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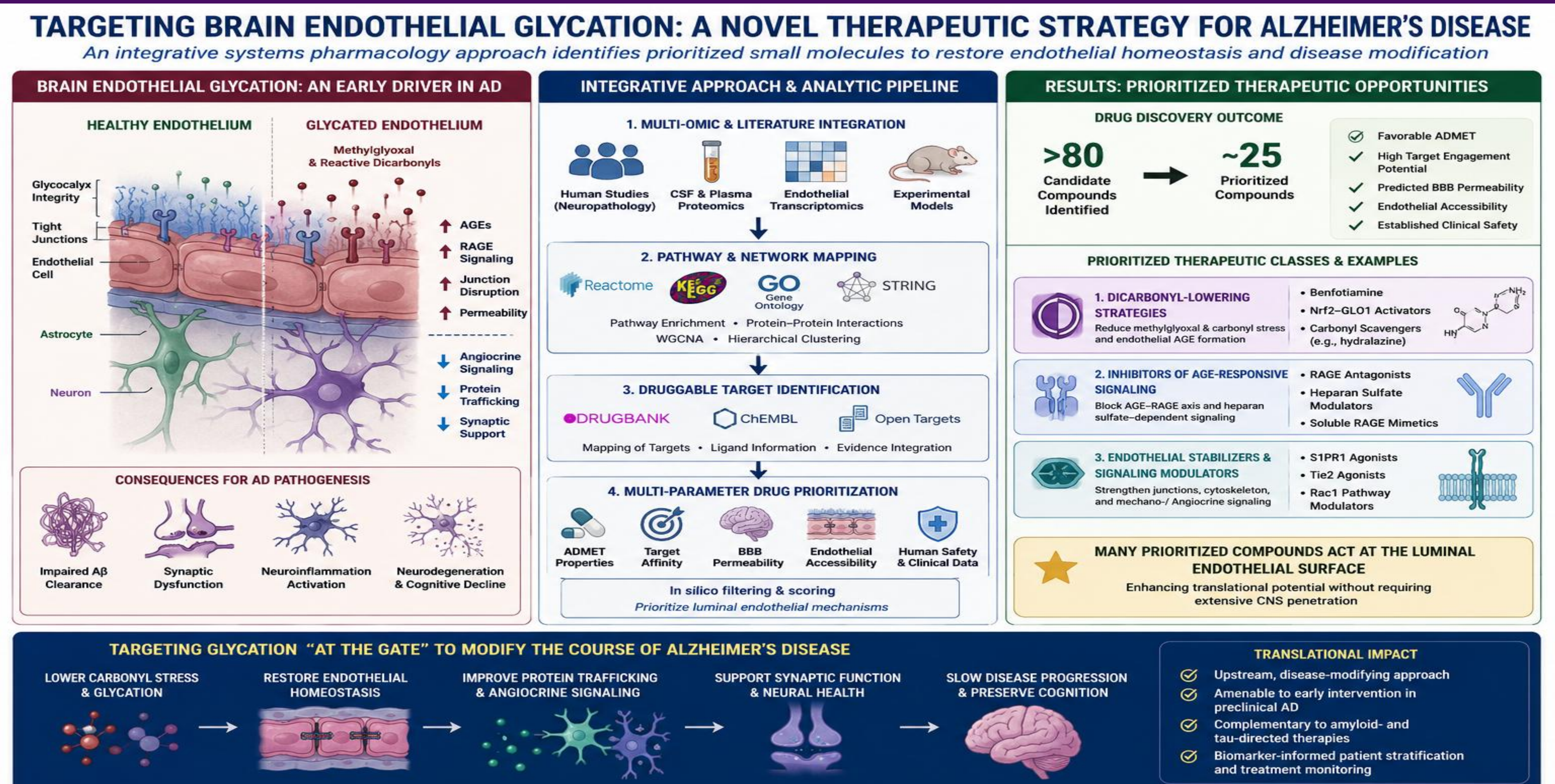
INTRODUCTION

Non-enzymatic glycation and carbonyl stress emerge as important mechanisms that disrupt the brain endothelial glycocalyx in preclinical AD. We here investigate brain endothelial glycation as a novel therapeutic target in AD.

METHODS

Network-level analyses were supported by WGCN and hierarchical clustering. Candidate drug targets were mapped using DrugBank, ChEMBL, and Open Targets platforms. Small molecules were prioritized through a multi-parameter filtering strategy incorporating predicted ADMET properties, target affinity, and blood-brain barrier permeability.

RESULTS



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