Cells as Tensegrity Structures: Architectural Basis of the Cytoskeleton

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Mechanotransduction - the cellular response to mechanical stress - is governed by the cytoskeleton (CSK), a network composed of different types of biopolymers that mechanically stabilizes the cell and actively generates contractile forces. To carry out certain behaviors (e.g., crawling, spreading, division, invasion), cells must modify their CSK to become highly deformable, whereas in order to maintain their structural integrity when mechanically loaded, the CSK must behave like an elastic solid. Over two decades ago, a model of the cell based on tensegrity architectur was introduced. The model proposes that prestress in the CSK is critical for cell shape stability. Key to this model is the concept that this stabilizing tensile prestress results from a complementary force balance between multiple, discrete, molecular support elements, including microfilaments, intermediate filaments and microtubules in the CSK, as well as external adhesions to the extracellular matrix and to neighboring cells. In this chapter, we review progress in the area of cellular tensegrity, including the mechanistic basis of the tensegrity model, and development of theoretical formulations of this model that have led to multiple a priori predictions relating to cell mechanical behaviors which have been confirmed in experimental studies with living cells.

Keywords: prestress, stiffness, cytoskeleton, acin filments, microtubules.

1. INTRODUCTION

It is well established that mechanical distortion of cell shape can impact many cell behaviors, including motility, contractility, growth, differentiation and apoptosis [4, 10, 14, 29, 33, 34, 37, 39]. Mechanical stresses produce these changes in cell function by inducing restructuring of the CSK and thereby impacting cellular biochemistry [3, 19, 31, 48]. Through largely unknown mechanisms, these mechanical signals are transduced into biochemical signals that lead to changes gene expression and protein synthesis [24]. This process, known as mechanotransduction, is governed by the cytoskeleton (CSK), a network composed of filamentous biopolymers (actin microfilaments, microtubules, intermediate filaments) that mechanically stabilizes the cell and actively generates contractile forces. Because the cytoskeletal filaments can chemically depolymerize and repolymerize, it was assumed in the past that cells alter their mechanical properties through sol-gel transitions [28, 48, 50]. However, cells can change shape from round to highly spread without altering the total amount of cytoskeletal polymer filaments in the cell [32], suggesting that it is not chemical remodeling but rather physical changes in mechanical forces across the CSK that govern cell deformability. During the past decade, a growing body of evidence has shown that preexisting mechanical distending stress (or prestress) borne by the CSK is a key determinant of cell deformability [20, 35, 36, 45, 47, 56, 57]. This prestress results from the action of tensional forces carried primarily by actin microfilaments and, to a lesser extent, by intermediate filaments and is resisted by external adhesive tethers to the extracellular matrix (ECM), known as focal adhesions (FAs), and to other cells, as well as by other cytoskeletal filaments (e.g., microtubules) inside the cell.

Standard continuum mechanics models that depict the cell as an elastic or viscoelastic solid cannot describe the influence of cytoskeletal prestress on cell deformability. The reason is that those models *a priori* assume that cells possess intrinsic stiffness in a stress-free state and therefore do not require prestress to stabilize them. Moreover, most of the existing continuum-based models of cells are *ad hoc* descriptions based on measurements obtained under particular experimental conditions, and these continuum models usually ignore contributions of subcellular structures and molecular components. Over two decades ago, Ingber introduced tensegrity architecture as a model of cytoskeletal mechanics [25, 26]. A hallmark property of tensegrity structures is that their structural stability is provided by tensile prestress carried by their cable-like elements. Key to the Ingber's cellular tensegrity model is the concept that this stabilizing tensile prestress results from a complementary force balance between multiple, discrete, molecular support elements, including microfilaments, intermediate filaments and

microtubules in the CSK, as well as external adhesions to the ECM and to neighboring cells.

In this article, we review progress in the area of cellular tensegrity. We describe how the CSK and the ECM form a single, tensionally integrated, system and how distinct biopolymers of the CSK may bear either tensile or compressive loads inside the cell. The cellular tensegrity model is a useful description of cellular mechanics because it provides a mechanism to link mechanics to structure at the molecular level, in addition to helping to explain how mechanical signals are transduced into biochemical responses within living cells and tissues.

