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Effect of SNX-2112 on proliferation of esophageal cancer cells via regulation of excision repair cross-complementing 1, epidermal growth factor receptor, and p53 expression

C.Y. Li[1], Y.J. Ren[2] and Y.D. Li[3]

[1]Department of Gastroenterology,
Yantai Liu Huangding Affiliated Hospital of Qingdao University,
Shandong, China
[2]Department of Emergency Medical Center,
Yantai Liu Huangding Affiliated Hospital of Qingdao University,
Shandong, China
[3]Department of Blood Nephrology, Wendeng Municipal Hospital,
Shandong Province, China

Corresponding author: Y.D. Li
E-mail: liyaodong1357@163.com

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ABSTRACT. SNX-2112 is a potential molecular targeted therapeutic drug against esophageal cancer (EC). However, its exact mechanism of action remains to be explained. The aim of this study was to investigate the effect of SNX-2112 on excision repair cross- complementing 1 (ERCC1), epidermal growth factor receptor (EGFR), and p53, to elucidate the mechanism of action of SNX-2112 on EC. Fresh tumor sections were surgically obtained from 65 patients with EC, and the expression of ERCC1, EGFR, and p53 was determined by immunohistochemical staining. Furthermore, the effect of SNX-2112 (0.2 μM) on the proliferation of EC-9706 cells and the expression

of ERCC1, EGFR, and p53 in these cells were analyzed by a cell proliferation assay and western blot, respectively. We observed a significant decrease and increase in ERCC1 ($P = 0.001$) and p53 ($P = 0.043$) expression, respectively, and no significant difference in EGFR ($P = 0.59$) expression, with the TNM stage of EC, which suggested that ERCC1 and p53 could be potential markers for the TNM stage of EC. We also observed a significant increase in ERCC1 expression, and decrease in p53 and EGFR expression, in EC-9706 cells treated with SNX-2112 ($P < 0.05$), indicating the regulation of EC by SNX-2112. Furthermore, SNX-2112 treatment induced a significant decrease in the proliferation of EC-9706, which confirmed the function of SNX-2112. In summary, SNX-2112 inhibits the proliferation of EC cells by regulating the expression of ERCC1, EGFR, and p53.

Key words: Esophageal cancer; SNX-2112; ERCC1; EGFR; p53; EC-9706 cells

