the presence of allergic (eosinophilic) mucin in 96% of consecutive chronic sinusitis patients. Thus, the eosinophils visualized in the tissue in the past were cells only in transit into the lumen.

We hypothesized in the article that the targets in the mucin are the extramucosal fungi. Recently, we were able to further validate this hypothesis and demonstrated that the eosinophils are indeed targeted against fungal organisms in CRS, a phenomenon that was absent in healthy controls as well as in patients with allergic rhinitis.[2]

We have visualized that the eosinophils were attacking and destroying noninvasive fungi in the mucin of CRS patients. The granule proteins released during that attack are also known to be highly toxic to the epithelium and have been shown to be essential in the damage of the epithelium.[3] Thus, the mere presence of fungi is nonspecific in this disease, but fungi are the target for the eosinophils in the CRS patient population and are essential as a trigger to stimulate the immunologic (eosinophilic) response to them in a sensitized individual. For these reasons we advocate the new terminology *eosinophilic fungal rhinosinusitis*. These findings and the conclusion we have drawn from them have been confirmed by a second research group.[4]

Criticisms like those published in the *Washington Post* are usually leveled by colleagues who are not informed about the work being done or have not paid close enough attention to the details provided. They also did not support their comments with data.

The European Rhinologic Society, the European Federation of Oto-Rhino-Laryngological Societies, and the International Rhinologic Society have planned plenary sessions on the subject at upcoming meetings. The International Federation of Oto-Rhino-Laryngological Societies has invited us to participate as faculty members at the Consensus Conference on Nasal Polyposis (scheduled for October 2000 in Siena, Italy) to help set standards and move research in new directions.

Our findings have led us to the development of new treatment options. Blinded, randomized, placebo-controlled drug trials have not been completed or published.

Jens U. Ponikau, MD
David A. Sherris, MD
Eugene B. Kern, MD
Mayo Clinic Rochester
Rochester, Minn

1. Ponikau JU, Sherris DA, Kern EB, et al. The diagnosis and incidence of allergic fungal sinusitis. *Mayo Clin Proc*. 1999;74:877-884.
2. Ponikau JU, Sherris DA, Kern EB. Chronic rhinosinusitis: an immune response to fungi. *Allergologie*. 1998;21:581.
3. Harlin SL, Ansel DG, Lane SR, Myers J, Kephart GM, Gleich GJ. A clinical and pathologic study of chronic sinusitis: the role of eosinophils. *J Allergy Clin Immunol*. 1988;81:867-875.
4. Braun H, Hofmann TH, Freudenschuss K, Buzina W, Ponikau J, Stammberger H. Eosinophilic fungal rhinosinusitis (EFRS): from fungus to chronic sinusitis. Paper presented at: Annual Meeting of the Austrian Medical Association; November 11, 1999; Graz, Austria.

Lactulose vs Sorbitol for Treatment of Obstipation in Hospice Programs

To the Editor: I appreciated Dr Kaur's Concise Review for Clinicians[1] on palliative care in hospice programs. In reference to Table 1 of this review, I would like to mention an article by Lederle et al[2] demonstrating that sorbitol is therapeutically equivalent to lactulose. Because sorbitol costs much less than lactulose, it should be the preferred agent.

Harrison G. Weed, MD
Ohio State University College of Medicine
Columbus

1. Kaur JS. Palliative care and hospice programs. *Mayo Clin Proc*. 2000; 75:181-184.
2. Lederle FA, Busch DL, Mattox KM, West MJ, Aske DM. Cost-effective treatment of constipation in the elderly: a randomized double-blind comparison of sorbitol and lactulose. *Am J Med*. 1990;89:597-601.

In reply: Dr Weed raises a good point. He is correct that both sorbitol and lactulose are osmotic laxatives and are therapeutically equivalent if constipation is the only indication for their use. The study he cites by Lederle et al[1] was done in an elderly ambulatory male population. Patients with metastatic cancer and those receiving narcotics were excluded from eligibility.

In our hospice program, about 80% of patients have metastatic cancer as a terminal diagnosis, and a large subgroup of these patients have liver metastases as a complicating factor. In this patient population, we prefer to use lactulose, if other less aggressive bowel programs have failed, because of its theoretical advantage in also treating hepatic encephalopathy. Lactulose, like sorbitol, is poorly absorbed from the gastrointestinal tract and reaches the colon virtually unchanged. However, lactulose, unlike sorbitol, is broken down primarily to lactic acid in the colon, exerting an acidic effect on colonic contents.[2] In this acidic environment, the reabsorption of ammonia and other substances thought to cause hepatic encephalopathy is impaired. Additionally, the acidic colonic environment inhibits the growth of colonic bacteria responsible for producing ammonia and other molecules linked to hepatic encephalopathy. The results are less ammonia and related compounds available to be reabsorbed from the colon and, therefore, presumably diminished hepatic encephalopathy. The acidic colonic environment created by lactulose is advantageous in treating the hepatic encephalopathy often associated with advanced malignant disease.[3]

In our institution, 16 oz of lactulose is $9.34 vs $2.21 for 16 oz of sorbitol. Many patients with hepatic metastases have unrecognized hepatic encephalopathy[4]; therefore, we prefer to use lactulose despite the cost difference. For patients without complications, sorbitol should certainly be considered the cost-effective option.

Judith S. Kaur, MD
Mayo Clinic Rochester
Rochester, Minn

1. Lederle FA, Busch DL, Mattox KM, West MJ, Aske DM. Cost-effective treatment of constipation in the elderly: a randomized double-blind comparison of sorbitol and lactulose. *Am J Med*. 1990; 89:597-601.
2. Kristalose [lactulose] for oral solution. In: *Physicians' Desk Reference*. 54th ed. Montvale, NJ: Medical Economics Co Inc; 2000:773.
3. Smith BC, James OF. The failing malignant liver. *Gut*. 1998;42:454-455.
4. Eras P, Sherlock P. Hepatic coma secondary to metastatic liver disease. *Ann Intern Med*. 1971;74:581-583.