tion was isolated from hog gastric mucosa which, on incubation with eggwhite or *ficus* lysozyme,[2] was hydrolyzed, yielding reducing sugars. The 2 known (neutral and acid) polysaccharides of gastric mucin[3] are not attacked by lysozyme.

Dodecyl sulfate, which has been reported to influence mucin production,[4] strongly inhibits eggwhite and gastric lysozyme. The latter, in extracts of human mucosa, is completely inactivated by M/1000 dodecyl sulfate.

The facts reported in this paper strongly suggest a role for lysozyme in the etiology of peptic ulcer.

[2] Meyer, K., Hahnel, E., and Steinberg, A., *J. B. C.*, 1946, **163**, 733.

[3] Meyer, K., Smyth, E. M., and Palmer, J. W., *J. B. C.*, 1937, **119**, 73.

[4] Shay, H., Komarov, S. A., and Siplet, H., *Science*, 1947, **105**, 128.

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Lysozyme in Chronic Ulcerative Colitis.*

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It has been previously shown in experimental animals that there is a low lysozyme content of the colonic mucosa in contrast to that of the stomach and in assays of apparently normal segments of 3 human large intestines, surgically removed for carcinoma, the lysozyme concentration was similarly low (mean = 3.5 units/g tissue). However, following the observation that lysozyme was able to remove the surface mucus from the dog stomach,[1] the present investigation was undertaken to determine whether abnormal concentrations of this mucolytic enzyme were present in the feces of patients with chronic nonspecific ulcerative colitis.

Table I summarizes the results of the stool lysozyme determinations. It is to be noted that the concentration of the enzyme in the feces of the control individuals was low whether the determinations were made on specimens obtained following a normal bowel movement or after purging with magnesium sulfate or castor oil. Similarly, in 3 chronic ulcerative colitis patients whose disease necessitated ileostomy and colectomy, the lysozyme concentration of the ileal stools was uniformly low. Also, in the single patient with idiopathic diarrhea who failed to show any organic change in the mucosa of the gastro-intestinal tract, there was little lysozyme present in the stool. In marked contrast to the above noted findings was the elevated lysozyme concentrations in the fecal excretions of 12 patients in whom the diagnosis of nonspecific chronic ulcerative colitis was established by roentgen and proctoscopic examination and in whom exhaustive search for pathogenic bacteria and/or parasites was negative. Even more striking was the high titer of lysozyme found in specimens of mucus obtained from the rectosigmoid region of patients with this disease.

In some instances it was possible to collect 24-hour stool specimens so that the total lysozyme excreted per day could be determined. The highest total lysozyme content noted in the feces of the control group was 528 units in 24 hours, whereas lysozyme excretion in the colitis patients was as high

* This work was supported in part by a grant from the Josiah Macy, Jr., Foundation, New York.

[1] Meyer, K., Prudden, J. F., Lehman, W. L., and Steinberg, A., PROC. SOC. EXP. BIOL. AND MED., 1947, **65**, 220.

TABLE I.
Lysozyme Assays of Normal and Pathological Stool Specimens.

Source	Individual lysozyme titers (units per g wet wt)			Mean lysozyme titer
Normal stools	0.5	9.4	4.4	2.7
	0.2	0.8	0.9	
Normal stools (after purging)	0.4	3.9	1.0	1.6
	0.8	1.3	0.1	
	1.6	3.8		
Chronic ulcerative colitis stools	28.3	48.7	22.3	56.0
	49.0	10.5	180.9	
	33.3	47.0	103.7	
	24.2	119.4	4.1	
"Mucus" from chronic ulcerative colitis patients	43.5	80.0	15.7	158.1
	167.0	466.0	176.5	
Ileal stools	0.1	2.0	3.6	2.8
Idiopathic diarrheal stool	5.0			

as 44,400 units in a comparable time period.

The experiments, although limited in number, indicate that lysozyme production is greatly increased in the diseased colon. The low titer of the ileal stools in the disease makes it unlikely that the enzyme is produced outside the colon. The high titer of the "mucus" points to the colonic mucosa as the source of the enzyme, although some microorganisms have been shown to produce small quantities of lysozyme.[2] We propose as a hypothesis that the pathogenesis of chronic ulcerative colitis may be due to a combination of local overproduction of lysozyme followed by a necrotizing action on the mucosa by the indigenous bacterial flora. We assume from analogy with the action of lysozyme on the gastric mucosa that the enzyme prepares the way for the necrotizing effect by a removal of the protective surface mucus. This hypothesis depends on the demonstration of a substrate of lysozyme in the colon.

[2] Meyer, K., Palmer, J. W., Thompson, R., and Khorazo, D., *J. B. C.*, 1936, **113**, 479.

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Effect of Caronamide upon Penicillin Therapy of Experimental Pneumococcus and Typhoid Infections in Mice.

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It is now agreed that one of the major disadvantages of penicillin as a therapeutic agent is the rapidity with which the human and animal kidney excretes the drug from the plasma into the urine. Because of this rapid excretion, which occurs both by way of the renal tubules and the glomeruli,[1] relatively large doses must be given every few hours if a detectable plasma concentration is to be maintained. To obtain higher concentrations of penicillin in the plasma, very

[1] Beyer, K. H., Peters, L., Woodward, R., and Verwey, W. F., *J. Pharm. and Exp. Therap.*, 1944, **82**, 310.