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Review Article

Taxis in archaea

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Microorganisms can move towards favorable growth conditions as a response to environmental stimuli. This process requires a motility structure and a system to direct the movement. For swimming motility, archaea employ a rotating filament, the archaellum. This archaea-specific structure is functionally equivalent, but structurally different, from the bacterial flagellum. To control the directionality of movement, some archaea make use of the chemotaxis system, which is used for the same purpose by bacteria. Over the past decades, chemotaxis has been studied in detail in several model bacteria. In contrast, archaeal chemotaxis is much less explored and largely restricted to analyses in halophilic archaea. In this review, we summarize the available information on archaeal taxis. We conclude that archaeal chemotaxis proteins function similarly as their bacterial counterparts. However, because the motility structures are fundamentally different, an archaea-specific docking mechanism is required, for which initial experimental data have only recently been obtained.

Principles of motility and taxis

Microorganisms respond to changes in the environment in order to optimize growth. One possibility to achieve this is to move towards a location with favorable conditions, a process named taxis. This movement along gradients requires both a sensory system and a motility machinery.

To achieve directed motility in liquid, bacteria and archaea use rotating filamentous motility structures to generate a propulsive force: bacterial flagella and archaella (formerly known as archaeal flagella) (Figure 1) [1–5]. These may generate different kinds of swimming behavior, such as (i) forward movement powered by a bundle of flagella interspersed with a tumbling non-directed motion (*Escherichia coli*) [6,7], (ii) polar flagella/archaella that push or pull the cell (*Vibrio alginolyticus* and *Halobacterium salinarum*) [8–10], (iii) unidirectional motor rotation alternating with stops (*Rhodobacter sphaeroides*) [11], and (iv) wrapping of the flagellum around the cell body resulting in a corkscrew motion of the cell (*Burkholderia* sp.) [12,13] and other mechanisms.

This swimming behavior depends on three states of the motility structure: forward motion, reverse motion, or stationary (cells are motionless or tumble). The effects of rotation direction on the swimming state vary between species and depend on the positioning and architecture of the motility structures. Motility is not by itself directional. Rather, directionality is generated in the form of a biased random walk [14]. As microorganisms generally are too small to sense spatial gradients, they rely on temporal sensing. In addition, they need some kind of memory in order to allow comparison with the situation a second ago. Bacteria and some archaea utilize the chemotaxis system as a sensory system to achieve tactic movement [14–17]. The main effect of the chemotactic system is an influence on the duration between subsequent switches between clockwise and counter-clockwise rotation of the motility structure [17–20]. This leads to the bias of the random walk and thus determines the directionality of the movement. Microorganisms may swim towards attractants and swim away from repellents [14]. Commonly, an increase in attractant concentration and a reduction of repellent concentration elicit an equivalent response.

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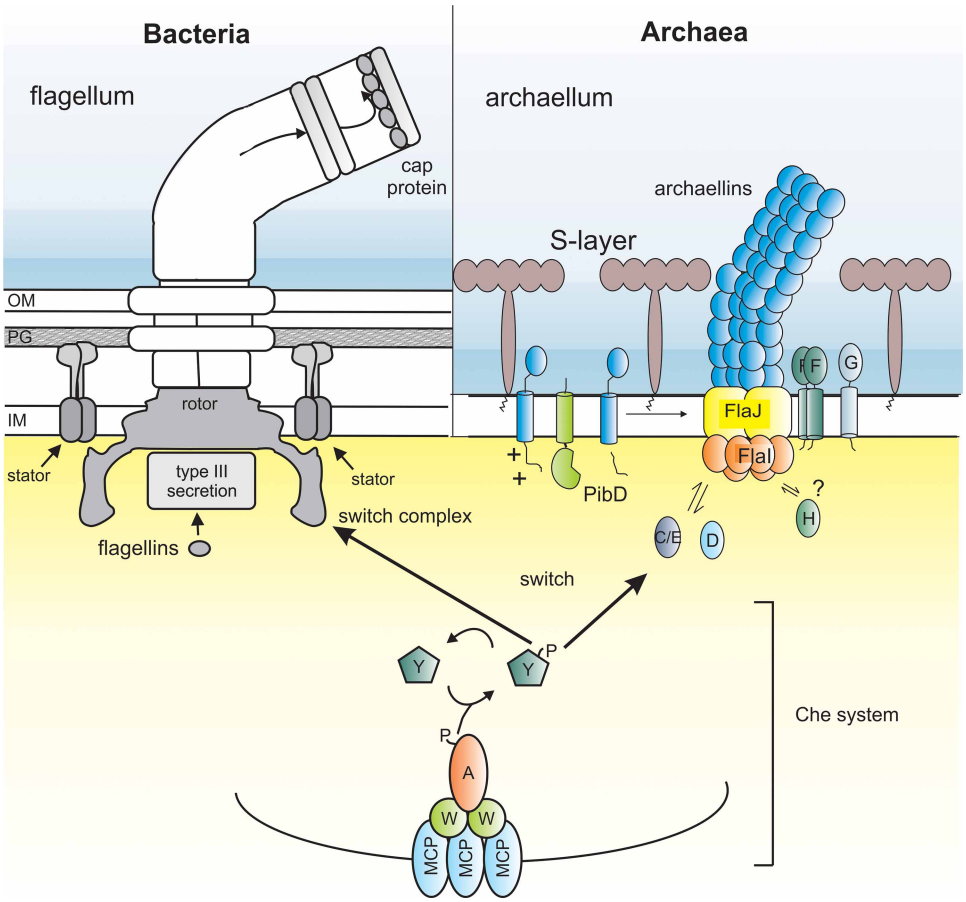

