LETTERS

Tryptophan-Poor Diets

The introductory paragraphs of the report by Lytle *et al.* (14 Nov., p. 692) tentatively relate their findings of "reduced levels of brain serotonin" and "increased responsiveness to electric shock" in "[r]ats fed tryptophan-poor corn diets" to the behavioral sequels of "protein-calorie malnutrition experienced early in life," or, more specifically, to those of "extreme kwashiorkor or marasmus." Indeed, they may be so related.

What seems remarkable to this reader is the absence of any mention of the "dementia" that, every spring for many years, filled virtually every available bed in the mental institutions of the American South with adults afflicted with pellagra. This disease was most surely the consequence of ingestion of a "tryptophan-poor corn diet." All symptoms, including the psychosis, were rapidly alleviated by the administration of nicotinic acid. The entire disease syndrome is prevented by dietary measures that increase the intake of all amino acids including tryptophan, as well as that of nicotinic acid. Tryptophan is the biological percursor of both nicotinic acid and serotonin.

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We welcome Handler's reminder regarding the "dementia" and pellagra observed in humans consuming diets that are poor in tryptophan, and that lack supplementary nicotinic acid, for long periods of time. However, we respectfully disagree with his suggestion that the increased electroshock sensitivity that we observed in rats consuming tryptophan-deficient, corn-based diets was caused by nicotinic acid deficiency.

1) Both of our test diets (that is, the casein control and corn diets) were supplemented with 45 milligrams of niacin per kilogram (dry weight), a concentration well within the range present in most standard lab chows. Hence, it seems unlikely that our corn-fed rats were deficient in this vitamin. Of course, only its direct measurement in plasma of corn-fed and casein-fed animals will suffice to establish the absence of a deficiency state.

2) We have shown that the diet-induced changes in both brain serotonin and electroshock sensitivity are reversed within 1 hour after the injection of tryptophan. We know of no evidence that the dementia of pellagra is reversed so rapidly after a single dose of tryptophan.

3) The injection of fluoxetine hydrochloride (Lilly 110140), a highly specific inhibitor of serotonin reuptake into neurons, reverses the changes in electroshock sensitivity among corn-fed rats (*1*). There seems little basis for believing that this drug also restores normal concentrations of nicotinic acid.

4) *p*-Chlorophenylalanine, a drug that decreases brain serotonin by inhibiting the enzyme tryptophan hydroxylase, exacerbates the diet-induced increases in electroshock sensitivity. We also know of no evidence that this drug decreases nicotinic acid.

5) Injections of the amino acids leucine, valine, or phenylalanine (which acutely lower brain tryptophan and suppress serotonin synthesis) in normal animals produces hyperalgesia within 1 hour after injection (*2*). It appears unlikely that these compounds would also produce pellagra in rats within 1 hour after injection.

6) Brain lesions that destroy serotonin axons and terminals in the telencephalon also produce hyperalgesia in rats (*3*). Here, too, we know of no relation between these manipulations and nicotinic acid.

In summary, although the consumption of tryptophan-deficient diets causes numerous biochemical changes besides decreasing brain serotonin, we believe that the most parsimonious explanation for our findings on pain sensitivity is that diet-induced reductions in brain serotonin underlie this effect.

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Good Scents?

The discovery and research of truly effective malodor counteractants (Research News, 28 Nov., p. 870) is undoubtedly welcomed by investigators concerned with olfactory systems, and probably by enterprising business, as market testing is reportedly in progress. The "other side of the coin" is not, however, mentioned in the article.

Would indiscriminant use of these chemicals as "air fresheners" deprive one of sampling some of the simpler pleasures of life (the distinctive aroma of a favorite cheese, the bouquet of a fine beverage)? Could not untimely or abusive use of malodor counteractants interfere with the detection of undesirable substances, such as the aromatics evolved from spoiling food or the mercaptans in escaping natural gas?

For a naive or unsuspecting consumer, would "good scents" mean good sense? It is to be hoped that public merchandising of these chemicals will not open Pandora's box.

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Biohazard: Virus-Contaminated Liquid Nitrogen

We wish to alert microbiologists to a potential source of laboratory infection which may be frequently overlooked. A variety of pathogenic and nonpathogenic microbes are commonly stored in glass ampules in liquid nitrogen storage tanks. This form of storage may present risks.

As a part of our routine laboratory duties, we store vesicular stomatitis virus (VSV), as well as other viruses, in flame-sealed glass ampules in a 240-liter liquid nitrogen storage tank. Recently, while removing a vial containing VSV from the storage tank, we noticed that an ampule had shattered within a storage cane. Several more broken ampules were found during a more thorough search. A review of the literature as well as several phone calls revealed that apparently no information was available on the survival of viruses under such conditions, but it was known that erythrocytes remained viable after direct contact with liquid nitrogen (*1*). We thus sought to determine if the contents of the broken vials had found their way into the surrounding liquid nitrogen and, if so, whether the contaminating viruses had survived.

Six 250-milliliter samples of liquid nitrogen obtained in sterile containers were taken from the storage tank in which VSV ampules had broken, placed in a hood, and evaporated to dryness. Small volumes of saline were used to rinse the interior surfaces of the sample vessels, and these rinses were examined for VSV. As many as 160 infectious virus particles were detected in one sample. By adding virus directly to liquid nitrogen, we demonstrated that there is no significant loss of virus infectivity.

The recovery of an infectious virus of minor clinical importance portends the potential biohazard of storing more virulent viruses under similar conditions. One