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Current Opinion in Genetics & Development

# Regulation of tissue morphodynamics: an important role for actomyosin contractility

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Forces arising from contractile actomyosin filaments help shape tissue form during morphogenesis. Developmental events that result from actomyosin contractility include tissue elongation, bending, budding, and collective migration. Here, we highlight recent insights into these morphogenetic processes from the perspective of actomyosin contractility as a key regulator. Emphasis is placed on a range of results obtained through live imaging, culture, and computational methods. Combining these approaches in the future has the potential to generate a robust, quantitative understanding of tissue morphodynamics.

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**Current Opinion in Genetics & Development** 2015, **32**:80–85

This review comes from a themed issue on **Developmental mechanisms, patterning and organogenesis**

Edited by **Deborah J Andrew** and **Deborah Yelon**

http://dx.doi.org/10.1016/j.gde.2015.01.002



## Introduction

The generation of tissue form during morphogenesis is coordinated across multiple length scales, from molecular interactions to cell-level deformations to tissue-level changes in shape. These length scales are often bridged by the transmission of cytoskeletal tension, which arises from the movement of myosin motors along actin filaments (Figure 1a). At the molecular scale, this actomyosin contractility is regulated by several signaling pathways, including those downstream of Rho kinase (ROCK) and myosin light chain kinase (MLCK). When forces produced by locally-activated actomyosin are transmitted along greater length scales *via* junctional domains, a cell can do work on or move within its surrounding tissue. For example, planar polarized contractility can instruct convergent extension (Figure 1b), whereas actomyosin contractility localized to apical cellular surfaces drives apical constriction (Figure 1c). Proteins that are sensitive to local stresses and strains (*e.g.* stretch-activated ion channels and other mechanosensing proteins) can also convert mechanical information back into molecular instructions (reviewed in [1]). In this way, actomyosin contractility regulates and can be regulated by tissue morphogenesis.

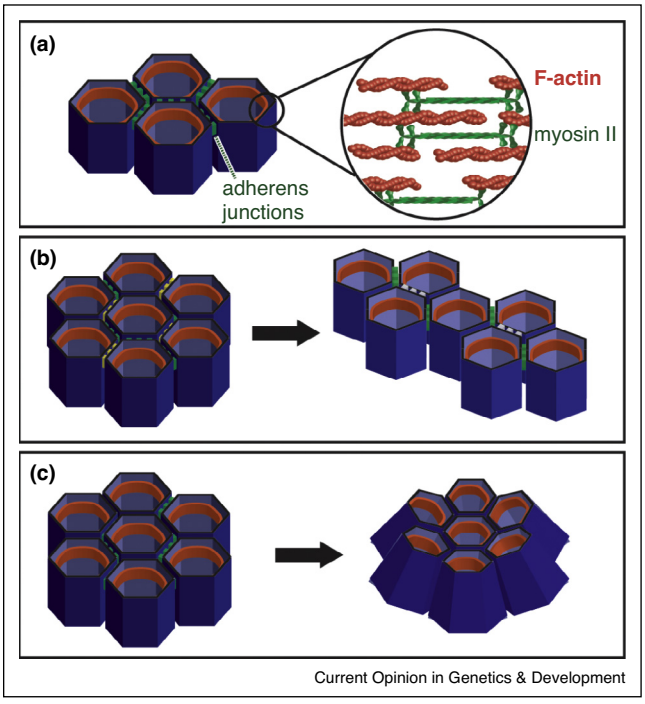
Given the architectural complexity of native epithelial tissues, protein signaling, and cellular biophysics, quantitative information about morphogenesis has been gleaned from a combination of *in vivo* imaging, experiments in culture, and computational modeling. Here, we review recent work that has used these techniques to elucidate the mechanics and dynamics of tissue morphogenesis, from the perspective of actomyosin contractility as a key regulator of these processes. The interested reader is directed to more comprehensive reviews describing the forces in morphogenetic patterning [2], contractility-induced changes in tissue shape [3,4], and epithelial junctional dynamics [5]. Combining computational, culture, and *in vivo* methods in the future will be important for generating a more robust, quantitative understanding of tissue morphodynamics.