Figure 1. Schematic representation of the chemotaxis system and its signaling cascade to the bacterial and archaeal motility structures.

As examples, a Gram-negative bacterium and a euryarchaeon are shown. Bacteria present the flagellum at their cell surface. New flagellins travel through the hollow interior of the filament to be added at the tip of the growing structure. In archaea, archaellins are N-terminally processed by PibD, which cleaves upstream of the signal peptide H domain. They are then added to the base of the growing structure, in a similar fashion as for type IV pili. A simplified version of the chemotaxis system is depicted, which shows how signals are transferred via the MCPs and CheW, resulting in autophosphorylation of CheA. The phosphate is transferred from CheA to CheY. In bacteria, phosphorylated CheY diffuses to the base of the flagellum where it binds to the switch complex, resulting in a change in the direction of rotation. In archaea, CheY requires the presence of CheF in order to bind to the archaellum. The exact composition and structural organization of the archaeal switch complex is yet not resolved. MCP, methyl-accepting chemotaxis protein; OM, outer membrane; IM, inner membrane; PG, peptidoglycan. Single letters refer to gene names with the prefix *che* (Che system) or *arl* (previously *fla*) (archaellum system).

This review has its focus on archaeal motility and chemotaxis. There are similarities and also major differences between this physiological process in bacteria and archaea. As many excellent reviews on taxis in bacteria exist [14,18,21], we cover bacterial systems only to the level necessary to understand the similarities and differences to the archaeal system.

Motility structures of archaea and bacteria

The rotating motility structures of bacteria (flagella) and archaea (archaella) are functionally similar, but have a fundamentally different molecular organization (Figure 1) [2,22]. The archaellum requires only 8–13 proteins, none of which shares homology with the ~30 proteins constituting the flagellum [23–25]. Instead, the assembly mechanism of the archaellum is similar to that of bacterial type IV pili [2,26]. The formation of type IV pili involves N-terminal cleavage of the major filament-forming proteins and their addition to the base of the

growing pilus via the type IV pilus assembly system [27,28]. The filament of the flagellum, in contrast, is made up of non-processed proteins, flagellins [29], which are secreted via a type III transport system, located at the base of the flagellum, and travel through the hollow interior of the flagellum to be added at the tip of the growing structure [29]. Rotation of bacterial flagella is driven by a proton motive force (or in some cases, a sodium motive force). The force-generating system is unrelated to the biogenesis mechanism. In contrast, rotation of the archaellum requires ATP hydrolysis [30–33]. Also, there is clear evidence that the component responsible for powering archaellar rotation is also involved in archaellar biogenesis [34–36]. In short, while bacteria and archaea possess functionally similar motility structures, at a structural level they are fundamentally different.

