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CORRELATION BETWEEN SERUM UIBC AND DISTRIBUTION OF GA-67. S. Nakano, Y. Hasegawa, K. Ibuka, T. Hashizume, A. Noguchi, T. Okishio, and S. Ishigami. The Center for Adult Diseases, Osaka.

In order to clarify clinically the relation between iron and Ga-67 metabolism, we studied the effect of serum unsaturated iron binding capacity (UIBC) on the distribution of Ga-67. Quantitation of uptake of liver, hepatoma, spleen, lumbar spine, and abdominal area of Ga-67 was carried out by the region of interest (ROI) technique using gamma camera. Abdominal area was chosen as reference and the ratios of the mean count rate of ROI of liver, hepatoma, spleen and lumbar spine to that of reference ROI were correlated with UIBC. One hundred and nineteen five cases were studied and consisted of hepatoma 44, malignant lymphoma 26, other malignant diseases 59, liver cirrhosis and chronic hepatitis 41, other benign diseases 25. There was good correlation between UIBC and liver to reference ratio ($r=0.66$, $p < 0.01$) and between UIBC and hepatoma to reference ratio ($r=0.51$, $p < 0.01$). The mean and standard deviation of spleen to reference ratio was 1.23 ± 0.24 ($n=74$), and that of lumbar spine to reference ratio was 1.51 ± 0.26 ($n=120$) and they did not show so much variation as the liver to reference ratio. There was no correlation between liver to spleen ratio of Ga-67 scan and that of Tc-99m Sn colloid scan.

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BINDING OF Ga-67 TO FERRITIN. K.Nakamura, H.Kawaguchi and H.Orii. Tokyo Metropolitan Institute of Medical Science. Tokyo.

Whether ferritin is involved in the distribution of Ga-67 has not been elucidated. We have investigated the binding of Ga-67 to ferritin in vitro, comparing with Fe-59. Ferritin prepared from rat liver or spleen (Fe-rich ferritin) had a strong affinity to Ga-67, while that from heart or kidney (Fe-poor ferritin) had a weak one. When Fe was removed from ferritin (i.e., apo-ferritin was formed), it could not bind to Ga-67, and the reverse was found. Binding of Ga-67 to ferritin was inhibited by transferrin ($+HCO_3^-$), suggesting that transferrin have a higher affinity to Ga-67 than ferritin. This fact was also found for Fe-59. Release of Fe from Fe-transferrin was enhanced by ATP, citrate or ascorbic acid, although these agents had no effect on the Ga-release from Ga-transferrin. These results indicate that both the Fe-core in ferritin and the release of Ga from transferrin are required for the Ga-ferritin binding. We have already found no accumulation of Ga-67, which was injected to rat *in vivo*, in the ferritin fraction of either liver, kidney, or tumor. The difference of the involvement of ferritin between *in vivo* and *in vitro* may give a clue to the species of Ga-67 presented in the cell, and it furthermore suggests that ferritin is one of points to distinguish Ga from Fe in the cell.

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CHANGES IN UPTAKE OF Ga-67 AND I-125-TRANSFERRIN BY TRANSFORMATION OF HAMSTER EMBRYO CELLS. A.Muranaka, M.Sawai, J.Saito and Y.Ito. Kawasaki Medical School. Kurashiki.

In the tumor accumulation of Ga-67-citrate (Ga), we reported already that the kinetics of Ga in tumor cells were different from those of I-125-transferrin (I-Tf) and another factors besides Tf might play important role. In present studies, using the malignant transformation of hamster embryo (HE) cells treated with 4NQO , changes in Ga and I-Tf uptake were investigated. Transformed HE cells (HEA-3) produced malignant and transplantable tumors in adult hamsters. The chromosome numbers of HEA-3 increased compared with those of normal HE cells. When $50\sim100$ $\mu g/ml$ of Tf was added, Ga uptake by HEA-3 increased about twice that in MEM only. On the other hand, Ga uptake by normal HE cells showed no marked increase. However, I-Tf uptake by HEA-3 was not greater than that by normal HE cells regardless of trypsinization. Therefore, changes in Ga uptake by transformation of HE cells are presumably affected by another factors (e.g. internalization into cells) rather than the quantity of Tf receptors.

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DISTRIBUTION OF Ga-67 IN THE CELL. K. Nakamura, H.Kawaguchi and H.Orii. Tokyo Metropolitan Institute of Medical Science.

Distribution of Ga-67 is consisted of 4 processes; transport in the serum, and through the membrane, transform and accumulate in the cell. Many factors, especially Fe, have influenced these four processes. In present paper, we have described the effect of Fe in the cell fraction on the distribution of Ga-67, so that the latter process has been clarified. Rat tissue homogenates were incubated with Ga-67 and the differential centrifugation was carried out. The results were obtained as follows; (1) In liver, kidney, heart, spleen, and tumor, Ga-67 was primarily accumulated in the lysosome fraction, which has played a major role in the Fe-binding capacity of the cell. (2) In kidney, Ga-67 was observed in almost all fractions. This fact has been explained by the comparatively low Fe-saturation ratio in these fractions. (3) In presence of Fe-chelating reagents, Fe in fractions were reduced, and Ga-67 was also decreased in these fractions but in the supernatant. It has suggested that the strong affinity to Ga-67 in the supernatant has inhibited the localization of Ga-67 in the lysosome fraction. (4) Fe-reagents has increased the Fe-content in all fractions, and where Ga-67 was found to be reduced. However, in some fractions containing Fe-colloids, Ga-67 was localized in a higher extent.