

Investigating TBI in Older Adults Using Age-Specific Finite Element Brain Models

Bashar L. Elkhatab, Ahmed A. Alshareef

I. INTRODUCTION

Traumatic brain injury (TBI) is a major public health concern, with approximately 1.7 million cases occurring annually in the United States [1]. The risk and severity of TBI are influenced by several factors ranging from impact conditions to individual anthropometry. TBI disproportionately affects older adults, who account for most TBI-related hospitalisations and deaths in the United States, mainly due to falls [2]. However, the underlying mechanisms contributing to these age-related differences in risk and outcomes remain poorly understood. With healthy aging, the brain undergoes pronounced structural and mechanical changes, including cerebral atrophy, ventricular enlargement, and reduced tissue stiffness (Fig. 1A) [3]. These alterations can potentially change the vulnerability of the brain to tissue deformations from head impact, yet most finite element (FE) brain models do not include age-specific material properties or structure for older adults. Understanding the effect of these anthropometric or mechanical factors can help stratify older adult injury risk based on deformation response.

This study aims to (1) investigate the age-dependent biomechanical response of the brain to injurious head impacts in older adults using subject-specific 3D finite element (FE) models, and (2) identify features of the older adult brain that increase injury risk.

II. METHODS

This study examines the effect of age-related cerebral atrophy and tissue softening on brain deformation under injurious head loading, integrating age-specific structural and mechanical information from a longitudinal magnetic resonance imaging (MRI) dataset.

Subject-Specific Older Adult Models

Longitudinal T1-weighted MRI data from 29 older adults (15M/14F, 68–96 years) in the Baltimore Longitudinal Study of Aging (BLSA) were used to build FE brain models that include structural changes with age (Fig. 1B) [4]. These 29 subjects, from a total of >1000 subjects, represent the highest rates of brain atrophy (Fig. 1C). Each subject had an initial baseline scan and at least three follow-up scans 1–3 years apart. The images were segmented to yield eight brain regions, and models were constructed for each scan session of the longitudinal data for a total of 82 subject-specific models.

Models were simulated with and without the material softening. The models without material softening used sex-specific older adult properties derived from a prior study that generated age- and sex-stratified biomechanical templates [5]. For models with material softening, age-dependent material properties were derived from a

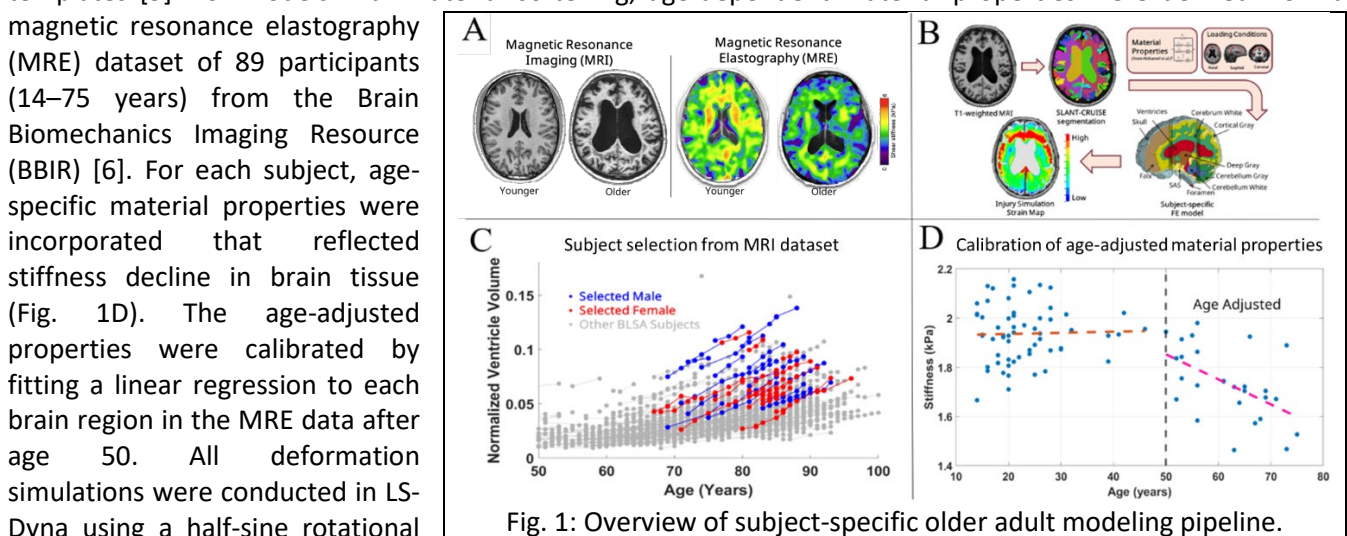


Fig. 1: Overview of subject-specific older adult modeling pipeline.

pulse (peak velocity 40 rad/s, acceleration 5000 rad/s²) similar to high severity falls onto hard surfaces from a standing height, applied across axial, sagittal, and coronal directions, totaling 164 simulations [7].