Diversity of chemotaxis systems

Motility structures are especially useful in concert with a system directing the movement of a cell towards an environment with better conditions. For this purpose, referred to as taxis, bacteria have adopted a two-component sensory system and a protein methylation-dependent system for adaptation. Whereas the motility structures of bacteria and archaea are fundamentally different, the two-component sensory system is conserved between bacteria and some archaea (mainly *Euryarchaea*, but also some *Thaumarchaea*) [15,16,37]. The conserved taxis signaling cascade is centered around the histidine kinase CheA and the response regulator CheY [17,18,21,38]. It represents an amplification cascade so that a moderate input signal will result in a strong output signal. Both the bacterial and archaeal chemotaxis system have components for adaptation [15,17,19,21]. However, the details of the adaptation system differ, not only between archaea and bacteria, but also within the bacterial domain [15,18,39]. This is evident when comparing the chemotactic systems of *E. coli* and *Bacillus subtilis*, the latter showing more similarities to the archaeal system.

Bacterial chemotaxis: example of *E. coli* and *B. subtilis*

Chemotaxis in *E. coli* has been extensively studied, and because of its simplicity, it serves as an attractive model [19]. *E. coli* cells are propelled forward by a bundle of about five flagella that rotate in a counter-clockwise direction [6,40]. When the flagella rotate in a clockwise direction, the bundle breaks apart and cells start to tumble, stopping forward movement [7,40]. The tumbling results in reorientation, such that when the flagella start rotating counter-clockwise again, the cell swims off in a different, randomly chosen direction [6,7]. The timing of this switch of rotation of the flagellar bundle relies on the chemotaxis system, in particular the concentration of phosphorylated CheY protein (CheY-P) [41,42]. Signal intensity (e.g. chemical concentration) can be sensed by receptors named methyl-accepting chemotaxis proteins (MCPs), which are organized in trimers of dimers that form large hexagonal arrays of thousands of receptors [21,43–45]. Based on the presence or absence of a transmembrane domain in the MCPs, the arrays can be anchored in the membrane or be cytosolic [44,46,47]. Binding of the signaling molecule to MCPs results in a conformational change of the receptors [48–50]. The histidine kinase CheA is, via the adaptor protein CheW, coupled to the MCPs and their conformational change can regulate the autophosphorylation activity of CheA. The phosphate on CheA is subsequently transferred to CheY, so that CheA autophosphorylation activity affects the level of phosphorylated response regulator, CheY [42,51]. Therefore, CheA functions as an integrator for diverse stimuli and delivers one unambiguous output. CheY-P diffuses to the flagellar motor where it interacts with proteins in the 'switch complex' (FliM, FliN and FliG) resulting in an oppositely rotating flagellum [52,53].

The key reaction for adaptation is a reversible methylation and demethylation of the MCPs, thus regulating their sensitivity. MCPs have a central signaling domain, forming a hairpin turn, which on both sides is embedded in coiled-coil domains consisting of heptad repeats [54–56]. Methylation takes place on selected Glu residues within such heptads [56–59], some of which are encoded as Gln and are subsequently deamidated to Glu by CheB in *E. coli* [60]. The CheR protein is responsible for methylation, while CheB is responsible for demethylation [61,62]. Bacterial CheB, which is composed of a catalytic and a CheY-like domain, is activated via phosphorylation by CheA [62]. In *B. subtilis*, CheD, instead of CheB, catalyzes deamidation of Gln residues to methylatable Glu residues [63]. In labeling studies, demethylation can be studied by analyzing the release of methanol [64–66]. In *E. coli*, a positive stimulus leads to an increase of methanol release and removal of the positive stimulus leads to a decrease [65]. The response of *B. subtilis* is different as methanol is released upon both, addition as well as removal of a positive stimulus [64]. This may indicate that opposite stimuli lead to adaptation via methylation of alternate sites in the MCPs of *B. subtilis* [56].

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In addition to the aforementioned proteins, in different bacteria, some other chemotaxis proteins play a role in the adaptation of the signal (CheC, CheV) and removal of the phosphate from CheY-P (CheZ, CheX, CheC, FliY) [61,62,67–73]. For example, *B. subtilis* possesses two extra signal adaptation mechanisms in addition to the CheB/CheR methylation system: (i) the CheC/CheD system and (ii) a system involving phosphorylation of CheV, a homolog of CheW, coupled to a response regulator domain [63,74,75]. The CheC/CheD system consists of the CheY-P phosphatase, CheC, and the receptor deamidase, CheD (see above). However, the interaction between these two proteins is more important for adaptation than their enzymatic activities [76]. CheD is thought to activate CheA by binding to the receptors. Increasing levels of CheY-P stimulate CheC–CheD interaction, leading to a reduction of CheA activation by CheD [63,74,75].

