Supplementary Note 1.

Our abundant structural data on rods (in vivo) and crystals (in vitro) show that rods and crystals share such strong resemblance to each other that their small differences may not be meaningful. Our diffraction patterns reveal they have the same architecture and molecular interfaces (Fig. 3 c,d,e,f). The only distinction between crystals and rods appears to be the degree of molecular order. GFP fusion molecules are more regularly ordered in the crystal than they are in rods. Hence, the rods are capable of bending, whereas crystals usually do not bend. Even this distinction between crystals and rods becomes blurred when the crystals are small. For example, our previous work with amyloid-like microcrystals shows that peptide molecules transition from crystal to fibrils without interruption. See Fig 1b,c in Sawaya et al., 2007.[1]

Our data indicate that it is unlikely that an endogenous protein in *E. coli* is required for GFP fusion protein rod formation. We have shown it is possible to produce crystals from purified GFP fusion protein *in vitro* without additional bacterial proteins or cofactors. Because these crystals are architecturally similar to rods (just more ordered), there is no reason to hypothesize that other bacterial proteins are necessary for rod assembly, although they may certainly participate. We imagine that a bacterial protein might conceivably play a role in lending the rod its limited flexibility, by randomly incorporating and interrupting the crystal-like lattice of the rods.

Supplementary Note 2.

We offer evidence that most of the required structural ingredients are already present in a majority of globular proteins: surfaces capable of forming a quasi-stable, screw-generated intermolecular interface (such as the patch of direct contact between GFP barrels observed in our GFP fusion protein protofilament), and a nearby terminus that, if appended with an appropriate structural element, could donate this element to bridge the interface. Screw symmetry is inherent to most natural filaments. It is also abundant in protein crystals. Over 95% of the 12,933 monomeric protein crystal structures in the Protein Data Bank (PDB) contain screw-generated intermolecular interfaces. Of these, 74% meet the criterion of a quasi-stable interface as defined by equaling or surpassing the 901 $Å^2$ of buried surface area (450 $Å^2$ per surface) observed between barrel domains of our GFP fusion protein protofilament. This area is only slightly larger than the average crystal lattice contact for monomeric proteins (400 $Å^2$ per surface). Including a constraint that one of the termini be located within 12 Å of the intermolecular interface (a threshold defined by our GFP fusion protein) leaves 53% of monomeric PDB entries meeting these combined criteria. That is, over half the monomer entries in the PDB (6816 entries) are poised for filament formation, waiting only for the genetic event that would append an appropriately shaped donor element, such as an α-helix, to stably bridge the protomer-protomer interface.

