light, by formation of a compound with lower solubility in water; this compound is ether-soluble. After evaporating off the ether, there remains a pink-brown residue with a strong smell of fæces.

It is possible that one step of the transformations brought about by light is the formation of skatole, by side-chain degradation of the indoleacetic acid molecule.

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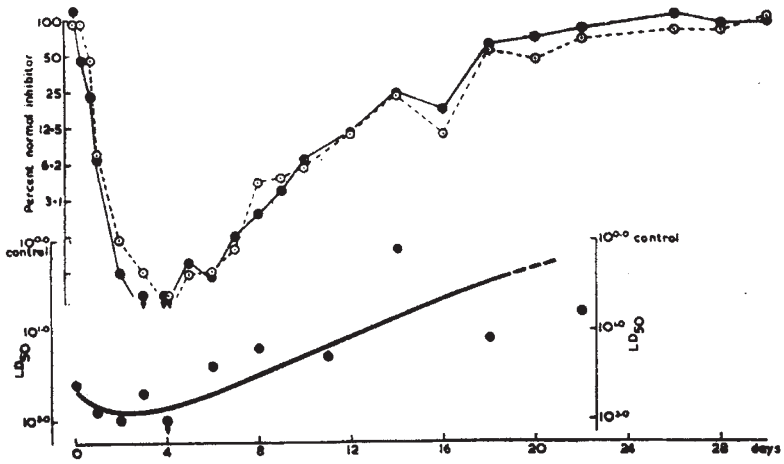
Protection by Receptor-destroying Enzyme against Infection with a Neurotropic Variant of Influenza Virus

IN 1948, Stone reported that infection by influenza viruses of embryonated hens' eggs after allantoic inoculation[1], and of mouse lungs after intranasal instillation[2], could be prevented by pretreatment of the exposed cells with the receptor-destroying enzyme of *Vibrio cholerœ*[3]. It seems worth adding to these observations the finding that death of mice from infection by a neurotropic variant of influenza virus, neuro-*WS*[4], can be similarly prevented by pretreatment of the mice with an intracerebral injection of receptor-destroying enzyme, and that the return of susceptibility to infection, following a single inoculation of receptor-destroying enzyme, runs roughly parallel to the return of inhibitory activity of the brain[5] to influenza virus hæmagglutination.

Groups of 4–5 weeks old mice were inoculated in the right cerebral hemisphere with 12,000 units[6] of purified[7] receptor-destroying enzyme in 0·03 ml., two hours to twenty-two days before being challenged by intracerebral inoculation of neuro-*WS* into the right hemisphere. The times of receptor-destroying enzyme inoculations were so arranged that challenge occurred on the same day for all groups and could, therefore, consist of titrating a single neuro-*WS* allantoic fluid in each group of mice (four mice being used per 0·5 $\log_{10}$ dilution). The mice were observed for twelve days after challenge, at the end of which time all surviving mice appeared perfectly healthy. Protection was estimated by the degree of reduction in the titre[8] (*LD*50) of the neuro-*WS* from the level found in a comparable group of mice which had received no pre-treatment. Afterwards, it was shown that receptor-destroying enzyme which had previously been inactivated by heating at 70° C. for one hour had no protective action.

The above experiment was done in conjunction with a study of the destruction *in vivo*, by a similar dose of receptor-destroying enzyme, of brain inhibitor to heated Lee[9] hæmagglutinin. Hæmagglutinin-inhibitor was measured in saline suspensions of each cerebral hemisphere of four mice, at various intervals after inoculation with receptor-destroying enzyme, and the geometric mean of the titres for each side was compared with the geometric mean titre of thirty-two normal hemispheres. So that dissemination of the receptor-destroying enzyme throughout the brain should not be aided by the process of preparing the brain suspensions, all brains were heated at 62° C. for one hour before they were ground up and suspended in saline. Suitable control experiments had shown that this procedure resulted in adequate inactivation of receptor-destroying enzyme. It follows that the observed degree of inhibitor destruction (more than 99·6 per cent) must represent the proportion of brain inhibitor which is accessible to receptor-destroying enzyme inoculated intracerebrally.



Upper graph. Destruction of *HA*-inhibitor in the right (continuous line) and left (dotted line) cerebral hemispheres, following inoculation of receptor-destroying enzyme into the right hemisphere. *Lower graph.* The number of control-mouse *LD*50 required to kill 50 per cent of the mice, when challenged with neuro-*WS* at various times after receiving receptor-destroying enzyme

The accompanying figure shows that approximately a hundred control-mouse *LD*50 were required to kill 50 per cent of the mice which had received receptor-destroying enzyme less than four days before challenge, and that susceptibility gradually returned after four days. The figure also shows that the same amount of receptor-destroying enzyme produced marked destruction of hæmagglutinin-inhibitor in both hemispheres; this destruction was greatest on the fourth day and was followed by a very gradual regeneration of inhibitor running roughly parallel to the return of susceptibility to infection.

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