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MINIREVIEW

Polarized Cells, Polar Actions

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INTRODUCTION

It has long been believed that the small size of bacteria, between 1 and 5 μm, obviates the need for subcellular domains, intracellular organelles, and a cytoskeleton. Recently, however, a bacterium, *Epulopiscium fishelsoni*, was discovered that is equivalent in size to a large eukaryotic cell (600 μm in length) but that still lacks any apparent intracellular organization, with the exception of a highly convoluted cytoplasmic membrane (4, 13). Are these membrane invaginations sufficient to allow this enormous bacterium to survive in a world thought to depend on diffusion? Perhaps this species, and other bacteria as well, possesses additional levels of organization that have not been observed by standard cytological examination of ultrastructure. This review explores the accumulating evidence that bacteria exhibit subcellular organization, with particular attention to organization at the cell poles.

There are numerous examples of polar organelles in bacteria, such as flagella, pili, and stalk-like appendages. Some of these external structures appear to border on the absurd, such as the 50 monopolar flagella in *Pyrococcus furiosus* (19). Often, the position of a polar appendage correlates with a physiological function, such as the attachment of *Pseudomonas aeruginosa* to the tracheal epithelium via a polar pilus. In this case, the bacterium not only attaches at the pole, but appears to burrow into the tracheal cells in a polar direction (57). Other observations that have appeared in the literature are more enigmatic. Some of these include the polar distribution of pili and hemagglutanin in *Myxococcus xanthus* (36, 38), the polar location of the maltose-binding protein in *Escherichia coli* (11), and the polar organelle of *Rhodopseudomonas palustris* (45). Recently, it has been demonstrated that periplasmic proteins in *P. aeruginosa* and *Erwinia herbicola* that participate in the conversion of chorismate to phenylalanine are localized at the cell poles (55, 56).

Are these observed polarities unusual events found in relatively few species, or does the capacity for polar localization of certain proteins and structures reflect a more general cellular organization that has been, for the most part, overlooked? In the following sections we describe three different but well-characterized examples of bacterial polar organization in an attempt to identify the unifying principles that establish polarity. We also address whether *Escherichia coli*, a bacterium that is not often regarded as exhibiting polarity, does in fact share some of these basic polarizing mechanisms.

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BRADYRHIZOBIUM JAPONICUM: AN EXAMPLE OF POLAR ATTACHMENT

Rhizobium and *Bradyrhizobium* species are legume symbionts. Establishment of the symbiosis between rhizobia and legumes is initiated by the intimate association of the bacterium with the root hair surface and culminates in the formation of a nitrogen-fixing nodule. In many cases, the initial attachment of rhizobia to the plant root hair is polar (10, 14, 42, 43, 49, 51). Polar attachment is rapid (51) and is mediated by an extracellular fibrillar material, located at the pole of the *Bradyrhizobium japonicum* cell, that bridges the gap between the bacterium and the root hair (49, 52). At least one component of this fibrillar material, a bacterial lectin called BJ38 (23), is localized to the extracellular fibrils that interact with the root hair (29). This lectin appears to be important for attachment, as mutants which do not produce BJ38 have a reduced ability to attach to root hairs (24).

The plant lectin soybean agglutinin has been implicated in the selective binding of *B. japonicum* (7, 12, 44, 50). The bacterial receptors for the plant agglutinin are polysaccharide components of the extracellular capsule. The capsule, including the soybean agglutinin receptors, is predominantly present at one pole of the cell (12, 50). Surprisingly, it has recently been demonstrated that these receptors are positioned at the bacterial pole opposite to that used for attachment (29). Furthermore, mutants without detectable capsules are still capable of adhering to root hairs (28); thus, receptor-lectin interaction is not involved in initial attachment. The polar positioning of the soybean agglutinin receptors may reveal a possible function during later stages of infection.

In addition to its asymmetric distribution of extracellular components, *B. japonicum* displays intracellular asymmetry. The end of the cell which contains the root hair attachment site, fibrils, and BJ38 lectin is occupied by reserve polymers (29, 49, 52). The opposite end of the cell, which is coated by the cellular capsule and agglutinin receptors, is occupied by the nucleoid and remaining cytoplasmic material (12, 49, 50).

