Supporting Information

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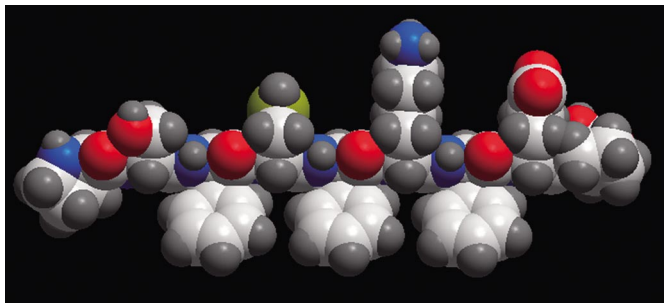

Fig. S1. Schematic molecular model of the amphiphilic peptide N-Pro-Ser-Phe-Cys-Phe-Lys-Phe-Glu-Pro-C. Carbon atoms are white, oxygen atoms are red, nitrogen atoms are blue, and hydrogen atoms are gray. In this conformation, side chains of the hydrophobic phenylalanine face in one direction, but the side chains of serine, cysteine, lysine, and glutamic acid face in the other direction, representing two distinct faces. The dimensions are ≈3.1 nm long, 1.2 nm high, and 0.4 nm thick.

Fig. S2. Pyrene crystals in water (*Left*) and pyrene crystals ([PY] = 2.47×10^{-3} M) in peptide solution ([peptide] = 4.38×10^{-4} M) (*Right*) after stirring both solutions for 1.5 h. Image shows that microcrystals of pyrene are stabilized by the amphiphilic peptide when suspended in aqueous solution.

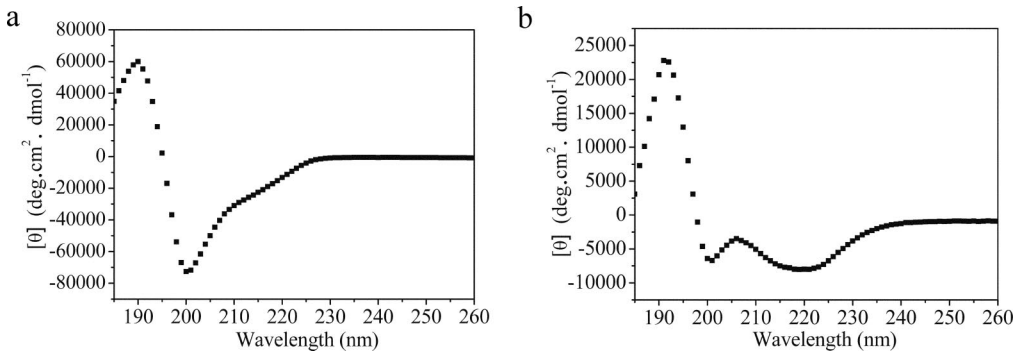

Fig. S3. CD spectra of mutated peptides containing single-residue replacement based on the original designed peptide. (*A*) CD spectrum of the mutant peptide P1, which contains replacement of Ser with Thr, N-Pro-Thr-Phe-Cys-Phe-Lys-Phe-Glu-Pro-C. The experimental conditions were exactly the same with a concentration of 0.23 mg/mL in water for 2 days before the CD spectrum test. A β-turn spectrum with a 200-nm minimum and a 189-nm maximum was detected, which was similar to the originally designed peptide. The spectrum shows a negative maximum ≈200 nm ($[\theta]_{200} = -73{,}000°$ per cm^2·dmol^{-1}) and a positive maximum near 189 nm ($[\theta]_{189} = 58{,}000°$ per cm^2·dmol^{-1}), with a slight intensity increase compared with the originally designed peptide. (*b*) CD spectrum of the mutant peptide P2 that contains replacement of Glu with Asp, N-Pro-Ser-Phe-Cys-Phe-Lys-Phe-Asp-Pro-C. The experimental conditions were the same with the originally designed peptide at 0.23 mg/mL in water for 2 days before the CD spectrum test. The spectrum shows a negative maximum ≈200 nm ($[\theta]_{200} = -6{,}400°$ per cm^2·dmol^{-1}) and 219 nm ($[\theta]_{219} = -8{,}000°$ per cm^2·dmol^{-1}), and a positive maximum near 189 nm ($[\theta]_{189} = 17{,}000°$ per cm^2·dmol^{-1}), with a distinct change compared with the originally designed peptide. The data indicate that the residue Glu plays an important role in the secondary structure conformation of the originally designed peptide.