2. BASIC PRINCIPLES OF TENSEGRITY ARCHITECTURE

Tensegrity architecture is a building principle introduced by R. Buckminster Fuller [15]. It describes a class of discrete network structures that maintain their structural integrity because of tensile prestress in their cable-like structural members. Fuller referred to this architecture as "tensional integrity", shortly "tensegrity". Ordinary elastic materials (e.g., rubber, polymers, and metals) by contrast, require no such prestress. A hallmark property that stems from this feature is that structural rigidity (stiffness) of the network is nearly proportional to the level of the prestress that it carries [44, 54]. As distinct from other prestressed structures, in tensegrity architecture the prestress in the cable network is balanced by compression of internal elements called struts (Fig. 1).
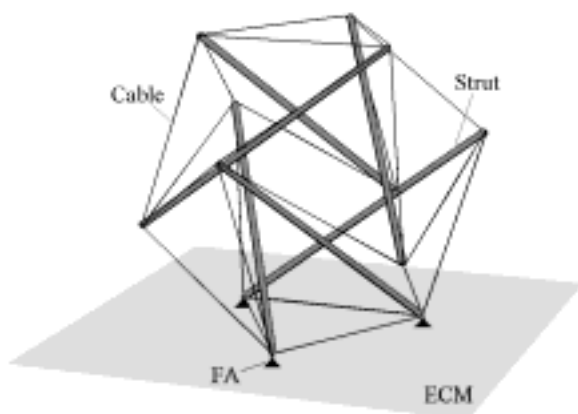

Figure 1. A simple tensegrity model composed of 24 tension supporting cables (black lines), which play the role of actin microfilaments, and 6 compression-supporting struts (gray bars), which play the role of microtubules. The cables carry pre-tension. The model is attached to the extracellular matrix (ECM) at three focal adhesions (FA) (black triangles). This particular model has been commonly used in the past as a conceptual model of cellular tensegrity [7, 43, 49, 54, 59].

In the Ingber's cellular tensegrity model, the CSK and the ECM are assumed to form a single, synergetic system mechanically stabilized by the cytoskeletal prestress. This prestress is generated actively by the cell's contractile machinery (molecular myosin motors), and passively by mechanical distension of the cell as it

adheres to the ECM and by swelling pressure of the cytoplasm. Two key premises of the model are: 1) that the prestress is primarily carried by the actin network and intermediate filaments, and 2) that this prestress is partly balanced by CSK-based microtubules and partly by FAs and other cells [23]. Thus, a disturbance of this complementary force balance would cause load transfer between these three distinct systems that would, in turn, affect cell deformability and alter stress-sensitive biochemical activities at the molecular level.

The central mechanism by which prestressed structures, including tensegrity architecture, develop restoring stress in the presence of external loading is primarily by geometrical rearrangement of their pre-tensed members. The greater the pre-tension carried by these members, the less geometrical rearrangement they undergo under an applied load, and thus, the less deformable (more rigid) the structure will be. This explains why the structural stiffness increases in proportion with the level of the prestress. To illustrate how these mechanisms arise from the cytoskeletal microstructure and how they predict various cell behaviors, we present in the following section a mathematical model of cellular tensegrity.

3. AN AFFINE TENSEGRITY MODEL OF THE CYTOSKELETON

The affine approximation effectively combines features of both continuum mechanics and discrete network modeling approaches, and allows developing a model of a complex structure without having to relay on a detailed description of microstructural geometry or boundary conditions. The key premise of the affine approximation is that microstructural strains are related to the global (continuum) strain according to the laws of continuum mechanics. This approach allows one to interpret mechanical properties of a discrete structure (such as the CSK) in terms of quantities that characterize a solid continuum (e.g., shear modulus). These quantities can be then used to study a particular boundary value problem in cellular mechanics using methods of continuum mechanics. Another advantage of the affine approach is that it yields explicit and mathematically transparent equations that describe various behaviors of cells and that can be easily implemented and experimentally tested. It does not require numerical and computationally-intensive calculations for obtaining those predictions.

