The structure and micromechanics of elastic tissue

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Elastin is a major component of tissues such as lung and blood vessels, and endows them with the long-range elasticity necessary for their physiological functions. Recent research has revealed the complexity of these elastin structures and drawn attention to the existence of extensive networks of fine elastin fibres in tissues such as articular cartilage and the intervertebral disc. Nonlinear microscopy, allowing the visualization of these structures in living tissues, is informing analysis of their mechanical properties. Elastic fibres are complex in composition and structure containing, in addition to elastin, an array of microfibrillar proteins, principally fibrillin. Raman microspectrometry and X-ray scattering have provided new insights into the mechanisms of elasticity of the individual component proteins at the molecular and fibrillar levels, but more remains to be done in understanding their mechanical interactions in composite matrices. Elastic tissue is one of the most stable components of the extracellular matrix, but impaired mechanical function is associated with ageing and diseases such as atherosclerosis and diabetes. Efforts to understand these associations through studying the effects of processes such as calcium and lipid binding and glycation on the mechanical properties of elastin preparations in vitro have produced a confusing picture, and further efforts are required to determine the molecular basis of such effects.

1. Introduction

Long-range elasticity is a requirement of many biological tissues. In mammals, a unique protein, elastin, is used to serve this function, although in other species a number of different proteins are used.

Elastin can constitute up to half the dry weight of tissues such as large blood vessels where, in association with a family of microfibrillar glycoproteins, it forms a lamellar structure whose biochemistry and contribution to vascular biomechanics have been exhaustively investigated. Similarly, the structure and biomechanics of elastic tissues in the skin and lung and the changes associated with ageing and disease are well understood [1,2]. However, fine fibres of elastin are difficult to detect by classical light and electron microscopic techniques, and, in consequence, the existence of networks of fine fibres of elastin in other tissues such as small blood vessels and adipose tissue has been largely overlooked. Fibrous elastin has been reported in cartilage [3,4]; however, its mechanical role has yet to be elucidated.

In §2, we summarize recent research on the distribution and organization of the fine fibril networks and the microstructure of denser elastin structures found in various tissues. The fibril networks are often found in close association with dense collagenous structures and, in §3, we discuss the implications of the coexistence of such structures of very different mechanical properties for tissue micromechanics.

The functional unit of elastin structure is the elastic fibre, generally containing elastin and a number of microfibrillar glycoproteins. In §4, we discuss the problem of relating the mechanical properties of these fibres to the organization and mechanical properties of the elastin molecule. The molecular bases of elastin mechanics have attracted interest since the 1960s, because long-range elasticity is generally associated with random polymers and is therefore an unusual property for a protein. Here, we present new insights derived from X-ray scattering and
Raman microspectrometry and discuss them in the context of molecular models of elastin and measurements of single molecule mechanics.

Elastin is a single gene-copy protein, but multiple isoforms may be produced by alternative splicing of its pre-mRNA. In §5, we summarize data on variations in molecular composition both between tissues and between species and consider the consequences of these variations on both intrinsic mechanical properties and the assembly of elastin into tissue-specific fibres and networks. Extending this theme, we also compare the structure and mechanical properties of elastin with those of the lamprins, a family of elastic proteins found in the agnathans, which may be evolutionary predecessors of the mammalian protein.

Very few diseases are associated with abnormalities in elastin, probably because defects in elastin are fatal in the perinatal period. However, elastin is the most stable of the extracellular matrix molecules and its rate of synthesis after puberty is almost unmeasurably small and, in consequence, it is susceptible to degradation or modification by chemical processes such as non-enzymatic glycation where the reaction rate is negligibly slow in comparison with the lifetime of most other matrix proteins. Loss of elasticity in structures such as blood vessels, lung and skin is a hallmark of the ageing process, and there are conflicting views on the extent to which it arises from fragmentation of elastin networks or from modification of the elastic properties of the elastin fibre itself. In §6, we summarize research relating changes in the mechanical properties of the elastin molecule and fibre mechanics to chemical interactions with the environment.

2. The distribution and structure of elastin fibres

In light microscopy, elastin is difficult to stain specifically and it is refractory to most of the stains used for electron microscopy. Probably for these reasons, existence of fine fibres of elastin in other tissues has often been overlooked. However, it is an intrinsic fluorophore, and two-photon excitation, which provides enhanced spatial resolution and depth penetration compared with single-photon stimulation, has revealed complex networks in a number of tissues. Figure 1 illustrates some representative structures.

