Canola oil toxicity in SHRSP and its sex difference

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Background
Canola oil ingestion gives rise to various untoward responses in SHRSP. The adverse effects include the life shortening with facilitated hypertension, decreased platelet count, shortened blood coagulation time, blunted peripheral glucose uptake, etc. and most of the responses are conspicuous in male animals. The aim of this study is to review the adverse effects of canola oil in SHRSP in detail, investigate sex differences in the responses and consider underlying mechanisms.

Methods
SHRSP of both sexes were examined for life-span, that is, 5-week of age the rats were fed on AIN-93 diet containing 10w/w% soybean oil (control) or canola oil as the sole dietary fat and tap water, given ad libitum. In another series of experiment animals were sacrificed after an 8-week ingestion of the soybean oil or canola oil diet and examined for hematological, pathological and biochemical changes.

Results and conclusion
Survival time of male SHRSP was shortened significantly by canola oil but not in female. In the animals of either sex pathological findings were similar between the control and canola oil ingestion groups, whereas canola oil-induced injuries in the heart and the kidney were found in the male of 8-week ingestion. In these animals facilitated hypertension, decreased platelet counts, blunted time-course of blood glucose reduction in OGTT, etc. were observed but neither were found in the female animals. In the male animals canola oil inhibited the gene and protein expressions of testicular CYP11a and CYP17. In contrast no downregulation of mRNA for CYP11a was found in the adrenal gland, but mRNA for CYP11b2 which leads to the increased plasma aldosterone was significantly increased. Thus, at least the testis is one of the target organs of canola oil toxicity. The reduced testosterone production especially due to the inhibition of CYP11a and CYP17 may play a key role in the adverse effects. Moreover, there are clear sex difference in the canola oil toxicity, and some regulation by testosterone of aldosterone production may underlie the sex difference in the toxicity.

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