How does the *B. japonicum* cell transmit its asymmetric pattern to its progeny? Cell division in bacteria produces inherently asymmetric progeny cells. The pole that arises from the division site is referred to as the "new" pole, whereas the distal pole is referred to as the "old" pole (Fig. 1A). It appears that the old pole carries the information concerning the prior history or state of the cell. In *Bradyrhizobium* cells, the reserve polymers are predominantly at the old pole, whereas the "nucleoid" portion is at the new cell pole. The cell undergoing division may first convert the nucleoid pole to an old cell pole by synthesizing and storing reserve polymers at that pole, yielding a predivisional cell with reserve polymers at both poles prior to division, as diagramed in Fig. 1A. The new poles produced after cell division contain the nucleoid portion of the

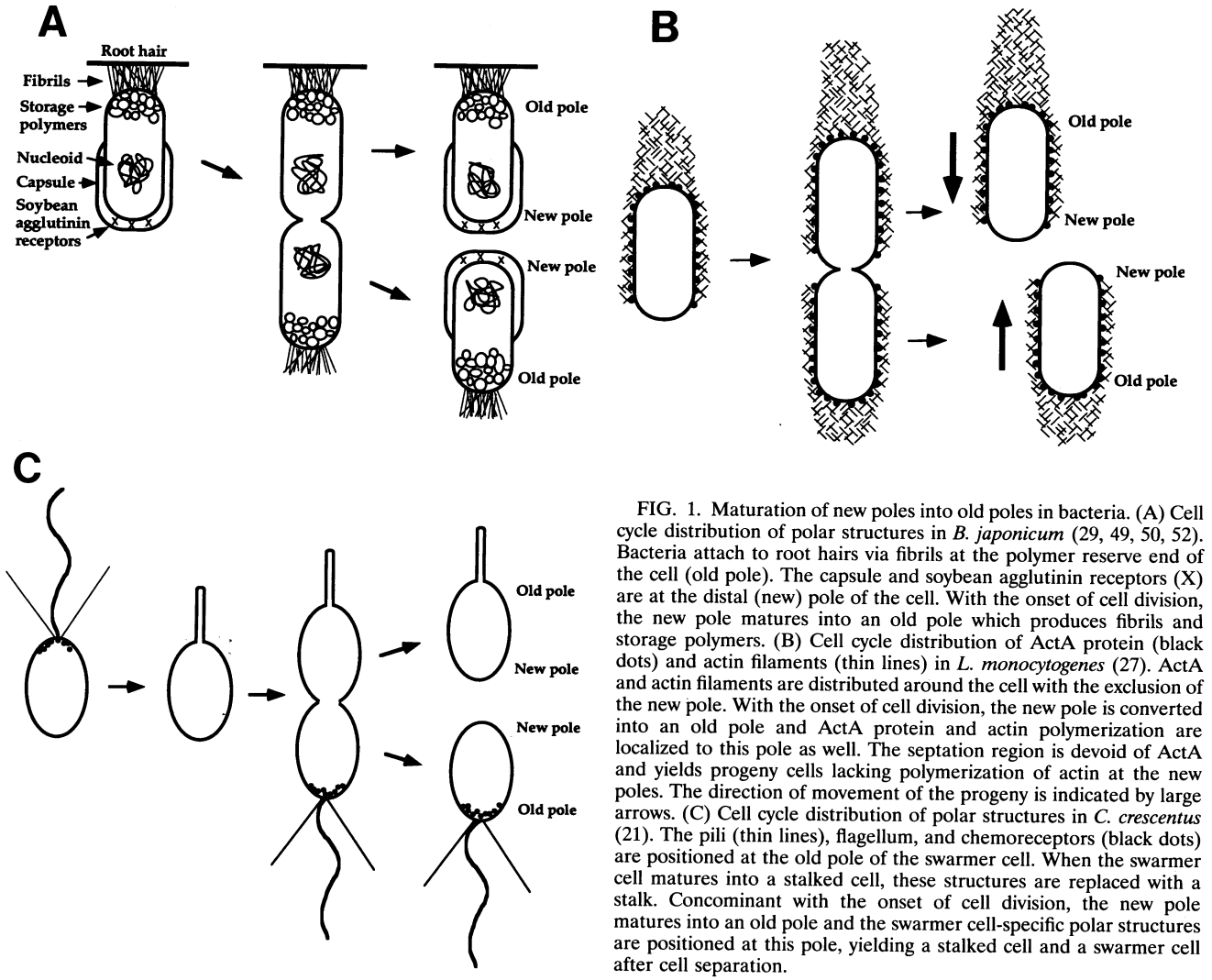

FIG. 1. Maturation of new poles into old poles in bacteria. (A) Cell cycle distribution of polar structures in *B. japonicum* (29, 49, 50, 52). Bacteria attach to root hairs via fibrils at the polymer reserve end of the cell (old pole). The capsule and soybean agglutinin receptors (X) are at the distal (new) pole of the cell. With the onset of cell division, the new pole matures into an old pole which produces fibrils and storage polymers. (B) Cell cycle distribution of ActA protein (black dots) and actin filaments (thin lines) in *L. monocytogenes* (27). ActA and actin filaments are distributed around the cell with the exclusion of the new pole. With the onset of cell division, the new pole is converted into an old pole and ActA protein and actin polymerization are localized to this pole as well. The septation region is devoid of ActA and yields progeny cells lacking polymerization of actin at the new poles. The direction of movement of the progeny is indicated by large arrows. (C) Cell cycle distribution of polar structures in *C. crescentus* (21). The pili (thin lines), flagellum, and chemoreceptors (black dots) are positioned at the old pole of the swarmer cell. When the swarmer cell matures into a stalked cell, these structures are replaced with a stalk. Concominant with the onset of cell division, the new pole matures into an old pole and the swarmer cell-specific polar structures are positioned at this pole, yielding a stalked cell and a swarmer cell after cell separation.

cell, and the older poles contain the reserve polymers (50). *B. japonicum* can apparently distinguish between old pole and new pole and, accordingly, can convert the nucleoid pole (new pole) to the reserve polymer pole (an old pole) following an uncharacterized cell division cue. These polar events require, in addition to intracellular reorganization, the loss of the polar capsule and the production and specific targeting of polar fibrils. How these