Iron Content and Cellular Proliferation in Thymus and Spleen of Hepatoma 22a Bearing Mice

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Tumor growth is known to induce depression of T-cell immunity, but on the other hand it also induces significant changes of iron metabolism. We hypothesized that iron deficiency may be one of the factors linked to the development of immunodepression observed in tumor growth. Here for the first time we assessed cell proliferation in two lymphoid organs – thymus and spleen, in comparison to iron status of these organs in mice bearing transplantable tumor. General iron status was evaluated on the basis of serum iron levels and liver iron. Thymus weight and cellularity were dramatically decreased from the third week of tumor growth, while spleen weight and cellularity increased. These animals also showed down-regulation of thymocyte proliferation but no decrease of splenocyte proliferation. But, at the same time thymus non-heme iron content was increased and in spleen – decreased. Thus, it may be supposed that spleen and thymus iron contents are sufficient to cover demands for cell proliferation in these organs. There was also no decline of surface transferrin receptor (CD71) expression on thymocytes and catalase activity, which confirm the absence of iron deficiency in the thymus. Parameters of iron metabolism were investigated in the thymus during the growth of transplantable tumor for the first time. Finely, iron deficiency is not the cause of immunological disorders, such as thymic involution and down-regulation of thymocyte proliferation, in mice bearing hepatoma 22a; iron-independent mechanisms may play a role in these processes. This data elucidate one, related to iron, aspect of tumor-induced metabolic influence on the immune system.

Keywords: iron, lymphocyte proliferation, thymic involution, experimental tumors, transferrin receptor, catalase