

Response to “Lncrna GAS5 Modulates the Progression of Glioma Through Repressing miR-135b-5p and Upregulating APC” [Letter]

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Dear editor

The article entitled “LncRNA GAS5 Modulates the Progression of Glioma Through Repressing miR-135b-5p and Upregulating APC” present a compelling study that elucidates the regulatory role of GAS5 and its interaction with miR-135b-5p in the context of glioma metastasis, invasion, and proliferation.¹ This research provides valuable insights into the molecular mechanisms underlying glioma pathogenesis and proposes potential therapeutic targets for intervention.

The study employs a range of robust methodologies, including Western blotting, RT-qPCR, CCK-8 assays, transwell migration, wound healing assays, and double luciferase experiments. These techniques collectively validate the findings and highlight the intricate regulatory network involving GAS5, miR-135b-5p, and APC in glioma cells. The APC gene, a tumor suppressor, is located on human chromosome 5q21-22.^{2,3} The discovery that GAS5 is downregulated in glioma tissues and that its upregulation suppresses glioma cell invasion, proliferation, and metastasis is particularly noteworthy. The elucidation of GAS5’s function in sponging miR-135b-5p which is a conserved transcript among mammals and is located in the gene locus of 1q32.1 in humans,⁴ thereby regulating APC expression, adds a significant layer of understanding to the molecular interplay in glioma progression.

While the findings are promising, there are several avenues for future research that could further enhance the understanding and therapeutic potential of this regulatory axis. One important area to explore is the long-term effects of GAS5 modulation in in vivo glioma models. Such studies would provide a more comprehensive understanding of GAS5’s therapeutic potential and its impact on glioma progression over extended periods. Additionally, investigating the possible side effects or unintended consequences of GAS5 upregulation in normal brain tissues would be crucial to ensure the safety and efficacy of potential therapeutic strategies.

Further research should also focus on the detailed signaling pathways downstream of APC that are influenced by GAS5 and miR-135b-5p. Understanding these pathways could reveal additional molecular targets and offer a broader perspective on how to effectively disrupt glioma progression. Moreover, comparative studies involving other non-coding RNAs and their interactions with miR-135b-5p and APC could provide a more holistic view of the regulatory networks at play.

In conclusion, the article presents significant advancements in understanding the molecular mechanisms of glioma progression and identifies GAS5 as a potential therapeutic target. The authors’ findings are well-supported by comprehensive experimental evidence and offer a promising direction for future research and therapeutic development. I look forward to seeing further investigations that build on these findings and advance the field of glioma research.

Disclosure

The authors report no conflicts of interest in this communication.

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