Table S1. Filament assemblies and their properties

System	Organism	Function	Symmetry	Polarity	Persistence length	Area Buried (Å^2)	Solvation Energy (cal/mol)[≠] [2]	Runaway Domain Swapping	Runaway Domain Coupling	Stacking	Bridging	Reference	PDB ID code
GFP-RNase(1-8) fusion	Synthetic fusion	Fluorescent marker	Two-fold screw symmetry along filament axis.	Yes	>1μm.	2054	14040	No.	Yes, donor domain is the C-terminal helix.	Yes, barrel-to-barrel.	Yes, helix i bridges protomers i+1 and i+2.	This work.	5HBD, **5HGE**, 5HW9, 6AS9
Nucleocapsid protein	Phlebo-viruses	Encapsidate RNA	Asymmetric.	Yes	<100 nm. Extremely flexible.	3147	28262	No.	Yes, donor domain is the N-terminal helix.	No, protomers are linked only through a flexible loop.	No, each protomer contacts only its two closest neighbors.	[3]	4V9E, 4H5Q, 4H5P, 4H5O, 4H5M, **4H5L**
Actin, Crenactin, MreB, ParM	Eukaryotes, Archaea, Prokaryotes	Cyto-skeleton	Screw symmetry along filament axis. Non-integral number of protomers per turn.	Yes	~9 μm with MgADP[4].	4819	27053	No.	No.	Yes, Multiple small patches of contact spread over four subdomains.	Yes, each protomer bridges two protomers on the opposite strand.	[5,6,7]	3JBJ, 3J8I, **5MW1**, 5AEY
Actin and Tropomyosin copolymer	Eukaryotes	Cyto-skeleton	Screw symmetry along filament axis. Non-integral number of protomers per turn.	Yes	~20 μm, approximately twice more rigid than isolated actin filaments[4]. Isolated tropomyosin filaments have persistence length ~100 nm [8].	3209‖	20882‖	No.	No.	Yes, Multiple small patches of contact spread over four subdomains.	Yes, tropomyosin is a pair of long helices which bridges 6 to 7 actin protomers longitudinally.	[9,10]	3J8A, **5JLF**
Septin	Eukaryotes	Cyto-skeleton, cell septation.	Two-fold rotation perpendicular to filament axis.	No	12 μm on membrane bilayers. Hinge motion (25°-30°) evident in electron micrographs, likely enabled by the swapped NC-dimer interface.	2175	19106	No.	Yes, donor domain is the N-terminal helices in the NC dimer interface. Further stabilized by C-terminal coiled-coils.	Yes, stacking between GTP-binding domains in the G-dimer interface.	No.	[11,12]	**2QA5**, 2QAG
RecA, Rad51, RadA.	Prokaryotes, Eukaryotes, Archaea	DNA recombination	Six-fold, screw symmetry along filament axis.	Yes	~1 μm with ssDNA and ATP[13].	3901	26544	No.	Yes, donor domain is the N-terminal helix; it accounts for 60% of protomer interface.	Yes, contacts between globular domains account for 40% of protomer interface.	Yes, DNA substrate bridges multiple protomers.	[14,15,16,17]	2REB, 3CMW, **5NP7**
T7 gp4 helicase domain	Synthetic fragment of viral gene	helicase	Six-fold, screw symmetry along filament axis.	Yes	~1 μm, estimated by homology with RecA	1869	10850	No.	Yes, donor domain is N-terminal helix. It accounts for 60% of protomer interface.	Yes, contacts between globular domains account for 40% of protomer interface.	No.	[18]	**1CR1**
Tubulin, FtsZ	Eukaryotes, Prokaryotes	Cyto-skeleton	Thirteen-fold screw symmetry along filament axis.	Yes	~5200 μm in vitro, 20 μm in vivo[19]. Extremely rigid.	5423	28011	No.	No.	Yes, all contacts between protomers occur through rigid domains.	No, not within a protofilament. However, bridging occurs through contacts between protofilaments.	[20]	**3JAK**
Fibrin	Eukaryotes	Blood clotting	Two-fold rotation perpendicular to filament axis.	No	~500 nm[21]	7120 A 8772 B 6879 C	55566 A 67805 B 57013 C	No.	No.	Yes, a D-D interface and an E-E interface, in addition to disulfide bonds.	Yes, through knob-hole interactions.	[22,23,24]	3GHG, **1M1J**, 1FZF