The CSK of an isolated adherent cell is modeled as a network composed of tension-bearing cables interconnected with compression-supporting struts [41]. The cables and struts are perfectly elastic. The structure is anchored to a rigid substrate. The cables play the role of actin microfilaments and intermediate filaments whereas the struts play the role of microtubules. The cables carry pre-tension which is partly balanced by the compression of the struts, and partly by anchoring forces of the substrate. All junctions are assumed frictionless. The variational statement of equilibrium for the model is

$$\delta U = \sum_{i=1}^{N} F_i \delta l_i - \sum_{i=1}^{M} Q_i \delta L_i \qquad (1)$$

where U is the potential of external macroscopic (continuum) stress, F_i and l_i are current forces and lengths of the cables ($i = 1, 2, 3 \ldots N$), Q_i and L_i are current forces and lengths of the struts ($i = 1, 2, 3 \ldots M$), and N and M denote the total number of cables and struts in the network, respectively. The negative sign in (1) indicates compression. Because cables and struts are two-force members, F_i and Q_i depend only on l_i and L_i, respectively. The struts are slender and may buckle under compression. In that case, L_i indicates the end-to-end length (chord-length) of a strut. For mathematical simplicity, we will not make a distinction in the following derivation between cables that describe actin microfilaments and those that describe intermediate filaments because they have the same mathematical form. In other words, the first term on the right hand side (1) can be split into two sums, one referring to microfilaments and one referring to intermediate filaments.

3.1. Force Balance between Actin Microfilaments, Microtubules and the ECM

For uniform volume change, $U = \sigma V$, where σ is an isotropic macroscopic stress and V is the current volume of the CSK. Then, according to the affine assumption, all lengths change in proportion to $V^{1/3}$, i.e., $l_i \propto V^{1/3}$, and $L_i \propto V^{1/3}$. Thus, it follows from (1) that

$$\sigma = \sum_{i=1}^{N} \frac{F_i l_i}{3V} - \sum_{j=1}^{M} \frac{Q_j L_j}{3V} = \frac{N \langle Fl \rangle}{3V} - \frac{M \langle QL \rangle}{3V}, \qquad (2)$$

where $\langle \cdot \rangle$ indicates the average over all filament orientations. At the reference state, the first term on the right-hand side of (2) represents the prestress (P) borne by the actin microfilament and intermediate filament networks; the second term represents the part of P balanced by microtubules (P_{MT}). If $P > P_{MT}$, then σ on the left-hand side of (2) indicates the part of P balanced by the ECM (P_{ECM}). Thus, $P_{ECM} = P - P_{MT}$. Mechanical equilibrium of a section of the cell (i.e., free-body diagram) demands that mean traction (T) at the cell-ECM interface and P_{ECM} are balanced, i.e.,

$$TA' = P_{ECM} A'' = (P - P_{MT})A'', \qquad (3)$$

where A' and A'' are the interfacial and the cross-sectional areas of the cell section, respectively (Fig. 2). (Strictly speaking, (3) holds only when the cross-sectional surface A'' is perpendicular to the substrate and the force balance is in the direction of the normal to A''.) Importantly, all variables in (3) are measurable, which makes possible to evaluate individual contributions of actin and intermediate filaments, ECM and microtubules to force balance across the CSK. For

example, experimental data show that the contribution of microtubules (i.e. P_{MT}) to changes in T can vary from a few percent in highly spread cells (i.e. big A'), to up to 80% in poorly spread cells (i.e. small A') while P and A'' are maintained nearly constant [18].
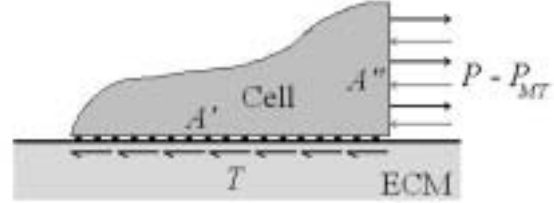

Figure 2. A free-body diagram of a section of the cell. Traction (T) (black semi-arrows) is balanced by the net prestress P_{ECM}, $TA' = P_{ECM}A''$, where A' and A'' are the interfacial and cross-sectional areas of the section, respectively. P_{ECM} equals the cytoskeletal prestress (P, black arrows) reduced by the portion balanced by microtubules (P_{MT}, gray arrows). ECM is the extracellular matrix; black dots are focal adhesions.

