This report describes the fatal case of a 13-year-old middle school girl (MSG) whose cause of death might be attributed to a intoxication involving Venlafaxine (VEN). MSG's mother had the history of depression for 11 years. She and her daughter were found dead in the same room of their apartment, with hanged herself. We analyzed the postmortem of MSG which were requested to our institute for the cause of her death. We couldn't get any blood samples from MSG's postmortem. So, we could detect unchanged VEN in the liver, spleen, brain and gastric contents using capillary gas chromatography with a nitrogen-phosphorus detector and gas chromatography-mass spectrometry (GC-MS). The following VEN concentrations were determined in MSG's postmortem tissues: 37.8 µg/kg (liver), 39.9 µg/kg (spleen) and 19.6 µg/kg (brain); other antidepressants, alcohol, and benzodiazepines couldn't be detected in specimens. The cause of her death was determined to be the intoxication of VEN resulting from its overdosage. The manner of death was postulated to be the homicide by her mother.

The Flavonoid Morin Inhibits Dimethylnitrosamine-Induced Liver Damage in Rats
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Morin, one of the major natural flavonoids has been reported to exhibit a wide range of pharmacological properties. In this study, we investigated the hepatoprotective effect of morin on the dimethylnitrosamine (DMN)-induced liver damage in rats. Oral administration of morin (10, 20mg/kg daily for 4 weeks) into the DMN-treated rats remarkably prevented the elevation of serum alanine transaminase, aspartate transaminase and alkaline phosphatase, and bilirubin levels. Morin also increased serum protein level and reduced the hepatic level of malondialdehyde in DMN-treated rats. Furthermore, DMN-induced elevation of hydroxyproline content was reduced by the treatment of morin and which result was consistent with a histochemical analysis of liver tissue stained with Sirius red. In conclusion, these results demonstrate that the in vivo hepatoprotective effect of morin against DMN-induced liver injury, and suggest that morin may be useful in the prevention of liver damage.

Induction of cyclooxygenase-2 by collagen and gelatin in murine macrophages
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Gelatin protein is derived from animal collagen tissues and is therefore present in many kinds of animal protein food. The biological origin and biocompatibility of gelatin has led to wide-ranging applications in the pharmaceutical and medical fields; for example, as sealants for vascular prostheses, bone-repairing materials, wound healing agents and scaffolds for tissue engineering purposes. In the present study, we investigated the effects of collagen and gelatin on the cyclooxygenase-2 (COX-2) gene which plays a crucial role in many physiological and pathological processes in macrophages. Collagen and gelatin significantly increased the production of prostaglandin E2 (PGE2) and the expression of COX-2 mRNA in dose-dependent manner. To investigate the significant cis-acting regions which COX-2 promoter, transient transfection experiments were carried out using reporter vectors harboring deleted COX-2 promoters. The transcriptional factor binding sites for activator protein 1 (AP-1) and NF-kB between -574 and -51 could be important for the induction of COX-2 mRNA by collagen and gelatin. The results of these studies suggest that induction of transcriptional activation of COX-2 by collagen and gelatin might be mediated through the AP-1 and NF-kB activation.