Actin Bundles

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Actin is one of the key and highly conserved elements of the cytoskeleton. It is indispensable for driving many cellular processes, including cell migration, cytokinesis, vesicle transport, and contractile force generation. To perform diverse functions, actin filaments assemble into higher-order structures such as branched actin networks and actin bundles. This entry describes different types of actin bundles present in cells, their locations, and the bundling proteins involved in their formation.

actin bundles fascin α-actinin espin plastin/fimbrin

1. Introduction

Actin, one of the key and highly conserved elements of the cytoskeleton, amounts to approximately 5–15% of total cell proteins [1][2]. It is indispensable for driving many cellular processes, including cell migration, cytokinesis, vesicle transport, and contractile force generation [3]. The globular actin monomers (G-actin) polymerize to form semi-flexible double-stranded helical filaments (F-actin), also known as microfilaments. These filaments assemble into higher-order structures, such as branched networks or bundles [5]. It is now recognized that complex actin networks and bundles are essential for many cellular functions. Lately, actin-bundling proteins have been attracting a lot of attention as their malfunction is linked to malignant cancers, muscular dystrophy, bone disease, and immunological disorders [4][5][6][7][8].

More than 150 actin-binding proteins (ABPs) are known to associate with the actin cytoskeleton, and many of them regulate actin functions [9]. These proteins are involved in: (1) regulation of actin assembly and disassembly, (2) actin-driven movements in cells, (3) connecting actin structures to plasma membrane/cell organelles or other cytoskeleton proteins, and (4) organizing actin filaments (by their crosslinking) into higher-order structures, such as branched actin networks or actin bundles [1][10][11]. The proteins forming higher-order structures are among the most represented and diverse functional families of actin-binding proteins.

2. Actin Bundles in the Cell

In the cells, actin is present either in monomeric, filamentous, or higher-order three-dimensional structures, such as bundles and branched networks [10]. The length of filaments varies from dozens of nanometers (e.g., in branched networks) to several dozen micrometers (e.g., in stress fibers, filopodia, and stereocilia) [12][13][14][15][16]. Together, they form a continuum of systems enabling the reception and transduction of mechanical stimuli across the cell and providing mechanical support for the shape and polarity of cells. The necessity of forming higher-order actin

structures is dictated by the immense variety of cellular functions supported by the actin cytoskeleton and a broad range of mechanical forces required to carry them out. Forces generated by these higher-order actin networks, ranging from piconewtons to nanonewtons, aid in cell migration and invasion, internal vesicle movements, endocytosis, exocytosis, phagocytosis, and cell division [17][18][19].

Actin bundles are linear arrays of actin filaments crosslinked by one or, more often, several different actin-bundling proteins (**Figure 1** A,B). The length and width of such bundles, and the number of filaments present in them are dictated by their unique set of proteins and by kinetic conditions under which these bundles are formed [20][21], giving each bundled complex a specific structure with different mechanical properties [19][22], **Figure 2, Table 1**. The size of the bundling proteins plays an important role in the topology and the compactness of the bundles (**Figure 1** B,C). For example, fascin, plastins/fimbrins, and small espins are small sized actin-bundling proteins that crosslink actin filaments to form compact and tighly packed bundles, whereas α-actinin is a larger-sized crosslinker inducing widely spaced bundles with distorted square lattice (**Figure 1** B,C).

There are two main types of actin bundles, with either parallel or mixed polarity filament orientations. In parallel (or uniform polarity) actin bundles, the filaments are ordered with consistent polarity, allowing them to conduct work (e.g., membrane deformation) due to the directional F-actin elongation. Parallel actin bundles are present in chemosensory and mechanosensory cell protrusions (microvilli) of most cell types [22][23], stereocilia of inner ear hair cells [24], bristles in the thorax of Drosophila melanogaster [25], and in ectoplasmic specializations of Sertoli cells (**Figure 2**, [26]). Filopodia, microspikes, focal adhesions, and distal ends of dorsal stress fibers, found in most cell types, consist also of parallel actin bundles (**Figure 2**, [7][27]). These bundles create the force for localized membrane protrusions, while helping cells to resist compressive forces from the membrane [28]. They facilitate cell movement in response to extracellular stimuli or intracellular signaling [29]. The length (1 to 100 μm) and number (one to hundreds) of these bundles per cell, their diameter and the number of actin filaments in them (a few to ~1000) vary depending on the cell type and the structures they support (microvilli, stereocilia, bristles, filopodia, (**Figure 2**, **Table 1**), and invadosomes (podosomes/invadopodia) [19][25][27][30][31][32][33][34].