feats are accomplished, and the mechanism by which the spatial organization is regulated, is not known. These observations imply that the initial root hair attachment of the bacterium is from the old pole (Fig. 1A). Maintaining the attachment at the old pole may be advantageous in that it ensures that the bacterium will remain attached to the plant cell regardless of future cell division events. If the bacterium utilized the new pole for attachment, then upon cell division and maturation of a new pole to old pole, the cell could detach before infection occurred.

POLYMERIZATION OF ACTIN BY *LISTERIA MONOCYTOGENES*

L. monocytogenes is an opportunistic pathogen that invades macrophages and other mammalian cells and spreads within the host tissue by direct cell-to-cell infection. *L. monocytogenes* enters the host by induced phagocytosis and subsequent lysis of the phagocytic vacuole. Once free in the host cytoplasm, the bacterium rapidly induces the polymerization of host cell actin filaments. After cell division, this results in a long actin tail extending from one pole of the bacterial cell (48). The polymerization of the asymmetrically distributed actin propels the bacterium through the host cell (Fig. 1B; 41, 46). Polymerization and cross-linking of short actin filaments is excluded at the leading pole, resulting in the formation of an actin tail at the trailing end of the bacterial cell (47). The direction of movement is determined by which pole of the cell is to be excluded from the actin polymerization process.

As in *B. japonicum*, *L. monocytogenes* polarity is established during cell division. Only the new poles are devoid of assembled actin (47). As the bacterium begins to divide, the new pole matures into an old pole. Actin assembly is then observed at both poles, thus producing a predivisional cell that has opposing actin tails and is devoid of actin filaments at the new cell division site. After cell separation, the progeny cells move in opposite directions (Fig. 1B).

One of the main players in the asymmetric assembly of actin appears to be the surface protein ActA. Cells with *actA*

mutations do not accumulate actin and do not move within the host (17, 26), suggesting that ActA is required for the bacterially induced actin assembly. Recently, it has been demonstrated that the distribution of ActA mirrors that of actin assembly (27) in that ActA is distributed around the cell except at the new pole following cell division. These new poles then mature into old poles as the cell begins a new cell division, with ActA now excluded from the septum (Fig. 1B). The gram-negative enteric pathogen *Shigella flexneri* uses a system of actin-based motility which appears to be remarkably similar to that of *L. monocytogenes*. In *S. flexneri*, an outer membrane protein (VirG) seems to be exclusively localized at one pole (22).

