NECROSTATIN-1 PROMOTES HEMATOMA RESOLUTION AND ATTENUATES CEREBRAL EDEMA FOLLOWING INTRACEREBRAL HEMORRHAGE

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Intracerebral Hemorrhage (ICH)

- Most prevalent type of hemorrhagic stroke
- Caused by the rupture of a diseased blood vessel
- Around 50% of all deaths occur within the first 48 hours
- 50-60% mortality within the first year
- Only 20% regain functional independence

Tumor necrosis factor α (TNF-α)

- Pro-inflammatory cytokine
- Increases neurological injury after ICH
- Activates RIP Kinase (RIPK) to induce necroptosis, a novel form of cell death

Necrostatin-1 (Nec-1)

- Novel allosteric inhibitor of RIP1 kinase
- Limits neurovascular damage in culture models of hemorrhagic injury
- Improve outcome after in pre-clinical TBI and cerebral ischemia models

Hypothesis

Necrostatin-1 reduces neurological injury after ICH

Necrostatin-1 Decreases Hematoma Size After ICH

Figure 1. Necrostatin-1 attenuates hematoma size after ICH. Coronal sections from vehicle, inactive analog, or Necrostatin-1 treated animals at 72hr post-ICH (a). Mice treated with either Necrostatin-1 or its inactive analog showed a significant reduction in brain hemoglobin content, a validated measure of hematoma size (b). Data are expressed as mean ± SEM. (*p<0.05, **p<0.001).
**Conclusions**

- Necrostatin-1 reduces hematoma size, brain edema, and neurological deficits after ICH

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