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BACKGROUND

- Aluminum nanoparticles (**Al NPs**) are widely used in consumer and industrial products and are linked to **neurotoxicity** and **cognitive dysfunction**.
- The **hippocampus**, essential for learning and memory, is highly vulnerable to disruptions in **energy metabolism** and **synaptic signaling**.
- **Glucose transporter-1 (GLUT-1)** supports cerebral glucose uptake and is critical for hippocampal energy supply.
- **Ca²⁺/calmodulin-dependent protein kinase II- α (CaMKII- α)** is essential for **synaptic plasticity** and memory formation.
- Both **GLUT-1** and **CaMKII- α** are implicated in early **Alzheimer's disease pathology**.
- Their temporal involvement in **Al NP-induced memory impairment** remains unknown.

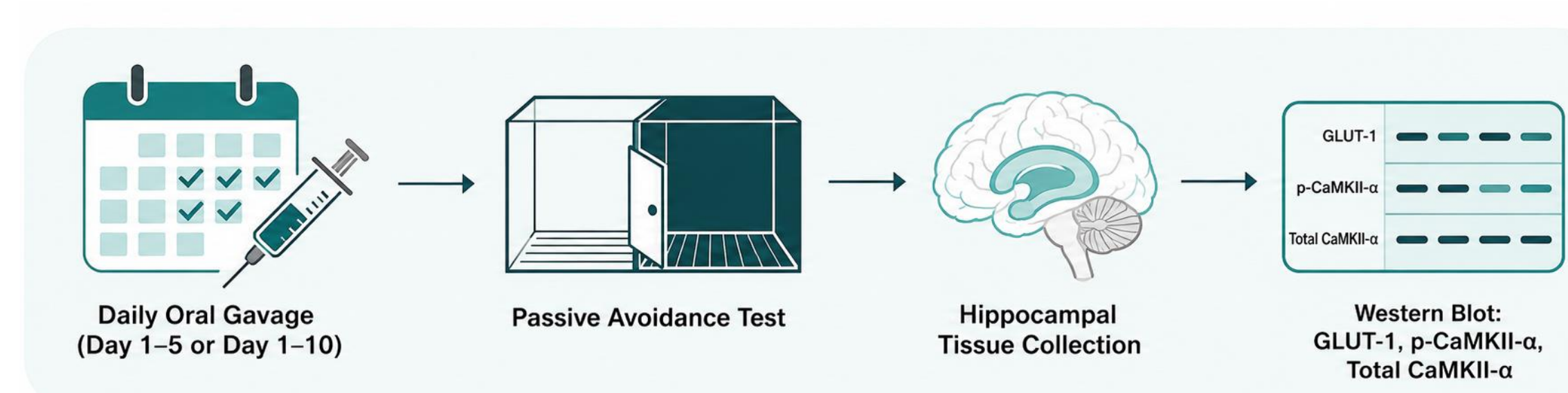
OBJECTIVE

- To examine whether short-term oral Al NP exposure causes **time-dependent** changes in hippocampal **GLUT-1** and **CaMKII- α** expression.
- To determine how these molecular changes relate to the **emergence of associative memory deficits** measured by passive avoidance.

METHODS

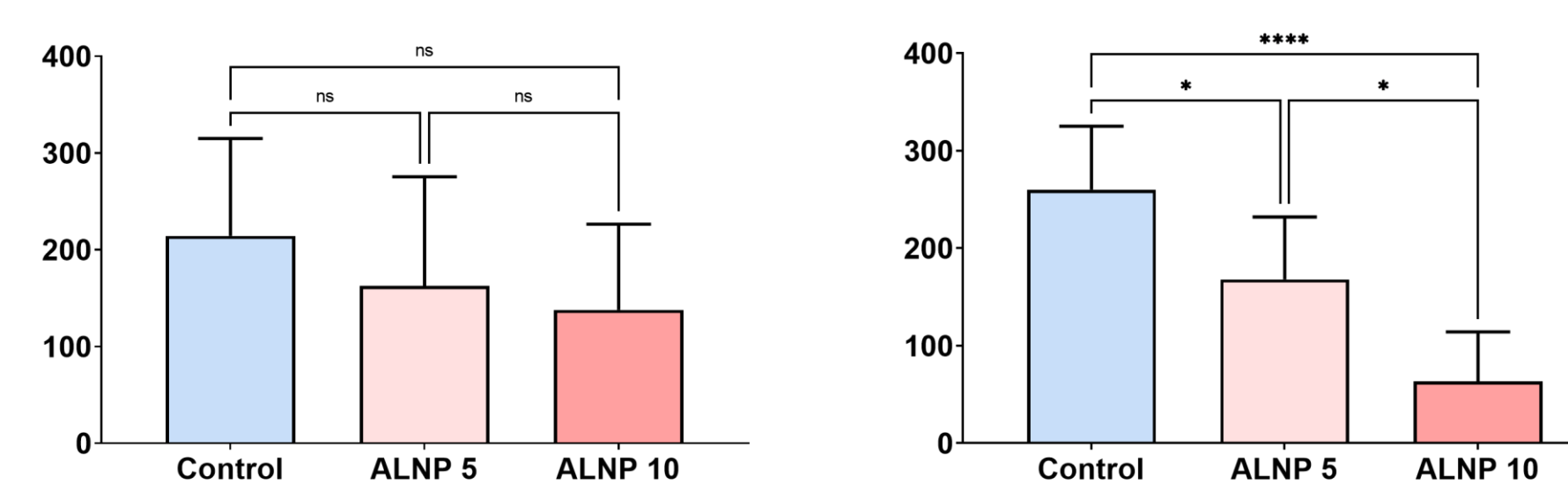
Animals: Male mice, n = 8 per group
Groups: Vehicle (Control) | Al NPs (10 mg/kg, oral)
Duration: 5-day or 10-day exposure
Behavioural Test:
Passive Avoidance Test: Measured step-through latency as an index of hippocampal-dependent associative memory (tested 24 h after last exposure).
Molecular Analysis (n = 5 per group):
Western blotting of hippocampal tissue
 Targets: **GLUT-1**, **Total CaMKII- α** , **phosphorylated CaMKII- α (p-CaMKII- α)**
 Normalized to **GAPDH**
Statistics: One-way ANOVA with Tukey's post hoc test, p < 0.05 considered significant.

