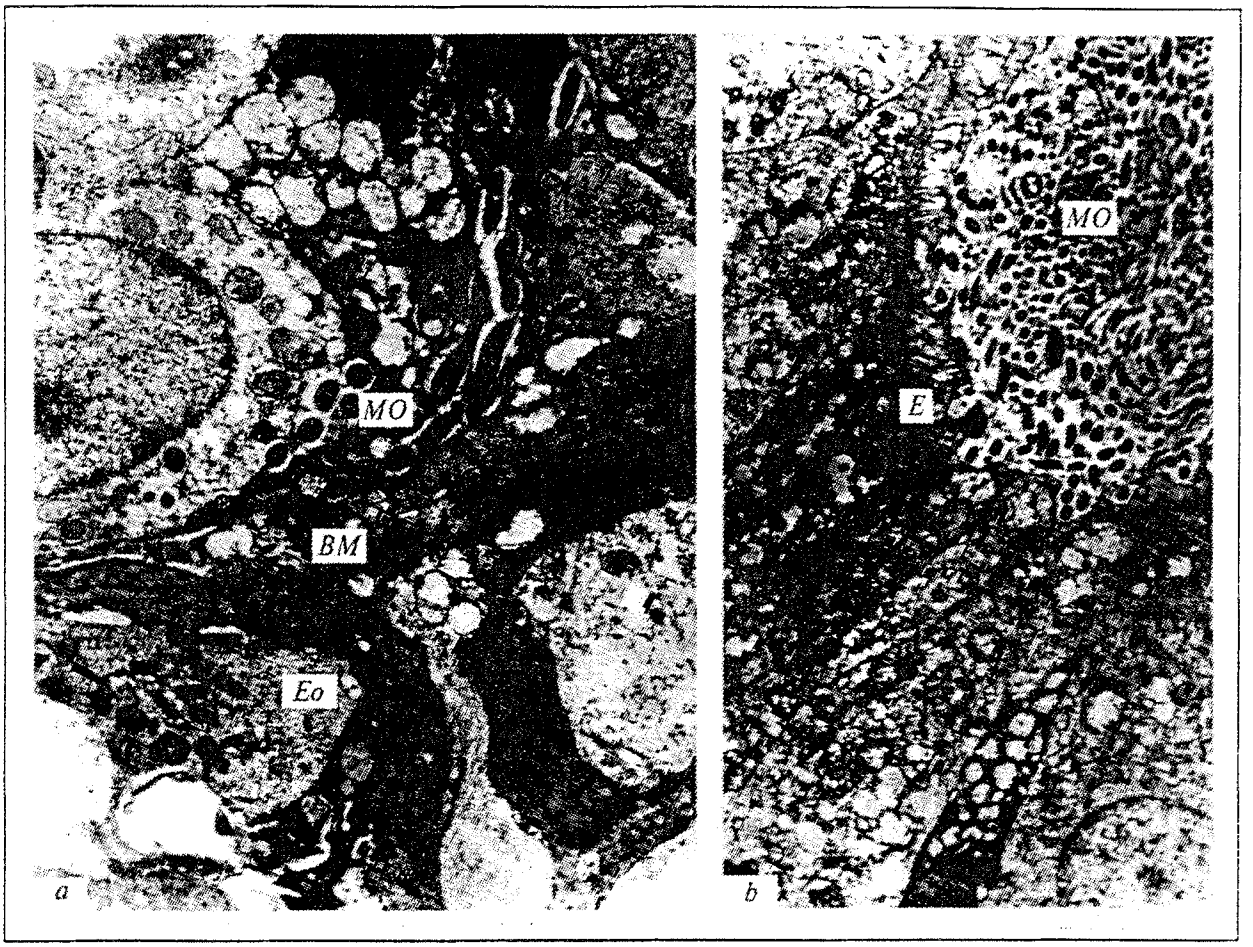

Fig. 2. Interaction of microorganisms with cecal enterocytes 24 h after oral administration of the pesticide Sumi-alpha. *a*) numerous microorganisms (*MO*) in the basal part of an enterocyte. *BM* — basal membrane; *Eo* — eosinophil of lamina propria, ×12,500; *b*) numerous microorganisms in the cryptal lumen without invading enterocytes (*E*), ×5500.

much higher (2- to 4-fold) numbers in all studied intestinal areas, particularly in the cecum (21.7± 0.2%) (Fig. 1, *a*).

The occurrence of PM was the highest 5 h after pesticide administration, particularly in rats given Sumi-alpha. In the ileum, they often invaded the epithelium. The microorganisms were usually present in secreting goblet cells.

PM often penetrated into cecal epithelial cells, forming large clusters, sometimes filling the entire cytoplasm depleted of secretion (Fig. 1, *a*). After crossing the lateral plasma membrane, they entered neighboring cells (sometimes prismatic cells, Fig. 1, *b*), where they were located in the cytoplasm or were surrounded by the plasma membrane (Fig. 1, *c*, *d*). Parietal microorganisms located in these peculiar vacuoles often had outgrowths in the form of fibrils on their surfaces (microbial flagella). Although PM rarely penetrated prismatic cells, the apical surfaces of these cells were changed considerably: their microvilli were thinned, deformed, and arranged in an irregular manner or sometimes were absent (Fig. 1, *d*).

Later, the enterocytes of Sumi-alpha-treated rats also contained large numbers of PM, which were seen in the basal part of these cells whose basement membranes were thickened and homogeneous (Fig. 2, *a*).

In prismatic cells, no marked structural abnormalities were noted other than the above-mentioned changes in the apical surface and some swelling and clarification of the mitochondrial matrix.

The invasion of enterocytes by PM in rats administered the pesticide Omait was less massive, although these cells contained PM in much higher numbers. Considerable numbers of PM were observed in the cryptae 24 h after administration of Omait. Changes observed in the microvilli and apical parts of enterocytes resembled those characteristic of clasmatosis (Fig. 2, *b*). Later on, PM were present in substantially lower numbers and were no longer seen in enterocytes.

Thus, the pesticides Omait and Sumi-alpha significantly increase the number of PM, particularly in epithelial cells. The microbes enter via secreting goblet cells.

Penetration of PM into enterocytes may be associated with various gastrointestinal disorders arising in pesticide poisoning.

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