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Modeling cytoskeletal and cell dynamics

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Here we give an overview of recent theoretical and experimental work on modeling the mechanics and dynamics of the cytoskeleton. The cytoskeleton is a multicomponent, complex and active material that is essential to cell mechanics and dynamics. We focus on one of the main components of this material, namely actin filaments. We discuss these filaments and their interactions with other proteins within the cytoskeleton. To fully understand the cytoskeleton, it is important to consider both theoretical and experimental work in calculo, in silico, in vitro, in vivo, and in situ. We review the current state of knowledge and look forward to further work to come on aspects not yet understood.

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Introduction

The cytoskeleton, made up of protein filaments and numerous regulatory proteins, is crucial for cell dynamics. It is required for cell division, migration, adhesion, and many more cellular processes [1]. In order to understand how the cytoskeleton controls cell mechanics and dynamics, we need a combination of modeling and experiments. Much work has been done on both approaches, but the multicomponent, complex cytoskeleton is still beyond our full comprehension. We now have a number of excellent models, but experimental observations, especially in living cells, reveal gaps in our ability to connect models to experiments. In this work, we review what is known, highlight exciting new work, and outline remaining open questions.

In the following, we consider the cytoskeleton, starting with actin only and building up its components section by section. We consider what has been found using analytical 'pen and paper' mathematical models (in calculo), computational simulations (in silico), experiments on components (in vitro), experiments in cells (in vivo or in cellulo) and finally, experiments in environments such as tissues (in situ) (Figure 1). In our opinion, all these different methods of study are necessary to gain a full understanding of how the cytoskeleton controls and affects cell dynamics.

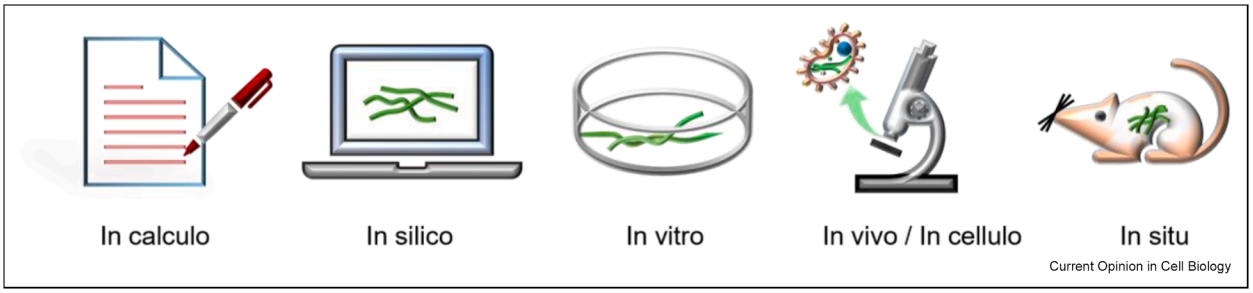
Actin

G-actin (globular actin) proteins polymerize to form filaments of F-actin (filamentous actin), with a thickness around 8 nm. This is an active process consuming energy provided by the hydrolysis of adenosine triphosphate (ATP). Cells are maintained out of equilibrium with an excess of ATP driving the polymerization of actin. The reverse process of depolymerization, disassembling filaments, also occurs in cells. The polymerization and depolymerization of actin filaments are biochemical reactions that happen stochastically, due to the underlying random nature of the Brownian motion of the molecules involved. In vitro, overall rates of polymerization and depolymerization of actin can be controlled by salt and ATP concentrations. We owe much of our understanding of this to the careful work of Pollard [2].

New, in vitro, work shows the effect on actin dynamics of a limited pool of actin monomers [3]. In this and many in vitro experiments, the issue of aging in actin monomers still remains a considerable unknown [3] and is therefore usually ignored in modeling.

Numerous actin-binding proteins regulate the (de) polymerization of actin filaments by activating nucleation of new filaments (formins) or branches (Arp2/3), and by activating/inhibiting (de)polymerization,

Figure 1



Schematic representation of methods to model and study the cytoskeleton and cell dynamics.

capping to prevent further dynamics at a tip, severing to cut a filament etc. [4–6] (Figure 2).

The Brownian ratchet model [7] elegantly explains the way cells can harness the process of active polymerization of actin to exert forces on cellular components, such as the lipid membrane. This can result in diverse dynamics such as deformations of the membrane (e.g. lamellipodium, filopodia, phagocytosis) or the beautiful transport mechanism used by Listeria, which can be reconstituted on beads [3].

