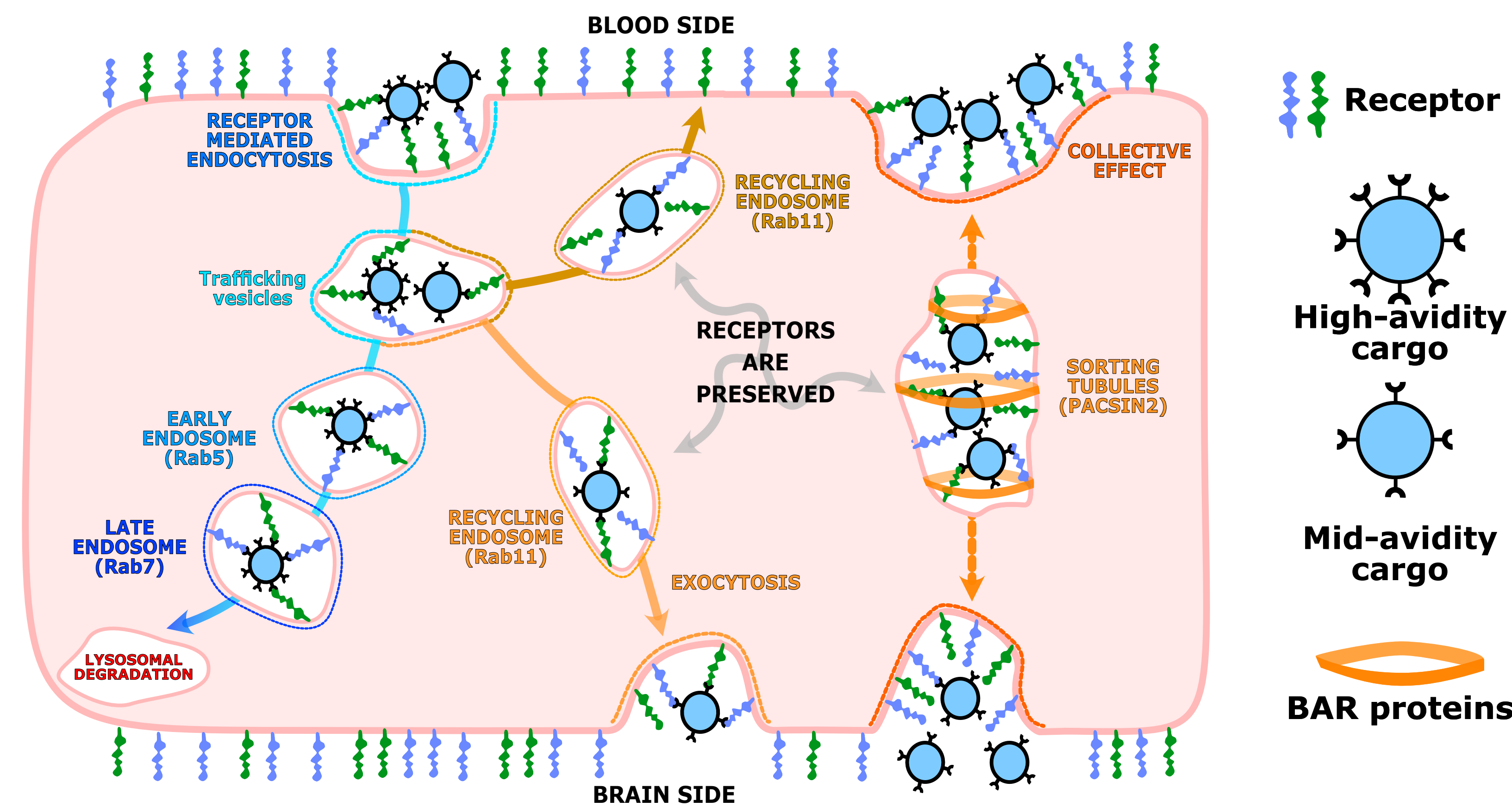


Unveiling the biological mechanisms underlying misfolded Amyloid- β clearance at the Blood-Brain Barrier