Intermediate Filaments: Vimentin Keratin Lamin Desmin (Crescentin)	Eukaryotes, (Prokaryotes)	Cyto-skeleton	Two-fold rotation symmetry perpendicular to filament axis.	No	~400 nm, high flexibility[25].	3874	39460	No.	No*.	Yes, coiled coils stack, or bundle*	Yes, each coiled coil contacts more than two neighbor protomers.	[26], [27]	3SWK, 3S4R, **3UF1**
Spectrins actinin, plakins, plectin, dystrophin	Eukaryotes	Cyto-skeleton	Two-fold rotational symmetry between protofilaments.	No	Rigid, short cross-linkers for actin. Protofilaments are single polypeptide chains composed of multiple helical repeats.	4249	26878	No.	Yes, neck helix of one protomer docks in EF34 domain of another protomer.	Yes, lateral stacking between helical domains.	No.	[28], [29]	**4D1E**
Shank3 SAM domain filament	Eukaryotes	Post synaptic density scaffold	Six-fold screw symmetry along filament axis.	Yes.		826	2827	No.	No.	Yes.	No.	[30]	**2F3N**
Myosin tail	Eukaryotes	Colocalize myosin motors to a thick filament.	Long coiled coils that bundle together.	Yes	642-1742 μm[31]	11344 A	91584 A	No.	No*.	Yes, coiled coils stack, or bundle*. Myosin heads do not stack together.	Yes, bridging between coiled-coils in thick filament.	[32], [33], [34]	1I84, **3JBH**, EMD-3301
Pilus (S. pyogenes gene 0128)	Prokaryotes (Gram positive)	Adhesion to host surfaces	Three-fold screw symmetry along filament axis.	Yes	220-280 nm[35]	829	6079	No.	No.	Yes, immunoglobulin-like domains stack end-to-end. Additionally, isopeptide bonds cross-link protomers.	No.	[36]	**3B2M**
Pilus Type IV	Prokaryotes	Twitching motility	Screw symmetry along filament axis. Rise of 10.3 Å and rotation of 100.8°	Yes	~5 μm[37]	4329	33083	No.	Yes, donor domain is the N-terminal helix.	Yes, a small interface between C-terminal domains, and a large interface between N-terminal domains.	Yes, among helical regions.	[38], [39]	**5VXX**, 5VXY, 5KUA
Pilus Type IV, sex F	Prokaryotes	Transfer of genetic material,	Five-fold screw symmetry along filament axis.	Yes	~5 μm[37]	2866	34987	No.	No.	Yes, short anti-parallel helical protomers stack laterally and longitudinally in a tube.	Yes, each protomer contacts 7 neighbors.	[40]	**5LEG**, 5LFB, 5LER
Pilus Type chaperone-usher, fimA	Prokaryotes	Adhesion to host surfaces				5978	21892	No.	Yes, N-terminal strand docks into incomplete immunoglobulin fold of neighboring protomer.	Yes, immunoglobulin domains stack along fibril axis.	Yes, each protomer contacts 8 to 10 neighbors.	[41]	**5OH0**
R-type Pyocin	Prokaryotes	Open pores in the cytoplasmic membrane.	Hexameric rings stacked with screw symmetry along tube axis.	Yes	Rigid	6068	36658	No.	Yes, C-terminal helix inserts into adjacent protomer of the hexameric ring.	Yes, rigid domains stack side-by-side in hexameric rings, and longitudinally.	Yes, each protomer shares interfaces with six surrounding protomers, some of these are in mutual contact.	[42]	**5W5E**
T4 Tail tube	Enterobacteria phage T4	Transfer DNA	Hexameric rings stacked with screw symmetry along tube axis.	Yes	Rigid	6967	41501	No.	Yes, an N-terminal helix and a β-hairpin inserts into adjacent subunits.	Yes, rigid domains stack side-by-side in hexameric rings, and longitudinally.	Yes, each protomer shares interfaces with six surrounding protomers, some of these are in mutual contact.	[42]	**5W5F**