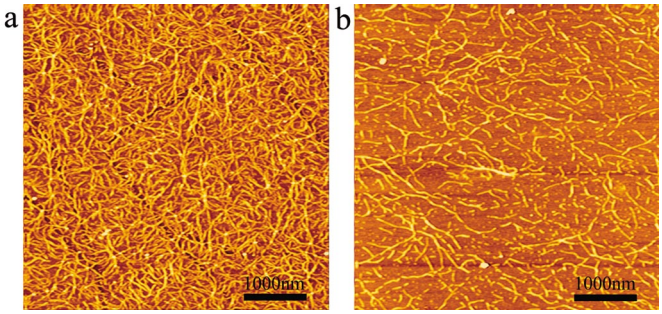

Fig. S4. Atomic force microscopy images of the mutated peptides P1 and P2. (*a*) P1 was dissolved in water at 0.5 mg/mL, and its image shows massive network containing many branching fibers. (*b*) P2 was dissolved in water at 0.5 mg/mL, and its image shows much shorter fiber formation with a less-branched network, indicating the important role of the residue Glu in fiber assembly. The scales are marked in each panel.

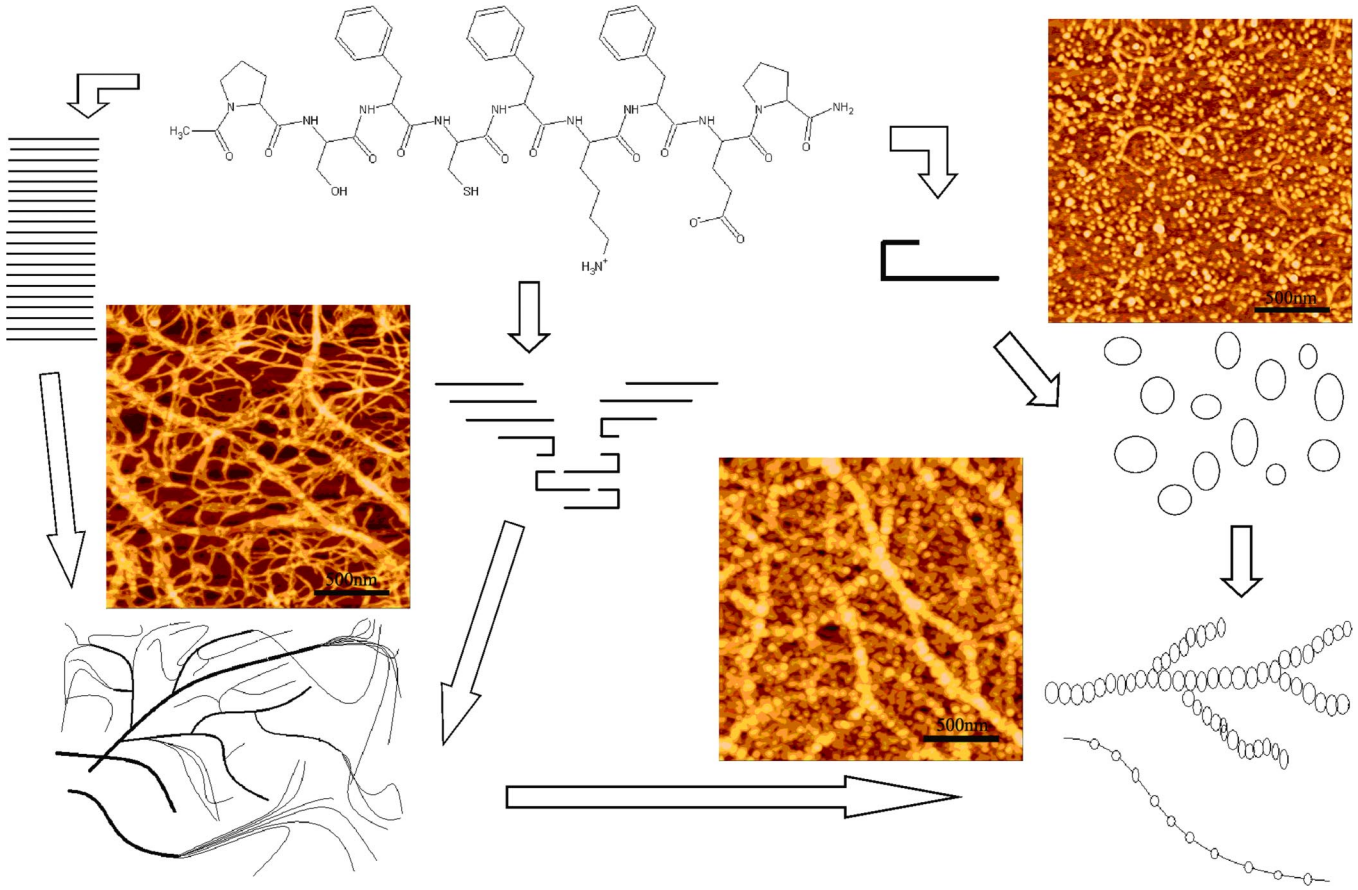

Fig. S5. Model for self-assembly. There are three stages for the peptide fibril formation. At the first stage, the peptide monomers self-assemble into protofibrils. The protofibrils can adopt two different nanostructures, i.e., globular aggregates or filaments. The filaments could be formed through the assembly β- sheets, whereas the globular beads could be formed based on combined interactions contributed by both β-turn and β-sheet structures. The second step involves the association of the filaments to form branched fibrils possibly around the terminal ends containing Pro residues, which usually curve peptide chains to create more branching points, allowing filling up the unpaired regions inside the hairpin structures. In addition, the β-turn structure also generates more bending forces and facilitates branching points to allow the unpaired filaments branching out stably to interact with either small aggregates or other filaments. At the third step, those globular aggregates could interact directly with