Results: The treatment of Centella spp. revealed significant reduction of lipid droplet deposition and decrease of the size of lipid droplet in the adipocyte. Molecular biological experiment suggested that the treatment of Centella spp. significantly inhibited the expression of Trib 3: more than 100% of Trib3 was decreased compared to the control. On the contrary, the expression of adiponectin, CTRP 6 and Glut4 were considerably increased by the treatment of these herbal extracts.

Conclusion: These results suggest that the adaptation of these herbal extracts have significant potential to inhibit fatal etiological triggering of cardiovascular disease causing by the obesity. Hence, these herbal extract might be a therapeutic target to fight against obesity and its associated diseases in modern industrial countries.

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Experimental Study on Protective and Anti-obesity Effects of Inulin from Chicory (Cichorium intybus L.) on Quail Model

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Purpose: Inulin from Cichorium intybus L., a kind of Chinese Materia Medica, is potential therapeutics that act alone or supplement on serum metabolic parameters in the prevention and treatment of obesity. The present investigation was undertaken to study the protective and anti-obesity effects of inulin from Cichorium intybus L. on serum lipid concentration and abdominal fat pad mass in quail model induced by protein and purine rich diet.

Methods: Quails were divided randomly into 5 groups according to body weight: normal group, model group, positive control group and chicory inulin high and low dosage groups. The normal group was fed with the common feedstuff, and the other groups were fed with protein and purine rich diet. Positive control group was given Fenofibrate 100 mg/(kg•d). Chicory inulin groups were given inulin 10, 5 g/(kg•d) respectively. All quails were given distilled water. Serum triglyceride (TG), abdominal fat pad mass and acetyl-Coa carboxylase (ACC), fatty acid synthase (FAS) activity were determined.

Results: Compared with normal group serum TG level of model group was significantly higher on 21d and 28d and abdominal fat pad mass increased on 28d. Compared with model, fenofibrate decreased serum TG level on 21d and 28d. Chicory inulin decreased TG significantly and abdominal fat pad mass on 28d. ACC protein expression and fatty acid synthase (FAS) activities were decreased in chicory inulin groups significantly. However, serum cholesterol (TC), low-density lipoprotein (LDL) and high-density lipoprotein (HDL) level were not significantly altered by treatment.

Conclusion: Inulin of Cichorium intybus L. significantly improved lipid metabolism of diet induced abdominal obesity in quails. The possible mechanism of anti-obesity activity appears to be either decreasing ACC protein expression and FAS activity, or decreasing serum TG level property.

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Aqueous extract of solanum nigrum activated programmed cell death and enhanced cisplatin/doxorubicin induced cytotoxicity on human hepatocellular carc

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Purpose: In traditional Chinese medicine, the aqueous extract of solanum nigrum (AESN) is a key ingredient presented in various formulas for dealing with cancer patients. Recent studies suggested AESN is capable of activating programmed cell death in many human cancer types both in vitro and in vivo. This study is to examine the antitumor potential of AESN in integration with standard chemotherapeutic drugs, cisplatin and doxorubicin, on human hepatocellular cancer cells.

Methods: Human hepatocellular carcinoma cells, Hep3B and HepJ5, were treated by 0 to 10 mg/ml AESN for 48 hr to determine the cytotoxicity. Hep3B and HepJ5 cells were treated with 0, 0.5 or 1.0 mg/ml AESN with 0 to 20 μM cisplatin or 0 to 10 μM doxorubicin respectively for 48 hr to evaluate the combined cytotoxic effects. The activation of programmed cell death markers, caspase-3 and caspase-7 for apoptosis and LC3 A/B for autophagy were also determined by western blotting assay on AESN-treated cells.

Results: The half-maximum inhibitory concentrations (IC50s) of AESN on Hep3B and HepJ5 cells were 0.96 and 0.97 mg/ml respectively. The co-treatment of AESN (0.5 mg/ml) reduced the IC50s of cisplatin from 6.75 to 2.75 μM on Hep3B cells and 8.71 to 2.84 μM on HepJ5 cells, whereas the IC50s of doxorubicin were reduced from 4.65 to 1.31 μM on Hep3B cells, and 6.39 to 1.42 μM on HepJ5 cells. AESN induced the accumulation of LC3 A/B II and the cleavage of caspase-7 but not caspase-3 on Hep3B and HepJ5 cells suggested the induction of autophagic and apoptotic cell death by AESN.

Conclusion: This study indicated that AESN activated programmed cell death including caspase-7 related apoptosis and autophagy to enhance cytotoxicity induced by cisplatin and doxorubicin in human hepatocellular carcinoma cells. These experimental evidences suggested AESN is a potential ingredient to develop the novel integrated chemotherapy with cisplatin or doxorubicin on treating hepatocellular carcinoma.

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