In articular cartilage (figure 1a), a network is present particularly in interterritorial matrix of the surface zone and in the pericellular matrix [4]. In the intervertebral disc (figure 1b), elastin fibres span the nucleus pulposus and are particularly prominent in the annulus, running both within and between collagen annuli [5,6]. Even in adipose tissue, which contains only a sparse extracellular matrix, there is a rich elastin network (figure 1c). Less unexpectedly, elastin is a significant component of small resistance arteries and veins, where it can be found in two morphologically distinct arrangements (figure 1d).

Finally, chordae tendinae are generally regarded as collagenous structures [7], but they contain a delicate elastin structure, particularly at their periphery (figure 1e).

As discussed in §6, the similar fluorescence of these fibres may conceal differences in biochemistry, but they show some structural similarities. They are generally of uniform diameter along their length. In disc and cartilage, the fibres are fine and typically less than or equal to 1 μm in diameter; however, in adipose, there is a larger range of fibre diameters (1–6 μm).

The fibres in cartilage, blood vessels and disc are predominantly long and straight; however, adipose and chordae tendinae fibres are both straight and wavy. Fibres in disc and cartilage normally follow the predominant direction of the collagen fibres although in areas with a less clear collagen arrangement a wider range of elastin fibre angles are seen. The fibres frequently bifurcate, but there does not appear to be a preferred angle of branching or any obvious difference in diameter between parent and daughter fibres. The elastin in small resistance arteries comprises a layer one fibre thick in the media, and fibres in this layer are highly organized and aligned longitudinally, and appear to be connected at regular intervals. Elastin fibres are present in the adventitia as well, where they are more abundant and more randomly oriented.

We presume that these fibres form in the same way as the more extensive structures in, for example, large blood vessels, by deposition of elastin onto a skeleton of microfibrillar glycoproteins. Although the structure of elastin in the large arteries is commonly described as lamellar, this is actually somewhat misleading as demonstrated in figure 1f. As shown in the elegant electron microscopic studies by Spina and co-workers on carefully prepared specimens of pure elastin, there is a complex hierarchical structure as shown in figure 2.

The predominant structure is of twisted rope-like fibres approximately 1 μm in diameter which are composed of fibrils 0.2 μm in diameter, which are clearly revealed in regions in which the ropes appear to fray. Also evident are
much finer (less than 0.1 μm), generally straight fibrils not unlike those described above which appear to run between the larger fibres.

The larger fibres have a diameter of approximately 1 μm. Early work by Partridge [8] revealed that the purified fibres contain internal water-filled spaces sufficiently uniform in size to allow the fibres to be used as a substrate for size exclusion chromatography for solutes up to 1000 Da in molecular weight. Studies using microbeam X-ray diffraction show that the fibres have limited structural order with d-spacings of 0.47 and 0.92 nm [9]. These values suggest that molecular conformation and molecular organization within the fibres are determined by intra- and intermolecular hydrophobic interactions.

In summary, it appears that elastin fibres approximately 1 μm in diameter are common building blocks in forming the elastic structures of many tissues. However, the fibre probably has a complex substructure that complicates the analysis of its mechanical properties, as we shall discuss below.

3. Tissue micromechanics: mechanical functions of elastic fibres

The coexistence of networks of collagen and elastin fibres in tissues such as blood vessels, which give rise to the nonlinear mechanical properties that are central to their biomechanical function, is well established (reviewed in [2,10]). More recent work has incorporated structural information on the aortic elastin network into anisotropic mechanical models, validated against mechanical testing data [11]. Nonlinear microscopy provides almost comparable visualization of both the elastin and collagen networks (figure 1f) in living tissue and should in the near future provide direct validation of these models and allow their extension to include interactions with collagen.

Another challenge which may yield to such microscopic investigations is the question of pre-stress in the elastin network. Fung [12] has drawn attention to the existence of residual stresses in blood vessels which result in the opening of a blood vessel when its wall is cut longitudinally. At the microscopic level, there are probably differential residual stresses between the collagen and elastin network components. We have observed that when collagen is digested from large elastic arteries the elastin network expands from the unpressurized diameter of the vessel to something close to the diameter of the vessel at physiological pressure. How these stresses are generated during vascular development is a question for the future.

