REVERSAL BY TRYPTOPHANE OF THE BIOLOGICAL EFFECTS
OF 3-ACETYLpyRIDINE

Sirs:

Woolley¹ has shown that the feeding of 3-acetylpyridine (a structural
analogue of nicotinic acid) to mice caused the production of a fatal disease
with many manifestations similar to those of human pellagra or of canine
blacktongue. This action of 3-acetylpyridine could be negated completely
by addition of nicotinic acid to the diet. At about the same time, Krehl
et al.² demonstrated that the inclusion of corn in a highly purified basal
ration for rats brought about retardation of growth and that this deleterious
effect of corn could be overcome either with nicotinic acid or with trypto-
phane. These authors postulated a selective action of the corn on the
intestinal flora as an explanation of their findings. However, an alternate
hypothesis is that the “pellagragenic” action of corn may be related to
the occurrence in it of a structural analogue of nicotinic acid which competes
with that vitamin just as 3-acetylpyridine does. Since tryptophane as
well as nicotinic acid counteracted the effects of corn, it was decided to
determine whether tryptophane would also overcome the action of 3-
acetylpyridine. The experiments described below will show that trypto-
phane did nullify the toxic effects of 3-acetylpyridine.

Responses of Mice to Added Tryptophane and 3-Acetylpyridine

<table>
<thead>
<tr>
<th>dl-Tryptophane added (per cent)</th>
<th>3-Acetylpyridine (mg. per day)</th>
<th>No. of animals</th>
<th>No. of deaths</th>
<th>Average change in weight (gm. per wk.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
<td>12</td>
<td>0</td>
<td>+4.0</td>
</tr>
<tr>
<td>0</td>
<td>4</td>
<td>12</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>2.0</td>
<td>4</td>
<td>6</td>
<td>0</td>
<td>+5.2</td>
</tr>
<tr>
<td>1.0</td>
<td>4</td>
<td>11</td>
<td>1</td>
<td>+6.1</td>
</tr>
<tr>
<td>0.3</td>
<td>4</td>
<td>11</td>
<td>1</td>
<td>+6.1</td>
</tr>
<tr>
<td>0.1</td>
<td>4</td>
<td>6</td>
<td>1</td>
<td>+4.6</td>
</tr>
</tbody>
</table>

Weanling mice were caged individually on screen floors and fed a diet
composed of sucrose 75 gm., casein 18 gm., salts³ 5 gm., heated starch
(amilgel)⁴ 20 gm., fortified corn oil⁵ 1 gm., thiamine 0.2 mg., riboflavin 0.5

(1945).
⁴ Supplied by the Corn Products Refining Company.
mg., pyridoxine 0.2 mg., calcium pantothenate 2 mg., choline chloride 10 mg., and inositol 100 mg. To this basal ration various amounts of tryptophane were added. Beginning on the 4th day each animal was given orally 4 mg. of 3-acetylpyridine per day. The experiment was continued for 10 days, and the animals were observed for signs of disease and were weighed twice weekly. In one experiment, to verify the previous finding that this vitamin would protect the animals, a group of six mice was fed the basal ration plus 0.5 per cent of nicotinamide.

The data in the table show that tryptophane nullified the toxicity of 3-acetylpyridine. It also prevented the pellagra-like manifestations called forth by the ketone. As little as 0.1 per cent of the amino acid was sufficient to protect the animals. Therefore, tryptophane was as active in this respect as was nicotinic acid. In view of these results and those of Krehl et al., it might be of interest for a study of the etiology of pellagra to examine corn for the presence of a structural analogue of nicotinic acid.

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New York

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Received for publication, December 17, 1945

With the technical assistance of A. Holloway.
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