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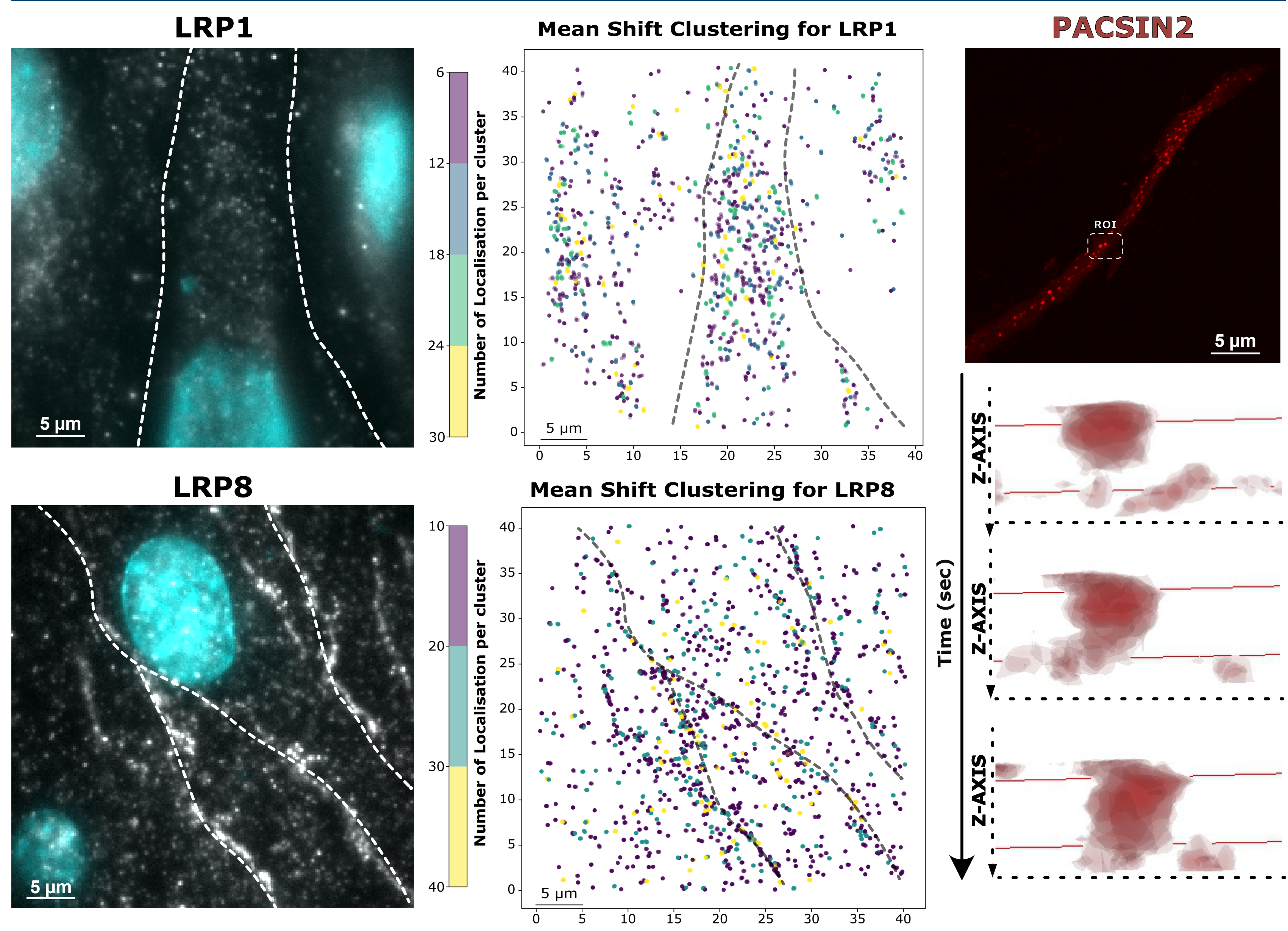
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Abstract

