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Interruptions between the triple helix peptides can promote the formation of amyloid-like fibrils[1] AVANISH PARMAR, EILEEN HWANG, BARBARA BRODSKY, UMDNJ-Robert Wood Johnson Medical School — It has been reported that collagen can initiate or accelerate the formation of amyloid fibrils. Non-fibrillar collagen types have sites where the repeating (Gly-Xaa-Yaa)n sequences are interrupted by non- Gly-Xaa-Yaa sequences, and we are investigating the hypothesis that some of these interruptions can promote amyloid formation. Our experimental data show that model peptides containing an 8 or 9 residue interruption sequence between (Gly-Pro-Hyp)n domains have a strong propensity for self association to form fibrous structures. A peptide containing only the 9-residue interruption sequence forms amyloid like fibrils with anti-parallel β sheet. Computational analysis predicts that 33 out of 374 naturally occurring human non-fibrillar collagen sequences within or between triple-helical sequences have significant cross-β aggregation potential, including the 8 and 9 residue sequences studied in peptides. Further studies are in progress to investigate whether a triple-helix peptide promotes amyloidogenesis and whether amyloid interferes with collagen fibrillogenesis.

[1]NIH

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