The target of canola oil toxicity in SHRSP and mechanisms underlying the toxicity

Naoki Ohara[1], Mai Nishikawa[1], Kenjiro Tatematsu[2], Yukiko Naito[3], Daisuke Miyazawa[1], Harumi Okuyama[1]

[1]Kinjo Gakuin Univ.Coll.Pharm., [2]Gifu Pharmaceut.Univ.Radiochem., [3]Kitasato Univ.Sch.Allied Health Sci.

[Aim of the study] Canola oil (CAN) ingestion is known to shorten the life-span of male SHRSP and the life-shortening is preceded by a decrease in plasma testosterone and an increase in aldosterone. The present study was conducted to clarify the target of the toxicity. [Methods] Male SHRSP were given an AIN-93G diet containing 10w/w % soybean oil (Control) or CAN and tap water *ad libitum* for 8 weeks. At the 8th week the animals were sacrificed and plasma concentrations of testosterone, aldosterone, LH and FSH were measured using ELISA kits. Testes were isolated and histopathologically examined. Gene expressions for ACE and ACE2 in the lung, kidney and testis were measured. [Results and discussion] CAN-induced decrease in plasma testosterone and increase in aldosterone were confirmed. Both, plasma LH and FSH were similar between the Control and CAN groups. Leydig cell counts in the testis were comparable between the two groups. The ratios of gene expression for ACEs to that for ACE (ACE2/ACE) in both, the testis and the kidney of the CAN group were significantly lower than those in the Control group, while ACE2/ACE in the lung of the two groups were similar.These results demonstrate that CAN ingestion does not affect gonadotropin secretion from the pituitary while suppresses the testicular function without pathomorphological changes nor Leydig cell count. The decreased ACE2/ACE in the testis and the kidney of the CAN group may affect the local renin-angiotensin-aldosterone system (RAAS). In this presentation the relationship among testosterone, aldosterone and RAAS in the CAN toxicity is discussed.