

EFFECT OF ALCOHOL-FEEDING ON GASTRIC MUCOSAL MAST CELL POPULATION AND GASTRIC TISSUE HISTAMINE CONCENTRATION IN ALBINO RATS

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Summary : Albino rats fed on 30% ethyl alcohol for 60 days demonstrated significant reduction in the histamine concentration of the gastric wall, estimated by bio-assay, as well as in the gastric mucosal mast cell population. It can, therefore be concluded that alcohol liberates histamine from the gastric mast cells even in the presence of alcohol-induced gastritis

Key words : gastric mucosal mast cells alcohol-fed rats gastric wall histamine

INTRODUCTION

Several neural and chemical factors are known to stimulate gastric secretion in man and other experimental animals (3,4,9). Enough evidence is available in the literature incriminating histamine as mediator in gastric secretion, provoked by hormonal and/or neural stimuli and these histamine liberators act by releasing histamine from the local mast cells (1). The degranulation of mast cells leading to histamine liberation results in loss of metachromasia of these cells which causes a decrease in their population on staining with Toluidine blue (10).

In man and other experimental animals, small amounts of ethyl alcohol (upto 15%-20% concentrations) given either orally or parenterally, cause a temporary increase in gastric secretion rich in Hydrochloric acid and mucin, probably acting directly on the gastric glands or through release of histamine from gastric mucosa, while higher concentrations cause gastritis and inhibit gastric secretion (8,13). The present experiment is, however, planned to study the effect of higher concentration of alcohol (30%) on the gastric mucosal mast cell population and gastric tissue histamine content in albino rats.

MATERIAL AND METHODS

Twenty healthy male albino rats weighing between 150-200 g and housed in separate cages were divided into 2 equal groups. Group I served as control and were

given food and water *ad libitum* for 60 days. Group II were treated in the same manner as Group I in addition to intra-gastric administration of 3 ml of 30% ethyl alcohol daily for the entire period. On the last day all the animals were allowed only water and after 24 hrs they were sacrificed and the stomach removed. Each stomach was divided into 2 equal halves by cutting along the greater and lesser curvatures.

From one half of each stomach the glandular portion alone was fixed in 4% aqueous solution of basic lead acetate for 48 hrs. Routine histological procedures followed and 10 μ m thick sections were made and stained in 1% aqueous solution of Toluidine blue for 1 min. The mast cells in the mucosal layer could be easily identified under the microscope by their metachromatic purple stain. Using a calibrated ocular micrometer, the cells were counted under high power objective and expressed for 1 mm² of gastric mucosa.

The other half of each stomach was weighed dry, cut into fine pieces in 2 ml/g of tissue of N-hydrochloric acid and ground up with a little sand in a mortar. 10 ml of distilled water per g of tissue was added during grinding. The extract was boiled for 1 min filtered and neutralised with N-NaOH (2). Histamine concentration of the extract was estimated by the standard biological assay method using terminal ileum of 24 h-fasted, medium-sized guinea pig, in an organ bath fluid, and the presence of histamine was confirmed by mepyramine maleate, 0.2 ml, 2.5×10^{-6} M. 3 point assay was performed in order to calculate histamine concentration which was expressed in μ g/g of tissue.

RESULTS

The results which are summarised in Table I show that there is an equally significant reduction in the mucosal mast cell population as well as in the tissue histamine concentration in the stomachs of alcohol-fed rats ($P < 0.01$).

TABLE I: Effect of alcohol-feeding on mucosal mast cell population (MCP) and tissue histamine concentration of stomach in albino rats (n=10).

Group	Mean MCP \pm S. D.	Mean gastric tissue histamine concentration μ g/g \pm S. D.
I. CONTROL	250 \pm 21	12.32 \pm 3.16
II. ALCOHOL-FED	221 \pm 18	9.14 \pm 2.92

P

<0.01

<0.01

DISCUSSION

It has been suggested that, in rats, about half of the whole body histamine formation takes place in the gastric wall (7). A strong positive correlation between mast cell population and histamine content of tissues in general (11), and stomach in particular (5,6), had been clearly demonstrated. It is obvious from the present findings that alcohol-feeding has significantly reduced the gastric mucosal mast cell population and this decrease is due to degranulation of the cells, which therefore results in significant depletion of histamine from the gastric wall. It can, therefore, be inferred that alcohol action on stomach involves release of histamine from the local mast cells. That alcohol-induced gastric secretion is not inhibited by therapeutic doses of atropine (12) further supports the link between alcohol and histamine. This fact does not, however, exclude the proven action of alcohol in releasing gastrin from the pyloric antrum (8). It is likely that the mechanisms co-exist.

On the other hand, it is well documented in the literature that ingestion of alcohol in concentrations of more than 15%-20% inhibits gastric secretion in man and experimental animals who also suffer from alcohol induced gastritis. A concentration as high as 30% was selected only with the aim of finding out whether alcohol affects gastric mucosal mast cell population even in higher concentrations, in spite of gastritis. In this study, no attempt was made to assess the degree of gastritis induced by alcohol in the animals used. Yet it is reasonable to conclude that ethyl alcohol, even in higher concentrations, can degranulate gastric mucosal mast cells liberating histamine, which, however cannot effect its gastric secretagogue action due to the concurrent gastritis.

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