The urine of male Swiss albino mice injected with [^{14}C]-histamine (7 μg/kg) was analysed for [^{14}C]-methylimidazoleacetic acids by gas chromatography, radioactive peaks being located as described by Robbins & Bakke (1967). Approximately 40% of the radioactivity was found in the peak corresponding to 1-MeIm4-AA, but none corresponding to 1-MeIm5-AA, thus confirming Schayer's results.

The mean 24 hr excretion of histamine and 1-MeIm4-AA in male mice during a control period of subcutaneous saline injections was 3 μg and 16 μg respectively. There was no excretion of 1-MeIm5-AA. During daily treatment with histamine (250 mg/kg subcutaneously) for 4 days the corresponding values were 3,000 μg and 213 μg respectively, but there was still no excretion of 1-MeIm5-AA. These results, therefore, do not confirm Karjala & Turnquest's (1955) findings.

Further evidence that 1-MeIm5-AA is not a histamine catabolite was obtained during some studies on the effect of enzyme inhibitors on histamine metabolism. Under standardized dietary conditions (Granerus, 1968) a healthy adult male volunteer excreted $3{\cdot}52 \pm 0{\cdot}07$ mg 1-MeIm4-AA and $1{\cdot}91 \pm 0{\cdot}12$ mg 1-MeIm5-AA per 24 hr (mean $\pm$ S.E. of mean, $N=5$). Treatment with tranylcypromine (10 mg orally every 8 hr) reduced the 24 hr excretion of 1-MeIm4-AA ($2{\cdot}68 \pm 0{\cdot}13$ mg, $N=3$, $P<0{\cdot}001$) but not that of the 1,5-isomer ($2{\cdot}33 \pm 0{\cdot}24$ mg). These results support the view that methylhistamine is oxidized to 1-MeIm4-AA by monoamine oxidase *in vivo*, because tranylcypromine is believed to be a relatively specific inhibitor of this enzyme, and also clearly indicate that the 1-MeIm4-AA and 1-MeIm5-AA arise through different biochemical pathways. It is therefore improbable that the 1-MeIm5-AA which occurs in human urine is a metabolite of histamine formed by oxidation of 1-methyl-5-(2-aminoethyl) imidazole.

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A model noradrenaline binding site.

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Immunoglobulins specific to noradrenaline have been prepared in rabbits by injection of a noradrenaline-bovine serum albumin complex. The immunoglobulins were extracted from the antisera by adsorption on to an insoluble polymer of the immunizing antigen with subsequent desorption by a related hapten. Complex formation between the immunoglobulin and various haptens structurally related to noradrenaline have been studied by a fluorescence quenching method. Association constants of the order of 10^6 to 10^7 l. mol^{-1} were obtained for the reaction : Hapten + immunoglobulin (IgC) $\rightleftharpoons$ IgC-hapten.