More than two actin-bundling proteins are often associated with these actin structures. Filopodia, a thin actin-rich plasma-membrane protrusions, help in chemosensing during cell migration, wound healing, and cell adhesion to the extracellular matrix [33]. They are enriched in fascin, but additional bundling proteins, such as α-actinin, fimbrin/plastin, filamin, and espin are also present in them under certain conditions [33][35]. Podosomes are actin-based dynamic structures near the plasma membrane of various cells (such as monocytic, endothelial, and smooth muscle cells). They contribute to cell migration, matrix invasiveness, bone remodeling, and mechanosensing [34][36][37] and contain fascin, L-plastin (a hematopoietic cell-specific plastin isoform), and α-actinin [34][38][39][40]. Invadopodia are functionally and structurally similar to podosomes of normal cells, but they are present in tumor cells [34]. Additionally, podosomes and invadopodia are unique in their ability to degrade ECM material by locally releasing proteolytic enzymes [34]. Ectoplasmic specializations of Sertoli cells, which are hybrid testis-specific cell–cell contacts (contributing to the blood–testis barrier), contain espin and T-plastin (a most abundant plastin isoform found in cells of most solid tissues) [19][41][42][43]. Microvilli—the finger-like projections on the surface of several types of cells - increase the total cell surface without substantially increasing its volume [19][23]. Most microvilli

contain I-plastin (a plastin isoform found in the intestinal and kidney microvilli and stereocilia of the inner ear), small isoforms of espin, and villin. Similarly, stereocilia are the modified microvilli that transduce mechanical signals into stimulus-dependent electrical signals. They predominantly contain I-plastin and fascin, but also have isoforms of espin and other proteins expressed in smaller quantities [44][45].

