**ABSTRACT**

Mycotoxins are the toxic metabolites produced by several fungi, with toxicity ranging from vomiting to carcinogenicity. Fungi and mycotoxins are present as contaminants in a wide range of food and feeds, posing health threat to humans and animals. In this study, medicinal herbs, spices and food materials were screened for contamination with fungi and mycotoxins. Inhibition of the growth of *Aspergillus flavus* and *Penicillium citrinum*, and mycotoxin production were carried out using spices. Mechanism of inhibition of Aflatoxin B$_1$ (AFB$_1$) production was studied. Further detoxification study of AFB$_1$ was carried out with organic acid and an herbal drug.

Analysis showed that 92% of the samples were contaminated with fungi. Fungal load in the samples was enumerated, and 45.3% were above the permissible limit set by World Health Organisation. Aspergillus and Penicillium were the predominant fungi isolated, which includes toxigenic strains producing AFB$_1$ and citrinin, respectively. AFB$_1$ was detected only in one sample i.e. peanut, whereas none of the medicinal herbs were detected for natural AFB$_1$ contamination. Spices were used for controlling the growth of *A. flavus* and AFB$_1$ production. Cinnamon and clove exhibited inhibitory effect and their minimum inhibitory concentrations were determined in culture medium. In rice, these spices inhibited AFB$_1$ synthesis without inhibiting fungal growth. The probable mechanism of AFB$_1$ inhibition by clove was correlated with reduced activity of the enzyme, xanthine oxidase. Furthermore, clove exhibited potential in degrading AFB$_1$ at elevated temperature. Clove inhibited *P. citrinum* growth and citrinin production efficiently. The herbal drug, Liv-52, showed the ability to degrade AFB$_1$, however it enhanced *A. flavus* growth and AFB$_1$ production in culture medium. Lactic acid was effective in degrading AFB$_1$ with the formation of two degradation products i.e. AFB$_2$ and AFB$_{2a}$. The degraded AFB$_1$ exhibited reduced cytotoxicity to HeLa cells.