to suppression of tumor growth and angiogenesis.

Keywords: Chemoprevention, Natural compounds

Room R-P4 Sep. 21 (Fri) 16:40 - 17:30

P23-5 Cancer prevention (3)
がんの予防 (3)

Chairperson: Yoshio Honma (Shimane Univ., Faculty of Med., Dept. of Life Science)
座長：本間 良夫（島根大学　医学部生命科学講座）

P-3366 Inhibition of Ultraviolet B-induced carcinogenesis by oral consumption of Brazilian medicinal plant

Harukuni Tokuda, Nobutaka Suzuki (Kanazawa Univ. Grad. Sch. of Med. Science)

ブラジル産薬用植物による紫外線照射発がんに対する抑制について

徳田 春邦、鈴木 信孝（金沢大学大学院医学系研究科）

Tabebuia avellanedae (Bignoniaceae) (TA), which is native to South America from Brazil to Argentina, is well known in traditional medicine as tea type used for the treatment of various disease. Previously, oral administration of extract of the inner bark (TA essence) inhibited the promotion stage of carcinogenesis in mouse skin (DMBA/TPA) suggesting that the extract might be a functional material for chemoprevention as well as vegetables. Several effects of UVB (290-320 nm) are thought to present in the solar spectrum and contribute to skin carcinogenesis. We present here that oral feeding of TA essence to skin elicits the inhibition of UVB induced cutaneous damage. In this experiment, SENCAR mice were exposed to UVB dose three times a week from FS20S lamps and oral feeding of 0.0025 % of TA essence, two weeks before and after tumor initiation resulted in decreased 70 % of the papillomas incidence and multiplicity when compared with the control after findings provide scientific evidence to support al in treatment of UVB irradiation damages.

, Facul of Agric. Kinki Univ.)

tural compounds

e aloe emodin on the development of s in Min mice

Takeshi Chihara[1], Takaaki Kaneko[1], Hidehiko Beppu[1], Kazumasa Wakamatsu[2], Masanori Shinzato[2], Shigeru Sonoda[1] ([1]Fujita Health Univ. Nanakuri Inst., [2]Fujita Health Univ. Sch. of Health Sciences)

Minマウスの大腸腫瘍発生に対する低用量アロエエモジンの影響

新保 寛[1]、千原 猛[1]、金児 孝晃[1]、別府 秀彦[1]、若松 一雅[2]、新里 昌功[2]、園田 茂[1] ([1]藤田保健衛生大学 七栗研究所、[2]藤田保健衛生大学 医療科学部)

Aloe emodin (AE), a natural anthraquinone compound, has been reported to have anticancer activity in various cancer cell lines and anti-inflammatory effects in murine macrophages. In this study, we investigated the cancer chemopreventive effects of AE in a Min mouse model. In the first experiment, male Min mice were fed a basal diet or diets containing 5 ppm AE and 10 ppm AE for 12 weeks. Only dietary administration of 5 ppm AE significantly reduced the numbers of colorectal tumors in Min mice. In the second experiment, we investigated the effect of AE on colitis-related colon carcinogenesis in Min mouse treated with dextran sodium sulfate (DSS). Female Min mice were exposed to 1% DSS in their drinking water for 7 days. AE was given to mice at a dose level of 5 or 50 ppm in their diet for 5 weeks. Although preliminary, feeding with 5 ppm and 50 ppm AE tended to decrease the numbers of colorectal tumors in Min mice. Further experiments are now underway. These results suggest that dietary administration of low-dose AE might have chemopreventive effects against colorectal tumor development in Min mice.

Keywords: Min mouse, Aloe emodin

P-3368 Anthraquinone Derivatives Induces Apoptosis in Human Ovarian Cancer Cells

YI-JUNG HSIEH[1], LI-MIN LIU[2], TZONG-DER WAY[1] ([1]Grad. Inst. of Biotechnology, China Medical Univ., Taiwan, [2]Grad. Inst. of Pharmaceutical Chemistry, China Medical Univ., Taiwan)

Human ovarian cancers cells overexpressing HER2 are more aggressive tumors with poor prognosis, and resistance to chemotherapy. This study investigates antiproliferation effects of anthraquinone derivatives of rhubarb root on human ovarian cancer cells. Emodin and aloe-emodin of anthraquinone derivatives showed antiproliferative effects on HER2-overexpressing SK-OV-3 cells. Two main signalling pathways that lead to apoptosis in mammalian cells: the intrinsic pathway and the extrinsic pathway. Binding of specific ligands to death receptors is the first step of the extrinsic apoptotic pathway, also known as the death receptor pathway. Emodin and aloe-emodin was also induced up-regulation of DR5. Therefore, this study highlighted emodin and aloe-emodin as processing anti-proliferative activity against HER2 overexpression or HER2-basal expression in ovarian cancer cells and playing important roles in apoptotic induction of human ovarian cancer cells.