It was noted many years ago by Wolinsky & Glagov [13] that blood vessels have a modular structure which differs around the circulation. Comparing a particular vessel between species shows that the modular structure is conserved, but the number of modules varies with vessel dimensions suggesting that each module is designed to support a particular load. To the best of our knowledge, the micromechanics of these structural units and the manner in which their properties are matched to local haemodynamic conditions have not been explored.

The biomechanical properties of the small blood vessels constituting the microcirculation have received remarkably little attention in comparison with large arteries and veins, not least because of the technical challenges in handling delicate vessels only tens or hundreds of micrometres in diameter. In large blood vessels, the primary biomechanical requirement is passive matching to local haemodynamic conditions, affected by an appropriate mix of collagen and elastin fibres, as discussed above. Small blood vessels determine vascular resistance, and the distribution of blood flow through changes in vessel calibre produced by contraction or relaxation of smooth muscle. This requires that the supporting matrix allows significant changes in internal radius in response to a cell-generated force as well as to changes in luminal pressure. A synergy between elastin and collagen, described theoretically as a ‘hook-on’ model [14] (several other models also exist), governs the nonlinear mechanical response of vessels in which elastin recruitment occurs at low distension, whereas collagen recruitment primarily occurs at high distension. Current approaches to modelling of small vessels are limited by the assumption that the vessel wall is homogeneous, meaning that the internal mechanical environment is not well understood. This work involves the use of nonlinear microscopy to image the elastin and collagen networks in small vessels mounted on a pressure myograph. Figure 3 shows two-photon fluorescence (TPF) and second harmonic generation (SHG) images of a resistance artery at zero transmural pressure and 30 mm Hg. The distribution of collagen and elastin is very different from that in larger vessels, with elastin present largely in the intimal region and very little interpenetration of the two networks. Under pressure, the collagen network becomes more densely packed owing to radial expansion of the vessel as a whole, but it is not yet clear whether fibres straighten. The internal layer of elastin, initially parallel and longitudinally aligned, deforms to a more isotropic arrangement. Work is in progress to determine quantitatively the deformations and rearrangements in the fibrous networks, and establish the interactions of these structures with the smooth muscle of the medial zone and the mechanisms of tonic contraction.

Another fundamental question is why many tissues whose primary biomechanical function is dependent on dense arrays of collagen fibres also contain networks of elastin. In order to investigate potential mechanical roles of the elastin fibres in articular cartilage, we have used multiphoton microscopy to image the displacement and change in morphology of cells, collagen and elastin networks in viable, unstained cartilage subjected to applied tensile loads [15]. The reorientation of individual elastin fibres could be
tracked between images taken at progressive strains as shown in figure 4. The changes in fibre angle at each strain were compared with those predicted by a simple model assuming that the cartilage was a homogeneous elastic material in which the elastin fibres were embedded in the matrix so that they did not slip and transduced no load. As shown in figure 4, the behaviour of most fibres differed from the model predictions demonstrating that the fibres are not a passive component of the matrix. The fibres therefore move relative to the local matrix when the cartilage is loaded. We therefore hypothesize that the fibres, which are predominantly distributed in the superficial zone of the cartilage, have a role in determining the response to the shear forces to which the tissue is subjected as one articular surface slides over another during joint articulation, possibly by maintaining collagen fibres in register or assisting recovery from strain. If this is true, loss of elastin fibres may be a factor in the development of osteoarthritis.

The mechanical role of elastin fibres in the intervertebral disc is also attracting increasing attention. In the intervertebral disc, at least in the young, the nucleus is relatively fluid and compressive loads are transmitted directly to the annulus. Both the relatively sparse network of predominantly type II collagen fibres and the elastin are randomly arranged and although no specific connections between the two networks have been observed, it seems likely that the elastin acts to assist the recovery of the collagen network after unloading of the disc [5]. The annulus bulges under compressive load and under bending and torsion there is a complex pattern of collagen fibre reorientation and shear between adjacent lamellae. The distribution of elastic fibres into relatively coarse interlamellar connections and

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**Figure 3.** False-colour images of a viable human subcutaneous small resistance artery at neutral luminal pressure (a) and 30 mm Hg (b). SHG (blue) reveals collagen, and TPF (green) reveals autofluorescent proteins. Fibres seen in green are elastin. Increased luminal pressure causes the innermost layer of elastin to stretch radially, revealing the interconnectivity of the elastin fibres. The vessel is oriented at an angle to the imaging plane, so that the top of the images is in the adventitia, and the bottom passes through the lumen.