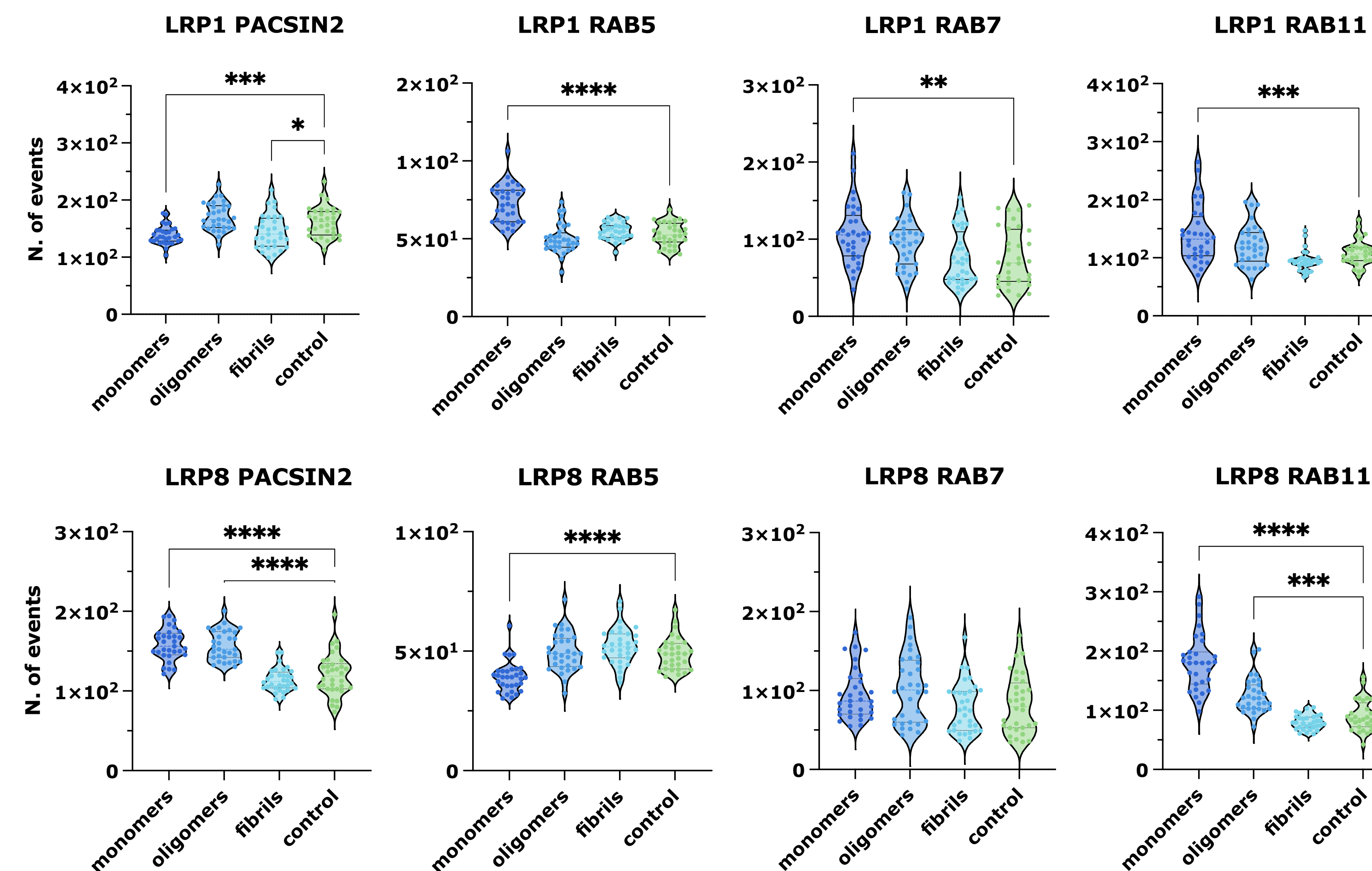


Misfolded Amyloid- β ($A\beta$) accumulation in the brain is a key feature of Alzheimer's disease, primarily linked to impaired clearance rather than overproduction. The blood-brain barrier (BBB) controls $A\beta$ transport via low-density lipoprotein receptors (LDLR), which sort cargo based on avidity. High-avidity molecules are retained and degraded, while mid-avidity cargoes are transcytosed through tubular vesicles stabilized by PACSIN2. Our research focuses on unveiling the cellular mechanisms triggered by different $A\beta$ assemblies at the BBB to identify potential therapeutic targets for Alzheimer's disease.

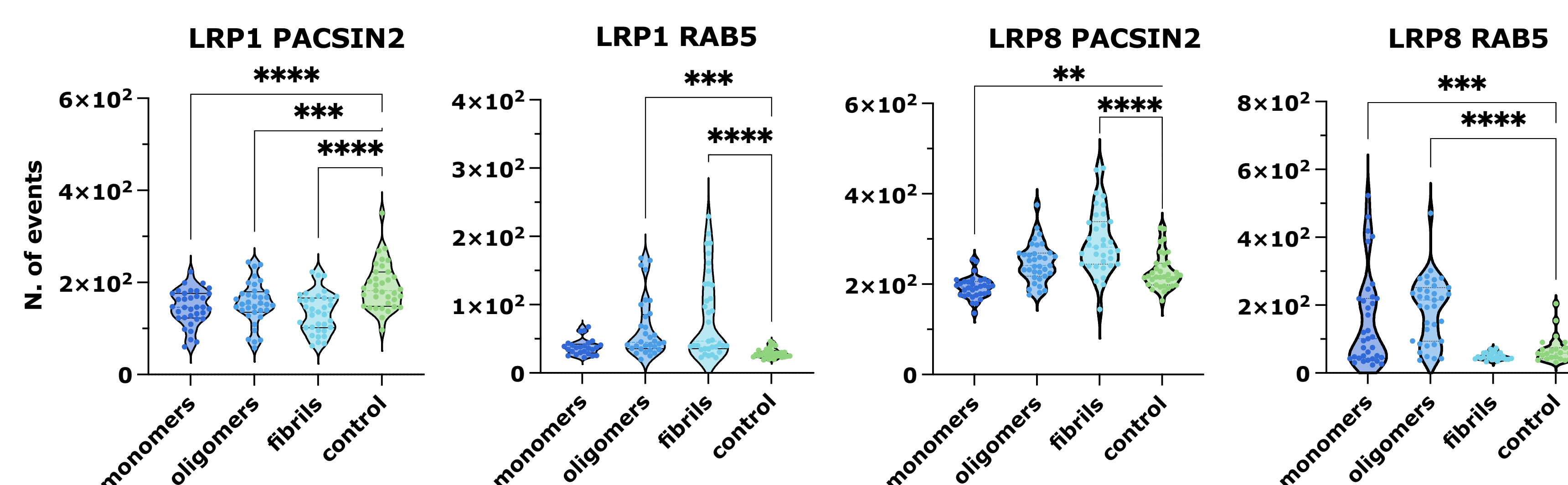
LRP1 and LRP8 clustering analysis by STORM and PACSIN2 tubulation by confocal