## Actomyosin contractility tissue elongation

Cell intercalation (reviewed in [6]) is a morphogenetic process in which cells remodel their intercellular contacts to significantly rearrange their relative positions within an array of neighboring cells. One of the most well-studied types of intercalation in the context of tissue morphogenesis, convergent extension, occurs when the tissue lengthens along one axis while simultaneously narrowing along the perpendicular axis (Figure 1b), a process requiring asymmetric distributions of force along cell boundaries. During *Drosophila* germband extension (Figure 2a), for example, planar polarized actomyosin contractility shortens dorsoventral junctions to drive intercalation in the anteroposterior direction [7]. Planar cell polarity (PCP) proteins within *Xenopus Laevis* mesoderm also direct convert extension by compartmentalizing cortical contractility along mediolaterally-aligned junctional domains [8] and so it is likely that similar mechanisms occur in vertebrates.

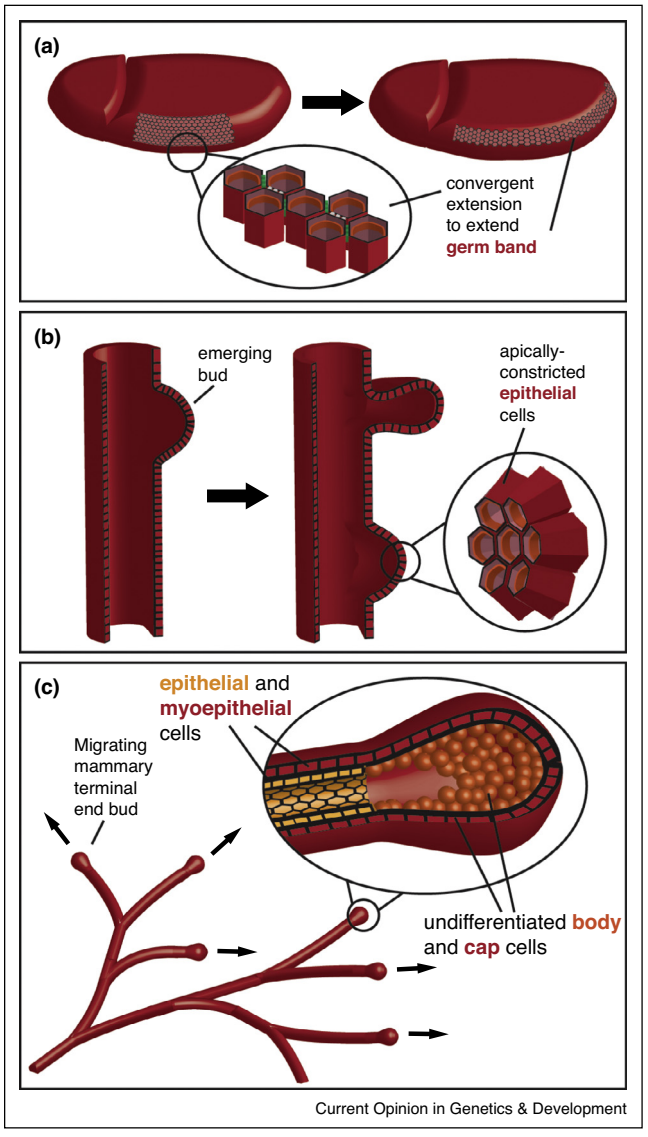
Obtaining a comprehensive, quantitative understanding of the dynamics of convergent extension remains an active area of research that has benefitted from computational modeling. Vertex models (reviewed in [9]) consider epithelial cells as polygons with the locations of the vertices specifying the state of the epithelial sheet (Figure 3a). Here, the equations of motion govern the

**Figure 1**




**Contraction of actomyosin filaments generates tissue-scale changes in shape**. **(a)** Filamentous actin, crosslinking proteins (not shown), and non-muscle myosin II form contractile actomyosin filaments, shown in the zonula adherens belt (orange structures) within an epithelium. **(b)** Local increases in planar polarized contractility (see yellow cell borders) result in preferential remodeling of cell junctions (to white cell borders) during convergent extension. **(c)** Apically-localized actomyosin contractility decreases the area of the apical membrane to drive budding and epithelial sheet bending.