A key assumption of the tensegrity model is that microtubules carry compression as they balance tension in the actin network. This is qualitatively supported by microscopic visualizations of microtubules of living cells that show that microtubules buckle as they oppose contraction of the actin network [56, 58]. It is not known, however, whether the compression that causes this buckling could balance a substantial fraction of the contractile prestress. To investigate this possibility, we carried out an energetic analysis of buckling of microtubules [46]. The assumption was that energy stored in microtubules during compression is transferred to a flexible substrate upon disruption (i.e., chemical depolymerization) of microtubules. Thus, an increase in elastic energy of the substrate following disruption of microtubules should indicate transfer of compression energy that was stored in microtubules prior to their disruption. Experimental data show that in spread human airway smooth muscle cells that are optimally stimulated with contractile agonists (i.e., the cytoskeletal contractile prestress is maintained constant at its optimal level), disruption of microtubules causes the energy stored in the substrate to increase on average by ~0.13 pJ [46]. This result was then compared with results from a theoretical analysis based on the model of Brodland and Gordon [2], in which the microtubules are assumed to be slender elastic rods laterally supported by intermediate filaments. Using the post-buckling equilibrium theory of Euler struts [51], we estimated that the energy stored during buckling of microtubules is ~0.18 pJ, which is close to the measured value of ~0.13 pJ [46]. This is further evidence in support of the idea that microtubules are intracellular compression-bearing elements.

Taken together, the above results confirmed the existence of a complementary force balance between contractile forces carried by the actin network, compression in microtubules and traction forces that

arise at the FA anchoring to the ECM, as predicted by the tensegrity.

We next we use the affine model to show how prestress confers structural stiffness to the CSK.

3.2. Prestress induced stiffness of the CSK

We consider distortion of the CSK due to macroscopic (continuum) simple shear strain (γ). In that case, the potential $U = V_0 \tau \gamma$, where τ is the macroscopic shear stress corresponding to γ and V_0 is the reference volume occupied by the CSK. Thus it follows from (1) that

$$\tau = \frac{1}{V_0} \left(\sum_{i=1}^{N} F_i \frac{dl_i}{d\gamma} - \sum_{j=1}^{M} Q_j \frac{dL_j}{d\gamma} \right) =$$

$$\frac{1}{V_0} \left(N \left\langle F \frac{dl}{d\gamma} \right\rangle - M \left\langle Q \frac{dL}{d\gamma} \right\rangle \right). \quad (4)$$

Taking the derivative of (4) with respect to γ and evaluating it at the reference state (i.e., $\gamma = 0$), we obtain the shear modulus (G) as follows

$$G = \frac{d\tau}{d\gamma}\bigg|_{\gamma=0} = \frac{1}{V_0} \left[N \left\langle F \frac{d^2 l}{d\gamma^2} \right\rangle + N \left\langle \frac{dF}{dl} \left(\frac{dl}{d\gamma} \right)^2 \right\rangle - \right.$$

$$\left. - M \left\langle Q \frac{d^2 L}{d\gamma^2} \right\rangle - M \left\langle \frac{dQ}{dL} \left(\frac{dL}{d\gamma} \right)^2 \right\rangle \right]_{\gamma=0} \quad (5)$$

To obtain quantitative predictions for G and compare them to experimental data, we assumed a) the affine strain field and b) that at the reference state, all orientations of cables and struts are equally probable. The first assumption yields l and L as functions of γ, i.e.,

$$\frac{l}{l_0} = \frac{L}{L_0} = \left[(1+\gamma)^2 \sin^2\theta \cos^2\psi + \right.$$

$$\left. + (1-\gamma)^2 \sin^2\theta \sin^2\psi + \cos^2\theta \right]^{1/2} \quad (6)$$

where ψ and θ are azimuth and latitude angles of the spherical coordinates, and l_0 and L_0 are reference lengths of cables and struts, respectively. The second assumption was used to calculate the average values, i.e.,