Keywords: Apoptosis, Emodin

P-3369 Koelreuteria elegans induces G2/M-phase arrest and autophagy attributing to cell death of human colon cancers

Lin Jiun-Shiang, Chen Pei-Ni, Hsieh Yih-Shou (Inst. of Biochemistry and Biotechnology, Chung Shan Medical Univ.)

Koelreuteria elegans (KE), a deciduous tree belonging to Taiwan indigenous species. The whole plant of KE has been reported to possess various therapeutic effects, including dispelling wind and clearing heat in traditional Chinese medcine, relieving cough, malaria. However, whether KE possesses anti-tumOral activity and its underlying mechanisms still remain unclear. Here, we prepared a *Koelreuteria elegans* extract (KEE) and tested the effects of KEE on colon cancer cell line DLD-1. Our results revealed that KEE induced arrest of G2/M-phase but not sub-G1 phase of DLD-1 cells, and consequently led to reduced cell proliferation and viability. In addition, KEE also triggered autophagy of DLD-1 cells, which was demonstrated by using electron microscopy analyses. Further investigation showed that KEE induced LC3 processing, elevated levels of ATG3 and ATG5, and diminished level of Bcl-2; but insignificantly affected activation of caspase-8 and caspase-9 in DLD-1 cells. Our results indicate that KEE diminishes cell viability of DLD-1cells via induction of G2/M-phase arrest and autophagy but not apoptosis, providingevidences that KEE should be beneficial to treat human colon cancer.

Keywords: autophagy, Chemoprevention

P-3370 Anti-carcinogenic potential of an Indian medicinal plant Swertia chirata

Prosenjit Saha, Debolina Pal, Subhayan Sur, Biswas Jaydip, Chinmay Panda (Chittaranjan National Cancer Inst.)

Swertia chirata, finds its mention in Charaka Samhita a classical medical text of ancient India. Experimental revalidation of the medicinal properties of this plant along with chemical analysis is likely to open up new avenues for its multispectrum use. The anticarcinogenic potential of *S. chirata* had remained unexplored until our group took up the investigations in collaboration with National Research Institute for Ayurvedic Drug Development, Kolkata. We have been reported that aqueous extract and amarogentin (Amg), the bitter component of this plant showed chemoprevtive activity on a DMBA induced mouse skin carcinogenesis model. We also found that Amg could restrict the mouse liver carcinogenesis at premalignant stage induced by tobacco related carcinogen NDEA. The restriction of carcinogenesis by Amg has been seen due to inhibition of cellular proliferation and induction of apoptosis. Moreover, our preliminary observation showed that Amg could reduce the expression of beta-catenin, c-myc and cyclinD1 along with increase in Bax:Bcl2 ratio. Our reports definitely point towards the promise that *Swertia chirata* as a future chemopreventive and therapeutic drug for cancer.

Keywords: Cancer prevention, Natural compounds

Room R-P4 Sep. 21 (Fri) 15:50 - 16:40

P23-6 Cancer prevention (4)
がんの予防 (4)

Chairperson: Masahiko Kanamori (Dept. of Human Science 1, Univ. of Toyama)
座長：金森 昌彦（富山大学　医学部人間科学 1 講座）

P-3371 A highly polar xanthophyll of fucoxanthinol induces apoptosis in colonosphere

Masaru Terasaki, Sonoko Masuda (Dept. Hlth. Environ. Sci., Hlth Sci. Univ. Hokkaido)

高極性キサントフィル fucoxanthinolは colonosphereの apoptosisを誘導する

寺崎 将、増田 園子（北海道医療大学　薬学部衛生薬学講座）

Many carotenoids have cancer chemopreventive potency. However, there are little reports on mechanism of carotenoid to cancer stem cell. Fucoxanthinol (FuOH) is a biotransformed type of fucoxanthin, which highly accumulated in brown alga, and has potent anticancer effect. We examined the effects of FuOH on disinteglation and apoptosis induction to spheroid containing colorectal cancer stem cell (CCSC) abundantly. The colonospheres (Csp) from HT-29 and HCT116 were identified to be CCSC rich cell population because of higher expression of CD44 and EpCAM, representative cell surface markers of CCSC, compared with their parental cells. The 0.1 - 5.0 μ mol/L of FuOH addition disinteglated Csp in both cells clearly, reduced their cell viabilities and induced apoptosis at dose dependent manner. The IC_{50} values for disinteglation of Csp were 1.8 and 1.3 μ mol/L at HT-29 and HCT116 cells, respectively. FuOH proved to be beneficial to apoptosis induction of Csp from two different characters of culture cells at the