Cancer Genetics

AB033. The role in cancer-related DNA damage repair of RNF43

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Background: Cancer is a chronic disease and currently the leading cause of death in Thailand. Functional defect of DNA repair pathway in cells plays role carcinogenesis. Conversely, cancer cells also remain the trace DNA repair pathway to survive under the genotoxic environment, i.e., chemotherapy, and result in resistance to the treatment. Previous reports have shown that DNA repair targeted therapy could improve the conventional treatment. The Ring Finger E3 ubiquitin ligase gene (*RNF43*) is of interest because the function in tumorigenesis related to DNA repair mechanism is ambiguous. Ubiquitin post-translational modification involves in many cellular processes, such as proteasomal degradation and DNA repair regulation. A number of studies indicated contribution of RNF43 toward proteasomal degradation of Frizzled receptor that is related to carcinogenesis. However, there has been no study investigating molecular mechanism of RNF43 on the DNA repair process which could participate in cancer.

Methods: The aim of this study was to investigate the functional role of RNF43 in DNA repair pathways that related to carcinogenesis. DT40 and related knockout strains were used as the model to study the function of RNF43. We first generated complete knockout cell line [DT40-RNF43(-/-)], and the abolition of RNF43 mRNA level was confirmed by qRT-PCR.

Results: Growth curve analysis revealed that DT40-RNF43(-/-) was comparable to the wild-type. The preliminary results of colony formation assay with the genotoxic compounds revealed that RNF43 might have a role in DNA double-strand break (DSB) repair pathways.

Conclusions: Further molecular study on RNF43 binding partner in response to DNA damage repair pathway would facilitate the understanding of RNF43 contribution in DNA-repair related carcinogenesis and provide a new target for personalized medicine in the future.

Keywords: DNA repair pathway; RING finger protein 43 (RNF43); cancer; ubiquitin post-translational modification; personalized medicine

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