$$\langle f \rangle = \frac{1}{2\pi} \int_0^{2\pi} \int_0^{\pi/2} f(\theta,\psi) \sin\theta \, d\theta \, d\psi , \quad (7)$$

where f is any function. By combining (2) and (5)-(7), we obtained that (for detailed derivation see [41])

$$G = 0.8(P - P_{MT}) + 0.2(BP - B_{MT}P_{MT}), \quad (8)$$

where $P = NF_0 l_0 / 3V_0$, $P_{MT} = MQ_0 L_0 / 3V_0$, $B = (dF/dl)_0 / (F_0/l_0)$, $B_{MT} = (dQ/dL)_0 / (Q_0/L_0)$, and subscript 0 indicates the reference state. The non-dimensional cable stiffness B was determined from tensile tests of isolated acto-myosin filament

interactions [27]; $B \approx 2.4$ for a wide range of tensile force. The non-dimensional strut stiffness B_{MT} was determined from buckling behavior of microtubules. We found experimentally that in highly spread cells $P_{MT} \approx 0.12P$ and the corresponding $B_{MT} \approx -0.7$ [41]. Substituting the above values for P_{MT}, B and B_{MT} into (8), we obtained that $G \approx 1.2P$. This prediction is consistent with our previously reported experimental data from cultured, highly spread airway smooth muscle cells [57] (Fig. 3).
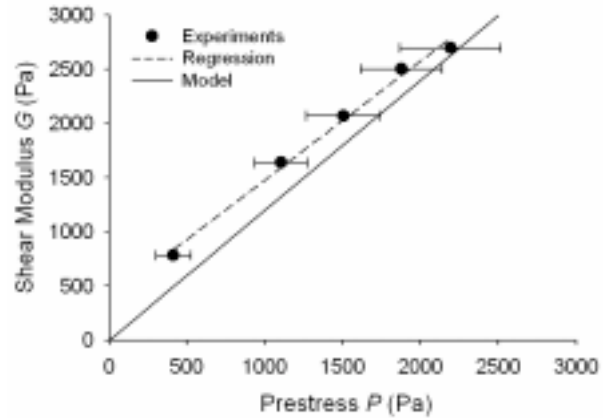
Figure 3. Cell shear modulus (G) increases linearly with increasing cytoskeletal prestress (P). Measurements were done in cultured human airway smooth muscle cells whose contractility was modulated by graded doses of histamine (constrictor) and isoproterenol (relaxant). G was measured using the magnetic cytometry technique [13] and P was measured by the traction cytometry technique [57]. Dots are data ±SE; the slope of the regression line is ~1.1 (dashed line). The affine tensegrity model predicts a slope of ~1.2 (solid line).

The model also predicts how microtubules contribute to the overall stability of the CSK. Since microtubules participate in balancing the overall contractile stress, their disruption would alter this balance and thus affect cytoskeletal shape stability. Experimental data from cultured adherent cells show that disruption of microtubules causes either cell softening [38, 55], stiffening [45, 60], or no change in stiffness [9, 52]. Using (8) and experimental data for the contribution of microtubules to the traction at the cell-ECM interface [18], we found that in highly spread cells stiffness slightly increases in response to disruption of microtubules whereas in poorly spread cells the stiffness decreases with disruption of microtubules [41]. This finding is quantitatively consistent with experimental data from cultured human airway smooth muscle cells which show that disruption of microtubules by colchicine causes a 10% increase in cell stiffness [45] whereas the model predicts an 8% increase [41].

One limitation of the affine approach is the assumption that local strains follow global strains, which leads to overestimate of elastic moduli [cf. 40]. Since it is not very likely that local strains of the CSK follow global strains applied to the cell, one should expect that the affine model predicts higher values of the elastic moduli than the measured ones. This can, in

part explain, the quantitative discrepancy between the slopes of the *G* vs. *P* relationship predicted by the model and the one calculated from the experimental data (solid vs. dashed lines in Fig. 6). Furthermore, this model cannot predict long-distance propagation of forces in the cytoplasm that has been observed in living cells [17, 31], since the model presumes a continuum behavior which, in turn, implies that local loads produce only local deformations (in continuum mechanics this is known as the principle of local action). Nevertheless, the affine model has been successful in describing and predicting a number of essential mechanical properties of living cells such as prestress induced stiffening, the contribution of microtubules to cell stiffness, and the load shift between the CSK and the ECM [41].