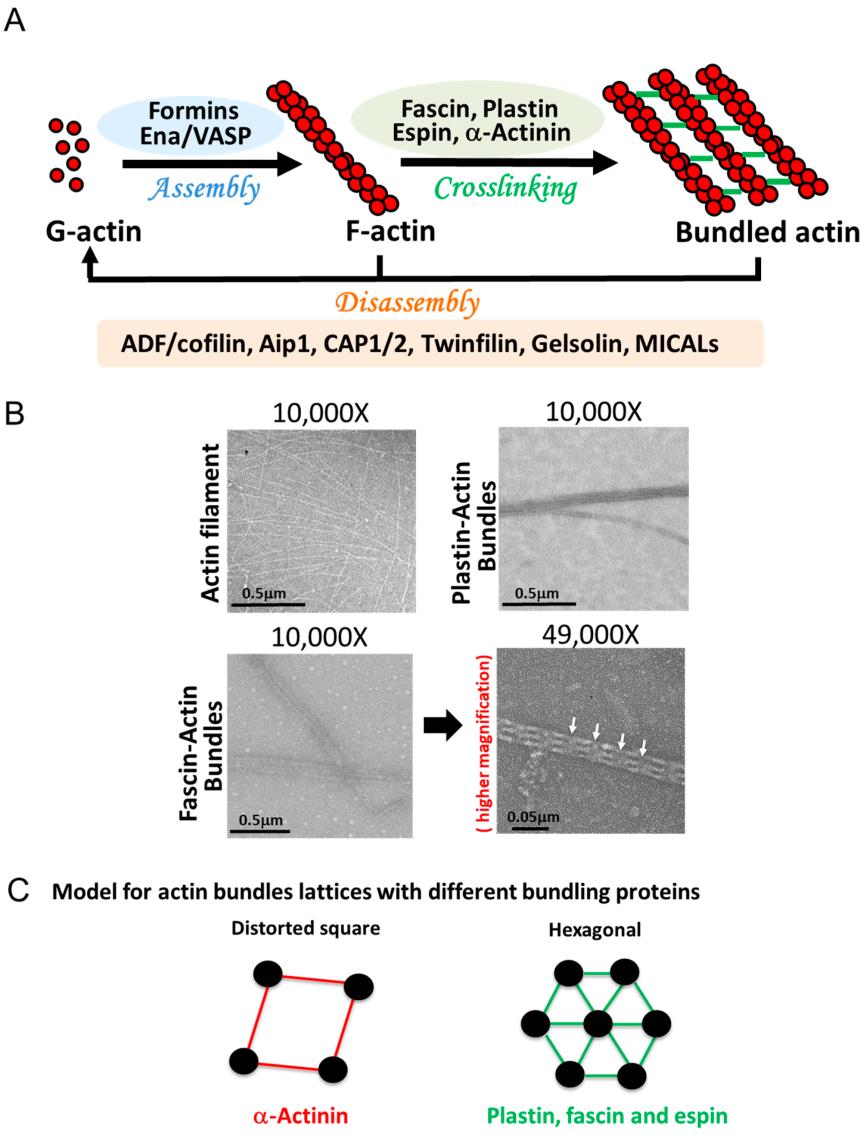

Figure 1. Overview of actin organization in the cell. (**A**) Assembly of actin monomers into linear filaments and higher-order actin structures. Actin monomers bind nucleation and elongation factors, such as Ena/VASP and formins, that assist in these processes, usually near the cytosolic side of plasma membranes. To form actin bundles in a spatially and temporally controlled manner, bundling proteins are recruited to crosslink these filaments. Actin filaments and bundles disassemble into actin oligomers (via severing) and monomers (via accelerated depolymerization) with the help of several disassembly/severing proteins, which contribute to actin turnover in cells. (**B**) EM micrographs of negatively stained actin filaments alone and in the presence of fascin or T-plastin. The magnification of the images is shown on the top of each micrograph. A high-magnification image (0.05 μm) shows an ordered fascin–actin bundle with periodic striations (indicated by arrows). These striations are formed by fascin bound to actin filaments. (**C**) Model of bundle lattices formed in the presence of different bundling

proteins. The black circles denote the actin filaments, and the colored lines denote the actin-bundling proteins. Notably, in the case of a hexagonal lattice, the inter-filament distance varies with the size of the bundling proteins involved.

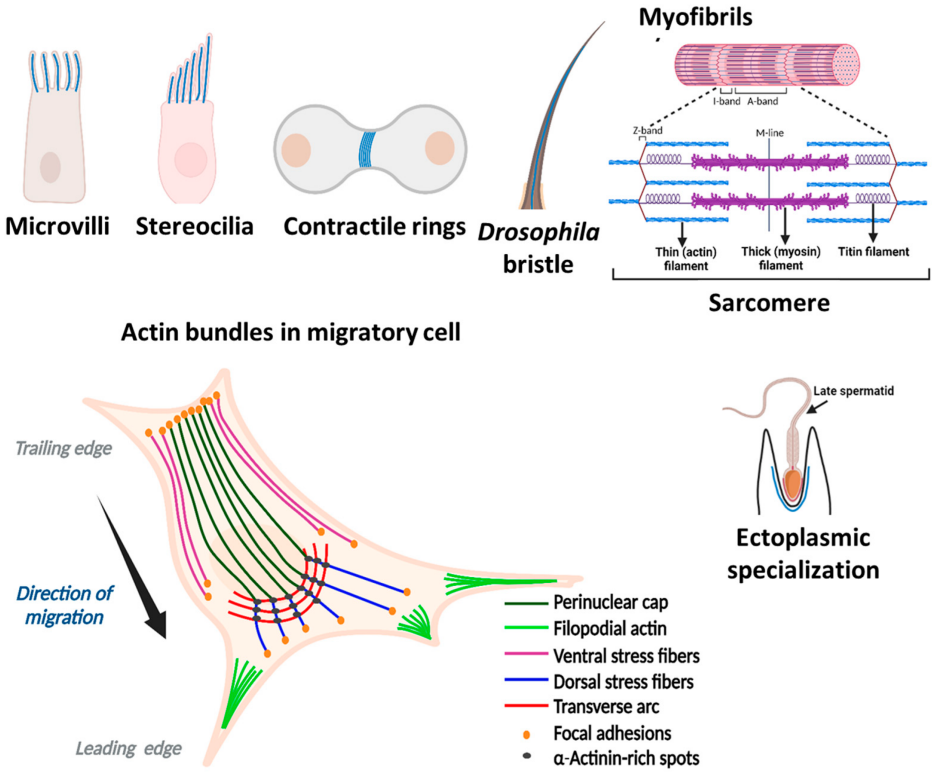

Figure 2. Schematic representation of a cell with different architectures of actin bundles. The blue lines show the actin bundles' location in different types of cells. In the sarcomere, "thick filaments" (purple) are composed of myosin, while "thin filaments" (blue) are actin bundles decorated with troponin and tropomyosin (decoration is not shown). The different actin bundles structures in migratory cells are shown using different colors. The thick brown line surrounding the perimeter of the cell denotes the cell cortex. This figure was created using BioRender.com.

Table 1. Properties of actin bundles in different structures.

