

Hypothesis on Targeted Modification of Human Individual Radiosensitivity

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According to the last investigations, formation of chromosomal aberrations in cell population is potentially oncogenic event, thus increased individual radiosensitivity in comparison with mean population level value is the factor of radiogenic cancer occurrence and development risk. New candidate genes of human individual radiosensitivity connected with the formation of radiosensitive cell phenotype (BRCA1, BRCA2, XRCC1 etc.) are known for today. The basis of the presented hypothesis is the position that targeted modification of individual radiosensitivity of human organism consists in the formation of normal mean population values of radiation effects reactions. Checkpoint in these processes may be individual radiation reaction on chromosomal level of peripheral blood lymphocytes known to be objective biodosimeters (G2 radiosensitivity assay). This approach radically differs from the traditional "radioresistance increasing", which assumes increased intensity of repair and other radioprotection systems functioning leading to their exhaustion in conditions of long-term irradiation and modification of individual radiation reactions. Pathology status is determined to be deviation from norm (increasing and reduction of parameter value is ment). Thus it is expedient to modify hypo- and hypersensitivity of human cells, tissues and organism in the case of radiation therapy, the strategy of which is directed to maximal lesion (deviation) of tumor cells and protection of surrounded normal tissues. In the case of revealing among healthy population individuals hypersensitive to radiation factor radiosensitivity modification is directed to the leading up of its parameters to normal/mean values.