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Parathyroid Hormone and Gastric Mucosal Surface Ultrastructure

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ABSTRACT

Examination in the SEM of gastric mucosa from rats given parathyroid hormone appeared to reveal an increased incidence of unspecific damage on the luminal surfaces of the gastric mucosal surface epithelial cells. The finding may be related to the suggested ulcerogenic effect of the hormone.

INTRODUCTION

A relationship between hyperfunction of the parathyroid glands and gastrointestinal disturbances and gastric ulcers is often suggested both in man and in experimental animals (8, 9, 10). There appears to be no difference between man and the rat in this respect, though the basal gastric secretion is increased in at least some of the patients with hyperparathyroidism (3, 4, 15, cf. 2), whereas it is decreased by treatment with parathyroid hormone (PTH) in the rat (1, 13).

In a previous investigation (13) an increased serum calcium level, a reduced secretion rate and reduced acidity in the basal gastric secretion were noted in rats treated with PTH for 14 days. This result might have been due to an inhibitory effect of this hormone on the gastric secretion, but an increased permeability of the gastric mucosa causing an increased back-diffusion of hydrogen ions would have given the same result (5, 11, 12, 14).

To examine the effects of PTH further gastric mucosa from rats treated with the hormone and from untreated controls were examined in the scanning electron microscope (SEM). A preliminary report of the findings is given in this paper.

MATERIAL AND METHODS

The experiments were performed on male albino rats weighing about 200 g. Five rats were given parathyroid hormone (Para-Thor-Mone, Ely, Lilly) s.c. twice a day for 14 days in a dose of 40 USP units/100 g body weight/day. Three control animals were given physiological saline s.c. twice a day.

Under general anaesthesia 2.5% glutaraldehyde in phosphate buffer was instilled intragastrically via a rubber tube. Five minutes later the stomachs were removed and specimens from the glandular portions were taken in duplicate. After the initial glutaraldehyde fixation postfixation was performed in 1% osmium tetroxide. The specimens were dehydrated in a graded acetone series. After drying they were mounted on brass stubs with an adhesive and electrical continuity was ensured by painting with colloidal silver. They were covered by cold-palladium in a vacuum-evaporator (Jeol, JEE 48) and examined in a Jeol SEM (JSM-U3). For details regarding the preparation procedure see (5) or (7).

RESULTS

The appearance of gastric mucosa from a rat given PTH is shown in Fig. 1. The surfaces of quite a few cells were irregular and appeared unspecifically damaged. No changes were observed along the intercellular borders. Fig. 2 shows a similar area from an untreated control animal. In Figs. 3 and 4 damaged and normal surface epithelial cells are seen at a higher magnification. In both these micrographs some microvilli are observed. Most of these structures appeared to be covered by mucus, however. It must be pointed out that signs of unspecific damage similar to those shown in Figs. 1 and 3 were occasionally seen on the