4. OTHER PESSTRESSED MODELS OF CELLULAR MECHANICS

There are other models in the literature that consider the effect of the prestress on cell deformability, most notably models based on a cortical membrane network [6,11], and a tensed cable network [42]. The former assumes that the prestress is carried by a thin cortical membrane that encloses pressurized liquid cytoplasm. The latter depicts the CSK as a network composed only of prestressed tension-bearing elements. While these models have been successful at explaining some particular aspects of cellular mechanics, they fail short of describing many other mechanical behaviors that are important for cell function. In particular, the cortical membrane network model ignores the contribution of the ECM to cellular mechanics, and cannot explain the observed transmission of mechanical signals from cell surface to the nucleus as well as to basal FAs [17, 31, and 56]. The tensed cable model ignores the role of compression-supporting microtubules [42].

On the other hand, all of these features (and many others) can be explained by the cellular tensegrity model [cf. 22, 23, 44]. Moreover, none of the other models provide a mechanism to explain how mechanical stresses applied to the cell surface result in force-dependent changes in biochemistry at discrete sites inside the cell (e.g., FAs, nuclear membrane, microtubules), whereas tensegrity can [24]. Thus, we believe that the cellular tensegrity model represents a good platform for further research on the cell structure-function relationship and mechanotransduction.

It is important to clarify that according to a mathematical definition of tensegrity that is based on considerations of structural stability [cf. 5], the cortical membrane model and the tensed cable network model also fall in the category of tensegrity structures. They differ only by the manner in which they balance the prestress. However, in the structural mechanics literature, this difference is used to make a distinction between various types of prestressed structures and consequently, tensed cable nets and tensegrity architecture are considered as two distinct types of structures [53]. Although this may be understandable from a theoretical modeling standpoint, a self-stabilized cable net (i.e., the one that is not attached to an external

world) cannot be 'tensed' unless it contains at least one compression element that balances these internal forces; hence, the more general definition of tensegrity may be relevant for describing real, three-dimensional structures in the living world.

5. TENSEGRITY AND CYTOSKELETAL RHEOLOGY AND FUTURE DIRECTIONS

During the past decade, biomechanical studies of the cell have been focused on its rheological behavior. This is important since the CSK is a dynamic system which undergoes continuous remodeling and in its natural habitat it is exposed to dynamic loads. Rheological studies on various cell types and with various techniques yielded two distinct features: 1) that rheological behaviors conform to a power law in both time (t^α) and frequency (ω^α) domains ($0 < \alpha < 1$); and 2) that this power law is influenced by cytoskeletal prestress [1, 13, 36, 47]. In particular, it has been observed that a power-law exponent (α) decreases with increasing cytoskeletal prestress. Since the power-law behavior is directly related to deformability, (i.e., when $\alpha \to 0$ or $\alpha \to 1$ we have a solid-like or fluid-like behaviors, respectively), then the observations suggest that cells use mechanical prestress to regulate their transition between a solid-like and a malleable behaviors [47]. This was quite a surprising finding since a standard paradigm was that this transition is regulated by chemical mechanisms that govern polymerization and depolymerization of the CSK [48].

A number of empirical and semi-empirical mathematically sophisticated models have been offered to explain these rheological behaviors of living cells [3, 12, 13, 30]. All these models could provide explanations and descriptions for the power-law behavior. However, none can explain the observed dependence of the cell rheological behavior on cytoskeletal prestress. Importantly, these models have no structural correlates in living cells, and thus cannot predict how specific cytoskeletal structural alterations (e.g., reorientation and rearrangement) might be related to cellular mechanical behaviors. To address this problem, we proposed a viscoelastic model based on tensegrity [49]; in a tensegrity model of the type shown in Fig. 1, elastic cables were replaced by simple Voigt spring-dashpot units. We showed that this model can account for the prestress-dependent rheological behavior of the cell. However, in order to explain the power-law behavior observed in cells, we had to assume *ad hoc* a very high degree of non-homogeneity between structural element properties in order to provide a wide spectrum of time constants that leads to the power-law.