Computational modeling of actin (de)polymerization often uses Monte Carlo methods to simulate polymerization and depolymerization of filaments as a stochastic process [8,9]. The package Cytosim [10], originally developed for microtubules, is now also widely used for actin [11].

Electron or optical microscopy with fluorescent actin can now trace the dynamics of individual filaments, and image analysis software that can automatically track filaments is available [12]. Once individual filaments meet, cross each other, or branch and begin to form a network, tracking becomes increasingly difficult. Improvements to super-resolution microscopy and image analysis will greatly enhance our knowledge of the structure of actin networks [13].

Actin filaments have a persistence length of order of 20 μm, meaning that on the scale of a cell they are semiflexible polymers. Much work [14] has been done in the field of polymer physics on the properties of networks of semiflexible polymers, with actin often used as an example. An important property of the structure of networks is their mesh size, which greatly affects their mechanical properties. In addition to the mechanical properties arising from the mesh size and semiflexible nature of the filaments, actin networks can be dynamic due to (de)polymerization. This means that over timescales of minutes they 'turnover' [2] i.e. filaments depolymerize and repolymerize in ways that can relax stresses and change the structure of the network [15].

Crosslinked actin

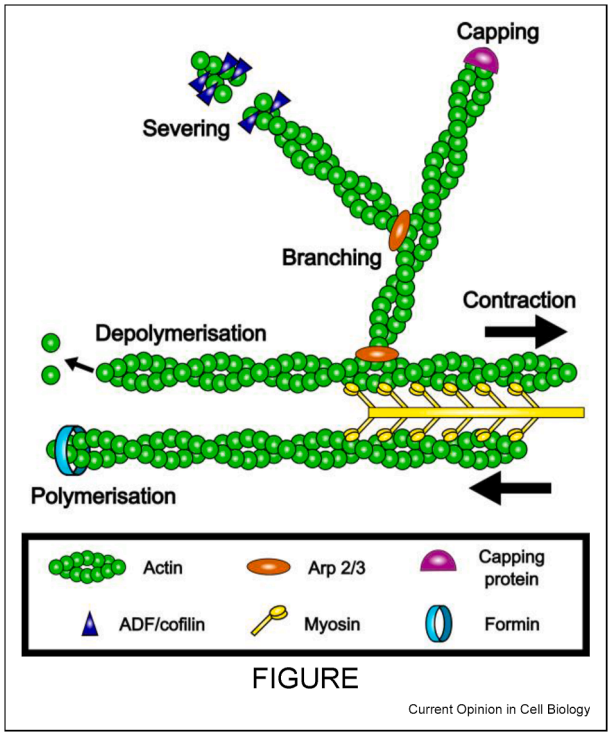
As well as actin filaments interacting sterically with each other, they can also be chemically crosslinked by actin-binding proteins. Actin filaments can be bound together in bundles [16], increasing their overall persistence length (rigidity) and force-generating power. Filaments can also be crosslinked to form an isotropic network. The mechanical properties of crosslinked networks are different from those that are not crosslinked. In particular, they are more rigid [17,18]. Actin filaments are dynamic, and so are the crosslinks in actin networks. Crosslinking proteins bind and unbind on particular timescales, allowing relaxation of stresses and changes to the structure of the network. Recent theoretical work has shown that active binding and unbinding of crosslinks to semiflexible polymers can generate contractility [19]. The balance of crosslinks and bundles within an actin network greatly affects the mechanical properties of the network [17,18]. Recently, it has become possible to begin to test the theory of these mechanical and structural properties in cells in vivo [12] using various experimental methods and models [1] (Figure 3).

However, there is much more happening in the cytoskeleton of cells than just a crosslinked semiflexible filament network with bundles. In particular, the action of myosin molecular motors transforms the material into an active gel with mechanical properties and dynamics beyond those possible in a passive material.