FIGURE 1 – Experimental Design Flowchart



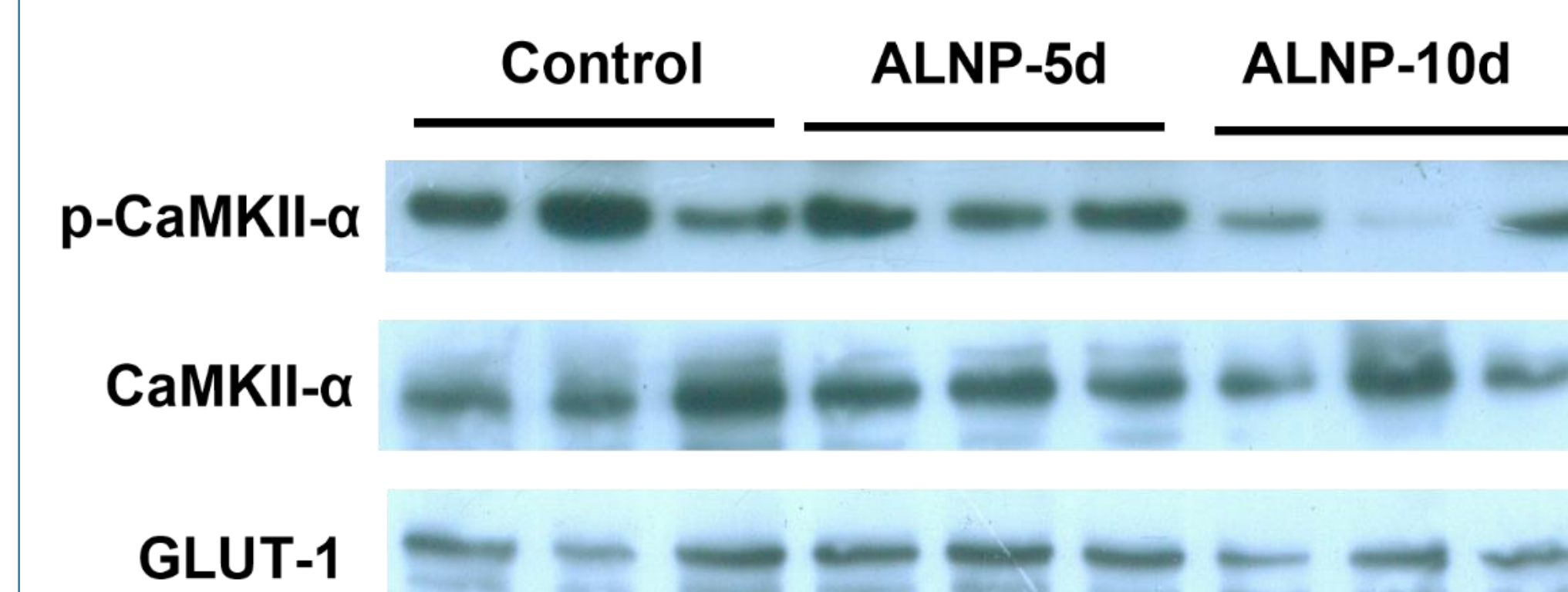
RESULTS

FIGURE 2 – Passive Avoidance Step-Through Latency (Day 5 & Day 10)



- **5-day exposure:** No significant difference in latency between Control and Al NP groups. Associative memory is **preserved**.
- **10-day exposure:** Marked reduction in step-through latency in the Al NP group (p < 0.01). **Associative memory impairment emerges only after sustained exposure.**

FIGURE 3 – Western Blot Quantification: GLUT-1, p-CaMKII- α , Total CaMKII- α (Day 5 & Day 10)



- **5-day exposure:** No significant molecular changes.
- **10-day exposure:** Significant reduction in **GLUT-1** and **p-CaMKII- α** .
- Mild decrease in **total CaMKII- α** .

CONCLUSION

- Although Recognition memory is impaired early (Day 5), passive avoidance memory needs more prolong time.
- Memory deficit in passive avoidance task following sustained Al NP exposure (Day 10) is associated with hippocampal **GLUT-1** and **CaMKII- α** signaling (while 5-day exposure had no significant effect).
- These molecular dysregulation coincides with the emergence of **associative memory impairment**.
- This **temporal pattern** links prolonged nano-aluminum exposure to disrupted **energy metabolism** and **synaptic kinase signaling**.
- Findings provide a **mechanistic framework** for Al NP contribution to **Alzheimer-related cognitive vulnerability**.

Contact

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