About 50% of chemotactic bacterial genomes contain more than one copy of the core chemotaxis genes [77]. Moreover, many organisms contain additional chemotaxis proteins with no homology to those in *E. coli* [15,37,77]. For example, *B. subtilis* possesses an extensive chemotaxis system with at least a copy of each characterized chemotaxis protein (except CheZ) [72]. Based on the variation of protein components, chemotaxis systems have been divided into 19 classes [37]. In addition to the limited set of chemotaxis proteins of the *E. coli* class, chemotaxis systems belonging to other classes contain an extensive number of proteins with additional functions. Therefore, the relative simplicity of the *E. coli* system is not representative of all bacteria.

Taxis in *Halobacterium* and other archaea

In contrast with the well-explored bacterial chemotaxis systems, studies on archaeal chemotaxis are limited. It was shown that several euryarchaea display tactic behavior and respond to acetate, some amino acids or light [78–80]. However, the majority of these studies seem not yet to have been combined with genetic or biochemical investigations.

Most of our understanding of archaeal taxis has been gleaned from studies of the halophilic model organism, *H. salinarum*. This microbe can perform aerobic respiration, arginine fermentation and use light as an energy source [81]. The latter requires the activity of light-driven ion pumps bacteriorhodopsin (BR) and halorhodopsin (HR) that pump out protons or import chloride ions, respectively [82]. As a result of these different growth strategies, *H. salinarum* can sense and respond to different stimuli such as light, oxygen, amino acids, osmolytes, and membrane potential [83–93].

H. salinarum contains 18 different MCP homologs named Halobacterial transducer proteins (Htrs), which either possess their own sensing domain or interact with other receptor proteins (Table 1) [56]. Six lack transmembrane helices, while the others contain between two and six transmembrane helices. For eight of these different Htrs, the stimuli have been identified (Table 1). HtrI and HtrII mediate the phototactic response, with light being sensed by their associated retinal proteins SRI and SRII, respectively [86,87,94,95,98]. Htr8 and Htr10 (HemAT) are required for attraction and phobic responses to oxygen, respectively [90,93]. Htr14 (MpcT) can detect changes in membrane potential [91], while the cytosolic Htr11 (Car) mediates taxis to the fermentable amino acid arginine, which is thus measured intracellularly. Htr3 (BasT) is responsible for the detection of branched and sulfur-containing amino acids (Leu, Ile, Val, Met, and Cys) [83,92], and Htr5 (CosT) mediates chemotaxis to compatible osmolytes of the betaine family [84]. Both BasT and CosT require binding proteins in order to transduce environmental stimuli. The binding proteins of BasT and CosT are closely related to periplasmic substrate-binding proteins from ABC transporter systems. They belong to the large set of secreted proteins which carry a lipobox and are retained by a covalently attached lipid anchor [84,99]. Bacterial-binding proteins have a dual function as they initiate solute uptake through substrate binding and interaction with the ABC transport system but also mediate chemotactic responses [100]. However, in *H. salinarum*, the binding proteins are exclusively involved in detection of substrates [84].

Phototaxis allows *H. salinarum* to move in the direction of optimal conditions for the two light-driven ion pumps, BR and HR, and at the same time avoid harmful UV radiation that might cause DNA damage. Phototaxis is mediated by Htr1 and Htr2 that receive signals from the photoreceptors SRI and SRII, respectively [86,87,94,95,98]. SRI is a photochromic receptor, which detects orange (attractant) as well as UV (repellent) light, while SRII detects blue light [101–103]. Htr1 and Htr2 are physically connected to the sensory rhodopsin. Besides mediating phototaxis, Htr2 is also involved in sensing of serine (Table 1) [89]. Light activation of SRI and SRII induces the release of membrane-bound fumarate, which was proposed to be an alternative switch factor of archaellum rotation [104,105]. Phototaxis offers boundless experimental possibilities. In contrast with chemical stimuli a light stimulus can be instantly switched on and off, allowing for a dynamic