Actomyosin

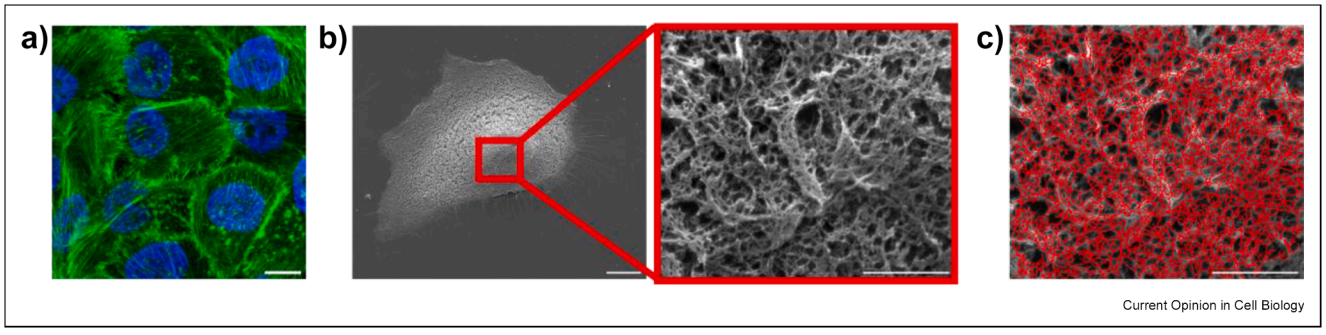
Myosin is a family of proteins that bind to actin. Myosins can act as passive crosslinkers, but can also act as active molecular motors. They can bind to actin filaments and move along them in a directional manner, sensing the direction inherent in the actin filaments. Some molecular motors are completely nonprocessive, which means

Figure 2



Schematic representation of actin and actin binding proteins.

Figure 3



Actin organization in cells. **a)** Fluorescence image of MCF 10 A cells. Actin is stained green, nucleus blue. The scale bar corresponds to 10 μm. **b)** Scanning electron microscopy image of a MCF 10 A cell from which the cell membrane has been extracted. The scale bar corresponds to 10 μm. Red inset: zoom on one area of the actin cortex. Scale bar represents: 1 μm. **c)** Outline of the actin cortex meshes in **b)**, generated with [20].

that they perform a single step along a filament before falling off. Others are very processive, performing many steps before unbinding from the filament. Myosin molecular motors often cluster together, forming 'mini-filaments' and can simultaneously bind to more than one actin filament in a network. If myosins are bound to two parallel actin filaments in a bundle, they can move along the bundle without causing stress to the actin network. However, when myosin binds to two filaments in opposite directions, the motors try to move in opposite directions, causing stress in the system. Bundles of antiparallel filaments with myosin bound are found in cell structures known as stress fibers, with the action of the myosin exerting contractile stress on the structure. A classical example of actomyosin contraction is in muscle sarcomeres. Recent molecular dynamics

simulations have provided insight into force generation at a submolecular level [21].

In an isotropic network, mini-filaments of myosin can bind to actin filaments in different directions, and this also causes contraction in the system. There has been some interesting theoretical work done on why, in practice, actomyosin networks are seen to be contractile not extensile [22]. Working with actomyosin in vitro is challenging. Meticulous work over the past couple of decades has developed experimental protocols, which are now routinely used to control and study in vitro actomyosin networks. In vivo experiments are harder to decipher due to our lack of knowledge of all the components and the interactions between actomyosin networks and their cellular surroundings. In cells, most actomyosin is positioned around the edge of the cell in what is known as the actin cortex, on the inside of the cell membrane [1,23].

Active gel or active matter theory at the continuum hydrodynamics level has been remarkably successful in describing the behavior of contractile actomyosin networks [24,25]. Recent applications of active gel theory include several studies [26–29]. The theory has been applied to a number of important cell processes including cell migration [30]. It is also used at a larger length scale for multicellular systems such as tissues [26]. The early theoretical work has led to a new field of biophysics known as active matter, which now has its own conferences and active research communities. This is a fruitful area of study with fundamental questions and numerous applications open to exploration.

Most applications of active gel theory focus on long time scales in the fluid limit to study cell migration, tissue dynamics etc. However, some work has focused on length and timescales in which the actin cytoskeleton acts as an active solid [31]. The nature of the full viscoelastic behavior of actomyosin remains a key open question. There are many possible models of viscoelasticity, and it is far from obvious which are most appropriate for the behavior of actomyosin, the actin cortex, the cytoskeleton, or tissues. Some work uses linear viscoelastic models such as the Maxwell or Kelvin-Voigt models, or combinations thereof [24,26,32]. Many studies describe the material as glassy (see the recent review [33]).

A new development consists of attempts to use machine learning methods to elucidate aspects of the cytoskeleton and cell dynamics [34–36].