**Figure 4.** TPF images of equine articular cartilage at (a) 0%, (b) 4% and (c) 8% applied tensile strain. (d) An overlay showing elastin fibres which can be individually tracked between the images. (e) A histogram showing the differences between the predicted and measured elastin fibre angles at both 4% and 8% strain. The shaded area shows the limited range where the values agree within the uncertainty of the measurements. Fibre orientation data before and after the application of tensile strain are included in the electronic supplementary material.
finer intralamellar ones suggests that they play a key role in the rapid recovery of disc shape after isovolumetric loading [5]. Recent demonstrations of an association between changes in the structure of the elastin network and disc degeneration [16] indicate that further exploration of this hypothesis may be of more than academic interest.

4. Fibre and molecular mechanics

The contribution of elastin to tissue mechanics is clearly modulated both by the total elastin content and by the type of network that is formed. However, the complexity of these networks makes it difficult to relate their mechanical properties to the intrinsic mechanical properties of the elastin fibre. Many fundamental studies have therefore used the uniform fibres of ligament elastin, sometimes dissected down to the level of individual fibre to circumvent possible issues arising from fibre heterogeneity or slippage between fibres. Most studies report an elastic modulus in the range 0.3–1.2 MPa, generally increasing with strain, with a breaking strain of up to 200% [17,18]. The mechanism of strain-stiffening has not been discussed, to the best of our knowledge, but the modulus of individual fibres is substantially higher than those measured in networks. A significant recent development has been the application of atomic force microscopy to determine the mechanical properties of tropoelastin (the soluble form of elastin which is secreted by the cell) [19]. The molecule was found to act as a near perfect spring, showing minimal hysteresis loss. However, the reported elastic modulus of 3 kPa is much lower than that of the formed fibre. In many physiological situations, the dynamic properties of elastin are important, but these have been less extensively investigated in purified preparations. Cyclic loading experiments show that resilience of fibres falls rapidly at higher frequencies [20] but, as discussed below, the mechanism may be quite complex.

The problem of reconciling the mechanical properties of the individual molecule to those of the fibre is a challenging task because of the complexity of the internal structure of the elastin fibre. The importance of entropic mechanisms of elasticity, described below, led to analogies with natural rubber and fostered a belief that elastin forms random networks. However, the microscopic images challenge this presumption and X-ray scattering data also indicate some level of organization. Early studies on dehydrated fibres [21,22] did not show changes with strain; however, a more recent study on hydrated fibres using microbeam diffraction showed that the 0.45 and 0.9 nm spacings became fainter under strain, suggesting that the hydrophobic forces had been overcome [9]. Molecular modelling, reflecting the preponderance of rather small and mobile amino acid side chains, does not appear to support this hypothesis [26,27].

Although the experiments summarized above have provided fundamental insights into the micromechanics of elastin fibres, it is important to remember that the mechanically functional unit in tissue is the elastic fibre, comprising both elastin and microfibrillar glycoproteins.

The principal glycoprotein is fibrillin and its mechanical properties have been studied, both in the form of the fibril-rich zonular fibrils of the eye [28] and in the networks forming the major structural component of blood vessels in crustaceans [29]. The former measurements show highly nonlinear stress–strain characteristics with an incremental elastic modulus of approximately 500 kPa at 20% strain, although the effective modulus is an order of magnitude lower in the network of lobster aorta. This zonular fibre modulus is close to that of a single ligament elastin fibre. Measurements have been made on the mechanical properties of the elastic tissue isolated from large blood vessels, with and without the microfibrillar component [30,31], showing that the microfibrils contribute to the mechanical properties. We speculated that this arises because the microfibrils influence the realignment of the elastin under applied load or through their binding to and altering the physico-chemical properties of the elastin monomer. However, the demonstration of the similarity of mechanical properties of the two components indicates that a more direct mechanical function of the fibrillin is possible, and this is supported by recent measurements on the mechanics of ligament elastic fibres with and without glycoproteins [32]. Resolution of this question requires knowledge of the organization of the microfibrillar network and the strains to which it is exposed. The Raman spectrum of fibrillin shows peak shifts under large strain [33] which might provide a probe of local matrix strain. However, attempts to do this in our laboratory have failed to resolve any of the microfibrillar modes from elastin, which dominate the spectrum of porcine aortic elastic tissue [23].