Table 1 Halobacterial transducer proteins: transducer proteins (Htrs, MCPs) from *H. salinarum* and *H. volcanii* are listed, together with their protein partners

htr number	Gene	#TM	Code (locus tag)	Partner	Stimulus	Reference
htr1	htrI	2	OE3347F	sopI	Attractant: orange light; repellent: UV light	[87,94,118]
htr2	htrII	2	OE3481R	sopII	Repellent: blue light; attractant: amino acid (Ser)	[89,95,118]
htr3	basT	2	OE3611R; HVO_0554	basB	Branched and sulfur-containing amino acids (Leu, Ile, Val, Met, Cys)	[83]
htr4		2	OE2189R	–		
htr5	cosT	2	OE3474R	cosB	Compatible osmolytes of betaine family	[84]
htr6		2	OE2168R	bdgProt		
htr7		3	OE3473F; HVO_1999	3TMprot		
htr8		6	OE3167F; HVO_1779	–	Attractant: oxygen	[90]
htr9		0	OE2996R	–		
htr10	hemAT	0	OE3150R; HVO_1484 + HVO_1126	–	Repellent: oxygen	[93]
htr11	car	0	OE5243F	–	Arginine	[92]
htr12		0	OE3070R	–		
htr13		0	OE2474R	–		
htr14	mpcT	2	OE1536R; HVO_0420	–	Membrane potential	[91]
htr15		0	OE2392R; HVO_0555 + HVO_3005	arlD (flaD)		
htr16		2	OE1929R	–		
htr17		3	OE3436R	3TMprot		
htr18		2	OE2195F	bdgProt		
htr36		2	HVO_2214	–		
htr37		2	HVO_2462	CBSdom		
htr38		0	HVO_2220	–		
htr39		1	HVO_0969	–		

Locus tags starting with OE are from *H. salinarum* strain R1 [96], and those starting with *HVO_* are from *H. volcanii* [97]. Partners are encoded in the same operon or gene cluster. 'bdgProt' refers to partners which belong to the ABC-type transport system periplasmic substrate-binding protein superfamily. '3TMprot' refers to an uncharacterized partner having three TM domains, while the MCP also has three TM domains. Htrs with both types of locus tags refer to ortholog sets. Htr numbers htr36–htr39 refer to Htrs from *H. volcanii* which do not have an ortholog in *H. salinarum*. Htr numbers between htr18 and htr36 are assigned to other species (e.g. *Natronomonas pharaonis*).

range of the duration of the stimulus as well as the intensity. This well-studied phototactic response is one of the use cases for systems biology applications [20,106,107].

The archaeal signal transduction cascade

Significant studies of the archaeal chemotaxis proteins remain confined to a few archaea: *H. salinarum* and recently also *Haloferax volcanii*. Archaea move by alternating forward and reverse swimming motility as

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facilitated by clockwise (CW) and counter-clockwise (CCW) rotation of the archaellum [8,9,31]. This mode of swimming behavior is different from *E. coli*, which involves tumbling, and instead appears more similar to the activity displayed by other bacteria such as *V. alginolyticus* [10]. In the absence of stimuli, *H. salinarum* and *Haloferax volcanii* cells were shown to perform a random walk [108,109]. As in bacteria, the concentration of CheY-P determines the switch frequency of the motility structure in archaea. In the absence of CheY, *H. salinarum* and *Haloferax volcanii* cells swim preferentially forward [17,108]. This may imply a bias for CW rotation in the absence of switching.

The protein composition of the *H. salinarum* and *Haloferax volcanii* chemotaxis systems (containing CheA, Y, W1, W2, R, B, C1, C2, C3, D, F1, and F2, or CheA, Y, W, R, B, C, D, F1, and F2, respectively) are more similar to the extensive set of *B. subtilis* than to the streamlined version of *E. coli* [15]. The individual role of some of these chemotaxis components has been genetically analyzed in *H. salinarum*, and their roles in taxis confirmed [17]. Deletion of *cheB* abolishes chemotaxis and results in cells with a high frequency of reversals, which is similar to phenotypes observed for an equivalent mutation in bacteria such as *E. coli* and *B. subtilis* [17]. While reversals were more frequent, the proportion of time spent in either CW or CCW rotation was still 50 : 50, similar to wild-type *H. salinarum*. This situation corresponds to that in *B. subtilis ΔcheB*, but is different from *E. coli ΔcheB*. A proteomic analysis showed that transducer methylation is absent from a Δ*cheR* mutant while overmethylation was encountered in a Δ*cheB* mutation, confirming their homology-assigned functions [56]. *H. salinarum* CheB is also involved in glutamine deamidation, which is equivalent to the dual activity of the *E. coli* enzyme, even though *H. salinarum* encodes a homolog of the *B. subtilis* glutamine deamidase CheD. Together, the CheB/CheR action is important for signal adaptation [110].

