ROLE OF PROα2(I) COLLAGEN CHAINS AND COLLAGEN CROSSLINKING IN THORACIC AORTIC BIOMECHANICAL INTEGRITY DURING AGING USING THE *OIM* MOUSE MODEL

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ABSTRACT

Type I collagen, normally a heterotrimeric molecule of two α1(I) and one α2(I) collagen chains, is the major contributor to aortic strength (F_{max}) and stiffness (IEM). We used the *oim* (osteogenesis imperfecta mouse) model, which synthesizes only homotrimeric type I collagen molecules [$\alpha1(I)_3$], to assess aortic integrity when α2(I) chains are absent from the type I collagen molecule and the effect of age-associated changes. We evaluated *oim*, heterozygote, and wildtype thoracic aortas at 3, 8, and 18-months of age for circumferential F_{max}/IEM, histology, aortic extracellular matrix (ECM) expression, collagen content, and collagen crosslinking. *Oim* thoracic aortas exhibited reduced F_{max} and IEM, collagen staining, and collagen content at each age class. Aortic ECM expression exhibited age-associated reductions in expression; no genotype-associated differences were shown. Despite increased collagen crosslinking in *oim* aortas, homotrimeric fibrils remained inherently weaker than heterotrimeric fibrils. All genotypes exhibited age-associated increases in F_{max}/IEM and collagen content; yet, collagen crosslinking did not correlate with increasing age, suggesting alternative mechanisms are responsible for the age-associated increases in aortic integrity.