CAULOBACTER CRESCENTUS AS A PROTOTYPICAL POLARIZED BACTERIUM

C. crescentus possesses many polar features whose presence and location are developmentally regulated. Unlike *B. japonicum* and *L. monocytogenes*, cell division in this organism does not produce two identical progeny. Instead, division produces a motile swarmer cell and a sessile stalked cell (Fig. 1C). At its old pole, the swarmer cell possesses a single flagellum, holdfast material, several pili, and chemoreceptors (2; for a review, see reference 21). In response to an unknown cue, the swarmer cell loses its flagellum and pili and specifically degrades its chemoreceptors (3; for a review, see reference 21). A stalk, with holdfast at its end, grows at the site previously occupied by the flagellum. As the stalked cell elongates and cell division initiates, a new flagellum, pili, phage receptors, chemoreceptors, and holdfast material are synthesized and positioned at the cell pole opposite the stalk.

The swarmer cell has its polar appendages at the old pole. As this cell matures into a stalked cell, the flagellum is released and a stalk is synthesized at the same site. When the stalked cell elongates and becomes a predivisional cell, the new pole matures into an old pole and assembly of the newly synthesized polar proteins and structures occurs at this pole. The mechanism of pole site selection is not known, but it may be dictated by a cellular marker that is laid down at the time of cell division (25). This hypothetical cellular marker, which has been referred to as an organizing center, may include parts of the cell division machinery. The organizing center must remain silent until appropriate cell cycle cues trigger maturation of the inactive organizing center into an active form (i.e., mature a new pole into an old pole). Such a mechanism may only require the synthesis of an activator at the appropriate time in the cell cycle. The activation of an organizing center, coupled to the temporally controlled expression of polar proteins, would ensure the appropriate placement and assembly of polar structures.

IS *E. COLI* A POLARIZED BACTERIUM?

Recently, new ways of looking at the bacterial cell have revealed that even the well-studied and apparently symmetric *E. coli* cell displays polarity. In the absence of an electric current, *E. coli* cells swim randomly. However, when exposed to a current, the cells travel toward the anode, thus exhibiting directed motility in an electric field. Most surprisingly, when the current is reversed, the cells first turn around, so that the same end of the cell is leading towards the new anode. Therefore, it appears that charge distribution within the *E. coli* cell is asymmetric (1).

It was suggested as early as 1970 that the periplasmic space at the poles of *E. coli* cells is enriched for various proteins (18). Induction of maltose-binding protein results in the physical alteration of the pole to form pole caps (16). Pole caps appear at one pole of small cells and at both poles of long cells, implying that it is specifically the old pole that accumulates large amounts of maltose-binding protein (16). This protein has been shown immunocytologically to be in the polar periplasm (11, 37). It should be stressed that not all periplasmic proteins are localized at the poles of the cells. For example, alkaline phosphatase (34) and alkaline phosphatase activity (33) have been shown to be randomly positioned.

Recently, chemoreceptor complexes in *E. coli* were shown to be clustered predominantly at the cell poles (37). Chemotaxis in bacteria involves the detection of a chemical stimulus by an inner membrane chemoreceptor (MCP), which in turn modulates the flow of phosphate groups through cytoplasmic signaling proteins to control the cell's pattern of flagellar rotation (for reviews, see references 30 and 39). At the membrane, MCPs form stable ternary complexes with CheA, a cytoplasmic kinase, and CheW, a protein that couples CheA to chemoreceptor control (20). These complexes aggregate as clusters that are predominantly at the cell poles (37). The polar clustering of any of these proteins requires all three. In the absence of the membrane proteins (MCPs), both CheA and CheW are randomly distributed in the cytoplasm. Similarly, the polar positioning of the MCPs requires the presence of CheA and CheW (37). It is not known at this time whether there is a preference for chemotaxis complex clustering to the old pole or new pole. However, since the incidence of cells possessing clusters at both poles increases with cells in the process of septating, it is likely that the old pole is the favored position (37).