"cab"-type carbonic anhydrase	Archaea	Carbonic anhydrase	Two-fold screw symmetry along filament axis.	Yes	Filaments not reported outside of crystal.	3631	30540	Yes, an N-terminal helix swaps into adjacent subunits.	No.	No.	No, bridging is not likely to exist outside of the crystal lattice.	[43]	**1G5C**
α_1-antitrypsin	Eukaryotes	Protease inhibitor.	None.	Yes	Highly flexible "beads-on-a-string."[44]	4874	41956	Yes, a strand inserts into and adjacent protomer.	No.	No.	No.	[45]	**1QMB**
Amyloid β, a disease-related amyloid	Eukaryotes	Hallmark of Alzheimer's disease.	None or two-fold symmetry along filament depending on the polymorph.	Yes	~50 nm[46]	4071	21319	No.	No, the protomer is a single, rigid 42-residue β-meander.	Yes.	No.	[47, 48, 49, 50, 51]	5KK3, 2NAO, 5AEF, **5OQV**
PSM-α3, a functional amyloid	Prokaryotes	Cell adhesion, biofilm formation.	Two-fold screw symmetry along filament axis.	Yes	~500 nm‡,[52]	1178	14824	No.	No, the protomer is a single, rigid 21-residue α-helix.	Yes, helices stack to form a sheet.	Yes, adjacent α-helices in one sheet are bridged by an α-helix in opposing sheet.	[52, 53]	**5I55**
HET-s, a functional prion.	Prokaryotes	Self/nonself recognition.	None.	Yes	7.5 +/-2.8 μm[54]	3789	14901	No.	No, the protomer core is a single, rigid 79-residue β-helix.	Yes, strands stack to form a sheet.	No.	[55]	2RNM, **2KJ3**
Collagen	Eukaryotes	Connective tissue	Three-fold screw axis	Yes	12-165 nm depending on mineralization[56].	1223	5658	No.	No*.	Yes, through main chain hydrogen bonding and van der Waals contact*.	Yes, each pair of strands is bridged by a third strand.	[57]	**1BKV**
Flagellar filament	Archaea, Prokaryotes	Cell motility, cell adhesion	Screw symmetry along filament axis. Ten subunits span three turns and one translational repeat.	Yes	~30 +/- 10 μm, rigid[58].	6620	58855	No.	No.	Yes, between N-terminal helical tails and between C-terminal globular domains.	Yes, each protomer bridges multiple neighboring protomers.	[59, 60, 61]	**5TFY**, 5O4U, 1UCU
CTP synthase	Prokaryotes, Eukaryotes	Regulation and storage of CTP synthase activity	Screw symmetry along filament axis.	No	Apparently rigid. [no length measurement?]	3091	19916	No.	Not in prokaryotic enzymes. Yes, in eukaryotic enzymes, involving a hinge between GAT and AL domains.	Yes, helical stacking of homo tetramers in both kingdoms.	Yes, bridging within and between subunits of tetramers in both kingdoms.	[62]	**5U6R**, 5U3C, 5U05, 5U03
Hemoglobin	Humans	Deterrent to malaria infection	Two-fold symmetry in projection	No	0.24 mm	2381	11427	No.	No.	Yes, native tetramer interface and additional side chain contacts between tetramers.	Yes, each longitudinal pair of protomers is bridged by contacts with a protomer from the adjacent strand.	[63, 64, 65]	**2HBS**

*The distinction between swapping and stacking is difficult to discern for coiled coil interactions such as seen in vimentin, the myosin tail, and collagen. To meet our requirement for runaway domain coupling, the protomer must: (1) contain two or more domains connected by a flexible hinge region and (2) each domain must interact with a separate protomer. While it is possible that hinges do exist in these long, slender helices, the resolution of these assemblies is too poor to establish their existence. Hence, we classify these coiled-coil interactions as stacking, until proven otherwise.

‡Rough estimate based on micrographs in this reference.

≠Energy of solvation calculated from PDB file indicated in bold. Values followed by a letter indicate which chain was used for the calculation.
‖ Note, value is underestimated due to side chains missing from model.

Supplementary References

1. Sawaya, M. R. *et al.* Atomic structures of amyloid cross-beta spines reveal varied steric zippers. *Nature* **447,** 453–457 (2007).
2. Eisenberg, D. & McLachlan, A. D. Solvation energy in protein folding and binding. *Nature* **319,** 199–203 (1986).
3. Raymond, D. D., Piper, M. E., Gerrard, S. R., Skiniotis, G. & Smith, J. L. Phleboviruses encapsidate their genomes by sequestering RNA bases. *Proc. Natl. Acad. Sci. U. S. A.* **109,** 19208–19213 (2012).
4. Isambert, H. *et al.* Flexibility of actin filaments derived from thermal fluctuations. Effect of bound nucleotide, phalloidin, and muscle regulatory proteins. *J. Biol. Chem.* **270,** 11437–11444 (1995).
5. Galkin, V. E., Orlova, A., Vos, M. R., Schröder, G. F. & Egelman, E. H. Near-atomic resolution for one state of F-actin. *Struct. Lond. Engl. 1993* **23,** 173–182 (2015).
6. Izoré, T., Kureisaite-Ciziene, D., McLaughln, S. H. & Löwe, J. Crenactin forms actin-like double helical filaments regulated by arcadin-2. *eLife* **5,** (2016).
7. Bharat, T. A. M., Murshudov, G. N., Sachse, C. & Löwe, J. Structures of actin-like ParM filaments show architecture of plasmid-segregating spindles. *Nature* **523,** 106–110 (2015).
8. Li, X. (Edward), Holmes, K. C., Lehman, W., Jung, H. & Fischer, S. The Shape and Flexibility of Tropomyosin Coiled Coils: Implications for Actin Filament Assembly and Regulation. *J. Mol. Biol.* **395,** 327–339 (2010).
9. von der Ecken, J. *et al.* Structure of the F-actin-tropomyosin complex. *Nature* **519,** 114–117 (2015).

