Alzheimer’s disease (AD) is the most common form of dementia in the elderly. The pathological hallmarks of the disease are characterized by the presence of senile plaques in form of extracellular β-amyloid (Aβ) deposition, accompanied by increases in inflammatory responses and impairment in learning and memory abilities. For a long time, a clear association between AD and hypertension has been reported, and memory abilities decreases inflammatory responses and impairment in learning and memory.

In order to investigate the potential link among hypertension and AD, we focused on a particular model of hypertension, obtained by transverse aortic coarctation (TAC) and showing a significant hippocampal and cortical Aβ deposition within four weeks. Thus, we explored molecular markers and behavioral traits associated to AD in genetically modified mice for RAGE. In particular, the process of microglia activation and the expression of typical pro- and anti-inflammatory genes as well as learning and memory abilities in the Morris Water Maze test, were assessed.

**EXPERIMENTAL DESIGN**

**BEHAVIORAL ANALYSIS**
- Morris Water Maze

**TREATMENT WITH RAGE INHIBITOR**
- TAC-induced cognitive impairment showed in Morris Water Maze test, were prevented by treatment with RAGE inhibitor. (*** p < 0.01 vs each other quadrant)

**CONCLUSIONS**
Overall, our findings point to a central role of hypertension-driven brain damage in the pathogenic mechanisms leading to amyloid pathology, confirming a role played by RAGE in the onset and progression of AD. Furthermore, the TAC model used here displays behavioral alterations having face validity with psychiatric symptoms of AD disease and thus appears as a valuable tool to explore the neural mechanisms underlying this pathology.

No potential conflict of interest

**REFERENCES**