In vivo experiments give a plethora of different results that can be described by a number of different models. Some experimental techniques, such as Atomic Force Microscopy (AFM), give results that are notoriously difficult to analyze and are model-dependent, often using overly simplified models that can give wildly different values for elastic parameters [37]. Experiments often do not agree with measurements using different techniques, and different cell types appear to have very different mechanical properties, further complicating comparisons [38]. Inhomogeneities and anisotropic properties of the actin cytoskeleton are additional complexities in measuring mechanical properties [12,39]. We expect the large amount of current work in this area will reveal important insights into understanding this complex material over the coming years.

Microtubules

Another important filament that makes up the cytoskeleton is microtubules. Like actin, microtubules are made up of protein subunits that polymerize and depolymerize using, in this case, Guanosine-5'-triphosphate (GTP) hydrolysis as their biochemical fuel. However, microtubules are considerably larger (25 nm diameter) and stiffer, with persistence length extending into the millimeter range. Microtubules and actin have been widely studied separately, but comparatively little is known about their interactions and joint action. In vitro experiments tend to be on a larger length scale than that of a cell. In vivo, it is known that there are proteins that connect microtubules and actin, as well as the physical structural connections. The importance of microtubules and other filaments interacting with or penetrating the actin cortex is increasingly being recognized [40–44] and modeled, for example, with a tensegrity model [37]. Much remains to be discovered as to how these cytoskeleton components work together in cells.

