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Radiation-induced Gastrointestinal Syndrome

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Editorial

In the case of a nuclear catastrophe or radiological terrorism, radiation-induced gastrointestinal syndrome (RIGS) is a limiting factor for therapeutic abdominopelvic radiation and is expected to be a major source of morbidity. We established an in vivo mouse-modeling platform in this study that allows for geographic and temporal modification of putative RIGS targets in mice after whole-abdomen irradiation without the confounding effects of simultaneous haematological syndrome that occurs after whole-body irradiation.

Acute radiation syndrome (ARS) is an acute illness that arises when people are exposed to a high dose of ionising radiation (>0.7 greys) in a short period of time for therapeutic or unanticipated reasons. Each organ's susceptibility to radiation varies greatly in humans, and distinct radiation syndromes occur at different amounts of irradiation. Hematopoietic stem cells are susceptible to severe harm with irradiation doses less than 8 grey, resulting in immunological weakness and hemorrhagic tendency. Within 30 days, patients exposed to irradiation develop illness, bleeding, and possibly death (hematopoietic syndrome; HPS). Villous epithelial cells and crypt stem cells, which are required for colon villi regeneration and epithelial integrity, were found to be growth-inhibited and even killed at doses of more than 10 greys, resulting in epithelial damage, loss of intestinal barrier function, inflammation, and even gut-derived sepsis. Subacute lethality results from severe gastrointestinal injury, which produces bacterial enteritis, malabsorption, diarrhoea, and fluid loss (gastrointestinal syndrome; GIS) [1,2].

Medical countermeasures against HPS include supportive care, infection control, and a bone marrow transplant, which can avoid death. However, because there are presently no viable medicinal treatments for GIS, its clinical use in abdominal radiation is severely limited. GIS is thought to be caused by the loss of epithelial stem cells in the crypts, hence it's critical to find new genes that regulate cell growth and cell death in villous epithelial cells and crypt stem cells.

Our lab was the first to clone N-Myc downstream regulated gene 2 (NDRG2), which belongs to the NDRG family, which is defined by a / hydrolase-fold motif and an esterase/lipase/thioesterase active site serine. NDRG2 is a tumour suppressor gene that has been linked to carcinogenesis, progression, and metastasis. NDRG2 has been linked to a variety of malignancies, including gastrointestinal tumours, breast cancer tumours, lung cancer tumours, neurologic tumours, and so on. The expression level of NDRG2 in tumour tissues was found to be significantly lower when compared to the level in paracarcinoma tissues of normal research groups. TNM stage and lymph node metastases have been shown to be inversely linked with NDRG2 expression levels [3].

Epithelial damage, loss of intestinal barrier function, inflammation, and even gut-derived sepsis result from the GIS, which inhibits and even kills villous epithelial cells and crypt stem cells. Because NDRG2 inhibits cell proliferation and induces apoptosis, it's possible that it plays a role in the progression of GIS. To investigate the involvement of NDRG2 in GIS, we crossed C57BL/6 mice carrying the floxed NDRG2 gene (Ndrg2fl/fl) with C57BL/6 mice carrying the Cre recombinase and villin promoter (Vil/Cre) to obtain Ndrg2G animals that lacked NDRG2 particularly in the intestinal epithelium. Overall, we discovered that $Ndrg2^{\Delta G}$ mice had significantly less symptoms of GIS after 8 grey of total body irradiation (TBI), leading to reduced irradiation-induced cell death and increased crypt cell proliferation. Our findings offer a fresh perspective on the pathophysiology of GIS, and they suggest that blocking NDRG2 could be a promising new target for preventing intestinal radiation injury.

Acute radiation syndrome is caused by high-dose radiation exposure, which can be accidental or therapeutic. It contains two life-threatening syndromes: HPS and GIS. Radiation-induced GIS is caused by the death of villous epithelial cells and crypt stem cells. However, there are no effective interventions or medications that can lower the mortality of radiation-injured individuals or ameliorate intestine damage. As a result, it's critical to identify the genes that control epithelial and stem cell cell proliferation and death. In this study, NDRG2 deficiency in the intestine protected mice against radiation-induced GIS and reduced irradiation-induced mortality by boosting cell proliferation and decreasing cell apoptosis in the intestinal villus and crypts [4].

In several cancer cells, we discovered that NDRG2 plays a role in apoptosis and cell proliferation. We discovered that when A-498 clear cell renal cell carcinoma (CCRCC) cells were infected with NDRG2 recombinant adenovirus, the proliferation rate of the cells fell dramatically, and that NDRG2 may cause the CCRCC cycle to be halted at the G1 phase. Furthermore, knockdown of NDRG2 reduced p53-mediated apoptosis in osteosarcoma cells and lung cancer cells. Similarly, overexpression of NDRG2 in hepatocarcinoma cells increased p53-induced apoptosis (HepG2 and Huh7). NDRG2 has been found to promote apoptosis in CCRCC cells, esophageal squamous-cell carcinoma (ESCC) cells, and MKN28 cells. These findings demonstrated that NDRG2 has an important function in cell proliferation and apoptosis, implying that it may have a role in radiation-induced GIS [5].

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Conflict of Interest

The author shows no conflict of interest towards this manuscript.

References

1. Dörr, Harald, and Viktor Meineke. "Acute radiation syndrome caused by accidental radiation exposure-therapeutic principles." *BMC Med* 9 (2011): 1-6.
2. Koenig, Kristi L., Ronald E. Goans, Richard J. Hatchett, and Fred A. Mettler Jr, et al. "Medical treatment of radiological casualties: current concepts." *Ann Emerg Med* 45 (2005): 643-652.
3. Melotte, Veerle, Xianghu Qu, Maté Ongenaert and Wim Van Criekinge, et al. "The N-myc downstream regulated gene (NDRG) family: Diverse functions, multiple applications." *FASEB J* 24 (2010): 4153-4166.
4. Cao, Wei, Ju-liang Zhang, Da-yun Feng and Xue-wu Liu, et al. "The effect of adenovirus-conjugated NDRG2 on p53-mediated apoptosis of hepatocarcinoma cells through attenuation of nucleotide excision repair capacity." *Biomater* 35 (2014): 993-1003.

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5. Lorentzen, Anders, Rikke H. Lewinsky, Jette Bornholdt and Lotte K. Vogel, et al. "Expression profile of the N-myc Downstream Regulated Gene 2 (NDRG2) in human cancers with focus on breast cancer." *BMC Cancer* 11 (2011): 1-8.

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