

changed (Table 1). The NE decrease affected all parts of the brain assayed, with the hypothalamus being most depleted (Table 1).

Correlative fluorescent histochemical observations were also obtained 7 days after 6-hydroxydopa treatment. There was a moderate to marked reduction of varicosities in the neocortex, septum, preoptic area, and hypothalamus (Fig. 2), but not in the caudatus putamen. The most pronounced alteration was the presence of numerous green fluorescent preterminal axons that were not observed in control brains. These fluorescing preterminal axons (Fig. 2) were observed in noradrenergic areas of the medulla oblongata, reticular formation, cerebellar peduncle, hypothalamus, preoptic region, septum, and cingulum. This is similar to that seen in the mouse brain after an intravenous injection of 6-hydroxydopa (5). Analogous to 6-hydroxydopamine treatment, the appearance of intensely fluorescent axons is highly suggestive of a proximal accumulation of catecholamines within monoaminergic neurons after degeneration of nerve terminals (11).

Thus, selective depletion of brain NE without DA depletion can be produced by 6-hydroxydopa. When it occurs, it is accompanied by an increase in shock-induced fighting. The time course of the onset of increased fighting behavior suggests that NE depletion is not the sole cause of the facilitation of shock-induced fighting. On the second day after an injection of 90 μ g of 6-hydroxydopa, at a time when no increase in shock-induced aggression is evident, brain NE is already as low as on day 7 (12).

In addition, however, 6-hydroxydopa produces degeneration of central NE terminals, as noted by accumulation of intensely fluorescent noradrenergic pre-

terminal axons (5). This is an effect similar to that produced by 6-hydroxydopamine (11). Ungerstedt (13) has shown that intracerebral injections of 6-hydroxydopamine produce supersensitivity of central DA receptors to exogenously administered L-dopa and apomorphine. It is plausible that, as with 6-hydroxydopamine, the NE terminal degeneration produced by 6-hydroxydopa is accompanied by an increase in sensitivity of NE receptors to endogenous amine which in turn induces an increase in shock-induced aggression (14).

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14. 6-Hydroxydopa was obtained from Regis Chemical Company, Chicago, Illinois.
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was made in the *Chemical Economics Handbook* (1, reference 6) or in their use of the published figures is not clear.

With respect to their assumption that the difference between the P/U ratio in fertilizers and rivers suggests that more than 90 percent of the applied phosphate has been taken up by plants, a look at the reaction product between soil and fertilizer would indicate that rarely is more than 20 percent of the applied phosphorus recovered in a given year. The remaining portion becomes an integral part of the soil, some of which will be available to subsequent crops.

The agricultural and environmental implications of Spalding and Sackett's work could be explored further, perhaps by the scientists at some public agency like the Tennessee Valley Authority's National Center for Fertilizer Development and Soils and Fertilizer Research Branch.

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Reference

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Munson points out a mistake in our article that occurred in copying; however, this mistake was not used in the calculation of 285 short tons. The article should read as follows: In a 5-year span from 1962 to 1965 fertilizer consumption increased from 25 million to 36 million short tons per year. In 1967 this amounted to over 2.9 million short tons of P_2O_5 applied to United States regions draining into the Gulf of Mexico. If we assume an average value of uranium in phosphate fertilizer to be 100 μ g/g, this amounts to approximately 290 short tons of uranium.

Since 100 μ g/g is a minimum uranium concentration in phosphate fertilizer, especially when one considers that all tabulations of tons of P_2O_5 applied are in terms of 100 percent P_2O_5 , a considerably higher uranium concentration could have been used in this calculation and still have been realistic. In short, the mistake noted by Munson did not change the uranium estimates or the implications of the report.

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Uranium in Runoff

In the report by Spalding and Sackett (1) we have to assume that the P/U ratios are correct. However, if we compare the phosphate and uranium values from the Brazos River (1, table 1) the resulting correlation coefficient would be very low, an indication that the two are not related. The relations shown in figure 2 (1) raise some questions.

Spalding and Sackett arrive at the erroneous value of 285 short tons of

U_3O_8 as being applied to those U.S. regions draining into the Gulf of Mexico because of the large error they made in the amount of P_2O_5 applied in 1967. They stated that "... this amounted to over 2.9 billion short tons of P_2O_5 ..." The facts are that the total P_2O_5 used in the 48 contiguous states was only 4.7 million tons for fiscal 1971. The amount used in the Gulf region would only be a fraction of that. Whether or not the initial error