BZL, GTN024 を添加後, 固定・染色し細胞内型虫体数を測定した. また BZL, GTN024 の活性酸素 (ROS) 産生を測定した. **【結果・考察】** BZL, GTN024 はいずれも細胞内型原虫に対して抑制効果を示し, IC50 値は 1.75μM, 0.16μM であった. 感染 24 時間後に化合物を添加すると, BZL では抑制効果が見られず, GTN024 では細胞内型虫体に抗原虫作用があることが示唆された. また, HT1080 で ROS 産生量を測定したところ, 両者ともに ROS 産生が認められたが, GTN024 の ROS 産生量は BZL の 7 割程度であった. 以上より, GTN024 は BZL とは異なる作用機序を持つことが示唆された.

19. Epithelial-mesenchymal Transition and Pathogenesis of Esophageal Carcinosarcoma

Takuro Nakazawa[1], Sumihito Nobusawa[1], Hayato Ikota[1], Hiroyuki Kuwano[2], Izumi Takeyoshi[3] and Hideaki Yokoo[1]

(1 Department of Human Pathology, Gunma University Graduate School of Medicine)

(2 Department of General Surgical Science, Gunma University Graduate School of Medicine)

(3 Department of Thoracic and Visceral Organ Surgery, Gunma University Graduate School of Medicine)

The pathogenesis of sarcomatous component in esophageal carcinosarcoma is unclear. To investigate the involvement of epithelial-mesenchymal transition (EMT) in sarcomatous differentiation, we performed immunohistochemistry for Slug, Twist, ZEB1, and ZEB2, transcription factors associated with EMT and E-cadherin, in 14 cases of SpCC of the esophagus. In order to verify the neoplastic nature of sarcomatous components, TP53 mutation status and protein expression were examined in each case. Nuclear ZEB1 expression was extensive in the sarcomatous component, greater than invasive front of carcinoma components ($p<0.0001$). Membranous E-cadherin expression was mostly lost in sarcomatous cells in all cases ($p<0.0001$). The p53 expression pattern was almost concordant between the two areas in all cases. TP53 mutation analysis revealed that 7 cases harbored identical mutations in both components. One case had mutations only in the sarcomatous component. It was noteworthy that none of them harbored mutation in exon 5, unlike conventional esophageal squamous cell carcinoma. These findings show that ZEB1 are widely expressed in sarcomatous area in esophageal carcinosarcoma, suggesting the involvement of EMT. The avoidance of exon 5 in terms of TP53 mutation may also be a feature of the tumor.

20. Sodium Selenite Supplementation Does Not Protect Cancerous Esophageal Cells from X-ray Irradiation Treatment

Irma M. Puspitasari[1,2], Chiho Yamazaki[1], Rizky Abdulah[2], Satomi Kameo[1], Takashi Nakano[3] and Hiroshi Koyama[1]

(1 Department of Public Health, Gunma University Graduate School of Medicine)

(2 Department of Pharmacology and Clinical Pharmacy, Faculty of Pharmacy, Universitas Padjadjaran, Indonesia)

(3 Department of Radiation Oncology, Gunma University Graduate School of Medicine)

【Background】 The role of radioprotective compounds is very important in clinical radiotherapy. Radioprotective compouds should protect normal tissues from both acute and late radiation damage without protecting the cancer tissues. Previous study revealed that 50nM of sodium selenite supplementation on non-cancerous human esophageal cells (CHEK-1 cells) before X-ray irradiation treatment can protect the cells from radiation induced-damage. However, the effects of sodium selenite supplementation on cancerous cells in X-ray irradiation treatment remain unknown. 【Objective】 To investigate the effects of sodium selenite supplementation on cancerous cells with X-ray irradiation treatment. 【Methods】 CHEK-1 cells and cancerous human esophageal cells (TE-8 cells) were cultured in RPMI medium enriched with 10% fetal bovine serum and 1% antibiotics (Penicillin and streptomycin). The IC50 of sodium selenite on CHEK-1 and TE-8 cells was determined by conducting citotoxicity assay. Cell survival of both cells post 2Gy X-ray irradiation was observed by using cell viability and colony formation assay. 【Results】 The cell IC50 of sodium selenite on CHEK-1 cells was determined to be 3.6μM and 7.4μM on TE-8 cells. Non-cancerous CHEK-1 cells with 50nM sodium selenite supplementation had a higher survival rate and cell viability at 72h post 2Gy X-ray irradiation compare to the control (0nM). In contrast, TE-8 cells with 50nM sodium selenite supplementation had a lower survival rate and cell viability at 72h post 2Gy X-ray irradiation compare to control. 【Conclusion】 The results suggest that sodium selenite supplementation at a dose 50nM for 72h before irradiation can protect non-cancerous human esophageal cells but does not protect cancerous esophageal cells from X-ray irradiation treatment.