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Sialyltransferase ST3GAL6 silencing reduces α2,3-sialylated glycans to regulate autophagy by decreasing HSPB8-BAG3 in the brain with hepatic encephalopathy

Key words: Hepatic encephalopathy; Hyperammonemia; Autophagy; ST3GAL6; HSPB8

Research Summary

This research focuses on the regulatory function of sialyltransferase ST3GAL6 in autophagy processes within the brain affected by hepatic encephalopathy（HE）. The specific aspects covered include the following:

- **Increased ST3GAL6 expression in brain with HE**
- **Silencing ST3GAL6 Decreases α2,3-Sialylation**
- **ST3GAL6 regulates autophagy through HSPB8-BAG3 complex**

Innovation points

- **ST3GAL6 as a Mediator: It may regulate the upregulation of α2,3-sialylated glycans during hyperammonemia, thereby influencing autophagy levels.**

- **Silencing ST3GAL6 decreases α2,3-sialylated glycans, thereby downregulating HSPB8-BAG3 to modulate autophagy**

- **Manipulating ST3GAL6 to regulate autophagy: offering a new perspective for intervention in HE**

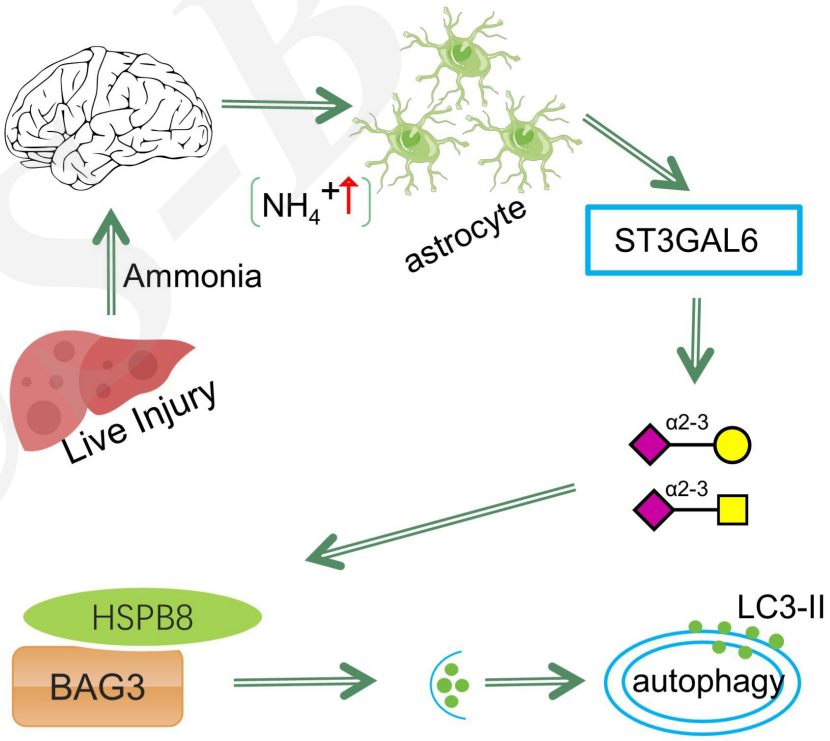

Figure 6k

Innovation points

A series of comprehensive figures were generated to summarize the influence of ST3GAL6 on autophagy in HE.

Figure 1 | Effect of ammonia on ST3GAL6 and α2, 3-sialylated glycans strocytes.

Figure 2 | Astrocyte autophagy levels upregulated by ammonia.

Figure 3 | Silencing ST3GAL6 expression attenuates autophagy levels in astrocytes.

Figure 4 | Effect of α2-3 sialylated glycans on autophagy.

Figure 5 | HSPB8-BAG3 complex upregulated by ammonia.

Figure 6 | Autophagy modulated by ST3GAL6 via HSPB8-BAG3 complex.