Archaeal chemotaxis operons also encode CheC/CheD homologs, in addition to the CheB/CheR adaptation system, reminiscent of the situation in *B. subtilis*. Furthermore, CheC and CheD from the euryarchaeon *Pyrococcus horikoshii* were identified as interaction partners in a large-scale protein interaction study [111]. Deletion of *cheC1* (previously *cheJ*) in *H. salinarum* led to reduced chemotactic activity, a lower frequency of reversals and the ratio between CW : CCW rotation was perturbed to 88 : 12, which is quite similar to the reported outcome in *B. subtilis* [17]. Nevertheless, none of the catalytic functions assigned to the bacterial homologs have yet been confirmed in *Halobacterium* [56].

Deletion of *cheY* and *cheA*, encoding the two core proteins of the two-component sensory system, have also been studied in *H. salinarum* [17,38]. These experiments showed that even though these archaeal chemotaxis proteins are homologous to those of *B. subtilis*, they lead to different effects on motility. Deletion of *cheY* and *cheA* genes in *H. salinarum* results in a phenotype whereby cells rarely switch swimming direction and progress in a straight trajectory, similar to the behavior seen in the deletion strains of *E. coli*, but notably different from the situation in *B. subtilis* [17,38,112,113]. Increasing concentrations of CheY-P lead to CW rotation in *E. coli* and *H. salinarum*, but conversely, result in CCW rotation in *B. subtilis* [15,17,114,115]. It seems that in archaea, like in *E. coli*, CheA is activated upon repellent binding, while in *B. subtilis* CheA activation occurs after attractant binding [15,17,114]. It was shown that CheA is responsible for CheY phosphorylation in *H. salinarum* [38]. CheY phosphorylation is strictly coupled to CheA dephosphorylation and, consequently, the half-life of archaeal CheY-P is probably very short [38]. In addition, the importance of the conserved CheY phosphorylation site was demonstrated by mutating the conserved aspartic acid (D53) of CheY in *Haloferax volcanii*, which resulted in a complete loss of chemotaxis [108]. Alignment of bacterial and archaeal CheY amino acid sequences indicated a high conservation [108]. Indeed, the recently resolved crystal structure of *Methanococcus maripaludis* CheY showed that the overall structure of the protein is very similar to that of bacterial CheY [108]. Specifically well conserved are the residues that are involved in the activation of CheY, such as those important for phosphorylation and for Tyr-Thr coupling, which is required for structural rearrangement during activation [108]. Mutation of these residues in *Haloferax volcanii* indicated that also in archaea, they are important for activation of CheY. In conclusion, archaeal chemotaxis proteins function in a similar fashion as their bacterial homologs, since the phosphorylation-dependent activation mechanism is conserved between them.

Communication between the archaeal motility machinery and the chemotaxis system

Despite the conservation of the bacterial and archaeal chemotaxis system, the dramatic differences in the two disparate motility systems pose the interesting question of how the chemotaxis system interacts with the