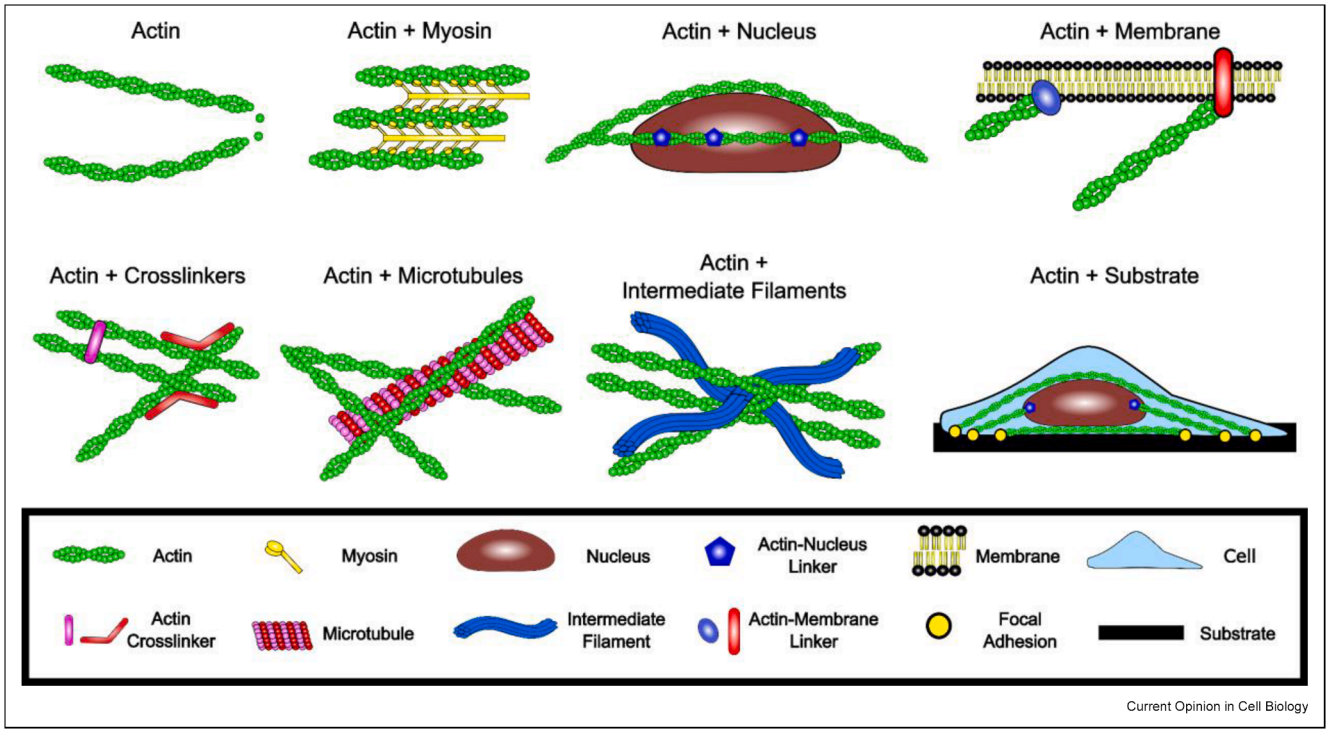
Intermediate filaments

Traditionally, intermediate filaments have been somewhat neglected compared to actin and microtubules. Unlike actin and microtubules, intermediate filaments do not actively turn over and are not known to interact with molecular motors, and are therefore passive filaments. However, their ubiquitous presence in vivo forms an important part of the mechanical properties of the cytoskeleton. An increasing amount of work is being done on these filaments, e.g. vimentin, and on their important effects on cell mechanics [45–48]. However, there is a large amount of work to be done to understand the role of these filaments in the cytoskeleton and how they interact with actin and microtubules.

Interactions with the nucleus

Interactions between the cytoskeleton and the nucleus is an area of increasing interest [49]. There are proteins that chemically connect cytoskeleton filaments to the nuclear membrane and the nuclear lamina on the inner side of the nuclear membrane. Models have shown how the cytoskeleton can exert mechanical forces to affect

Figure 4



Schematic representation of the interactions of actin with linker proteins, molecular motors, and cell organelles.

the position and shape of the nucleus [50,51]. However, there remains a large amount still to be done, especially in connection with in vivo experimental work. How mechanical forces transmitted by the cytoskeleton to the nucleus affect gene expression is a fascinating area for future work.

Interactions with the cell membrane

The actin cortex is closely associated with the cell membrane [52]. In some in vivo experiments, it can be difficult to disentangle the two, with results quoted for membrane tension being dominated by the tension of the actin cortex rather than the lipid membrane. Some models assume a composite of the membrane and cortex as an elastic sheet [15]. More detailed models of the interaction between the actin cortex and the membrane allow for the study of processes such as blebbing, in which the connection is broken and then reformed [53], and phagocytosis, in which large membrane deformations occur [54].

Experiments on actin reconstituted in Giant Unilamellar Vesicles (GUVs), liposomes, or on supported lipid bilayers are now contributing to understanding actin-membrane interactions [53,55–58].

Adhesion with substrates and other cells

The cytoskeleton is crucial for adhesion with substrates and with other cells. Cells on rigid substrates form stress fibers connected to the substrate by focal adhesions, a machinery of proteins that is mechanosensitive and has long been an area of interest for biophysicists. Models such as the molecular clutch or catch bonds [18,59,60] have been influential in understanding mechanosensitivity. The place of such substrate stiffness sensing in situ is a largely unexplored landscape [61]. Fascinating work on this in the brain is being undertaken by Ref. [62].

Adhesion between cells in tissues is of clear importance in development, life, and disease, such as cancer. Via adhesion between cells, large multicellular mechanical connections can be set up, controlling mechanics and dynamics of whole organs. Active matter theory can be applied to the tissue level [63,64].

Another model that has been remarkably successful in describing such multicellular tissues is the surprisingly simple vertex model, in which vertices are connected together in polygons with area and perimeter constraints [65,66].

Conclusion

In this article we have briefly overviewed many aspects of the cytoskeleton and raised open questions for current and future research. A common feature of these questions is the challenge of bridging the gap between theoretical modeling and experiments. This is essential for experimental data analysis and interpretation, and for testing our theoretical understanding. As we move from in vitro to in vivo or in situ experiments and from observations to biophysical measurements, this challenge becomes harder. Tackling this will bring us closer to answering the overarching question of how the mechanical and dynamical behavior of the cytoskeleton affects its interactions with its cellular environment. The future prospects for the field are bright due to advancing technology for biophysics experiments and developments in models and data analysis techniques. The increasing recognition of the importance of interdisciplinary collaborations between theory and experiments will bring deep insights to the field.

In conclusion, we now have many building blocks (Figure 4) of understanding from in calculo and in silico modeling, combined with in vitro experiments. These are just beginning to be brought together to discover the inner workings of the cytoskeleton in vivo and in situ. The coming years are full of promise for understanding this essential part of living organisms.

Credit author statement

Christoph Anton: Investigation; Writing – original draft; Visualization.

Franziska Lautenschläger: Conceptualization; Project administration; Supervision; Writing – review & editing.

Rhoda J. Hawkins:

Conceptualization; Project administration; Supervision; Writing – original draft; Writing – review & editing.

Declaration of competing interest

There are no competing interests to disclose.

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Data availability

No data was used for the research described in the article.

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