10. Khaitlina, S. Y. Tropomyosin as a Regulator of Actin Dynamics. *Int. Rev. Cell Mol. Biol.* **318,** 255–291 (2015).

11. Mostowy, S. & Cossart, P. Septins: the fourth component of the cytoskeleton. *Nat. Rev. Mol. Cell Biol.* **13,** 183–194 (2012).

12. Sirajuddin, M. *et al.* Structural insight into filament formation by mammalian septins. *Nature* **449,** 311–315 (2007).

13. Hegner, M., Smith, S. B. & Bustamante, C. Polymerization and mechanical properties of single RecA–DNA filaments. *Proc. Natl. Acad. Sci.* **96,** 10109–10114 (1999).

14. Prentiss, M., Prévost, C. & Danilowicz, C. Structure/function relationships in RecA protein-mediated homology recognition and strand exchange. *Crit. Rev. Biochem. Mol. Biol.* **50,** 453–476 (2015).

15. Story, R. M., Weber, I. T. & Steitz, T. A. The structure of the E. coli recA protein monomer and polymer. *Nature* **355,** 318–325 (1992).

16. Chen, Z., Yang, H. & Pavletich, N. P. Mechanism of homologous recombination from the RecA–ssDNA/dsDNA structures. *Nature* **453,** 489–494 (2008).

17. Short, J. M. *et al.* High-resolution structure of the presynaptic RAD51 filament on single-stranded DNA by electron cryo-microscopy. *Nucleic Acids Res.* **44,** 9017–9030 (2016).

18. Sawaya, M. R., Guo, S., Tabor, S., Richardson, C. C. & Ellenberger, T. Crystal structure of the helicase domain from the replicative helicase-primase of bacteriophage T7. *Cell* **99,** 167–177 (1999).

19. Pallavicini, C. *et al.* Lateral Motion and Bending of Microtubules Studied with a New Single-Filament Tracking Routine in Living Cells. *Biophys. J.* **106,** 2625–2635 (2014).

20. Nogales, E. An electron microscopy journey in the study of microtubule structure and dynamics. *Protein Sci. Publ. Protein Soc.* **24,** 1912–1919 (2015).

21. Storm, C., Pastore, J. J., MacKintosh, F. C., Lubensky, T. C. & Janmey, P. A. Nonlinear elasticity in biological gels. *Nature* **435,** 191–194 (2005).

22. Spraggon, G., Everse, S. J. & Doolittle, R. F. Crystal structures of fragment D from human fibrinogen and its crosslinked counterpart from fibrin. *Nature* **389,** 455–462 (1997).

23. Doolittle, R. F., Spraggon, G. & Everse, S. J. Three-dimensional structural studies on fragments of fibrinogen and fibrin. *Curr. Opin. Struct. Biol.* **8,** 792–798 (1998).

24. Doolittle, R. F. X-ray crystallographic studies on fibrinogen and fibrin. *J. Thromb. Haemost.* **1,** 1559–1565 (2003).

25. Schopferer, M. *et al.* Desmin and Vimentin Intermediate Filament Networks: Their Viscoelastic Properties Investigated by Mechanical Rheometry. *J. Mol. Biol.* **388,** 133–143 (2009).

