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THE INFLUENCE OF NEURAMINIDASE ON GROWTH AND METASTATIC ABILITY OF A NEOPLASTIC TUMOR

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Neuraminidase is an alpha-glycosidase enzyme, which cleaves terminal sialic acid residues from sialylated substrates, such as glycoproteins and glycolipids. By doing so, neuraminidase remodels cell surface charge and surface antigens, and regulates many biological processes. In the present study we set out to asses neuraminidase influence on the development of an experimentally transplantable tumor and its metastatic action in the lungs. The aim was to answer the question of whether neuraminidase, while administered both in vivo and in vitro, could influence the growth of Morris hepatoma and its metastatic ability in the rat. 124 female Buffalo breed rats, weighing 160±10 g, 12 weeks old were used for the study. The experiment was continued for 21 days. The rats ware administered Clostridium perfringens neuraminidase (Sigma) into the caudal vein or into tumor mass. Biometric measurements were taken of the tumor site in the left posterior extremity for the assessment of a tumor mass and volume. Internal organs were macroscopically inspected for the presence metastases. A scrap of the left posterior extremity from the implanted site, lungs (in which Morris hepatoma metastases are usually found), and other organs were taken for histopathological examination. Some of the histological material was preserved for NM-23-H2 protein assay - a marker of Morris hepatoma growth and metastatic abilities. Blood was taken to determine Cathepsin B activity -a marker of hepatoma progression, and GOT, GPT, GGTP- as typical markers of liver damage. In all groups of neuraminidase-treated rats, which obtained different doses of it, only single metastases were found in the lungs, while in the control group 80% of the rats had metastases in this location. This trend was not observed in case of peritoneal metastases, which were similar in both control and experimental groups. GOT, GPT and GGTP levels were higher in the investigated groups with Morris hepatoma than those in the healthy rats. There was no correlation found between the levels of those enzymes and neuraminidase administration. The experiment also revealed an increased concentration of cathepsin B in all rat groups with Morris hepatoma implanted, which might indicate cathepsin B as a new auxiliary marker of Morris hepatoma growth in similar experimental models. Any influence of neuraminidase, administered in vivo or in vitro, on cathepsin B levels in this model was not detected. Thus, the results obtained in the experiment confirmed the GOT, GTP and GGTP abnormalities in a neoplastic disease. We conclude that neuraminidase influences hepatoma Morris growth and its metastatic abilities.