Cancer Genetics

AB064. *TRIM29*: a novel gene involved in DNA repair mechanisms

Rakkreat Wikiniyadhanee[1], Tassanee Lerksuthirat[2], Wasana Stitchantrakul[2], Sermsiri Chitphuk[2], Donniphat Dejsuphong[1,2]

[1]Program in Translational Medicine, [2]Research Center, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Bangkok, Thailand

Background: Cytotoxic chemotherapy and/or radiation therapy inducing DNA damage is a part of cancer treatment. Tripartite motif 29 (*TRIM29*) is highly expressed in many malignancies; for example, pancreatic cancer which is notorious resistant to cytotoxic chemotherapy and radiation therapy. *TRIM29* is a member of the TRIM protein family composed of more than 70 members associated with a broad of biological processes. Originally, *TRIM29* gene was described as a candidate gene responsible for ataxia- telangiectasia (AT); however, *TRIM29* was dismissed as AT-causing gene after ataxia- telangiectasia mutated (ATM) was discovered as a causative gene for AT. A few studies about *TRIM29* suggested that it was involved in DNA damage response and high expression of *TRIM29* promoted resistance to ionizing radiation (IR), which induces DNA double strand breaks (DSB). Nevertheless, the functions of *TRIM29* in DNA damage responses and/or DNA repair mechanisms are still unclear.

Methods: To investigate the functions of *TRIM29* in DNA repair mechanisms, wild-type DT40 (WT) and mutant strains have been selected and used as a model. Firstly, the researchers generated the *TRIM29* knockout (TRIM29-/-/-/+).

Results: The growth analysis showed that *TRIM29*-/-/-/+ was comparable to WT. The results of DNA-damaging agent sensitivity using clonogenic survival assays indicated that *TRIM29*-/-/-/+ clones displayed increased sensitivity to topoisomerase 2 inhibitors which induce DNA DSBs repaired by non-homologous end joining (NHEJ) pathway. The *TRIM29*-/-/-/+ clones also exhibited mild sensitivity to camptothecin and cisplatin, indicating that TRIM29 plays a role in DNA DSB repair mechanisms.

Conclusions: Further study of *TRIM29* in response to DNA DSBs may help improve the understanding of functions of *TRIM29*. In the future, *TRIM29* might be a target for anti-cancer drug, leading to improvement of cancer treatment effectiveness.

Keywords: Tripartite motif 29 (*TRIM29*); DNA repair; DNA double strand breaks (DSB); DT40

doi: 10.21037/atm.2017.s064

Cite this abstract as: Wikiniyadhanee R, Lerksuthirat T, Stitchantrakul W, Chitphuk S, Dejsuphong D. *TRIM29*: a novel gene involved in DNA repair mechanisms. Ann Transl Med 2017;5(Suppl 2):AB064. doi: 10.21037/atm.2017.s064