**Figure 2**

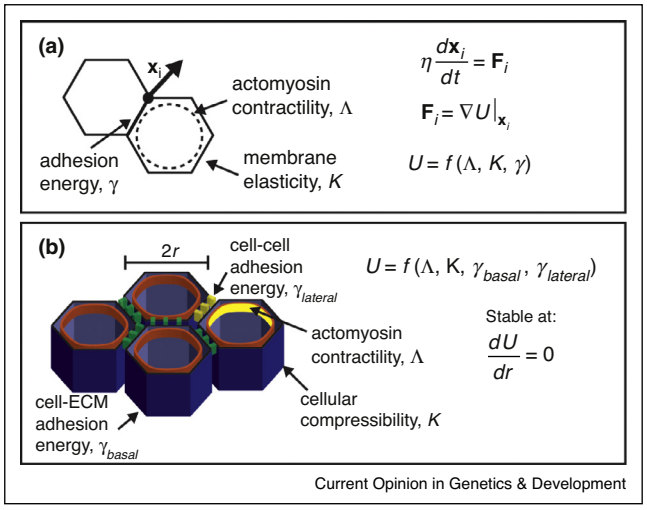



**Actomyosin contractility regulates many developmental processes**. **(a)** Convergent extension elongates the germband during *Drosophila* gastrulation. **(b)** Apical constriction initiates budding during airway branching morphogenesis in the embryonic chicken lung. **(c)** Contractility-dependent collective migration sculpts the 3D mammary ductal architecture during mammary branching morphogenesis.

movement of the vertices, and the forces associated with cellular contractility can be either specified explicitly or follow from the minimization of an energy function. The latter approach has been used to reveal that during germband extension, convergent extension results from the tissue minimizing its potential energy when driven by planar polarized contractility [10]. These cellular rearrangements occur with a characteristic, limiting velocity of $\sim$2 μm/min, as calculated from a linear dependence of tissue cohesion, a function of actomyosin contractility and intercellular adhesion, on the resistance of the tissue to flow [11]. It will be interesting to determine how incorporating additional complexities, such as feedback between contractility and adhesion, competition between contractility and other cytoskeletal filaments [12], or mechanical communication with nearby morphogenetic events [13], into computational models quantitatively affects the final tissue form. In addition to elucidating rate-limiting steps in morphogenetic processes, such insight might clarify the degree to which certain mechanisms are context-dependent.

Axis elongation in *Drosophila* highlights the importance of controlled, spatiotemporal regulation of actomyosin contractility and the associated generation of force during tissue extension. Myosin II must be spatially and temporally regulated to achieve an efficient change in tissue shape [14], as increasing or decreasing its expression results in less efficient intercalation. This is consistent with studies *in vitro* in which it is seen that motor activity must be regulated to coordinate global contractions [15]. Otherwise, motor activity causes crosslinks to unbind,

**Figure 3**




**Computational modeling can quantify the underlying physical forces acting on cells during morphogenesis. (a)** In vertex models, cells are often modeled as 2D polygons representing a slice through the cell at the adherens belt. The movement of each vertex is related to the force acting on that vertex, which is a function of actomyosin contractility, adhesion to neighboring cells, and the elasticity of the membrane. See [9] for more information. **(b)** A recent model of epithelial morphology expands the force balance into 3D to represent changes in tissue shape as stable points in the underlying mechanical equations.

thereby driving an initially well-connected network to a critical, ruptured state. Thus, actomyosin contractility may be thought of as one regulator of a tissue-level potential energy function that, when properly maintained, drives cellular rearrangement and tissue extension.

