Age-adaptive Fracture and Toughening Behaviors of Cortical Bone at the Microscale

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Summary

Bone possesses different toughening mechanisms to resist fracture at different ages. The study aims to investigate the fracture behaviors of cortical bone during aging and its correlation with microstructural alterations, with an emphasis on mineralized collagen fibril organization. Bone micropillars were manufactured from the femur of young, adult, and aged mice, and in-situ compression testing was conducted to quantify the fracture behavior. SEM, TEM, and electron diffraction were adopted to assess the micro-/nanoscale structural alterations and toughening mechanisms. The results showed that the young and adult groups were prone to shearing, while the aged group preferred splitting. Surprisingly, the young and aged groups demonstrated comparable toughness to the adult group in their dominant fracture modes. The dominant toughening mechanisms shifted from fibril pull-out and bridging to fibril kinking and crack deflection with aging. The study highlights the crucial roles of mineralized collagen fibril arrangement in adaptive toughing mechanisms during aging.

Introduction

Aging increases bone fracture susceptibility. Bone might develop different toughening mechanisms to resist fracture at different ages, potentially by altering the multi-scale organization of bone components. However, the relationship between fracture resistance and multi-scale structural alterations during aging remains unclear. The study aims to investigate the fracture behaviors of cortical bone at the microscale during aging and its correlation with microstructural alterations, with an emphasis on mineralized collagen fibrils' organization.

Methods

BALB/c male mice with different ages were used and categorized into three groups: young (3-month), adult (8-month) and aged (18-month). The right femurs were harvested. Bone micropillars were manufactured with focused ion beam (FIB) from the femur of young, adult, and aged mice, and in-situ compression testing was conducted to quantify the fracture behavior. Scanning electron microscopy, transmission electron microscopy, and electron diffraction were adopted to assess the micro-/nanoscale structural alterations and toughening mechanisms.

Results and Discussion

The results showed that the dominant fracture modes of bone micropillars varied with age. The young and adult groups were prone to shearing, while the aged group preferred splitting. The adult group exhibited superior compressive properties and progressive post-yield behavior regardless of the fracture mode. Surprisingly, the young and aged groups demonstrated comparable toughness to the adult group in their dominant fracture modes, with 49 ± 18 MJ/m3 (young) vs. 53 ± 23 MJ/m3 (adult) in shearing, and 49 ± 21 MJ/m3 (aged) vs. 41 ± 16 MJ/m3 (adult) in splitting, respectively. Moreover, the dominant toughening mechanisms shifted from fibril pull-out and bridging to fibril kinking and crack deflection with aging.

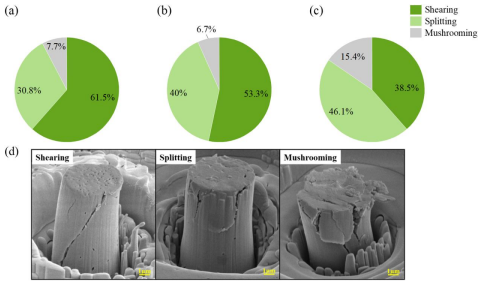

Figure 1: Proportions of three different fracture modes of bone micropillars across the (a) young, (b) adult, and (c) aged groups. (d) shows typical SEM images of three fracture modes: shearing, splitting, and mushrooming..

Conclusions

The present study demonstrated that both the young and aged cortical bones are capable of generating significant strain-hardening behavior under compression with a toughness comparable to that of the adult bones at the microscale, which can be attributed to the age-related changes in the microstructure and associated toughening mechanisms. The study highlights the crucial roles of the arrangement of mineralized collagen fibrils in the toughening behaviors of cortical bone. Investigating the fracture and toughening behaviors of bone at the microscale is essential for bridging gaps in the field of bone biomechanics. The findings here can help researchers gain a better understanding of how and where age affects clinical fracture risk and facilitate the development of targeted therapies.

Acknowledgments

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References

[1] Liu F et al. (2025). *ACS Biomater Sci Eng*, **11**: 1038-50.
[2] Depalle B et al. (2021). *Acta Biomater*, **120**: 194– 202.
[3] Liu F et al. (2022). *Acta Biomater*, **152**: 345– 354.