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- Large-scale survey, animal models and computational modeling identify histological neurodegenerative biomarkers for traumatic optic neuropathy
- Wet-adhesive and antibacterial PAH-TPP coacervates with self-mineralizing capability for cranial flap fixation
- Evaluating burnout syndrome among healthcare workers: Prevalence and risk factors
- Visualization of subthalamic nucleus on susceptibility weighted imaging and the verification of accuracy by microelectrode recording
- Uncovering the effects of ATBC plasticizers on intracerebral hemorrhage outcomes based on network toxicology and in vitro and in vivo experimental validation
- miR-210 Regulates Autophagy Through the AMPK/mTOR Signaling Pathway, Reduces Neuronal Cell Death and Inflammatory Responses, and Enhances Functional Recovery Following Cerebral Hemorrhage in Mice
- Patient-centered insights into virtual reality rehabilitation for stroke: a systematic review and qualitative meta-synthesis
- Effects of NGF-chitosan on alleviating secondary degeneration and repairing primary degeneration after expanded partial optic nerve transection

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In a preclinical experimental study Wang et al. aims to explore the neuroprotective role of miR-210 in a Murine Model of Intracerebral Hemorrhage, focusing on its effect on autophagy, apoptosis, and inflammation, mediated via the AMPK/mTOR signaling pathway.

 Strengths:

Addresses a clinically relevant and understudied area (ICH lacks effective neuroprotective treatments).

Uses both in vivo (mouse ICH model) and in vitro (HT22 neuronal cells) experiments, enhancing translational relevance.

Incorporates molecular, cellular, behavioral, and histological endpoints, enabling mechanistic insight and functional correlation.

Applies pharmacological modulators (AICAR and rapamycin) to probe the pathway.

⚠ Limitations:

No long-term follow-up: Outcomes are only measured up to 72 hours; longer-term neurological recovery is not addressed.

Lack of dose–response or time–response curves for miR-210 or inhibitors weakens the causal interpretation.

No human data or validation in human tissues, limiting immediate translational impact.

2. Methodological Rigor Good use of multiple experimental groups: sham, ICH, ICH + LV-miR-210, etc.

 Multiple techniques applied: qPCR, Western blot, immunofluorescence, behavioral tests (Morris Water Maze), TUNEL, brain edema assessment.

⚠ Randomization and blinding are not clearly described, potentially introducing bias.

⚠ Small sample sizes in some groups (n=6) may compromise statistical power, especially in behavioral assays.

3. Mechanistic Insights The study demonstrates that miR-210 overexpression reduces inflammation and neuronal apoptosis by downregulating autophagy via inhibition of AMPK/mTOR activation.

Rescue experiments with AICAR (AMPK activator) and rapamycin (mTOR inhibitor) support the mechanistic link.

 The dual validation in vitro and in vivo strengthens the proposed pathway.

⚠ However, the specific targets of miR-210 upstream of AMPK/mTOR were not identified, leaving a mechanistic gap.

4. Interpretation and Novelty The paper highlights a novel role for miR-210 in regulating autophagy following ICH.

 Suggests therapeutic potential of miR-210 modulation in acute brain injury.

⚠ The results confirm rather than substantially extend previous findings on miR-210’s protective role in ischemia and other CNS injuries.

⚠ Lacks comparative analysis with other neuroprotective miRNAs or pathways in ICH.

5. Writing and Presentation ⚠ The manuscript would benefit from better structure and clarity,

particularly in the abstract, where experimental steps are listed in detail without synthesis.

Figures and data presentation were not assessed in this review but are key to full appraisal.

 Conclusion This is a well-conceived preclinical mechanistic study that provides valuable insight into the neuroprotective role of miR-210 in cerebral hemorrhage via the AMPK/mTOR/autophagy axis. While the data support the proposed mechanism and therapeutic potential, the study would be strengthened by:

Clearer methodological transparency (randomization, blinding),

Inclusion of long-term outcomes,

Functional target identification upstream of AMPK/mTOR,

Comparative or combinatory analyses with other neuroprotective interventions.

 Recommendation Publishable with minor revisions. The study lays solid groundwork for future translational research but requires additional validation in more clinically relevant models. [1)].

1)
Wang Y, Jiang L, Tian JJ, Zhu LL, Dai HJ, Guo C, Zhou LY, Wang L, Lu Y, Zhang Y. miR-210 Regulates Autophagy Through the AMPK/mTOR Signaling Pathway, Reduces Neuronal Cell Death and Inflammatory Responses, and Enhances Functional Recovery Following Cerebral Hemorrhage in Mice. Neurochem Res. 2025 Jun 5;50(3):180. doi: 10.1007/s11064-025-04434-7. PMID: 40471451.