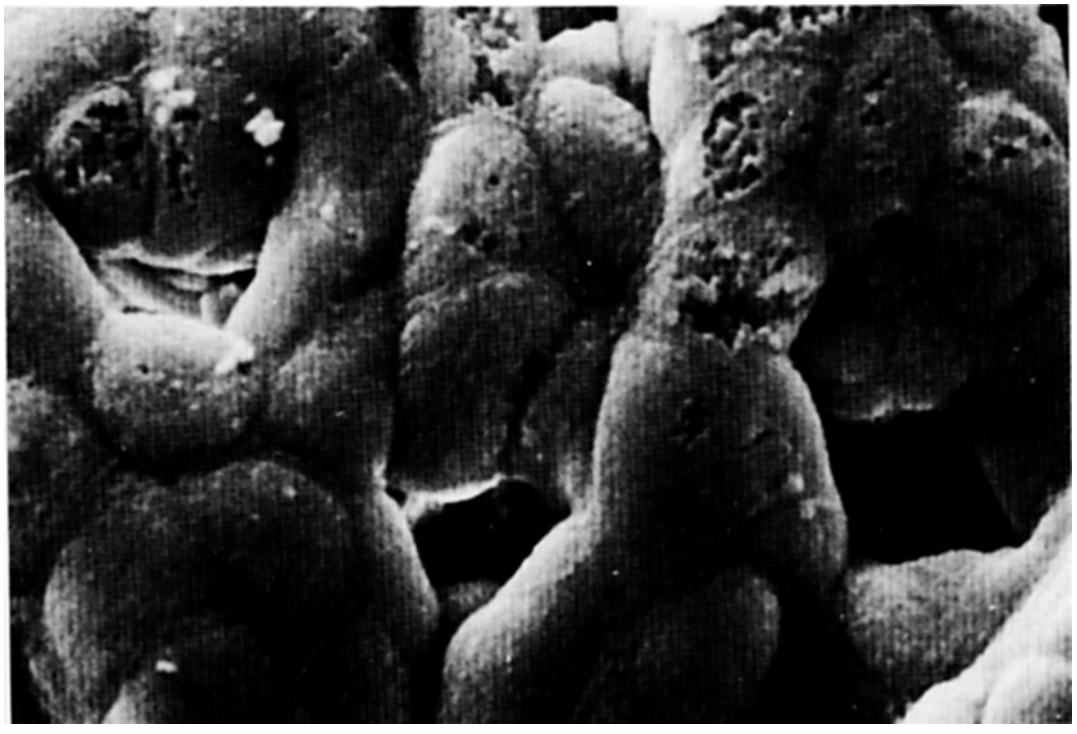

Fig. 1. Micrograph of gastric mucosa from a rat treated with parathyroid hormone for 14 days. The openings of three crypts are seen. Some cells appear unspecifically damaged. (×3000.)

control specimens. The changes observed on specimens from PTH treated rats seemed to be more pronounced, however, and the damaged cells more numerous.

DISCUSSION

The type of damage noted on gastric mucosa from PTH treated rats was not observed on cat gastric mucosa exposed to aspirin, acetic acid or hyperosmotic sodium chloride solutions (5, 6, 7). Subsequent to exposure of gastric mucosa to these weak acids (7) or to large osmolality variations (6) the appearance of the gastric mucosal surface epithelium was also changed from normal. In these cases the changes were of different natures, however.

As to the origin of the observed changes it is only possible to speculate. One suggestion is that PTH treatment may make the cell surfaces more susceptible for peptic digestion. The changes appear to be sufficient to explain the decreased gastric secretion observed after PTH treatment of rats as an effect of an increased back-diffusion of hydrogen ions (5, 11, 12, 14). They may be related to the suggested ulcerogenic effect of the hormone.

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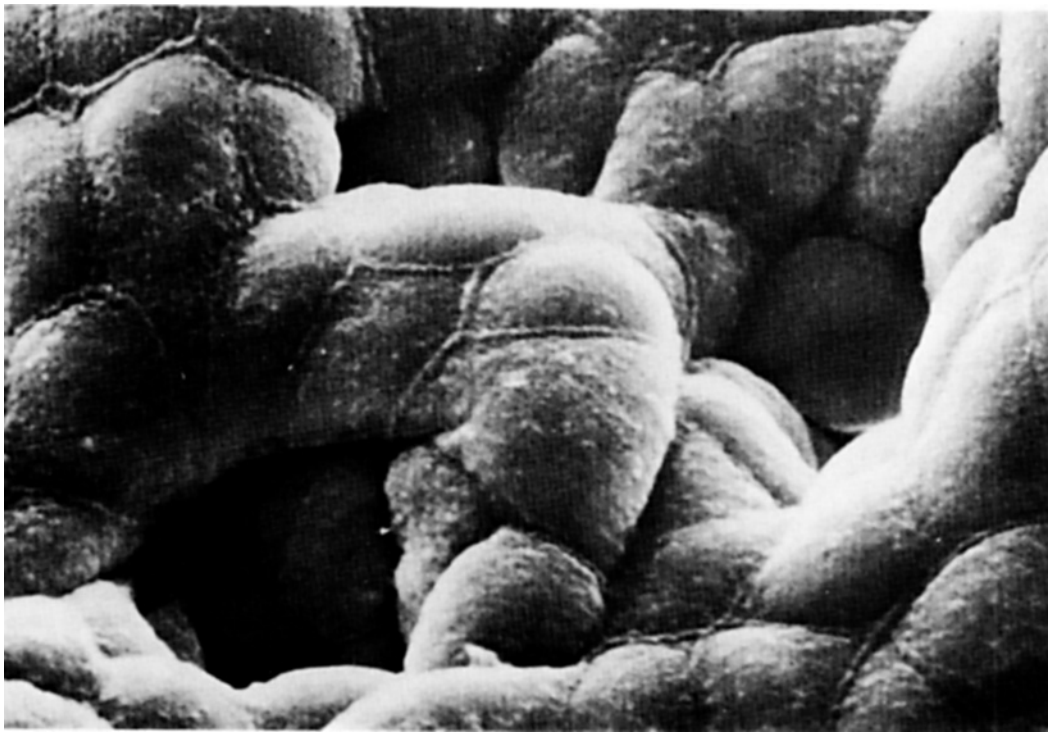