Moving to molecular mechanisms of elasticity, the curiosity of a protein which displays long-range elasticity has attracted attention for over 50 years. Until recently, however, most experiments were conducted on elastin fibres where attempts to infer molecular mechanisms of elasticity are complicated by the uncertainties, already discussed, surrounding molecular organization within the fibres. Thermomechanical testing [34] and application of the Flory [35,36] theory of entropic elasticity reveal a significant component of entropy elasticity at higher temperatures, although at room or physiological temperatures its energetic contribution is less than 50% (figure 5). That the remainder comes from solvent interactions is demonstrated both by experiments on the mechanical effects of changing solution composition and by calorimetry. Early experiments using the former approach were compromised by failure to appreciate the partitioning of mixed solutions between the bulk medium and the intrafibrillar space. However, experiments with panels of probes of increasing hydrophobicity, for example primary alcohols, show a steady transition from
Elongation,  

are included in the electronic supplementary material. An increase in length. See [37] for further details. The raw thermoelasticity data  together with shifts in side chain modes, indicating changes in amide bands, indicative of changes in secondary structure, in lateral spacing [33]. Raman spectrometry reveals changes in range), there is an increase in axial periodicity and a decrease at higher strains (which may be beyond the physiological limit). As elastin fibres are stretched, there is an increase in entropic elasticity at higher temperatures, attributed to increased hydrogen bonding, though this remains to be experimentally verified. 

Elastin is a single gene-copy protein, but multiple isoforms can be produced by alternative splicing. Consistent differences in amino acid composition have been observed, both between tissues and between species. An interesting mechanical perspective on interspecies variation is provided by Gosline’s [25] comparison of aortic elastin between warm- and cold-blooded, where he argued that the differences in composition give rise to variations in glass transition temperature that are related to the normal operating temperature of the elastin. 

Sandberg [42] drew attention to variations in the content of hydroxyproline between tissues. It is highest in the very fine network of elastin fibres in the lung parenchyma, twofold lower in large blood vessels and another twofold lower in ligamentum nuchae, and Sandberg speculated that the more heavily hydroxylated elastin would be ‘stiffer’ because of increased hydrogen bonding, though this remains to be experimentally verified. 

We described above the existence of networks of fine elastin fibres in cartilaginous tissues. Although these fibres show the fluorescence, histological and immunohistochemical properties of elastin as well as stability against cyanogen bromide extraction, a number of groups have reported difficulty in recovering the recognized amino acid composition in ‘elastin’ extracted from cartilaginous tissues [5]. It is probable that this reflects the presence of contaminating protein fragments. However, it is striking that in some respects the amino acid sequences reported resemble lamprin. The lamprins are a family of proteins that can be isolated by cyanogen bromide digestion from lamprey cartilages [43]. Each of the lamprey matrix proteins is very similar in amino acid composition [44], but recent work in our laboratory has demonstrated that they differ significantly in mechanical properties (figure 6). Proteins extracted from branchial and pericardial cartilages closely resemble elastin in stress–strain behaviour, whereas those from annular and piston cartilages have nonlinear, but highly reproducible, stress–strain behaviour and notable hysteresis. The former resemble elastin in secondary structure, as determined by Raman spectrometry and form open-cell networks,
6. Mechanical changes in ageing and disease

Elastin is the most stable of the extracellular matrix molecules, with negligible synthesis after skeletal maturity. That lung and blood vessels maintain their function over 10^6 cycles of stretch and relaxation is therefore a significant mechanical performance. Nevertheless, loss of tissue elasticity is one of the hallmarks of ageing. It has long been recognized to be associated with fragmentation and thinning of elastin structures in skin and blood vessels and recent work in our laboratory shows that the fine fibres of cartilage elastin are broken in osteoarthritis.

