Procont agent in vivo experiments in consider of microRNAs expression patterns

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Background: *:*

In previous experiment have represented that certain onco- and supressorgenes (Ha-ras, K-ras, c-myc, P53, bcl-2) shown increased expression after carcinogene agent (DMBA) exposition in vital organs. This molecule are functionally connected with microRNAs (let-7a, miR-21, miR-146a) which regulate the oncogen translation.

Aim:

In vivo ivestigate the Procont agent effectmechanism in terms of special microRNAs expression

Methods:

For the experiment we used 5-week-old CBA/CA $H2^k$ inbread mice of both sexes. *We investigated the* chemopreventive effect of Procont comparing with the mices on normal laboratory diet. We fed the Procont agent before 7 days the intraperitoneal injection of DMBA. After one week of the beginning of Procont diet we analized the let7a, miR-21, miR-146a gene expression in vital organs of mice. The gene expression was determined by absolute nucleic acid quantification method in the case of miRNAs, with 4.0 Light Cycler software. Student's t test was performed between the groups and 95 % confidence interval of the differendce was calculated in the case of each organ. Values were expressed as the mean ± 2 SD. The calculation was performed using Statistical Program for Social Science 19.0 (SPSS) software.

Results:

The DMBA exposition caused significant increased expression of all micro RNAs in the vital organs (lungs, kidneys, spleen and liver). The Procont influenced primarily the let-7a expression compared the DMBA treatment especially in the liver, kidneys and lungs. From the investigated oncogenes K-ras radical decreased, which plays important role in the initiation step of chemical carcinogenesis.

Discussion:

In the Procont threated animals we measured significant changes on the level of microRNAs and mRNA. This significant changes shown a repair mechanism of changes induced by chemical carcinogene and prove the Procont previously assumed proapoptotic mechanism. Our further studies goes on evidence the apoptotic effectmechanism extended the NFkappaB and P53 signal transduction system.