In Cervical Cancer Cells, Increased O-Glcnacylation Enhances the IGF-1 Receptor/Phosphatidyi Inositol-3 Kinase/Akt Pathway

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Commentary

Cervical squamous cell carcinoma (CSCC) is a kind of cancer that a ects millions of women and their families around the world. CSCC is caused by the human papillomavirus (HPV), and squamous intraepithelial lesions (SILs) caused by high-risk HPV (HR-HPV) infection are considered precancerous lesions. In a prior study, HPV-infected cancer cells were able to survive by combating lipid peroxidation. According to recent study, ferroptosis kills cancer cells by an iron-dependent lipid peroxidation mechanism, and it has been recommended as a potential method for female cancer therapy [1]. e role of ferroptosis in SIL progression into CSCC was examined in this study. Ferroptosis was detected in SIL, but anti-ferroptosis was reported in CSCC. Our ndings also revealed that persistent ferroptosis resulted