OLD POLE VERSUS NEW POLE

Although the examples of polarity described in this minireview are phenotypically very different, their patterns of development have a common theme. In *C. crescentus*, it is the old pole that bears polar components. In *B. japonicum*, both the old pole and the new pole bear characteristic components. In *L. monocytogenes*, ActA protein and actin polymerization are absent at the new pole. Clearly, these bacteria are all able to distinguish between the old pole and the new pole and to display polar features accordingly.

What physiological determinants could be used by bacteria to distinguish old pole from new pole? An obvious difference between the cell poles is that the new pole was, most recently, a septum. Cell division is the culmination of an orchestrated event involving a large number of proteins that tag the site of division, rearrange the cell wall, redirect membrane growth, and physically constrict the cell within a specific domain and at a specific time in the cell cycle (see reference 31). FtsZ, which is required for septum formation in *E. coli* (9) and *Bacillus subtilis* (53), and amidase, an autolysin involved in degrading the bacterial cell peptidogycan in *Streptococcus pneumoniae* (15), are positioned at the septum during cell division. There are undoubtedly other cell division proteins that are similarly localized. It is likely that the new pole retains some of the properties, and perhaps specific proteins, left from the division site.

There are several examples of specific cellular features shared by both the septum and the new pole following cell division. We have already described the ActA and actin polymerization in *L. monocytogenes* and the cytoplasmic asymmetries in *B. japonicum*. Unlike *B. japonicum*, many bacteria lack capsular material at the septum and new poles of the progeny. The lack of capsular material at the new pole has even been described for cocci (round bacteria), demonstrating

a "polarity" after cell division. In *Staphylococcus aureus*, progeny from a cell division are unencapsulated at the new "pole" that results from cell separation (6). Similarly, the aggregation substance in *Enterococcus faecalis* is lacking at the septum and new poles of daughter cells (54).

Newly synthesized phage attachment sites are often located at the cell poles (8). Lambda phage attach at the septum and at one pole of progeny cells in *E. coli*, implying that newly synthesized LamB or LamB trimers are localized to these positions (40). The sites of phage attachment have been used as markers for new cell wall and membrane synthesis. The septum and resulting new pole are clearly sites for rapid cell growth, whereas the old pole may be more inert, incorporating new wall material very slowly or not at all (5).

A structural remnant of the cell division process, termed the periseptal annulus, has been observed at cell poles. However, these periseptal annuli appear to remain at the pole as the new pole matures into an old pole (35), so it could not be used for discriminating between old poles and new poles. Cytological examination of cell division in *Salmonella typhimurium* revealed that a septal attachment site (a murein-membrane attachment) remains at the new pole after cell division. The septal attachment site is not present at both poles of the cell, indicating that pole maturation is accompanied by loss of this attachment site (32). The relationships between the murein-membrane attachment sites, cell growth, protein insertion sites, and localized cell division proteins are unknown, but are clearly worth further attention.

SUMMARY

The recognition of polar bacterial organization is just emerging. The examples of polar localization given here are from a variety of bacterial species and concern a disparate array of cellular functions. A number of well-characterized instances of polar localization of bacterial proteins, including the chemoreceptor complex in both *C. crescentus* and *E. coli*, the maltose-binding protein in *E. coli*, the *B. japonicum* surface attachment proteins, and the actin tail of *L. monocytogenes* within a mammalian cell, involve proteins or protein complexes that facilitate bacterial interaction with the environment, either the extracellular milieux or that within a plant or mammalian host. The significance of this observation remains unclear.

Polarity in bacteria poses many problems, including the necessity for a mechanism for asymmetrically distributing proteins as well as a mechanism by which polar localization is maintained. Large structures, such as a flagellum, are anchored at the pole by means of the basal body that traverses the peptidogycan wall. But for proteins and small complexes, whether in the periplasm or the membrane, one must invoke a mechanism that prevents the diffusion of these proteins away from the cell pole. Perhaps the periplasmic proteins are retained at the pole by the presence of the periseptal annulus (35). The constraining features for membrane components are not known. For large aggregates, such as the clusters of MCP, CheA, and CheW complexes, perhaps the size of the aggregate alone prevents displacement.

In most cases of cellular asymmetry, bacteria are able to discriminate between the new pole and the old pole and to utilize this information for localization specificity. The maturation of new pole to old pole appears to be a common theme as well. Given the numerous examples reported thus far, we propose that bacterial polarity displays specific rules and is a more general phenomenon than has been previously recognized.

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