

Effect of safranal, a constituent of *Crocus sativus* (saffron), on methyl methanesulfonate (MMS)-induced DNA damage in mouse organs: An alkaline single-cell gel electrophoresis (comet) assay

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Abstract: The influence of safranal, a constituent of *Crocus sativus* L. stigmas, on methyl methanesulfonate (MMS)-induced DNA damage was examined using alkaline single-cell gel electrophoresis (SCGE), or comet, assay in multiple organs of mice (liver, lung, kidney, and spleen). NMRI mice were divided into five groups, each of which contained five mice. The animals in different groups were received the following chemicals: physiological saline (1 mL/kg, ip), safranal (363.70 mg/kg, ip), MMS (120 mg/kg, ip), safranal (72.70 mg/kg, ip) 30 min prior to MMS administration, and safranal (363.70 mg/kg, ip) 30 min prior to MMS administration. Mice were sacrificed about 2 h after the administration of direct mutagen MMS, safranal, or saline, and the alkaline comet assay was used to evaluate the influence of safranal on DNA damage in different mouse organs. Increase in DNA migration was varied between 9.08 times (for spleen) and 22.12 times (for liver) in nuclei of different organs of MMS-treated mice, as compared with those of saline-treated animals ($p < 0.001$). In control groups, no significant difference was found in the DNA migration between safranal- and saline-pretreated mice. The MMS-induced DNA migration in safranal-pretreated mice (363.70 mg/kg) was reduced between 4.04-fold (kidney) and 7.31-fold (liver) as compared with those of MMS-treated animals alone ($p < 0.001$). This suppression of DNA damage by safranal was found to be depended on the dose, and pretreatment with safranal (72.70 mg/kg) only reduced DNA damage by 20.29%, 21.08%, 31.32%, and 20.88% in liver, lung, kidney, and spleen, respectively ($p < 0.001$ as compared with saline-treated group). The results of the present study showed that safranal clearly repressed the genotoxic potency of MMS, as measured by the comet assay, in different mouse organs, but the mechanism of this protection needs to be more investigated using different in vitro system assays and different experimental designs.

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