

Social Exposome and Epigenetic Age Acceleration in Dementia

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INTRODUCTION

RESULTS

CONCLUSIONS

The social exposome encompasses the cumulative burden of socially patterned exposures across the life course¹. These exposures are increasingly recognized as major contributors to dementia risk, yet the biological mechanisms through which they influence biological aging remain incompletely understood. Epigenetic age acceleration (EAA), derived from DNA methylation clocks, provides a molecular marker of biological aging that may capture the long-term impact of adverse social experiences². However, evidence integrating multidimensional social exposome measures with epigenetic aging in dementia, particularly in Latin American populations, remains limited. To investigate the associations between multidimensional social exposome measures and epigenetic age acceleration across multiple DNA methylation clocks in individuals with dementia and cognitively unimpaired controls from five Latin American countries.

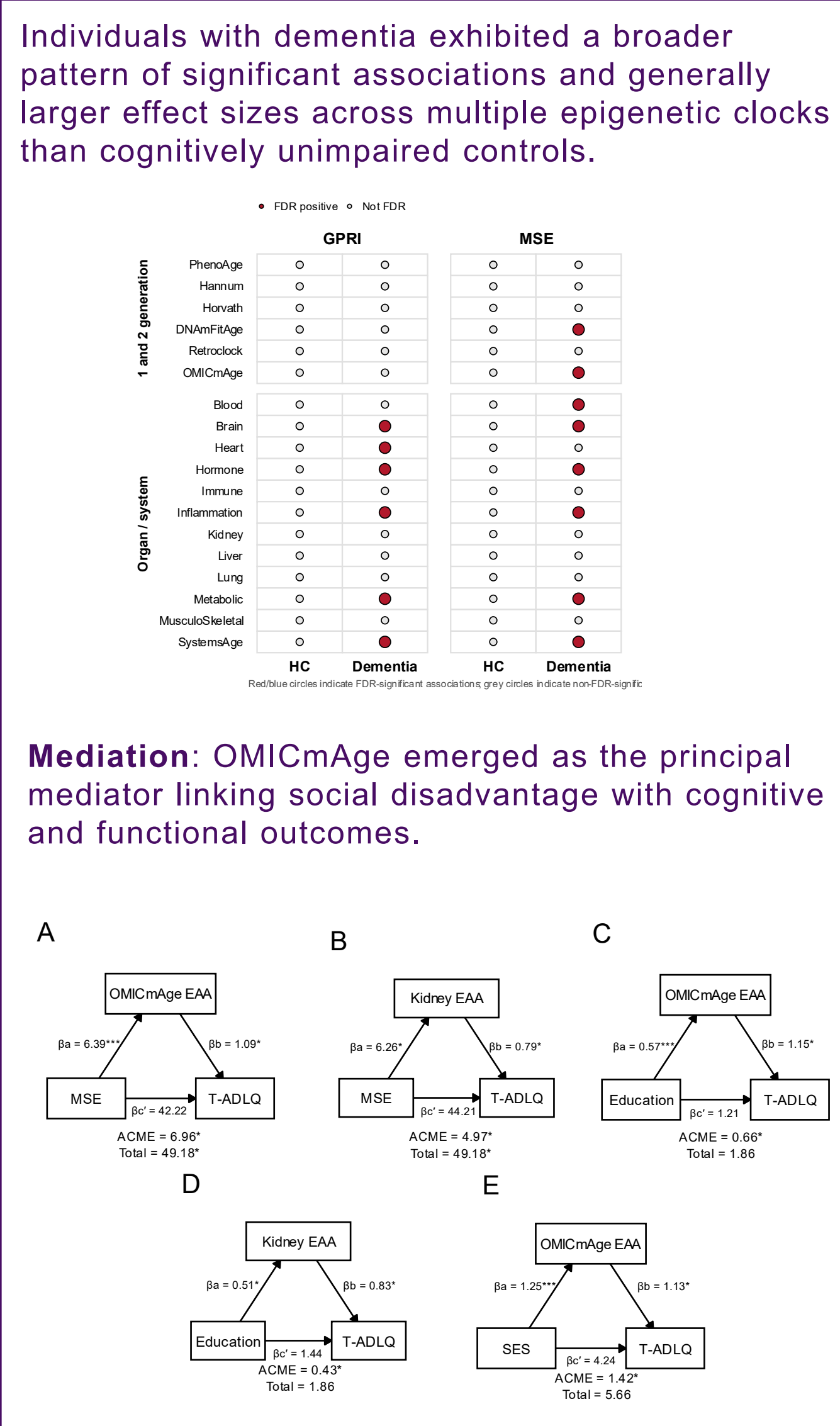
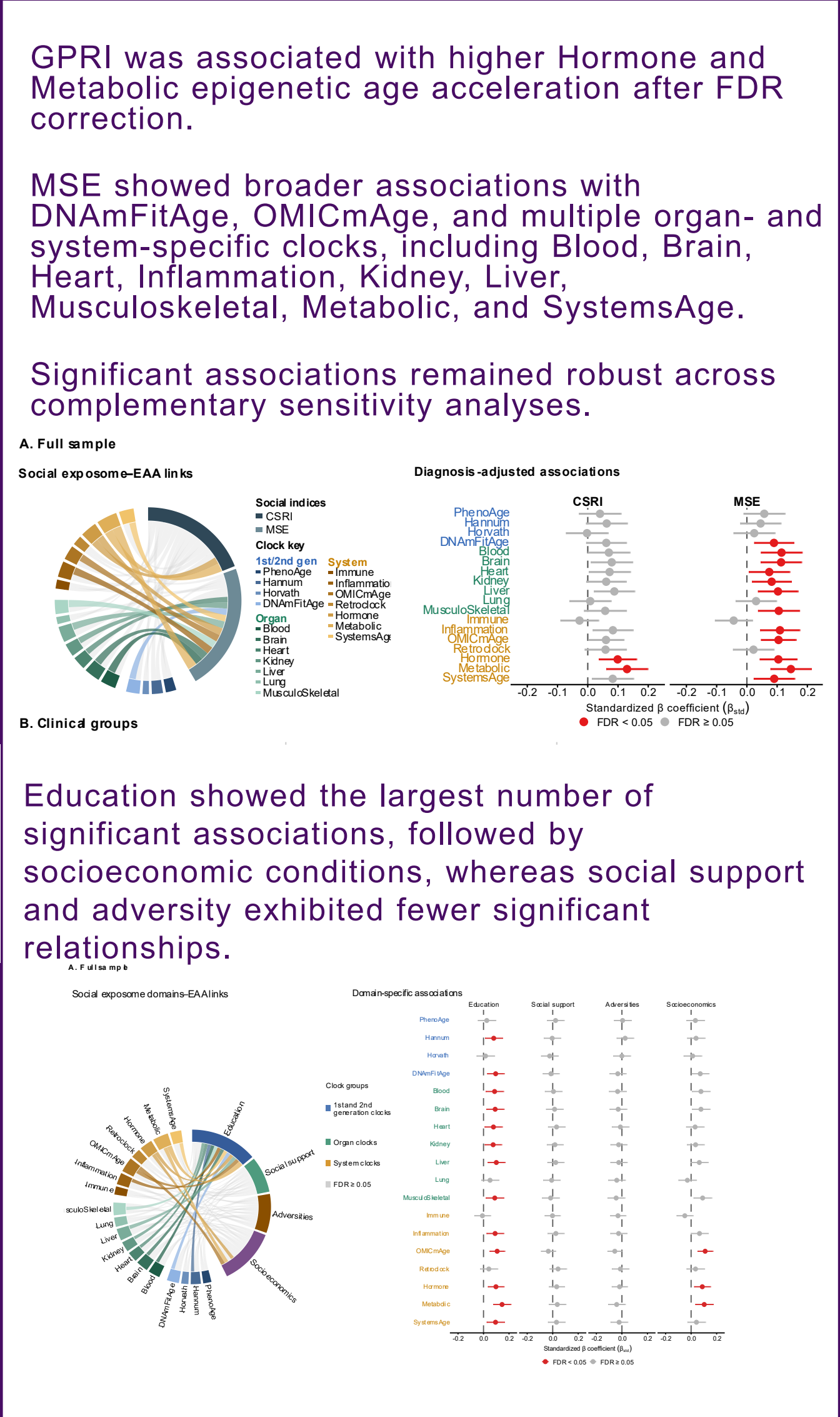
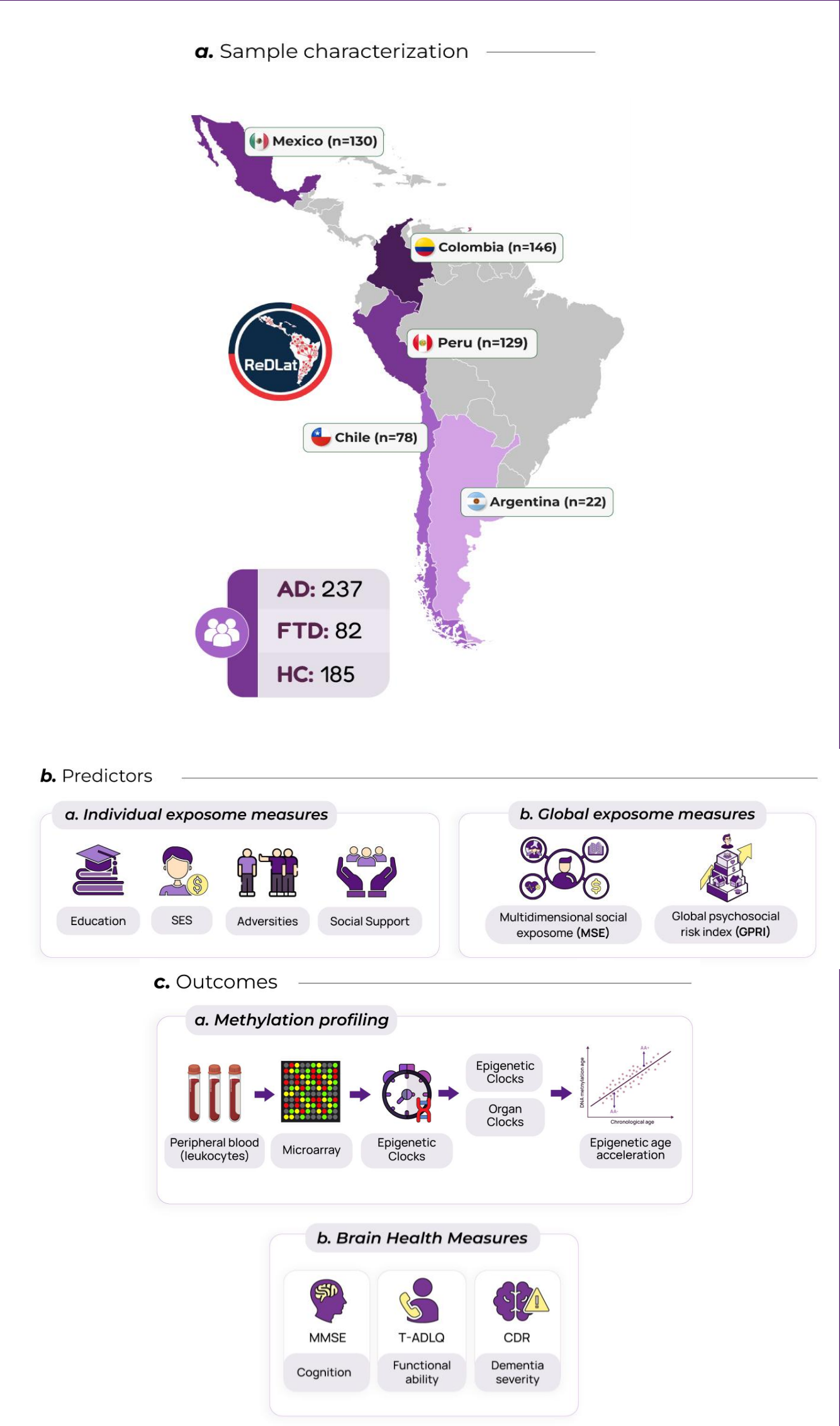
METHODS

-participants: 854 ReDLat participants from five Latin American countries.

Social exposome: Global psychosocial risk index (GPRI) and Multidimensional Social Exposome (MSE).

Epigenetic aging: Blood DNA methylation (Illumina EPIC); EAA estimated across multiple DNA methylation clocks.

Analysis: Association analyses • Mediation analyses • Interaction analyses • Stratified analyses • Sensitivity analyses



CONCLUSIONS

-Greater social disadvantage was associated with higher EAA across multiple biological systems.

-MSE showed broader and more consistent associations than GPRI.

-Individuals with dementia exhibited broader and stronger associations between the social exposome and EAA than cognitively unimpaired controls.

-These findings support the social exposome as a determinant of biological aging and identify EAA as a potential biomarker linking social adversity to dementia.

REFERENCES

1. Genon S, Ibanez A, Tahmasian M, Eickhoff SB. *Linking the exposome to the brain-behaviour phenotype*. Nature Reviews Neuroscience. 2026.

2. Migeot J, et al. *Social exposome and brain health outcomes of dementia across Latin America*. Nature Communications. 2025.

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