To provide a mechanistic explanation for the observed rheological behavior of living cells, we recently initiated an investigation that would link the cytoskeletal prestress to molecular dynamics of polymers of the CSK. Our rationale is as follows. The rheological behavior of the CSK must necessarily reflect dynamics of polymer chains of the CSK. The dynamics of long chain molecules is characterized by

thermally driven fluctuations that result in a power law-like rheological behavior [8, 21]. However in the living CSK, polymer chains are under tension due to prestress forces. This tension, in turn, should impact cytoskeletal polymer dynamics in such a way that thermally-driven fluctuations diminish with increasing tension. This would push the cytoskeletal rheology closer to the solid-like behavior and thus, the power-law dependence should diminish. Our preliminary statistical models of fluctuating polymer chains under sustained tension yielded behaviors that are qualitatively consistent with the observations in living cells. This leads us to believe that this approach provides a good physical basis to explain how cytoskeletal prestress may affect the rheology of molecules within the CSK, and how these molecular scale features feed back to alter the mechanical properties of the entire cell through the unifying mechanism of cellular tensegrity.

It is well known that living cells exhibit significant regional differences in mechanical stiffness [16]. However, since the whole cell responds to an external mechanical stimulus as an integrated unit, these local units within the cytoplasm must be mechanically connected via the CSK, possibly via the prestress-bearing elements. A more comprehensive analytical tensegrity model needs to be developed in order to capture the behavior of mechanical heterogeneity and anisotropy observed in living cells [17, 19].

6. SUMMARY

In this chapter, we have shown that the tensegrity model is a useful framework for studying mechanics of living adherent cells. The model identifies mechanical prestress borne by the CSK as a key determinant of shape stability within living cells and tissues. It also shows how mechanical interactions between the CSK and ECM come into play in the control of various cellular functions. Furthermore, the model provides a way to channel mechanical forces in distinct patterns, to shift them between different load-bearing elements in the CSK and ECM, and to focus them on particular sites where biochemical remodeling may take place. If successful, this approach may show the extent to which prestress plays a unifying role in terms of both determining cell rheological behavior, and orchestrating mechanical and chemical responses within living cells. Moreover, it will elucidate potential mechanisms that link cell rheology to the mechanical prestress of the CSK, from the level of molecular dynamics and biochemical remodeling events, to the level of whole cell mechanics.

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ЋЕЛИЈЕ КАО ТЕНЗЕГРИТИ СТРУКТУРЕ: АРХИТЕКТОНСКА ОСНОВА ЦИТОСКЕЛЕТА

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Механо-хемијски пренос – ћелијски одговор на механичке напоне – одвија се преко унутарћелијске биополимерне мреже познате као цитоскелет, која обезбеђује механичку стабилност ћелије и производи затезне силе. Да би обезбедио нормално функционисање ћелије, цитоскелет мора да прилагођава своју деформабилност биолошким захтевима. С једне стране, у току кретања, ширења и деобе, ћелија мора да буде веома деформабилна, готово као флуид. С друге стране, да би одржала структурни интегритет под дејством механичких напрезања, ћелија мора да се понаша као чврсто еластично тело. Пре више од две деценије, појавио се у литературе модел цитоскелета заснован на тензегрити архитектуру. У моделу се сматра да је механички преднапон, који карактерише тензегрити структуре, чинилац који одређује и регулише деформабилност цитоскелета, Онсновна премисе модела је да цитоскелетни преднапон настаје кроз равнотежу и пренос механицких сила измеdjу биополимерних влакана цитоскелета (актинских микровлакана, микротубула и средњих влакана) и спољашњих адхезионих тачака којима је ћелија везана за екстрацелуларну матрицу и суседне ћелије. У овом раду дат је преглед развоја у истраживању и примени тензегрити архитектуре у ћелијској биомеханици, укључујући механистичку основу тензегрити модела, као и илустративни пример којим се приказују главне особине и предвиђања модела и њихово поређење са резултатима добијеним из експерименаталних мерења на живим ћелијама.