516

DEVELOPMENT OF IRMA WITH NEW MONOCLONAL ANTIBODIES, 130-22 AND 145-9. M.Kunimatsu, K.Endo,T.Nakashima , Y.Watanabe, H.Ohta, Y.Kawamura,H,Sakahara,M.Koizumi,K.Nakajima*, K.Torizuka*,Y.Matsuoka**,T.Yoshida**, T.Nakagawa**,N.Yamaguchi**,Koto Univ.,Kyoto *Fukui Medical Univ.,Fukui,**Mie Univ.,Tsu.

Recently,we have produced two new monoclonal Abs,130-22 and 145-9,against pulmonary carcinoma cell line (PC-9).Using these two monoclonal Abs,fundamental study of new IRMA is reported.The assay was performed by one step method using polystyrene beads coated with 130-22 or 145-9.

Both 130-22 and 145-9 bind CA125 antigens which is well known as a tumor marker of ovarian carcinoma.From competitive binding inhibition study,both 130-22 and 145-9 recognize distinct different antigenic determinants with OC125 developed by the immunization with ovarian cancer cells.130-22 and 145-9 recognized very similar antigen epitopes to each other.In the 9 IRMAs using combinations of three monoclonal Abs,130-22, 145-9 and OC125,the best standard curve was obtained in the case of I-125 labeled OC125 and 130-22 or 145-9 coated beads.

In the assay using I-125 labeled 130-22 and 145-9 coated beads, serum antigen levels in patients with ovarian carcinoma were found to be increased in 19 of 43 cases.However,in patients with benign ovarian tumors, abnormal values were seen only in 4 of 34 cases. Therefore,IRMA with new monoclonal Abs,130-22 and 145-9 seemed to be promising for clinical use in ovarian cancer.

518

THE EFFECT OF FERRIC CITRATE ON Ga-67 ACCUMULATION IN TUMOR CELLS. H.Murahashi,H.Wakao,H.Otsuka,S.Tachikawa, H.Ikuta and T.Higashi. Kanagawa Dental College,Yokosuka.

It is well known that the mechanism of gallium accumulation into tumor cells is mediated with transferrin receptor as well as iron. The present study was designed to explore the difference between the mechanism of gallium accumulation and that of iron. Tumor cells (Mouse Leukemia, L-5178Y,2.5×10^5cell/ml) were suspended in Fischer's medium containing 10% horse serum and were incubated at 37°C. Ga-67 was administrated in the medium which transferrin in the medium was saturated with ferric citrate (0.5,5.0μg/ml), then, Ga-67 uptake into the tumor cells was counted at 1,3,6, hours following Ga-67 administration. The effect of ferric citrate administration in the medium on the cell growth was not recognized. However, Ga-67 accumulation in the control group was inhibited about 38% by ferric citrate administration. Furthermore, Monensin which inhibits the recycle of transferrin receptor was added in the medium, thus, Fe-59 accumulation was diminished compared with Ga-67 accumulation. From these results, it was suggested that the mechanism of Ga-67 uptake into tumor cells differ from that of Fe-59.

519

THE EFFECTS OF ANTI-TUMOR DRUG (ADRIAMYCIN) ON Ga-67 AND Fe-59 UPTAKE IN CULTURED TUMOR CELLS. S.Tachikawa,H.Motohashi, H.Murahashi,H.Wakao and T.Higashi. Kanagawa Dental College, Yokosuka.

Previously, we reported that the Ga-67 uptake into cultured tumor cells (Mouse leukemia L5178Y) increased by adriamycin treatment. In this study, we were designed to explore the mechanism of increase of Ga-67 uptake into cultured tumor cells by adriamycin treatment. In addition to, we studied also the Fe-59 uptake into cultured tumor cells by adriamycin treatment increased as comparison with that of control.

However, the increase of Fe-59 uptake was less than that of Ga-67. Futhermore, we studied the effect upon the cell population following adriamycin treatment using flow cytometry.

Thus, we found that the cell population is synchronized on G2+M phase of cell cycle following adriamycin treatment.

Pleviously, we reported that the Ga-67 uptake increased at G2+M phase of cell cycle. In addition, we discovered that the microvilli of tumor cell surface is disappered following adriamycin treatment using scanning electron microscopy. From these results, we postulated that the increase of Ga-67 uptake into tumor cell following adriamycin treatment is due to the fact that cell population of tumor cell is synchronized on G2+M phase by adriamycin.

520

THE RELATIONSHIP BETWEEN CYTOSKELETAL FILAMENTS AND Ga-67 UPTAKE IN MOUSE LEUKEMIA CELLS. H.Otsuka[1],H.Ikuta[1], T.Higashi[1],T.Takahashi[2],K.Takahashi[2], Y.Jinbu[3]and Y.Akasaka[3]. 1.Department of Radiology. Kanagawa Dental College. 2.Department of Oral Anatomy. Kanagawa Dntal College. 3.Department of Dentistry and Oral Surgery.Jichi Medical School.

In this study,we investigated the effect of cytoskeletal filaments,constituent element of cytoskeleton,on Ga-67 uptake into the cultured mouse leukemia cells. At first cytochalasin B,specific inhibitor of microfilaments,was added into the cultured cell suspensions. We can't found decrease of Ga-67 uptake into tumor cells following addition of 10uM cytochalasin B. However,the Ga-67 uptake significantly decreased following addition of 50uM cytocahlasin B. Furthermore,we examined the effect of cytochalasin B on the structure of cell surface using electron microscopy. After addition of 50uM cytochalasin B into cultured tumor cell suspension,a disappearance of mictovilli in these tumor cell surfaces was observed. Subsequently,vinblasine(0.5~50uM), specific inhibitor of microtubles,was added to the cultured tumor cell suspension. In spite of complete desruption of microtubles following vinblastine addition,the Ga-67 uptake into tumor cells only slightly decreased as compared with control cells. From these results,we postulated that a microfilaments play an important role in Ga-67 uptake within the tumor cells.