

ferent input from the small intestine by way of neurons with cell bodies that form the inferior vagal (nodose) ganglion. Neurons constituting the nucleus of the solitary tract are situated entirely within the brain.^{2,3} The figure in Korner and Leibel's article raises the question of exactly where peptide YY₃₋₃₆ acts on neurons associated with the hindbrain to exert its inhibitory effect.

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THE AUTHORS REPLY: We appreciate the feedback from Dr. DeSantis about our figure, which depicts interactions among hormonal and neural pathways that regulate energy homeostasis. In this schematic diagram, we indicated that the nucleus of the soli-

tary tract provides efferent innervation to the small intestine. Actually, the nucleus of the solitary tract is a mix of sensory neurons located within the dorsal vagal complex of the medulla. The dorsal vagal complex is a primary site of action of several neuropeptides mediating gastrointestinal activities; in addition, it contains efferent neurons that constitute the dorsal motor nucleus of the vagus. The effect of peptide YY₃₋₃₆ on gastric motility is mediated, at least in part, by direct inhibition of vagal efferent neurons in the dorsal motor nucleus through actions at Y₂-type peptide YY receptors.¹ Thus, in addition to its effects within the arcuate nucleus of the hypothalamus, peptide YY₃₋₃₆ acts as an "ileal brake" that suppresses gastric motility, thereby possibly contributing to a sense of satiety.

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False Positive Test for Aspergillus Antigenemia Related to Concomitant Administration of Piperacillin and Tazobactam

TO THE EDITOR: The detection of aspergillus galactomannan antigenemia is a method used in the diagnosis of invasive aspergillosis.^{1,2} Some transient false positive reactions have been reported with the use of enzyme-linked immunosorbent assays in relation to the passage of galactomannan of food origin through the intestinal mucosa or in relation to possible interactions with antibiotics, including piperacillin.³ We report a recent marked increase in the frequency of false positive tests and its relation to the concomitant administration of piperacillin and tazobactam.

Between May 1 and July 15, 2003, 931 serum samples were examined for the presence of galactomannan with the use of the Platelia aspergillus kit (Bio-Rad). The charts of all the patients with positive results for galactomannan antigenemia (optical-density index, >1.5) and of 37 patients with persistently negative results were reviewed for the diagnosis of invasive aspergillosis, according to the criteria of the European Organization for Research and Treatment of Cancer–Mycoses Study Group.⁴ Concomitant administration of drugs, including antibi-

otics, was recorded as a possible cause of false positive reactions.³ During the study period, 103 of the serum samples, obtained from 37 patients, were positive. The diagnosis of invasive aspergillosis was probable in three of these patients and possible in one. Among the remaining 33 patients and among the 37 patients with persistently negative results on tests for antigenemia, there were no clinical or radiologic signs of invasive aspergillosis. In most cases, galactomannan antigen levels abruptly increased to high positive values in consecutive samples — a finding confirmed with different batches of Platelia kits.

A chart review showed that 25 of the 37 patients with positive tests for galactomannan antigenemia were receiving a combination of piperacillin and tazobactam, as compared with 2 of 37 patients with persistently negative tests (chi-square=28.22; P<0.001). In the patients treated with this drug combination, galactomannan antigen levels increased as early as one day after the initiation of treatment and became undetectable one to six days after cessation.

Four batches of the piperacillin–tazobactam

used during this period were found to be positive for galactomannan (optical-density index, >1.5) at concentrations between 6 and 200 mg per milliliter (i.e., usual plasma concentrations after intravenous administration). Piperacillin alone yielded negative results; data for tazobactam alone were unavailable.

These results are highly suggestive of strong cross-reactivity of the *Platelia aspergillus* test with the piperacillin–tazobactam combination. This phenomenon seems to be recent and may be restricted to a limited number of batches of the drug combination. While further investigations are in progress to determine the origin of these false positive reactions and to explore cross-reactivity with other beta-lactam antibiotics, we urge clinicians and biologists to be aware of the possible occurrence of false positive results, which might lead to inappropriate invasive investigations or treatments.

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Keratoderma Blennorrhagicum

TO THE EDITOR: We report the case of a 34-year-old man who presented with a two-month history of painful keratotic plaques that involved the skin surrounding the ears, the nose, and the soles in association with polyarthritis. He had had a diarrheal illness several months before the onset of symptoms. He reported that he had not had symptoms of urethritis or conjunctivitis. His mother had psoriasis. Physical examination revealed cachexia and oral candidiasis, but no organomegaly or lymphadenopathy. Pustular and scaling keratotic papules and plaques were distributed over the extensor surfaces of the arms and legs as well as the palms and soles. Onychodystrophy affected both the fingernails and the toenails. The beard area and the nasolabial folds showed erythema and scaling, findings consistent with the presence of seborrheic dermatitis. Figure 1 shows the psoriasiform, hyperkeratotic plaques typical of keratoderma blennorrhagicum (i.e., the acral pustular and hyperkeratotic lesions seen in Reiter's syndrome and pustular psoriasis¹).

The results of a urinalysis were normal. A complete blood count revealed mild leukopenia (white-cell count, 3500 per cubic millimeter [reference range, 3600 to 9000]), with 56 percent neutrophils and 14 percent lymphocytes). Flow-cytometric analysis of venous blood showed absolute lymphopenia (lymphocyte count, 490 per cubic millimeter [reference range, 1100 to 3400]), with a marked

reduction in CD3+CD4+ lymphocytes (5 per cubic millimeter [reference range, 440 to 1600]). An enzyme-linked immunosorbent assay and a Western blot study were positive for the human immunodeficiency virus (HIV).

The patient was treated with clobetasol propionate ointment, applied to the skin lesions twice daily, and 25 mg of acitretin daily. Antiretroviral therapy was begun. Follow-up examination four weeks later showed improvement of the hyperkeratotic plaques. The patient did not return for follow-up for



Figure 1. Psoriasiform, Hyperkeratotic Plaques on the Soles of the Feet of a 34-Year-Old Man.