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Paroxetine is Pro-inflammatory in Human Brain Microvascular Endothelial Cells

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BACKGROUND

- Paroxetine is an antidepressant that is widely used.
- Paroxetine possesses anticholinergic properties which may contribute to cognitive decline in older adults.
- Surprisingly, the mechanisms of the cognitive decline of anticholinergic drugs in older adults remains elusive.

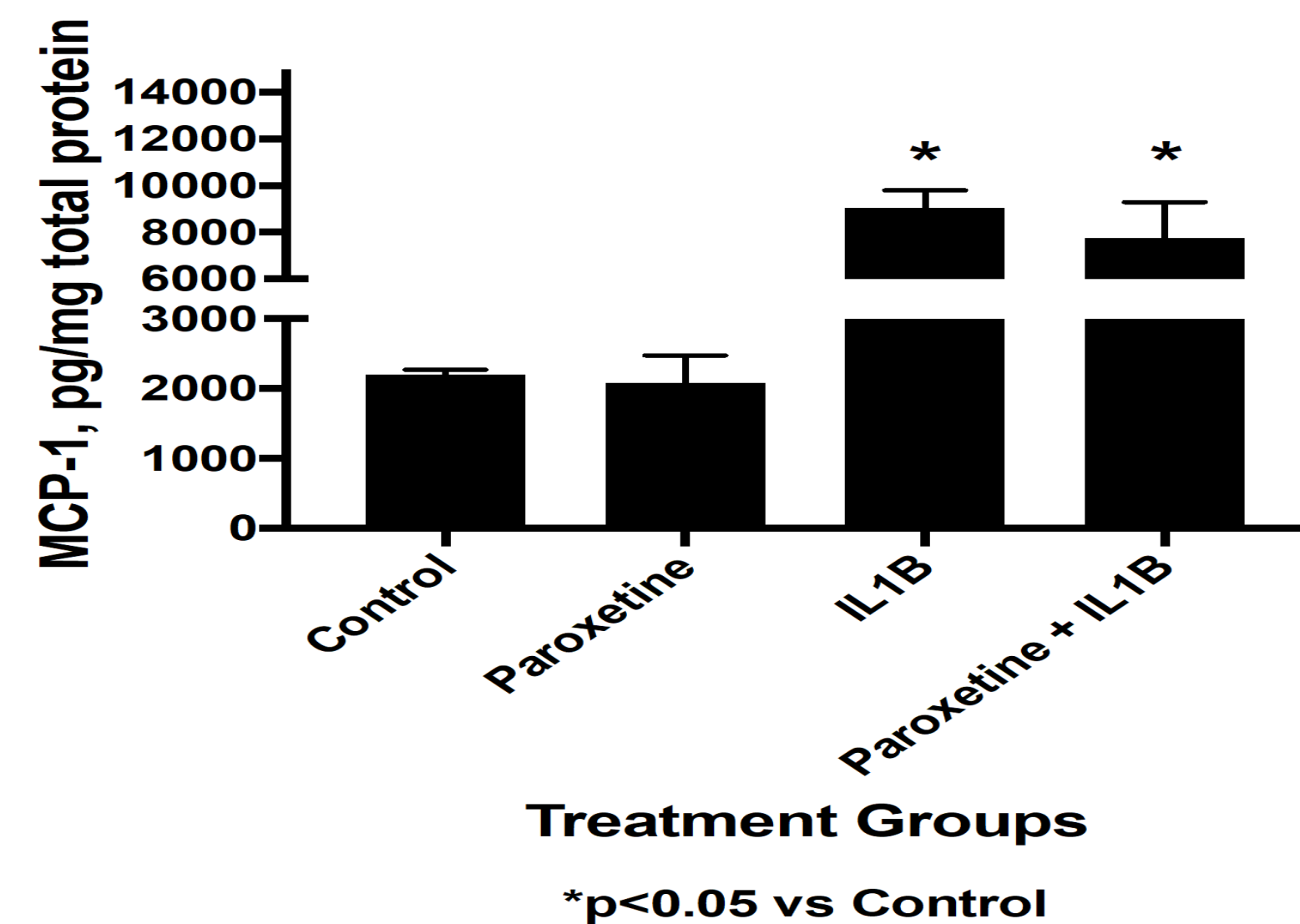
RESEARCH QUESTION OR HYPOTHESIS

- A hypothesis is that a loss of cholinergic signaling is pro-inflammatory in the brain.
- We designed experiments to determine if Paroxetine is pro-inflammatory in Normal Human Brain Microvascular Endothelial Cells (NHBMECs).