Processing and Analysis

Maximum principal strain (MPS) was calculated for each simulation and the 95th percentile MPS (MPS95) value across the brain was used as the primary response variable. The MPS95 was compared across longitudinal scans and between models with and without softening to identify which features influence the deformation response.

III. INITIAL FINDINGS

Across longitudinal scans for the subject-specific simulations, MPS95 decreased with increased normalised ventricle volume, indicating lower global brain deformation with greater atrophy (Fig. 2A). The models with material softening had increased MPS95 relative to the same subjects without material softening (mean increase of 0.07 MPS95; $p < 0.05$), indicating a larger effect of material softening on deformation and vulnerability of the older adult brain. The increase due to softening, however, was not uniform even for subjects that had the same decrease in material properties (e.g., they were the same age and sex). This interaction was investigated by plotting the average MPS95 across the longitudinal scans for each subject (Fig. 2B). Although both atrophy and brain softening are correlated to age, the deformation response is not very well correlated ($R^2=0.011$). The amount of atrophy using the normalised ventricle volume as a measure, however, correlated well with age ($R^2=0.625$). This correlation shows that models with higher degrees of atrophy showed greater sensitivity to softening, suggesting that geometric and material aging processes interact to shape overall deformation behaviour and brain vulnerability. The results were consistent for the axial, sagittal, and coronal loading conditions with sagittal loading having the highest variation among the models.

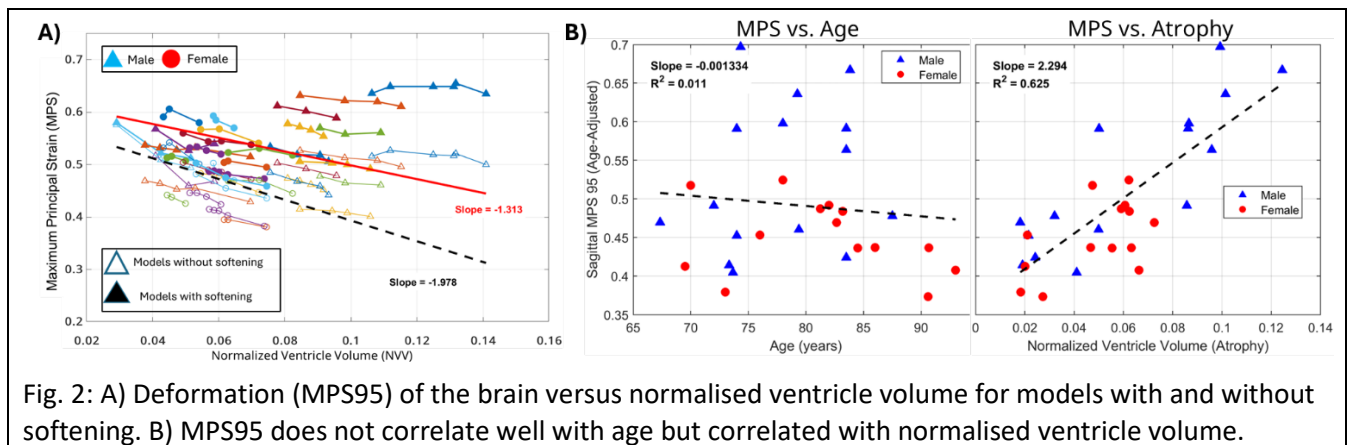


Fig. 2: A) Deformation (MPS95) of the brain versus normalised ventricle volume for models with and without softening. B) MPS95 does not correlate well with age but correlated with normalised ventricle volume.

IV. DISCUSSION

The results demonstrate that structural and mechanical changes with aging affect the deformation response of the brain, with competing influences on brain deformation. While atrophy reduces strain magnitude slightly, brain softening amplifies deformation, leading to greater variability in biomechanical response among older adults. Integrating MRI- and MRE-derived datasets enables a data-driven modeling framework that captures these coupled effects and provides a more accurate representation of the aging brain. This study is limited by the number of subjects from a single dataset, as well as having only one kinematic loading condition. Ongoing work will conduct a regional analysis to identify focal patterns of vulnerability that increase TBI risk with age. It will also include a larger cohort with more loading conditions. to identify risk factors for TBI in older adults.

V. REFERENCES

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