fferent input from the small intestine by way of neu-
rons with cell bodies that form the inferior vagal (nodose) ganglion. Neurons constituting the nucle-
us of the solitary tract are situated entirely within the
brain. The figure in Korner and Leibel’s article
raises the question of exactly where peptide YY
3–36 acts on neurons associated with the hindbrain to ex-
ert 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 dor-
sal vagal complex of the medulla. The dorsal vagal
complex is a primary site of action of several neuro-
peptides mediating gastrointestinal activities; in ad-
dition, it contains efferent neurons that constitute
the dorsal motor nucleus of the vagus. The effect of
peptide YY
3–36 on gastric motility is mediated, at
least in part, by direct inhibition of vagal efferent
neurons in the dorsal motor nucleus through ac-
tions at Y
2-type peptide YY receptors.

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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 galac-
tomannan antigenemia is a method used in the di-
agnosis of invasive aspergillosis. Some transient
false positive reactions have been reported with the
use of enzyme-linked immunosorbent assays in re-
lation to the passage of galactomannan of food
origin through the intestinal mucosa or in relation
to possible interactions with antibiotics, including
piperacillin. 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 galacto-
mannan 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 persis-
tently negative results were reviewed for the diagno-
sis of invasive aspergillosis, according to the criteria
of the European Organization for Research and
Treatment of Cancer–Mycoses Study Group. Con-
comitant administration of drugs, including antibi-
otics, was recorded as a possible cause of false pos-
itive reactions. 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 ra-
diologic signs of invasive aspergillosis. In most cas-
es, galactomannan antigen levels abruptly increased
to high positive values in consecutive samples — a
finding confirmed with different batches of Plate-
lia 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 com-
bination, 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

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N ENGL J MED 349;24 WWW.NEJM.ORG DECEMBER 11, 2003

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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 psoriasis).

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.