26. Herrmann, H. & Aebi, U. Intermediate Filaments: Structure and Assembly. *Cold Spring Harb. Perspect. Biol.* **8,** a018242 (2016).

27. Chernyatina, A. A., Nicolet, S., Aebi, U., Herrmann, H. & Strelkov, S. V. Atomic structure of the vimentin central α-helical domain and its implications for inter-

mediate filament assembly. *Proc. Natl. Acad. Sci. U. S. A.* **109,** 13620–13625 (2012).

28. Le Rumeur, E., Hubert, J.-F. & Winder, S. J. A new twist to coiled coil. *FEBS Lett.* **586,** 2717–2722 (2012).

29. Ribeiro, E. de A. *et al.* The Structure and Regulation of Human Muscle α-Actinin. *Cell* **159,** 1447–1460 (2014).

30. Baron, M. K. *et al.* An Architectural Framework That May Lie at the Core of the Postsynaptic Density. *Science* **311,** 531–535 (2006).

31. Miller, M. S., Tanner, B. C. W., Nyland, L. R. & Vigoreaux, J. O. Comparative Biomechanics of Thick Filaments and Thin Filaments with Functional Consequences for Muscle Contraction. *J. Biomed. Biotechnol.* **2010,** (2010).

32. Woodhead, J. L. *et al.* Atomic model of a myosin filament in the relaxed state. *Nature* **436,** 1195–1199 (2005).

33. Alamo, L. *et al.* Conserved Intramolecular Interactions Maintain Myosin Interacting-Heads Motifs Explaining Tarantula Muscle Super-Relaxed State Structural Basis. *J. Mol. Biol.* **428,** 1142–1164 (2016).

34. Hu, Z., Taylor, D. W., Reedy, M. K., Edwards, R. J. & Taylor, K. A. Structure of myosin filaments from relaxed Lethocerus flight muscle by cryo-EM at 6 Å resolution. *Sci. Adv.* **2,** e1600058 (2016).

35. Rheinlaender, J., Gräbner, A., Ott, L., Burkovski, A. & Schäffer, T. E. Contour and persistence length of Corynebacterium diphtheriae pili by atomic force microscopy. *Eur. Biophys. J. EBJ* **41,** 561–570 (2012).

36. Kang, H. J., Coulibaly, F., Clow, F., Proft, T. & Baker, E. N. Stabilizing isopeptide bonds revealed in gram-positive bacterial pilus structure. *Science* **318,** 1625–1628 (2007).

37. Skerker, J. M. & Berg, H. C. Direct observation of extension and retraction of type IV pili. *Proc. Natl. Acad. Sci.* **98,** 6901–6904 (2001).

38. Wang, F. *et al.* Cryoelectron Microscopy Reconstructions of the Pseudomonas aeruginosa and Neisseria gonorrhoeae Type IV Pili at Sub-nanometer Resolution. *Structure* **25,** 1423-1435.e4 (2017).

39. Kolappan, S. *et al.* Structure of the Neisseria meningitidis Type IV pilus. *Nat. Commun.* **7,** 13015 (2016).

40. Costa, T. R. D. *et al.* Structure of the Bacterial Sex F Pilus Reveals an Assembly of a Stoichiometric Protein-Phospholipid Complex. *Cell* **166,** 1436-1444.e10 (2016).

41. Hospenthal, M. K. *et al.* The Cryoelectron Microscopy Structure of the Type 1 Chaperone-Usher Pilus Rod. *Structure* **25,** 1829-1838.e4 (2017).

42. Zheng, W. *et al.* Refined Cryo-EM Structure of the T4 Tail Tube: Exploring the Lowest Dose Limit. *Structure* **25,** 1436-1441.e2 (2017).

43. Strop, P., Smith, K. S., Iverson, T. M., Ferry, J. G. & Rees, D. C. Crystal structure of the 'cab'-type beta class carbonic anhydrase from the archaeon Methanobacterium thermoautotrophicum. *J. Biol. Chem.* **276,** 10299–10305 (2001).

44. Lomas, D. A., Evans, D. L., Finch, J. T. & Carrell, R. W. The mechanism of Z alpha 1-antitrypsin accumulation in the liver. *Nature* **357,** 605–607 (1992).