the branched fibrils and form the ''beads-on-a-thread'' nanostructures. The coexistence of both β-turn and β-sheet structures and terminal Pro residues enables the peptide interaction in a much more complicated way, such as massively staggered fiber formation caused by branched points at turn or bending regions. Turning or bending features embedded inside this peptide contributed by the elements mentioned above can create more branched and knotted fibers that tether them together to form an interwoven network. Because this interaction belongs to heterogeneous process and each step in the model may occur at any given time during assembly, this model aims to outline main possible events to explain how turns and sheets as well as Pro residues enable nanostructure formation without sequential relationships.

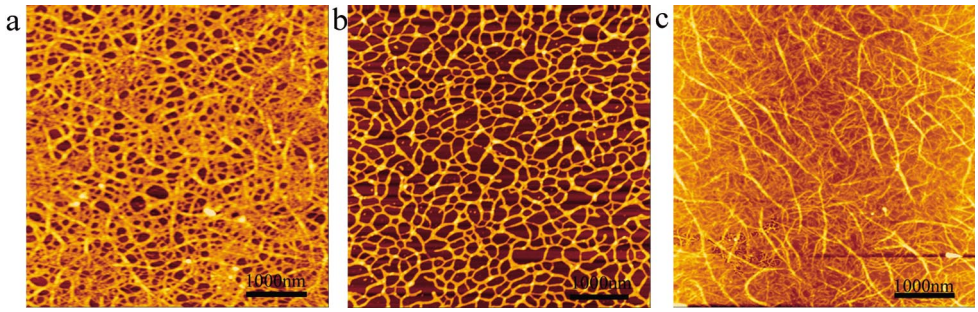

Fig. S6. AFM images of the peptide (0.5 mg/mL) nanofiber at different stages of sonication. (*a*) The image, before sonication, shows staggered fibers, and some of them are thin fibrillar branches within the network. (*b*) The image collected immediately after sonication shows much of the large-sized meshwork, indicating that the sonication did not destroy the mainframe structure. (*c*) The image collected 2 days after sonication shows a high density of fibers. The meshwork has been fully filled up to form long fibers and sheets, indicating dynamic process of disassembly and reassembly in response to sonication disruption. The scales are marked in each panel.