LETTER TO THE EDITOR

Histamine Release from Mast Cells by Lecithinases A and C

SIR,—In a letter on page 271 of the current volume of this Journal Dr. J. F. Riley comments upon a theory about the mechanism of the disrupting action of compound 48/80 on rat mast cells proposed by Högberg and Uvnäs[1]. According to this hypothesis compound 48/80 attacks the mast cell and liberates histamine by activating a lytic enzyme localized on the mast cell membrane. Högberg and Uvnäs[1] also describe some observations on the disrupting action of lecithinase A, prepared from various sources, on mast cells and they found a surprizingly good parallelism between the action of various enzyme blocking substances on lecithinase A, and on the disrupting action of compound 48/80. The observations were considered to support the hypothesis that compound 48/80 might act by activating a lytic enzyme. But which type of lytic enzyme was involved was left an open question. Dr. Riley has now gone a little further and has suggested that the lytic enzyme might be of the type lecithinase C. This might well be the case since as also pointed out by Riley lecithinase C disrupts mast cells. In fact, we have also observed that a lecithinase preparation from *Clostridium perfringens* (a strain pathogenic to man) which probably is a lecithinase C is very active in disrupting mast cells. And what is of special interest, the action of this lecithinase shows a pH optimum and a temperature sensitivity similar to those previously observed to be valid for the disrupting action of compound 48/80. Lecithinase A, on the other hand, lacks a distinct pH optimum above 7 and is very resistant to heat. More information about the influence of various factors on the disrupting action of the lecithinases A and C will be published shortly from our laboratories. But, since our knowledge about the chemistry of the mast cell membrane is so scanty, it is in my opinion very difficult to visualise the possible nature of a cell membrane phospholipase.

BÖRJE UVNÄS.

Department of Pharmacology,
Karolinska institutet,
Stockholm 60, Sweden.

March 20, 1958.

REFERENCE

1. Högberg and Uvnäs, *Acta. physiol. scand.*, 1957, **41**, 345.