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In vitro, interaction of homotrimers with heterotrimers of type I collagen SEJIN HAN, WOLFGANG LOSERT, University of Maryland, SERGEY LEIKIN, NIH — The dominant mutations in type I collagen cause a group of diseases, often termed collagen, or connective tissue, diseases: for example, Osteogenesis Imperfecta (OI) characterized by bone fragility and skeletal deformity. The mechanism in which collagen mutations affect on the diseases is still unknown. To understand the fibril assembly and their interactions might provide a key to approaching the cause of the collagen diseases. This study demonstrates that the self-assembly, termed fibrillogenesis, of type I collagen homozygous mutations revealed substantial differences in the kinetics with the absence of lag time and in the morphology of 3D fibril network structure. The heterotrimers (normal) and homotrimers (mutant) in mixtures were segregated within the same fibrils during fibrillogenesis, in correspondence between confocal microscopy and thermodynamic measurements. The efficiency for self-assembly of the homotrimers into fibrils was markedly reduced, while that of the heterotrimers was not affected by the presence of homotrimers with no change in solubility.

Sejin Han
University of Maryland

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