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NOX4-Driven ROS Formation Regulates Proliferation and Apoptosis of Gastric Cancer Cells through the GLI1 Pathway Chao-Tao Tang^{a,1}, Xiao-Lu Lin^{b,1}, Shan Wu^{a,1}, Qian Liang^a, Li Yang^a, Yun-Jie Gao^a, Zhi-Zheng Ge^a

^a Division of Gastroenterology and Hepatology, Key Laboratory of Gastroenterology and Hepatology, Ministry of Health, Renji Hospital, School of Medicine, Shanghai Jiao Tong University, Shanghai Institute of Digestive Disease, Shanghai 200001, China

^b Department of Digestive Endoscopy, Provincial Clinic Medical College, Fujian Medical University, Fujian Provincial Hospital, Fuzhou 350001, China

¹These authors have contributed equally to this work Correspondence to: Zhi-Zheng Ge, email: zhizhengge@aliyun.com

Keyword: NADPH oxidase 4, ROS, GLI1, gastric cancer, proliferation, apoptosis

Abstract: NADPH Oxidase 4 (*NOX4*), a member of the NOX family, has emerged as a significant source of reactive oxygen species, playing an important role in tumor cell proliferation, apoptosis, and other physiological processes. However, the potential function of *NOX4* in gastric cancer (GC) cell proliferation is yet unknown. The aim of this study was to illustrate whether *NOX4* plays a role in regulating gastric cancer cell growth. First, the clinical information from 90 patients was utilized to explore the clinical value of NOX4 as a predictive tool for tumor size and prognosis. Results showed that *NOX4* expression was correlated with tumor size and prognosis. In vitro assays confirmed that knockdown of *NOX4* expression blocked cell proliferation and the expression of Cyclin D1, BAX, and so on. Interestingly, *NOX4* promoted cell proliferation via activation of the GLI1 pathway. *GLI1*, a well-known transcription factor in the Hedgehog signaling pathway, was overexpressed to test whether NOX4 activates downstream signaling via GLI1. Overexpression Download English Version:

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