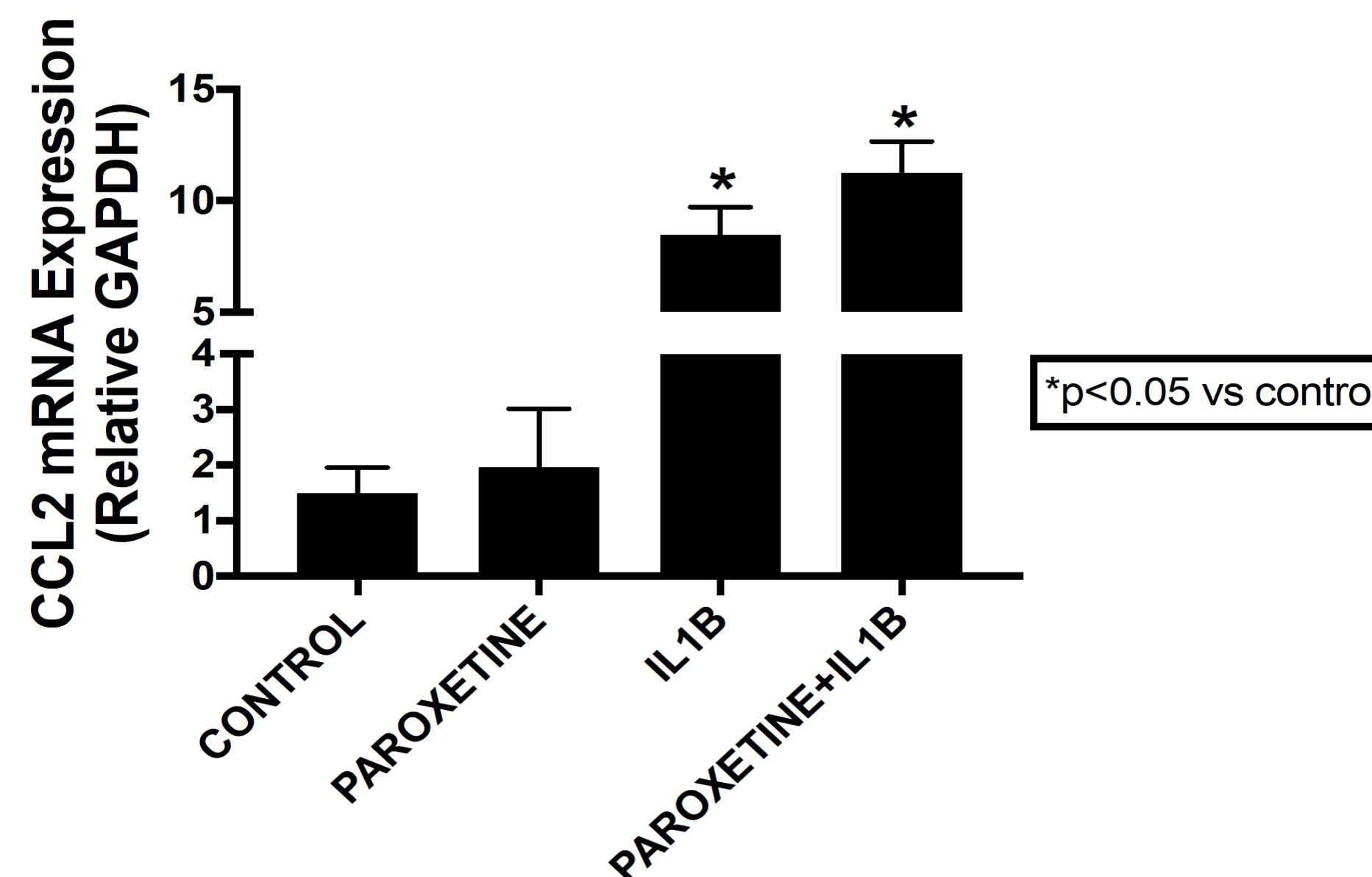
METHOD

- NHBMECs were grown at 5% CO₂, 37C and 95% relative humidity until 90% confluent.
- NHBMECs were treated with the RNA free water as control, Paroxetine as 1.67x 10⁻⁴ mM/mol, IL1B 2ng/ml for 24 hours.
- RNA was isolated and converted to cDNA via RNA-cDNA conversion kit.
 - Gene expression determined via RT-PCR.
- Statistical analysis was performed via ANOVA and post-hoc Tukey's with a significant p-value < 0.05.

Inflammatory Biomarker production in Normal Human Brain Microvascular Endothelial Cells.



Gene expression studies.



RESULTS

- A statistically significantly increased production of inflammatory biomarker, MCP1 was found.
- NHBMECs when exposed to Paroxetine for 24 hours demonstrated elevated expression of CCL2 and CXCL5.
- NHBMECs exposed to Paroxetine + IL1B demonstrated synergistic elevations in CCL2 and CXCL5.

CONCLUSION

- Our result suggests that Paroxetine may have pro-inflammatory properties within this subset of human brain cells.
- Our preliminary data suggests the possibility of pathological pro-inflammatory mechanisms associated with Paroxetine.

Further evaluation of different populations of normal human brain cells are warranted.