Location	Microvilli	Stereocilia	Filopodia	Stress fibers	Bristles (*drosophila*)
Cell type	Most cells (frequent in epithelial cells)	Auditory and vestibular sensory cells	Motile cells	Most cells (prominent in fibroblasts, smooth muscle, and endothelial cells)	Sensory organ precursor cells
Function	Increase apical surface area for absorption	Mechano-electrical signaling	Sensory and guiding	Contraction and adhesions	Mechanosensing

Location	Microvilli	Stereocilia	Filopodia	Stress fibers	Bristles (*drosophila*)
Length	100 nm to 2 µm	1.5–15 µm	≤10 µm	≥2 µm	Macrochaetes: 250–300 µm, Microchaetes: 70 µm (non-continuous 1–5 µm units)
Diameter	50–100 nm	~200 nm	20–200 nm	Varies from cell to cell	Varies
Number of actin filaments	30–40	~400–3000	10–30	10–30	7–18 bundles with hundreds of filaments
Actin filament organization	Parallel (unipolar)	Parallel (unipolar)	Parallel (unipolar)	Mixed (bipolar)	Parallel
Bundling proteins	Espin, plastin, villin	Fascin, espin, plastin	Fascin, α-actinin, plastin, espin	α-Actinin, fascin filamin	*singed* (fascin), *forked* (espin)

n a longer distance between their actin-binding domains (e.g., α-actinin). Therefore, these filaments are typically packed less densely than in parallel bundles, leaving space for the intercalation of myosin thick filaments. Mixed polarity bundles are the constituents of stress fibers of non-muscle cells, myofibrils in muscle cells, cytokinetic contractile rings, and the cell cortex (**Figure 2**). The filament-forming motor protein myosin II is usually associated with these actin bundles and enables their contractile functions [46][47]. Stress fibers are the primary mediators of cell contraction in non-muscle cells, governing some of their vital processes, including migration, adhesion, and mechanosensing. Although α-actinin is the major actin-bundling protein identified in stress fibers, other bundling proteins (fascin, espin, fimbrin/plastin, and filamin) can also be present (**Table 1**, [47][48]). A likely role of fascin and espin in such bundles is the stabilization of a subset of filaments with uniform polarities; fimbrin/plastin, despite their compact size, can directly stabilize antiparallel actin assemblies [49][50]. Based on their origin, subcellular location, and protein composition, stress fibers can be grouped into four classes: ventral, dorsal, transverse arcs, and perinuclear actin caps (**Figure 2**).

Ventral stress fibers are >2 µm long thick actomyosin bundles, whereas dorsal fibers are shorter (~1 µm), devoid of myosin, and unable to contract [51]. These fibers usually contain 10–30 actin filaments per cross-section [47][52]. Ventral stress fibers anchor to focal adhesions from both ends, while dorsal stress fibers anchor to them only from one end, with the other end being embedded into transverse arcs. Ventral stress fibers are the most abundant contractile structures in the cell. They are made of bipolar actin filaments that display alternating patterns of myosin- and α-actinin-enriched regions. In migration, they help retract the motile cells' trailing edge and establish the cells' front–rear polarity [53]. Transverse arcs are curved, thin actin filament bundles with repeated α-actinin–myosin patches formed just behind the lamellipodia [47]. They are mainly involved in the persistence of cell motility by acting as a link between the lamella and the focal adhesion-connected dorsal stress fibers [54]. The perinuclear actin cap is a contractile structure (surrounding the nucleus) that emanates from focal adhesions at the leading edge. It influences the nucleus shape and position in the cell by transducing environmental signals to the nucleus of cells via LINC complexes [55][56].

Myofibrils of cardiac and skeletal muscles are assembled into highly organized periodic structures (sarcomeres; **Figure 2**). In contrast to that, myofibrils in smooth muscles are less ordered and more reminiscent of the ventral stress fibers of non-muscle cells. However, these three types of myofibrils share a striated pattern when immuno-stained with antibodies against myosin II and α-actinin. Each sarcomere contains thick (myosin) filaments in the center and is flanked by regions that contain thin filaments (actin filaments decorated by tropomyosin and troponin) (**Figure 2**). Actin filaments are embedded with their barbed (plus) ends in the Z-band regions separating sarcomeres and containing α-actinin as the major crosslinking protein (**Figure 2**, [57]). Therefore, while notably more ordered and with a better-controlled filament length, sarcomeres are organized similarly to stress fibers, which may serve as precursors of sarcomere assembly [58].

In cytokinetic contractile rings of dividing cells, actin filaments form bundles at the division plane (**Figure 2**) along with the intercalated myosin II filaments. The motor activity of myosin drives the contraction and separation of cells into two daughter cells. α-Actinin, fimbrin/plastin, and anillin are the bundling proteins identified in these structures [59][60][61]. In addition to these actin-rich structures, actin bundles are also present in the cell cortex (**Figure 2**). The cell cortex is clearly detectable in rounded mitotic or amoeboid cells, as a continuous layer of actin and non-muscle myosin II-enriched networks under the cell membrane. Its thickness varies from ~190 nm in human mitotic cells to 4 μm in some oocyte cells [62][63]. The actin-bundling proteins associated with the cell cortex include α-actinin, fimbrin/plastin, fascin, and filamin [62].

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