45. Huntington, J. A. *et al.* A 2.6 A structure of a serpin polymer and implications for conformational disease. *J. Mol. Biol.* **293,** 449–455 (1999).

46. Witte, T. *et al.* Time resolved structure analysis of growing beta-amyloid fibers. *J. Struct. Biol.* **159,** 71–81 (2007).

47. Riek, R. & Eisenberg, D. S. The activities of amyloids from a structural perspective. *Nature* **539,** 227–235 (2016).

48. Colvin, M. T. *et al.* Atomic Resolution Structure of Monomorphic Aβ42 Amyloid Fibrils. *J. Am. Chem. Soc.* **138,** 9663–9674 (2016).

49. Wälti, M. A. *et al.* Atomic-resolution structure of a disease-relevant Aβ(1-42) amyloid fibril. *Proc. Natl. Acad. Sci. U. S. A.* **113,** E4976-4984 (2016).

50. Schmidt, M. *et al.* Peptide dimer structure in an Aβ(1-42) fibril visualized with cryo-EM. *Proc. Natl. Acad. Sci. U. S. A.* **112,** 11858–11863 (2015).

51. Gremer, L. *et al.* Fibril structure of amyloid-β(1–42) by cryo–electron microscopy. *Science* **358,** 116–119 (2017).

52. Tayeb-Fligelman, E. *et al.* The cytotoxic Staphylococcus aureus PSMα3 reveals a cross-α amyloid-like fibril. *Science* **355,** 831–833 (2017).

53. Taglialegna, A., Lasa, I. & Valle, J. Amyloid Structures as Biofilm Matrix Scaffolds. *J. Bacteriol.* **198,** 2579–2588 (2016).

54. Lamour, G. *et al.* Mapping the Broad Structural and Mechanical Properties of Amyloid Fibrils. *Biophys. J.* **112,** 584–594 (2017).

55. Wasmer, C. *et al.* Amyloid Fibrils of the HET-s(218–289) Prion Form a β Solenoid with a Triangular Hydrophobic Core. *Science* **319,** 1523–1526 (2008).

56. Lovelady, H. H., Shashidhara, S. & Matthews, W. G. Solvent specific persistence length of molecular type I collagen. *Biopolymers* **101,** 329–335 (2014).

57. Bella, J. Collagen structure: new tricks from a very old dog. *Biochem. J.* **473,** 1001–1025 (2016).

58. Trachtenberg, S. & Hammel, I. The rigidity of bacterial flagellar filaments and its relation to filament polymorphism. *J. Struct. Biol.* **109,** 18–27 (1992).

59. Poweleit, N. *et al.* CryoEM structure of the Methanospirillum hungatei archaellum reveals structural features distinct from the bacterial flagellum and type IV pili. *Nat. Microbiol.* **2,** 16222 (2016).

60. Daum, B. *et al.* Structure and in situ organisation of the Pyrococcus furiosus archaellum machinery. *eLife* **6,** (2017).

61. Yonekura, K., Maki-Yonekura, S. & Namba, K. Complete atomic model of the bacterial flagellar filament by electron cryomicroscopy. *Nature* **424,** 643–650 (2003).

62. Lynch, E. M. *et al.* Human CTP synthase filament structure reveals the active enzyme conformation. *Nat. Struct. Mol. Biol.* **24,** 507–514 (2017).

63. Wang, Z., Kishchenko, G., Chen, Y. & Josephs, R. Polymerization of deoxysickle cell hemoglobin in high-phosphate buffer. *J. Struct. Biol.* **131,** 197–209 (2000).

64. Harrington, D. J., Adachi, K. & Royer Jr, W. E. The high resolution crystal structure of deoxyhemoglobin S1. *J. Mol. Biol.* **272,** 398–407 (1997).

65. Wang, J. C. *et al.* Micromechanics of isolated sickle cell hemoglobin fibers: bending moduli and persistence lengths. *J. Mol. Biol.* **315,** 601–612 (2002).