## Actomyosin contractility tissue budding and bending

In addition to changes in tissue shape mediated by junctional remodeling, morphogenetic events also proceed when changes in cellular shape propagate into changes in tissue form. For example, apical constriction (reviewed in [16]) within adjacent cells produces a bend or bud within an epithelial sheet (Figure 1c). This change in cell shape, mediated by apically localized actomyosin contractility, is critical for many processes including initiation of monopodial branches in embryonic chicken lungs (Figure 2b) [17••]. A similar role for actomyosin contractility has subsequently been suggested for domain branching during murine airway branching morphogenesis [18]. In this developing organ, the degree of budding scales with the degree of contractile activity [19] and bifurcation of lung buds is also driven by MLCK-regulated actomyosin contractility [20].

Live imaging has revealed many dynamic features of apical constriction, especially during *Drosophila* gastrulation. During formation of the ventral furrow, apical domain polarization localizes actomyosin activity in prospective mesodermal cells to drive apical constriction in a ratchet-like mechanism [21•,22]. In this process, pulses of myosin II activation are associated with pulses of ROCK without a detectable lag time [23]. Forces generated by apical constriction in ventral furrow formation can then produce a tissue-scale hydrodynamic flow of cytoplasm to mediate tissue elongation and propagate forces deeper into the tissue [24••].

Despite this tremendous insight, much less is known quantitatively about how force generation regulates apical constriction. Mechanical models have demonstrated that apical constriction is sufficient to give rise to budding [17••] and folding of an epithelium [25], yet much remains to be learned about apically-localized contractility and how it varies in different contexts. It will be interesting to investigate force generation and transmission across multiple length scales during the ratchet-like mechanism of apical constriction with vertex or vertex-like models. Emerging techniques using fluorescence resonance energy transfer (FRET)-based force sensors [26,27] will also likely help provide quantitative data against which to test these models.

Actomyosin contractility is also an important regulator of epithelial bending, which can convert a two-dimensional (2D) sheet into a three-dimensional (3D) structure. Recent developmental studies of the chick heart tube [28], *Drosophila* wing disk [29] and eggshell [30•] have implicated regulatory roles for cellular tension in producing bent, 3D tissue structures. It will be interesting to characterize the dynamics of such changes in shape in these and future cases with regard to recent models of epithelial morphogenesis (Figure 3b) [31••]. Here, *in vivo* morphologies are understood as stable points of mechanical equations, with epithelial sheet curvature and bending resulting from cell adhesion and contractility. In the future, quantitative measurements of tension should be incorporated into such models. For example, pN-scale forces have been measured across focal adhesions [26] and adherens junctions [27]. Other emerging quantification tools include laser nanosurgery [32], laser ablation [33], and deformable microdroplets of fluorescent oil [34]. The latter method has been used to measure anisotropic stresses on the order of nN $\mu m^{-2}$ in 3D aggregates.

Similar to tissue elongation, apical constriction requires precise regulation of the actomyosin force-generating machinery. During *Drosophila* dorsal closure, amnioserosa cells propel epidermal cell migration by rapidly fluctuating their apical membrane area. The cycle lengths of these fluctuations shorten with the onset of net tissue contraction, followed by a damping of fluctuation amplitude until the amnioserosa cells contract rapidly [35]. Here, a low level of myosin activity is required to generate

efficient contraction, as increased myosin phosphorylation results in an apparently stiffer amnioserosa [36]. It will be interesting to study quantitatively how force generation, cytoskeletal architecture, and adhesion complex formation are optimized for this and similar morphogenetic events.

## Actomyosin contractility collective migration

As discussed above, a tissue can change shape through junctional rearrangements and cell deformations. Changes in tissue form also occur during collective cellular migration (reviewed in [37]). For example, collective epithelial cell movement through the mammary stroma during branching morphogenesis (Figure 2c) produces the characteristic mammary ductal architecture [38]. Here, the cells within the migrating cohort maintain junctional attachments to each other as the population remodels the surrounding tissue and extracellular matrix.

