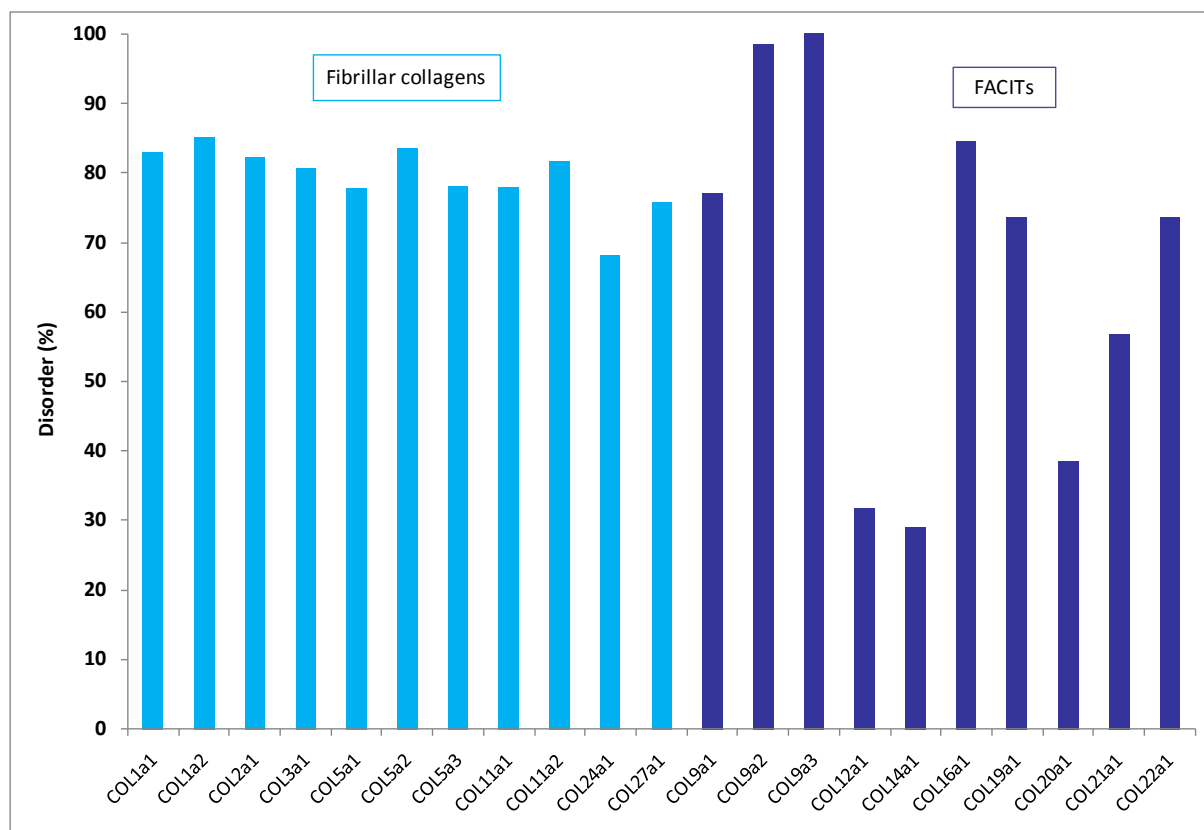
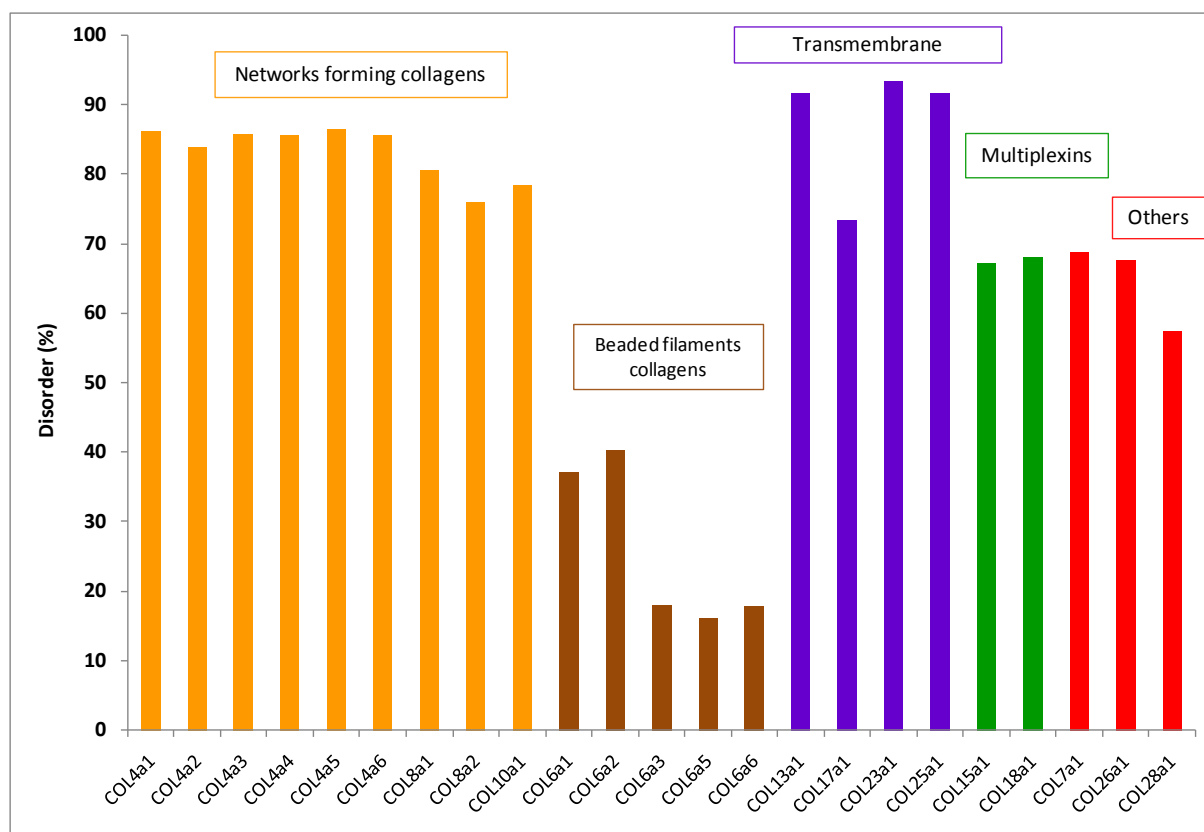


INTRINSIC DISORDER OF THE EXTRACELLULAR MATRIX

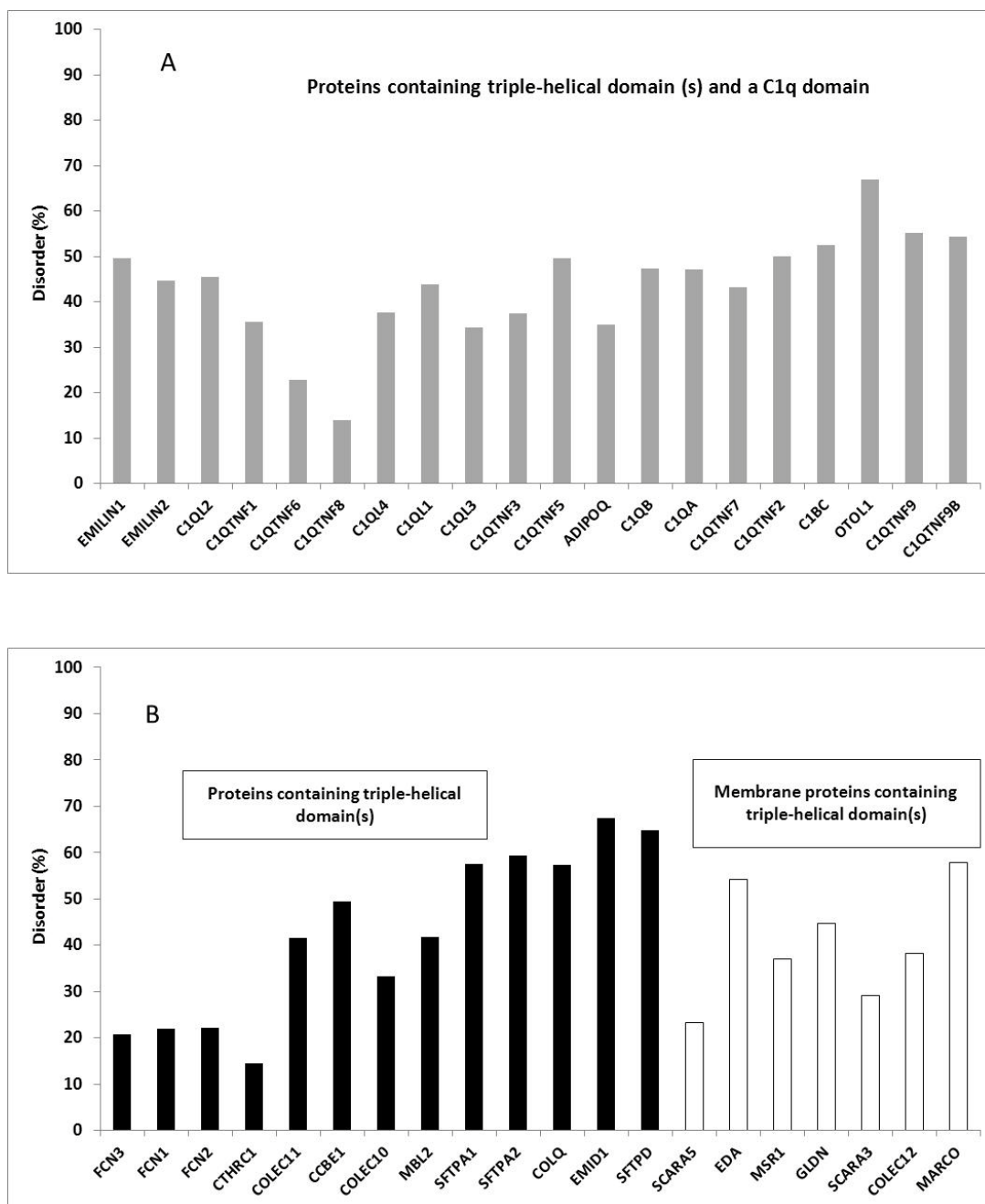
Franck Peysselon¹, Bin Xue², Vladimir N. Uversky^{2,3*}, Sylvie Ricard-Blum^{1*}



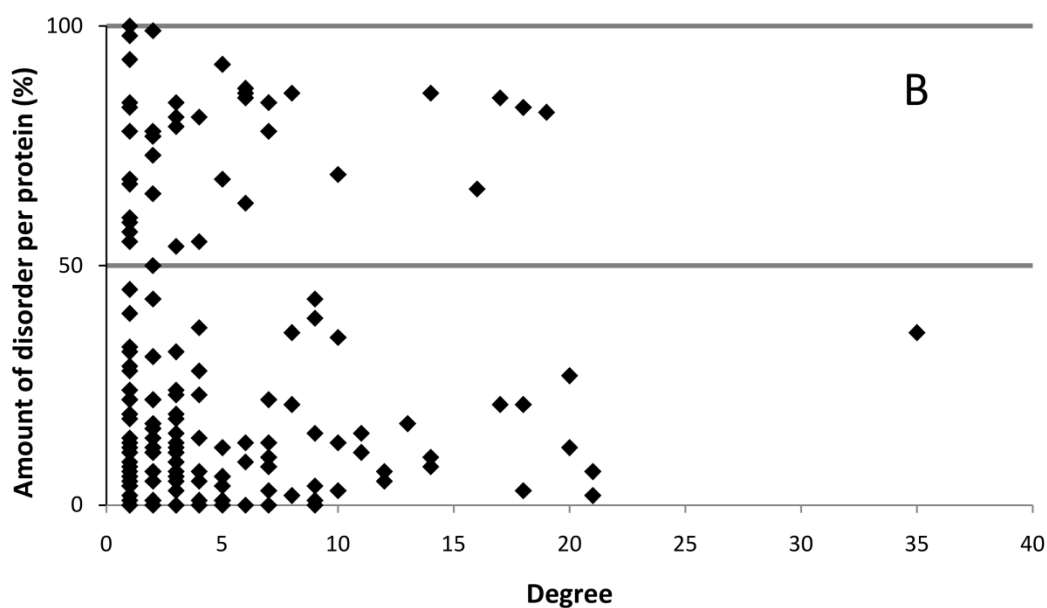
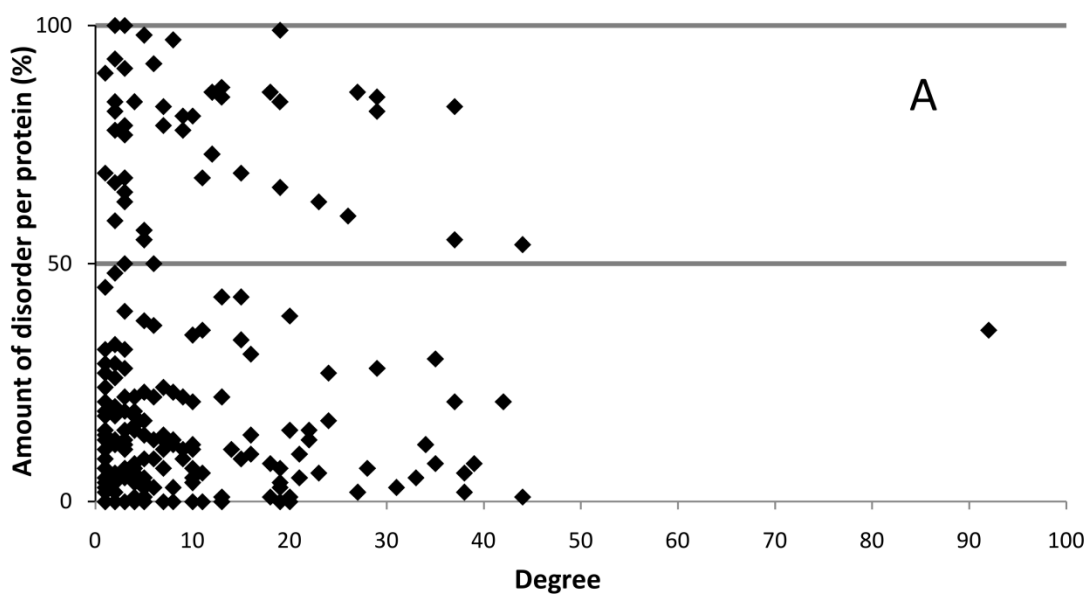
Supplementary Figure 1S: Amount of predicted disorder (%) in the α chains of fibril-forming collagens and Fibril-Associated Collagens with Interrupted Triple-Helices (FACITs). The polypeptide chains are designed by the gene name.



Supplementary Figure 2S: Amount of predicted disorder (%) in the α chains of network-forming collagens, collagen forming beaded filaments, membrane collagens, multiplexins, and other collagens. The polypeptide chains are designed by the gene name.



Supplementary Figure 3S: Amount of predicted disorder (%) in the individual chains of proteins containing triple-helical domain(s) and one C1q domain (A, ■), and in proteins that do not contain a C1q domain (B, □). The polypeptide chains are designed by the gene name and displayed in the increasing order of triple-helical content.



Supplementary Figure 4S: Amount of disorder (% of disordered residues) in extracellular proteins versus their degree in protein-protein interaction networks comprising all the protein partners of extracellular proteins (ECM_1, A) or only the extracellular partners of extracellular proteins (ECM_2, B).