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# Unraveling the influence of LncRNA in gastric cancer pathogenesis: a comprehensive review focus on signaling pathways interplay

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## Abstract

Gastric cancers (GCs) are among the most common and fatal malignancies in the world. Despite our increasing understanding of the molecular mechanisms underlying GC, further biomarkers are still needed for more in-depth examination, focused prognosis, and treatment. GC is one among the long non-coding RNAs, or lncRNAs, that have emerged as key regulators of the pathophysiology of cancer. This comprehensive review focuses on the diverse functions of long noncoding RNAs (lncRNAs) in the development of GC and their interactions with important intracellular signaling pathways. lncRNAs affect GC-related carcinogenic signaling cascades including pathways for EGFR, PI3K/AKT/mTOR, p53, Wnt/ $\beta$ -catenin, JAK/STAT, Hedgehog, NF- $\kappa$ B, and hypoxia-inducible factor. Dysregulated long non-coding RNA (lncRNA) expression has been associated with multiple characteristics of cancer, such as extended growth, apoptosis resistance, enhanced invasion and metastasis, angiogenesis, and therapy resistance. For instance, lncRNAs such as HOTAIR, MALAT1, and H19 promote the development of GC via altering these pathways. Beyond their main roles, GC lncRNAs exhibit potential as diagnostic and prognostic biomarkers. The overview discusses CRISPR/Cas9 genome-modifying methods, antisense oligonucleotides, small molecules, and RNA interference as potential therapeutic approaches to regulate the expression of long noncoding RNAs (lncRNAs). An in-depth discussion of the intricate functions that lncRNAs play in the development of the majority of stomach malignancies is provided in this review. It provides the groundwork for future translational research in lncRNA-based whole processes toward GC by highlighting their carcinogenic effects, regulatory roles in significant signaling cascades, and practical scientific uses as biomarkers and therapeutic targets. © The Author(s), under exclusive licence to Springer Science+Business Media, LLC, part of Springer Nature 2024.

## Author keywords

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