3D culture models have provided quantitative insight into how collective migration is regulated by actomyosin contractility. In a 3D model of mammary branching morphogenesis, tissue regions under higher mechanical stress (~0.1 kPa) show changes in gene expression [39] and molecular signaling [40], resulting in multicellular invasion into the surrounding matrix [41]. Additionally, contractility within the tissue generates surrounding mechanical heterogeneities [42] and locally aligns matrix fibrils to instruct ductal elongation [43]. These findings are congruent with recent observations of branching morphogenesis of the murine salivary gland, where actomyosin contractility regulates basement membrane remodeling [44]. Actomyosin contractility is thus central to branching morphogenesis, and so it will be exciting to uncover more quantitative details about how intercellular forces temporally vary as the branch extends.

A full understanding of the mechanics of collective migration will require quantitative observation into how the material properties and behavior of individual cells within a migrating cohort influence migration and morphogenesis. Indeed, as cell density increases in a migrating epithelial sheet, cell movements transition from ballistic (that is, the cellular mean square displacement is proportional to the square of the observation time, $\langle r^2 \rangle \sim t^2$) to sub-diffusive ($\langle r^2 \rangle \sim t^\alpha$ where $\alpha < 1$) as the cells become trapped in cages formed by their neighbors [45]. This increases the stiffness of the epithelium, possibly due to increased transmission of stress between cells and a concomitant strengthening of the cytoskeleton. The formation of adherens junctions also coincides with an increase in the apparent stiffness of epithelial monolayers, reflecting the generation of tissue-level tension [46]. These observations provide further mechanical means by which actomyosin contractility is involved in regulating morphogenesis.

## Outlook and concluding remarks

Actomyosin contractility both regulates and is regulated by tissue morphogenesis, as is briefly described here for 2D tissue elongation, 3D budding and bending, and collective migration. A more complete understanding of the regulation of tissue morphodynamics will require better linking of molecular signaling to current mechanical models that describe changes in tissue shape based on localized contractility. A notable recent example combines approaches to demonstrate oscillations of myosin contractile activity in the observed spatiotemporal pattern in the elongating Drosophila egg chamber [47]. Potential topics for mechanochemical modeling include a wingless-int chemical gradient specifying the precise domains of localized nonmuscle myosin II activity during chick feather morphogenesis [48] and feedback between ROCK and Shroom signaling to amplify planar polarized actomyosin contractility during *Drosophila* germband extension [49]. It will also be interesting to incorporate more complex feedback between intercellular forces and cellular biochemistry. For example, myosin-dependent contractility can decrease the mobility of cadherin molecules, thus concentrating them at adherens junctions in culture [50]. Finally, calcium channels also regulate force [51], indicating a further unexplored coupling between cellular bioelectrochemistry and tissue mechanics.

Recent advances have expanded our quantitative understanding of how contractility influences the dynamics of morphogenetic processes. Overcoming current challenges will require quantifying the forces generated by actomyosin filaments in tissues undergoing morphogenesis and incorporating these into more advanced models describing changes in tissue shape. Where this fails, better culture models must be developed to physiologically recapitulate *in vivo* development. Quantitative insight gained from these processes can then be used to test computational models that describe 3D tissue morphogenesis as a function of subcellular actomyosin regulation. Conversely, newly developed models should be able to aid in experimental design. By incorporating all three approaches, we will be able to use culture and modeling results to verify mechanisms postulated from *in vivo* data, while using *in vivo* observations to identify the physiological relevance of culture and modeling data. *In vivo* imaging, culture models, and computational approaches are thus well poised to generate a robust, quantitative understanding of tissue morphodynamics.

## Acknowledgements

Work from the authors' group was supported in part by grants from the NIH (GM083997, HL110335, HL118532, HL120142), the NSF (CMMI-1435853), the David & Lucile Packard Foundation, the Alfred P. Sloan Foundation, and the Camille & Henry Dreyfus Foundation. C.M.N. holds a Career Award at the Scientific Interface from the Burroughs Wellcome Fund. M.J.S. was supported in part by the NSF Graduate Research Fellowship Program.

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