Fig. 2. Micrograph of gastric mucosa from a control animal. Here also the openings of three crypts are observed. (×3000.)

Fig. 3. Damaged surface epithelial cells on gastric mucosa from a PTH treated rat. Along the intercellular borders some microvilli are seen. (×6000.)

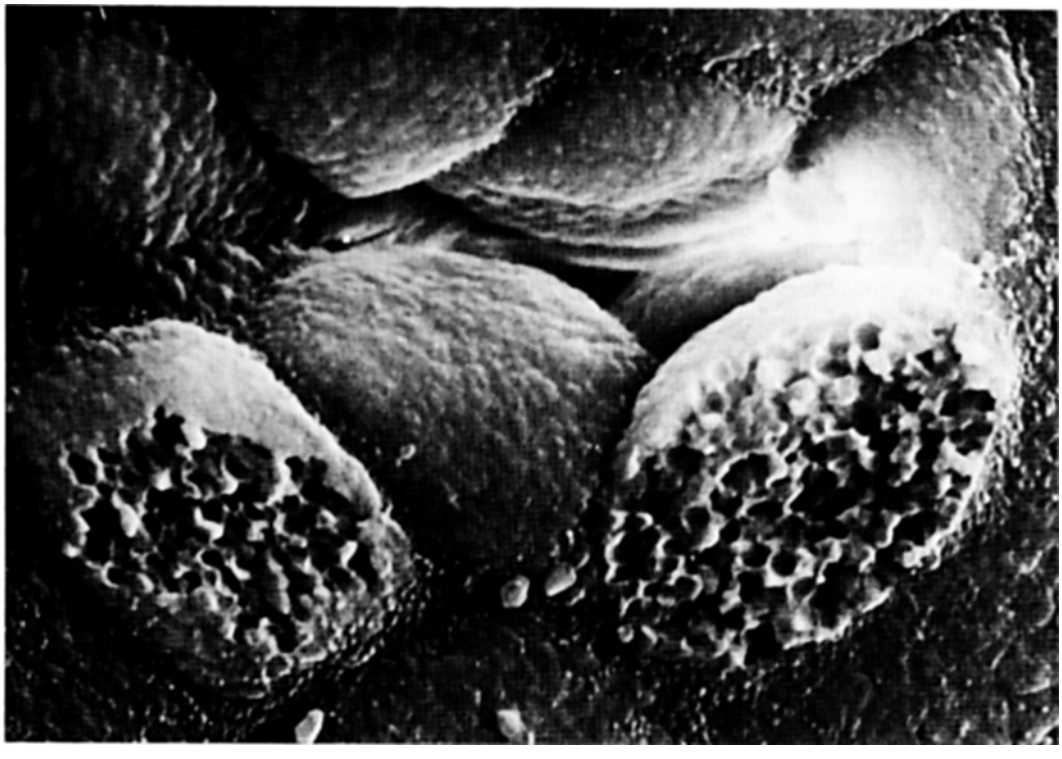

Fig. 4. Normal surface epithelial cells. Some microvilli can be observed in this micrograph also. Most of these structures appear to be covered by mucus, however. (×6000.)

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