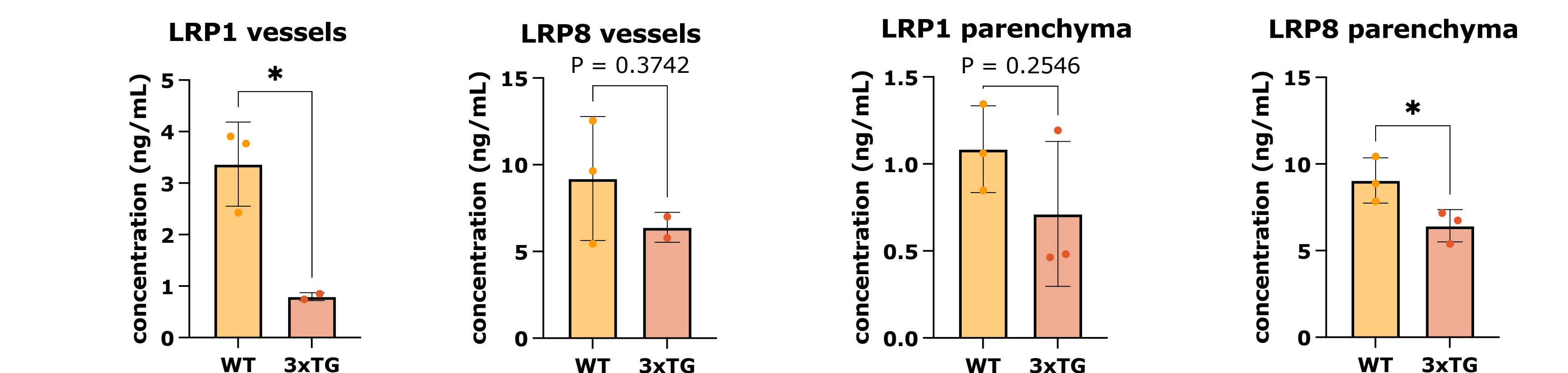
Colocalization analysis of LRP1 and LRP8 following A β 42 treatment



Colocalization analysis of LRP1 and LRP8 following A β 40 treatment



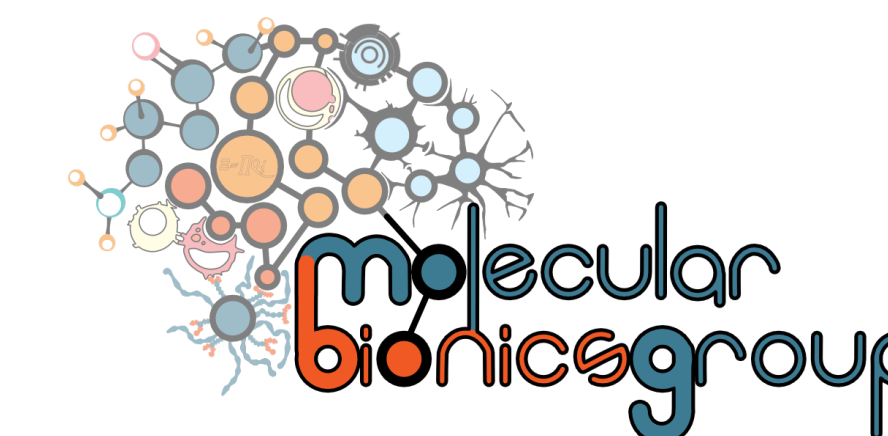
LRP1 and LRP8 protein expression in brain vasculature and parenchyma of 3xTg mice



Acknowledgement and contact



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