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CORRECTION

Correction: The nuclear hypoxia-regulated NLUCAT1 long non-coding RNA contributes to an aggressive phenotype in lung adenocarcinoma through regulation of oxidative stress

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The original version of this article unfortunately contained a mistake. The following correction has therefore been made in the original: The term "LUCAT1" was missing in the abstract. The corrected abstract is given below. The original article has been corrected.

Abstract

Lung cancer is the leading cause of cancer death worldwide, with poor prognosis and a high rate of recurrence despite early surgical removal. Hypoxic regions within tumors represent sources of aggressiveness and resistance to therapy. Although long non-coding RNAs (lncRNAs) are increasingly recognized as major gene expression regulators, their regulation and function following hypoxic stress are still largely unexplored. Combining profiling studies on early-stage lung adenocarcinoma (LUAD) biopsies and on A549 LUAD cell lines cultured in normoxic or hypoxic conditions, we identified a subset of lncRNAs that are both correlated with the hypoxic status of tumors and regulated by hypoxia in vitro. We focused on a new transcript, Nuclear LUCAT1 (NLUCAT1), which is strongly upregulated by hypoxia in vitro and correlated with hypoxic markers and poor prognosis in LUADs. Full molecular characterization showed that NLUCAT1 is a large nuclear transcript composed of six exons and mainly regulated by NF-κB and NRF2 transcription factors. CRISPR-Cas9-mediated invalidation of NLUCAT1 revealed a decrease in proliferative and invasive properties, an increase in oxidative stress and a higher sensitivity to cisplatin-induced apoptosis. Transcriptome analysis of NLUCAT1-deficient cells showed repressed genes within the antioxidant and/or cisplatin-response networks. We demonstrated that the concomitant knockdown of four of these genes products, GPX2, GLRX, ALDH3A1, and PDK4, significantly increased ROS-dependent caspase activation, thus partially mimicking the consequences of NLUCAT1 inactivation in LUAD cells. Overall, we demonstrate that NLUCAT1 contributes to an aggressive phenotype in early-stage hypoxic tumors, suggesting it may represent a new potential therapeutic target in LUADs.