Abstract

Colorectal cancer (CRC) is one of the most common types of cancer happening to both men and women in all race and ages, accounting 9% of all cancer incidence worldwide. In 2018 alone, there are 1.8 million new cases and almost 900,000 deaths. Causes of CRC varies into environmental and non-modifiable factors. As for the non-modifiable factors, the most established risk is inherited genetic risk where the cause of it is due to inherited mutation of specific gene called GREM1, a gene from a protein Gremlin-1 (Grem1). Grem1 is an antagonist to a secreted signaling molecule called bone morphogenetic protein (BMP) that work in tandem with its antagonist to regulate development of kidney, lung, limbs, digestive organs, and some other systems. Despite knowing Grem1's role in human organ development, its role in carcinogenesis is yet to be studied in detail. In this project, three main experiments, with HeLa cells used as initial cancer cell model and tissues of mouse intestine for visualization, were set up to understand better Grem1's role in cancer cells. Result showed that Grem1 was able to bind with HeLa cells as transfection was confirmed through western blotting. pSmad 1/5 immunohistochemistry staining showed that Grem1 was in abundance at the base of the mouse colonic crypt but sparse to none found at the villi towards the tip. MTT assay results showed that Grem1 was capable to increase cell viability of HeLa cells, even after damaged by apoptosis inducing agent, H_2O_2.

Keywords: *Colorectal cancer, non-modifiable factors, GREM1, Gremlin-1, antagonist, bone morphogenetic protein (BMP), regulate development, carcinogenesis, HeLa cells, transfection, pSmad 1/5, immunohistochemistry, colonic crypt, MTT assay, cell proliferation, cell viability, H_2O_2.*