Because of its longevity, elastin is susceptible to modification by chemical reactions that, for other matrix components, are negligibly slow, and a number of these appear to modify mechanical properties. Elastic tissue calcifies focally in atherosclerotic plaques and more extensively in rare conditions such as Monkenberg’s sclerosis, but there is still controversy about whether the interaction with calcium is electrostatic or involves binding to a neutral site [45]. Although there is now strong evidence that much of the interaction is purely electrostatic the latter mechanism is interesting in the context of mechanics, because it could influence the hydrophobicity of the molecule, and thereby change its mechanical properties. However, experiments in our laboratory using Raman spectrometry have so far failed to detect any changes in conformation or hydrophobicity following in vitro exposure to calcium salts at concentrations up to 2 M, and evidence of changes in mechanical properties has been obtained only at similarly high calcium concentrations [46]. By contrast, fibrillin contains extensive calcium binding domains, which influence its structure and mechanical properties [29]. The possibility that it is responsible for the pathophysiological observations merits consideration.

Elastic tissue generally contains a significant amount of lipid, particularly in the vicinity of atherosclerotic plaques [47]. The mechanisms of lipid binding are reported to involve interaction with the hydrophobic domains of the elastin molecule and to result in conformational changes [48]. It is possible therefore that lipid binding will modify the mechanical properties of elastin but, to the best of our knowledge, there has been no direct demonstration of this.

Diabetes gives rise to widespread circulatory complications which have stimulated investigations of the changes in elastin produced by reaction with high concentrations of glucose. Like collagen, elastin is susceptible to non-enzymatic glycation via the Amadori reaction. This results in changes both in the conformation of the elastin network resulting in an increase in zero-strain dimensions and an increase in its elastic modulus, either consequent on the conformational change or arising from the formation of additional cross-links. Studies of composite vascular networks containing both collagen and elastin showed that although both networks stiffened the collagen network contracted introducing differential strains in the matrix [49].

Diabetes is but one example of a wide range of clinical conditions characterized by high levels of oxidative stress, which may affect the mechanical properties of elastin in similar ways. Most of these conditions remain unexplored, but one situation that has received some attention is the effect of exposure to light and ionizing radiation. The specific effects of light, as distinct from age, on skin elastin have been extensively described [50]. The principle observation is fibre breakage associated with attack by free radicals, which results in skin deformation and loss of elasticity. Experiments on purified elastin exposed acutely to ionizing radiation have shown a decrease in elastic modulus [51]. This is consistent with fibre breakage in contrast to the cross-linking which occurs in collagen fibres. The latter leads to an increase in stiffness, and so it is clear that irradiation generates a complex pattern of change in the micromechanics of tissue.

7. Conclusion

Recent research has demonstrated elastin, generally in the form of networks of fine fibres, is a more widely distributed component of tissue than previously appreciated. This realization has largely come about because TPF microscopy has provided a more sensitive detection technique than classical histological methods. Multi-photon microscopy has also proven a valuable tool in informing mechanical models to determine the contribution of these networks to tissue micromechanics. Although much remains to be done, it is clear that the networks are mechanically functional and are implicated in the development of various pathological conditions.

The structure of the elastic fibre, or of the elastin component itself, and the molecular mechanisms of elasticity are long-standing questions, but important insights have been obtained from modern methods of microscopy and spectroscopy. We are now close to final resolution of long-standing controversies and obtaining a secure basis for analysis of changes in mechanical properties in diseases such as diabetes and atherosclerosis. Because of the complex composition of the extracellular matrix and the mechanical interactions between many of its components these questions must be addressed by means of micromechanical measurements. One specific challenge we alluded to was investigating the structural units of large blood vessels. In addition to characterizing the mechanics of the formed structure, there are questions relating to its formation. In each vessel, the same cells are responsible for its formation and maintenance, and presumably the differences in structure arise from the perception and response to site-specific chemical or mechanical signals.

Elastin is unique among mammalian proteins in possessing long-range elasticity. Its evolution was a slow process and we have discussed its similarities to some of its immediate precursors, the lamprey proteins. Other elastic proteins from even more primitive species such as byssal fibres used by molluscs to attach themselves to solid surfaces and ab ductin which forms a compression spring in the hinges of bivalves have with few exceptions [52–55] received little attention from the biomechanics community. An outstanding exception is the silk proteins which, although operating in a very different environment, show remarkable similarities in structure and properties and mechanics [56–59]. A powerful driver for research on silks has been the potential commercial exploitation of their remarkable mechanical properties. Similar promises have been made for synthetic constructs based on amino acid sequences found in elastin [60,61], and similar claims could be made for its evolutionary precursors, but much remains to be done.
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References