archaeal motility structure (Figure 1). As components of the bacterial switch complex, such as FliM, are absent from archaea, CheY-P in archaea must have a different binding partner. A protein pull-down approach of different chemotaxis proteins in *H. salinarum* indicated that CheY can interact with two proteins, named CheF1 and CheF2 [116,117]. These proteins can additionally bind euryarchaeal-specific archaellum proteins, ArlD and ArlCE (previously named FlaD and FlaCE) [116,117]. Thus, the homologous CheF1 and CheF2 proteins were suggested to represent adaptors to connect the archaellum with the motility machinery [116]. Indeed, deletion of CheF1 in *H. salinarum* and *Haloferax volcanii* resulted in cells impaired in chemotaxis, with a low frequency of reversals and an almost 100% CW rotation bias [108,116]. CheF1 is conserved in almost all archaea with a chemotaxis system and its gene is usually located within the *che* operon. A duplication of *cheF1* probably gave rise to *cheF2*, which is only present in a few haloarchaea [116]. Deletion of *cheF2* had just a mild effect on chemotaxis [108,116]. *In vitro* interaction assays between purified CheY and CheF of *M. maripaludis* showed that phosphorylation of CheY is important for interaction [108]. Moreover, the crystal structure of archaeal CheY showed that, although there is a high conservation of the protein fold, some residues in the archaeal α-4 helix were, in contrast with bacterial CheY, carrying a prominent negative charge. These residues were mutated in *Haloferax volcanii* and *in vivo* analysis showed that cells carrying these mutations had impaired chemotaxis and cells displayed an increased frequency of reversals of rotation compared with the wild type [108]. Moreover, *in vitro* binding studies showed that mutation of these residues diminishes the binding affinity between CheY and CheF. Interestingly, it was shown that CheC2 in *H. salinarum* interacts with both CheF adaptor proteins, instead of a direct interaction with CheY as is the case in the bacterial system [72,116,117]. This again indicates the important role of CheF in archaeal chemotaxis.

Thus, the structure and activation mechanism of archaeal and bacterial CheY is highly conserved. The only additional requirement for connection of the chemotaxis system to the archaeal motility machinery seems to be a slightly different surface charge of archaeal CheY and the presence of the adaptor protein CheF. The structural organization and composition of the archaeal switch complex has not been elucidated yet.

Future perspectives

Archaea, like bacteria, perform tactic behavior. In the model euryarchaea, *H. salinarum* and *Haloferax volcanii* taxis relies on the chemotaxis system, which might have been obtained from bacteria (most likely from *Firmicutes* or *Thermotogae*) by horizontal gene transfer [16,37]. The available molecular and genetic analyses of the haloarchaeal chemotaxis system indicate a high similarity with that of bacteria. However, archaeal chemotaxis is far from being completely understood. The structural differences between the archaellum and flagellum necessitate subtle changes in the archaeal response regulator CheY as well as an adaptor protein for connection to the archaeal motility machinery. The structure of CheF and its cellular localization have yet to be elucidated. CheF might be cytosolic or found semi-permanently bound to the archaellum motor. The composition and structural organization of the archaeal switch complex will be an important research topic to understand the functioning of the archaeal chemotaxis system. In recent years, studies on the cellular positioning of bacterial chemosensory arrays and flagella have provided major insights into the regulation of bacterial cell shape and organization. The current development of stable archaea-compatible fluorescent proteins is expected to finally allow the first direct observation of the positioning of the archaeal chemotaxis machinery and to open an exciting new line of research.

A major remaining mystery in this field is the apparent absence of a functional chemotaxis apparatus in several archaeal phyla. Although these are readily identifiable in the euryarchaea and thaumarchaea, these systems have not been found in other phyla, including the crenarchaea. Representative species of almost all archaeal phyla encode the archaellum. As a motility structure is especially useful in combination with a sensory system, there is the exciting possibility that crenarchaea might possess an undiscovered sensory system, developed independently from the classical chemotaxis system.

Summary

- To direct their movement towards favorable growth conditions, a process named taxis, microorganisms use a motility structure and a sensory system.

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- The motility structures of archaea and bacteria, archaella and flagella, respectively, have a fundamentally different composition and structural organization.
- Bacteria and some archaea use the chemotaxis system to direct their movement.
- The available experimental data on archaeal chemotaxis proteins suggest that they function similarly to their bacterial counterparts.
- The euryarchaeal chemotaxis system is more closely related to that of *Bacillus subtilis* than to that of *Escherichia coli.*
- The chemotaxis proteins involved in communication with the motility structure are specific for either the bacterial or archaeal motility structure. Initial insights into the connection of these systems in archaea have emerged only recently.

Abbreviations

BR, bacteriorhodopsin; CCW, counter-clockwise; CW, clockwise; HR, halorhodopsin; Htrs, Halobacterial transducer proteins; MCPs, methyl-accepting chemotaxis proteins.

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Competing Interests

The Authors declare that there are no competing interests associated with the manuscript.

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